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PHYSIOLOGICAL PARAMETERS AND PHYSICAL ACTIVITY DATA FOR EVALUATING EXPOSURE MODELING PERFORMANCE: A SYNTHESIS

Thomas McCurdy
Exposure Modeling Research Branch
Human Exposure and Modeling Division
National Human Exposure Research Laboratory
Research Triangle Park NC 27711

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Foreword and Acknowledgements

I hope that this report provides the basis for evaluating selected critical outputs of EPA's exposure/intake dose models. It represents about two years of part-time work, the last one working as an unpaid "guest researcher." Even so, I do not view this as a finished job, as frankly I ran out of steam. I also do not consider it to be a "labor of love;" the tedious nature of the work precluded that, but I was almost compelled to do what I could, as no one else here at EPA would be able to undertake the task for a number of reasons. I have long felt that EPA exposure modelers should undertake more evaluation of the Agency's exposure/intake dose model performance than is typically done. This report, I hope, will help in that regard.

I acknowledge the contributions that EPA colleagues have made to improving to the APEX and SHEDS models over the years. They are Dr. Janet Burke, Dr. Stephen Graham, Dr. Kristin Isaacs, John Langstaff, Ted Palma, Harvey Richmond, and Dr. Jianping Xue. The long-term, fundamental involvement with APEX and its predecessor models, NEM and pNEM, is also acknowledged. The people who were so involved the longest were Jim Capel, Ted Johnson, Roy Paul, and Luke Wijnberg. Ted Johnson should be singled out in this regard since he generally led the contractual work that constituted early exposure/intake dose model development and numerous applications. More recent contractor involvement involved staff of Alion Technology (and ManTech before that), they include: Dr. Glen Graham, Dr. Kristin Isaacs, Yeshpal Lakadi, and Dr. Luther Smith, and Casson Stalling. Drs. William Biller and Thomas Feagans are recognized for their fundamental contributions to the logic of exposure modeling as an integral part of a probabilistic, time-series, risk assessment process suitable for EPA's NAAQS-standard review/setting activities. I also thank John Langstaff and Ted Palma of the Office of Air Quality Planning and Standards (OAQPS) for reviewing this report and providing comments that improved the final project.

Several student contractors assisted me with basic data-gathering and review of published papers that contributed to many of the detailed tables sprinkled throughout this report. They were: Jennifer Hutchinson, A'ja Moore, and Melissa Smart. It was fairly tedious work that they were given, and they did it with aplomb. Ms. Kriti Sharma developed tables of resting metabolic rate that were not used here; both she and Ms. Hutchinson were good at developing the "spider diagrams" mentioned in Section 1, the logic of which was used on various sets of papers contained in this report.

Finally, I thank HEASD management for letting me be a guest researcher so that I could get the report to its current state, that--while not totally complete---can be used by exposure/intake dose modelers as a starting point, at least, for evaluating model performance. The managers most involved were Drs. Timothy Buckley and Roy Fortmann. Dr. Fortmann expedited report publication. I again thank Dr. Kristin Isaacs for being my project leader as a guest researcher. Her patience with bureaucratic procedures during my guest worker tenue is commendable.

Limitations

Citations to journal article titles generally follow ISSN (International Standard Serial Numbering) conventions, but deviate in a few ways. I usually use at least a 4-letter abbreviation for country names, rather than the 2-3 letters often used. Thus, for instance, *American* is abbreviated Amer. rather than Am. Also, single-word journal titles are always spelled out rather than being abbreviated (e.g., Ergonomics rather than "Ergonom.").

Listing of multiple-author articles in the References seemingly follows a random pattern: sometimes only one author's name is provided, other times all co-author names are included. Probably the main reason for this is that many people have worked on my bibliography over the 20 years that it has existed, and different people put entries into the list in their own way. (I did not stress uniformity, as I could deal with the differences, and was glad for the "outside" assistance.) I certainly did not want to take the time to redo the bibliography by following a strict rule (such as including all authors up to three, followed by et al., unless there were four authors total and then all four would be included, etc.). In addition, given that there were so many citations in my bibliography, I tried not to make any single citation take up more than two lines of 8-point type. All references used in this report should be available in Room E253 of EPA's Office of Research and Development, either as a paper copy (in whole or in part) or as a PDF file, so they are easy to obtain and standardize (if desired).

The intent of this report is to present data, and not to formulate testable hypotheses, etc. Thus, there is little speculation about the etiology and functioning of the physiological parameter for which data are provided. Such explanatory variables as body composition, race or ethnic origination of subjects, lean body mass, and the like are not presented or discussed in any detail. Race/ethnicity does not seem to be very important causes of differential basal metabolism and other physiological parameters. Being overweight or obese, on the other hand, greatly affects

those parameters—even on a per kilogram basis—and are mentioned where appropriate. Being overweight, obese, or having health issues are treated basically as "gating variables" in some of the tables presenting cohort-specific data (see Tables 1-2, 6-10, 12, and 25).

Even though there is a lot of information and data contained in this report, I got "burned out" after about a year as a guest researcher on this project. While I consider many parts of the report to be synoptic and dispositive, I did cut corners on some Sections. I did not do a synoptic review of the following topics/Sections: $METS_{MAX}$ in Section 6, physical activity levels of asthmatics (Section 9), and—in particular—activity-specific energy expenditure estimates (EEACT) in Section 11. The latter are needed to improve upon the METS distribution data in CHAD (and in a larger sense, data in the exercise physiologist's Compendium of physical activities that are the basis for CHAD's EEACT estimates). I feel badly about not doing more on intra-individual variability inherent in every physiologic and metabolic parameter that underlies our intake dose modeling procedures. I was going to attempt to rectify that shortcoming by back-calculating intra-individual variance from papers that provide both an ICC and inter-individual variance statistics, but those data are rare and difficult to obtain. I believe that this would be a fruitful effort for someone to attempt, although much more valuable would be to undertake de novo longitudinal studies designed explicitly to address both intra- and inter-individual variability in physiological and metabolic parameters.

I also regret the lack of statistical analyses and graphical display of the data contained in the report. While I did more analyses than I include here, it was taking me too long to do what others here at EPA can do quickly and very efficiently. If there is any interest in doing these tasks, the major data tables are in Excel and can easily be migrated into a standard statistical package and analyzed/displayed. (It will take a bit more work to separate standard deviations from their means in Tables 6, 8, and 10 so that statistical analyses can be undertaken, but that really is a "mechanical" task.) The content of this report really should be based upon formal meta-analyses of the important physiologic and time use variables used in our models, explicitly accounting for possible causal attributes of the variables, and inversely weighting the sample means by their sample size (among other statistical techniques needed to address unequal sample variances and non-random sampling). I have neither the time nor expertise needed to do such a task. Hopefully someone will want to undertake that job.

Future Contact

Since my plan is to finally really retire after this report is released (and a short "fun" paper is written), I won't be around to answer questions or address concerns. I probably--and eventually (by that I mean the response may be delayed if I am out of town)--can be reached at 919-383-3052 or landtmccurdy@gmail.com. Feel free to contact me related to anything in this report. As mentioned, all the studies cited in the report presently are available in E253 as a paper copy (in whole or in part) or as a PDF file. Since there is a lot of useful information in those papers/files, I hope that EPA makes some effort to save this material (unlike what happened when I formally retired and all my computer files were removed by "user support".) I still have not intellectually recovered from that event; I lost a lot of information because of the unnecessary and unexpected dumping of those files.

Disclaimer

This report was independently conceived and authored by a "guest researcher" to the U.S. Environmental Protection Agency (EPA). It has been subjected to Agency review and has been approved for publication. Nothing in it should be construed to represent Agency policy. The mentioning of commercial product names or services does not imply endorsement by EPA.

1.0 Introduction

The purpose of this report is to develop a database of physiological parameters needed for understanding and evaluating performance of the APEX and SHEDS exposure/intake dose rate model used by the Environmental Protection Agency (EPA) as part of its regulatory activities. The APEX model is the Air Pollution Exposure Model and SHEDS is the Stochastic Human Exposure and Dose Simulation model. APEX is used by both EPA's Office of Air Quality Planning and Standards (OAQPS) and the National Exposure Research Laboratory (NERL) in EPA's Office of Research and Development (ORD), while SHEDS mostly is used by ORD. Both models have been used by non-EPA organizations.

In spirit this paper follows the intent of *Data Sources Available for Modeling Exposures in Older Adults* (McCurdy, 2011) but is expanded to include all age groups. Because a nationally-applicable repository of data for physiological factors does not exist, we looked to the clinical nutrition and exercise physiology literatures for relevant information on age- and gender-specific variables used in our exposure models. Much of these data come from "panel" or convenience studies of specific population subgroups, many of them focused on people with a health or weight issue. Since these studies—especially newer ones--often include a similar age/gender control group of approximately the same sample size (*n*), information for "normal" subjects also is reported. Control groups generally are defined to be subjects with no *known* health/weight issues *relevant to the topic being investigated*, which of course is not the same as having no physical problem(s) or being healthy, although study authors often labeled them as such. In our data tables, we generally used the original author(s) delineation of the tested groups that was identified even though the designations often were not precise.

Combining data from disparate studies having different objectives and using a variety of protocols and subjects results in considerable uncertainty regarding general applicability of the information gathered. A formal meta-analysis of the data often is attempted in that situation (Egger & Smith, 1997; Egger et al., 1997), but that is not possible to undertake at the present time. Perhaps this compilation can become the basis for such an effort, since it provides for each study its group mean, standard deviation (where possible), and its sample size. (Where only standard errors of the estimate [SE] are provided in an article, they are converted to SD by multiplying by square root of the sample size: $SD = SE * \sqrt{n}$.) Additional information would have to be obtained for each study, however, in order to undertake a complete meta-analysis of data contained in this report.

Single-gender data are emphasized in this report. Rather large differences in oxygen consumption and ventilation rate measures by gender are seen in the literature for the same age cohort as a perusal of Tables 1 & 2 indicates. Rowland et al. (1997) discusses the reasons for these differences at some length. The same disparity is seen in maximal ventilation rates (Section 4), daily energy expenditure (Section 8), and time spent in moderate/vigorous physical activity (Section 10). Combining data from females and males results in an average value being presented that does not reflect characteristics of either group, even on a per body mass basis. As a general statement, $VO_{2.MAX}$ and $V_{E.MAX}$ on both an absolute and relative body mass basis is higher in males than in females for the same age grouping and fitness level, as might be expected due to larger lung and oxygen-carrying capacities sizes in males. Since many studies in exercise physiology and clinical nutrition combine data from females and males, particularly for young children and older adults, ignoring gender removes over 100 papers from our database. In the future, probably mixed-gender studies could be utilized for children <8 y old or so, but physiological changes associated with older children and going through puberty definitely affect a number of important physiological parameters for boys and girls at different rates (Rudroff et al., 2013).

Literature Search Procedures

Tabular data in this report are only from U.S. studies unless otherwise noted. Non-U.S. papers are used mostly to document statements concerning theory, relationships, and concepts. The focus on U.S. studies is due to (1) important cultural aspects of diet and physical activity patterns that affect some of our variables of interest, and (2) who we are: a U.S. governmental agency (EPA). While we believe that human physiological *relationships* generally are similar among all people regardless of culture or country of origin, there is a cultural and geographical component of diet and time use behavior that is societal-specific. Even resting metabolism and body composition metrics show cultural influences, although "developed" countries across the globe in general are seeing similar rates of obesity and inactivity (Andersen et al., 2003). Physical activity patterns in a population are greatly affected by social factors, including occupational type, educational level, and income (Welk, 2002). Geographic and climatic factors also affect physical activity patterns. Certainly the use of time is culturally dependent (Robinson 1977). Because we are a U.S. governmental organization working *only* on analyses affecting our country's population, it is prudent for

us to focus entirely on characteristics and lifestyles of U.S. citizens. There are copious amounts of U.S. data for almost every physiological component included in our exposure model, so we don't have to "go abroad" to obtain sufficient relevant data.

The information presented here is from a literature search designed by staff of EPA's library in RTP, NC. The review strategy was developed by Ms. Susan Forbes of the University of North Carolina's School of Library Science. The search was first undertaken in 2005 and focused on 1990-2005 papers, and has been repeated periodically ever since. Non-English papers were included if they provided unique information on a physiological relationship (as opposed to measurements). Even then they had to be in Spanish, Portugese, or French, which could be read locally.

Databases searched originally included Dissertation Abstracts Online, EMBASE, ExtraMED, MEDLINE, PASCAL, SciSearch and 19 others. Only MEDLINE has been used since 2010. Abstracts produced by the search were reviewed using a philosophy of rejecting only obviously inapplicable articles (presenting non-U.S. data; data from a study occurring in a confined living or experimental chamber; or being documented only in an abstract or a conference presentation). Articles that made it through this stage—about 73% of them, were reviewed and further culled if they were simply a review of other papers--presenting secondary data--or were "redundant" (discussed below). Over 5,000 articles or books were reviewed at this stage of the process.

After narrowing our search results as described, we excluded studies providing "redundant" data on a particular topic. Redundant data is the same numerical information for a particular parameter that appears in more than one paper. We have frequently identified up to four different papers that have published essentially the same data, often with different first authors and appearing in different journals (e.g., the work of Pollock and colleagues). Institutional affiliation of the first author sometimes changes from paper-to-paper, as does the order of authors. Occasionally, there are slight differences in the sample sizes used, as later articles usually—but not always--include more people in a study. Thus, it is difficult to separate out papers with unique data from multiple papers describing the same measurement study, particularly because study locations and date of the measurements are often not provided. Including the same data from multiple papers would distort "real" variability inherent in a physiological parameter, making it seem less variable than it is. Egger and Smith (1998) call this "multiple publication bias." We tried to minimize multiple listings of the same data by devising a "spider web" of all authors involved in a paper (a diagram linking all authors of possibly redundant data papers), checking details of the clinical testing protocol used and its frequency of testing, variables obtained, etc., and (infrequently) by contacting first authors to ascertain if their data were published in other papers. That last effort was not very fruitful due to the difficultly of obtaining current contact information, lack of response to our emails, and—frankly—lack of candor on the part of some authors. Over 50 papers were removed from our review due to perceived redundancy. Even so, we think that some data presented in more than one paper has crept into this report. Hopefully these data will not significantly distort our assessment due to the sheer number of the studies included in our data base.

Finally, 17 papers first-authored by Dr. Eric T. Poehlman were removed from our review due to their being retracted from the scientific literature as a result of a fraud investigation undertaken by the U.S. Department of Health and Human Services, who funded much of his work (Wikipedia, 2007).

The 4,000 or so papers that remained after these efforts were read and *some* of their cited references were obtained for further review even if they were published before 1990. Thus, inclusion of pre-1990 data in this report is rather eclectic, depending more on availability and personal interest than rigorous adherence to a search strategy. Of the papers reviewed, 49.0% of them were rejected due to (1) presenting only "absolute" data (i.e., non-V_E data were provided without being on a body mass-specific basis: see below), (2) providing only "mixed gender" data (data were not separated by sex); or (3) measuring data using a "non-conventional" protocol (e.g., oxygen consumption data not coming from either a treadmill or a cycle ergometer, but from an arm cranking protocol). The use of alternative protocols usually results in physiological parameter estimates being significantly different than those obtained using generally-accepted methods. As an example of the measurement problems associated with different protocols, see Appendix A for a discussion of the oxygen consumption testing protocols used by different researchers. As can be seen there, there are a number of ways in which data are obtained for physiological parameters of interest to us.

When a paper provides data from a "before-and-after" experiment or trial, only the baseline (pre-experiment) data are presented here. No post-exercise improvement in oxygen consumption or fitness, etc. or any other "after" data appears are used in this report.

With respect to temporal changes that may occur in physiological parameters of interest, such as changes in time spent in physical work or exercise (or changes in anthropometric factors such as body mass), we formally evaluated change over time for those parameters where we had enough data to do so. Undertaking these temporal change analyses is described in context of the parameter being discussed. We could not formally evaluate temporal change in most of the parameters used in our exposure models due to a lack of longitudinal studies on most topics.

Although we use data from both clinical nutrition and exercise physiology studies, there is a dichotomy between these disciplines on the emphasis placed on VO_2 measurements in their work. Generally, exercise physiologists (and cardiologists) measure $VO_{2.MAX}$ in their subjects, but not resting oxygen consumption ($VO_{2.REST}$). Nutritionists, on the other hand, usually measure $VO_{2.REST}$, which is often reported as basal metabolism in

energy expenditure units (EE in kcal kg^{-1} min^{-1}), but do not measure $VO_{2.MAX}$ (Patterson et al., 2005). This dichotomy has resulted in a lack of emphasis in the literature on *reserve* metrics—the difference between minimum and maximum physiological states--and a loss of a "bounded physiological anchor" in much of exercise and nutritional work. This point will become clearer later. Basically, use of reserve metrics allows the analyst to construct activity-specific energy expenditure (EE_A) work rates relative to both of a person's physiological limits (lower and upper), thus fostering more relational stability among parameters. This is one of the fundamental points of this report.

Conventions Used in the Report Concerning Metrics

Units and metrics in exercise physiology are tedious to express in *Word*. Most importantly, since Word does not allow you to overstrike symbols except in "Equation Writer," a number of normal notational conventions are **not** used here. In the biological sciences, "rate" metrics—those involving *time* in the denominator—are presented *with a dot over the parameter:* usually a "*V dot*" for ventilatory-oriented metrics. Such a convention cannot be done "gracefully" in Word. Not using the dot convention might confuse readers conversant with human physiological studies since *volume* metrics often use the same letter symbol but have no dot over them. Thus, not using dots over rate variables means that we cannot differentiate symbolically between *rate* metrics and *volume* metrics. As a general statement, we *only present rate metrics* in this report. If a volume metric is discussed, we make it clear that volume, not rate, is the parameter of concern.

Along that line, we only utilize VO_2 and most other physiological parameters on a **per-body mass** (BM) basis—which rigorously should be depicted as $VO_{2/BM}$ or $V_{E/BM}$, etc. We consistently shorten the metric to just VO_2 or V_E to reduce subscripts. Thus, $VO_{2.MAX}$ in this report, unless otherwise noted, really is maximal oxygen consumption *per body mass* having units of mL kg^{-1} min^{-1} (also cited as mL/kg-min in the Tables since superscripts cannot be depicted in Excel). We focus on body mass-adjusted physiological metrics, as doing so reduces—but does not eliminate--gender and age variability in most parameters, and allows a more intuitive comparison of the parameters for disparate population groups. However, it is well known that BM-normalized metrics are not without problems themselves (Vanderburgh & Katch, 1996). Rowland (1996) succinctly enumerates the limitations associated with per-body mass "ratio" metrics. He says that there is no "universal standardizing factor" devised that allows an analyst to definitively compare population subgroups with respect to aerobic capacity and most other physiological measures, especially for children as they develop over time (Rowland, 1996). That being said, however:

> *"…Body mass is the dimensional measure adopted by comparative biologists as the usual standard for physiologic comparisons—assumed in this discussion to be equivalent to body weight when subjects are in the same gravitational condition…" (Rowland, 1996; p. 22).*

See also Rowland (1991) for an interesting discussion of "normalizing" oxygen consumption for use in exercise physiology research. We also never present physiological data using body surface area (BSA) as a normalizing metric because there is no biological reason why doing so improves generality of the parameter, especially for children (Livingston & Lee, 2001; Rowland, 1996). Most of the body's energy expenditure is used to keep the brain and other body organs functioning, which are not a function of BSA (McCurdy, 2000). The "hidden" BM convention used in this report for VO_2 estimates is **not** used for ventilation—or breathing rate--metrics (V_E). V_E data usually are presented in the literature only in absolute terms, with units of L min^{-1} (L/min).

Rarely do we discuss lean body mass (LBM)-adjusted metrics because of the dearth of information available to exposure modelers regarding population-level LBM measurements. (LBM is often called "fat-free mass" [FFM] in the literature, but there are subtle differences in meaning, so that term is not used here.) On occasion, BM to the 0.67 or 0.75 exponent will be discussed, as that adjustment--also called "allometric scaling"—often reduces inter-individual variability in many physiological and pharmacokinetic variables (Nevill, 1994, 1997). When so discussed, the full metric and units will be used: e.g., $VO_{2/BM}^{0.67}$ and mL $kg_{BM}^{-0.67}$ min^{-1}.

Resting Energy Expenditure (REE); Resting Metabolic Rate (RMR); Basal Metabolic Rate (BMR)

Resting energy expenditure (REE), variously called resting metabolic rate (RMR) or basal metabolic rate (BMR), is an important physiologic metric, as will become abundantly clear in the discussion below on METS (metabolic equivalents of work). When RMR is discussed as a rate, we use units of kcal kg^{-1} d^{-1} in this report. Again, the per-BM subscript is to be implied. When basal metabolism data are presented or discussed in conjunction with **daily total energy expenditure** (DTEE) its units are kcal d^{-1}, the same as used for DTEE. In this case, we label basal metabolism as REE, resting energy expenditure to hopefully minimize confusion. Basically, REE = RMR * 1,440 minutes, which implies that BMR does not vary within a day. While this is known to be incorrect as there is a circadian pattern to RMR data (Reilly et al., 1997, 2000), there is no practical way to measure BMR over an entire day except for comatose, hospitalized patients or inactive people confined to a direct calorimeter. REE data for selected special cohorts appear in Tables 13-15, 16, 18, and 20.

Some authors distinguish between BMR and RMR based upon different measuring protocols used to ascertain resting energy expenditure, but we treat them as synonyms. It is

impractical to *not* do so, since the terms are used rather interchangeably in the nutrition literature, and analyses of possible practical *differences* among them are rare. RMR usually is measured by oxygen consumption techniques (indirect calorimetry) and often is reported in units of mL VO_2 kg^{-1} min^{-1}. Infrequently RMR is measured directly in a calorimeter based on temperature change measurements and is reported in units of kcal kg^{-1} min^{-1}, but doing so is rare.

Even though REE/RMR/BMR is a fundamental physiological metric, we do not present specific tabular data for this parameter, however measured, in this report. The reader interested in data on basal metabolism is directed toward McCurdy & Graham (2006) or the vast literature that exists on the topic. A recent one is McMurray et al. (2014), which presents BMR data from 197 studies published between 1980 and 2011. There literally are hundreds of prediction equations for RMR available in the literature. Many compare predictions from one equation (or sets of equations) to others, usually developed anew in the paper cited. Most papers find that existing equations do not adequately work for certain age/gender subgroups or those cohorts with a physical or mental handicap. Shortcomings with the Schofield (1985) equations used in the APEX and SHEDS models have been extensively noted. The Schofield equations certainly are based upon subjects that are, for the most part, more active than people are currently; and, in addition, his equations are not based upon many North Americans, so the ethnic composition of his subjects is quite different than contemporary United States inhabitants. The Schofield equations used in EPA models should be revised to incorporate updated information concerning REE in contemporary times and ethnic composition.

Intra- and inter-variability in physiological parameters

It is important to address intra-individual variability in exposure assessments to better address longitudinal variability in physiologic and time use parameters. Only in this manner can we address uncertainty due to individual characteristics *per se* in our models (Chikaraishi et al., 2010; Isaacs et al., 2013; Xue et al., 2004). The same is true for physiological parameters. Failure to do so results in incorrect understanding of important dosimetric, metabolic, and pharmacokinetic processes in the human body (Jamei et al., 2009). It also results in downward biased estimates of both the product-moment (Pearson) and rank-order (Spearman or Kendall) correlation coefficients among variables in an association.

One way to account for intra-individual variability is to base the intra-individual COV on the ICC metric obtained from an estimate of longitudinal data for a **set** of individuals using repeated-measures statistical techniques. The ICC metric describes the ratio of between-group variance to total variance (between-group + within-group) explained, and knowing inter-individual variability, you can approximate intra-individual variability in the sample. However, except for studies focused on *reliability* of physiological measurement protocols, there are very little longitudinal analyses of physiological data that allow rigorous characterization of the ICC or intra-individual variability. Even those studies that do investigate temporal changes in physiological parameters in a sample over time—such as the work of Pollock and colleagues (Pollock, 1974; Pollack et al., 1987, 1997)—really only provide "sequential cross-sectional" data rather than individual-specific longitudinal data. The work of Asmussen et al. (1975), Sidney and colleagues (1998) and Van Pelt et al. (1994) also is of this type. While rate-of-change statistics are sometimes supplied for the time periods analyzed, they are on a group-mean basis (Pollock et al., 1997), so an estimate of intra-individual variability is impossible to obtain. This is a major shortcoming of the physiological databases used as input to the APEX and SHEDS models. It also is a major hindrance in evaluating distributional aspects of our model outputs to determine if a "proper" amount of intra-individual variability is adequately captured. Grouped variability in a sample for a physiological parameter can be approximated from cross-sectional data by investigating the sample's coefficient of variation (COV), but individual variability—a major source of exposure modeling uncertainty—cannot.

We try to characterize intra-individual variability in important physiological parameters wherever possible. Unhappily however, there is little out there, as will be seen by reviewing the tables presented in this report.

2.0
Absolute and Relative General Physiological Metrics

There are three generalized physiological metric formulations used in this report, whether or not they are normalized by body mass.

1. *Absolute metrics*, not being relative to any other *physiological* measure. They may, however, be normalized to an *anthropogenic* measure, such as height or weight. An example is activity-specific oxygen consumption ($VO_{2.ACT}$) or activity-specific energy expenditure (EE_{ACT}), with units of mL kg_{BM}^{-1} min^{-1} and kcal kg^{-1} min^{-1}, respectively.
2. *"One-sided" relative metrics,* related to some defined construct, such as maximal level achieved. Examples are $\%VO_{2.MAX}$ or $\%VO_{2.MAX/BM}$. One-sided relative metrics may also be anchored to a minimal level, such as basal or resting metabolic rate (RMR). METS are an example of this type, where activity-specific energy expenditures (kcal min^{-1} or kcal kg^{-1} min^{-1}) are divided by a person's RMR to produce a unitless metric caused by canceling of units. Daily Physical Activity Level (PAI) is another example of a metric being anchored to the basal metabolic rate, in this case the ratio of total daily energy expenditure to RMR needed to support the activities undertaken (McCurdy, 2000).
3. *"Two-sided" relative metrics*, bounded by limits on both the low- and high-end. These generally are called *"reserve metrics,"* as briefly mentioned above and more fully explored below. Reserve metrics retain their original units. Relating exercise data to both resting and maximal exercise limits explicitly adjusts for differences in fitness and age in children, in particular (Logan et al., 2000). Examples are oxygen consumption reserve ($VO_{2.RES}$) which is equal to $VO_{2.MAX}$ - $VO_{2.REST}$, ventilation reserve ($V_{E.RES}$, which = $V_{E.MAX}$ - $V_{E.REST}$), heart rate reserve (HRR, which = HR_{MAX} – HR_{REST}), and $METS_{RES}$ = $METS_{MAX}$ – 1.

Appendix B contains an extended discussion of these three types of general physiological metrics using heart rate as an example. We do not highlight HR in this paper as it is not used in either the APEX or SHEDS models. However, it is the physiological parameter having the most data—probably due to the fact that there is a lot of concern regarding people with cardiovascular problems and how their condition can best be evaluated and treated in a clinical research setting. How the general HR metrics relate to those of more interest to us is also discussed in Appendix B.

3.0
Maximal Oxygen Consumption (VO_{2max})—the "Controlling Parameter"

Overview of Tables 1 & 2

One of the most important parameters of interest to us is $VO_{2.MAX}$. $VO_{2.MAX}$ is alternately known as "aerobic capacity," "maximal oxygen uptake," "maximal aerobic power," or just "aerobic power" (Armstrong, 2013; McArdle et al., 2001). Strickland et al. (2012) call it the "gold standard measure of aerobic fitness." It usually is expressed in units of mL kg^{-1} min^{-1} or mL min^{-1}. It is frequently defined to be the point in an exercise test where oxygen consumption plateaus—or increases only slightly—with increasing work rate. As mentioned in Appendix A, however, many subjects never attain such a plateau, especially children, people with cardiovascular problems, and the elderly (Armstrong, 2013; White et al., 1998), so additional—and usually "relaxed"--criteria of $VO_{2.MAX}$ attainment are used. Some authors then call this measure of aerobic capacity "$VO_{2.PEAK}$" instead of $VO_{2.MAX,}$ but this terminology is not universally used.

In general and for most people, undertaking steady-state exercise at VO_2 levels less than 55-60% of $VO_{2.MAX}$ causes little lactate accumulation (McArdle et al., 2001). Thus, energy can be expended at that rate for relatively long periods of time (4+ h). Increasing $VO_{2.MAX}$ due to an exercise program results in—for a period of time, anyway—the ability to accomplish the same amount of work using less oxygen consumption, and also increases endurance (Åstrand, 1992). These improvements are not solely due to an improved oxygen transport system (increased density and size of capillaries), but also due to an increased use of free fatty acids to supplement glycogen usage, and increased mitochondrial enzyme activity (Åstrand, 1992). A review of important factors that determine maximal oxygen uptake/consumption in individuals is found in Lamb (1984).

VO_2 values, including $VO_{2.MAX}$, generally are obtained using a progressive treadmill or cycle ergometer tests, with good agreement between them on a group basis, but not so good on an individual basis (Bassett & Boulay, 2003). We generally only provide $VO_{2.MAX}$ data for exercise tests using one of these two methods. However, there are many different protocols used for each one of these general approaches, as highlighted in Appendix A.

$VO_{2.MAX}$ data from U.S. studies are presented in Tables 1 (females) and 2 (males) for various *author-defined* fitness, health, or weight categories. We do not provide any $VO_{2.MAX}$ data for "mixed-gender" studies, although a number are reported in the literature. There are statistically significantly differences in $VO_{2.MAX}$, either on an absolute or relative basis, between the sexes at all ages except the very young or old (Armstrong, 2013; Graves et al., 2013; Rowland, 2013; Weiss et al. 2006). Values of $VO_{2.MAX}$ in Tables1 and 2 are provided only as *relative estimates* in terms of mL kg_{BM}^{-1} min^{-1} metrics (mL/kg-min). A reader interested in absolute—non-body mass normalized--$VO_{2.MAX}$ estimates can find them for particular gender/age cohorts in hundreds of published articles in the exercise literature. Likewise, $VO_{2.MAX}$ estimates on a lean body mass (fat-free mass) basis can also be found: e.g., Graves et al (2013).

A paper that presents a summary of $VO_{2.MAX}$ data similar to that presented in the Tables is contained in Smith & Gilligan (1989) for 133 studies. About 30 of the studies in paper are included in Tables 1 & 2. The other studies were excluded here mostly because they were from non-US citizens. Another published source of $VO_{2.MAX}$ data from scores of studies on females is found in Wells (1991). We do not include any $VO_{2.MAX}$ data from the Wells (1991) "meta-analysis" article in Tables 1 & 2, but do include data from some of the U.S. studies used in that article if we could obtain the cited article ourselves. Another published source of $VO_{2.MAX}$ data from scores of studies on females is found in Patil et al. (1993). Other meta-analyses articles of U.S. citizen's $VO_{2.MAX}$ data exist but are not cited here as we only used data from "original sources." An early paper that provides the mean of maximal oxygen consumption by age data *in trained males* from the US, two named countries and "all-other" countries is Shephard (1966). The US data are similar to residents of all the countries except for the Scandinavian data, which has considerably higher grouped $VO_{2.MAX}$ data than the rest-of-the-world, at least prior to age 50. His values for U.S. residents are not significantly different than those shown in Table 1 and 2.

Table 1. Estimates of $VO_{2.MAX}$ in females seen in the literature

Body Mass (BM)-Adjusted Estimates: Mean ± SD VO2Max Estimate (mL/kg-min)							
Age /Range Mean	SD	Health Status	Mean	SD	COV (%)	Citation	Comment
Females: Normal, Healthy, or Not-Specified							
a. Mean & SD statistics are provided for age							
7.8	0.3	N	46.4	2.9	6.3	Livingstone et al. 1992	n=5
7.9	2.7	NS	49.1	6.5	13.2	DM Rogers et al. 1995a	n=15
8.2	1.2	N	45.5	4.7	10.3	Cureton et al. 1997	n=20; mixed fitness groups
8.5	0.8	H	45.3	4.0	8.8	Treuth et al. 2003	n=6
8.7	0.7	N	39.7	8.6	21.7	McMurray et al. 2003	n=403; CA
8.8	0.7	NS	50.2	3.6	7.2	DM Rogers et al. 1995b	n=21
8.8	0.7	N	38.4	9.8	25.5	McMurray et al. 2003	n=103; AA
9.0	2.0	N	38.0	7.0	18.4	Cooper et al. 1984	n=24
9.1	1.5	NS	33.9	2.3	6.8	Gilliam et al. 1977	n=15
9.4	0.5	N	41.9	2.5	6.0	Livingstone et al. 1992	n=4
9.4	1.0	NS	45.4	5.8	12.8	Cureton et al. 1995	n=106; multicenter study
9.8	0.6	H	35.4	7.5	21.2	KE Swain et al. 2010	n=20
9.8	0.7	NS	46.8	7.1	15.2	Loftin et al. 1998	n=19
10.0	0.8	N	38.3	9.1	23.8	McMurray et al. 2003	n=381; CA
10.0	1.8	N	42.5	6.7	15.8	McMurray et al. 1998	n=18
10.1	1.2	N	49.1	3.1	6.3	Chausow et al. 1984	n=3
10.1	0.8	N	37.2	9.8	26.3	McMurray et al. 2003	n=98; AA
10.2	1.0	N	41.0	7.0	17.1	Janz et al. 1998	n=62
10.2	2.8	N	44.8	6.2	13.8	Skinner et al. 1971	n=20; treadmill protocol #3
10.3	0.3	H	42.3	5.1	12.1	N Hopkins et al. 2011	n=70 (Summer only)
10.4	2.5	N	45.7	5.1	11.2	Skinner et al. 1971	n=20; treadmill protocol #2
10.4	2.8	N	43.0	6.9	16.0	Skinner et al. 1971	n=21; treadmill protocol #1
10.6	0.9	N	43.2	8.6	19.9	Iannotti et al. 2004	n=33
10.6	1.4	H	41.4	5.2	12.6	Roemmich et al. 1998	n=12
10.8	0.6	N	49.2	3.5	7.1	Mahon et al. 1997a	n=15; Tanner 1
11.2	1.0	N	40.0	6.0	15.0	Janz et al. 1998	n=61
11.6	1.2	N	54.3	4.7	8.7	Mahon et al. 1997a	n=11; Tanner 2
11.6	2.8	NS	36.7	5.4	14.7	Golden et al. 1991	n=101
12.0	0.8	N	35.0	5.4	15.4	McMurray et al. 2003	n=403; CA
12.0	0.7	N	52.5	5.0	9.5	Peyer et al. 2011	n=55
12.1	0.8	N	33.6	5.8	17.3	McMurray et al. 2003	n=103; AA
12.1	1.0	N	38.0	7.0	18.4	Janz et al. 1998	n=62
12.1	3.5	H	34.9	6.5	18.6	JK Murphy et al. 1988	n=42; CA
12.2	2.7	NS	45.4	7.3	16.1	DM Rogers et al. 1995	n=15
12.5	0.4	N	42.3	3.5	8.3	Livingstone et al. 1992	n=5
12.5	0.7	N	48.1	2.6	5.4	Mahon et al. 1997a	n=8; Tanner 3
12.9	1.0	N	31.9	5.1	16.0	Peyer et al. 2011	n=105
13.0	0.8	N	35.0	6.3	18.0	McMurray et al. 2003	n=349; CA
13.0	1.0	N	45.8	5.9	12.9	Cureton et al. 1997	n=26; mixed fitness groups
13.1	0.8	N	33.5	6.1	18.2	McMurray et al. 2003	n=36; AA
13.1	0.8	N	41.1	6.9	16.8	Boiarskaia et al. 2011	n=74
13.1	1.8	N	40.5	7.6	18.8	Mahar et al. 2011	n=90; validation sample
13.2	0.1	N	36.0	6.8	18.9	Peyer et al. 2011	n=128
13.2	1.0	N	38.0	7.0	18.4	Janz et al. 1998	n=58
13.2	1.5	NS	38.6	7.5	19.4	Mahar et al. 2011	n=36; cross-validation sample
13.2	3.3	H	33.2	5.9	17.8	JK Murphy et al. 1988	n=47; AA

Table 1. Estimates of $VO_{2.MAX}$ in females seen in the literature (continued)

Body Mass (BM)-Adjusted Estimates: Mean ± SD VO2Max Estimate (mL/kg-min)							
Age /Range Mean	SD	Health Status	Mean	SD	COV (%)	Citation	Comment
13.3	5.7	NS	45.0	5.7	12.7	Cureton et al. 1995	n=45; multicenter study
13.4	1.1	N	47.7	5.4	11.3	Peyer et al. 2011	n=13; Tanner 4
13.4	1.6	N	38.5	6.8	17.7	Pivarnik et al. 1995	n=53; CA
13.5	0.8	N	37.3	6.3	16.9	Peyer et al. 2011	n=63
13.7	0.6	H	39.8	4.0	10.1	Grossner et al. 2005	n=10
13.7	1.3	H	39.3	5.1	13.0	Roemmich et al. 1998	n=18
13.7	1.7	N	41.2	5.1	12.4	Peyer et al. 2011	n=32
14.0	0.7	N	33.3	6.4	19.2	McMurray et al. 2003	n=312; CA
14.1	1.0	N	34.0	5.0	14.7	Janz et al. 1998	n=57
14.1	2.2	NS	39.5	4.2	10.6	Drinkwater et al. 1975	n=10
14.2	0.8	H	45.3	4.0	8.8	Treuth et al. 2003	n=6
14.2	0.8	N	31.4	6.5	20.7	McMurray et al. 2003	n=74; AA
14.3	1.8	N	41.5	6.4	15.4	Mahon et al. 1997a	n=13; Tanner 4
14.6	0.5	N	31.4	4.8	15.3	Pivarnik et al. 1998	n=19; AA
14.6	0.7	N	34.6	9.4	27.2	Crowhurst et al. 1993	n=9
15.0	0.6	N	36.0	5.1	14.2	Rowland et al. 2011	n=9
15.0	0.8	N	32.4	6.7	20.7	McMurray et al. 2003	n=297; CA
15.0	2.0	N	34.0	4.0	11.8	Cooper et al. 1984	n=27
15.1	0.8	N	30.3	5.9	19.5	McMurray et al. 2003	n=75; AA
15.2	1.5	N	47.3	5.2	11.0	Peyer et al. 2011	n=57
15.3	1.1	NS	40.4	5.1	12.6	Murray et al. 1993	n=32
15.6	3.4	N	38.2	6.9	18.1	Moffatt et al. 1984	n=13; controls
15.6	0.4	N	38.7	2.9	7.5	Livingstone et al. 1992	n=3
16.2	1.1	NS	34.2	7.0	20.5	Gutin et al. 2005	n=104; white adolescents
16.3	1.2	NS	29.6	6.8	23.0	Gutin et al. 2005	n=121; black adolescents
16.7	1.1	NS	46.0	4.7	10.2	Dill et al. 1972	n=10
16.9	3.0	NS	46.6	6.0	12.9	Loftin et al. 1998	n=?
18.9	0.5	N	31.7	2.5	7.9	Burke 1977	n=8; experimental group
18.9	0.5	N	34.9	5.0	14.3	Burke 1977	n=7; control group
19.1	2.8	NS	37.5	5.9	15.7	Dolgener et al. 1994	n=45; cross-validation group
19.2	6.2	NS	23.4	5.1	21.8	AM Miller et al. 2012	n=13; siblings of survivors
19.4	3.1	NS	36.6	4.7	12.8	Dolgener et al. 1994	n=100; validation group
19.5	1.4	NS	36.6	5.2	14.2	Darby & Pohlman 1999	n=15
19.7	2.4	N	49.2	9.8	19.9	K Sell et al. 2008	n=12; game players
19.8	2.5	N	39.5	6.7	17.0	Kaminsky et al. 1993	n=28
19.9	1.8	NS	36.7	10.2	27.8	Lepp et al. 2013	n=27
20.1	1.6	N	34.0	6.0	17.6	Hu et al. 2007	n=14; group #1
20.3	0.9	H	38.9	4.4	11.3	Deschenes et al. 2009	n=10
20.5	1.6	N	44.2	3.2	7.2	Bransford & Howley 1977	n=10; untrained
20.6	2.0	N	35.8	5.1	14.2	Hu et al. 2007	n=14; group #2
20.8	1.8	H	38.7	4.2	10.9	McComb et al. 2006	n=13
20.8	2.0	NS	37.5	6.6	17.6	Darby & Pohlman 1999	n=63
20.8	3.0	NS	39.0	8.1	20.8	Mole & Hoffmann 1999	n=38
21.0	3.0	N	42.4	10.4	24.5	J Kang et al. 2007	n=11
21.1	3.3	N	44.9	6.9	15.4	Latin & Elias 1993	n=25
21.2	1.0	N	27.9	4.6	16.5	Chitwood et al. 1996	n=11;black subjects
21.5	2.2	NS	39.2	4.2	10.7	JD George et al. 1998	n=49; test of protocol
21.6	2.9	NS	41.6	5.2	12.5	JD George et al. 1996	n=50

Table 1. Estimates of $VO_{2.MAX}$ in females seen in the literature (continued)

Body Mass (BM)-Adjusted Estimates: Mean ± SD VO2Max Estimate (mL/kg-min)							
Age /Range Mean	SD	Health Status	Mean	SD	COV (%)	Citation	Comment
21.7	1.6	NS	44.2	5.6	12.7	Cureton et al. 1995	n=23; multicenter study
21.8	1.3	N	39.3	3.4	8.7	Kaminsky & Whaley 1993	n=5
22.2	3.6	N	29.3	2.9	9.9	Chitwood et al. 1996	n=11; white subjects
22.2	1.8	N	37.5	6.1	16.3	J Kang et al. 1999	n=7
22.3	2.8	NS	32.7	2.9	8.9	Drinkwater et al. 1975	n=10
22.8	3.2	H	38.7	8.7	22.5	Grossner et al. 2005	n=10
23.0	4.0	N	44.2	4.9	11.1	DW Hill 1996	n=12
23.5	5.0	N	34.8	5.3	15.2	Gonzales & Scheur.2006	n-11
23.7	6.6	NS	44.8	7.8	17.4	DM Rogers et al. 1995	n=15
23.8	2.4	N	32.2	6.6	20.5	Ridout et al. 2005	n=12
23.9	3.4	H	49.5	6.7	13.5	Porcari et al. 1997	n=16; control group
23.9	5.6	NS	47.3	8.3	17.5	Mole & Hoffmann 1999	n=44
24.0	3.6	N	36.2	7.0	19.3	S.-C. Chung et al. 1999	n=11
24.3	1.6	NS	34.8	2.7	7.8	Frey et al. 1993	n=7; untrained students
24.5	3.9	N	35.6	4.7	13.2	Kohrt et al. 1998	n=18
24.8	4.8	N	40.1	6.8	17.0	Blessinger et al. 2009	n=26
25.0	2.0	H	45.1	10.5	23.3	Pettitt et al. 2008	n=7
25.0	3.0	NS	34.3	3.8	11.1	Horton et al. 1998	n=6; untrained
25.0	4.9	NS	38.2	8.3	21.7	Swain et al. 1998	n=24
25.0	9.0	NS	40.1	7.2	18.0	Swain et al. 1994	n=81
25.1	4.3	N	36.3	4.6	12.7	Steffan et al. 1999	n=15; normal weight
25.6	4.9	N	40.0	6.1	15.3	BJ Sawyer et al. 2010	n=29
26.3	1.8	H	36.1	7.1	19.7	KJ Melanson et al. 1997	n=8
25.6	1.6	N	56.8	10.7	18.8	K Sell et al. 2008	n=7; not game players
26.6	5.5	N	38.6	4.4	11.4	Browning et al. 2006	n=10
27.3	5.0	N	47.6	6.5	13.7	Ballor & Poehlman 1992	n=13; resistance trained
27.3	5.1	H	30.3	4.7	15.5	Lovelady et al. 1990	n=8; lactating; controls
27.4	5.1	H	42.2	3.1	7.3	Thomsen & Balor 1991	n=8; group 1
27.5	5.1	H	41.0	4.7	11.5	Thomsen & Balor 1991	n=10; group 2
27.8	3.5	H	35.6	7.8	21.9	Dionne et al. 2004	n=19
27.9	5.7	H	31.4	2.7	8.6	Thomsen & Balor 1991	n=9; group 3
27.9	6.8	N	31.0	8.5	27.4	McMurray et al. 1998	n=286
28.0	6.1	N	43.6	7.9	18.1	Sheaff et al. 2010	n=7; firefighters
28.1	4.3	H	39.3	10.4	26.5	Treuth et al. 1996	n=8
28.3	7.7	NS	43.6	1.8	4.1	Foster 1975	?
28.9	7.8	NS	44.6	3.7	8.3	L Kravitz et al. 1997	n=9
29.0	3.5	H	34.3	5.5	16.0	BE Hunt et al. 1997	n=12
29.0	5.0	N	39.9	5.8	14.5	Horton et al. 2002	n=10
29.4	4.4	N	28.8	19.3	67.0	Soultankis et al. 1996	n=10
30.0	3.5	H	46.4	2.4	5.2	Lovelady et al. 1990	n=8; lactating; exercise group
30.0	4.0	H	45.7	2.6	5.7	Kaminsky et al. 1990	n=6
30.0	7.2	N	39.0	4.2	10.8	Horvath & Drink.1982	n=4
30.4	8.2	H	47.5	5.2	10.9	Olson et al. 1991	n=9
30.5	5.0	H	33.0	4.9	14.8	Byrne et al. 1996	n=28
31.0	6.8	N	34.2	5.6	16.4	Horton et al. 1994	n=5; controls
31.8	11.1	NS	36.3	7.9	21.8	Flint et al. 1974	n=7; non-exercisers
32.1	11.7	N	38.8	7.5	19.3	Warr et al. 2013	n=12; pre-deployed NG
33.0	3.0	N	46.8	4.0	8.5	Beidleman et al. 1999	n=8; follicular stage (Note 1)

Table 1. Estimates of $VO_{2.MAX}$ in females seen in the literature (continued)

Body Mass (BM)-Adjusted Estimates: Mean ± SD VO2Max Estimate (mL/kg-min)							
Age /Range Mean	SD	Health Status	Mean	SD	COV (%)	Citation	Comment
33.5	4.9	N	47.7	4.3	9.0	Schraff et al. 1992	n=11
33.6	5.5	N	29.5	4.8	16.3	GR Hunter et al. 2011	n=49; African-American
34.0	6.3	H	47.1	4.6	9.8	Horton et al. 2006	n=11
34.2	2.7	N	30.1	5.7	18.9	Ridout et al. 2005	n=9
34.2	6.3	N	33.7	5.5	16.3	GR Hunter et al. 2011	n=47; European American
34.8	3.4	NS	31.7	3.0	9.5	Drinkwater et al. 1975	n=14
35.1	10.5	H	41.0	8.2	20.0	Engels et al. 1998	n=101
37.5	12.0	N	34.4	5.4	15.7	Nieman et al. 2005	n=15; age range: 20-55
37.6	3.4	N	32.8	0.7	2.1	Shvartz 1996	n=5
40.0	14.0	H	27.6	7.9	28.6	NP Greene et al. 2011	n=25
41.8	5.9	N	33.5	6.2	18.5	Evans 1990	n=20
42.0	14.0	H	30.0	9.0	30.0	Ardestani et al. 2011	n=102
42.4	9.0	N	31.9	7.5	23.5	LT Weir et al. 2006	n=384
42.6	17.6	H	37.8	12.0	31.7	Gardner & Poehl. 1993	n=111; validation sample
43.4	2.9	NS	29.5	2.7	9.2	Drinkwater et al. 1975	n=13
43.4	4.9	NS	23.0	5.7	24.8	Lind et al. 2005	n=23
43.6	2.4	N	26.5	2.7	10.2	Ridout et al. 2005	n=9
43.8	16.0	H	36.4	9.5	26.1	Gardner & Poehl. 1993	n=56; cross-validation sample
45.7	7.9	N	25.0	5.0	20.0	Motl & Fernhall 2012	n=16; controls
48.3	11.6	NS	32.2	7.5	23.3	Kline et al. 1987	n=86; cross-validation group
48.5	11.4	NS	31.4	8.5	27.1	Kline et al. 1987	n=92; validation group
48.6	16.0	H	29.4	7.1	24.1	Fleg et al. 2005	n=375
49.0	4.0	N	27.0	6.8	25.2	NA Lynch et al. 2002	n=18; perimenopausal
49.4	16.3	N	28.6	7.1	24.8	Talbot et al. 2000	n=497; BLSA participants
50.4	6.0	H	29.1	5.4	18.6	Byrne et al. 1996	n=375
50.9	9.5	H	24.1	4.5	18.7	Tosti et al. 2011	n=7; control group
51.5	6.6	H	20.2	3.3	16.3	Duscha et al. 2001	n=11
52.0	2.0	H	22.3	3.2	14.3	NA Lynch et al. 2002	n=18; postmenopausal
52.5	3.4	NS	23.7	3.5	14.8	Drinkwater et al. 1975	n=6
53.0	2.8	N	24.4	6.6	27.0	Ridout et al. 2005	n=10
53.2	4.8	H	28.6	5.0	17.5	JS Green et al. 2001	n=12; exer. no est.replace.
54.3	3.2	N	24.6	4.1	16.7	Johannessen et al. 1986	n=10; exercisers
54.3	8.2	N	33.1	7.5	22.7	Guderian et al. 2010	n=10; in an exercise program
55.4	5.4	N	21.3	7.2	33.8	Johannessen et al. 1986	n=5; controls
55.5	5.1	H	28.0	5.6	20.0	JS Green et al. 2001	n=10; exercisers, est. replace.
56.9	5.1	NS	25.9	4.7	18.1	Stefanick et al. 1998	n=117
57.1	4.3	NS	23.3	4.2	18.0	DR Young et al. 1994	n=160
61.0	3.0	N	26.0	3.0	11.5	Hagberg et al. 2003	n=9
61.0	3.8	H	22.2	4.6	20.7	BE Hunt et al. 1997	n=15; post-menopausal
61.0	4.0	N	22.2	4.7	21.2	Hunt et al. 1997	n=15
62.0	3.0	N	33.4	7.6	22.8	Hagberg et al. 1998	n=22
62.0	6.0	N	23.2	3.3	14.2	Sheldahl et al. 1996	n=9
62.0	6.0	NS	22.8	4.2	18.4	Tanaka et al. 1998	n=9; lean, mostly sedentary
62.0	7.0	NS	21.7	3.3	15.2	Sheldahl et al. 1996	n=11
63.0	5.0	NS	30.1	8.5	28.2	Hagberg et al. 1998	n=22
63.3	2.9	N	21.8	2.6	11.9	Kohrt et al. 1991	n=16; control group
64.0	3.1	N	21.6	2.9	13.4	Kohrt et al. 1991	n=57; experimental group
64.0	4.0	NS	24.3	4.3	17.7	Proctor et al. 2003	n=13

Table 1. Estimates of $VO_{2.MAX}$ in females seen in the literature (continued)

Body Mass (BM)-Adjusted Estimates: Mean ± SD VO2Max Estimate (mL/kg-min)							
Age /Range Mean	SD	Health Status	Mean	SD	COV (%)	Citation	Comment
64.9	2.5	N	19.2	2.2	11.5	Ridout et al. 2005	n=10
65.0	5.0	H	27.0	4.0	14.8	Gonzales et al. 2011	n=21
65.5	7.8	N	16.1	4.8	29.8	Carter et al. 1994	n=16; control group
66.6	4.9	H	22.2	3.6	16.2	Dionne et al. 2004	n=12
67.0	3.7	N	19.4	3.4	17.5	ND Parker et al. 1996	?
67.0	3.9	H	19.3	3.9	20.2	Treuth et al. 1995	n=15
68.0	7.0	n	23.2	5.3	22.8	Pescatello et al. 1994	n=11
68.6	5.7	N	21.9	4.2	19.2	Panton et al. 1996	n=36
70.0	6.1	N	21.5	4.3	20.0	Parise et al. 2004	n=117
70.3	7.3	H	21.3	5.0	23.5	Byrne et al. 1996	n=?
70.4	3.9	N	17.5	2.8	16.0	Sergi et al. 2009	n=81
70.4	6.1	NS	17.6	5.0	28.4	Ainsworth et al. 1993	n=18
70.9	8.1	N	20.3	4.1	20.2	Simonsick et al. 2006	n=46
71.0	3.0	H	22.9	3.7	16.2	Blackman et al. 2002	n=14; test group 1
71.0	4.0	H	23.1	5.9	25.5	Blackman et al. 2002	n=12; test group 2
71.0	5.0	H	21.7	3.2	14.7	Blackman et al. 2002	n=16; test group 3
71.0	6.0	N	24.8	3.6	14.5	Stachenfeld et al. 1998	n=9; exercise group
71.1	5.1	N	17.3	4.0	23.1	Peterson et al. 2003	n=114
71.2	3.5	H	22.6	3.2	14.2	Fehling et al. 1999	n=42
71.3	4.4	H	23.7	4.7	19.8	Audette et al. 2006	n=8; walking group
71.5	4.6	H	21.6	5.2	24.1	Audette et al. 2006	n=11; Tai Chi group
72.0	5.0	H	21.4	4.5	21.0	Blackman et al. 2002	n=14; control group
72.3	2.1	H	21.0	4.3	20.5	KJ Melanson et al. 1997	n=8
73.0	8.5	N	25.1	6.2	24.7	Stachenfeld et al. 1998	n=8; control group
73.0	9.0	N	25.2	6.2	24.6	Stachenfeld et al. 1999	n=8
73.3	2.7	N	16.7	3.3	19.8	Perini et al. 2000	n=11; VO2 range: 12.0-21.7
73.5	5.7	H	26.8	8.3	31.0	Audette et al. 2006	n=8; sedentary controls
74.5	7.8	NS	17.3	3.4	19.7	Fiser et al. 2010	n=24
74.6	4.0	N	18.0	4.0	22.2	Ridout et al. 2005	n=8
75.5	3.8	H	19.6	3.8	19.4	Deschenes et al. 2009	n=10
b. Complete age statistics are not provided							
6.0		N	36.5	2.9	7.9	DW Morgan et al. 1999	n=20
8.0		N	42.3	4.9	11.6	Treuth et al. 2004	n=91
9.0		N	42.3	5.4	12.8	Treuth et al. 2004	n=88
10.0		N	41.9	6.1	14.6	Treuth et al. 2004	n=84
12 - 13		NS	39.3	-		Pate et al. 2006 APAM	50th per. CI: 37.8-39.9
12.7		NS	42.7	6.1	14.3	Eisenman & Golding 1975	n=8; experimental group
12.7		NS	44.5	6.2	13.9	Eisenman & Golding 1975	n=8; control group
14 -15		NS	38.0	-		Pate et al. 2006 APAM	50th per. CI: 37.2-38.4
15.0		N	46.2	8.3	18.0	Cureton et al. 1997	n=7; mixed fitness groups
16-17		NS	37.6	-		Pate et al. 2006 APAM	50th perc.CI: 36.5-38.8
17-28		NS	33.8	4.6	13.6	Fringer & Stull 1974	n=44
18-19		NS	36.7	-		Pate et al. 2006 APAM	50th perc. CI: 35.7-37.8
20's		NS	34.1	5.0	14.7	Fleg et al. 1995a	
18 - 21		N	34.4	3.4	9.9	SB Parker et al. 1989	n=14; exercise group
18 - 21		N	37.5	5.7	15.2	SB Parker et al. 1989	n=10; control group
18 - 34		N	43.2	4.1	9.5	Beidleman et al. 1995	n=10; control group
19.5		H	38.5	3.6	9.4	Humphrey & Falls 1975	n=15

Table 1. Estimates of $VO_{2.MAX}$ in females seen in the literature (continued)

Body Mass (BM)-Adjusted Estimates: Mean ± SD VO2Max Estimate (mL/kg-min)							
Age /Range Mean	SD	Health Status	Mean	SD	COV (%)	Citation	Comment
19.6		NS	38.1	3.8	10.0	Eisenman & Golding 1975	n=8; experimental group
19.6		NS	39.0	3.9	10.0	Eisenman & Golding 1975	n=8; control group
20.0		N	37.7	2.9	7.7	Blessing et al. 1987	n=13; group 1
20.0		N	36.5	3.1	8.5	Blessing et al. 1987	n=13; group 2
29.0		NS	40.5	8.7	21.5	Diaz et al. 1978	n=5; treadmill only
30'3		NS	31.5	4.9	15.6	Fleg et al. 1995a	n=17
40's		NS	29.4	3.4	11.6	Fleg et al. 1995a	n=12
50's		NS	27.1	5.4	19.9	Fleg et al. 1995a	n=13
55 - 59		N	24.5	5.5	22.4	Hollenberg et al. 1998	n=100
60's		NS	25.7	4.4	17.1	Fleg et al. 1995a	n=12
60 - 64		N	22.7	4.4	19.4	Hollenberg et al. 1998	n=96
60-69		H	25.7	4.4	17.1	Fleg et al. 1995b	n=12
65 - 69		N	21.6	3.8	17.6	Hollenberg et al. 1998	n=109
65.0		N	21.9	4.5	20.5	Hollenberg et al. 2006	n=339; exercise group #1
60 - 77		H	19.4	3.6	18.6	ND Parker et al. 1996	n=16; control group
68.0		N	19.7	4.1	20.8	Hollenberg et al. 2006	n=293; exercise group #2
70's		NS	18.0	2.4	13.3	Fleg et al. 1995a	n=7
70 - 74		N	20.3	3.3	16.3	Hollenberg et al. 1998	n=88
75 - 79		N	19.2	3.1	16.1	Hollenberg et al. 1998	n=36
80's		NS	21.2	1.3	6.1	Fleg et al. 1995a	n=2
80 - 84		N	17.8	3.2	18.0	Hollenberg et al. 1998	n=18
>85		N	18.1	6.0	33.1	Hollenberg et al. 1998	n=7
Females: Active, Fit, or Athlete							
a. Mean & Statistics are provided for age							
9.1	1.5	Act	33.9	2.3	6.8	Gilliam et al. 1974	n=15; exercisers
11.3	1.1	Act	48.5	8.0	16.5	Rowland & Green 1988	n=18
13.0	2.0	Fit	58.7	4.5	7.7	Drinkwater et al. 1975	n=11
14.6	0.7	Ath	43.5	3.4	7.8	Rowland et al. 2011	n=13
15.2	4.1	Ath	45.2	5.3	11.7	Moffatt et al. 1984	n=13; gymnasts
15.6	1.1	Ath	50.8	4.6	9.1	Butts 1982	n=127; cross-country runners
15.9	1.0	Ath	61.7	7.1	11.5	Cunningham 1990	n=24; cross-country runners
19.0	1.0	Ath	52.1	5.1	9.8	Hill & Rowell 1997	n=13; track team members
19.0	3.6	Ath	49.0	10.8	22.0	Wenner et al. 2006	n=13; amenorrheic
19.6	1.1	Ath	46.9	5.6	11.9	Dellavalle & Haas 2012	n=24; rowers with low iron levels
20.1	1.1	Ath	49.5	5.6	11.3	Dellavalle & Haas 2012	n=24; rowers
20.0	1.4	Ath	45.7	4.9	10.7	Enemark-Miller et al. 2009	n=24; Lacrosse players
20.0	3.0	Ath	51.8	4.5	8.7	AS Ryan et al. 1996	n=14; exercise group
20.1	1.5	Ath	44.2	3.3	7.5	MS Green et al. 2013	n=39; soccer players
20.1	1.7	Fit	46.2	2.9	6.3	Getchell et al. 1977	n=21; joggers
20.5	1.6	Fit	44.0	4.7	10.7	Pintar et al. 2006	n=15; normal weight
20.6	2.8	Ath	48.8	4.1	8.4	Branford & Howley 1977	n=10; distance runners
20.7	3.3	Act	46.9	5.2	11.1	Nindl et al. 1998	n=20; Army personnel
20.7	3.2	Act	36.9	3.8	10.3	Sharp et al. 2002	n=122
20.7	3.6	Act	41.3	4.0	9.7	Rowland & Green 1988	n=18
21.0	3.0	Act	42.4	10.4	24.5	J Kang et al. 2007	n=11
21.0	3.6	Ath	51.0	7.2	14.1	Wenner et al. 2006	n=13; eumenorrheic
21.3	1.2	NS	44.8	5.5	12.3	Gist et al. 2014	n=3; mod.-trained college
21.4	3.4	Act	39.2	5.1	13.0	Sharp et al. 2002	n=155

Table 1. Estimates of $VO_{2.MAX}$ in females seen in the literature (continued)

Body Mass (BM)-Adjusted Estimates: Mean ± SD VO2Max Estimate (mL/kg-min)							
Age /Range Mean	SD	Health Status	Mean	SD	COV (%)	Citation	Comment
21.9	2.0	Fit	48.7	4.5	9.2	Jeans et al. 2011	n=8; range of VO2: 45.0-52.5
21.9	2.4	Fit	51.1	6.5	12.7	Drinkwater et al. 1975	n=10
22.0	2.5	Ath	34.1	4.5	13.2	AS Ryan et al. 1996	n=8; control group
22.0	3.6	Act	35.3	2.5	7.1	Proctor et al. 2004	n=13
22.8	4.5	Ath	50.7	9.0	17.8	Faria & Faria 1998	n=12; college rowers
22.9	3.2	Act	40.7	5.5	13.5	Astorino et al. 2010	n=17; recreationally active
23.0	2.7	Act	39.1	2.1	5.4	Astorino et al. 2012	n=4
23.0	3.0	Ath	55.0	6.0	10.9	Wenner et al. 2006	n=9; eumenorrheic/oral contrac.
23.0	3.7	NS	49.6	3.5	7.1	Darby et al. 1995	n=16; exercise dancers
23.0	8.5	Act	39.9	7.0	17.5	Kist et al. 2013	n=11; aerobically trained
23.4	2.1	Ath	53.4	2.7	5.1	Drenowatz & Eisen. 2011	n=10; endurance runners
23.5	6.4	Fit	33.9	4.5	13.3	Meyers & Sterling 2000	n=24; equestrians
25.0	3.0	Act	42.9	5.0	11.7	CB Scott 1997	n=10
25.0	4.6	Act	51.9	5.1	9.8	Sparling & Cureton 1983	n=34; distance runners
25.2	3.1	Act	41.1	6.1	14.8	Astorino et al. 2012	n=9; recreationally active
25.7	7.2	Act	44.0	9.6	21.8	Beckham & Earnest 2000	n=18; 79% are active
26.0	3.0	Act	52.1	3.1	6.0	Ogawa et al. 1992	n=13
26.0	3.3	Ath	66.0	4.0	6.1	LO Schultz et al. 1992	n=9; endurance trained
26.0	3.7	Act	55.0	3.7	6.7	Tanaka et al. 1997	n=14; endurance-trained
26.3	4.2	Ath	53.8	2.8	5.2	Laughlin & Yen 1996	n=8; amenorrhic
26.3	5.9	Fit	44.9	4.2	9.4	Nicklas et al. 1989	n=6; eumenorrheic
26.7	5.5	Fit	54.5	4.1	7.5	Ballor & Poehlman 1992	n=21 aerobically trained
26.9	5.3	Ex	45.3	4.2	9.3	SD Fox et al. 1993	n=9; recreational aerobics
27.0	2.1	Ath	63.0	4.6	7.3	Gojanovic et al. 2012	n=5
27.0	2.8	Ath	51.8	4.0	7.7	Proctor et al. 1998	n=8
27.0	5.0	Fit	55.3	6.6	11.9	Horton et al. 1998	n=8; competitive cyclists
27.8	2.0	Ath	53.5	1.1	2.1	Frey et al. 1993	n=6; cycling team members
28.0	3.4	Fit	45.4	4.5	9.9	Sandoval & Matt 2002	n=14
28.0	5.0	Act	42.5	5.1	12.0	Dean et al. 2003	n=8; mid-luteal phase (Note 2)
29.5	5.1	Act	42.6	3.7	8.7	EL Melanson et al. 2002	n=8; lean exercisers
30.0	3.7	Fit	53.0	5.6	10.6	Seals et al. 1999	n=14; endurance trained
30.0	3.9	Fit	53.4	5.0	9.4	BE Hunt et al. 1997	n=15; runners
30.0	5.5	Ath	57.0	5.1	8.9	Schaal et al. 2011	n=5; eumenorrheic
30.2	5.0	Fit	47.6	9.1	19.1	Quinn et al. 1994	n=8
30.7	3.4	Ath	60.8	8.5	14.0	Laughlin & Yen 1996	n=8; regular cycles
31.0	5.0	Ath	60.3	4.8	8.0	Thompson & Man.1996	n=13; endurance runners
31.0	9.6	Ath	56.0	3.4	6.1	Schaal et al. 2011	n=5; amenorrheic
31.7	9.2	Fit	41.1	7.1	17.3	Dalleck & Kravitz 2006	n=12; moderate exercisers
32.9	5.0	Fit	46.7	6.7	14.3	Dalleck & Kravitz 2006	n=24; moderate exercisers
32.9	4.0	Pg	27.7	1.4	5.1	Szymanski & Satin 2012	n=15; highly active
33.0	5.4	Fit	53.6	5.2	9.7	Horton et al. 1994	n=5; cyclists
34.0	3.3	Ath	56.5	5.1	9.0	AS Ryan et al. 1996	n=9; exercise group
34.0	4.6	Act	55.2	4.6	8.3	Tanaka et al. 1997	n=21; endurance-trained
34.3	4.0	Pg	23.8	2.2	9.2	Szymanski & Satin 2012	n=15; active
35.2	3.2	Fit	52.4	5.4	10.3	Drinkwater et al. 1975	n=10
35.7	4.2	Fit	50.2	1.7	3.4	Shvartz 1996	n=6
38.7	1.4	Ath	54.1	7.2	13.3	Wells et al. 1992	n=11; runners
39.7	10.1	Fit	43.6	7.6	17.4	Malek et al. 2004	n=49; aerobically trained

Table 1. Estimates of $VO_{2.MAX}$ in females seen in the literature (continued)

Body Mass (BM)-Adjusted Estimates: Mean ± SD VO2Max Estimate (mL/kg-min)							
Age /Range Mean	SD	Health Status	Mean	SD	COV (%)	Citation	Comment
41.3	1.4	Ath	47.4	6.7	14.1	Wells et al. 1992	n=11; runners
42.8	2.0	Ath	48.7	7.8	16.0	SA Hawkins et al. 2001	n=24; visit 1
44.8	3.7	Fit	50.4	2.8	5.6	Drinkwater et al. 1975	n=7
45.0	3.5	Ath	49.6	4.7	9.5	AS Ryan et al. 1996	n=10; exercise group
45.0	3.6	Act	51.4	6.5	12.6	Tanaka et al. 1997	n=3; endurance-trained
46.0	4.4	Ath	26.9	4.9	18.2	AS Ryan et al. 1996	n=6; control group
47.1	1.3	Ath	43.6	5.1	11.7	Wells et al. 1992	n=11; runners
49.8	2.8	Ath	46.7	5.2	11.1	SA Hawkins et al. 2001	n=16; visit 1
51.2	2.4	Ath	45.2	5.9	13.1	SA Hawkins et al. 2001	n=24; visit 2
52.3	2.7	Fit	46.1	9.7	21.0	Drinkwater et al. 1975	n=6
52.6	1.5	Ath	41.2	5.9	14.3	Wells et al. 1992	n=10; runners
54.0	4.3	Act	42.7	7.2	16.9	Tanaka et al. 1997	n=23; endurance-trained
55.7	7.8	Act	30.6	6.7	21.9	Nikolai et al. 2009	n=7; in a water exercise class
57.0	3.0	Act	35.3	3.3	9.3	Ogawa et al. 1992	n=13
58.0	1.9	Ath	43.8	6.6	15.1	AS Ryan et al. 1996	n=10; exercise group
58.0	3.5	Fit	39.1	5.9	15.1	Seals et al. 1999	n=12; endurance trained
58.0	3.7	Fit	38.7	5.6	14.5	BE Hunt et al. 1997	n=14; post-menopausal runners
58.0	6.3	Fit	40.0	6.0	15.0	Tanaka et al. 1998	n=10; runners
58.3	3.2	Ath	40.8	7.2	17.6	SA Hawkins et al. 2001	n=16; visit 2
59.0	6.3	Fit	30.7	6.6	21.5	Tanaka et al. 1998	n=10; swimmers
60.0	7.0	O	15.0	2.8	18.7	Jordan et al. 2005	n=24
61.0	8.0	Fit	40.0	4.8	12.0	Proctor et al. 1997	n=8; endurance trained
61.7	4.7	Ath	39.5	3.9	9.9	Wells et al. 1992	n=6; runners
63.3	2.0	Ath	46.2	9.0	19.5	SA Hawkins et al. 2001	n=13; visit #1
64.0	3.5	Act	24.6	4.2	17.1	Proctor et al. 2004	n=12
64.6	3.9	Ath	39.4	4.8	12.2	SA Hawkins et al. 2001	n=9; visit 1
64.7	2.0	Fit	35.6	4.4	12.4	Drinkwater et al. 1975	n=6
66.0	3.6	Act	32.5	4.7	14.5	Tanaka et al. 1997	n=13; endurance-trained
66.8	15.9	Ath	29.4	14.5	49.3	Wilund et al. 2008	n=6; Master athlete
73.2	5.7	Ath	31.8	8.4	26.4	SA Hawkins et al. 2001	n=9; visit 2
b. Complete age statistics are not provided							
9--10		Ath	56.3	6.6	11.7	Eisenmann et al. 2001	n=9; distance runners
11.0		Ath	57.9	5.2	9.0	Eisenmann et al. 2001	n=11; distance runners
12.0		Ath	57.1	5.3	9.3	Eisenmann et al. 2001	n=15; distance runners
13.0		Ath	54.8	6.3	11.5	Eisenmann et al. 2001	n=17; distance runners
14.0		Ath	56.9	8.4	14.8	Eisenmann et al. 2001	n=14; distance runners
15.0		Ath	56.2	7.0	12.5	Eisenmann et al. 2001	n=911; distance runners
14 - 15		Ath	48.5	4.6	9.5	Drinkwater & Horvath1971	n=11; track athletes
16.0		Ath	54.3	6.8	12.5	Eisenmann et al. 2001	n=12; distance runners
17--18		Ath	51.8	6.4	12.4	Eisenmann et al. 2001	n=16; distance runners
18 - 21		Act	44.2	4.8	10.9	WL Daniels et al. 1982	n=7; Army cadets
18 - 23		Act	44.1	1.5	3.4	Kamon & Pandolf 1972	n=6
18--34		Fit	59.7	5.3	8.9	Fay et al. 1989	n=13; distance runners
18 - 34		Fit	60.2	4.7	7.8	Beidleman et al. 1995	n=10; endurance runners
19 - 21		Fit	47.7	2.8	5.9	Kamon & Pandolf 1972	n=4
20.0		Act	38.9	5.9	15.2	Sonna et al. 2001	n=97; non-participants
21.0		Act	39.6	5.1	12.9	Sonna et al. 2001	n=71; participants group
24.0		Ath	64.7	-		Wilhite et al. 2013	n=1; elite distance runner

Table 1. Estimates of $VO_{2.MAX}$ in females seen in the literature (continued)

Body Mass (BM)-Adjusted Estimates: Mean ± SD VO2Max Estimate (mL/kg-min)							
Age /Range Mean	SD	Health Status	Mean	SD	COV (%)	Citation	Comment
25 - 34		Act	31.7	4.6	14.5	Bruce 1984b	n=?
26.0		Act	51.5	3.2	6.2	Proctor et al. 1997	n=8; endurance trained
35 - 44		Act	29.9	5.3	17.7	Bruce 1984b	n=?
55 - 64		Act	29.7	4.7	15.8	Bruce 1984b	n=?
Females: Sedentary, Overweight, or Obese							
a. Mean & SD statistics are provided for age							
9.1	1.4	O	25.8	4.9	19.0	Gutin et al. 1995	n=12; AA exercise group
9.4	1.6	O	25.3	4.7	18.6	Gutin et al. 1995	n=10; AA control group
11.2	1.8	OW	32.1	5.1	15.9	Byrd-Williams et al. 2008	n=76; Hispanic youth
15.2	1.2	O	21.5	4.4	20.5	Gutin et al. 2002	n=39; black adolescents
15.3	1.2	O	24.7	3.9	15.8	Gutin et al. 2002	n=15; white adolescents
19.4	1.5	OW	42.8	3.3	7.7	Pintar et al. 2006	n=15; high fit
21.0	0.8	O-Sed.	28.3	1.4	4.9	Szmedra et al. 1998	n=7; AA
21.0	3.6	Sed.	38.2	3.0	7.9	Loucks et al. 1998	n=9
21.0	4.0	OW	32.4	3.1	9.6	Potteiger et al. 2008	n=18; control group
21.1	3.0	OW	30.9	5.0	16.2	Pintar et al. 2006	n=15; low fit
21.9	2.0	Sed.	30.4	4.3	14.1	Pintar et al. 2006	n=15; normal weight
22.0	3.0	Sed.	28.0	4.8	17.1	Croley et al. 2005	n=11
22.8	2.7	OW	24.4	6.4	26.2	MK Thornton et al. 2011	n=10; AA
23.0	2.0	Sed.	37.0	4.3	11.6	Ogawa et al. 1992	n=14
23.3	4.6	O-Sed.	32.7	3.8	11.6	Washburn et al. 2003	n=29
23.4	3.6	O	26.8	2.4	9.0	Kaminsky & Whaley 1993	n=5; Hispanic
24.0	5.0	OW	32.8	4.2	12.8	Potteiger et al. 2008	n=25; exercise group
25.0	3.3	Sed.	34.9	4.6	13.2	Tanaka et al. 1997	n=11
25.0	4.0	Sed.	34.2	5.2	15.2	Schiller et al. 2001	n=14; Caucasian
25.0	3.0	Sed.	34.0	7.6	22.4	Schiller et al. 2001	n=14; Hispanic
25.1	3.1	Sed.	38.5	4.0	10.4	MJ Turner et al. 1999	n=10
25.3	7.3	O	25.9	3.3	12.7	Browning et al. 2006	n=9
27.5	5.1	Sed.	39.0	2.3	5.9	Laughlin & Yen 1996	n=8; regular cycles
28.0	3.0	O	22.1	2.1	9.5	Henson et al. 1987	n=7
28.6	12.4	Sed.	23.9	9.4	39.3	Rynders et al. 2011	n=74
28.7	6.9	Sed.	42.1	4.8	11.4	Ballor & Poehlman 1992	n=48
28.7	6.9	Sed.	32.6	3.8	11.7	Dowdy et al. 1985	n=10; control group
29.0	3.5	Sed.	34.3	5.5	16.0	Seals et al. 1999	n=12
29.8	5.8	O	27.6	5.4	19.6	Steffan et al. 1999	n=20
30.1	4.7	OW	30.9	1.9	6.1	Lennon et al. 1985	n=8; exercise group #2
31.5	4.4	Sed.	32.4	6.4	19.8	Westerlind & Will. 2007	n=24
31.5	5.6	Sed.	33.8	3.9	11.5	Dowdy et al. 1985	n=18; experimental group
32.1	10.7	Sed.	24.7	5.4	21.9	Skinner et al. 2001	n=120; black
32.8	5.9	OW	25.0	3.8	15.2	Nehlsen et al. 1991	n=18; exercise group
33.0	3.3	Sed.	33.7	6.3	18.7	Tanaka et al. 1997	n=11
33.0	4.0	Sed.	29.5	6.4	21.7	Branch et al. 2000	n=18
33.0	4.0	Sed.	33.4	5.6	16.8	Schiller et al. 2001	n=14; Caucasian
34.0	4.0	Sed.	30.3	5.8	19.1	Schiller et al. 2001	n=13; Hispanic
34.5	13.7	Sed.	29.8	6.8	22.8	Skinner et al. 2001	n=226; white
35.9	7.2	OW	30.9	1.9	6.1	Lennon et al. 1985	n=11; exercise group #1
36.0	6.8	OW	25.7	3.8	14.8	Nehlsen et al. 1991	n=18; control group
37.1	4.0	OW	24.7	2.7	10.9	Nieman et al. 1988	n=11; exercise group

Table 1. Estimates of $VO_{2.MAX}$ in females seen in the literature (continued)

Body Mass (BM)-Adjusted Estimates: Mean ± SD VO2Max Estimate (mL/kg-min)							
Age /Range Mean	SD	Health Status	Mean	SD	COV (%)	Citation	Comment
37.4	8.3	OW	29.6	2.9	9.8	Lennon et al. 1985	n=11; control group
38.0	6.3	OW	25.6	3.2	12.5	Nieman et al. 1988	n=10; control group
38.0	6.3	NF	25.8	3.7	14.3	Shvartz 1996	n=6
39.4	9.5	O	22.4	4.5	20.1	Jakicic et al. 1995	n=121
41.7	7.3	OW-O	27.0	13.4	49.6	CW Hall et al. 2012	n=24
43.0	11.0	OW	24.0	4.6	19.2	KA Snyder et al. 1997	n=15
43.4	4.9	Sed.	23.0	5.7	24.8	E Lind et al. 2005	n=23
44.0	3.0	Sed.	26.8	2.3	8.6	Schiller et al. 2001	n=8; Hispanic
45.0	3.7	Sed.	27.0	4.5	16.7	Tanaka et al. 1997	n=14
45.0	5.0	Sed.	28.0	5.0	17.9	Schiller et al. 2001	n=21; Caucasian
51.7	2.6	OW-O	16.9	2.5	14.8	Earnest et al. 2010	n=82
51.9	4.3	Sed.	20.7	3.6	17.4	JS Green et al. 2001	n=14; estrogen replacement
52.0	6.0	O	22.0	5.5	25.0	Lost citation 2012	n=8
52.0	6.6	OW	23.5	3.4	14.5	JL Robbins et al. 2009	n=12
53.0	4.0	Sed.	25.5	5.8	22.7	Schiller et al. 2001	n=15; Hispanic
54.0	4.5	Sed.	26.2	3.6	13.7	Tanaka et al. 1997	n=20
54.0	5.0	Sed.	26.2	3.9	14.9	Schiller et al. 2001	n=26; Caucasian
54.0	12.5	OW-O	27.0	7.0	25.9	Dionne et al. 2001	n=15; carriers of 2 genotypes
55.0	1.9	Sed.	25.5	5.7	22.4	Zarins et al. 2009	n=10; postmenopausal
56.6	6.6	OW-O	16.0	2.9	18.1	Church et al. 2007	n=103;sedentary group 3
56.3	6.0	OW	16.1	3.0	18.6	Sisson et al. 2009	n=88; Sed. Group 3
56.7	6.4	OW	14.9	2.3	15.4	Sisson et al. 2009	n=84; Sed. Group 2
56.8	12.2	OW-O	24.7	6.8	27.5	Dionne et al. 2001	n=16; carriers of 1 genotype
56.9	2.7	Sed.	21.1	3.9	18.5	JS Green et al. 2001	n=9; no estrogen replacement
57.2	5.8	OW-O	15.6	2.9	18.6	Church et al. 2007	n=102; sedentary control group
57.3	6.6	OW-O	14.9	2.4	16.1	Church et al. 2007	n=104;sedentary group 2
57.5	1.5	OW-O	16.0	2.6	16.3	Earnest et al. 2010	n=76
57.7	6.6	OW-O	15.5	2.9	18.7	Church et al. 2007	n=155;sedentary group 1
57.8	6.4	O-Sed.	15.3	2.0	13.1	AN Jordan et al. 2005	n=27; Exercise group #2
58.0	4.9	OW	19.8	3.9	19.7	AS Ryan et al. 2000	n=24
58.0	6.5	OW	15.4	3.0	19.5	Sisson et al. 2009	n=138; Sed. Group 1
58.3	5.9	O-Sed.	15.6	2.3	14.7	AN Jordan et al. 2005	n=60; exercise group #1
59.0	4.0	Sed.	27.5	4.5	16.4	Fielding et al. 1999	n=17; protocol test 1
60.0	7.0	O-Sed.	15.0	2.8	18.7	AN Jordan et al. 2005	n=24; exercise group #3
60.0	8.0	O-Sed.	21.1	1.6	7.6	Tanaka et al. 1998	n=9
62.0	4.5	Sed.	22.6	4.0	17.7	Seals et al. 1999	n=20
64.0	4.0	Sed.	22.2	3.1	14.0	Ogawa et al. 1992	n=14
64.0	4.0	Sed.	22.4	4.8	21.4	Tanaka et al. 1997	n=16; carriers of 1 genotype
64.0	4.0	Sed.	21.5	4.7	21.9	Schiller et al. 2001	n=18; Caucasian
64.0	5.0	OW-O	36.3	8.2	22.6	Nicklas et al. 2003	n=29
64.4	3.2	Sed.	22.0	2.2	10.0	MJ Turner et al. 1999	n=10
64.9	4.2	OW-O	14.8	2.4	16.2	Earnest et al. 2010	n=93
65.0	4.0	Sed.	20.7	2.9	14.0	Schiller et al. 2001	n=5; Hispanic
66.0	6.0	OW	20.2	3.6	17.8	JL Thompson et al. 1997	n=40; postmenopausal
66.0	4.0	Sed.	19.9	3.1	15.6	Kohrt et al. 1998	n=112
69.2	11.0	Sed.	20.3	7.6	37.4	Wilund et al. 2008	n=6
67.0	4.9	Sed.	16.2	3.5	21.6	White et al. 1998	n=60; exercise group #1
70.0	8.0	Sed.	17.6	4.5	25.6	Croley et al. 2005	n=9

Table 1. Estimates of $VO_{2.MAX}$ in females seen in the literature (continued)

Body Mass (BM)-Adjusted Estimates: Mean ± SD VO2Max Estimate (mL/kg-min)							
Age /Range Mean	SD	Health Status	Mean	SD	COV (%)	Citation	Comment
72.0	3.0	Inact.	21.4	3.9	18.2	DiPietro et al. 2006	n=9; exercise group 3
72.0	8.0	Sed.	19.1	3.6	18.8	EP Weiss et al. 2006	n=83
73.0	3.0	Inact.	21.2	3.4	16.0	DiPietro et al. 2006	n=9; exercise group 2
74.7	3.4	Sed.	12.1	3.7	30.6	Church et al. 2008	n=20
75.0	5.0	Inact.	18.3	4.2	23.0	DiPietro et al. 2006	n=7; exercise group 1
72.0	8.0	Sed.	19.1	3.6	18.8	EP Weiss et al. 2006	n=83; non-smokers
b. Complete age statistics are not provided							
17 - 18		Sed.	26.2	-	-	MA Edwards 1974	n=6
17 - 18		Sed.	38.2	0.1	0.3	Kamon & Pandolf 1972	n=?
17 - 22		Sed.	38.4	4.7	12.2	Kearney et al. 1976	n=14; exercise group 1
17 - 22		Sed.	38.5	5.0	13.0	Kearney et al. 1976	n=13; exercise group 2
18 - 20		Sed.	27.3	-	-	MA Edwards 1974	n=6
18 - 23		OW	43.6	6.8	15.6	O'Leary & Stav. 2012	n=9
25 - 34		Sed.	26.1	6.4	24.5	Bruce 1984b	n=?
35 - 44		Sed.	34.1	3.2	9.4	Bruce 1984b	n=?
45 - 54		Sed.	23.1	4.0	17.3	Bruce 1984b	n=?
55 - 64		Sed.	20.2	4.3	21.3	Bruce 1984b	n=?
Females: Health & Other Issues							
21.9	2.7	MR	30.8	7.7	25.0	Draheim et al. 1999	n=13
22.3	6.9	CC	19.8	5.1	25.8	AM Miller et al. 2012	n=34; 12 y mean post-treatment
29.3	6.3	CHD	22.4	5.3	23.7	GK Lui et al. 2011	n=40; pregnant CI=0.61 (0.15)
29.3	6.3	CHD	26.1	5.2	19.9	GK Lui et al. 2011	n=38; pregnant CI=0.89 (0.07)
30.4	6.7	MR	28.1	7.1	25.3	Fernhall et al. 1996	n=20; no DS
31.7	7.2	DS	22.2	4.3	19.4	Fernhall et al. 1996	
32.9	5.8	Pg	21.3	2.5	11.7	Szymanski & Satin 2012	n=15; non-exercisers
33.3	7.1	Pg	26.9	5.2	19.3	Soultankis et al. 1966	n=20
41.8	9.7	MS	21.7	6.0	27.6	Petruzzello & Motl 2011	n=25
43.6	7.8	MS	22.1	5.8	26.2	Motl & Fernhall 2012	n=32; relapsing-remitting MS
49.9	11.6	MI	19.9	4.8	24.1	Pinkstaff et al. 2011	n=146
50.6	8.7	BC	22.0	4.0	18.2	Tosti et al. 2011	n=7
52.5	11.5	CHF	21.9	2.7	12.3	Duscha et al. 2001	n=13
59.2	11.0	HP	28.2	7.7	27.3	Shultz et al. 2010	n=49
62.0	6.6	CAD	21.7	3.3	15.2	Sheldahl et al. 1996	n=11
62.0	11.0	HP	14.5	3.9	26.9	Ades et al. 2006	n=815; multicenter study
63.7	5.8	COPD	11.3	3.0	26.5	Carter et al. 1994	n=58; severe airflow limitation
64.8	6.4	COPD	17.0	5.6	32.9	Carter et al. 1994	n=23; mild airflow limitation
65.0	5.2	COPD	13.9	3.5	25.2	Carter et al. 1994	n=42; moderate airflow limitation
69.0	6.0	HP	17.0	5.0	29.4	Ades et al. 1993	n=15
72.9	6.1	Cardio	14.2	2.9	20.4	Ades et al. 2005	n=21

Abbreviations:	
A	Asthmatic
AA	African-American
Act	Active (but non-athletes)
Ath	Athletes
BC	Breast cancer patient
BLSA	Baltimore Longitudinal Study of Aging
CA	Caucasian

Abbreviations:	
CC	Cancer survivor
CF	Cystic Fibrosis
CHD	Congenital heart disease
CI	Chronotropic Index
CHF	Chronic heart failure
COPD	Chronic obstructive pulmonary disease
DS	Down syndrome

Abbreviations:	
EX	Exercisers (regular)
Fit	Very active healthy exercisers
Frail	Mild-to-moderate frailty
H	Healthy
HP	heart patients
Inact.	Inactive (not necessarily sedentary)
MI	Myocardial iscemia
MR	Mentally retarded (some with Down syndrome)
MS	Multiple sclerosis
N	Normal (mostly healthy)
NF	Not fit; poor fitness
NG	National Guard (all types)

Abbreviations:	
NS	Not specified
O	Obese
OW	Overweight
Pg	Pregnant
Sed	Sedentary
Notes:	
1.0	The study investigated VO2 at sea level (shown) and at altitude for two menstrual phases; There was no statistical differences between phases; luteal: 46.3 ± 5.6 mL/kg-min
2.0	VO2max was measured in 3 phases of the menstrual cycle. VO2max in the other 2 phases was slightly higher (not statistically significant).

Table 2. Estimates of $VO_{2.MAX}$ in males seen in the literature

Body Mass (BM)-Adjusted Estimates: Mean ± SD VO2Max Estimate (mL/kg-min)							
Age /Range Mean	SD	Health Status	Mean	SD	COV (%)	Citation	Comment
Males: Normal, Healthy, or Not-Specified							
a. Mean & SD statistics are provided for age							
7.3	1.0	H	47.8	9.1	19.0	Treuth et al. 2003	n=6
7.5	0.3	N	52.4	19.3	36.8	Livingstone et al. 1992	n=6
8.3	1.1	N	52.7	4.2	8.0	Cureton et al. 1997	n=27; mixed fitness group
8.3	1.9	NS	50.7	9.6	18.9	DM Rogers et al. 1995a	n=15
8.8	0.7	N	45.9	9.8	21.4	McMurray et al. 2003a	n=403; CA
9.0	0.7	NS	55.0	6.3	11.5	DM Rogers et al. 1995b	n=21
9.0	0.9	N	44.5	8.1	18.2	McMurray et al. 2003a	n=87; AA
9.1	1.0	NS	49.6	6.4	12.9	Cureton et al. 1995	n=200; multicenter study
9.3	0.2	N	48.9	12.9	26.4	Livingstone et al. 1992	n=5
9.4	1.7	NS	42.7	5.5	12.9	Gilliam et al. 1977	n=32
9.5	0.7	N	39.0	6.4	16.4	Becker & Vaccaro 1983	n=13; experimental group
9.6	2.6	NS	52.0	9.3	17.9	Fahey et al. 1979	n=7; Tanner 1
9.8	0.6	H	35.4	7.5	21.2	KE Swain et al. 2010	n=20
9.9	1.0	NS	46.7	8.0	17.1	Iannotti et al. 2004	n=10
10.0	0.6	N	41.7	5.7	13.7	Becker & Vaccaro 1983	n=13; control group
10.0	1.0	H	45.6	4.0	8.8	Rogowski et al. 2012	n=19
10.0	2.0	N	42.0	6.0	14.3	Cooper et al. 1984	n=37
10.1	0.8	N	44.4	10.1	22.7	McMurray et al. 2003a	n=381; CA
10.2	1.2	N	48.7	5.5	11.3	Kanaley & Boileau 1988	n=10
10.3	0.3	N	44.6	9.4	21.1	McMurray et al. 2003a	n=79; AA
10.3	2.5	N	53.2	15.8	29.7	Chausow et al. 1984	n=8
10.4	0.3	H	47.2	6.0	12.7	N Hopkins et al. 2011	n=46 (Summer only)
10.4	1.1	N	45.9	2.6	5.7	Mayers & Gutin 1979	n=8
10.5	0.7	N	47.4	5.4	11.4	Mahon et al. 1997b	n=9
10.5	1.2	N	48.1	6.0	12.5	McMurray et al. 1998b	n=15
10.6	1.0	N	50.0	9.0	18.0	Janz et al. 1998	n=61
10.6	1.3	H	47.5	6.4	13.5	Roemmich et al. 1998	n=18
10.6	2.3	N	53.0	4.3	8.1	Skinner et al. 1971	n=26; treadmill protocol #3
10.7	0.6	N	52.3	6.0	11.5	JD Brown et al. 2002	n=16
10.7	0.7	NS	39.4	7.1	18.0	Fahey et al. 1979	n=7; Tanner 2

Table 2. Estimates of $VO_{2.MAX}$ in males seen in the literature (continued)

Body Mass (BM)-Adjusted Estimates: Mean ± SD VO2Max Estimate (mL/kg-min)							
Age /Range		Health			COV		
Mean	SD	Status	Mean	SD	(%)	Citation	Comment
10.7	2.6	N	51.6	7.0	13.6	Skinner et al. 1971	n=31; treadmill protocol #1
10.8	0.4	H	38.4	2.4	6.3	Haffor et al. 1990	n=5
10.9	0.3	N	56.8	6.8	12.0	Paterson et al. 1986	n=19
10.9	1.3	H	50.9	8.3	16.3	Rowland et al. 1997 MSSE	n=15
10.9	2.6	N	50.0	5.5	11.0	Skinner et al. 1971	n=26; treadmill protocol #2
11.0	0.8	N	52.1	8.5	16.3	Mahon et al. 1997a	n=15; Tanner 1
11.1	2.4	H	44.1	7.9	17.9	JK Murphy et al. 1988	n=60; CA
11.1	3.0	H	42.6	8.6	20.2	JK Murphy et al. 1988	n=64; AA
11.2	2.6	H	37.4	12.1	32.4	Boas et al. 1999	n=23; control group
11.4	1.2	N	51.8	5.4	10.4	Mahon et al. 1997a	n=11; Tanner 2
11.6	1.0	N	49.0	6.0	12.2	Janz et al. 1998	n=58
11.6	1.8	N	45.2	5.0	11.1	Rogowski et al. 2012	n=19
11.8	2.8	N	44.3	7.4	16.7	Golden et al. 1991	n=101
12.0	0.3	N	60.0	5.7	9.5	Paterson et al. 1986	n=19
12.1	0.8	N	40.5	7.0	17.3	McMurray et al. 2003a	n=403; CA
12.3	0.9	N	39.9	6.9	17.3	McMurray et al. 2003a	n=87; AA
12.6	1.0	N	46.0	8.0	17.4	Janz et al. 1998	n=61
12.7	0.3	N	51.8	5.1	9.8	Livingstone et al. 1992	n=5
12.7	1.0	N	53.8	5.7	10.6	Cureton et al. 1997	n=27; mixed fitness group
12.8	1.1	N	48.7	5.3	10.9	Boileau et al. 1977	n=21; treadmill value
12.8	1.8	NS	48.7	9.1	18.7	Mahar et al. 2011	n=48; validation sample
12.8	1.8	NS	49.5	8.2	16.6	Mahar et al. 2011	n=34; cross-validation sample
12.8	2.1	N	52.5	2.7	5.1	Kwee & Wilmore 1990	n=181; "above average fitness"
12.9	0.3	N	60.9	5.3	8.7	Paterson et al. 1986	n=19
12.9	1.2	NS	41.3	9.1	22.0	Fahey et al. 1979	n=6; Tanner 3
13.0	0.7	N	48.5	8.3	17.1	Boiarskaia et al. 2011	n=61
13.1	0.8	N	42.8	8.2	19.2	McMurray et al. 2003	n=349; CA
13.2	1.2	N	55.7	5.0	9.0	Mahon et al. 1997a	n=8; Tanner 3
13.3	0.8	N	40.8	7.3	17.9	McMurray et al. 2003	n=72; AA
13.4	1.9	NS	53.4	5.9	11.0	DM Rogers et al. 1995b	n=15
13.6	1.0	N	48.0	7.0	14.6	Janz et al. 1998	n=56
13.6	1.4	N	44.4	2.7	6.1	Kwee & Wilmore 1990	n=163; "below average fitness"
13.6	1.5	NS	50.9	6.6	13.0	Cureton et al. 1995	n=99; multicenter study
13.7	0.5	N	51.8	4.8	9.3	Kanaley & Boileau 1988	n=10
13.9	0.3	N	61.1	5.7	9.3	Paterson et al. 1986	n=19
14.1	0.7	N	42.4	9.0	21.2	McMurray et al. 2003	n=312; CA
14.3	0.8	N	41.0	8.3	20.2	McMurray et al. 2003	n=66; AA
14.3	1.0	H	52.8	9.2	17.4	Treuth et al. 2003	n=6
14.6	1.0	N	46.0	7.0	15.2	Janz et al. 1998	n=53; exercise group
14.6	1.3	H	51.4	5.6	10.9	Roemmich et al. 1998	n=11
14.7	1.0	N	54.4	3.4	6.3	Mahon et al. 1997a	n=9; tanner 4
14.9	0.3	N	63.5	6.4	10.1	Paterson et al. 1986	n=19
15.1	0.7	N	42.6	10.8	25.4	McMurray et al. 2003	n=297; CA
15.1	1.0	NS	46.4	7.7	16.6	Murray et al. 1993	n=43
15.1	2.6	NS	45.2	11.3	25.0	Fahey et al. 1979	n=3; Tanner 5
15.3	1.1	N	44.7	5.3	11.9	Rogowski et al. 2012	n=20
15.4	0.4	N	51.3	2.9	5.7	Livingstone et al. 1992	n=3
15.4	0.8	N	41.1	10.1	24.6	McMurray et al. 2003	n=62; AA

Table 2. Estimates of $VO_{2.MAX}$ in males seen in the literature (continued)

Body Mass (BM)-Adjusted Estimates: Mean ± SD VO2Max Estimate (mL/kg-min)							
Age /Range		Health			COV		
Mean	SD	Status	Mean	SD	(%)	Citation	Comment
15.4	1.8	NS	48.4	6.9	14.3	Fahey et al. 1979	n=5; Tanner 4
15.8	0.8	N	52.8	6.4	12.1	Cureton et al. 1997	n=38; mixed fitness group
15.9	1.1	ND	43.7	10.6	24.3	Gutin et al. 2005	n=94; black adolescents
16.0	1.0	NS	50.0	8.0	16.0	Cooper et al. 1984	n=33
16.2	1.3	NS	47.6	8.8	18.5	Gutin et al. 2005	n=102; white adolescents
17.9	1.6	NS	52.9	5.0	9.5	Dill et al. 1972	n=11
18.7	0.6	N	52.4	4.6	8.8	Wolfe et al. 1976	n=9; lean group
19.0	1.9	NS	43.5	4.7	10.8	Fox et al. 1975	n=26; exercise group 1
19.2	0.4	N	41.4	4.0	9.7	Burke 1977	n=7; control group
19.2	0.4	N	41.8	4.9	11.7	Burke 1977	n=9; experimental group
19.2	2.2	NS	44.2	4.9	11.1	Fox et al. 1975	n=23; exercise group 2
19.3	0.5	N	44.1	5.7	12.9	Golden & Vaccaro 1984	n=9 group 3
19.3	2.3	NS	46.3	8.0	17.3	Dolgener et al. 1994	n=96; validation group
19.4	1.3	NS	44.3	5.0	11.3	Dolgener et al. 1994	n=33; cross-validation group
19.5	0.9	H	59.6	3.5	5.9	Kasch et al. 1986	n=11
19.6	2.7	NS	45.1	3.5	7.8	Fox et al. 1975	n=16; control group
19.7	2.1	H	49.2	9.8	19.9	K. Sell et al. 2008	n=12
19.7	2.7	N	42.9	8.1	18.9	Golden & Vaccaro 1984	n=9; group 2
19.8	1.1	N	51.3	5.2	10.1	Kaminski et al. 1993	n=15
19.8	3.9	NS	43.2	4.8	11.1	Fox et al. 1975	n=10; exercise group 3
19.9	0.9	NS	55.8	3.5	6.3	Harms et al. 1995	n=8; low fat content
20.6	1.3	N	48.6	4.4	9.1	Browning et al. 2006	n=10
20.8	1.7	H	45.1	6.7	14.9	McComb et al. 2006	n=19
20.8	2.2	N	52.6	6.3	12.0	Wiley & Shaver 1972	n=35
20.8	2.4	NS	45.6	9.0	19.7	Lepp et al. 2013	n=22
20.9	1.9	H	42.0	7.2	17.1	Deschenes et al. 2006	n=9
21.0	0.9	NS	48.2	4.7	9.8	Ziemann et al. 2011	n=11; control group
21.0	9.3	NS	30.9	4.9	15.9	AM Miller et al. 2013	n=19; cancer siblings
21.1	1.5	N	54.3	4.2	7.7	Kanaley & Boileau 1988	n=10
21.2	1.6	N	54.8	4.9	8.9	FI Katch et al. 1974	n=50; treadmill data
21.4	1.4	NS	53.9	6.4	11.9	Cureton et al. 1995	n=22; multicenter study
21.4	2.4	H	46.2	6.2	13.4	V Katch & Henry 1972	n=35
21.6	1.1	NS	50.1	3.1	6.2	Ziemann et al. 2011	n=10; exercise group
21.8	3.4	H	46.2	4.7	10.2	Baldwin et al. 2000	n=6; control group
22.5	2.0	N	52.5	5.1	9.7	JD Brown et al. 2002	n=21
22.5	2.6	NS	52.9	4.7	8.9	JA Davis et al. 1976	n=39; treadmill; Note 2
22.6	2.5	N	44.7	9.9	22.1	AT Peterson et al. 1999	n=16
22.7	3.7	N	32.7	7.6	23.2	McDowell et al. 2003	n=16
22.9	2.5	NS	43.4	4.2	9.7	Rogowski et al. 2012	n=19
23.0	3.1	NS	46.5	7.0	15.1	JD George 1996	n=50
23.0	4.0	N	48.3	12.8	26.5	DW Hill 1996	n=12
23.0	4.0	N	50.1	8.4	16.8	McMiken 1976	n=30
23.0	5.0	N	44.0	8.3	18.9	J Kang et al. 2007	n=11
23.2	7.4	N	70.7	12.0	17.0	Trappe et al. 1996	n=15
23.3	2.8	H	58.9	6.7	11.4	Porcari et al. 1997	n=16; control group
23.6	4.8	N	43.7	10.0	22.9	Kendall et al. 2009	n=42
23.6	6.6	NS	55.0	5.3	9.6	DM Rogers et al. 1995a	n=15
24.0	2.6	N	45.7	7.9	17.3	CM Thomas et al. 1999	n=7; exercise group 3

Table 2. Estimates of $VO_{2.MAX}$ in males seen in the literature (continued)

Body Mass (BM)-Adjusted Estimates: Mean ± SD VO2Max Estimate (mL/kg-min)							
Age /Range		Health			COV		
Mean	SD	Status	Mean	SD	(%)	Citation	Comment
24.0	3.0	N	47.7	10.5	22.0	Kaminsky et al. 1987	n=14; ex group #1
24.0	3.0	N	40.2	6.3	15.7	Proctor et al. 2005	n=11
24.0	4.9	H	45.0	9.8	21.8	Zappe et al. 1996	n=6
24.0	9.0	NS	49.9	8.1	16.2	Swain et al. 1994	n=81
24.1	5.0	N	41.2	8.3	20.1	Gonzales & Scheun. 2006	n=11
24.6	3.4	N	35.5	3.1	8.7	KR Segal et al. 1985	n=10
24.8	2.9	H	42.2	3.7	8.8	JF Nichols et al. 1990	n=9
25.0	1.0	H	49.3	3.9	7.9	Davey et al. 1995	n=6
25.0	2.0	N	47.6	4.8	10.1	Coggan 1993	n=6
25.0	2.2	N	45.3	3.1	6.8	CM Thomas et al. 1999	n=5; exercise group 1
25.0	2.8	N	47.9	5.4	11.3	Kohrt et al. 1991	n=28
25.0	3.0	NS	44.1	5.7	12.9	Proctor et al. 1995	n=6
25.0	3.0	H	57.1	10.5	18.4	Pettitt et al. 2008	n=7
25.0	3.2	N	55.5	6.6	11.9	BE Hunt et al. 2001	n=10; untrained
25.3	2.0	N	50.2	6.2	12.4	Mahon et al. 1997b	n=9
25.6	1.6	H	56.8	10.7	18.8	K Sell et al. 2008	n=7
26.0	2.6	N	46.8	3.2	6.8	CM Thomas et al. 1999	n=7; control group
26.0	5.1	H	51.8	11.1	21.4	Swain et al. 1998	n=26
26.0	6.0	N	43.0	4.0	9.3	JO Hill et al. 1984	n=4; low-VO2max group
26.3	3.8	N	51.4	4.1	8.0	Bransford & Howley 1977	n=10; untrained
26.4	6.4	N	45.0	6.2	13.8	BJ Sawyer et al. 2010	n=23
26.4	8.5	NS	46.0	11.3	24.6	J Kang et al. 1999	n=17
26.6	6.3	N	48.3	7.4	15.3	Warr et al. 2013	n=76; pre-deployed NG
26.6	7.4	N	34.8	9.2	26.4	McMurray et al. 1998a	n=1396
26.8	6.4	N	42.2	6.1	14.5	Blessinger et al. 2009	n=19
26.9	3.8	N	43.0	5.1	11.9	Bullough et al. 1995	n=10
27.0	3.0	NS	42.9	3.7	8.6	Horton et al. 1998	n=6; untrained
27.0	7.1	N	40.9	8.7	21.3	Sheaff et al. 2010	n=26
27.0	7.8	NS	48.0	4.6	9.6	JQ Zhang et al. 1998	n=21
27.3	5.8	N	56.1	6.9	12.3	Latin & Elias 1993	n=28
27.6	3.8	H	46.1	8.5	18.4	Maresh et al. 1992	n=8
27.6	5.6	N	42.7	5.9	13.8	Katch & Katch 1973	n=75
27.8	5.8	H	46.0	7.0	15.2	LO Schulz et al. 1991	n=43
28.0	2.0	N	50.6	6.5	12.8	Kaminsky et al. 1987	n=10; ex. Group #2
28.0	7.0	N	29.0	5.6	19.3	Beere et al. 1999	n=13
28.4	4.8	H	41.8	10.4	24.9	Byrne et al. 1996	n=15
29.0	1.7	N	39.6	4.3	10.9	CM Thomas et al. 1999	n=3; exercise group 2
29.0	2.4	N	44.0	7.1	16.1	Tankersley et al. 1991	n=7
29.0	4.9	H	45.0	10.0	22.2	Sheffield-Moore et al. 2004	n=6
29.2	7.9	NS	49.9	7.5	15.0	L Kravitz et al. 1997	n=9
30.7	5.1	H	47.4	7.5	15.8	Rowland et al. 1997 MSSE	n=16
31.0	6.6	N	38.3	6.3	16.4	KR Segal et al. 1990	n=11
31.4	3.9	NS	41.9	4.3	10.3	Fox et al. 1975	n=10; exercise group 4
33.0	4.0	H	40.0	7.0	17.5	Fleg et al. 1993	n=21
33.8	6.2	H	53.8	7.4	13.8	Horton et al. 2006	n=13
34.0	3.0	N	37.2	8.0	21.5	Proctor et al. 2005	n=11
36.0	2.6	N	38.7	6.3	16.3	Kastello et al. 1993	n=7
36.5	2.3	H	40.6	7.3	18.0	Nelson et al. 2010	n=141; 30-39 cohort

Table 2. Estimates of $VO_{2.MAX}$ in males seen in the literature (continued)

Body Mass (BM)-Adjusted Estimates: Mean ± SD VO2Max Estimate (mL/kg-min)							
Age /Range		Health			COV		
Mean	SD	Status	Mean	SD	(%)	Citation	Comment
37.0	10.0	N	47.5	10.3	21.7	Convertino & Ludwig 2000	n=66
37.8	3.4	N	39.7	2.0	5.0	Shvartz 1996	n=6
38.0	12.8	H	44.0	9.1	20.7	Engels et al. 1988	n=104
39.0	6.0	N	41.7	3.7	8.9	Spielmann et al. 2011	n=102; multi-site study
39.0	9.0	H	47.0	6.0	12.8	Donovan et al. 2009	n=182
39.0	15.0	H	40.0	9.0	22.5	Ardestani et al. 2011	n=92
39.1	7.4	NS	34.8	9.0	25.9	JA Davis et al. 1999	n=7; control
42.0	14.0	H	33.0	8.6	26.1	NP Greene et al. 2011	n=24
43.0	7.2	NS	31.0	7.2	23.2	JA Davis et al. 1999	n=9; experimental
44.6	2.8	H	38.4	7.0	18.2	Nelson et al. 2010	n=398; 40-49 cohort
44.6	12.5	N	36.2	7.6	21.0	Talbot et al. 2002	n=522; BLSA
45.3	8.9	N	46.7	8.5	18.2	Trappe et al. 1996	n=15
46.0	4.0	N	34.6	6.6	19.1	Proctor et al. 2005	n=12; exercise group C
46.2	2.2	H	39.2	5.5	14.0	Dehn & Bruce 1972	n=26
46.4	11.9	NS	42.4	10.5	24.8	Kline et al. 1987	n=83; cross-validation group
46.5	10.7	NS	42.2	9.8	23.2	Kline et al. 1987	n=82; validation group
47.7	7.3	N	38.8	5.4	13.9	FW Kasch 1984	n=83
47.8	8.5	NS	37.5	6.7	17.9	Stefanick et al. 1998	n=190
48.7	6.0	H	36.9	7.9	21.4	Byrne et al. 1996	n=25
50.0	10.7	N	36.5	8.1	22.2	LT Weir et al. 2006	n=2417
50.1	5.8	N	35.5	7.9	22.3	McDonough et al. 1970	n=16
51.9	16.0	H	35.3	8.4	23.8	Fleg et al. 2005	n=435
52.0	10.0	N	36.2	4.4	12.2	Heath et al. 1981	n=9; lean, untrained
52.1	16.7	N	34.1	8.4	24.6	Talbot et al. 2000	n=619; BLSA
53.5	2.8	H	35.2	6.5	18.5	Nelson et al. 2010	n=235; 50-59 cohort
54.0	3.0	N	32.5	5.7	17.5	Proctor et al. 2005	n=101
54.6	3.0	H	37.7	4.5	11.9	Dehn & Bruce 1972	n=21
56.2	4.1	NS	29.9	5.0	16.7	DR Young et al. 1994	n=197
58.0	3.0	N	32.3	4.1	12.7	Proctor et al.1995	n=6
60.0	4.7	N	22.5	5.2	23.1	Carter et al. 1994	n=13; control group
60.6	6.3	N	20.4	6.3	30.9	Duscha et al. 2001	n=10
61.0	2.8	N	42.0	4.8	11.4	Kastello et al. 1993	n=8
61.0	3.0	NS	33.4	7.6	22.8	Hagberg et al. 1998	n=22
61.6	2.7	H	31.6	4.2	13.3	Nelson et al. 2010	n=42; 60-69 cohort
61.8	8.2	N	33.5	6.8	20.3	Guderian et al. 2010	n=10
62.0	6.0	N	34.9	3.3	9.5	Sheldahl et al. 1996	n=9
62.8	1.0	H	27.4	3.7	13.5	Seals et al. 1984	n=8
63.0	3.0	N	29.2	5.7	19.5	Coggan 1993	n=6
63.0	3.0	N	26.5	3.5	13.2	Proctor et al. 2005	n=10
63.0	6.0	H	30.5	5.0	16.4	Fleg et al. 1995b	n=23; control group
63.0	7.3	H	35.2	5.4	15.3	BE Hunt et al. 2001	n=6; untrained
63.7	3.1	N	27.5	4.2	15.3	Kohrt et al., 1998	n=53; experimental group
63.9	1.7	H	31.3	6.9	22.0	Dehn & Bruce 1972	n=10
64.0	4.0	N	17.7	6.5	36.7	Beere et al. 1999	n=10
64.8	3.6	N	28.3	4.3	15.2	Kohrt et al., 1998	n=19; control group
65.0	2.0	H	31.5	5.6	17.8	Davy et al. 1995	n=6
65.0	5.0	H	36.0	8.0	22.2	Gonzales et al. 2011	n=19
65.0	5.0	NS	27.1	5.8	21.4	Hagberg et al. 1998	n=21

Table 2. Estimates of $VO_{2.MAX}$ in males seen in the literature (continued)

Body Mass (BM)-Adjusted Estimates: Mean ± SD VO2Max Estimate (mL/kg-min)							
Age /Range		Health			COV		
Mean	SD	Status	Mean	SD	(%)	Citation	Comment
66.0	3.0	N	32.9	5.6	17.0	Tankersley et al. 1991	n=6
67.0	1.2	N	27.4	4.0	14.6	CM Thomas et al. 1999	n=4; exercise group 2
67.0	1.7	N	23.9	1.0	4.2	CM Thomas et al. 1999	n=3; exercise group 3
67.0	2.4	H	38.0	7.3	19.2	Zappe et al. 1996	n=6
67.7	3.7	N	27.0	7.2	26.7	McDowell et al. 2003	n=18
68.0	2.6	N	29.9	7.4	24.7	CM Thomas et al. 1999	n=7; control group
68.7	5.1	N	27.7	3.7	13.4	Panton et al. 1996	n=19
69.0	2.4	H	39.0	7.3	18.7	Sheffield-Moore et al. 2004	n=6
69.0	2.6	N	24.8	3.4	13.7	CM Thomas et al. 1999	n=7; exercise group 1
69.5	6.3	NS	20.7	6.7	32.4	Ainsworth et al. 1997	n=10
70.1	3.8	H	28.9	4.9	17.0	Fehling et al. 1999	n=44
70.6	9.0	NS	22.7	5.5	24.2	Fiser et al. 2010	n=25
71.4	6.3	N	28.2	5.0	17.7	Parise et al. 2004	n=95
72.1	7.6	N	23.7	4.0	16.9	Simonsick et al. 2006	n=56
72.5	4.9	N	21.7	4.8	22.1	Peterson et al. 2003	n=59
72.9	5.1	H	24.2	5.4	22.3	Deschenes et al. 2006	n=9
72.9	7.0	H	27.1	5.5	20.3	Byrne et al. 1996	n=35
73.5	5.9	N	27.6	6.0	21.7	Talbot et al. 2002	n=167; BLSA
74.0	4.0	N	24.6	5.6	22.8	Proctor et al. 2005	n=141; 30-39 cohort
74.0	5.0	H	29.0	5.0	17.2	Fleg et al. 1993	n=16; control group
74.7	2.8	N	23.5	3.8	16.2	Perini et al. 2000	n=12; 18.6-31.4
75.5	2.8	N	27.0	2.5	9.3	Benestad 1965	n=13
b. Complete age statistics are not provided							
6.0	-	N	39.1	2.8	7.2	DW Morgan et al. 1999	n=15
12 - 13	-	NS	43.0	-		Pate et al. 2006 APAM	50th percentile (CI: 42.1-44.5)
14 - 15	-	NS	45.8	-		Pate et al. 2006 APAM	50th percentile (CI: 44.2-48.1)
16 - 17	-	NS	46.2	-		Pate et al. 2006 APAM	50th percentile (CI: 45.1-47.3)
18 - 19	-	NS	46.3	-		Pate et al. 2006 APAM	50th percentile (CI: 45.2-47.6)
18 - 21	-	N	58.3	3.5	6.0	WL Daniels et al. 1982	n=11; Army cadets
19 - 47	-	H	47.9	6.0	12.5	Lukaski et al. 1989	n=16; treadmill value
20's	-	NS	43.8	9.4	21.5	Fleq et al. 1995a	n=13
20 - 29	-	N	44.7	3.9	8.7	Mitchell et al. 1958	n=36
28.6	-	NS	50.7	4.2	8.3	Diaz et al. 1978	n=7; treadmill only
20 - 39	-	N	45.8	4.8	10.5	Milesis et al. 1976	n=16; control group
20 - 39	-	N	45.0	7.4	16.4	Milesis et al. 1976	n=15; exercise group A
20 - 39	-	N	41.5	5.2	12.5	Milesis et al. 1976	n=17; exercise group B
20 - 39	-	N	45.4	6.5	14.3	Milesis et al. 1976	n=12; exercise group C
30's	-	NS	40.3	7.6	18.9	Fleq et al. 1995a	n=30
30 - 39	-	N	35.4	3.3	9.3	Mitchell et al. 1958	n=8
40's	-	NS	33.5	4.9	14.6	Fleq et al. 1995a	n=12
40 - 44	-	H	40.5	4.7	11.6	McDonough et al. 1970	n=10
40 - 49	-	N	35.4	3.3	9.3	Mitchell et al. 1958	n=8
44.4	-	H	34.5	5.2	15.1	Blumenthal et al. 1988	n=18
45 - 49	-	H	38.4	5.3	13.8	McDonough et al. 1970	n=24
45 - 59	-	H	31.0	5.0	16.1	DA Meyers et al. 1991	n=68
50's	-	NS	35.7	6.6	18.5	Fleq et al. 1995a	n=25
50 - 54	-	H	37.5	5.3	14.1	McDonough et al. 1970	n=20

Table 2. Estimates of $VO_{2.MAX}$ in males seen in the literature (continued)

Body Mass (BM)-Adjusted Estimates: Mean ± SD VO2Max Estimate (mL/kg-min)							
Age /Range Mean	SD	Health Status	Mean	SD	COV (%)	Citation	Comment
55 - 59	-	N	30.3	5.9	19.5	Hollenberg et al. 1998	n=79
55 - 59	-	H	36.2	5.7	15.7	McDonough et al. 1970	n=19
60's	-	NS	30.4	8.2	27.0	Fleq et al. 1995a	n=26
60 - 64	-	N	29.7	6.1	20.5	Hollenberg et al. 1998	n=66
60 - 64	-	H	32.6	4.7	14.4	McDonough et al. 1970	n=9
60 - 79	-	H	27.0	5.0	18.5	DA Meyers et al. 1991	n=64
65 - 69	-	N	26.9	5.8	21.6	Hollenberg et al. 1998	n=73
66.0	-	N	28.5	6.0	21.1	Hollenberg et al. 2006	n=253; exercise group #1
65 - 69	-	H	27.7	4.2	15.2	McDonough et al. 1970	n=3
70's	-	NS	30.2	5.6	18.5	Fleq et al. 1995a	n=14
70 - 74	-	N	26.5	4.3	16.2	Hollenberg et al. 1998	n=81
70.0	-	N	23.8	5.3	22.3	Hollenberg et al. 2006	n=189; exercise group #2
75 - 79	-	N	22.4	3.2	14.3	Hollenberg et al. 1998	n=42; 60-69 cohort
80's	-	NS	23.2	5.8	25.0	Fleq et al. 1995a	n=3
80 - 84	-	N	22.1	2.5	11.3	Hollenberg et al. 1998	n=18
>85	-	N	18.3	2.1	11.5	Hollenberg et al. 1998	n=4
Males: Active, Fit, or Athlete							
a. Mean & SD statistics are provided for age							
9.4	1.7	Act	42.7	5.5	12.9	Gilliam et al. 1977	n=32; exercisers
10.2	1.3	Act	49.0	5.7	11.6	Sady & Katch 1981	n=21
10.5	1.1	Ath	56.6	2.0	3.5	Mayers & Gutin 1979	n=8; cross-country runners
10.8	2.2	Fit	61.0	2.4	3.9	Kwee & Wilmore 1990	n=24
17.3	0.9	Ath	73.4	4.9	6.7	AS Cole et al. 2006	n=15; cross-country runners
19.5	0.8	Ath	66.6	3.7	5.6	McMiken & Daniels 1976	n=8
19.7	1.1	Fit	57.4	3.6	6.3	Ribisl & Kachadorian 1969	n=11
19.9	2.7	Act	50.7	4.8	9.5	Sharp et al. 2002	n=122
20.0	1.6	Ath	69.3	2.8	4.0	Bransford & Howley 1977	n=10; distance runners
20.0	4.0	Fit	62.0	6.0	9.7	JO Hill et al. 1984	n=4; high-VO2 group
20.4	2.0	Ath	50.2	5.3	10.6	Vander et al. 1984	n=7; national-class fencers
20.1	1.6	Act	50.4	4.0	7.9	Dolezal & Potteiger 1998	n=10
20.1	1.6	Act	50.7	5.8	11.4	Dolezal & Potteiger 1998	n=10
20.1	1.6	Act	52.3	4.4	8.4	Dolezal & Potteiger 1998	n=10
20.5	1.8	Ath	59.2	3.9	6.6	Peyer et al. 2011	n=13; hockey forwards
20.5	1.9	Fit	56.0	6.8	12.1	Jeans et al. 2011	n=6; range of VO2: 51.5-64.0
20.5	2.1	Ath	56.2	1.9	3.4	Peyer et al. 2011	n=11; hockey defensemen
20.7	2.8	Act	53.8	5.3	9.9	Nindl et al. 1998	n=20; Army personnel
21.0	2.0	Fit	61.5	7.7	12.5	Darling et al. 2005	n=10; range=43-71
21.0	3.3	Act	44.5	5.0	11.2	Proctor et al. 2003	n=11
21.1	1.7	Ath	63.0	7.0	11.1	SR Hopkins et al. 1998	n=6
21.8	3.4	Act	50.6	6.2	12.3	Sharp et al. 2002	n=171
22.0	2.0	Ath	69.0	2.3	3.3	Heath et al. 1981	n=16
22.1	2.4	Fit	56.5	3.2	5.7	Gist et al., 2014	n=8; moderately-trained
22.6	3.1	Act	45.6	7.2	15.8	Astorino et al. 2012	n=5; recreationally active
22.7	3.7	Ath	57.1	6.0	10.5	McDowell et al. 2003	n=21; track runners
23.0	4.0	Ath	54.8	4.1	7.5	Gale & Flynn 1974	n=8; competitive wrestlers
23.0	5.0	Act	44.0	8.3	18.9	J Kang et al. 2008	n=11
23.4	4.5	Fit	69.1	5.0	7.2	Pereira & Freedson 1997	n=7; highly trained
23.5	1.0	Ath	70.6	4.5	6.4	Wilhite et al. 2013	n=6; wlite distance runners

Table 2. Estimates of $VO_{2.MAX}$ in males seen in the literature (continued)

Body Mass (BM)-Adjusted Estimates: Mean ± SD VO2Max Estimate (mL/kg-min)							
Age /Range		Health			COV		
Mean	SD	Status	Mean	SD	(%)	Citation	Comment
23.5	4.5	Act	43.8	6.1	13.9	Astorino et al. 2010	n=13; recreationally active
23.9	1.3	Fit	59.2	8.4	14.2	C Kaufman et al. 2006	n=?; high-fit (active)
23.9	1.3	Ath	63.4	6.6	10.4	Drenowatz & Eisenman 2011	n=10; endurance run.
24.0	2.0	Ath	67.0	3.0	4.5	Romijn et al. 1993	n=5; endurance cyclists
24.0	5.7	Ath	62.0	4.0	6.5	Proctor et al. 1998	n=8
24.0	7.8	Act	54.8	10.5	19.2	Kist et al. 2013	n=11; aerobically trained
24.1	2.5	Act	55.8	4.2	7.5	Duncan et al. 1997	n=10; continuous treadmill
24.2	2.7	NS	47.8	6.3	13.2	JD George et al. 1998	n=36; test of protocol
24.2	5.7	Act	53.4	9.7	18.2	Beckham & Earnest 2000	n=12; 75% active
24.2	6.0	Act	48.5	6.2	12.8	Kolkhorst et al. 1994	n=9
24.3	3.1	Fit	68.1	5.7	8.4	JF Nichols et al. 1990	n=8; runners
24.3	3.8	Fit	44.7	7.6	17.0	Bloomer 2005	n=10; resistance & aerobic fit
24.5	4.7	Ath	77.0	8.0	10.4	LO Schultz et al. 1991	n=20; endurance trained
25.0	2.0	Fit	66.3	2.5	3.8	Proctor et al. 1995	n=6
25.0	4.0	Fit	64.4	3.7	5.7	Horton et al. 1998	n=8; competitive cyclists
25.0	5.0	Act	64.9	5.3	8.2	Hagberg et al. 1988	n=11
25.0	4.2	Fit	61.0	4.9	8.0	Sparling & Cureton 1983	n=34; distance runners
25.0	7.0	Fit	64.0	2.8	4.4	JM Wilson et al. 2010	n=10; endurance runners
25.3	5.5	Act	45.6	4.0	8.8	Astorino et al. 2012	n=11; recreationally active
25.7	2.3	Ath	61.1	4.1	6.7	JL Thompson et al. 1995	n=6; group 1
25.7	3.5	Fit	68.8	5.3	7.7	Trappe et al. 1996	n=10
26.0	4.9	Fit	42.7	2.7	6.3	Kenny & Ho 1995	n=?; VO2 range: 39.9-46.8
26.0	6.3	Ath	61.1	5.7	9.3	Sedlock et al. 1989	n=10; triathletes
26.1	5.7	Fit	62.9	4.7	7.5	Bullough et al. 1995	n=10
26.1	6.9	Ath	68.6	6.3	9.2	Powers et al. 1983	n=9; distance runners
26.5	2.2	Ath	75.6	3.2	4.2	DW Morgan et al 1995	n=22; elite runners
26.6	6.1	Fit	52.2	5.4	10.3	Crawford et al. 2011	n=44; Army personnel BF≤18%
26.8	4.5	Ath	62.5	5.0	8.0	JL Thompson et al. 1995	n=4; group 2
27.0	4.0	Ath	66.3	4.4	6.6	Coggan 1993	n=6; endurance runners
27.0	2.8	Fit	62.0	5.7	9.2	Monahan et al. 2001	n=8
27.0	6.2	Act	61.3	5.6	9.1	Van Pelt et al. 2001	n=39; exercisers
27.1	5.0	Act	56.7	5.8	10.2	Quindry et al. 2013	n=12; recreationally active
27.1	6.7	Act	64.1	11.0	17.2	Trappe et al. 1996	n=18
27.3	3.6	Fit	70.5	4.0	5.7	DW Morgan et al. 1989	n=13
27.6	4.6	Fit	48.2	6.1	12.7	Dalleck & Kravitz 2006	n=12; moderate exercisers
27.6	7.2	Fit	63.4	6.6	10.4	DW Morgan et al 1995	n=41
27.7	6.3	Ath	63.4	6.7	10.6	Gojanovic et al. 2012	n=9; distance runners
28.0	3.0	Act	63.5	4.4	6.9	Ogawa et al 1992	n=14
28.0	4.0	Ath	54.3	6.5	12.0	Gale & Flynn 1974	n=9; Olympic team wrestlers
28.0	4.0	Fit	58.4	8.7	14.9	DeSouza et al. 2000	n=12; endothelial dysfunction
29.0	4.0	Ath	61.9	4.9	7.9	J Thompson & Manore 1996	n=24; endurance trained
29.2	6.8	Fit	27.6	7.2	26.1	Dalleck & Kravitz 2006	n=24; moderate exercisers
28.3	4.5	Ath	65.8	6.3	9.6	Baldwin et al. 2000	n=7; endurance trained
29.0	4.5	Fit	62.0	4.5	7.3	Tanaka et al. 2002	n=20; endurance-trained
29.8	3.9	Fit	49.9	4.3	8.6	Sandoval & Matt 2002	n=15
29.9	9.1	Act	63.5	6.4	10.1	Weltman et al. 1990	n=31; runners
30.0	5.6	Act	54.7	6.6	12.1	Sady & Katch 1981	n=21
30.0	6.0	Act	53.6	10.0	18.7	Fedel et al. 1995	n=12; competitive skaters

Table 2. Estimates of $VO_{2.MAX}$ in males seen in the literature (continued)

Body Mass (BM)-Adjusted Estimates: Mean ± SD VO2Max Estimate (mL/kg-min)							
Age /Range		Health			COV		
Mean	SD	Status	Mean	SD	(%)	Citation	Comment
30.4	7.6	ACT	45.0	3.7	8.2	EL Melanson et al. 2002	n=8; lean exercisers
30.6	7.2	Fit	44.1	6.8	15.4	Crawford et al. 2011	n=55; Army men BF >18%
31.0	5.4	Fit	58.3	5.4	9.3	Pereira & Freedson 1997	n=8; moderately trained
31.8	9.2	Fit	56.1	7.9	14.1	Dumke et al. 2006	n=10; trained cyclists
32.3	6.4	Act	54.8	6.3	11.5	TE Ward et al. 1998	n=17; Bruce treadmill test
32.8	6.7	Fit	66.0	7.0	10.6	McDaniel et al. 2002	n=9; trained cyclists
34.6	2.8	H	50.6	10.0	19.8	CB Scott & Bogdanffy 1998	n=12
35.0	6.2	Ath	66.6	8.8	13.2	Costill et al. 1973	n=12; distance runners
36.9	5.0	Fit	59.2	4.1	6.9	DW Morgan et al. 1995	n=16
37.3	3.9	Fit	63.3	2.2	3.5	Shvartz 1996	n=6
39.0	3.0	Ath	60.7	5.1	8.4	Peiffer et al. 2008	n=14; Cyclist (CI 52.3-69.8)
39.0	11.1	Fit	53.4	8.4	15.7	Malek et al. 2004	n=93; aerobically trained
39.9	6.2	Fit	48.6	5.8	11.9	Ribisl & Kachadorian 1969	n=24; moderately well-trained
44.5	2.8	Ath	58.7	9.5	16.2	SA Hawkins et al. 2001	n=31; visit 1
44.6	6.9	Act	44.6	6.7	15.0	FW Kasch et al. 1985	n=15;longitudinal yr 1 study
46.5	6.1	Ath.	47.2	7.2	15.3	Barnard et al. 1979	n=13; sprinters
46.8	9.8	Ath	60.3	13.3	22.1	Trappe et al. 1996	n=10
47.2	3.8	Fit	59.2	4.9	8.3	Trappe et al. 1996	n=10
47.2	5.8	Act	37.0	5.3	14.3	Loftin et al. 1996	n=12
48.7	7.6	Act	48.9	5.9	12.1	Trappe et al. 1996	n=18
49.0	3.0	Ath	55.2	6.6	12.0	Peiffer et al. 2008	n=10; cyclist CI: 43.7-64.6
49.0	4.4	Fit	52.0	4.4	8.5	Tanaka et al. 2002	n=19; endurance-trained
50.8	4.1	Act	51.0	13.5	26.5	TE Ward et al. 1998	n=10; Bruce treadmill test
51.4	6.8	Fit	38.0	6.5	17.1	McDonough et al. 1970	n=60
53.2	7.8	H	37.7	7.6	20.2	McDonough et al. 1970	n=34; runners
53.5	3.3	Ath	50.4	8.4	16.7	SA Hawkins et al. 2001	n=31; visit 2
53.9	2.9	Ath	53.4	8.2	15.4	SA Hawkins et al. 2001	n=34; visit 1
56.2	6.9	Act	45.2	10.0	22.1	FW Kasch et al. 1985	n=15; longitudinal study: yr 2
55.2	8.6	Fit	45.0	5.4	12.0	Coyle et al. 1983	n=6; exercisers
55.3	11.2	Ath	54.4	10.8	19.9	Barnard et al. 1979	n=13; endurance trained
57.0	4.0	Act	52.7	3.3	6.3	Proctor et al. 1995	n=6
59.0	6.0	Ath	58.7	4.3	7.3	Heath et al. 1981	n=16; Masters athlete
59.0	6.9	Act	54.8	8.0	14.6	BE Hunt et al. 2001	n=12
59.1	7.6	Act	31.4	10.2	32.5	Nikolai et al. 2009	n=7; in a water exercise class
59.6	8.5	Fit	49.9	5.4	10.8	Schulman et al. 1996	n=8; endurance trained
60.0	6.9	Act	40.2	9.3	23.1	FW Kasch et al. 1985	n=15;longitudinal study: yr 3
60.0	8.6	Fit	53.3	5.4	10.1	Pollock et al. 1987	n=11; competitive athlete
62.0	2.0	Fit	52.0	2.7	5.2	Coggan et al. 1993	n=6; endurance runners
62.0	8.9	Ath	54.0	6.6	12.2	MA Rogers et al. 1990	n=15
62.2	3.5	Ath	46.2	8.2	17.7	SA Hawkins et al. 2001	n=34; visit 2
62.3	2.9	Ath	46.2	9.0	19.5	SA Hawkins et al. 2001	n=13; visit 1
63.0	5.7	Act	42.3	7.4	17.5	Van Pelt et al. 2001	n=32; exercisers
63.0	3.0	Act	47.6	4.3	9.0	Ogawa et al 1992	n=14
63.0	5.6	Fit	45.0	2.8	6.2	Monahan et al. 2001	n=8
63.0	9.0	Ath	42.6	8.9	20.9	DeSouza et al. 2000	n=20; endothelial dysfunction
63.1	6.9	Act	43.1	8.4	19.5	FW Kasch et al. 1985	n=13;longitudinal study: yr 4
63.4	6.5	Ath	49.6	5.8	11.7	Katzel et al. 2001	n=42
63.7	5.0	Act	31.9	3.6	11.3	Hageman et al. 2000	n=9; resistance group

Table 2. Estimates of $VO_{2.MAX}$ in males seen in the literature (continued)

Body Mass (BM)-Adjusted Estimates: Mean ± SD VO2Max Estimate (mL/kg-min)							
Age /Range Mean	SD	Health Status	Mean	SD	COV (%)	Citation	Comment
64.0	4.9	Fit	41.8	2.9	`	WL Kenny & Ho 1995	n=6; VO2 range: 39.1-45.6
64.0	5.7	Ath	45.9	4.5	9.8	Proctor et al. 1998	n=8
64.2	9.4	Fit	45.9	6.7	14.6	Pollock et al. 1987	n=13; post-competitive athlete
65.0	4.0	Ath	45.9	4.6	10.0	Peiffer et al. 2008	n=8;Cyclists: CI 38.2-51.7
65.0	4.0	Act	50.0	4.8	9.6	Hagberg et al. 1988	n=11
65.0	6.0	Fit	43.3	6.3	14.5	JF Nichols et al. 1990	n=9
65.0	8.0	Ath	47.2	5.9	12.5	Fleg et al. 1995b	n=16;Endurance runners
66.0	3.0	Ath	46.4	5.1	11.0	Tankersley et al. 1991	n=7; Master runners
66.0	5.6	Act	31.4	4.8	15.3	Proctor et al. 2003	n=11
66.0	8.5	Ath	48.0	4.2	8.8	Goldberg et al. 2000	n=18;Endurance runners
66.2	6.5	Act	33.1	6.2	18.7	Hageman et al. 2000	n=9; control group
66.3	11.6	Ath	36.5	17.2	47.1	Wilund et al. 2008	n=7; Master athlete
67.0	8.2	Fit				Tanaka et al. 2002	n=17; endurance-trained
67.7	3.7	Act				McDowell et al. 2003	n=18; endurance runners
68.0	3.7	Act				Shi et al. 2008	n=8
68.4	9.8	Ath				Trappe et al. 1996	n=10
69.0	3.5	Act	45.0	6.9	15.3	BD Johnson et al. 1991	n=12
70.4	8.8	Ath	40.5	8.9	22.0	Pollock et al. 1997	n=21; still competes
71.1	3.2	Ath	36.4	9.4	25.8	SA Hawkins et al. 2001	n=13; visit 2
76.0	4.8	Ath	41.5	8.8	21.2	SA Hawkins et al. 2001	n=8; visit 1
82.8	4.0	Ath	28.4	7.6	26.8	SA Hawkins et al. 2001	n=8; visit 2
b. Complete age statistics are not provided							
6--10	-	Ath	62.7	6.1	9.7	Eisenmann et al. 2001	n=13; distance runners
10.0	-	Ath	61.1	4.9	8.0	Daniels et al. 1978	n=4; mid-dist runner
11.0	-	Ath	63.6	7.1	11.2	Eisenmann et al. 2001	n=12; distance runners
12.0	-	Ath	63.3	6.3	10.0	Eisenmann et al. 2001	n=14; distance runners
12.0	-	Ath	59.0	6.6	11.2	Daniels et al. 1978	n=4; mid-dist runner
12.0	-	Ath	62.7	5.2	8.3	Daniels et al. 1978	n=7; mid-dist runner
13.0	-	Ath	60.8	7.2	11.8	Eisenmann et al. 2001	n=16; distance runners
14.0	-	Ath	63.5	5.2	8.2	Eisenmann et al. 2001	n=20; distance runners
15.0	-	Ath	62.7	6.3	10.0	Eisenmann et al. 2001	n=16; distance runners
16.0	-	Ath	64.8	5.0	7.7	Eisenmann et al. 2001	n=14; distance runners
17.0	-	Ath	67.5	5.6	8.3	Eisenmann et al. 2001	n=20; distance runners
17.0	-	Ath	61.2	4.4	7.2	Daniels et al. 1978	n=7; mid-dist runner
18.0	-	Ath	67.3	8.0	11.9	Eisenmann et al. 2001	n=14; distance runners
20.0	-	Act	50.2	6.5	12.9	Sonna et al. 2001	n=116; non-participants
21.0	-	Act	51.1	6.5	12.7	Sonna et al. 2001	n=66; participants
20 - 33	-	Ath	63.3	8.0	12.6	Ferley et al. 2013	n=12; group 1
20 - 33	-	Ath	59.4	8.9	15.0	Ferley et al. 2013	n=12; group 2
20 - 33	-	Ath	59.9	8.6	14.4	Ferley et al. 2013	n=8; group 3
25 - 34	-	Act	42.5	5.1	12.0	Bruce 1984	n=?
35 - 44	-	Act	39.9	5.4	13.5	Bruce 1984	n=?
40 - 49	-	Ath	57.5	-	-	Pollock et al. 1974	n=11; VO2 range 46-64
45 - 54	-	Act	37.0	5.3	14.3	Bruce 1984	n=?
50 - 59	-	Ath.	54.4	-		Pollock et al. 1974	n=5; VO2 range 49-57
55 - 64	-	Act	33.3	4.4	13.2	Bruce 1984	n=?
60 - 69	-	Ath	51.4	-		Pollock et al. 1974	n=6; VO2 range 40-61
70 - 79	-	Ath	40.0	-		Pollock et al. 1974	n=3; VO2 range 38-41

Table 2. Estimates of $VO_{2.MAX}$ in males seen in the literature (continued)

Body Mass (BM)-Adjusted Estimates: Mean ± SD VO2Max Estimate (mL/kg-min)							
Age /Range Mean	SD	Health Status	Mean	SD	COV (%)	Citation	Comment
Males: Sedentary, Overweight, or Obese a. Mean & SD statistics are provided for age							
11.2	1.6	OW	37.5	6.9	18.4	Byrd-Williams et al. 2008	n=84; Hispanic
13.3	1.5	Sed	35.2	2.7	7.7	Kwee & Wilmore 1990	n=31; "low fit"
14.1	1.3	O	25.6	3.0	11.7	Gutin et al. 2002	n=16; black adolescents
14.5	1.3	O	31.0	8.5	27.4	Gutin et al. 2002	n=10; white adolescents
19.1	1.6	O	34.4	3.8	11.0	Wolfe et al. 1976	n=12; "moderately obese"
19.8	3.2	Sed	31.6	5.6	17.7	Lawrenson et al. 2003	n=6
17.7	3.6	OW	47.1	3.6	7.6	Harms et al. 1995	n=8; high fat content
22.0	4.0	OW-Sed	39.2	5.2	13.3	Potteiger et al. 2008	n=16; exercise group
22.9	5.0	O	31.0	5.6	18.1	Kyriazis et al. 2007	n=7; control group
23.9	3.8	O-Sed	39.6	5.7	14.4	Washburn et al. 2003	n=17
24.0	4.0	OW-Sed	39.5	5.7	14.4	Potteiger et al. 2008	n=15; control group
24.1	1.6	Sed	40.1	3.0	7.5	C Kaufman et al. 2006	n=?; low-fit
24.5	3.3	Sed	46.7	2.1	4.5	MJ Turner et al. 1999	n=9
25.4	4.5	O	28.9	5.4	18.7	KR Segal et al. 1985	n=10
25.6	7.0	O	35.1	7.5	21.4	Browning et al. 2006	n=10
26.0	3.0	Sed	46.4	4.0	8.6	Hagberg et al. 1988	n=13
26.0	3.0	Sed	49.3	4.7	9.5	Hoetzer et al. 2007	n=10
26.0	5.7	Sed	44.2	6.8	15.4	Van Pelt et al. 2001	n=32
26.3	3.6	Sed	51.4	3.9	7.6	DW Morgan et al. 1995	n=10
26.4	5.4	O	33.6	5.4	16.1	Kyriazis et al. 2007	n=8; exercise group
27.0	2.8	Sed	45.0	5.7	12.7	Monahan et al. 2001	n=8
27.0	3.0	Sed	45.9	6.1	13.3	Ogawa et al 1992	n=14
27.0	4.0	Sed	41.8	6.9	16.5	DeSouza et al. 2000	n=12; endothelial dysfunction
28.0	5.4	Sed	41.0	10.7	26.1	Tanaka et al. 2002	n=29
30.7	8.6	OW	39.7	4.7	11.8	Lennon et al. 1985	n=11; control group
31.4	3.1	Sed	42.6	9.5	22.3	Lieber et al. 1989	n=12; exercise group #1
31.8	6.9	O	35.4	6.6	18.6	Sopko et al. 1984	n=21
31.9	3.0	Sed	40.0	4.9	12.3	Lieber et al. 1989	n=12; exercise group #2
32.0	6.6	O	29.2	4.0	13.7	KR Segal et al. 1990	n=11
32.2	1.8	NF	26.3	3.6	13.7	Shvartz 1996	n=5
32.2	7.2	OW	40.5	3.4	8.4	Lennon et al. 1985	n=12; exercise group #2
32.3	2.2	Sed	40.0	4.1	10.3	Lieber et al. 1989	n=10; control group
32.3	15.2	Sed	32.9	9.8	29.8	Rynders et al. 2011	n=74
32.9	2.5	Sed	38.5	5.2	13.5	Maliszewski et al. 1995	n=10; range=30.3-48.6
33.9	11.7	Sed	33.3	6.1	18.3	Skinner et al. 2001	n=78; black
36.4	15.0	Sed	37.3	9.0	24.1	Skinner et al. 2001	n=209; white
37.0	5.5	OW	37.6	2.3	6.1	Lennon et al. 1985	n=12; exercise group #1
41.0	6.0	O	28.3	5.5	19.4	Greene et al. 2012	n=10
43.3	6.6	OW-O	37.2	7.2	19.4	CW Hall et al. 2012	n=9
45.7	2.1	Sed	33.2	4.5	13.6	Dehn & Bruce 1972	n=15
47.0	4.0	Sed	37.7	7.0	18.6	Hoetzer et al. 2007	n=15
47.7	7.2	H	32.0	8.0	25.0	McDonough et al. 1970	n=16
50.0	5.3	Sed	34.0	5.3	15.6	Tanaka et al. 2002	n=28
50.0	6.0	OW	30.4	2.8	9.2	Heath et al. 1981	n=9; sedentary
52.1	9.1	Sed	24.2	6.9	28.5	Costill et al. 1974	n=24; control group
52.2	6.6	OW-Sed	32.1	3.9	12.1	JL Robbins et al. 2009	n=9

Table 2. Estimates of $VO_{2.MAX}$ in males seen in the literature (continued)

Body Mass (BM)-Adjusted Estimates: Mean ± SD VO2Max Estimate (mL/kg-min)							
Age /Range		Health			COV		
Mean	SD	Status	Mean	SD	(%)	Citation	Comment
55.0	2.7	Sed	31.2	4.7	15.1	Dehn & Bruce 1972	n=10
58.0	10.0	Sed	31.8	5.4	17.0	DeSouza et al. 2000	n=24; endothelial dysfunction
60.0	5.1	Sed	32.1	4.4	13.7	Schulman et al. 1996	n=10
61.1	6.2	Sed	30.1	5.5	18.3	Katzel et al. 2001	n=47
61.4	5.2	Sed	33.9	6.4	18.9	MA Rogers et al. 1990	n=14
62.0	5.8	Sed	31.0	6.4	20.6	Van Pelt et al. 2001	n=34
63.0	3.0	Sed	27.2	5.1	18.8	Ogawa et al 1992	n=3
63.0	5.0	Sed	33.6	6.4	19.0	Hoetzer et al. 2007	n=21
63.0	6.9	Sed	34.0	.		Goldberg et al. 2000	n=12; lean body mass
63.0	5.1	O,Sed	26.0	5.1	19.6	Goldberg et al. 2000	n=26; obese
64.0	3.0	Sed	29.6	4.1	13.9	Ehsani et al. 2003	n=10
64.7	1.5	Sed	28.8	3.1	10.8	Dehn & Bruce 1972	n=3
65.0	2.8	Sed	29.0	2.8	9.7	Monahan et al. 2001	n=8
65.0	3.0	Sed	24.9	5.0	20.1	DeSouza et al. 1997	n=11; normotensive
65.0	4.9	Sed	29.0	4.9	16.9	Tanaka et al. 2002	n=24
66.0	5.0	Sed	27.0	2.2	8.1	Hagberg et al. 1988	n=10
66.4	5.6	Sed	28.0	3.6	12.9	MJ Turner et al. 1999	n=11
66.5	5.1	Sed	20.3	4.4	21.7	Lawrenson et al. 2003	n=6
66.7	14.9	Sed	25.4	13.7	53.9	Wilund et al. 2008	n=6
67.5	5.4	Sed	25.2	3.4	13.5	Shi et al. 2008	n=8
68.7	4.8	Sed	26.3	5.2	19.8	JF Nichols et al. 1990	n=19
69.0	4.0	Sed	23.6	3.5	14.8	DeSouza et al. 1997	n=12; hypertensive
76.0	9.0	Sed	21.4	6.3	29.4	EP Weiss et al. 2006	n=33
b. Complete age statistics are not provided							
18 - 23	-	OW	54.4	6.9	12.7	O'Leary & Stavrianeas 2012	n=8
25 - 34	-	Sed	36.7	5.6	15.3	Bruce 1984	n=?
35 - 44	-	Sed	36.6	4.3	11.7	Bruce 1984	n=?
45 - 54	-	Sed	32.7	4.7	14.4	Bruce 1984	n=?
55 - 64	-	Sed	29.8	4.8	16.1	Bruce 1984	n=?
60 - 72	-	Sed	26.9	2.8	10.4	Frontera et al. 1990	n=12
Males: Health & Other Issues							
11.5	2.9	A	35.8	9.5	26.5	Boas et al. 1999	n=22
11.6	2.8	CF	41.4	7.5	18.1	Boas et al. 1999	n=25
18.8	-	SC	33.8	-	-	JR Robinson et al. 1976	n=16; A anemia only
20.0	-	SC	33.2	-	-	JR Robinson et al. 1976	n=16; Hbs trait & anemia
21.4	2.6	MR	41.2	11.2	27.2	Draheim et al. 1999	n=10
21.4	8.4	CC	28.5	5.8	20.4	AM Miller et al. 2012	n=38
26.7	5.9	DS	27.6	6.4	23.2	Fernhall et al. 1996	n=31; 12 y mean post-treatment
26.9	6.4	MR	32.7	7.2	22.0	Fernhall et al. 1996	n=35;no DS
41.0	9.0	MS	42.0	5.0	11.9	Donovan et al. 2009	n=32
43.9	9.4	HBP	31.0	6.7	21.6	Eicher et al. 2010	n=45
45.7	5.0	Blind	24.0	2.9	12.1	W Siegel et al. 1970	n=9; cycle ergometry
49.9	11.6	MI	25.5	7.1	27.8	Pinkstaff et al. 2011	n=157
52.1	9.1	IHD	20.0	6.4	32.0	Costill et al. 1974	n=24; experimental group
52.1	9.1	IHD	18.7	4.9	26.2	Costill et al. 1974	n=20; no experiment group
55.6	6.6	IHD	37.0	4.7	12.7	Coyle et al. 1983	n=6; trained (exercise program)
56.0	10.0	HF	15.4	-		Keteyian et al. 2010	n=160; COV=5.9%
57.4	12.5	CHF	14.8	2.5	16.9	Duscha et al. 2001	n=25

Table 2. Estimates of $VO_{2.MAX}$ in males seen in the literature (continued)

Age /Range		Health	Body Mass (BM)-Adjusted Estimates: Mean ± SD VO2Max Estimate (mL/kg-min)				
Mean	SD	Status	Mean	SD	COV (%)	Citation	Comment
61.0	11.0	IHD	19.3	6.1	31.6	Ades et al. 2006	n=2081; multicenter study
61.2	12.0	HP	31.0	9.3	30.0	Shultz et al. 2010	n=60
64.0	3.0	CAD	27.6	5.7	20.7	Sheldahl et al. 1996	n=9; exercise group #2
64.0	11.0	CHF	14.2	2.6	18.3	Bowen et al. 2012	n=24; med. ramp protocol
65.3	6.5	COPD	17.0	5.6	32.9	Carter et al. 1994	n=32; mild airflow limitation
65.9	6.0	COPD	9.9	2.7	27.3	Montes de Oca et al. 1996	n=25; severe obstruction
66.3	6.2	COPD	16.2	4.9	30.2	Carter et al. 1994	n=57; mod. airflow limitation
66.6	6.5	COPD	13.5	3.8	28.1	Carter et al. 1994	n=176; severe airflow limitation
68.0	5.0	HP	21.0	5.0	23.8	Ades et al. 1993	n=30
68.0	5.7	CAD	25.3	2.8	11.1	Sheldahl et al. 1996	n=8; exercise group #1
69.0	3.3	CAD	26.0	5.3	20.4	Sheldahl et al. 1996	n=11; exercise group #3
76.0	9.0	MI	24.0	4.0	16.7	Fleg et al. 1993	n=8; "silent" MI

Abbreviations	
A	Asthmatic
AA	African American
Act	Active (but non-athletes)
Ath	Athletes
BF	Body fat
BLSA	Baltimore Longitudinal Study of Aging
CAD	Coronary artery disease
CC	Cancer survivor
CF	Cystic Fibrosis
CHD	Chronic heart disease
CHF	Chronic heart failure
COPD	Chronic obstructive pulmonary disease
COV	Coefficient of variation
DS	Down syndrome
Fit	Very active healthy exercisers
Frail	Mild-to-moderate frailty
H	Healthy
HBP	High blood pressure
HP	heart patients
IHD	Ischemic heart disease

Abbreviations	
MI	Myocardial iscemia
MR	Mentally retarded (some with Down syndrome)
MS	Metabolic syndrome
N	Normal (mostly healthy)
NF	Not fit; poor fitness
NG	National Guard (all types)
NS	Not specified
O	Obese
OW	Overweight
Pg	Pregnant
SC	Sickle cell
Sed	Sedentary

Overall, we reviewed 1,649 papers for compilation of extant $VO_{2.MAX}$ data. Our cut-off date for a paper to be included in Tables 1 & 2 was January 1, 2015. Useable data—having both age- and gender-specific attributes--were obtained and entered into the Tables from 381 of the papers that we reviewed (23.1%). These papers provided 1,025 "lines" (entries) of data: 909 entries having sample mean age information, and 116 having only sample age range data. Of the 1,268 papers that were not used (76.9% of the total), 507 were from non-US studies and 761 had some type of data "issue." Data issues included presenting only mixed-gender results, using a protocol other than treadmill or cycle ergometer to estimate $VO_{2.MAX}$, or providing $VO_{2.MAX}$/ $VO_{2.PEAK}$ data only in absolute or in per-LBM units.

It should be noted that all of the data in Tables 1 and 2 are cross-sectional in nature, even though some are from "longitudinal" studies—usually a single measurement for multiple time periods, often separated by years. Basically these are treated as separate cross-sectional studies.

The coefficient of variation (COV), which is the standard deviation of the sample divided by its mean, is an indicator of the relative amount of variation in a sample. It usually is expressed as a percentage, and it is a useful metric for describing innate "inter-individual" (across subjects) variability in the sample. COV's are provided in Table 1 & 2 —where possible—for the $VO_{2.MAX}$ metric. As can be seen, the range of inter-individual variation in $VO_{2.MAX}$ among the various samples is large, generally between 10-30%. Studies having a $VO_{2.MAX}$ mean with a COV <7% and >35%

should be viewed skeptically; studies with a $VO_{2.MAX}$COV outside of the 7-35% range probably have a highly biased sample.

The studies in Tables 1 & 2 rarely provide distributional information regarding variability in $VO_{2.MAX}$ measurements seen in the tested samples, but Safrit & Wood (1995) and Pate et al. (2006) do so. Their data appear here as Table 3. The 5th-95th *range* for college-aged students is ~55% of the median male value and ~36% of the median female value. The equivalent COV's for these two samples is about 22% and 18%, respectively, not very different than those seen in Tables 1 & 2. The 10th-90th range for youth listed in Table 3 varies between 43-51% of the median estimate, with no trend discernable by age grouping. Basically there is a lot of age-/gender-specific inter-individual variability in the $VO_{2.MAX}$ metric, and our exposure models should capture that. With respect to distributional aspects of the Safrit & Wood (1995) data, for females they follow a normal probability distribution between the 10th and 95th percentiles, while they are normally distributed for males between the 5th and 95th percentile (data not shown). For adolescents, males have a longer-tail $VO_{2.MAX}$ distribution than females; otherwise they are parallel (data not shown).

Concepts

$VO_{2.MAX}$ (or $VO_{2.PEAK}$) is considered by many to be the "standard" measure of physical fitness and/or the functional limit of a person's cardiopulmonary system (Balady et al., 2010; Foucquirer et al., 2013; Jacobs et al., 1997; Kemper & Verschuur, 1985; McArdle et al., 2001, Mitchell et al., 1958; Sharkey 1984; Wagner, 1996). It is a reliable measure of impairment of oxygen delivery to cardiovascular and muscular systems (Wagner, 2010), and thus is an indicator of heart problems irrespective of their cause (Weber et al., 1988). $VO_{2.MAX}$ is much lower in people with decreased exercise capacity due to cardiovascular issues (Forman et al., 2009), and it is this capacity that more accurately predicts mortality in people with a variety of coronary risk factors than alternative respiratory metrics that have been evaluated (Franklin, 2000, 2007).

It is possible to work at rates greater than $VO_{2.MAX}$ for short periods of time, but this can only be accomplished via energy transfer due to glycolysis, resulting in lactate accumulation after about 4-5 minutes and an inability to continue further. The important point to note is that it is possible to record >100% $VO_{2.MAX}$ values for short periods of time (Ogita et al., 1999; Rowland, 1993).

A good, succinct review of the limiting factors associated with the $VO_{2.MAX}$ metric appears in Bassett Jr. and Howley (1997 & 2000) and Rowell (1974). Good reviews of $VO_{2.MAX}$ studies from all over the world are available that contain "secondary data" from previously published studies, although none of their information appears in Tables 1 & 2. Older reviews of female maximum oxygen consumption are contained in three Drinkwater papers (1973, 1984, & 1989). More recent reviews of female data appear in Arena et al. (2007), Eisenmann & Malina (2002), Kelley & Kelley (2006), and Wolfe & Weissgerber (2003). Reviews of male $VO_{2.MAX}$ data are contained in Eisenmann & Malina (2002), FitzGerald et al. (1997), and Hartung et al. (1992). Eisenmann et al. (2011) provide percentile distributions—difficult to come by--for both female and male adolescent $VO_{2.MAX}$ values (aged 12-18 y). A good review of $VO_{2.PEAK}$ measurements in people who experienced one or more strokes in the past (hemiparetic stroke patients) is found in Ivey et al. (2006). Their review of seven studies found that $VO_{2.PEAK}$ in stroke patients was approximately one-half that of age-matched non-stroke "controls."

Table 3. Percentile distribution of $VO_{2.MAX}$ by age grouping

	VO2.MAX Estimate (mL/kg-min)									
	College Age		12-13 y		14-15 y		16-17 y		18-19 y	
Percentile Ranking	Fem.	Male	Fem.	Male	Fem.	Male	Fem.	Male	Fem.	Male
5	29.6	34.1								
10	31.8	36.6	31.0	34.7	30.6	38.1	30.5	36.4	28.9	37.6
20	32.6	39.1	33.2	37.3	32.1	40.0	32.8	38.9	31.0	40.3
30	34.0	41.6	35.8	39.0	34.5	41.9	34.5	42.0	33.5	43.0
40	34.4	43.3	37.2	41.0	36.2	43.8	36.1	44.4	35.4	44.4
50	35.1	45.8	39.3	43.0	38.0	45.8	37.6	46.2	36.7	46.3
60	35.7	47.5	40.4	45.0	38.9	48.2	39.4	47.9	38.3	48.7
70	36.3	49.2	43.2	47.3	40.5	50.2	41.4	50.2	39.6	50.8
80	37.0	52.5	45.1	51.5	43.2	52.5	44.2	53.8	41.9	53.7
90	38.5	57.6	48.4	56.2	48.8	58.8	49.8	58.3	47.2	58.4
100	42.2	60.9								

Sources: Safrit & Wood (1995). Introduction to Measurement in Physical Education and Exercise Science (3rd ed).

Pate et al. (2006). "Cardiorespiratory fitness levels among U.S. youth 12 to 19 years of age." Arch. Pediatr. Adoles. Med. 160: 1005-1012.

Note: Fem. = Female

$VO_{2.MAX}$ and Age

Many articles state that $VO_{2.MAX}$ on a body-mass basis declines with age for adults (Bruce, 1984; Minson & Denney, 1997; White et al. 1998), but exactly what the rate of decline is with age is difficult to quantify. Some estimates state that the rate of decline is 3-10% per decade for non-athletes before the age of 70, and accelerates to >20% per decade afterward (Fiser et al., 2010; Renlund & Gerstenblith, 1987). Other papers find that the decline of $VO_{2.MAX}$ with age is modest between 20 and 50 yrs (Ceaser et al., 2013; Wang et al., 2010). A summary of the Wang et al. (2010) findings appears here as Table 4. Note the quite high COV for all of the decadal groupings for both genders.

A large cross-sectional study of Canadian residents aged 20-65 indicates that VO_{2MAX} declined about 55.5% (14.5% per decade) in females and about 39.0% (8.6% per decade) in males (Bailey, et al., 1974). One interesting finding in their study is that the decline in females was concave for the 45-y period, while it was convex for males; in other words, the rate of change is different for the genders. VO_{2MAX} measured cross-sectionally drops faster during the 20-49 age range for males and then decreases more slowly after that; for females, the opposite is true (Bailey et al., 1974).

Another estimate of the decline in $VO_{2.MAX}$ is on the order of 8-34% over 20 years in middle-aged subjects, with a larger decrease in less-active people (Pollock et al., 1997; Smith & Gilligan, 1989; Tanaka et al., 1997). At age 75 y, $VO_{2.MAX}$ has been found to be 50% of individual peak values (Barnard et al., 1979). A reduction rate for $VO_{2.MAX}$ of 10% per decade is often seen in the literature (Schiller et al., 2001). A $VO_{2.MAX}$ <18 mL kg^{-1} min^{-1} in the elderly is used by the Social Security Administration as an indicator of severe disability and the need for non-independent living arrangements (AS Jackson et al., 2009).

Middle-aged and older people who participate in aerobic fitness programs have $VO_{2.MAX}$ values 14-43% higher than inactive people at the same age (Barnard et al., 1979). Athletes who maintain their physical activity levels over time as they age have significantly higher $VO_{2.MAX}$ than less active contemporaries (Hagberg, 1987; Hawkins et al., 2001, 2003; MA Rogers et al. 1990). Active individuals who maintain their physical fitness have a slower rate of decline in $VO_{2.MAX}$ (Barnard et al., 1979), although other authors disagree: "the present cross-sectional meta-analytic findings do not support the hypothesis that the rate of decline in $VO_{2.MAX}$ with age is related to habitual aerobic exercise status in men" (Wilson & Tanaka, 2000).

In general, $VO_{2.MAX}$ on a body mass basis is stable during childhood (Rowland, 1989) and peaks around puberty, and like HR_{MAX} and muscle mass, declines after that (Åstrand, 1992; Barnard et al., 1979; Buskirk & Hodgson, 1987; Freedson et al., 2000; Goodman & Thomas, 2002; Pollock et al., 1997; Posner et al., 1987; Schiller et al., 2001). In females, peak $O_{2.MAX}$ per body mass occurs around 10-12 y, while in boys, the peak occurs somewhat later, around 14 y (Armstrong, 2013; Rowland, 2013). These findings are different from those presented in Janz et al. (1998), a longitudinal study of adolescents in Muscatine, Iowa that found decreases in $VO_{2.PEAK}$ per body mass as early as 11 y of age in both females and males that continued over a five year period: from 47 ± 7 to 34 ± 5 mL kg^{-1} min^{-1} in females and from 50 ± 9 to 46 ± 7 (same units) in males (Janz & Mahoney, 1997; Janz et al., 1998, 2000). McMurray et al. (2002) also show relatively monotonic decreases in $VO_{2.MAX/BM}$ in children and adolescents beginning at age 7-9, in both African-American and Caucasian youth.

Table 4. Means and selectied percentiles of VO_2 values from the 1990-2004 NHANES surveys (All Ethnic Groups)

				20th		80th		
Age Range	**Mean**	**SD**	**COV**	**Value**	**95th CI**	**Value**	**95th CI**	**n**
				Females				
20 - 29	36.5	9.6	26.3	30.6	30.0-31.5	41.7	40.6-42.5	576
30 - 39	35.4	9.3	26.3	29.0	28.2-30.3	41.1	40.1-42.6	542
40 - 49	34.4	10.3	29.9	28.1	27.1-28.9	40.0	38.7-40.9	425
				Males				
20 - 29	44.5	10.4	23.4	37.9	37.3-38.6	50.2	48.9-51.4	675
30 - 39	42.8	12.0	28.0	36.4	35.5-37.3	48.0	47.1-49.3	574
40 - 49	42.2	12.8	30.3	35.5	34.7-37.0	47.2	46.0-49.1	458

Abbreviations:

CI:	Confidence Interval
COV:	Coefficient of variation
NHANES:	National Health and Nutrition Examination Survey

Abbreviations:

SD:	Standard deviation

Source: C.-Y. Wang, et al. (2010). Amer. J. Epidem. 171: 426-435.

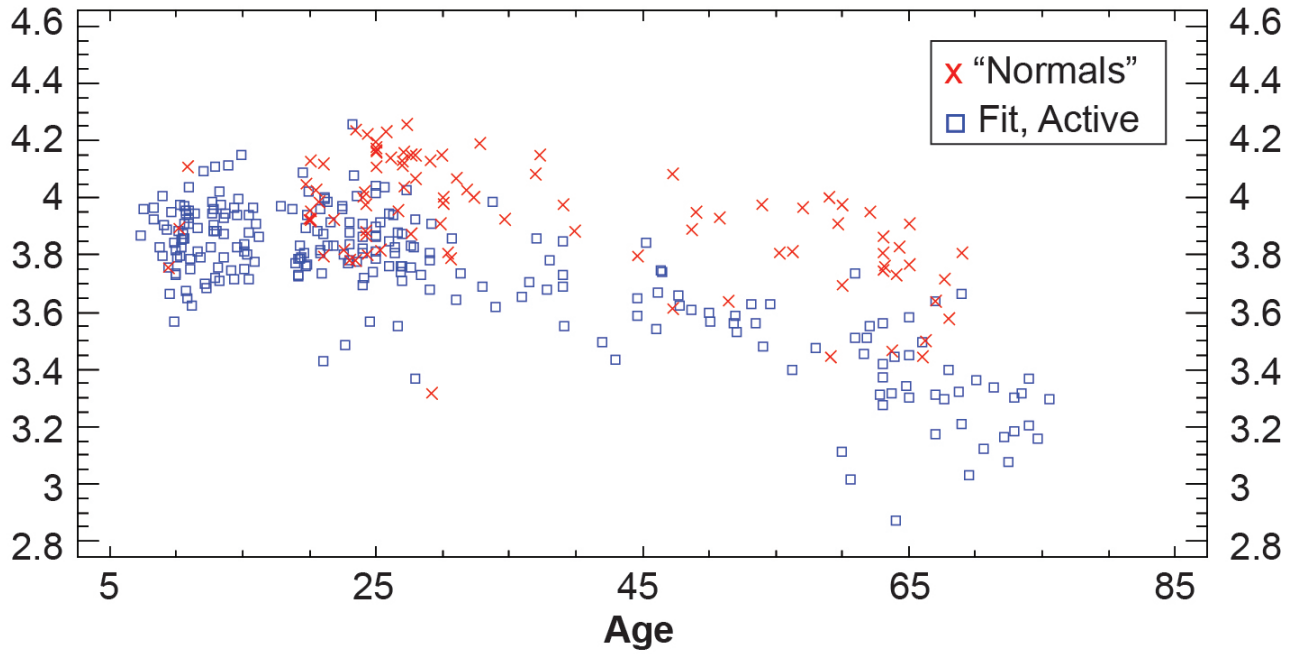

Figure 1. Plot of LN VO2 MAX versus age for two groups

In body mass-adjusted units the cross-sectional decline for normal-weight adult males is about 0.45 mL kg^{-1} min^{-1} per year regardless of baseline physical activity level, and about 0.30 mL kg^{-1} min^{-1} per year in adult females (Hodgson & Buskirk, 1977). The rate of decline is less in some longitudinal studies, with a regression-based slope of 0.40 mL kg^{-1} min^{-1} per year for males obtained by Dehn & Bruce in 1972 (reviewed in Hodgson & Buskirk, 1977). However, one study that provided both cross-sectional and longitudinal rates of decline in a sample of males states that the cross-sectional decline was 0.4 mL kg^{-1} min^{-1} per year and the average longitudinal decline in individuals was 0.9 mL kg^{-1} min^{-1} per year (Larson & Bruce, 1987). Another study showing a high longitudinal decline in $VO_{2.MAX}$ is McClaran et al. (1995), which found a decrease of 0.75 kg^{-1} min^{-1} per year for a mixed gender cohort aged 67 y at the beginning.

Not all longitudinal studies show a monotonic decline in $VO_{2.MAX}$ over the years, and one actually showed a small increase in the 18th year of the study compared to the 10th year, with very little variability over the entire 18 y period (Kasch et al., 1985). Kasch & Wallace (1976) present $VO_{2.MAX}$ data for 13 exercising males followed over 11 years, their starting ages were between 32-56 y. There was no discernible trend seen in most of the men and an Intraclass Correlation Coefficient (ICC) analysis of their data undertaken by me indicated that most of the variance seen in the data was *between* individuals and *not within* an individual; the ICC was 0.87 ($p \approx 0.011$), indicating that only ~5% of total explained variance was due to intra-individual variability. Figure 1 depicts the natural logarithm of $VO_{2.MAX}$ data shown in Table 2 for normal and fit males.

$VO_{2.MAX}$ and Fitness Level

As introduced above, besides age and gender, fitness level also affects $VO_{2.MAX}$ measures as evidenced by the data on oxygen consumption "standards" provided by the American College of Sports Medicine (Sanders & Duncan, 2006). Their standards are reproduced here as Table 5. Added to

Table 5. Age and gender specific "CUTPOINTS" of aerobic fitness levels

Age Range/ Fitness Level	VO2.MAX Cutpoints (mL/kg-min) Females ACLS	Females NHANES	Males ACLS	Males NHANES
20-29				
Low	≤ 30.63	≤ 30.63	≤ 37.13	≤ 3.94
Mod.	30.64 - 36.64	30.64 - 37.49	37.14 - 44.22	37.95 - 45.71
High	> 36.64	> 37.49	> 44.22	> 45.71
30-39				
Low	≤ 28.70	≤ 29.08	≤ 35.35	≤ 36.88
Mod.	28.71 - 34.59	29.09 - 36.45	35.36 - 42.41	36.89 - 44.90
High	> 34.59	> 36.45	> 42.41	> 44.90
40-49				

Table 5. Age and gender specific "CUTPOINTS" of aerobic fitness levels (continued)

Age Range/ Fitness Level	$VO_{2.MAX}$ Cutpoints (mL/kg-min) Females ACLS	Females NHANES	Males ACLS	Males NHANES
Low	≤ 26.54	≤ 28.95	≤ 33.04	≤ 37.00
Mod.	26.55 - 32.30	28.96 - 35.40	33.05 - 39.88	37.01 - 44.36
High	> 32.30	> 35.40	> 39.88	> 44.36

Source: Sanders & Duncan (2006). *Med. Sci. Sports Exer.* 38: 701-707.

	Females	Males
60 - 69		
Poor	≤ 12.9	≤ 15.9
Fair	13.0 - 20.9	16.0 - 22.9
Average	21.0 - 32.9	23.0 - 35.9
Good	33.0 - 36.9	36.0 - 40.9
Excellent	≥ 37.0	≥ 37.0

Source: McArdle et al. (2001). Exercise Physiology (5th ed.). Philadelphia: Lippincott .

Notes:

ACLS	Aerobics Center Longitudinal Study (Blair et al. 1989).
MOD.	Moderate fitness level
NHANES	National Health and Nutrition Examination Survey (Duncan et al. 2005)

it is information on similar age/gender groups from the Aerobics Center Longitudinal Study (Blair et al., 1989) and data for older adults obtained from McArdle et al. (2001). The Table clearly shows the effect that different fitness levels have on $VO_{2.MAX}$ by age and gender; the difference in $VO_{2.MAX}$ between low- and high-fit categories are in the range of 20-30% for the various age categories, and even more for the elderly.

Predicting $VO_{2.MAX}$ Using Anthropomorphic Inputs

There are a number of $VO_{2.MAX}$ prediction equations in the literature using only age, gender, and/or body mass as independent variables. They will not be reviewed in this report. The reader is directed to Armstrong & Welsman (1994, 1997), Armstrong et al. (1999), and Bonen et al. (1979) for a discussion of $VO_{2.MAX}$ prediction equations in children and adolescents. $VO_{2.MAX}$ prediction equations for older groups appears in Bradsford and Howley (1977), Darby & Pohlman (1999), Dolenger et al. (1994); Fleg (1994), Fleg et al. (2005), Latin & Elias (1993), and Rosen et al. (1998). In addition, $VO_{2.MAX}$ often is predicted using non-maximal testing, such as measuring HR at sub-maximal rates (George et al., 1993, 1997; Kline et al., 1987). See these citations—really only a small sample of the information available—for additional information regarding $VO_{2.MAX}$ prediction equations seen in the literature. There also are scores of activity-specific $VO_{2.ACT}$ prediction equations. Numerous independent variables are used in these equations, such as age, gender, fitness level, health status, body composition (lean body mass, total body fat, fat distribution, etc.) and body mass index. Many of these variables are not available to EPA's exposure modelers in the data bases available to the Agency. None of the prediction equations will be reviewed here.

Alternative (Allometric) Scaling Approaches

$VO_{2.MAX}$ scales most accurately with body mass to the 0.872 power ($BM^{0.872}$); see Weibel et al. (2004). Alternative exponents have frequently been presented in the comparative physiology literature; the most prevalent values that are seen are $BM^{0.67}$ (Kleiber, 1947) and $BM^{0.75}$ (Kleiber, 1950). Additional power values could be cited, but suffice to say that scaling to body mass provides lower standard errors when $VO_{2.MAX}$ is regressed against other feasible anthropogenic or physiological metrics, such as body surface area or basal metabolic rate (Weibel et al., 2004). Variation of $VO_{2.MAX}$ in the majority of mammals is tightly associated with aerobic capacity, the volume of capillaries and the total volume of mitochondria. Athletes and other highly fit individuals are more proficient than "ordinary" individuals in all three of these attributes, which is why they have a higher $VO_{2.MAX}$ for identical body weights (Weibul et al., 2004; Weibul & Hoppeler, 2005).

The work by McCann and colleagues on scaling of VO_2 metrics to account for body size differences when comparing children and adults and females and males also is informative (McCann, 2004; McCann & Adams, 2002a & b, 2003). Markovic et al. (2007) compared empirically-derived exponents for BM with respect to $VO_{2/BM}$ at different work rates from resting to maximal oxygen consumption; they found that the exponents behaved differently in athletes and normal (control) males. They also found that the best-fit exponents varied between 0.67-0.98, so there was no single value that performed best in either group or at varying work rates (Markovic et al., 2007).

It should be noted that allometric scaling is theoretically correct only if there is **no** correlation between the ratio metric (say, $VO_{2/BM}$) and the anthropometric variable(s) of interest (BM, HR, BSA, etc.). For a discussion of this issue, see Davies et al. (1995) and Katch & Katch (1974). Kolokotrones et al. (2010) state that the relationship between mass and metabolic rate has a convex curvature on a logarithmic scale, and therefore does not follow a pure power law. Thus, the theory behind allometric scaling does not fit with $VO_{2/BM}$ or $VO_{2MAX/BM}$ data (Kolotrones et al., 2010: alternative hypotheses have to be evaluated. One alternative hypothesis that has been evaluated is heat flow rather than a purely allometric approach (Roberts et al., 2010). An even harsher critic of allometric scaling is Dr. I. Mahood of the Food and Drug Administration. He states that: "the notion of a fixed exponent is theoretical and there is no evidence that the exponent of a physiological or pharmacokinetic parameter resolves around a fixed number" (Mahood, 2010: p.2927).

Allometric scaling is also known as "fractals" when applied to resting metabolic rate that scales to the quarter-power of body mass (Rowland, 2007). The interested reader is referred to Rowland (2007), Suarez & Darveau (2005), or Weibel et al. (2004) and the references cited in them for additional information on the topic.

Relative $VO_{2.MAX}$ Metrics: One and Two-Sided

$VO_{2.MAX}$ is an absolute oxygen consumption metric, as we have defined it in Section 2. Discussion of it as a one-sided VO_2 metric is contained in Appendix B, where the focus is on its relationship with various heart rate metrics. In general, the most common form of a one-sided VO_2 metric seen in the literature is $\%VO_{2.MAX}$. Basically, $\%VO_{2.MAX}$ is not linear with either %HRR or $\%HR_{MAX}$ metrics (Brawner et al., 2002; Bruce, 1984b; Davis & Covertino, 1975; Swain 2000; Swain & Franklin, 2002a).

As mentioned above, the most common form of a two-sided VO_2 metric seen in the literature is $VO_{2.RES}$. It more closely follows a linear relationship with various other reserve metrics, especially HRR (Brawner et al., 2002; Carvalho et al., 2008, 2009; Swain & Franklin, 2002b). Even so, the linear relationship is not tight, having mean differences between %HRR and $\%VO_{2.MAX}$ of 6-8% in a set of exercising individuals (Cunha et al., 2011a, b). Additional information on the $VO_{2.RES}$ metric appears below in the discussion of the metabolic chronotropic relationship (Section 7). Data on $VO_{2.RESERVE}$, or for both $VO_{2.MAX}$ *and* $VO_{2.REST}$ (so that $VO_{2.RESERVE}$ can be calculated), are depicted in Table 6. Units of both mL/kg-min and mL/min are provided. As seen, there is not much information in the literature on the $VO_{2.RESERVE}$ metric.

Table 6. Estimates of $VO_{2.Reserve}$ or both $VO_{2.Rest}$ and $VO_{2.Max}$ seen in the same article

		Oxygen Consumption (in mL/kg-min)				
Age Range (Mean±SD)	Health Status	VO2.Rest (Mean±SD)	VO2.Max (Mean±SD)	Mean Diff.	Citation	Comments
Females: Normal, Healthy, or Not Specified						
6	N			28.9	DW Morgan et al. 1999	n=20; SD of VO2.Res= 2.5
13.1 ± 2.0	N	3.2 ± 0.7	41.6 ± 3.6	38.4	Hui & Chan 2006	n=21; Chinese data
14 ± NS	N	4.0 ± 1.6	28.7 ± 5.4	24.7	Wilson et al. 1985	n=34; controls
24.3 ± 4.2	NS	2.98 ± 0.40	29.0 ± 5.3	26.0	Frey et al. 1993	n=7; untrained
31.1 ± 8.8	N	3.6 ± 0.4	39.7 ± 5.5	36.1	Dalleck & Kravitz, 2006	n=24; cross trainer
55.7 ± 7.8	N	3.4 ± 0.3	30.6 ± 6.7	27.2	Nikolai et al. 2009	n=7
Females: Active, Fit, or Athlete						
13 - 19	Ath	3.5 ± 0.1	52.7 ± 4.7	49.2	Guidetti et al. 1999	n=9; competitive gymnast
27.8 ± 2.6	Fit	3.45 ± 0.64	45.1 ± 5.4	41.6	Frey et al. 1993	n=6; trained exercisers
Females: Sedentary, Overweight, Obese, or Health Issues						
14 ± NS	EBP	3.2 ± 1.4	22.7 ± 5.9	19.5	Wilson et al. 1985	n=34; subjects
26.8 ± 7.9	Para.	3.02 ± 0.64	28.0 ± 6.	24.8	M Lee et al. 2010a	n=19
Males: Normal, Healthy, or Not Specified						
6	N			31.1	DW Morgan et al. 1999	n=15; SD of VO2.Res=2.8
13.9 ± 1.9	N	3.9 ± 0.7	48.4 ± 6.1	44.5	Hui & Chan 2006	n=28; Chinese data
14 ± NS	N	3.2 ± 1.4	22.7 ± 5.9	33.8	Wilson et al. 1985	n=56; controls
19.7 ± 2.1	N			45.9	Sell et al. 2008	n=12; Calculated
25.6 ± 1.6	H			53.3	Sell et al. 2008	n=7; Calculated
29.2 ± 6.8	N	4.4 ± 1.8	38.2 ± 9.2	43.0	Dalleck & Kravitz, 2006	n=24; cross trainer
59.1 ± 7.6	N	3.5 ± 0.2	31.4 ± 10.0	27.9	Nickolai et al. 2009	n=7

Table 6. Estimates of $VO_{2.Reserve}$ or both $VO_{2.Rest}$ and $VO_{2.Max}$ seen in the same article (continued)

Oxygen Consumption (in mL/kg-min)						
Age Range (Mean±SD)	**Health Status**	**VO2.Rest (Mean±SD)**	**VO2.Max (Mean±SD)**	**Mean Diff.**	**Citation**	**Comments**
Males: Sedentary, Overweight, Obese, or Health Issue						
14 ± NS	EBP	4.7 ± 1.8	36.2 ± 9.1	31.5	Wilson et al. 1985	n=68; subjects
22.5 ± 4.4	Sed	3.02 ± 0.64	27.9 ± 6.29	24.8	M Lee et al. 2010a	n=19
Both Genders: Sedentary, Overweight, Obese, or Not Healthy						
24.07 ± 6.28	Para	3.12 ± 0.50	26.3 ± 6.2	23.2	M Lee et al. 2010b	n=19
27.8 ± 5.6	SCI	3.8 ± 22.9	22.9 ± NS	19.1	PL Jacobs et al. 1997	n=11; assisted-walk test
58.0 ± 7.0	Diet	2.9 ± 1.3	19.0 ± 6.6	16.1	Colberg et al. 2003	n=10; no DAN
62.9 ± 10.1	Diet	2.9 ± 1.4	15.1 ± 4.7	12.2	Colberg et al. 2003	n=13; DAN
Females: Normal, Healthy, or Not Specified						
21.8 ± 6.0	Active	0.2 ± 0.02	2.4 ± 0.2	2.2	Blanksby & Reidy 1988	n=10; competitive dancers
Males: Active, Fit, or Athlete						
23.2 ± 6.3	Active	0.30 ± 0.05	3.2 ± 0.4	2.9	Blanksby & Reidy, 1988	n=10; competitive dancers
Males: Sedentary, Overweight, Obese, or Health Issue						
28.1 ± 5.8	Para.	0.24 ± 0.05	2.24 ± 0.54	2.0	Davis & Shephard, 1988	n=15; active
27.4 ± 8.1	Para.	0.21 ± 0.08	1.56 ± 0.35	1.4	Davis & Shephard, 1988	n=15; inactive
Both Genders: Normal, Healthy, or Not Specified						
6 - 14	N	0.15 ± 0.08	1.23 ± 0.17	1.1	Cabrera et al., 2002	n=14; BSA<1.1
6 - 14	N	0.25 ± 0.09	1.64 ± 0.54	1.4	Cabrera et al., 2002	n=12; BSA 1.1-1.4
6 - 14	N	0.36 ± 0.01	2.90 ± 0.29	2.5	Cabrera et al., 2002	n=12; BSA>1.4

Abbreviations	
Ath	Athlete
BSA	Body Surface Area (meters**2)
DAN	Diabetic Autonomic Neuropathy
DCI	Spinal Cord Injury
Diet	Diabetic
EBP	Elevated Blood Pressure
Fit	An individual who is “fit” (active and has good VO2)

Abbreviations	
n	Sample size
N	Normal
NS	Not Specified
Para.	Paraplegic
SCI	Spinal Cord Injury
SD	Standard Deviation
VO2.Res	Oxygen consumption reserve

4.0
Ventilation Rate (V_E) Considerations

V_E is the ventilation rate associated with a specific oxygen consumption. It also is known as the minute ventilation rate, which is equal to breathing rate (f_R, in breaths per minute: bpm) * Tidal Volume (V_T, in liters). Note that V_T is one of the few times in this report that the "V" symbol is for a volume, not a rate. (See the glossary.) V_E also = alveolar ventilation rate (V_A) + dead space ventilation rate (V_D). See McArdle et al. (2001) for more information on these relationships. V_E is an indicator of the body's ability to provide oxygen to exercising muscles, but is not a limiting factor in exercising at sea level. That is due to the fact that normal healthy people do not approach diffusion limitations even at maximal work rates (Beidleman et al., 1999). The indicator role that V_E plays is not as clear-cut for people exercising at altitude.

The units of V_E are usually in L/min; only infrequently are they presented as L/kg-min (Wilmore & Sigerseth, 1967). Since there are so few articles using body mass-normalized V_E, we do not present any data using that metric. This is not to say that body mass is not important in characterizing V_E--it is, but >95% of the articles available to us report data V_E in L/min units only.

V_E increases linearly with increasing VO_2 up to about 60-70% of $VO_{2.MAX}$ in adults, approximately equal to a person's ventilatory anaerobic threshold (VAT) (Burnley et al., 2011; Hansen et al., 1984; Hebestreit et al., 2000; Washington, 1989, 1993). That threshold is variously called the aerobic or the gas exchange threshold by different authors (Wasserman, 1984). Above that inflection point, V_E increases faster than VO_2 resulting in an upward-increasing slope for the relationship (Bernard & Franklin, 1979). In adults, VAT occurs around 58-65% of $VO_{2.MAX,}$ and this inflection point can be increased due to exercise and increasing fitness (Haffor et al., 1990). Part of the non-linear increase in V_E with workload is due to the "cost of breathing," where the energy needed to meet additional VO_2 demands increases non-linearly with workload (Lorenzo & Babb, 2012; McArdle et al., 2001). The percentage of total oxygen consumption needed for the cardiovascular, peripheral circulation, and respiratory systems is around 3-5% at low workloads, but can be 10-15% at high workloads. There does not seem to be a gender difference in the changes in relative workload regardless of the sex-related differences in ventilatory capacity (Lorenzo & Babb, 2012). Vella et al. (2006) present data that indicates that the average oxygen cost of breathing is 8.8 ± 3.3% at $VO_{2.MAX}$, and ranged from 5.0–17.6% in different individuals. The metric of oxygen cost is $\Delta VO_2 / \Delta V_E$ with units of mL/L. Increased oxygen cost is met by increasing breathing and ventilation rates, which in turn increases VQ. See Section 5. The increased cost of breathing is not primarily responsible for pulmonary function decrements often seen in exercise studies, which are different for females than males (Coast et al., 1999; Sheel & Guenette, 2008).

VAT for children and adolescents occurs at a higher percentage of $VO_{2.MAX}$. In children 6-15, VAT appears between 71 ± 10% to 75 ± 13% in males and between 68 ± 10% to 72 ± 13% in females (Washington et al., 1988). The overall range of these percentages were 37-97% for males and 42-95% for females, so there is a wide range within youth where relative VAT occurs (Washington et al., 1988). The highest percentage values are for fit individuals.

V_E and $V_{E.MAX}$ measurements, like oxygen consumption data, are protocol-dependent, varying considerably depending upon the method used to estimate them (Garner et al., 2011; Katch et al., 1974; Magel & Faulkner, 1967; Mahon et al., 1998; Phillips et al., 2008; Price & Campbell, 1997; Toner et al., 1990). $V_{E.MAX}$ obtained using a treadmill is higher than that estimated from a cycle ergometer, and the difference usually is statistically significant (Katch et al., 1974; Lukasi et al., 1989; McArdle & Magel, 1976; Rivera-Brown & Frontera., 1998). Some studies, on the other hand, show small differences in estimated $V_{E.MAX}$ between the two exercise modes (McArdle et al., 1973). Where possible, $V_{E.MAX}$ values shown in this report are from treadmills using a continuous protocol without a mouthpiece. If a "sports mouthpiece" is used for protecting an athlete's teeth, subsequent V_E estimates using it will be slightly lower than estimates obtained without a mouthpiece. When subjects can breathe through their nose only, V_E estimates are significantly higher (Garner et al., 2011). Continuous protocols generally produce higher V_E estimates than discontinuous exercise protocols using the same piece of equipment and nose/mouth breathing method (McArdle et al., 1973).

$V_{E.MAX}$ estimates obtained from the same subjects at different times using the same measuring method and protocol are reasonably reproducible on a group-mean basis (Rivera-Brown & Frontera, 1998). The within-subject COV for both treadmill and ergometer testing procedures (COV's were not presented separately for each method) was estimated to be 9% on average for patients with pre-existing heart failure problems. Their $V_{E.MAX}$'s were estimated for a symptom-limited maximum workload, considered also to be the point where $VO_{2.MAX}$ occurred. Individual COV's for a test on the same piece of equipment ranged from 1.3-16.5%, quite a wide range (Keteyian et al., 2010). I could not uncover

additional papers providing the same type of data for "normals," even when doing a literature search specifically on the topic. Quite surprising, actually.

In general, $V_{E.MAX}$ is higher in males than in females of the same age and fitness level (Beals et al., 1996; Kamon & Pandolf, 1972; Krahenbuhl et al., 1977, 1978, 1979). $V_{E.MAX}$ declines with age in both genders, as do most physiological metrics (Pollock, et al., 1997). V_E is more highly correlated with BM than age. Correlations of V_E with age for submaximal activities are 0.11-0.76 (median=0.40), and are 0.30-0.73 with BM (median=0.57) (Beals et al., 1996).

Overall, we reviewed 543 papers for compilation of $V_{E.MAX}$ data. Useable data—having both age- and gender-specific $V_{E.MAX}$ and/or VQ data that utilized U.S. citizens--were obtained and entered into Table 7 from 135 of the papers that we reviewed (24.9%). These papers provided estimates for 376 samples depicted on individual "lines" of $V_{E.MAX}$ data: 252 entries having sample mean and SD age information, and 124 having only sample age range data. Of the 408 papers that were not used, 165 were from non-US studies (30.4%), and 119 did not provide $V_{E.MAX}$ data for any metric (21.9%). Other data issues included presenting only mixed-gender results (42 papers: 7.7% of the total reviewed); using a protocol other than a treadmill or cycle ergometer to estimate $V_{E.MAX;}$ or presenting $V_{E.MAX}$ data only graphically (31 papers: 5.7%). Other unused papers presented only sub-maximal, activity-specific ventilation rate ($V_{E.ACT}$) information (11 papers: 2.0%). Finally, there were 29 review or conceptual papers (5.3%) that were not used for Tables 7-9, and 11 "redundant" papers (2%) that presented relevant information but were previously included in our Tables.

There is a temporal and gender pattern to the articles cited in Table 7. Articles on females are almost equally distributed among three temporal categories: before 1990, in the 1990s, and in the 2000-2010s. There are between 61-66 articles for each time span. The majority of articles on males, however, occurred before 1980 (104), with 63 published in the

Table 7. Estimates of $V_{E.Max}$ seen in the literature

Age			VEmax (L/min)					
Mean	SD	Cond.	Mean	SD	COV (%)	Citation	Sample Size (n)	Comments
Females: Normal, Healthy, or Not-specified								
a. Mean & SD statistics are provided for age								
7.6	1.0	NS	40.5	10.0	24.7	Krahenbuhl et al. 1978	49	
8.2	1.0	N	37.6	7.7	20.5	Treuth et al. 1998	12	
8.5	0.8	N	52.7	12.7	24.1	Wilmore& Sigerseth 1967	20	
8.7	1.1	H	54.6	12.6	23.1	Krahenbuhl et al. 1977	20	
9.1	1.5	NS	45.2	7.2	15.9	Gilliam et al. 1977	15	
9.8	0.7	N	53.2	8.8	16.5	Loftin et al. 1998	19	
10.4	0.5	N	59.5	15.8	26.6	Wilmore& Sigerseth 1967	20	
12.4	0.5	N	70.1	10.9	15.5	Wilmore& Sigerseth 1967	22	Pregnant
13.7	0.6	N	85.0	13.0	15.3	Grossner et al. 2005	10	
15.6	3.4	N	82.2	12.4	15.1	Moffatt et al. 1984	13	Controls
16.9	3.0	N	88.6	16.7	18.8	Loftin et al. 1998	19	
18.9	2.5	N	70.7	14.6	20.7	Burke 1977	8	Group 1
18.9	2.5	N	81.3	22.6	27.8	Burke 1977	7	Control
19.0	0.9	H	67.0	13.0	19.4	Mahler et al. 2001	14	
19.5	1.6	N	86.5	17.4	20.1	A Perry et al. 1988	24	Group 2
19.6	1.6	N	99.1	16.7	16.9	A Perry et al. 1988	21	Group 1
19.6	2.0	N	85.9	14.8	17.2	A Perry et al. 1988	24	Control
19.7	1.0	N	88.5	10.6	12.0	Lesmes et al. 1978	8	Group 1
19.7	1.6	N	95.6	18.3	19.1	Lesmes et al. 1978	8	Group 2
19.7	1.9	N	88.6	15.7	17.7	Vogel et al. 1986	212	VO2max=46-132
19.9	2.0	N	80.5	9.5	11.8	Lesmes et al. 1978	8	Group 3
20.5	1.6	N	68.3	14.0	20.5	Pintar et al. 2006	15	Normal weight
20.8	1.1	N	81.9	11.7	14.3	McArdle et al. 1972	35	
22.4	3.5	N	79.7	26.2	32.9	Lesmes et al. 1978	8	Group 4
22.8	3.2	N	82.0	20.0	24.4	Grossner et al. 2005	10	
23.8	3.7	N	95.7	13.3	13.9	Gonzales 2002	8	
24.1	3.5	N	87.8	14.9	17.0	Beidleman et al. 1995	10	Control group
26.2	10.4	N	85.1	12.9	15.2	Stephenson et al. 1982	6	Mean of all cycle days

Table 7. Estimates of $V_{E.Max}$ seen in the literature (continued)

Age			VEmax (L/min)				Sample	
Mean	SD	Cond.	Mean	SD	COV (%)	Citation	Size (n)	Comments
29.4	3.8	n	81.4	14.6	17.9	Jaque-Fort. et al. 1996	22	Pregnant
30.3	4.3	H	78.0	16.0	20.5	Treuth et al. 2005	17	Normal BMI (<19.8)
30.4	4.3	N	74.9	15.0	20.0	Khodiguian et al. 1996	13	
30.4	4.3	N	67.0	14.0	20.9	Treuth et al. 2005	34	Normal BMI; 6 wk PP
30.8	3.9	N	75.0	16.0	21.3	Treuth et al. 2005	17	Low BMI (<19.8)
30.8	4.4	H	74.0	15.0	20.3	Treuth et al. 2005	34	Normal BMI; 27 wk PP
30.9	3.9	N	58.0	12.0	20.7	Treuth et al. 2005	17	Low BMI; 6-wk PP
31.0	3.8	N	78.6	10.3	13.1	Jaque-Fort. et al. 1996	7	Postpartum
31.4	4.0	N	69.0	18.0	26.1	Treuth et al. 2005	17	Low BMI; 27 wk PP
31.8	11.1	H	72.2	12.7	17.6	Flint et al. 1974	7	VE range=56.4-89.9
33.0	3.0	H	100.0	14.0	14.0	Beidleman et al. 1999	8	
33.5	4.9	N	77.4	8.8	11.4	Scharff-Olsen et al. 1992	11	
37.5	12.0	NS	90.7	19.0	20.9	Nieman et al. 2005	15	Walkers aged 20-55
59.0	4.1	NS	46.8	9.1	19.4	Fielding et al. 1997	17	Probably sedentary
62.0	7.0	N	58.3	10.3	17.7	Sheldahl et al. 1996	11	
63.3	2.9	H	50.1	9.8	19.6	Kohrt et al. 1991	16	Experimental Group
64.0	3.1	H	48.8	9.4	19.3	Kohrt et al. 1991	57	Control group
65.5	7.8	N	42.6	16.5	38.7	Carter et al. 1994	16	
68.6	5.7	H	56.5	15.4	27.3	Panton et al. 1996	36	
b. Complete age statistics are not provided								
8-11		N	64.1	11.9	18.6	Rowland & Cunningham 1997	9	Longitudinal Study
9-11		N	50.5	8.9	17.6	Vaccaro & Clarke 1978	15	3 males
9-12		N	71.0	14.1	19.9	Rowland & Cunningham 1997	9	Longitudinal Study
11.1		N	55.9			Girandola et al. 1981	15	Pre-pubertal
10-13		N	76.3	19.8	26.0	Rowland & Cunningham 1997	9	Longitudinal Study
12.7		N	56.5	11.1	19.6	Eisenman & Golding 1975	8	Group 1
12.7		N	57.6	11.1	19.3	Eisenman & Golding 1975	8	Controls
11-14		N	83.1	18.0	21.7	Rowland & Cunningham 1997	9	Longitudinal Study
12-15		N	95.3	22.7	23.8	Rowland & Cunningham 1997	9	Longitudinal Study
15.9		N	76.9			Girandola et al. 1981	15	Pubertal
14-17		N	69.5	4.1	5.9	Drinkwater & Horvath 1972	7	
17-28		N	80.3	18.4	22.9	Fringer & Stull, 1974	44	
<19		N	70.7	18.5	26.2	Drinkwater et al. 1975	10	VQ=32.0
19.5		H	91.0	13.6	14.9	Humphrey & Falls 1975	15	VE @ HR.Max
19.6		N	64.5	11.5	17.8	Eisenman & Golding 1975	8	Group 2
19.6		N	60.2	10.4	17.3	Eisenman & Golding 1975	8	Control
19-24		NS	58.3	13.3	22.8	Rockenfeller & Burke 1979	21	
29.0		N	72.0	15.1	21.0	Drinkwater et al. 1975	10	VQ=34.6
29.0		N	96.8	23.7	24.5	Diaz et al. 1978	5	Protocol study
30-39		N	68.4	10.6	15.5	Drinkwater et al. 1975	14	VQ=34.9
40-49		N	65.2	6.3	9.7	Drinkwater et al. 1975	13	VQ=36.2
50- 59		N	56.6	10.0	17.7	Drinkwater et al. 1975	6	VQ=36.6
55-59		N	56.5	13.4	23.7	Hollenberg et al. 1998	100	
> 60		N	45.4	11.8	26.0	Drinkwater et al. 1975	6	VQ=29.1

Table 7. Estimates of $V_{E.Max}$ seen in the literature (continued)

Age			VEmax (L/min)				Sample	
Mean	SD	Cond.	Mean	SD	COV (%)	Citation	Size (n)	Comments
60-64		N	51.2	9.3	18.2	Hollenberg et al. 1998	96	
60-67		NS	56.0	14.0	25.0	Blackie et al. 1991	20	
60-69		N	49.0	12.4	25.3	Hollenberg et al. 2006	339	
65-69		N	49.3	9.6	19.5	Hollenberg et al. 1998	109	
67.0		N	47.0	12.2	26.0	Hollenberg & Tager 2000	579	
68.0		N	38.8	11.4	29.4	Hollenberg et al. 2006	293	
70-74		N	45.8	9.7	21.2	Hollenberg et al. 1998	88	
70-79		NS	48.0	12.0	25.0	Blackie et al. 1991	20	
75-79		N	43.5	10.6	24.4	Hollenberg et al. 1998	36	
80-84		N	40.6	10.6	26.1	Hollenberg et al. 1998	18	
>85		N	34.9	6.0	17.2	Hollenberg et al. 1998	7	
Females: Active, Fit, Athlete								
a. Mean & SD statistics are provided for age								
12.0	0.0	Ath	77.2	12.9	16.7	GD Wells et al. 2006	7	Elite swimmers
13.0	0.0	Ath	78.7	13.3	16.9	GD Wells et al. 2006	30	Elite swimmers
14.0	0.0	Ath	83.2	13.1	15.7	GD Wells et al. 2006	18	Elite swimmers
15.0	0.0	Ath	81.2	17.8	21.9	GD Wells et al. 2006	30	Elite swimmers
15.2	4.1	Ath	92.5	14.6	15.8	Moffatt et al. 1984	13	Gymnasts
16.0	0.0	Ath	87.9	9.5	10.8	GD Wells et al. 2006	10	Elite swimmers
17.0	0.0	Ath	72.5	10.5	14.5	GD Wells et al. 2006	6	Elite swimmers
18.0	0.0	Ath	99.2	4.1	4.1	GD Wells et al. 2006	2	Elite swimmers
19.0	1.0	Ath	119.0	18.0	15.1	DW Hill & Rowell 1997	13	Track team members
19.7	1.4	Fit	88.9	9.4	10.6	Astorino et al. 2004	13	VQ=36.7; preseason
19.7	1.4	Ath	96.2	13.0	13.5	Astorino et al. 2004	9	VQ=36.7; post-season
20.4	1.6	Ath	109.6	12.4	11.3	Wilmore et al. 1990	8	Amenorrheic runners
20.7	3.2	Fit	84.9	15.6	18.4	MA Sharp et al. 2002	155	Army recruits
21.2	2.3	Ath	98.2	8.7	8.9	McArdle et al. 1972	6	Misc. sports events
21.4	3.4	Fit	99.6	15.0	15.1	MA Sharp et al. 2002	122	Army recruits
21.5	1.9	Act	107.5	13.9	12.9	Beidleman et al. 1995	10	Runners
23.0	3.0	Ath	128.1	16.7	13.0	Kozak-Collins et al. 1994	7	Competitive cyclists
23.3	3.7	Fit	94.0	11.3	12.0	Williford et al. 1989	10	Aerobic dancers
23.5	6.4	Act	90.3	16.0	17.7	Meyers & Sterling 2000	24	Equestrians
23.6	5.7	Ath	111.6	13.8	12.4	Wilmore et al. 1990	5	Eumenorrheic runners
25.0	4.6	Fit	93.4	5.9	6.3	Wilmore et al. 1990	8	Eumenorrheic controls
25.2	3.1	Act	97.6	12.5	12.8	Astorino et al. 2011	9	VQ=39.0
26.0	3.7	Act	118.6	16.8	14.2	Tanaka et al. 1997	14	Endurance trained
26.9	5.3	Act	111.4	17.9	16.1	SD Fox et al. 1993	9	
32.4	4.5	Ath	108.9	8.6	7.9	Wilmore et al. 1974b	11	Enduranced runners
34.0	4.6	Act	117.7	17.4	14.8	Tanaka et al. 1997	21	Enduranced trained
42.8	2.0	Ath	88.9	12.7	14.3	Hawkins et al. 2001	24	Master's athlete
45.0	3.6	Act	109.7	14.8	13.5	Tanaka et al. 1997	13	Enduranced trained
49.8	2.8	Ath	83.9	10.0	11.9	Hawkins et al. 2001	16	Master's athlete
51.2	2.4	Ath	83.8	14.2	16.9	Hawkins et al. 2001	24	Master's athlete
54.0	4.8	Act	103.3	19.2	18.6	Tanaka et al. 1997	23	Enduranced trained
58.3	3.2	Ath	76.3	13.6	17.8	Hawkins et al. 2001	16	Master's athlete
63.0	3.0	Fit	78.4	15.8	20.2	Kohrt et al. 1991	19	
64.6	3.9	Ath	80.3	9.0	11.2	Hawkins et al. 2001	9	Visit #1
66.0	3.6	Ath	86.7	20.2	23.3	Tanaka et al. 1997	13	
73.2	5.7	Ath	61.2	13.5	22.1	Hawkins et al. 2001	9	Visit #2

Table 7. Estimates of $V_{E.Max}$ seen in the literature (continued)

Age			VEmax (L/min)				Sample	
Mean	SD	Cond.	Mean	SD	COV (%)	Citation	Size (n)	Comments
b. Complete age statistics are not provided								
9-11		Fit	45.7	9.4	20.6	Vaccaro & Clarke 1978	15	3 males
12-13		Ath	75.2	8.4	11.2	Drinkwater & Horvath 1971	2	
14-15		Ath	80.2	11.4	14.2	Drinkwater & Horvath 1971	11	
14-17		Ath	77.5	10.3	13.3	Drinkwater & Horvath 1972	7	
16-18		Ath	90.9	4.4	4.8	Drinkwater & Horvath 1971	2	
< 19		Fit	71.4	14.2	19.9	Drinkwater et al. 1975	11	VQ=31.9
20.0		Act	73.9	13.2	17.9	Blesssing et al. 1987	13	Group 1
20.0		Act	75.1	13.2	17.6	Blesssing et al. 1987	13	Group 2
18-21		Fit	90.8	17.4	19.2	Daniels et al. 1982	7	Army cadets
18-23		Act	76.9	8.5	11.1	Kamon & Pandolf 1972	4	
19-21		Fit	85.6	8.1	9.5	Kamon & Pandolf 1972	4	
19-29		Fit	77.0	9.6	12.5	Drinkwater et al. 1975	16	VQ=34.7
30-39		Fit	83.1	16.8	20.2	Drinkwater et al. 1975	10	VQ=36.4
40-49		Fit	82.6	11.2	13.6	Drinkwater et al. 1975	7	VQ=35.0
50-59		Fit	60.7	6.4	10.5	Drinkwater et al. 1975	6	VQ=30.4
Females: Sedentary, Overweight, Obese, or Health Issues								
a. Mean & SD statistics are provided for age								
8.7	0.7	OW	50.2	8.9	17.7	Treuth et al. 1998	12	
19.4	1.5	OW	76.8	7.4	9.6	Pintar et al. 2006	15	Fit
21.1	3.0	OW	58.6	3.0	5.1	Pintar et al. 2006	15	Low fit also
21.9	2.0	Sed	53.4	14.8	27.7	Pintar et al. 2006	15	Normal weight
25.0	3.3	Sed	88.1	15.6	17.7	Tanaka et al. 1997	11	
25.0	4.0	Sed	63.5	12.0	18.9	Schiller et al. 2001	12	Caucasian
25.0	3.0	Sed	63.2	13.2	20.9	Schiller et al. 2001	12	Hispanic
31.2	4.5	OW	78.0	10.0	12.8	Treuth et al. 2005	12	High BMI (>28.6)
31.3	4.5	OW	65.0	11.0	16.9	Treuth et al. 2005	12	High BMI; 6 wk PP
31.7	4.6	OW	70.0	9.0	12.9	Treuth et al. 2005	12	High BMI; 27 wk PP
31.9	4.1	Sed	74.4	14.3	19.2	JLP Roy et al. 2006	20	AA
32.4	5.8	Sed	83.3	15.4	18.5	JLP Roy et al. 2006	30	Caucasian
32.8	5.9	OW	81.8	13.6	16.6	Nehlsen et al. 1991	18	Control group
33.0	3.3	Sed	94.9	9.9	10.4	Tanaka et al. 1997	11	
33.0	4.0	Sed	66.4	9.7	14.6	Schiller et al. 2001	14	Causcasian
34.0	4.0	Sed	63.3	10.8	17.1	Schiller et al. 2001	13	Hispanic
34.9	7.2	Sed	83.3	15.8	19.0	GR Hunter et al. 2004	39	White
35.5	7.0	Sed	74.5	13.8	18.5	GR Hunter et al. 2004	35	Black
36.0	6.8	OW	81.1	11.5	14.2	Nehlsen et al. 1991	18	Exercise group
43.7	11.3	O	76.2	17.8	23.4	Utter et al. 1998	22	Control group
44.0	3.0	Sed	56.1	15.8	28.2	Schiller et al. 2001	8	Hispanic
44.6	11.5	O	79.1	17.0	21.5	Utter et al. 1998	21	Caucasian
45.0	3.7	Sed	78.5	11.6	14.8	Tanaka et al. 1997	11	Group1
45.0	5.0	Sed	64.2	12.3	19.2	Schiller et al. 2001	21	Caucasian
45.4	9.7	O	76.5	14.3	18.7	Utter et al. 1998	26	Group 2
48.0	7.1	OW	76.0	18.0	23.7	VK Phillips et al. 2008	20	Treadmill
48.7	10.3	O	76.3	14.1	18.5	Utter et al. 1998	22	Group 3
53.0	4.0	Sed	57.0	12.4	21.8	Schiller et al. 2001	15	Hispanic
54.0	4.5	Sed	80.5	15.2	18.9	Tanaka et al. 1997	20	
54.0	5.0	Sed	66.0	18.4	27.9	Schiller et al. 2001	26	Caucasian

Table 7. Estimates of $V_{E.Max}$ seen in the literature (continued)

Age			VEmax (L/min)				Sample	
Mean	SD	Cond.	Mean	SD	COV (%)	Citation	Size (n)	Comments
64.0	4.0	Sed	52.9	22.5	42.5	Schiller et al. 2001	18	Caucasian
64.0	4.0	Sed	64.7	16.4	25.3	Tanaka et al. 1997	16	
64.0	4.0	Sed	64.7	16.4	25.3	DeVito et al. 1997	16	
65.0	4.0	Sed	54.0	15.7	29.1	Schiller et al. 2001	5	Hispanic
75.2	4.6	Sed	58.7	9.6	16.4	Kent-Braun & Ng 2000	9	
b. Complete age statistics are not provided								
17.5		Sed	75.9	0.9	1.2	Kamon & Pandolf 1972	2	
17 - 22		Sed	79.0	11.9	15.1	Kearney et al. 1976	14	Exercise group 1
17 - 22		Sed	75.5	4.4	5.8	Kearney et al. 1976	13	Exercise group 2
20 - 29		Sed	63.2	13.2	20.9	Schiller et al. 2001	12	Hispanic
20 - 29		Sed	63.5	12.0	18.9	Schiller et al. 2001	14	Caucasian
25 - 44		Sed	68.0	10.0	14.7	Dowdy et al. 1985	18	Group 1; VQ=32
25 - 44		Sed	62.6	12.4	19.8	Dowdy et al. 1985	10	Controls; VQ=32
30 - 39		Sed	67.3	10.8	16.0	Schiller et al. 2001	13	Hispanic
30 - 39		Sed	66.4	9.7	14.6	Schiller et al. 2001	14	Caucasian
35.5		Sed	69.6	9.2	13.2	Getchell 1975	11	Age range: 28-51
40 - 49		Sed	56.1	15.8	28.2	Schiller et al. 2001	8	Hispanic
40 - 49		Sed	64.2	12.8	19.9	Schiller et al. 2001	21	Caucasian
50 - 59		Sed	57.0	12.4	21.8	Schiller et al. 2001	15	Hispanic
50 - 59		Sed	66.0	18.4	27.9	Schiller et al. 2001	26	Caucasian
> 60		Sed	52.9	22.5	42.5	Schiller et al. 2001	18	Caucasian
> 60		Sed	54.0	15.7	29.1	Schiller et al. 2001	5	Hispanic
Females: Health or Other Issues								
29.4	3.8	Preg.	81.4	14.5	17.8	Khodiguian et al. 1996	22	30 weeks pregnant
30.4	6.7	MR	65.4	13.1	20.0	Fernhall et al. 1996	29	
31.7	7.2	MR-D	47.3	9.5	20.1	Fernhall et al. 1996	16	
62.0	6.6	CAD	59.2	9.9	16.7	Sheldahl et al. 1996	9	
63.7	5.8	COPD	26.1	7.2	27.6	Carter et al. 1994	58	Severe
64.8	6.4	COPD	39.9	8.2	20.6	Carter et al. 1994	23	Mild
65.0	5.2	COPD	33.9	8.2	24.2	Carter et al. 1994	42	Moderate
Males: Normal, Healthy, or Non-specified								
a. Mean & SD statistics are provided for age								
7.9	0.9	NS	44.5	11.6	26.1	Krahenbuhl et al. 1978	49	
9.4	1.7	NS	50.3	8.0	15.9	Gilliam et al. 1977	35	
9.5	0.7	H	59.3	11.3	19.1	Becker & Vaccaro 1983	13	Experimental Group
9.6	2.6	N	51.7	9.2	17.8	Fahey et al. 1979	7	Pubertal stage 1
10.0	0.6	H	60.7	12.0	19.8	Becker & Vaccaro 1983	13	Control Group
10.2	1.2	NS	57.8	11.4	19.7	Kanaley & Boileau 1988	10	Prepubescent
10.7	0.7	N	57.1	14.9	26.1	Fahey et al. 1979	6	Pubertal stage 2
10.8	0.4	H	60.3	10.1	16.7	Haffor et al. 1990	5	
12.5	0.9	NS	85.7	14.1	16.5	Maksud & Coutts 1971	17	
12.8	0.9	H	70.6	16.8	23.8	Williford et al. 1996	5	A-A
12.8	1.1	N	67.4	16.1	23.9	Boileau et al. 1977	21	Part of a test/retest
12.8	1.5	H	80.3	35.2	43.8	Williford et al. 1996	12	A-A
12.9	1.2	N	67.9	14.9	21.9	Fahey et al. 1979	6	Pubertal stage 3
13.7	0.5	NS	87.2	14.1	16.2	Kanaley & Boileau 1988	10	Pubescent
15.1	2.6	N	90.9	15.8	17.4	Fahey et al. 1979	5	Pubertal stage 4
15.4	1.8	N	80.2	12.7	15.8	Fahey et al. 1979	3	Pubertal stage 5
18.7	0.6	N	101.3	7.7	7.6	Wolfe et al. 1976	9	

Table 7. Estimates of $V_{E.Max}$ seen in the literature (continued)

Age			VEmax (L/min)				Sample	
Mean	SD	Cond.	Mean	SD	COV (%)	Citation	Size (n)	Comments
19.2	4.9	N	97.7	24.7	25.3	Burke 1977	9	Group 1
19.2	4.9	N	116.0	19.9	17.2	Burke 1977	7	Control
19.7	2.2	N	139.1	21.3	15.3	Fogel et al. 1986	210	VO2.Max range-84-194
19.9	0.9	N	148.7	7.9	5.3	Harms et al. 1995	8	FM<7 kg
20.0	1.5	N	147.0	30.0	20.4	Misplaced citation	9	
20.7	3.1	H	132.0	14.8	11.2	Pollock 1977	10	Lean
20.8	6.5	H	107.0	23.0	21.5	Mahler et al. 2001	14	
21.0	8.5	H	92.4	23.6	25.5	Kang et al. 1997	8	
21.1	1.5	NS	135.4	15.5	11.4	Kanaley & Boileau 1988	10	Adult
21.1	1.6	N	153.9	19.1	12.4	FI Katch et al. 1974	50	Treadmill test
21.4	2.4	H	151.7	17.4	11.5	V Katch & Henry 1972	35	
21.9	4.0	N	168.0	10.4	6.2	McArdle et al. 1973	15	Has 3 athletes
22.3	2.3	N	141.1	13.9	9.9	JA Davis et al. 1976	39	
23.2	7.4	N	136.2	6.2	4.6	Trappe et al. 1996	15	Longitudinal study (T1)
23.8	3.4	H	123.2	26.0	21.1	Schelegle et al. 1989	20	O3 sensitive
25.2	5.1	H	115.3	12.7	11.0	Schelegle et al. 1989	20	Not O3 sensitive
26.1	5.1	N	134.8	24.6	18.2	Gonzales 2002	8	
27.6	5.6	N	149.0	20.8	14.0	VL Katch & FI Katch 1973	75	
30.2	9.2	N	132.0	18.8	14.2	Simon et al. 1983	5	
39.1	7.4	NS	108.1	31.5	29.1	JA Davis et al 1979	7	Control
43.0	7.2	NS	105.8	15.3	14.5	JA Davis et al. 1979	97	Experimental
45.3	8.9	H	111.2	5.4	4.9	Trappe et al. 1996	15	Longitudinal study (T2)
60.0	4.7	N	71.3	13.4	18.8	Carter et al. 1994	13	
62.0	6.0	N	102.4	15.9	15.5	Sheldahl et al. 1996	9	
63.7	3.1	H	80.2	15.5	19.3	Kohrt et al. 1991	53	Experimental group
64.8	3.6	H	75.8	14.0	18.5	Kohrt et al. 1991	19	Control group
64.2	9.4	NS	144.0	25.0	17.4	Pollock et al. 1987	13	Ex-Athlete
68.7	5.1	H	87.0	22.5	25.9	Panton et al. 1996	19	
b. Complete age statistics are not provided								
3 - 4		H	22.8	5.3	23.2	Shuleva et al. 1990	9	33% female
5 - 6		H	27.1	6.1	22.5	Shuleva et al. 1990	13	23% female
8.0		N	58.7	5.1	8.7	Krahenbuhl et al. 1979	10	Group 2
8.0		N	61.5	7.4	12.0	Krahenbuhl et al. 1979	10	Group 1
8 -11		N	65.9	8.0	12.1	Rowland & Cunningham 1997	11	Longitudinal study
9 -12		N	78.2	9.3	11.9	Rowland & Cunningham 1998	11	Longitudinal study
10 - 13		N	85.5	12.3	14.4	Rowland & Cunningham 1999	11	Longitudinal study
11 - 14		N	94.4	13.1	13.9	Rowland & Cunningham 2000	11	Longitudinal study
12 - 15		N	105.1	16.3	15.5	Rowland & Cunningham 2000	11	Longitudinal study
18 - 23		NS	145.8			Seals & Mullin 1982	12	Untrained
19 - 47		H	124.6	24.4	19.6	Lukasi et al. 1989	16	Bruce protocol
20 - 29		N	94.9	15.9	16.8	Mitchell et al. 1958	36	
20 - 35		N	123.2	16.7	13.6	Milesis et al. 1976	16	Control group
20 - 35		N	113.5	15.3	13.5	Milesis et al. 1976	14	Exercise group 1
20 - 35		N	127.5	15.5	12.2	Milesis et al. 1976	17	Exercise group 2

Table 7. Estimates of $V_{E.Max}$ seen in the literature (continued)

Age			VEmax (L/min)				Sample	
Mean	SD	Cond.	Mean	SD	COV (%)	Citation	Size (n)	Comments
20 - 35		N	117.5	17.2	14.6	Milesis et al. 1976	12	Exercise group 3
21 - 35		N	109.5	16.4	15.0	Gettman et al. 1976	11	Controls-prisoners
22 - 35		N	123.0	22.2	18.0	Gettman et al. 1976	11	Group 1-prisoners
23 - 35		N	117.6	14.2	12.1	Gettman et al. 1976	20	Group 2-prisoners
24 - 35		N	114.4	16.1	14.1	Gettman et al. 1976	13	Group 3-prisoners
28.6		N	132.1	17.7	13.4	Diaz et al. 1978	7	Protocol test
30 - 39		N	89.4	17.3	19.4	Mitchell et al. 1958	18	
40 - 49		N	88.8	21.3	24.0	Mitchell et al. 1959	8	
45 - 59		N	114.0	24.0	21.1	Meyers et al. 1991	68	
55 - 59		N	88.7	15.2	17.1	Hollenberg et al. 1998	79	
60 - 64		N	85.8	22.1	25.8	Hollenberg et al. 1998	66	
60 - 67		NS	83.2	7.3	8.8	Saltin & Grimby 1968	5	Ex-Ath., 10 y no train.
60 - 69		NS	83.0	14.0	16.9	Blackie et al. 1991	20	
60 - 79		N	91.0	21.0	23.1	Meyers et al. 1991	64	
65 - 69		N	78.8	20.3	25.8	Hollenberg et al. 1998	73	
66.0		N	80.4	21.5	26.7	Hollenberg et al. 2006	253	
68.0		N	75.8	21.6	28.5	Hollenberg & Tager 2000	419	
70.0		N	62.4	15.9	25.5	Hollenberg et al. 2006	189	
70 - 74		N	75.6	14.0	18.5	Hollenberg et al. 1998	81	
70 - 79		NS	66.0	12.0	18.2	Blackie et al. 1991	11	
75 - 79		N	63.6	15.7	24.7	Hollenberg et al. 1998	42	
80 - 84		N	56.2	8.6	15.3	Hollenberg et al. 1998	189	
>85		N	53.5	6.0	11.2	Hollenberg et al. 1998	4	
Males: Active, Fit, or Athlete								
a. Mean & SD statistics are provided for age								
12.1	1.2	Act	85.3	15.6	18.3	J Davis & Oldridge 1971	6	Longitudinal study T1
13.0	0.0	Ath	93.1	18.1	19.4	GD Wells et al. 2006	8	Elite swimmers
14.0	0.0	Ath	101.6	19.3	19.0	GD Wells et al. 2006	24	Elite swimmers
14.0	1.2	Act	103.2	26.9	26.1	J Davis & Oldridge 1971	6	Longitudinal study T2
15.0	0.0	Ath	99.7	13.3	13.3	GD Wells et al. 2006	40	Elite swimmers
16.0	0.0	Ath	99.7	19.2	19.3	GD Wells et al. 2006	10	Elite swimmers
16.0	1.3	Fit	128.5	17.0	13.2	Rivera-Brown & Frontera 1998	20	Treadmill test
17.0	0.0	Ath	113.6	17.4	15.3	GD Wells et al. 2006	9	Elite swimmers
18.0	0.0	Ath	119.8	10.5	8.8	GD Wells et al. 2006	7	Elite swimmers
18.0	2.6	Ath	133.0	19.9	15.0	Rundell 1996	7	Olympic speed skaters
19.0	0.8	Ath	142.5	17.5	12.3	McMiken & Daniels 1976	8	Distance runners
19.5	2.5	Ath	156.5	13.7	8.8	Mahood et al. 2001	13	Cross-country skiers
19.8	1.0	Ath	127.0	13.0	10.2	Magel & Faukler 1967	26	Swimmers; 49 bpm
19.9	2.7	Fit	132.0	19.7	14.9	MA Sharp et al. 2002	171	Army recruits
20.4	1.8	Ath	127.9	27.4	21.4	McArdle & Magel 1979	23	Treadmill test
21.1	3.4	Fit	161.4	13.4	8.3	McArdle et al. 1978	11	Experimental group
21.2	1.6	Act	153.9	19.1	12.4	FI Katch et al. 1974	50	Treadmill values
21.3	2.6	Act	169.0	10.7	6.3	Pollock 1977	8	Fit, good runners
21.4	2.4	Act	151.7	17.4	11.5	V Katch & Henry 1972	35	Part of the above?
21.4	2.6	Fit	167.2	8.4	5.0	McArdle et al. 1978	8	Control group
21.8	3.4	Fit	141.6	20.2	14.3	MA Sharp et al. 2002	122	Army recruits
23.0	3.0	Fit	132.9	12.4	9.3	LaFrenz et al. 2008	10	Endurance trained
24.0	2.5	Act	158.0	23.1	14.6	Glass et al. 1997	6	

Table 7. Estimates of $V_{E.Max}$ seen in the literature (continued)

Age			VEmax (L/min)				Sample	
Mean	SD	Cond.	Mean	SD	COV (%)	Citation	Size (n)	Comments
24.8	5.7	Act	144.1	27.9	19.4	Wallick et al. 1995	16	Roller skaters
25.3	5.5	Act	147.5	25.4	17.2	Astorino et al. 2011	11	
24.5	4.0	Ath	173.4	14.6	8.4	Lounana et al. 2007	11	Elite amateur cyclists
25.5	3.5	Ath	174.1	24.0	13.8	Lounana et al. 2007	15	Professional cyclists
25.7	3.5	Fit	150.5	3.8	2.5	Trappe et al. 1996	18	
26.0	3.0	Ath	170.0	12.0	7.1	Mahler et al. 1984	8	Olympic rowers
26.2	3.0	Ath	168.0	14.6	8.7	Pollock 1977	20	Elite runners
27.1	6.7	Act	130.4	5.1	3.9	Trappe et al. 1996	18	
28.6	3.3	Act	145.7	16.4	11.3	Harms et al. 1997	7	Control case
30.4	7.4	Fit	150.8	21.1	14.0	Yuen et al . 2011	14	Active cyclists
42.4	14.0	Ath	146.5	35.0	23.9	Faria et al. 1996	16	Cross-country skiers
44.5	2.8	Ath	143.7	18.4	12.8	Hawkins et al. 2001	31	Master's athlete
46.5	6.1	Ath	122.5	24.5	20.0	Bernard et al. 1979	13	Master's sprinter
46.8	9.8	Ath	124.5	7.3	5.9	Trappe et al. 1996	10	Highly fit
47.2	3.8	Fit	121.0	2.8	2.3	Trappe et al. 1996	10	
47.2	5.8	Act	123.4	21.4	17.3	Loftin et al. 1996	12	Handball players
48.7	5.9	Act	109.0	3.8	3.5	Trappe et al. 1996	18	
50.5	3.5	Fit	144.0	23.4	16.3	Pollock et al. 1997	21	
53.5	3.3	Ath	126.5	26.7	21.1	Hawkins et al. 2001	31	Master's athlete
53.9	2.9	Ath	131.3	19.2	14.6	Hawkins et al. 2001	34	Master's athlete
55.3	11.2	Ath	116.1	25.2	21.7	Bernard et al. 1979	13	Master's endurance
60.0	8.6	Ath	148.0	18.0	12.2	Pollock et al. 1987	11	
60.2	8.8	Ath	151.4	20.0	13.2	Pollock et al. 1997	21	
61.0	8.0	Ath	98.0	11.0	11.2	Proctor et al. 1998	8	
62.0	8.9	Ath	116.2	17.8	15.3	MA Rogers et al. 1990	15	
62.2	3.5	Ath	120.3	23.3	19.4	Hawkins et al. 2001	34	Master's athlete
62.3	2.9	Ath	84.0	14.0	16.7	Hawkins et al. 2001	13	Visit #1
64.0	6.0	Ath	135.0	25.0	18.5	Proctor et al. 1998	8	
65.0	3.0	Ath	106.9	27.4	25.6	Hagberg et al. 1988	10	
68.4	9.8	Fit	87.5	11.7	13.4	Trappe et al. 1996	10	Highly fit
70.4	8.8	Ath	117.4	24.7	21.0	Pollock et al. 1997	21	Followup
71.1	3.2	Ath	88.0	27.4	31.1	Hawkins et al. 2001	13	Visit #2
72.7	1.5	Ath	97.8	12.1	12.4	Wilmore et al. 1974	3	Endurance runners
76.0	4.8	Ath	93.9	27.4	29.2	Hawkins et al. 2001	8	Visit #1
82.8	4.0	Ath	73.8	23.2	31.4	Hawkins et al. 2001	8	Visit #2
b. Complete age statistics are not provided								
17 - 26	-	Fit	117.7	12.3	10.5	Kamon & Pandolf 1972	5	
18 - 23		Ath	173.7			Seals & Mullin 1982	12	Crew team
18 - 23		Ath	153.1			Seals & Mullin 1982	10	Gymnastics team
18 - 23		Ath	169.0			Seals & Mullin 1982	11	Swimming team
18 - 23		Ath	173.1			Seals & Mullin 1982	10	Wrestling team
22.0		Act	149.6	20.7	13.8	Maksud & Coutts 1971	20	
18 -21	-	Fit	140.1	19.3	13.8	WL Daniels et al. 1982	11	Army cadets
19 -34	-	Act	122.1	13.0	10.6	Kamon & Pandolf 1972	5	
40 -49	-	Ath	150.9	-	-	Pollock 1974	11	Runners; 112-162
50 -59	-	Ath.	139.9	-	-	Pollock 1974	5	Runners; 111-159
60 -69	-	Ath.	140.0	-	-	Pollock 1974	6	Runners; 113-160

Table 7. Estimates of $V_{E.Max}$ seen in the literature (continued)

Age			VEmax (L/min)				Sample	
Mean	SD	Cond.	Mean	SD	COV (%)	Citation	Size (n)	Comments
70 -75	-	Ath.	97.8	-	-	Pollock 1974	3	Runners;84-106
Males: Sedentary, Overweight, or Obese								
a. Mean & SD statistics are provided for Age								
19.1	1.4	O	102.0	18.2	17.8	Wolfe et al. 1976	12	
19.7	1.6	OW	148.0	9.3	6.3	Harms et al. 1995	8	FM>13 kg
21.5	1.9	Sed.	148.7	19.9	13.4	Wilmore et al. 1970	17	
23.5	2.9	Sed.	104.8	18.4	17.6	Poole & Gaesser 1985	6	Group 3
23.8	3.6	Sed.	103.7	11.0	10.6	Poole & Gaesser 1985	6	Group 2
24.6	6.7	Sed.	109.6	26.5	24.2	Poole & Gaesser 1985	5	Group 1
29.7	2.9	Sed.	146.1	16.0	11.0	Wilmore et al. 1970	15	
39.1	7.4	Sed.	108.1	31.5	29.1	JA Davis et al. 1979	7	Control group
40.5	3.1	Sed.	142.7	27.3	19.1	Wilmore et al. 1970	16	
43.0	7.2	Sed.	105.8	15.3	14.5	JA Davis et al. 1979	9	Group 1
52.9	4.4	Sed.	133.5	27.5	20.6	Wilmore et al. 1970	7	Control group
61.4	5.2	Sed	95.8	22.1	23.1	MA Rogers et al. 1990	14	
66.0	5.0	Sed	85.0	11.0	12.9	Hagberg et al. 1988	10	
75.7	4.7	Sed	98.3	21.9	22.3	Kent-Braun & Ng 2000	9	
b. Complete age statistics are not provided								
20 - 35		Sed	123.2	16.7	13.6	Milesis et al. 1976	16	Control
20 - 35		Sed	113.5	15.3	13.5	Milesis et al. 1976	14	Group 1
20 - 35		Sed	127.5	15.5	12.2	Milesis et al. 1976	17	Group 2
20 - 35		Sed	117.5	17.2	14.6	Milesis et al. 1976	12	Group 3
28 - 39		Sed	126.9	12.2	9.6	Pollock et al. 1969	8	Control group
28 - 39		Sed	126.9	16.6	13.1	Pollock et al. 1969	9	Group 2
28 - 39		Sed	127.2	14.1	11.1	Pollock et al. 1969	10	Group 1
30 - 45		Sed	125.6	18.8	15.0	Pollock et al. 1972	10	Group 2
30 - 45		Sed	132.3	14.5	11.0	Pollock et al. 1972	12	Group 1
41.6		Sed	101.0	14.9	14.8	Getchell 1977	12	Ages 30-57
48.9		Sed	86.9	18.6	21.4	Pollock et al. 1971	16	
49 - 65		Sed	104.9	19.1	18.2	Pollock et al. 1976	22	Group 1
49 - 65		Sed	108.1	26.2	24.2	Pollock et al. 1976	7	Control
60 - 72		Sed	75.2	17.3	23.0	Frontera et al. 1990	12	
Males: Health/Other Issues								
26.7	5.9	MR-D	70.4	16.7	23.7	Fernhall et al. 1996	35	
26.9	6.4	MR	85.4	21.6	25.3	Fernhall et al. 1996	31	
27.4	8.1	Para	69.0	16.0	23.2	Davis & Shephard 1988	15	Inactive
28.1	5.8	Para	106.0	22.0	20.8	Davis & Shephard 1988	15	Active
55.0	9.0	CHF	60.1	12.8	21.3	J Myers et al. 2012	24	Exercise Group
55.2	10.1	HT	79.9	25.2	31.5	Olivari et al. 1996	11	1 female age=36
56.0	10.0	HF	55.2	-	-	Keteyian et al. 2010	160	VO2 COV=9%
57.0	7.0	CHF	54.4	12.2	22.4	J Myers et al. 2012	26	Control Group
59.0	9.0	CAD	92.7	18.0	19.4	J Milani et al. 1996	15	
63.3	6.4		48.9	14.5	29.7	Mador et al. 1995	6	COPD
64.0	3.0	CAD	68.0	9.3	13.7	Sheldahl et al. 1996	10	Exercise group 2
64.0	11.0	CHF	59.0	15.0	25.4	Bowen et al. 2012	24	Mild heart problem
65.3	6.5		51.5	18.5	35.9	Carter et al. 1994	32	Mild COPD
66.3	6.2		48.3	14.2	29.4	Carter et al. 1994	57	Moderate COPD

Table 7. Estimates of $V_{E.Max}$ seen in the literature (continued)

Age Mean	SD	Cond.	VEmax (L/min) Mean	SD	COV (%)	Citation	Sample Size (n)	Comments
66.3	6.3		37.1	11.4	30.7	Carter et al. 1994	176	Severe COPD
68.0	5.7	CAD	68.2	6.8	10.0	Sheldahl eta al. 1996	8	Exercise group 1
69.0	3.0	CAD	74.6	10.3	13.8	Sheldahl eta al. 1996	11	Exercise group 3

Abbreviation & Symbols:	
A-A	African-Americans
Act	Active
Alt	Altitude
Ath	Athlete
BMI	Body Mass Index (kg/m**2)
Bpm	Breaths per minute
BSA	Body Surface Area (m**2)
CAD	Coronary artery disease
CHF	Chronic Heart Failure
COPD	Chronic Obstructive Pulmonary Disease
COV	Coefficient of Variation (SD/mean)
D	Down Syndrome
Fit	Fit or trained individuals
FM	Fat mass
H	Healthy
Heart	Heart disease or coronary artery disease
HF	Heart Failure
HT	Heart Transplant recipient

Abbreviation & Symbols:	
♀	Females
♂	Males
MR	Mental Retardation
N	Normal health
N	Sample size
NS	Not specified (unknown)
O	Obese
OW	Overweight
Para	Paraplegic
PP	Post-partum
Preg	Pregnant
Sed	Sedentary
T	Time (followed by a label)
VE	Ventilation rate (L/min)
VO2	Oxygen consumption (L/min)
VQ	Ventilatory Equivalent (VE/VO2) (unitless)
Wk	Week(s)

1990s, and only 34 after the Millennium. About 66% of the articles published after 1990 used females for their subjects exclusively. It is unknown if this time/gender bias affects our $V_{E.MAX}$ or other ventilatory metrics. It should be noted that all of the data in Table 7 are cross-sectional in nature, even though some V_E estimates are from "longitudinal" studies—usually consisting of a single measurement for multiple time periods in the same individual, often separated by years. Basically these are treated as separate sequential cross-sectional studies.

In general, people who are sedentary, overweight, and/or have health problems have lower $V_{E.MAX}$ levels than "normal," healthy people, who have lower levels than fit, active, or athletes. Individuals with mental issues, including mental retardation—with or without Down syndrome—also have lower $V_{E.MAX}$ levels than "normals" (Baynard et al., 2004, 2008).

Pregnant females do not have significantly different $V_{E.MAX}$ levels than non-pregnant females of approximately the same age, although women who are pregnant have higher V_E recordings for rest, 25W, 50W, and 75W exercise levels (Khodiguian et al., 1996). Even given that situation, the authors state that there was no pregnant condition/workload interaction in an ANOVA of all of the data, "suggesting that pregnancy did not lead to augmentation in the ventilatory response to increasing levels of work (Khodiguian et al., 1996; p. 234). Thus, the impact of pregnancy on V_E is mixed, and no other similar study could be found to shed light on the issue. It does appear that post-partum V_E values are significantly lower than pre-pregnancy values, but the decrease becomes less over time post-delivery (Jaque-Fortunato et al., 1996; Treuth et al., 2005). I could not find any information regarding the length of time post-partum that is required before pre-pregnancy V_E values are attained.

There is no difference in $V_{E.MAX}$ over the menstrual cycle for females (Stephenson et al. 1982), nor is there any difference in activity-specific V_E in the different phases of the cycle either at sea level or at altitude (Beidleman et al., 1999; Bemben et al., 1995).

In attempting to address $V_{E.RES}$ values for various age/gender cohorts, we run into a problem. There are very little data on resting ventilation rate reported in the literature ($V_{E.REST}$) and even less on $V_{E.RESERVE}$ ($V_{E.RES}$). Unlike the $VO_{2.RES}$ metric, there is no well-accepted approach used to derive $V_{E.RES}$ from $V_{E.MAX}$ data, so there are few $V_{E.RES}$ values reported in the exercise physiology literature. What data I could find on $V_{E.REST}$ and $V_{E.RES}$ appears in Table 8. $V_{E.RES}$ at maximal oxygen consumption appears to be on the order of 60-70 L/min in females without health issues, and 70-110 L/min in males.

Also included in that Table are resting V_E data. $V_{E.REST}$ does not vary much with age or gender on an absolute basis, but does on a per-BM basis. $V_{E.REST}$ values in Table 8 appear to be higher than resting values provided in Tables 5-6 and 5-14 of EPA's *Exposure Factors Handbook* (EPA, 1997b) and a Summary Table found in Adams (1993).

There is a difference in V_E values between predominately arm-work (upper body), predominately leg-work only, or a combination of the two work modes (Adams et al., 1998). Arm-work only at low-to-moderate intensities require ~10% less V_E than leg-work at the same intensity; arms-only work at high intensities elicit greater V_E at any given HR than legs-only work at equivalent workloads. The impact that static (isometric) work alone, or in combination with various levels of dynamic work, has on the HR→ V_E relationship and V_E itself needs systematic investigation (Adams et al., 1998).

Breathing Rate

Breathing rate (f_B) is an innate function of oxygen consumption demands, including tidal volume (V_T) and ventilation rate V_E. One formula for $\mathbf{f_B}$ is that it is = V_E / V_T (McArdle et al., 2001). Breathing rate increases with workload. Tidal volume does also, in such a matter that V_E increases even faster. An insert presented in McArdle et al. (2001) provides "typical values" for pulmonary ventilation values in *fit males* from rest to vigorous exercise (p. 261). The insert is reproduced here: brpm = breaths per minute.

Condition	f_B Breathing Rate (brpm)	V_T Tidal Volume (L/breath)	V_E Pulmonary Ventilation Rate (L/min)
Rest	12	0.5	6
Moderate Exercise	30	2.5	75
Vigorous Exercise	50	3.0	150

For adult females of "normal fitness," the $\mathbf{f_B}$ values shown above are higher at low workloads but lower at high: 14 bpm at rest and 40 bpm at maximum workload (Jaque-Fortunato et al., 1996). The resting tidal volume values for females are similar to the values depicted above, but are <2.1 for maximum workload levels due to their smaller lung size relative to males. The same finding with respect to higher values for f_B at rest and lower V_E values at peak exercise is seen in Stephenson et al. (1982). Tobin et al. (1983) present resting $\mathbf{f_B}$ values that are 16.6 ± 2.8 bpm in both males and females. Treuth et al. (2004) report that $\mathbf{f_B}$ at rest and at peak exercise increases with age in girls, going from 13 to 15 bpm at rest, and from 41 to 57 bpm at peak exercise.

Adams (1993) conducted a series of tests on respiratory functions that involved children aged 3-6 y up to older adults as old as 78 y. His resting values are higher than listed above for children—between 20-26 bpm—and somewhat higher for adolescents and adults of both genders. Measured resting $\mathbf{f_B}$'s for the latter groups were 13-14 bpm (Adams, 1993). Breathing rates for walking @ 2.5 mph were somewhat higher for children and young teenagers—about 32-37 bpm, but were significantly lower for adults and the elderly for both genders: 23-25 bpm (Adams, 1993). His running @ 4 mph measured $\mathbf{f_B}$'s were about 10% lower than the "vigorous exercise" estimate provided above.

Table 8. Estimates of $V_{E.Reserve}$ or both $V_{E.Rest}$ and $V_{E.Max}$ seen in the same article

		Ventilation Rate (L/min)				VE.Reserve Calculated				
		VE.Rest		VE.Max		VE.Reserve				
Age Range (Mean±SD)	Health Status	Mean	SD	Mean	SD	Mean	SD	Citation	(n)	Comments
Females, Normal, Healthy, or Not Specified										
a. Mean & SD statistics are provided for age										
26.2 ± 10.4	N	8.9	5.3	85.1	12.9	76.2		Stephenson et al. 1982	6	Mean of all cycle days
28.0 ± 4.6	N	7.7	0.9					Schoene et al. 1981	6	Folicular phase
28.0 ± 4.6	N	10.0	0.7					Schoene et al. 1981	6	Luteal phase
29.4 ± 3.8	N	9.6	1.6	81.4	14.6	71.8		Jaque-Fortunato et al. 1996	22	Pregnant
30.4 ± 4.3	N	7.6	2.6	74.9	15.0	67.3		Jaque-Fortunato et al. 1996	16	Control group
31.0 ± 3.8	N	6.9	1.9	78.6	10.3	71.7		Jaque-Fortunato et al. 1996	7	Post-partum

Table 8. Estimates of $V_{E.Reserve}$ or both $V_{E.Rest}$ and $V_{E.Max}$ seen in the same article (continued)

		Ventilation Rate (L/min)				VE.Reserve Calculated				
		VE.Rest		VE.Max		VE.Reserve				
Age Range (Mean±SD)	Health Status	Mean	SD	Mean	SD	Mean	SD	Citation	(n)	Comments
Females: Active, Fit, or Athlete										
a. Mean & SD statistics are provided for age										
22.3 ± 4.8	Ath	10.7	1.1					Schoene et al. 1981	6	Amen. ath. (follicular)
22.3 ± 4.8	Ath	11.6	1.1					Schoene et al. 1981	6	Amen. ath. (luteal)
27.8 ± 8.4	Ath	8.8	0.6					Schoene et al. 1981	6	Mens ath. (follicular)
27.8 ± 8.4	Ath	10.7	0.7					Schoene et al. 1981	6	Mens. ath. (luteal)
Males: Normal, Healthy, or Not Specified										
a. Mean & SD statistics are provided for age										
8.8 ± 1.1	NS	5.2	1.6					X Wang & Perry 2006	21	Video game VE=7.9(2.1)
12.5 ± 0.9	NS	13.3	2.5	85.7	14.1	72.4		Maksud & Coutts 1971	17	
54.3 ± 9.2	NS	11.8	4.5	93.3	23.0	87.5		Hansen et al. 1984	77	Shipyard workers
b. Complete age statistics are not provided										
18 - 24	N	8.6		115.2		106.6		Hansen et al. 1967	18	Sea level Group 1
18 - 24	N	9.2		107.7		98.5		Hansen et al. 1967	18	Sea level Group 2
18 - 24	N	8.9		109.8		100.9		Hansen et al. 1967	18	Sea level Group 3
18 - 24	N	8.8		113.2		104.4		Hansen et al. 1967	18	Sea level Group 4
65 - 75	N	11.6	2.9					HE Wood et al. 2010	11	
Males: Active, Fit, or Athlete										
21.1 ± 1.7	Ath	11.6	2.6	39.5	15.4	27.9		Hopkins et al. 1998	7	VE @ 30% VO2Max
21.1 ± 1.7	Ath	11.6	2.6	71.9	7.9	60.3		Hopkins et al. 1998	7	VE @ 65% VO2Max
21.1 ± 1.7	Ath	11.6	2.6	146.2	26.2	135		Hopkins et al. 1998	7	VE @ 90% VO2Max
Males: Health Issues										
	COPD	15.0	15.0	28	7.0	13		Montes Oca et al. 1996	25	
Both Genders: Normal, Healthy or Not Specified										
"Young"	N	10.4	2.6	93.3	40.4	82.9		Did not record!	8	Mixed fitness
30 ± 7	NS	8.0	1.7	63.8	17.2	55.8		Keyser et al. 1999	6	NWC user
Both Genders: Sedentary, Overweight, Obese, or Health Issues										
40 ± 9	Sed.	7.0	1.8	46.8	18.7	39.8		Keyser et al. 1999	18	WC for 16+ years

Abbreviations:	
Ath	Athletes
Amen	Amenorrheic
CHF	Chronic Heart Failure
COPD	Chronic Obstructive Pulmonary Disease
Mens	Menstruating
n	Sample size
N	Normal

Abbreviations:	
NS	Not specified
NWC	Non-wheelchair (ambulatory)
SD	Standard Deviation
VE	Ventilation rate (L/min)
VE.Max	Maximum ventilation rate (L/min)
VO2.Max	Maximum oxygen consumption rate (L/min)
WC	Wheelchair user (manual)

Adams' resting V_E measures were slightly higher than the 6 bpm value shown above: the measured values were between 6.2-7.1 L/min at rest for the various age/gender cohorts evaluated (Adams, 1993). Walking and running V_E estimates in Adams (1993) are significantly lower than the moderate/vigorous estimates in the "typical values" table. Adams (1993) did not contain any information on tidal volume (V_T), so comparisons could not be made for that parameter.

In general, female V_E's will be lower than those shown above, with $V_{E.MAX}$'s in the 75-85 L/min range for "normal fitness" females. That is what is seen in Table 7 for most normal, healthy adult females, although fit, active, or athletic females between the ages of 18 and 55 y can have considerably higher ventilation rates.

The relative ratio change from rest to vigorous exercise in the above data is 4 for breathing rate, 6 for V_T, and 25 for V_E. Athletes and fit persons can have $\mathbf{f_B}$'s as high as 60-70 breaths/min, making for a ratio change of 5.0 - 5.8 for $\mathbf{f_B}$. (However, the depth of breathing at these rates is shallow, causing V_T to decrease relative to the values shown in the McArdle et al. (2001) insert. Since V_E's greater than160 L/min or so are uncommon—even in elite athletes—the maximum ratio change of V_E is on the order of 26-30 times the resting rate.)

Beals et al. (1996) took the Adams (1993) data and used cluster analysis to apportion activities into similar groupings from a V_E perspective using a "nearest neighbor" approach (and two others methods). They then classified them into low, moderate, and high groups for children, adult females, and adult males. None of these groups were explicitly defined (Beals et al., 1996). For children, moderate activities were walking at 2-3 mph and "playing"; V_E's for these activities were in the 16-18 L/min for a 5 min duration (mean/SD=16.7±2.8). Vigorous activities included walking at 3-4 mph and running; V_E's for those activities were 24-29 L/min (28.6±3.3) (Beals et al., 1996). The V_E's for typical activities for the same two groupings were higher for adults, with males having higher ventilation rates than females. V_E distributions for all activities and all age/gender categories were best fit with a log-normal or gamma distribution (Beals et al., 1996).

If breathing with increasing workload becomes labored or inadequate, the usual V_E-to-VO_2 convex relationship discussed earlier can curve downward, decreasing VQ with increasing workload among other changes (McArdle et al. 2001). That response is common in COPD patients, and indicates a failure of ventilation to keep pace with oxygen demands (McArdle et al., 2001). For more on VQ, see Section 5.

Activity-Specific Estimates of V_E ($V_{E.ACT}$)

Prior to 2000, OAQPS and NERL used estimates of activity-specific ventilation rate directly in its exposure/intake dose-rate models. Since then, we "build" activity-specific $V_{E.ACT}$ estimates using the METS→VO_2→ V_E relationships documented in Appendix D. In order to facilitate a "quick and dirty" evaluation of activity-specific V_E estimates developed in the APEX and SHEDS models, I wanted to include in Appendix E a compilation of $V_{E.ACT}$ estimates appearing in various articles, reports, and books. This was not done, however, due to author fatigue. A definitive evaluation of activity-specific V_E's should be undertaken. Subsequently, a "hard look" should be taken for each factor in the appropriate METS→VO_2→ V_E relationship used to estimate intake dose rate in our exposure models. That would assist in determining and evaluating which parameter in the above relationship affects estimated $V_{E.ACT}$ estimates the most.

Equations for Predicting $V_{E.A}$ from $VO_{2.A}$ Estimates

As just mentioned, $METS_{ACT}$ estimates are converted into $VO_{2.ACT}$ values and then into $V_{E.ACT}$ metrics. We no longer use general distributions of V_E, but have developed a new set of age- and gender-specific VO_2→V_E equations based upon a re-analysis of the Adams data (Graham & McCurdy, 2004). These equations are used for all activities to obtain an estimate of $V_{E.ACT}$ from $VO_{2.ACT}$ | $METS_{ACT}$. For example, for people <20 years old, we use the following equation to estimate V_E from VO_2 for individual i, in units of L kg^{-1} min^{-1}, and that it applies to all activities undertaken by individuals in that cohort.

$$\text{Ln}\,[_E\,BM^{-1}]_I = 4.433 + (1.086 * \text{Ln}\,[VO_2\,BM^{-1}]) + (0.283 * \text{Ln}\,[AGE_I]) + (0.051 * GENDER_I) + \{e_{\text{Within}}: 0, 0.096\} + \{e_{\text{Between}}: 0, 0.112\}$$

Where: $VO_2\,BM^{-1}$ = Body mass adjusted VO_2 in mL kg^{-1} min^{-1} (S.E. = 0.010)

AGE_I = Age of the individual in years (S.E. = 0.012)

$GENDER_I$ = Sex of the individual: -1 ♀ and +1 ♂ (S.E. = 0.005)

e_{Within} = Intra-individual variation, a normal distribution {N} with a mean of 0 and a SD = 0.096

e_{Between} = Inter-individual variation, a {N} with a mean of 0 and SD = 0.112

The R^2 of the equation is 0.925 and $p<0.001$. All independent variables are significant at $p<0.001$. The sample size of the equation is 1,085. See Graham & McCurdy (2004) for additional information and V_E prediction equations for other age groupings (20 £ 34, 34 £61, and 61+).

There are numerous alternative equations to estimate V_E for general and specific activities available in the exercise physiology literature, including standard textbooks on the subject; e.g., Anderson et al. (1978), Åstrand & Rodahl (1986), McArdle et al. (2001), Nieman (1990), and Wells (1991).

EVR: Equivalent Ventilation Rate

Prior to the development of the APEX model, and for some versions of the pNEM model, OAQPS used the EVR form of breathing rate: VE divided by body surface area (BSA). The units of this metric are liters/m2 – min. This metric was used to facilitate the linking of exposure model outputs to the ozone clinical data that were on a per-BSA basis. For a discussion of the previously used approach to estimate EVR, see Johnson (2002), Johnson & McCoy (1995), and Johnson et al. (2005).

Nasal/Oral Patterns Associated with V_E Levels

As a person undertakes more and more work, oral inhalation becomes an ever more important source of oxygen for the lung as nasal resistance increases with airflow (Chadha et al., 1987; Kleinman & Mautz, 1988). The changing patterns of nasal/oral breathing greatly affect the "scrubbing efficiency" of the nose, usually a very effective filter of "incidentally inhaled toxic particles and gases" (p. 101), resulting in more deposition of pollutants in the lung via oral breathing (Kabel et al., 1994; Kirkpatrick et al., 1982; Vass et al., 2003). While most humans are oral-nasal breathers at even relatively low V_E levels, the fraction of air entering through the mouth is relatively small in most individuals (0-10%) until 25-45 L/min V_E, where it increases to 50-60% or so (Kleinman & Mautz, 1988). This alteration of nasal/oral components of V_E affects the pattern of intake dose received and the distribution of dose in sensitive regions of the lung (and subsequently the blood stream). Factors that determine the type and amount of biological responses due to these changes in dosing pattern and amount of xenobiotic material absorbed in the body depend upon a number of chemical, physical, and physiological parameters (Kleinman & Mautz, 1988); they are:

1. The relative air-to-liquid phase partitioning of the inhaled chemical entering the mucus layer lining of the respiratory airway.
2. Residence time of the gas in each airway segment, a direct function of V_E and segment volume
3. Degree of turbulence in the airstream
4. The effective area of absorbing surface in each segment of the airway
5. Diffusivity of the gas
6. The presence of particles in the "carrier" airstream, which alters the site and type of deposition in the airway (and may enhance transport of a toxic substance deeper into the respiratory tract)
7. Modification of mucus production rate due to altered physiological states
8. Alteration of chemical and physical properties in the mucus "sheath" itself
9. Changes in airway volumetric dimensions due to airway dilution/constriction due to biological responses affected by inhalation of the irritant itself.

Data on these effects have been obtained *in vivo* through animal experiments using different gases, with and without particulate aerosols. Functional relationships between the amounts of pollutant absorbed in the lung by nasal and oral breathing routes and by different ventilation rates have been developed in humans for selected pollutants (Kleinman & Mautz, 1988). For relatively low V_E's, the ratio of dose of pollutant reaching the lung drops for oral breathing-to-nasal breathing from 1.8 at low V_E to 1.3 at higher V_E (Kleinman & Mautz, 1988).

Apparently, body size, body composition, and nose volume all affect the physiological and other parameters mentioned above for nose and mouth breathing rates, more so than gender *per se* (Hall, 2005). Obviously the effects of biological and physiological parameters on V_E are complex, but could be important in modeling intake dose rate associated with human exposures. More work should be undertaken on this subject to determine if nasal/oral changes due to increasing work rates make a significant difference in absorbed dose to target organs.

5.0
VQ: the Ratio of V_E to VO_2

The ratio of V_E-to-VO_2 is called the "ventilatory equivalent" (VQ). It is fairly linear with work rate up to the ventilatory threshold, (also known as the "anaerobic threshold") but increases non-linearly above it (Simon et al., 1983). VQ measurements, like its two constituents V_E and VO_2, depend upon the exercise protocol used to ascertain them, and statistically significant differences in VQ have been observed using different procedures in a number of fitness groups and in both genders (Kamon & Pandolf, 1972). VQ is affected by exercise mode and the specific protocol used within an exercise mode (McArdle et al., 2001). VQ observed using a cycling protocol generally is higher than that obtained using a treadmill; VQ's obtained from cycling is on the order of 2.0-5.0 ratio units over a running VQ of 30.0-35.0 (about 6-17% higher: data from Kamon & Pandolf, 1972).

McArdle et al. (2001) state that this ratio in healthy people is on the order of 20-32 (L min^{-1}/L min^{-1}, unitless in other words) at moderate exercise levels, but those values seem to be low compared with some of the data shown in Table 9. VQ values at $VO_{2.MAX}$ in the 40's are seen in the literature (Åstrand & Rodahl, 1986). Miyashita et al. (1981) present data for 5 adult males aged 23-26 y having a VQ mean/SD of 40.7 / 3.3 (COV=8.2%); the VQ range of the group is 37.1- 44.3. The measured VQ at the anaerobic/ventilatory threshold in that study was only about 23.5 (SD=2.3), with a narrow range of 22.0-27.6 (Miyashita et al., 1981).

In general, VQ for sedentary individuals is higher than for more active people (Tanaka et al., 1997). In addition, high VQ values are a marker of inefficient ventilation due to hyperventilation, increased dead space, and/or the "oxygen cost of breathing."

Subjects with heart failure or other respiratory problems have a consistently high VQ (Luks et al., 2012). The VQ for hospital patients with heart failure was 46 ± 11 at peak exercise (which was at a work rate <$VO_{2.MAX}$) and 39 ± 9 at the anaerobic threshold (Mejhert et al., 2002). Nixon et al. (1995) present "mixed gender" VQ data for youths aged 7-30 (mean=13 y) with severe respiratory and/or cardiovascular problems; they also presented VQ data for 41 normal, healthy controls. The testing was done at "peak exercise," and not maximal exercise, due to the nature of the group's health issues: heart and/or lung transplants. The VQ for the control group was 37.1 (range: 30.5-49.8), and with associated $V_{E'S}$ of 72.9 L/min (range: 46.8-138.8) (Nixon, 1996). On the other hand, VQ's for the heart, heart-lung, and lung transplant groups respectively were 52.2 (range: 36.2-63.9), 42.7 (range: 35.4-75.7), and 51.2 (range: 34.9-65.0). The high VQ's for the health-compromised groups were measured even though their corresponding V_E's were lower than the control group, as would be expected. $V_{E.PEAK}$ in L/min for them was—in the same order—51.0 (range: 26.1-87.6), 37.4 (range: 26.2-84.1), and 41.3 (range: 23.4-63.7) (Nixon et al., 1995).

Lean young adults have a higher VQ than moderately obese males of the same age (Wolfe et al., 1976). Children have a higher VQ for the same relative workloads than adults, with VQ averaging around 32 (McArdle et al., 2001). VQ is highly reproducible in children, with a test/retest correlation >0.85 ($p<0.05$) seen in a number of studies reviewed by Mahon & Cheatham (2002). VQ's for elderly females is significantly higher than that for elderly males (Panton et al., 1996).

Some authors state that VQ is non-linear at low VO_2 rates, but that non-linearity does not significantly affect intake dose estimates in our exposure models. In some papers, VQ is defined relative to $VCO_{2.MAX}$ but since EPA's exposure models do not utilize VCO_2 parameters, they are not reviewed here. For one such paper, see McConnell & Davies (1992).

Table 9. Estimates of the ventilatory equivalent (VQ) seen in the literature

Age/Age Range (y)			VQ @ VO2.Max (unitless)					
Mean	SD	Health Status	Mean	SD	COV	Citation	Sample Size (n)	Comment
Females: Normal, Healthy, or Not Specified								
a. Mean & SD statistics are provided for age								
8.5	0.8	N	33.1		*	Wilmore & Sigerseth 1967	20	
9.8	0.7	N	34.8		*	Loftin et al. 1998	19	
10.4	0.5	N	31.8		*	Wilmore & Sigerseth 1967	20	
12.4	0.5	N	29.2		*	Wilmore & Sigerseth 1967	22	

Table 9. Estimates of the ventilatory equivalent (VQ) seen in the literature (continued)

Age/Age Range (y)			VQ @ VO2.Max (unitless)					
Mean	SD	Health Status	Mean	SD	COV	Citation	Sample Size (n)	Comment
16.9	3.0	N	32.8		*	Loftin et al. 1998	19	
19.7	1.0	N	38.3		*	Lesmes et al. 1978	8	Group 1
19.7	1.6	N	40.3		*	Lesmes et al. 1978	8	Group 2
19.9	2.0	N	37.4		*	Lesmes et al. 1978	8	Group 3
20.8	1.1	N	38.1		*	McArdle et al. 1972	35	
22.4	3.5	N	35.7		*	Lesmes et al. 1978	8	Group 4
62.0	6.0	N	39.2		*	Sheldahl et al. 1996	9	Controls
68.6	5.7	N	41.3	7.7	18.6	Panton et al. 1996	36	
68.6	5.7	N	41.3	7.7	18.6	Panton et al. 1996	55	Both genders
68.6	5.7	N	41.3	7.7	18.6	Panton et al. 1996	55	Both genders
b. Complete age statistics are not provided.								
8 - 11		N	39.6	2.3	5.8	Rowland 1997	9	Longitudinal Study
9 - 12		N	39.2	3.4	8.7	Rowland 1997	9	Longitudinal Study
10 - 13		N	38.0	2.3	6.1	Rowland 1997	9	Longitudinal Study
11 - 14		N	39.5	2.0	5.1	Rowland 1997	9	Longitudinal Study
12 - 15		N	39.4	3.9	9.9	Rowland 1997	9	Longitudinal Study
19.5		H	32.6	2.9	8.9	Rowland 1997	15	
Females: Active, Fit, or Athlete								
15.6	1.1	Act	36.0	4.5	12.5	Butts 1982	127	
20.7	3.2	Fit	40.7			MA Sharp et al. 2002	122	Army recruits
21.2	2.3	Ath	36.9		*	McArdle et al. 1972	6	
21.4	3.4	Fit	39.9		*	MA Sharp et al. 2002	155	Army recruits
23.3	3.7	Fit	34.9		*	Williford et al. 1989	10	Aerobic dancers
26.0	3.7	Ath	39.5		*	Tanaka et al. 1997	14	Endurance trained
34.0	14.6	Ath	38.0		*	Tanaka et al. 1997	21	Endurance trained
45.0	3.6	Ath	40.6		*	Tanaka et al. 1997	13	Endurance trained
54.0	4.8	Ath	43.0		*	Tanaka et al. 1997	23	Endurance trained
66.0	3.6	Ath	48.2		*	Tanaka et al. 1997	13	Endurance trained
Females: Sedentary, Overweight, or Obese **a. Mean & SD statistics are provided for age**								
25.0	3.3	Sed	42.0		*	Tanaka et al. 1997	11	
31.9	4.1	Sed	41.3		*	JLP Roy et al. 2006	20	A-A
32.4	5.8	Sed	39.7		*	JLP Roy et al. 2006	30	Caucasian
33.0	13.3	Sed	45.2		*	Tanaka et al. 1997	11	
45.0	3.7	Sed	46.2		*	Tanaka et al. 1997	14	
54.0	4.5	Sed	44.7		*	Tanaka et al. 1997	20	
64.0	4.0	Sed	40.4		*	Tanaka et al. 1997	16	

Table 9. Estimates of the ventilatory equivalent (VQ) seen in the literature (continued)

Age/Age Range (y)			VQ @ VO2.Max (unitless)					
Mean	SD	Health Status	Mean	SD	COV	Citation	Sample Size (n)	Comment
b. Complete age statistics are not provided.								
20 - 29		Sed	30.2		*	Schiller et al. 2001	14	Caucasian
20 - 29		Sed	30.1		*	Schiller et al. 2001	12	Hispanic
30 - 39		Sed	31.6		*	Schiller et al. 2001	14	Caucasian
30 - 39		Sed	35.4		*	Schiller et al. 2001	13	Hispanic
40 - 49		Sed	35.7		*	Schiller et al. 2001	21	Caucasian
40 - 49		Sed	33.0		*	Schiller et al. 2001	8	Hispanic
50 - 59		Sed	36.7		*	Schiller et al. 2001	26	Caucasian
50 - 59		Sed	35.6		*	Schiller et al. 2001	15	Hispanic
> 60		Sed	35.3		*	Schiller et al. 2001	18	Caucasian
> 60		Sed	36.0		*	Schiller et al. 2001	5	Hispanic
Females: Health Issues								
32.0	5.0	Preg.				McMurray et al. 1995	10	Sub-maximal workload
62.0	6.6	CAD	38.9		*	Sheldahl et al. 1996	11	
Males: Normal, Healthy, or Not Specified **a. Mean & SD statistics are provided for age**								
10.8	0.4	H	33.9	2.5	7.4			
12.5	0.9	NS	38.8		*	Maksud & Coutts 1971	17	Age range: 11-14
19.9	0.9	NS			*	Harms et al. 1995	8	FM<7 kg
22.7	4.2	H	41.2		*	Toner et al. 1990	6	
22.7	4.2	H	41.2		*	Toner et al. 1990	6	
22.7	4.2	H	41.2		*	Toner et al. 1990	6	
22.7	4.2	H	41.2		*	Toner et al. 1990	6	
23.2	7.4	NS	29.5		*	Trappe et al. 1996	15	
23.8	3.4	H	32.6		*	Schelegle et al. 1989	20	O3 sensitive
25.5	5.1	H	32.1		*	Schelegle et al. 1989	20	Non O3 sensitive
45.3	8.9	H	29.4		*	Trappe et al. 1996	15	
54.3	9.2	NS	37.7	6.9	18.3	Hansen et al. 1984	77	Shipyard workers
62.0	6.0	N	39.4		*	Sheldahl et al. 1996	9	Controls
64.2	9.4	N	44.2		*	Pollock et al. 1987	13	Ex-athlete
68.7	5.1	N	39.8	8.7	21.9	Panton et al. 1996	55	Both genders
68.7	5.1	N	39.8	8.7	21.9	Panton et al. 1996	55	Both genders
68.7	5.1	N	39.8	8.7	21.9	Panton et al. 1996	55	Both genders
b. Complete age statistics are not provided.								
8 - 11		N	37.2	3.5	9.4	Rowland 1997	9	n=9; Longitudinal Study

Table 9. Estimates of the ventilatory equivalent (VQ) seen in the literature (continued)

Age/Age Range (y)			VQ @ VO2.Max (unitless)					
Mean	SD	Health Status	Mean	SD	COV	Citation	Sample Size (n)	Comment
9 - 12		N	35.3	1.7	4.8	Rowland 1997	9	Longitudinal Study
10 - 13		N	34.4	2.3	6.7	Rowland 1997	9	Longitudinal Study
11 - 14		N	35.9	2.8	7.8	Rowland 1997	9	Longitudinal Study
12 - 15		N	34.1	2.6	7.6	Rowland 1997	9	Longitudinal Study
18 - 23		NS	38.3		*	Seals & Mullin 1982	12	Untrained
19 - 47		H	35.1		*	Lukaski et al. 1989	16	Bruce protocol
Males: Active, Fit, or Athlete								
a. Mean & SD statistics are provided for age								
16.0	1.3	Fit	32.1		*	Rivera-Brown et al. 1997	20	
18.0	2.6	Ath	31.5		*	Rundell 1996	7	Olympic speed skaters
19.9	2.7	Fit	37.4		*	MA Sharp et al. 2002	122	Army recruits
21.8	3.4	Fit	36.1		*	MA Sharp et al. 2002	171	Army recruits
24.8	5.7	Act	32.5		*	Wallick et al. 1995	16	VQ range: 26.2-33.4
25.7	3.5	Act	32.9		*	Trappe et al. 1996	10	
27.1	6.7	Act	28.9		*	Trappe et al. 1996	18	
42.4	14.0	Ath	43.3	5.1	11.8	IE Faria et al. 1996	16	Cross-country skiers
46.8	9.8	Fit	31.3		*	Trappe et al. 1996	10	
47.2	3.8	Ath	29.0		*	Trappe et al. 1996	10	
47.2	5.8	Act	33.7		*	Loftin et al. 1996	12	Handball players
48.7	7.6	Act	29.0		*	Trappe et al. 1996	18	
60.0	8.6	Ath	41.9		*	Pollock et al. 1987	11	Master's athlete
68.4	9.8	Fit	31.9		*	Trappe et al. 1996	10	
b. Complete age statistics are not provided								
18 - 23		Ath	35.7		*	Seals & Mullin 1982	12	row team
18 - 23		Ath	38.8		*	Seals & Mullin 1982	10	Gymnastics team
18 - 23		Ath	36.2		*	Seals & Mullin 1982	11	Swimming team
18 - 23		Ath	36.9		*	Seals & Mullin 1982	10	Wrestling team
24.3		Ath				S Robinson et al. 1976	13	Champion runners
40 - 49		Ath	36.8		*	Pollock 1974	11	
47.9		Ath				S Robinson et al. 1976	13	Champion runners
56.6		Ath				S Robinson et al. 1976	13	Champion runners
50 - 59		Ath	38.5		*	Pollock 1974	5	
60 - 69		Ath	40.8		*	Pollock 1974	6	
70 - 75		Ath	36.5		*	Pollock 1974	3	

Table 9. Estimates of the ventilatory equivalent (VQ) seen in the literature (continued)

Age/Age Range (y)			VQ @ VO2.Max (unitless)					
Mean	SD	Health Status	Mean	SD	COV	Citation	Sample Size (n)	Comment
Males: Sedentary, Overweight, or Obese								
								See Note 1.
a. Mean & SD statistics are provided for age								
19.7	1.6	OW				Harms et al. 1995	8	FM>13 kg
23.5	1.2	Sed	27.9	3.2	11.5	Poole & Gaesser 1985	6	Group 3
23.8	3.6	Sed	32.6	2.9	8.9	Poole & Gaesser 1985	6	Group 2
24.6	6.7	Sed	36.1	5.1	14.1	Poole & Gaesser 1985	5	Group 1
39.1	7.4	Sed	38.8	6.1	15.7	JA Davis et al. 1979	7	Control
43.0	7.2	Sed	39.4	7.2	18.3	JA Davis et al. 1979	9	Group 1
b. Complete age statistics are not provided								
30 - 47		Sed	42.4		*	Pollock et al. 1975	9	Group 1
30 - 47		Sed	42.8		*	Pollock et al. 1975	8	Group 3
30 - 47		Sed	43.3		*	Pollock et al. 1975	9	Group 2
30 - 47		Sed	43.3		*	Pollock et al. 1975	7	Control Group
49 - 65		Sed	39.0		*	Pollock et al. 1976	7	Control Group
49 - 65		Sed	42.5		*	Pollock et al. 1976	22	Group 1
Males: Health Issues								
55.0	9.0	CHF	39.0	6.7	17.2	J Myers et al. 2012	24	Exercise Group
57.0	7.0	CHF	29.1	9.4	32.3	J Myers et al. 2012	26	Control Group
56.0	7.3	RP				Furuike et al. 1982	23	See Note 2
64.0	3.0	CAD	33.2		*	Sheldahl et al. 1996	9	
68.0	5.7	CAD	34.7		*	Sheldahl et al. 1996	8	
69.0	3.3	CAD	39.1		*	Sheldahl et al. 1996	11	
Both Genders: Normal, Healthy, or Non-Specified								
3 - 4		N	30.8		*	Shuleva et al. 1990	9	33% female
5 - 6		N	29.8		*	Shuleva et al. 1990	13	23% female
25.0	8.0	H	38.0	8.0	21.1	Shah et al. 1998	17	Controls (41% female)
70+		NS	38.1	8.8	23.1	BD Johnson et al. 1991	30	
70+		NS	38.1	8.8	23.1	BD Johnson et al. 1991	30	
70+		NS	38.1	8.8	23.1	BD Johnson et al. 1991	30	
70+		NS	38.1	8.8	23.1	BD Johnson et al. 1991	30	
Both Genders: Health Issues								
25.0	10.0	CF	38.0	8.0	21.1	Shah et al. 1998		
32.0	9.0	CHF	38.0	7.0	18.4	Weber et al. 1982	5	Group A
50.0	13.0	CHF	45.0	17.0	37.8	Weber et al. 1982	19	Group C

Table 9. Estimates of the ventilatory equivalent (VQ) seen in the literature (continued)

Age/Age Range (y)			VQ @ VO2.Max (unitless)					
Mean	SD	Health Status	Mean	SD	COV	Citation	Sample Size (n)	Comment
53.0	14.0	CHF	38.0	15.0	39.5	Weber et al. 1982	14	Group D
55.0	17.0	CHF	40.0	8.0	20.0	Weber et al. 1982	17	Group B
62.0	7.0	COPD	43.0	6.0	14.0	JL Larson et al. 1999	12	Group 1
66.0	5.0	COPD	42.0	9.0	21.4	JL Larson et al. 1999	13	Group 2
66.0	6.0	COPD	40.0	9.0	22.5	JL Larson et al. 1999	14	Group 3
68.0	6.0	COPD	40.0	4.0	10.0	JL Larson et al. 1999	14	Group 4

Symbols & Abbreviations:	
♀	Females
♂	Males
*	Calculated from VE & VO2 Max data provided
A-A	African-American
Act	Active
Ath	Athlete
CF	Cystic Fibrosis
CHF	Chronic Heart Failure
COPD	Chronic Obstructive Pulmonary Disease
CV	Coefficient of Variation
FM	Fat Mass
Max	Maximum oxygen consumption (L/min)
n	Sample size
N	Normal

Symbols & Abbreviations:	
Preg	Pregnant
RP	Respiratory problems (patients)
SD	Standard deviation
Sed	Sedentary
VE	Ventilation rate (L/min)
VO2	Oxygen Consumption (L/min)
y	Years

Notes:

1. If no value is shown in the “Specified Work Rate” column, it means that it was at VO2.Max.

2. VE value shown is the mean ventilation rate (SD=5.8; range: 20.9-1.7 L/min); the VQ range was 25.9-43.3 (unitless).

6.0
METS Considerations

METS, the metabolic equivalent of work, can function as a reserve metric due to its definition. METS are the *ratio* between activity-specific energy expenditure (EE_{ACT}) to RMR or oxygen consumption ($VO_{2.ACT}$) to RMR (in VO_2 units, of course). Since RMR is defined to be a MET of 1.0, $METS_{RES} = METS_{MAX} - 1.0$. It is a unitless metric.

As noted earlier, to simplify the estimation of activity-specific METS, exercise physiologists have developed the concept of a "standardized" MET (Ainsworth et al., 1993, 2000, 2011) and set it equal to 3.5 mL kg^{-1} min^{-1} in oxygen consumption units. Pettitt et al. (2007) states that this factor was based on observations from only one person, and cites Byrne et al. (2005) as the source for this statement. However, the "one-person" statement is not discussed in that source, so there is uncertainty concerning validity of the Petitt et al. (2007) statement.

We do not use standardized METS in this paper. In fact, examining the reasons why standardized METS should *not* be used is one of the main themes of this work. In short, EE rates based on a standardized MET under-estimates activity specific METS ($METS_{ACT}$) in most adults for all but the most sedentary activities (Kozey et al., 2010; Manore et al., 1991) because it over-estimates RMR (Bryne et al., 2005; Lee et al., 2010a, b; McMurray et al., 2014). For example, RMR is 3.1 mL kg^{-1} min^{-1} in people with paraplegia, which is statistically different than 3.5 mL kg^{-1} min^{-1} (Lee et al., 2010a). Conversely, $METS_{ACT}$ are over-estimated in overweight and obese individuals using a standardized MET because it under-estimates RMR on a body-mass basis in those people (Rachette et al., 1995). RMR in children and adolescents generally is higher than 3.5 mL kg^{-1} min^{-1}, although it approaches that value in males around puberty (Son'kin & Tambovtseva, 2012), further complicating use of the standard METS concept.

In a large homogeneous sample of subjects aged 18-74 using the 3.5 value overestimates $VO_{2.REST}$ by 35%, on average (Byrne et al., 2005). While a range of measured $VO_{2.REST}$ versus 3.5 was not provided in their paper, only 14 (2%) of the 769 subjects in the study had a resting $VO_2 \geq 3.5$ mL kg^{-1} min^{-1}. These 14 subjects were heterogeneous in age and included both genders, but all had a low relative body weight, with a Body Mass Index (BMI) between 16-22 kg m^{-2} (Byrne et al., 2005). The authors obtained better results using the 1 kcal kg^{-1} h^{-1} $VO_{2.REST}$ "constant" that is equivalent to 3.5 mL kg^{-1} min^{-1}. Doing so still overestimates resting oxygen consumption by 20%, on average; better, but still not good. A paper by KS Hall et al. (2013) confirms that measured REE is 31.6% on average lower than the 3.5 resting METS value, which results in METS estimates in the Compendium that are lower than measured METS by 71%, on average, for 60% of walking activities (Hall et al., 2013).

McMurray et al. (2014) reviewed REE data from 197 studies of adults that clearly show that measured REE is <1 kcal kg^{-1} h-1 in the vast majority of age/gender cohorts that were investigated. Overall, the mean REE was 0.863 kcal kg^{-1} h^{-1} (95% CI=0.852-0.874), or about 14% lower than that normally used for REE (McMurray et al., 2014). REE for females was 0.839 kcal kg^{-1} h^{-1} (CI=0.825-853) and was 0.892 kcal kg^{-1} h^{-1} (0.872-0.912) for males, highlighting another problem of using the same value for both genders.

One alternative to the standardized MET approach is to use "corrected MET" values, which are defined in the following webpage: (http://sites.google.com/site/compendiumofphysicalactivities/home). That definition, however, has its own set of problems, since it is explicitly based on using the Harris-Benedict BMR equation (H-B BMR) developed in 1918 (Harris & Benedict, 1918). The corrected METS equation from the website is:

Corrected METS = Compendium METS Value * (3.5 ml kg^{-1} min^{-1} / H-B BMR ml kg^{-1} min^{-1})

In general, corrected METS will be larger than the Compendium-METS estimates as the H-B BMR estimate is <3.5 ml kg^{-1} min^{-1}. See the webpage for examples.

Activity-specific METS ($METS_{ACT}$) are often estimated from HR-monitoring studies using the Karvonen approach, as clearly described in Strath et al. (2000). The Strath example uses the 3.5 factor for $VO_{2.REST}$, but does not have to, since $VO_{2.REST}$ could have been measured directly. Their logic steps follow:

1. Obtain activity-specific HR (HR_{ACT}). HR_{REST} and HR_{MAX} have previously been measured.
2. Obtain equivalent %HRR for an activity ($\%HR_{ACT}$).
3. $HRR_{ACT} = [(HR_{ACT} - HR_{REST}) / (HR_{MAX} - HR_{REST})] * 100$
4. Assume for an activity that: $\%VO_{2.RES.ACT} = \%HRR_{ACT}$
5. Estimate $VO_{2.ACT}$:
6. $VO_{2.ACT} = [(\%VO_{2.RES.A}/100)* (VO_{2.MAX} - VO_{2.REST})] + VO_{2.REST}$
7. Calculate $METS_{ACT}$: $METS_{ACT} = (VO_{2.ACT} / VO_{2.REST})$

Where HRR=Heart Rate Reserve.

Activity-specific METS are briefly discussed in Section 11 of this report. To link the METS approach to Section 10 that is focused on moderate and vigorous physical activity defined by accelerometers, it should be recognized that the relationship between accelerometer counts and METS estimates are non-linear. In general, METS estimates decrease with accelerometer counts as counts increase (Agiovlasitis et al., 2012). Thus, higher accelerometer counts will underestimate activity-specific METS associated with an undertaking.

We are most interested in using the bounded $METS_{RES}$ metric as a way to improve intake dose rate estimation procedures used in our exposure models. We present what little data on $METS_{MAX}$ is seen in the literature in Table 10. Estimates of $METS_{.RESERVE}$ can easily be obtained by subtracting 1 from the values shown. Many of the values seen in the Table are from studies that reported both $VO_{2.REST}$ and $VO_{2.MAX}$ data. Using the metabolic chronotrophic relationship discussed in the next Section, $VO_{2.RESERVE} = METS_{.RESERVE}$, thus:

$((VO_{2.MAX} - VO_{2.REST}) / VO_{2.REST}) = ((METS_{MAX} – METS_{REST}) / METS_{REST})$.

Since $METS_{REST} = 1$ by definition, $((VO_{2.MAX} - VO_{2.REST}) / VO_{2.REST}) = (METS_{MAX} -1)$. Rearranging and simplifying terms, we get: $METS_{MAX} = VO_{2.MAX} / VO_{2.REST}$. This is the approach used to obtain many of the data entries appearing in Table 10.

$METS_{MAX}$ has long been used in prescribing exercise limits in cardiovascular patients (Bourque et al., 2009; Morris et al., 1993; and see Appendix B). In general, these researchers first identify people, who are mostly male, who cannot achieve 85% of their age-predicted HR_{MAX} and then subject them to a graded treadmill exercise test that is indexed as METS. Those that pass the HR criterion, are then categorized into 3 groups: <7 METS, 7.0-9.9 METS, and ≥10 METS. A cutpoint of 10 METS or higher predicts low mortality, even when coronary artery disease is present (Bourque et al., 2009). Patients in the lower two groups have a higher prevalence of ischemia, and that is inversely related to the METS levels attained (Bourque et al., 2009). It should be noted that these METS levels are all quite high when compared to the "standard" METS criteria of 3-5.9 METS for moderate physical activity (PA) and ≥6 METS for vigorous PA.

There are a number of studies that confirm the "protective role" of a higher $METS_{MAX}$ capacity in these patients, even in the presence of other risk factors; the risk of death from any cause in subjects with a $METS_{MAX}$ <5 was double that of subjects with a $METS_{MAX}$ of 8 or higher (Myers et al., 2002). Their sample size was about 6,200 subjects. Their findings are similar to those of previous researchers (Blair et al., 1989, 1995; Ekelund et al., 1988; Franklin & Swain, 2003; Haskell et al., 1992).

The $METS_{MAX}$, concept, by the way, has been used by EPA since 2003 to "cap" youth $METS_{MAX}$ as a part of the CHAD database (www.epa.gov/heasd/chad.html). The cap was calculated from a "means-of means" analysis undertaken in 2001, and was slightly modified in the McCurdy & Graham (2004) report. The caps are provided—as $METS_{MAX}$ limits--on the CHAD webpage. In the 2004 report, we recommended that in an exposure modeling application using the usual algorithms, if any $METS_{ACT}$ estimate was obtained that was >$METS_{MAX}$ limits for the age/gender cohort, then the $METS_{ACT}$ estimate be set at the limit. We were fully aware that doing so may result in a "skewed" distribution of $METS_{ACT}$ values for a particular activity code, but data were—and still are--weak concerning those distributions anyway, and there are no data available to us to test the practical impact of using the maximum $METS_{ACT}$ limit decision rule. More rigorously: $METS_{ACT}$ are always ≤$METS_{MAX}$. The CHAD $METS_{MAX}$ caps follow.

Age	Females	Males
6	5.4	6.2
7	5.8	6.7
8	6.3	7.2
9	6.7	7.7
10	7.2	8.2
11	7.7	8.8
12	8.3	9.5
13	9.0	10.1
14	9.6	10.8
15	10.3	11.6
16	11.2	12.4

Jakicic et al. (2010) provide interesting data on total METS-minutes that obese adults (both genders) participate in versus their $METS_{MAX}$ categories. These data do not fit into any of the Tables in this report and so will be only discussed here. One important finding is that total daily METS-minutes is monotonically related to $METS_{MAX}$ in obese adults with diabetes. Diabetics—both diagnosed and undiagnosed--comprise about 7.8% of the total US population, so the susceptible group of concern is quite large. The study from which the following data are taken comprise 5,145 individuals, 2,145 of whom carried a RT3 accelerometer. The average age of the sample was 59.0 ± 6.8. The relative

Table 10. Estimates of METS.$_{Max}$ seen in-or calculated from--the literature

METS.Max Estimate (unitless)			
Age Range (Mean ± SD)	**Mean ± SD**	**Citation**	**Comment**
Females: Normal, Healthy, or Not Specified			
13.1 ± 2.0	13 ± NS	Hui & Chen 2006	Calc. from VOmax & VOrest in Table 6.
14 ± NS	7.0 ± NS	Wilson et al. 1985	Calc. from VOmax & VOrest in Table 6.
24.3 ± 4.2	9.7 ± NS	Frew et al. 1993	Calc. from VOmax & VOrest in Table 6.
31.1 ± 8.8	11.1 ± NS	Dalleck & Kravitz 2006	Calc. from VOmax & VOrest in Table 6.
55.7 ± 7.8	9.0 ± NS	Nikolai et al. 2009	Calc. from VOmax & VOrest in Table 6.
Females: Active, Fit, or Athlete			
13 - 19	15.1 ± NS	Guidetti et al. 2000	Calc. from VOmax & VOrest in Table 6.
21.8 ± 6.0	12.0 ± NS	Blanksby & Reidy 1988	Competitive dancers
27.8 ± 2.6	13.1 ± NS	Frey et al. 1993	Calc. from VOmax & VOrest in Table 6.
Females: Sedentary, Overweight, Obese, or Health Issues			
14 ± NS	7.1 ± NS	Wilson et al. 1985	Calc. from VOmax & VOrest in Table 6.
26.8 ± 7.9	9.3 ± NS	M Lee et al. 2010a	Calc. from VOmax & VOrest in Table 6.
Males: Normal, Healthy, or Not Spec.			
13.9 ± 1.9	12.4 ± NS	Hui & Chen 2006	Calc. from VOmax & VOrest in Table 6.
14 ± NS	7.1 ± NS	Wilson et al. 1985	Calc. from VOmax & VOrest in Table 6.
29.2 ± 6.8	8.7 ± NS	Dalleck & Kravitz 2006	Calc. from VOmax & VOrest in Table 6.
41.7 ± 8.8	10.1 ± 2.1	Blair et al. 1995	Measured via a maximal/resting fitness test.
59.1 ± 7.6	9.0 ± NS	Nickolai et al. 2009	Calc. from VOmax & VOrest in Table 6.
Males: Active, Fit, or Athlete			
23.2 ± 6.3	10.7 ± NS	Blanksby & Reidy 1988	Competitive dancers
Males: Sedentary, Overweight, Obese, or Health Issues			
14 ± NS	7.7 ± NS	Wilson et al. 1985	Calc. from VOmax & VOrest in Table 6.
22.5 ± 4.4	9.2 ± NS	M Lee et al. 2010a	Calc. from VOmax & VOrest in Table 6.
27.4 ± 8.1	7.4 ± NS	Davis & Shephard 1988	Calc. from VOmax & VOrest in Table 6.
28.1 ± 5.8	9.3 ± NS	Davis & Shephard 1988	Calc. from VOmax & VOrest in Table 6.
46.8 ± 9.6	11.0 ± 2.2	Blair et al. 1995	From a maximal/resting fitness test.
Both Genders: General Estimates			
Young	13	McArdle et al. 2001	Brochard et al. 1990 Table 9.5
Middle Age	10	McArdle et al. 2001	Brochard et al. 1990 Table 9.5
Old	7	McArdle et al. 2001	Brochard et al. 1990 Table 9.5
Very Old	4	McArdle et al. 2001	Brochard et al. 1990 Table 9.5
20 - 39	12	U.S. Dept. Health & HS	Table 2-4; females 1-2 METS lower
40 - 64	10	U.S. Dept. Health & HS	Table 2-4; females 1-2 METS lower
65 - 79	8	U.S. Dept. Health & HS	Table 2-4; females 1-2 METS lower
80+	5	U.S. Dept. Health & HS	Table 2-4; females 1-2 METS lower
30 (Sed.)	10	Franklin 2000	

Table 10. Estimates of $METS._{Max}$ seen in-or calculated from--the literature (continued)

METS.Max Estimate (unitless)			
Age Range (Mean ± SD)	**Mean ± SD**	**Citation**	**Comment**
30 (Athlete)	23	Franklin 2000	
Both Genders: Normal, Healthy, or Not Specified			
6 - 14	8.2 ± NS	Cabrera et al. 2002	BSA < 1.1
6 - 14	6.7 ± NS	Cabrera et al. 2002	BSA=1.1-1.4
6 - 14	8.1 ± NS	Cabrera et al. 2002	BSA> 1.4
Both Genders: Sedentary, Overweight, Obese. Or Not Healthy			
24.1 ± 6.3	8.4 ± NS	M Lee et al. 2010b	Calc. from VOmax & VOrest in Table 6.
27.8 ± 5.6	6.0 ± NS	PL Jacobs et al. 1997	Calc. from VOmax & VOrest in Table 6.
58.0 ± 7.0	6.6 ± NS	Colberg et al. 2003	Calc. from VOmax & VOrest in Table 6.
62.9 ± 10.1	5.2 ± NS	Colberg et al. 2003	Calc. from VOmax & VOrest in Table 6.

Abbreviations:	
HS	Human Services
NS	Not specified/not calculated
Sed.	Sedentary

number of accelerometer participants was not proportional to people in the different $METS_{MAX}$ categories, so there is a disjunct in extrapolating the data, limiting conclusions that can be drawn from the analysis. By $METS_{MAX}$ category, the following METS-minute data can be obtained:

$METS_{MAX}$ Category	Average MET_Min of Activity Undertaken	Percent of Group that Partic. in Bouts
< 6.0	16.9	59.2
6.0 - 7.9	19.8	72.1
8.0 – 9.9	22.2	79.5
≥ 10.0	25.8	88.4

(Partic. = Participates)

Both the total MET-minutes and the average energy expended per one MET-minute increased with $METS_{MAX}$ category (Jakicic et al., 2010), so the relationship is not linear. The last column reflects the percentage of people (both females and males) that undertook exercise for one or more 10 min bouts with at least a 3 METS level of energy expenditure as estimated by a RT3 accelerometer (Jakicic et al., 2010). Thus, the higher a person's $METS_{MAX}$ fitness level is, the more frequent and/or the longer they participate in higher energy expenditure activities. These are intuitively appealing findings.

Morris et al. (1993), practicing cardiologists, developed regression equations for males using subjects between the ages of 18-89 y. They disaggregate their subjects into four groups: 1,338 patients referred to them for evaluation of possible coronary artery disease, divided into sedentary and active patients; and, 196 volunteers who undertook the same type of treadmill VO_2 tests, also divided into active and sedentary subjects. The referral subjects did not include severe heart patients, who were excluded for safety reasons (Morris et al., 1993). A reproduction of their $METS_{MAX}$ prediction regression equations follows.

Referral patients, active:

$$METS_{MAX} = 18.7 – (0.15 * Age)$$
n=346, SEE = 3.0, r= -0.49, p<0.001

Referral patients, sedentary:

$$METS_{MAX} = 16.6 – (0.16 * Age)$$
n=253, SEE = 3.2, r= -0.43, p<0.001

Volunteers, active:

$$METS_{MAX} = 16.4 – (0.13 * Age)$$
n=122, SEE = 2.5, r= -0.58, p<0.001

Volunteers, sedentary:

$$METS_{MAX} = 11.9 – (0.07 * Age)$$
n= 74, SEE = 1.8, r= -0.47, p<0.001

These equations produce consistently lower $METS_{MAX}$ for sedentary individuals than active people for the two groups, and also predict higher $METS_{MAX}$ for volunteers than for referral patients. Applying these equations results in pretty high $METS_{MAX}$ estimates at the 95% prediction interval (e.g., 15 METS for 60 y olds, and 10 METS for 80 y old people having possible coronary patients). These values are considerably higher than those appearing in Table 10. It is quite obvious that additional data on cohort-specific $METS_{MAX}$ limits are needed, especially if we anchor our $METS_{ACT}$ estimates using the metabolic chromotropic relationship. A concerted effort should be made to thoroughly review the literature to come up a more complete database of $METS_{MAX}$ value than I was able to undertake.

7.0
Metabolic Chronotropic Relationship

A unifying approach to linking HR and other physiological parameters to VO_2 and METS is called the metabolic chronotropic relationship (MC relationship). It also is known as the heart rate/work rate ratio (HR/WR) (Lewather et al., 1999; Wilkoff & Miller, 1992). The MC relationship is functionally very similar to the Karvonen approach and is based upon using reserve metrics to relate submaximal exercise stages. Proponents of the relationship state that it adjusts for age, physical fitness, and functional capacity of an individual and appears to be unaffected by exercise testing mode or protocol (Brubaker & Kitzman, 2011). It often is stated in the physiological literature that energy expenditure for a specific activity will elicit the same *relative* HRR and $VO_{2.RES}$. This seems to be particularly true when a *single* exercise protocol is used for work rates lower than the lactate threshold.

In short, the MC relationship implies that %HHR = $\%METS_{RES}$ = $\%VO_{2RES}$, and implies that the slope of relationships between any two of these metrics is = 1.0 and that their intercept = 0.0 (Lewalter et al., 1999). For instance, Dalleck & Kravitz (2006) regress %HRR and $\%VO_{2.RES}$ for different exercise rates in 48 adults of both genders on an elliptical crosstrainer and obtained the following relationship: %HRR = -0.7 + [1.01 * $\%VO_{2.RES}$], R^2=0.99, p<0.001 (SE not provided). The intercept was not significantly different than 0.0 and the slope was not different than 1.0 (Dalleck & Kravitz, 2006). Another example of good regressions for the two metrics is shown in Hui & Chan (2006) for Chinese youth aged 10-17. They report an R^2 of 0.92 for a treadmill equation for females of the form %HHR = 22.9% + (0.79 * $\%VO_{2.RES}$) [SEE=0.01%]. For males, it was %HHR = 15.7% + (0.85 * $\%VO_{2.RES}$) [R^2=0.90; SEE=0.01%]. Even though these results seem to be excellent, the authors state that "the equivalency between %HRR and $\%VO_2R$ [their symbol for $\%VO_{2.RES}$] was not confirmed in the present study" (Hui & Chan, 2006; p. 48), perhaps because they estimated HR_{MAX} using the 220-Age formula; see the discussion below regarding this way of estimating HR_{MAX}. A study of 6 Japanese female black-belt karate participants aged 20 y also reported a very good relationship between HRR and $VO_{2.RES}$. The regression was %HRR = 5.72 + (1.01 * $\%VO_{2.RES}$) [R^2=0.98; no SEE presented] (Imamura et al., 2002). Swain (2000) and Swain et al. (1998) also develop %HRR→$\%VO_{2.RES}$ regressions with very high R^2's.

The strong association of the two reserve metrics deteriorates somewhat when a *set* of disparate activities is evaluated or people with health issues are tested. Carvalho et al., (2008) regressed %HRR against $\%VO_{2.RES}$ in heart failure patients using optimized beta blocker regimes; the subsequent regression equation had an R^2 of 0.90 (SE= 1.8%). The same results for patients without an optimized drug regime was R^2=0.83; SE=2.1%. Not bad in either case, and Carvalho et al. (2009) found a good relationship between the two reserve metrics in a study of heart transplant patients. The regression equation in that study (%HRR=13.3 + [0.88* $\%VO_{2.RES}$]) had an R^2=0.89 (no SE provided). A study of older diabetics also found a close relationship between %HRR and $\%VO_{2.RES}$ (Colberg et al., 2003).

However, it appears that the %HRR to $\%VO_{2.RES}$ relationship is imprecise for people with a *blunted* HR response to exercise, whether it is due to physical problems or to age (Patterson et al., 2005). In a study of heart disease patients, both the slope and intercept of a number of %HRR→$\%VO_{2.RES}$ regressions are significantly different at $p \leq 0.01$ (Brawner et al., 2002). In addition, Cunha et al. (2010, 2011) have shown that the VO_2 testing protocol used itself significantly affects the %HRR→ $\%VO_{2.RES}$ relationship in healthy individuals, depending upon how fit they were and their prior physical activity patterns. They found that in a number of individuals, there was a better relationship between %HRR and $\%VO_{2.MAX}$ than between %HRR and $\%VO_{2.RES}$ (Cunha et al., 2010). The %HRR to $\%VO_{2.RES}$ relationship is also not very good for non-traditional exercise protocols not involving large muscles (Rothstein & Meckel, 2000). That also is the finding of Mendez-Villanueva et al. (2010) for arm-paddling exercise in highly-trained surfboard riders; both the mean slope of the regression between the two metrics and the intercept is significantly different than 1 and 0, respectively, for arm-exercise, contrary to what was found for lower-body exercise (Mendez-Villanueva et al., 2010).

Like the absolute HR-to- VO_2 relationship, the association between %HRR and $\%VO_{2.RES}$ is not linear over the entire range. %HRR and $\%VO_{2.RES}$ is approximately linear up to the "gas exchange threshold" (Pettit et al., 2008), which seems to be a new name for anaerobic threshold, but the relationship becomes non-linear above it. (See comments below regarding this threshold concept.) The relative variability (coefficient of variability: COV) in the two reserve metrics is different for people with similar age and gender characteristics: on the order of 19-24% for $VO_{2.RES}$ and 24-26% for HRR (Nikolai et al., 2009). This finding does not indicate a consistently linear relationship between HRR and $VO_{2.RES}$. Lounana et al. (2007) state that predicted $\%VO_{2.RES}$ values were equivalent to %HRR values in the 35-95% HRR range, but diverged on either side of that range.

This infers that a linear relationship among the three reserve parameters exists (Lewather et al., 1999), but in reality there is variability among persons at different relative work rates, and variability along the work rate continuum within an individual (Lewather et al., 1999) Thus the MCR function is not a smooth one, but has a slope only ≈1.0 over the work rate range. The inter- and intra-individual variability of the MC relationship results in a 95th confidence interval for the slope of 0.8-1.3 (Brubaker & Kitzman, 2011; Coman et al., 2008). Wilkoff & Miller (1992) state that the 95th confidence interval for the relationship is 0.79-1.33 in CEAP patients (those having chronic heart rate issues and using the chronotropic assessment exercise protocol) and 0.97-1.02 in "normals" using the Bruce protocol. Not only is the MC relationship only approximately linear, it seems to be so for most people only at exercise rates between 35-95% of $VO_{2.RES}$ (Lounana et al., 2007). The divergence from linearity can be about 8% for an individual at a particular work rate depending upon the protocol used (Cunha et al., 2011a, b). In fact, Cunha et al. (2011b) present a Table showing 25 regression equations relating %HRR (dependent variable) to % $VO_{2.RES}$ with slope values between 0.83 and 1.08. Most slope values are within 3% of 1.00, however. Wilkoff et al. (1989) provide the following regression equations for CEAP patients and "normals" that are informative: %HRR = 4.6 + (0.94 * %$METS_{RES}$; R^2=0.96) and %HRR = 3.4 + (0.94 * %$METS_{RES}$; R^2=0.98), respectively. Their relative deviations around the 0.0 intercept were 7.7% and 5.0%, respectively (Wilkoff et al., 1989). A study of the MC relationship in sedentary heart transplant patients by Carvalho et al. (2010) produced a weaker %HRR-%$VO_{2.RES}$ regression equation (R^2=0.89) and a higher intercept value (13.3). A much more positive finding from a regression of %HRR on % VO_{2RES} is found in Brawner et al. (2002), also involving patients with heart disease. For three different subgroups of that population, none of the slopes were significantly different from the line of identity. Thus, there is a wide variety of findings in the literature regarding the %HRR-%$VO_{2.RES}$ MC relationship.

Cardiologists use the MC relationship to identify people with cardiovascular, particularly cardiac, problems, such as myocardial ischemia (Lauer et al., 1998). In these cases, the MC relationship is called the "cardiac chronotropic relationship" or the "chronotropic index" (Brubaker & Kitzman, 2007). Since it is dangerous to exercise people with heart problems at maximal rates, HRMax is estimated by 220-Age rather than being measured; this introduces a lot of uncertainty into the concept. A person with an MC relationship <0.8 (or, alternatively, <0.85) is said to have chronotropic incompetence (CIComp), defined to be an inability of the heart to increase its rate commensurate with increased activity or demand (Brubaker & Kitzman, 2011; Chin et al., 1979; Coman et al., 2008; Wilkoff & Miller, 1992). Alternative ratios have also been used (0.70, 0.75. 0.85), resulting in a fairly wide range of CIComp prevalence rates in the general population when coupled with different incremental dynamic exercise protocols (Brubaker & Kitzman, 2011; Lauer et al., 1998, 1996, 1999). Lewather et al. (1999) states than *any* deviation of the slope 1.0 between HR and metabolic reserve (METS) "can be used to define a potentially abnormal rate response to exercise" (p. 361). Thus, while the numerical definition of CIComp is not a settled concept, the MC *relationship* is receiving wide acceptance as a method for better relating physiological parameters over the range of exercise rates possible in humans (Coman et al., 2008; Cunha et al., 2011b; Panton et al., 1996; Swain et al., 1994, 1998; Swain & Leutholtz, 1997). We are considering using the MC relationship in the future to better relate activity-specific work rates (EE_A in kcal/kg-min) that are used in the APEX and SHEDS models to model intake dose rates of exposed persons.

With respect to oxygen consumption (VO_2) and metabolic equivalents of work (METS), the MC relationship can be stated as:

$$VO_{2.ACT} \mid VO_{2.RES} = (VO_{2.CT} - VO_{2.REST}) / (VO_{2.MAX} - VO_{2.REST}) = METS_{ACT} \mid METS_{RES} = METS_{ACT} \mid (METS_{MAX} - 1)$$

Where:

$METS_{ACT}$ =	Activity-specific METS
$METS_{MAX}$ =	Maximum METS
$METS_{RES}$ =	METS Reserve ($METS_{MAX}$ – 1.0)
$VO_{2.ACT}$ =	Activity-specific VO_2
$VO_{2.MAX}$ =	Maximal VO_2
$VO_{2.REST}$ =	Resting VO_2

Notes:| ="given" or "conditioned upon"
By definition, resting METS=1.0

There is an idea that changes in body size alter the metabolic requirements of *relative work rates* rather than *absolute work rates*, and this results in a constant "per kg" metric. There is intuitive support for this concept, backed up observations from animal and human experiments (Rowland, 2012). Subtracting resting (basal) metabolic rate from activity-specific energy expenditure minimizes age and gender differences in activity-specific EE at similar work rates (Rowland, 2011, 2013). However, it has been known for a long time that "normalizing" work rates by a fat-free or lean-body mass minimizes these differences even more. See Åstrand & Rodahl (1986), Boileau & Horswill (2000), or McArdle et al. (2001), for additional information. Some information relating $METS_{MAX}$, HRR, and $VO_{2.MAX}$ "workload intensities" for differing fitness levels is presented in Table 11.

Table 11. "WORKLOAD INTENSITY" for differing METS.$_{Max}$ fitness level

Intensity Level Category	Corresponding VO2Reserve & HRR Levels %	METS & Corresponding VO2Max Percentages			
		Max METS = 5.0		Max METS = 8.0	
		METS	%VO2 Max	METS	%VO2 Max
Light	20 - 39	1.8 - 2.5	36 - 51	2.4 - 3.7	30 - 47
Moderate	40 - 59	2.6 - 3.3	52 - 67	3.8 - 5.1	48 - 64
Hard	60 - 84	3.4 - 4.3	68 - 87	5.2 - 6.9	65 - 86
Very Hard	≥ 85	≥ 4.4	≥ 88	≥ 7.0	≥ 87
		Max METS = 10		Max METS = 12.0	
		METS	%VO2 Max	METS	METS
Light	20 - 39	2.8 - 4.5	28 - 45	3.2 - 5.3	27 - 44
Moderate	40 - 59	4.6 - 6.3	46 - 63	5.4 - 7.5	45 - 62
Hard	60 - 84	6.4 - 8.6	64 - 86	7.6 - 10.2	63 - 85
Very Hard	≥ 85	≥ 8.7	≥ 87	≥ 85	≥ 86
		METS Reserve Levels Associated with the Above Max METS			
		5	8	10	12
Light	20 - 39	0.8 - 1.6	1.4 - 2.7	1.8 - 3.5	2.2 - 4.3
Moderate	40 - 59	1.6 - 2.4	2.8 - 4.1	3.6 - 5.3	4.4 - 6.5
Hard	60 - 84	2.4 - 3.4	4.3 - 5.9	5.4 - 7.6	6.6 - 9.2
Very Hard	≥ 85	≥ 3.4	≥ 6.0	≥7.7	≥9.3

Abbreviations:	
HR	Heart Rate
METS	Metabolic equivalents (of work)
VO2	Oxygen consumption

Source: Kesaniemi et al. (2001).

Note: Kesanniemi et al. (2001) also provide an estimate of "maximal" workload intensity, but it simply is the Max METS value seen in the corresponding column at 100% of VO_2 & HR Reserve. Their "very light" category was deleted;it equaled <20% of HRR & VO_2reserve (=50% HRmax).

8.0
Daily Total Energy Expenditure (DTEE)

In reality we would like to have estimates of intake energy (EI) for purposes of exposure modeling, especially for dietary exposure analyses. The equating of energy intake with energy expenditure--plus change in body stores--is a "fundamental property" of thermodynamics and is the basis for estimating nutritional needs of mammals (Schoeller, 2009). In humans, energy input is the amount of chemical energy entering the body that can be liberated via metabolism; it is measured by metabolizable energy. It is very difficult to estimate EI in practice and most methods used to do so lead to systematic errors in reporting calories consumed, especially among overweight and obese individuals (Champagne et al., 2002). Thus, energy expenditure is measured or estimated in order to accurately estimate EI in the general population (Livingstone & Black, 2003; Schoeller, 2009).

Methods used to estimate EE intake on a daily (DTEE) or other time-basis include dietary records (diary), ex post food frequency questionnaires, and duplicate food portion studies. The latter studies generally are relatively accurate but are very expensive to undertake. Studies that compare DTEE measured by DLW (see below) with that determined by a dietary diary approach, the second-most accurate method of estimating EI, have found that EI is systematically under-reported by 20-30%, on average by youth (Champagne et al., 1998) and by 10-30% in adults (Hebert et al., 2002). In overweight or obese females and those having a high need for "social approval," the underestimate of EI is even higher (Hebert et al., 2002). Studies that compare DLW measures against food-frequency questionnaires or seven-day dietary recall estimates show low correlations between EE and EI measures. For example, low and statistically insignificant correlations of 0.12-0.14 between EE and EI are reported by Hebert et al. (2002). The only group tested that can accurately estimate EI from a food diary compared to DLW measures of EE, is professional dietitians (Champagne et al., 2002). See also Trabulski & Schoeller (2001) and Tran et al. (2000) for dietary intake and DLW comparisons.

The literature review used to obtain DTEE data in US citizens followed the pattern explained above in the VO_2 and subsequent Sections. Literature searches supplied by the EPA Library originally identified about 1,200 papers that focused on energy expenditure concepts in human, and their abstracts were reviewed. A number of these were acquired and further reviewed, and relevant references were identified as being of potential interest. These papers (or books; hereafter "papers") were obtained and evaluated for relevancy. At this point, 1,617 papers were identified as being of potential interest. All of them were obtained—in whole or in part (abstract only) and were systematically, but briefly, reviewed to determine which might contain DTEE data derived from doubly labeled water (DLW) studies of US citizens. Of the total papers, 736 (45.5%) were identified for detailed review. Of the 881 papers that were rejected for further review at this stage, 379 contained only resting energy data or REE estimates using only statistical prediction equations (23.4% of the total pursued); 207 did not use DLW to estimate daily energy expenditure (12.8%) or were a methods comparison study; and 174 papers used non-US subjects (10.8%). The remaining 121 papers were rejected because they provided redundant data (for example, DeLany et al., 2002, which also appeared in other guises as DeLany (1998) and DeLany et al. 2004 & 2006). Additional papers were rejected if they presented only "mixed-gender" (i.e., did not distinguish between females and males) data, were a review of other studies, or provided EE data only on a per-kg basis. A number of papers by Eliakim et al. (1996, 1997, and 2001a, b) were rejected because they were intervention studies with no baseline measurements.

The above procedure was supplemented by a *de novo* Google Scholar search plus review of selected references cited in the 736 papers that were reviewed in detail. Frankly, I lost track of what DTEE papers were reviewed and rejected or accepted after that, but Tables 12-21 contain over 220 unique citations providing over300 lines of DTEE data (and its components, where appropriate). These studies provided data used in this report. The data in Table 12 all come from DLW studies of US citizens unless otherwise noted. Almost 100% of the studies listed in Tables 12-21 are cross-sectional in nature. Very few longitudinal studies of DTEE involving the same set of subjects exist, and many studies purported as being longitudinal do not provide tabular data for all of the years (e.g., Spandano et al. 2005). Essentially they are treated as sequentially cross-sectional samples in this Report.

Table 12. Estimates of DTEE & PAEE seen in the literature

Ages				DTEE (kcal/day)			PAEE (kcal/d)				
Mean	**SD**	**Type**	**(n)**	**Mean**	**SD**	**COV**	**Mean**	**SD**	**COV**	**Citation**	**Comment**
Females: Normal, Healthy, or Not-Specified											
a. Mean & SD statistics are provided for Age											
5.5	0.9	N	35	1410	263	18.7	382	239	62.6	Nguyen et al. 1996	
5.5	0.4	H	13	1347	184	13.7				Fontvielle et al. 1993	From individual data
6.4	1.0	N	12	1536	363	23.6				Johnson et al. 1996	C
6.5	2.3	H	11	1453	534	36.8				Motil et al. 1998	Controls
7.6	1.7	N	25	1648	475	28.8	485	160	33.0	Nagy et al. 1997	AA: Tanner 1
7.9	1.2	N	9	1614	401	24.8	479	164	34.2	Nagy et al. 1997	C: Tanner 2
8.1	1.0	H	11	1934	201	10.4	641	213	33.2	Dugas et al. 2008	EA
8.1	1.7	N	24	1715	428	25.0	346	254	73.4	Johnson et al. 2000	Fairly fat group; AA
8.1	1.4	N	55	1566	399	25.5	238	312	131.1	Johnson et al. 2000	Fairly fat group; C
8.2	1.0	N	12	1574	218	13.9	372	243	65.3	Treuth et al. 1998	
8.3	1.2	H	10	1640	222	13.5	351	146	41.6	Dugas et al. 2008	MA
9.7	0.8	N	123	1846	247	13.4				Bandini et al. 2002	Pre-pubertal
10.1	1.0	N	45	2002	335	16.7	722	239	33.1	Craig et al. 1996	Premenarchal
10.2	1.4	N	13	2123	206	9.7	693	112	16.2	Roemmich et al. 2000	Pre-pubertal
10.6	0.4	H	25	2314	351	15.2	860	239	27.8	DeLany et al. 2006	C
10.7	0.7	H	28	2182	246	11.3	741	167	22.5	DeLany et al. 2006	AA
10.7	0.9	N	73	2098	257	12.2				Bandini et al. 2002	Pubertal
12.3	1.0	H	13	2429	327	13.5	773	313	40.5	Calabro et al. 2013	Ages 11-14
12.6	0.7	M	53	2196	399	18.2	559	296	53.0	DeLany et al. 2004	AA & C
12.7	2.3	N	27	2304	387	16.8				Perks et al. 2000	
12.8	1.9	N	18	2237	263	11.8	654	148	22.6	Roemmich et al. 2000	Pubertal
13.2	1.8	H	9	2321	281	12.1				Wong 1994	Caucasian
14.3	1.0	N	14	2385	446	18.7				Bandini et al. 1990	
18.4	0.6	N	91	2448	351	14.3				Stice et al. 2011	PAI data not reported
22.1	4.3	H	32	2596	421	16.2				Hise et al. 2002	
24.1	3.5	N	10	2224	386	17.4	731	367	50.2	Beidleman et al. 1995	
24.8	6.9	N	6	1985	351	17.7	590	293	49.7	Casper et al. 1991	
25.2	3.5	H	10	2368	124	5.2				Sawaya et al. 1995	9-day study
25.8	5.8	N	13	2371	397	16.7				Leenders et al. 2006	13 accel. equations
28.0	5.7	N	33	2409	574	23.8	747	408	54.6	Johannsen et al. 2008a	
31.0	6.0	N	9	1993	427	21.4	610	347	56.9	Hibbert et al. 1994	PAI range: 1.34-2.15
31.3	5.0	LM	9	2414	237	9.8				Lovelady et al. 1993	PAI range: 1.51-2.09
31.7	4.8	N	27	2221	368	16.6	702	354	50.4	Weinsier et al. 2002	Group 1: Maintainers

Table 12. Estimates of DTEE & PAEE seen in the literature (continued)

Ages				DTEE (kcal/day)			PAEE (kcal/d)				
Mean	SD	Type	(n)	Mean	SD	COV	Mean	SD	COV	Citation	Comment
31.8	5.5	N	20	2017	237	11.8				Walsh et al. 2004	White
31.9	4.7	N	14	1992	340	17.1				Walsh et al. 2004	Black
32.6	13.1	NS	16	1765	625	35.4	1010	400	39.6	Luke et al. 2005	
33.0	6.0	N	12	2261	208	9.2				Welle et al. 1992	Control group
34.0	6.1	H	83	2008	333	16.6	524	282	53.8	Hunter et al. 2002	Premen.; ages 23-47 y
34.0	6.3	N	14	2259	192	8.5				Amatruda et al. 1993	Ages 21-45
37.6	5.7	N	20	1959	303	15.5	491	291	59.3	Weinsier et al. 2002	Group2: Gainers
38.0	8.0	H	15	2199	215	9.8				Schoeller et al. 1997	Moderately active
39.6	5.9	H	10	2519	418	16.6	820	411	50.1	Johannsen et al. 2008b	
48.0	14.0	H	20	3883	1732	44.6				Roubenoff et al. 2002	Control group
49.1	6.8	N	80	2118	404	19.1				Herbert et al. 2002	Ages 40-65
49.7	7.3	N	136	2306	455	19.7	705	323	45.8	Masse et al. 2004	PAI range: 1.2-2.5
59.4	3.5	N	34	2141	363	17.0	684	280	40.9	Bathalon et al. 2001	Restrain. Eaters
60.0	4.0	H	33	2156	329	15.3				Hays et al. 2002	PAI range: 1.22-2.29
60.3	3.1	N	26	2268	280	12.3	805	232	28.8	Bathalon et al. 2001	Unrest. Eaters
60.8	3.1	H	29	2229	325	14.6				Vinken et al. 1999	Ages: 55-65
62.1	11.9	H	27	2282	167	7.3				Seale 2002	Age range: 41-80
64.0	5.0	H	6	2092	231	11.0	410	251	61.2	Goran & Poehlman 1992	PAI range: 1.25-1.82
64.0	7.0	NS	37	2090	411	19.7	207	211	101.9	Starling et al. 1998a	Ages: 52-79; AA
65.0	8.0	H	37	1987	396	19.9	397	290	73.0	Carpenter et al. 1998	AA
66.0	8.0	H	96	2115	360	17.0	600	260	43.3	Brochu et al. 1999	Ages: 50-88
67.0	4.0	H	13	1447	162	11.2	682	325	47.7	Treuth et al. 1996	
67.0	6.0	H	52	1946	371	19.1	469	305	65.0	Carpenter et al. 1998	C
67.6	4.1	NS	10	2065	NS					Roberts 1996	Meta-analysis
68.0	6.6	NS	43	1997	403	20.2				Tomoyasu et al. 1999	White
69.0	5.4	N	29	2233	404	18.1	711	275	38.7	Johannsen et al. 2008a	
70.0	3.9	N	15	2293	682	29.7	767	558	72.8	Frisard et al. 2007	
71.5	4.8	N	21	2213	429	19.4	547	360	65.8	Ades et al. 2005	
73.0	3.0	H	13	2103	837	39.8				Rutgers et al. 1997	
73.5	4.2	H	13	2256	215	9.5				Seale et al. 2002	Rural residents
74.0	2.0	NS	10	1852	214	11.6				Roberts 1996	Meta-analysis
74.0	4.4	H	10	1814	212	11.7				Sawaya et al. 1995	9-day study
74.0	4.4	H	10	1813	215	11.9				Vinken et al. 1999	Ages: 68-80
74.1	3.2	NS	67	1904	369	19.4	620	272	43.9	Blanc et al. 2004	AA
74.5	2.8	N	40	1892	271	14.3	568	181	31.9	Cooper et al. 2013	
74.6	3.1	N	40	1839	175	9.5	436	386	88.5	Manini et al. 2009	Ages: 71-79
74.8	2.8	NS	77	1885	286	15.2	584	197	33.7	Blanc et al. 2004	AA

Table 12. Estimates of DTEE & PAEE seen in the literature (continued)

Ages				DTEE (kcal/day)			PAEE (kcal/d)				
Mean	**SD**	**Type**	**(n)**	**Mean**	**SD**	**COV**	**Mean**	**SD**	**COV**	**Citation**	**Comment**
82.0	2.8	N	40	1814	337	18.6	540	277	51.3	Cooper et al. 2013	
92.0	2.0	N	49	1626	222	13.7	436	180	41.3	Johannsen et al. 2008a	
93.0	3.3	N	11	1608	206	12.8	381	179	47.0	Frisard et al. 2007	
b. Complete age statistics are not provided											
5-10		H	19	1779	257	14.4	373	248	66.5	Trowbridge et al. 1997	AA
5-10		H	14	1780	273	15.3	403	262	65.0	Trowbridge et al. 1997	C
8 - 9		H	27	1710	281	16.4	463	213	46.0	Treuth et al. 2003a	2 lean parents
8 - 9		H	38	1738	290	16.7	483	265	54.9	Treuth et al. 2003a	1 lean/1 obese parent
8 - 9		H	23	1790	297	16.6	511	230	45.0	Treuth et al. 2003a	2 obese parents
8-12		H	196	1940	161	8.3	511	133	26.0	Bandini et al. 2004	Premenarchal
8-12		NS	90	1851	213	11.5				Bandini et al. 2013	Relatively low active
33.4		NS	10	2315	285	12.3				Champagne et al. 2002	Non-dietitians; 26-41
36.4		NS	10	2154	332	15.4				Champagne et al. 2002	Dietitians; ages 28-45
30-69		NS	180	2190	406	18.5				Tooze et al. 2013	
49-79		NS	21	2357	807	34.2				Mahabir et al. 2006	Postmenopausal
60-69		N	48	2042	343	16.8				Roberts & Dallal 2005	
70-79		NS	14	1888	295	15.6				Roberts & Dallal 2005	
80-89		NS	6	1382	152	11.0				Roberts & Dallal 2005	
90-97		NS	9	1356	166	12.2				Roberts & Dallal 2005	
Females: Active, Fit, or Athlete											
a. Mean & SD are provided for Age											
20.0	2.0	Fit	20	4732	191	4.0				Castellani et al. 2006	Winter military act.
21.5	1.9	Ath	10	2937	709	24.1	1355	647	47.7	Beidleman et al. 1995	
23.4	4.7	At.	5	5593	2510	44.9				Trappe et al. 1997	Olympic trials training
25.0	1.3	Fit	9	3541	718	20.3	1754	625	35.6	Ruby et al. 2002	Wildfire firefighters
26.0	3.3	Ath	9	2826	312	11.0				L.O. Shulz et al. 1992	Elite distance runners
40.0	7.0	Act	9	2462	167	6.8				Schoeller et al. 1997	
74.2	2.7	Act	39	2106	263	12.5	805	206	25.6	Manini et al. 2009	Ages: 70-79
b. Complete age statistics are not provided											
8-12		Act	71	2097	249	11.9				Bandini et al. 2013	Relatively active
19-22		Ath	9	2038	298	14.6				Edwards et al. 1993	Cross-country runners

Table 12. Estimates of DTEE & PAEE seen in the literature (continued)

Ages				DTEE (kcal/day)			PAEE (kcal/d)				
Mean	SD	Type	(n)	Mean	SD	COV	Mean	SD	COV	Citation	Comment
Females: Sedentary, Overweight, or Obese a. Mean & SD statistics are provided for Age											
8.5	2.0	OW	14	1554	319	20.5	313	275	87.9	Johnson et al. 1998	Mohawk & Caucasian
8.7	0.7	OW	12	2009	316	15.7	525	193	36.8	Treuth et al. 1998	
10.2	0.7	OW	27	2156	338	15.7				Champagne et al. 1998	AA
10.2	0.7	OW	31	2308	373	16.2				Champagne et al. 1998	C
10.5	0.3	OE	51				2602	655	25.2	Bunt et al. 2003	Pima Indians
13.4	0.8	OW	20	2835	336	11.9				R.Singh et al. 2009	
15.2	1.8	O	16	3282	558	17.0				Bandini et al. 1990	
29.0	4.0	O	5	2963	135	4.6	1212	570	47.0	Hibbert et al. 1994	
31.3	13.0	O	9	2789	440	15.8				L.O. Schulz et al. 1994	Pima Indians
32.0	10.0	OW	28	2684	309	11.5				Tataranni et al. 2003	Pima Indians
34.6	10.6	M	172	2428	433	17.8	793	272	34.3	Ebersole et al. 2008	2/3 were OW or O
35.2	7.4	Sed	17	2177	267	12.3				Hunter et al. 2000	Premenopausal C
35.6	6.9	Sed	18	1969	342	17.4				Hunter et al. 2000	Premenopausal AA
36.0	5.8	OW	20	2118	343	16.2				Walsh et al. 2004	Black
36.0	7.0	OW	26	2677	428	16.0				Welle et al. 1992	
36.5	6.1	OW	21	2234	396	17.7				Walsh et al. 2004	White
38.0	5.0	Sed	8	1960	191	9.7				Schoeller et al. 1997	
38.5	6.1	O	10	2593	319	12.3	673	304	45.2	Johannsen et al. 2008b	
38.6	8.1	OW	30	2559	295	11.5				Roberts et al. 2012	CALERIE Study
38.7	6.0	O	15	2703	339	12.5	1028	251	24.4	Kushner et al. 1995	C
39.5	5.2	O	18	2704	449	16.6				Amatruda et al. 1993	Ages 31-51
39.8	5.0	O	14	2452	361	14.7	834	253	30.3	Kushner et al. 1995	AA
40.8	4.5	O	13	2616	422	16.1				Racette et al. 1995	
43.8	9.2	OW	35	2353	611	26.0	1333	498	37.4	Staten et al. 2001	(Question the PAI)
48.0	10.0	OW	47	2462	454	18.4	980	335	34.2	Paul et al. 2004	
57.5	4.2	O	15	2639	378	14.3	761	359	47.2	Rawson et al. 2002	Trp64Arg Non-Carriers
57.8	6.6	O	19	2752	513	18.6	864	360	41.7	Rawson et al. 2002	Trp64Arg Carriers
61.2	15.3	OW	27	3071	361	11.8				Seale 2002	Ages: 32-82
62.1	11.9	OW	27	2282	167	7.3				Seale 2002	
64.0	8.0	OW	37	2090	411	19.7	493	297	60.2	Starling et al. 1998b	AA
65.0	3.5	O	25	1999	385	19.3	435	310	71.3	Nicklas et al. 1997	AA
73.5	4.2	OW	13	2256	215	9.5				Seale 2002b	
75.5	2.8	OW	72	1930	395	20.5	605	302	49.9	Manini et al. 2009	AA
75.5	2.8	OW	80	1891	296	15.7	549	192	35.0	Manini et al. 2009	C

Table 12. Estimates of DTEE & PAEE seen in the literature (continued)

Ages				DTEE (kcal/day)			PAEE (kcal/d)				
Mean	SD	Type	(n)	Mean	SD	COV	Mean	SD	COV	Citation	Comment
b. Complete age statistics are not provided											
40-69		OW	206	2308	474	20.5	750	799	106.5	Tooze et al. 2007	62% was OW or O
49-79		OW	25	2665	631	23.7				Mahabir et al. 2006	Post-menopausal
49-79		OW	19	2730	1185	43.4				Mahabir et al. 2006	Post-menopausal
60-69		OW	46	2061	294	14.3				Roberts & Dallal 2005	
70-79		OW	19	1868	402	21.5				Roberts & Dallal 2005	
80-89		OW	6	1748	464	26.5				Roberts & Dallal 2005	
90-97		OW	7	1766	292	16.5				Roberts & Dallal 2005	
Females: Health & Other Issues											
6.2	2.1	RS	14	845	251	29.7				Motil et al. 1998	Lack of use of hand
24.5	6.9	ANP	6	1972	644	32.7	888	468	52.7	Casper et al. 1991	Amenorrheic
39.9	11.9	CP	12	1986	363	18.3	340	601	176.8	RK Johnson et al. 1997	
47.0	14.0	RA	20	2849	1075	37.7				Roubenoff et al. 2002	Stable, with drugs
72.9	6.1	CHD	21	2207	402	18.2	498	314	63.1	Ades et al. 2005	
Males: Normal, Healthy, or Not-Specified **a. Mean & SD statistics are provided for Age**											
5.2	0.7	N	36	1554	335	21.6	454	287	63.2	Nguyen et al. 1996	
5.4	0.3	H	15	1416	252	17.8				Fontvielle et al. 1993	From individual data
5.5	0.7	N	12	1678	603	35.9				Johnson et al. 1996	C
7.4	1.6	N	22	1698	479	28.2	432	215	49.8	Nagy et al. 1997	AA: Tanner 1
7.6	1.0	H	10	1804	215	11.9	523	186	35.6	Dugas et al. 2008	MA
7.6	1.5	N	19	1799	480	26.7	355	324	91.3	Johnson et al. 2006	Fairly fat group; AA
8.0	1.0	H	16	1893	359	19.0	588	239	40.6	Dugas et al. 2008	EA
8.3	1.6	N	20	1660	349	21.0	429	173	40.3	Nagy et al. 1997	C: Tanner 1
8.7	1.8	N	17	1783	377	21.1	307	195	63.5	Johnson et al. 2006	Fairly fat group; C
10.9	0.6	H	29	2576	330	12.8	932	239	25.6	DeLany et al. 2006	C
10.9	0.7	H	31	2572	382	14.9	1004	287	28.6	DeLany et al. 2006	AA
10.9	1.0	N	14	2174	236	10.9	712	191	26.8	Roemmich et al. 2000	Pre-puberal
12.5	1.6	N	23	2412	476	19.7				Perks et al. 2000	
12.8	0.8	M	61	2651	392	14.8	758	299	39.4	DeLany et al. 2004	AA & C
12.9	2.1	H	15	2710	658	24.3	937	272	29.0	Calabro et al. 2013	Ages 10-16
13.4	1.2	n	14	2555	251	9.8	673	210	31.2	Roemmich et al. 2000	Pubertal
14.5	1.5	N	14	3109	506	16.3				Bandini et al. 1990	
22.3	1.9	N	14	3494	182	5.2				Roberts et al. 1991	Sed. Occup + active

Table 12. Estimates of DTEE & PAEE seen in the literature (continued)

Ages				DTEE (kcal/day)			PAEE (kcal/d)				
Mean	SD	Type	(n)	Mean	SD	COV	Mean	SD	COV	Citation	Comment
22.7	2.5	H	17	3461	641	18.5				Roberts et al. 1995	Same as above?
22.7	3.8	H	22	3379	1353	40.0				Hise et al. 2002	
23.1	2.4	N	24	3356	635	18.9				Vinken et al. 1999	Age range: 18-28
27.0	4.4	N	20	3476	880	25.3	1265	679	53.7	Johannsen et al. 2008a	
35.9	13.4	NS	16	3245	565	17.4	1300	370	28.5	Luke et al. 2005	
41.2	9.8	NS	24	3172	410	12.9				Conway et al. 2002	
42.0	16.0	H	30	2892	548	18.9	788	414	52.5	Rising et al. 1994	Pima Ind.; some O
61.2	15.3	H	27	3071	351	11.4				Seale 2002	Age range: 32-82
64.0	7.0	H	28	2642	537	20.3	746	438	58.7	Carpenter et al. 1998	AA
64.0	8.0	NS	28	2772	556	20.1	410	320	78.0	Starling et al. 1998a	Ages: 52-79
67.0	8.0	H	84	2755	511	18.5	860	355	41.3	Brochu et al. 1999	Ages: 45-90
67.8	6.1	H	20	2580	566	21.9				Vinken et al. 1999	Ages: 60-81
68.0	6.4	H	18	2691	547	20.3				Roberts et al. 1995	
68.0	6.0	H	7	2675	394	14.7	692	402	58.1	Goran & Poehlman 1992	PAI range: 1.25-2.11
69.0	5.4	N	29	2970	458	15.4	1057	307	29.0	Johannsen et al. 2008a	
69.0	7.0	NS	15	2495	352	14.1				Roberts 1995	Meta-analysis
70.0	6.9	H	9	2349	300	12.8				Roberts et al. 1996	Same as above?
71.0	4.1	H	17	2852	462	16.2	940	349	37.1	Frisard et al. 2007	
70.0	6.2	NS	39	2701	528	19.5				Tomoyasu et al. 1999	White
70.0	7.0	H	47	2584	506	19.6	743	375	50.5	Carpenter et al. 1998	
71.0	5.0	NS	16	2412		0.0				Roberts 1996	
74.4	4.1	H	14	2971	390	13.1				Seale et al. 2002	Rural residents
74.7	3.2	N	47	2482	476	19.2	832	308	37.0	Cooper et al. 2013	
74.8	2.9	NS	72	2324	436	18.8	865	284	32.8	Blanc et al. 2004	W; ages: 70-79
75.1	3.1	N	43	2395	214	8.9	737	544	73.8	Manini et al. 2009	
75.1	3.2	NS	72	2521	396	15.7	775	313	40.4	Blanc et al. 2004	Ages: 70-79
82.0	3.0	NS	23	1657	209	12.6				Fuller et al. 1996	Ages: 76-88
82.2	3.3	N	47	2208	376	17.0	666	243	36.5	Cooper et al. 2013	
92.0	2.0	N	46	2002	326	16.3	539	227	42.1	Johannsen et al. 2008a	
93.0	3.3	N	11	2052	265	12.9	551	196	35.6	Frisard et al. 2007	
b. Complete age statistics are not provided											
5.0		N	41							Salbe et al. 1997	Pima Indians
5-10		H	12	1871	260	13.9	387	249	64.3	Trowbridge et al. 1997	C
5-10		H	17	1837	260	14.2	347	251	72.3	Trowbridge et al. 1997	AA
30-69		NS	189	2877	498	17.3				Tooze et al. 2013	
60-69		NS	14	2397	437	18.2				Roberts & Dallal 2005	

Table 12. Estimates of DTEE & PAEE seen in the literature (continued)

Ages				DTEE (kcal/day)			PAEE (kcal/d)				
Mean	SD	Type	(n)	Mean	SD	COV	Mean	SD	COV	Citation	Comment
70-79		NS	30	2407	374	15.5				Roberts & Dallal 2005	
80-89		NS	4	1700	239	14.1				Roberts & Dallal 2005	
90-97		NS	6	1935	156	8.1				Roberts & Dallal 2005	
Males: Active, Fit, or Athlete											
a. Mean & SD are provided for Age											
20.0	2.0	Fit	30	6142	191	3.1				Castellani et al. 2006	Winter military act.
21.0	2.9	Act	13	3031	627	20.7				Haggerty et al. 1997	Construction workers
22.5	3.3	Fit	4	4750	531	11.2				Forbes-Ewan et al. 1989	Military training
22.6	3.5	Fit	19	4116	719	17.5				Tharion et al. 2004	Military training
24.5	1.8	Fit	7	4878	716	14.7	2628	714	27.2	Ruby et al. 2002	Wildfire firefighters
25.0	3.0	Fit	10	5378	678	12.6				Hoyt et al. 2001	Cold military training
25.0	5.0	Fit	7	3480	220	6.3				DeLany et al. 1989	Military training
27.1	4.2	Fit	27	3477	816	23.5				Hoyt et al. 1991	High-alt. military train.
27.9	7.3	Act	10	4716	435	9.2	2422	375	15.5	Heil 2002	Woodland firefighters
28.0	5.0	Fit	7	3220	280	8.7				DeLany et al. 1989	Military training
31.0	4.0	Fit	6	4558	566	12.4				Hoyt et al. 1994	High-alt. mitary train.
45.5	4.8	Fit	13	2964	676	22.8				Lane et al. 1997	Astro.; ground-study
45.6	4.8	Fit	13	2796	452	16.2				Lane et al. 1997	Astro.space-study
74.5	3.3	Act	43	2788	293	10.5	1079	183	17.0	Manini et al. 2009	Ages: 70-79
b. Complete age statistics are not provided											
19-20		Fit	18	4281	721	16.8				Burstein et al. 1996	Winter military training
19-20		Fit	12	3937	551	14.0				Burstein et al. 1996	Summer military training
Males: Sedentary, Overweight, or Obese											
a. Mean & SD statistics are provided for Age											
8.2	1.9	OW	17	1922	501	26.1	598	353	59.0	RK Johnson et al. 1998	Mohawk & Caucasian
10.3	0.7	OW	29	2537	339	13.4				Champagne et al. 1998	AA
10.3	0.7	OW	31	2574	367	14.3				Champagne et al. 1998	C
13.7	0.7	OW	14	3332	312	9.4				R. Singh et al. 2009	
14.4	1.9	O	18	3612	643	17.8				Bandini et al. 1990	
35.4	13.8	O	12	3172	707	22.3				Paul et al. 2004	Pima Indians
37.0	13.0	OW	64	2985	481	16.1				Tataranni et al. 2003	Pima Indians

Table 12. Estimates of DTEE & PAEE seen in the literature (continued)

Ages				DTEE (kcal/day)			PAEE (kcal/d)				
Mean	SD	Type	(n)	Mean	SD	COV	Mean	SD	COV	Citation	Comment
47.0	11.0	OW	44	3035	335	11.0	1171	311	26.6	Paul et al. 2004	
64.0	7.0	OW	28	2772	556	20.1	865	451	52.1	Starling et al. 1998	AA
66.0	4.6	OW	21	2679	591	22.1	817	472	57.8	Nicklas et al. 1997	AA
75.2	2.9	OW	74	2327	431	18.5	733	302	41.2	Manini et al. 2009	AA
75.5	3.1	OW	76	2511	390	15.5	804	273	34.0	Manini et al. 2009	
b. Complete age statistics are not provided											
40-69		OW	244	2899	469	16.2	893	328	36.7	Tooze et al. 2007	75% were OW or O
60-69		OW	30	2851	420	14.7				Roberts & Dallal 2005	
70-79		OW	34	2624	461	17.6				Roberts & Dallal 2005	
80-89		OW	6	2294	357	15.6				Roberts & Dallal 2005	
90-97		OW	2	1863	46	2.5				Roberts & Dallal 2005	
Males: Health & Other Issues **a. Mean & SD statistics are provided for Age**											
35.1	11.5	CP	18	2455	622	25.3	307	453	147.6	RK Johnson et al. 1997	
62.0	8.0	Park	16	2214	460	20.8	339	366	108.0	Toth et al. 1997a	
72.9	7.9	Park	20	2237	510	22.8	521	310	59.5	Delikanaki et al. 2009	

Notes & Abbreviations:	
*	Calculated as: mean DTEE/mean REE.
AA	African-American
Accel.	Accelerometers
Act.	Activite
AI	American Indian
ANP	Anorexia Nervosa Patients
Astro.	Astronauts
Ath.	Athlete
C	Caucasian
CHD	Chronic Heart Disease
COV	Coefficient of Variation (SD/Mean*100)
CP	Cerebral palsy
DTEE	Daily Total Energy Expenditure (kcal/day)
EA	European-American
H	Healthy
LM	Lactating Mothers

Notes & Abbreviations:	
M	Mixed lean and obese subjects
MA	Mexican-American
(n)	Sample Size
N	Normal
NS	Not Specified
PAEE	Physical Activity Energy Expenditure
PAI	Physical Activity Index (DTEE/REE; also known as PAL: Physical Activity Level)
Park	Parkinson Disease patient
RA	Rheumatoid Arthritis
REE	Resting Energy Expenditure
RH	Rett Syndrome (a neurodevelopmental disorder)
O	Obese
OW	Overweight
SD	Standard Deviation
Sed	Sedentary

A number of studies using fairly narrowly-defined cohorts and/or uncommon physical problems were not included in the tabular data. Most of these involved in-patient or hospital-based research studies, but not all. The excluded studies comprised infants with (1) ventricular septal defects (Ackerman et al., 1988); (2) cyanotic congenital heart disease (Leitch et al., 1998); (3) pre-symptomatic cystic fibrosis (Bronstein, 1995); and pulmonary insufficiency (Denne, 2001). Finally, studies involving preterm babies still in the hospital were also excluded; these studies were Jensen et al. (1992) and Leitch et al. (1999). In general, disease-related weight loss reflects a chronic whole-body energy imbalance that is difficult to model successfully (Toth, 1999), and has a low probability of ever being of interest to EPA's exposure modelers.

Even though DLW studies are treated as being the "gold standard," in a comparison of DLW estimates of DTEE in a 7-day whole-body respiratory chamber study of 12 young male adults, it was found that the DLW estimates on average were -2.5% ± 5.8% lower than the VO_2 chamber studies. See information relating $METS_{MAX}$, HRR, and $VO_{2.MAX}$ chamber estimates contained in Ravussin et al. (1991). The range of the differences was -14% to +4%, with the larger underestimates being observed in heavier, fatter subjects (Ravussin et al., 1991).

The units of DTEE are usually presented as kcal/d or MJ/d. If MJ/d units are provided, they are converted to kcal/d units by multiplying them by 239. Rarely—for some odd reason—are *only* kcal/kg data presented, even though doing so probably would reduce age/gender variability in energy expenditure estimates. Thus, the data in Tables 11-19 are presented as kcal/d and are not BM-normalized. Extreme DTEE measures are seen in polar expeditions and in strenuous military training, where values as high as 7,500 kcal/d were recorded, although 3,300-4,000 kcal/d was a more common high range (Burstein et al., 1996; Hoyt et al., 1991). Usually participants in artic expeditions lose significant weight at even these high DTEE rates (Stroud et al., 1993). Many studies of male-only military training exercises report DTEE in the 4,000-5,000 kcal/d range (Tharion et al., 2005). A few of these "special studies" are included in Table 12 for contrast. Hoyt et al. (2001) and Tharion et al. (2005) contain good reviews of military training studies in the U.S. and other countries that use DLW to estimate DTEE.

There have been a number of multi-study analyses of DLW-measured DTEE. These include purely descriptive and meta-analytic studies. Cambridge University in England has compiled a descriptive analysis of 74 studies in "affluent societies" that used DLW to estimate DTEE (Black 1996, 2000; Black et al. 1996). The studies produced 574 group means/SD's estimates for both genders and ages between 2 y to 90 y olds. Fifty of the studies are of non-US citizens and are not included in Table 12. Also not included in the Table were 24 U.S. studies that provided incomplete information, were in units that could not be converted to kcal/d estimates, or used a non-standard protocol. The Black et al. papers cited above are the most synoptic review of DLW studies that I know of. Summary data from all of the studies appears here as Table 13. Besides DTEE data, Black and colleagues present REE and PAI data for the same cohorts included in their review. Their work is not a meta-analysis of EE data *per se*, but could be used as an input to one.

Table 13. Group mean estimates of DTEE, REE, & PAI from BLACK (2000)

Age Range	DTEE (kcal/day)			REE (kcal/day)			PAI			
	Mean	SD	COV (%)	Mean	SD	COV (%)	Mean	SD	COV (%)	n
Females										
1 - 6	1315	215	16.3	860	167	19.4	1.57	0.30	19.1	21
7 - 12	1912	430	22.5	1147	239	20.8	1.68	0.16	9.5	24
13 - 17	2725	621	22.8	1601	359	22.4	1.73	0.24	13.9	26
18 - 29	2486	526	21.2	1482	263	17.7	1.70	0.28	16.5	89
30 - 39	2390	406	17.0	1434	143	10.0	1.68	0.25	14.9	76
40 - 64	2342	406	17.3	1386	167	12.0	1.69	0.23	13.6	47
60 - 74	2055	382	18.6	1267	167	13.2	1.62	0.28	17.3	24
≥ 75	1458	263	18.0	980	143	14.6	1.48	0.23	15.5	12
Males										
1 - 6	1458	239	16.4	908	191	21.0	1.64	0.39	23.8	29
7 - 12	2342	382	16.3	1362	239	17.5	1.74	0.22	12.6	32
13 - 17	3370	645	19.1	1936	359	18.5	1.75	0.19	10.9	31
18 - 29	3298	717	21.7	1793	286	16.0	1.85	0.33	17.8	56
30 - 39	3418	741	21.7	1960	430	21.9	1.77	0.31	17.5	36

Table 13. Group mean estimates of DTEE, REE, & PAI from BLACK (2000) (continued)

Age Range	DTEE (kcal/day) Mean	SD	COV (%)	REE (kcal/day) Mean	SD	COV (%)	PAI Mean	SD	COV (%)	n
40 - 64	2749	406	14.8	1673	191	11.4	1.64	0.17	10.4	15
60 - 74	2629	382	14.5	1649	215	13.0	1.61	0.28	17.4	22
≥ 75	2199	311	14.1	1434	167	11.6	1.54	0.24	15.6	34

Notes & Abbreviations:	
COV:	Coefficient of Variation (SD/Mean)
DTEE:	Daily Total Energy Expenditure (kcal/d)
n:	Sample Size
PAI:	Physical Activity Index (DTEE/REE)
REE:	Resting Energy Expenditure (kcal/d)
SD:	Standard Deviation

Sources:

Black, A.E. (2000). "Critical evaluation of energy intake using the Goldberg cut-off for energy intake.

Inter. J. Obesity 24: 1119-1130. Black, A.E. (1996). "Physical activity levels from a meta-analysis of doubly water studies for validating energy intake as measured by dietary assessment." *Nutr. Rev.* 54: 170-174.

Black, A.E., Coward, W.A., et al. (1996). "Human energy expenditure in affluent societies: an analysis of 574 doubly-labeled water measurements." *Euro. J. Clin. Nutr.* 50: 72-82.

The same is true for Brooks et al. (2004), which is a condensation of a NIH study by the (US) National Academy of Sciences' Institute of Medicine (IOM). The IOM looked at *approximately* 80 studies (the exact number is not provided in Brooks et al., 2004) of people with a "healthy" BMI, which is defined to be between 18.5 and 25.0 kg/m^2 for adults and <85th percentile for youth 3-18 y old. Their data—mean estimates of DTEE, REE, and PA--are reproduced here as Table 14. REE for youth 3-18 was estimated by regression equations using weight and height as independent variables. PAI estimates were derived by dividing group mean DTEE by group mean REE. The mean DTEE and REE values presented in Table 14 generally are higher than those seen in Table 13, but the mean PAI values are lower. Most of the differences among any of the measures, however, are <10% or so for all of the age/gender groups used.

A true meta-analysis of DTEE/PAI data is described in Dugas et al. (2011). They included 98 studies from both developed and under-developed countries. The studies reported data for 183 cohorts including almost 5,000 individuals.

DTEE, not surprisingly, is inversely related to age and positively related to BM in both genders; there was no association of DTEE (and PAI) with development status of the country where the subjects resided (Dugas et al., 2011).

An informative and visually interesting discussion of DTEE as people age appears in Manini (2010). Basically both REE and DTEE decrease over time, but the decrease in DTEE is due more to a decrease in physical activity (PA) rather than to a reduction in organ sizes or tissue metabolic rates (Manini, 2010; Manini et al., 2009). There does not seem to be a difference in DTEE (or PAI) between pre- and post-menopausal status in older women, adjusted for age (Tooze et al., 2007).

The Food and Nutrition Board (2005) of the Institute of Medicine, part of the U.S. National Academies of Science, reviewed a number of DTEE studies using DLW. None of its data appear in this report.

Components of DTEE

DTEE for weight-stable persons is decomposed into a limited number of components because they cannot readily be separated. Basal, or resting, metabolism always is identified in any typology of DTEE and it is by far the largest component of DTEE in most individuals (Goran & Treuth, 2001). REE accounts for 60-80% of DTEE in sedentary people, 55-70% in "normal" individuals, and 45-60% in active people (McCurdy, 2000). REE is the minimal metabolic activity needed to maintain bodily functions and temperature at rest. This includes circulation, respiration, transport and movement of liquids, cellular activity, maintenance of electrolyte gradients, and central nervous functioning (McCurdy, 2000). There are numerous ways that REE is measured (or predicted from a variety of anthropogenic observations), and each one has multiple protocol variations. This report is not the place to expand upon that complicated subject; REE data that underlies PAI data in this report all come from direct (chamber) or indirect calorimetry measures unless otherwise noted. For narrowly defined cohorts, variability within the cohort (between-person variability [COV_B]) for REE is about 3.0-6.0%, about the same as within-person variability (COV_W) of approximately 4% for individuals comprising a narrow age/gender cohort (Black, 2000). However, these estimates seem to be low according to data provided by Black & Cole (2000) for specific studies—with an implied wider grouping of subjects—that states that the mean within-person variance over multiple days is 11.8% as compared with the mean

Table 14 Group mean estimates of DTEE, REE, & PAI from BROOKS ET AL. (2004)

Age Range (y)	DTEE (kcal/d) Mean	REE (kcal/d) Mean	PAI Mean	Sample Size (n)
		Females		
3 - 8	1487	1004	1.48	227
9 - 13	1907	1186	1.60	89
14 - 18	2302	1361	1.69	42
19 - 30	2436	1361	1.80	82
31 - 50	2404	1322	1.83	61
51 - 70	2066	1226	1.70	71
> 70	1564	1183	1.33	24
		Males		
3 - 8	1441	1035	1.39	129
9 - 13	2079	1320	1.56	28
14 - 18	3116	1729	1.80	10
19 - 30	3081	1769	1.74	48
31 - 50	3021	1675	1.81	59
51 - 70	2469	1524	1.63	24
>70	2238	1480	1.52	39

Notes & Abbreviations:	
DTEE:	Daily Total Energy Expenditure (kcal/d)
n;	Sample size
PAI:	Physical Activity Index (DTEE/REE)
REE:	Resting Energy Expenditure (kcal/d)

Source:
Brooks, G.A., et al. (2004). "Chronicle of the Institute of Medicine physical activity recommendation: how a physical activity recommendation came to be among dietary recommendations." *Amer. J. Clin. Nutr.*79(Supp.): 921S-930S.

between-person variance of 13.0%. These are quite different within and between variances estimates reported by the same first author.

Infrequently, REE is divided into sleeping EE (SEE) and "arousal" EE, where the latter is ill-defined and often treated as a "residual" (REE-SEE) representing EE spent in waking up or going from one state to another (Manini, 2010; Ravussin & Rising, 1992). I do not see any advantage to making this distinction, and since it cannot be accurately measured, arousal EE is not used in this report. SEE also is not considered further except to note that REE in babies and infants usually is measured as the child sleeps; so SEE plays a major role in measurements of DTEE (and PAI) for that cohort.

Another component of DTEE usually identified is Dietary Induced Thermogenesis, or DIT. It also is called the thermic effect of food (TEF; Hibbert et al., 1994), but that term is not used here. DIT is energy needed to digest food and fluids, and is difficult to measure directly. Usually it simply is estimated to be 5-15% of DTEE and is subtracted from DTEE to identify non-DIT daily energy expenditure (Martin et al., 2011; Westerterp, 2004). Frequently 10% is used as the estimate of DIT, but careful monitoring studies indicate that DIT values of 7.6-8.0% are reasonable, at least for females of widely varying BMI (Hibbert et al., 1994). The impact of using of a constant—of any value—is unexplored in DTEE studies that I have seen in the literature. Thus, DIT essentially has an imprecise impact on daily DTEE estimates and their variability over time, both within an individual and in a group of individuals. You would expect DIT to vary by the caloric value of individual meals, macronutrient composition (Wilson & Morely, 2003), the type of activity taken both before and after ingesting food, the type of food consumed (particularly protein and alcohol), and personal characteristics, such as body composition, FFM, etc. (Westerterp, 2004). DIT certainly is an under-defined aspect of daily total energy expenditure.

That remainder (often calculated by DTEE - [0.10*DTEE] - REE) is known by a number of terms: physical activity energy expenditure (PAEE), non-exercise thermogenesis (NEAT; see below), shivering & twitching, retained energy (gaining weight), work EE, daily living EE, etc. (Luke et al. 2005; McCurdy, 2000). Because there is no method to estimate these components of DTEE in "free-living" individuals, generally only three components of DTEE are discussed: REE, PAEE, and DIT (treated as a constant proportion of DTEE itself as just noted). PAEE, then, is a catch-all term that is used in the exercise literature and is not focused on what most people would consider to be actual physical activity. See the Glossary of Terms for formal definitions of PA. Because of the uncertainty in what exactly

PAEE includes in any one study, the PAEE values provided in the literature should be taken as an approximation. S. Liu et al. MSSE (2012) discuss computational methods used to estimate EE for physical activities.

Physical activity energy expenditure is the most variable component of DTEE on a daily basis in most individuals, and is largely determined by genetic factors (Westerterp & Plasqui, 2004). PAEE greatly affects the overall (absolute) amount of DTEE in individuals (Westerterp, 2008), and is estimated as being from 3% to 50% of DTEE in adult males (Rising et al., 1994). This proportion is lower in elderly individuals and people with health problems: about 10-30% (Goran & Poehlman, 1992; Toth, 1999). PAEE itself is often disaggregated into activity-specific EE, such as walking, carrying things, gardening, etc. (Passmore & Durnin, 1955), but data on these cannot come from DLW studies that integrate whole-body EE over time. DLW, then, cannot describe the intensity, duration, frequency, or pattern of physical activity in a subject without some type of additional monitoring being used (Roemmich et al., 2000). Understanding EE from specific activities requires the use of methods discussed below in Section 10. The vast majority of PAEE estimates contained in tables included in this report are *calculated* and not directly measured. Almost all of these are calculated using the formula presented above, usually in the form: PAEE = (DTEE * 0.9) – REE (e.g., Tooze et al., 2007).

NEAT is a term that is used by some Mayo Clinic researchers to represent energy expended in an individual by all physical activities *other than* sleeping, eating, and volitional sports or exercise (Levine, 2003, 2004; Levine et al., 1999, 2000, 2006). Thus, EE expended in working, walking for transport, watching TV, cleaning the home, etc. is considered to be NEAT. It accounts for the most *variance* in daily physical activity (Donahoo et al., 2004), which—as mentioned—is the most variable part of DTEE. Since NEAT can only be roughly estimated using the "factorial method" (see the Glossary) to **subtract** activity-specific EE from estimated PAEE (itself estimated as (DTEE – [REE + DIT]) -- the concept causes a sense of false precision in daily energy expenditure components and is not used further in this report. In *confined* respiratory chamber studies of exercise using strict protocols, NEAT was 89-92% of PAEE for youth aged 4-19 y (Butte et al., 2007).

The above discussion assumes that subjects are weight-stable. If weight is changing, either higher or lower for whatever reason (e.g., energy intake >energy expenditure from eating too much and/or not undertaking enough physical activity or both; energy expenditure<energy intake due to dieting, exercising more, sickness; or growth and development in growing youth), it has to be accounted for in the energy balance equation (Butte, 2000). Even in babies, however, energy expended in growth—called energy deposition—decreases quickly with age as a percentage of everyday total energy expenditure. Energy deposition goes from about 22% at 3 months, either in bottle- or breast-fed babies, to <2% at 24 months (Butte et al., 2000). Thus, growth EE is a relatively short-term phenomenon. For exposure modeling purposes, we have to assume that the modeled population is weight-stable or we would have to undertake dynamic modeling of cohort weight changes. This would be quite a difficult undertaking (McCurdy, 2000). On the other hand, we probably should account for weight changes in babies <1 y old.

Butte et al. (2000) provides data on DTEE in babies and infants; it is abstracted and reproduced here as Table 15. As seen, DTEE increases meaningfully with age in both genders. There is a significant difference in DTEE for breast-fed versus bottle-fed (formula) at most ages, but data on breast-feeding is not available for our exposure modeling work and so are not discussed further. On an absolute basis, DTEE, REE, and PAEE do not differ significantly between lactating and non-lactating mothers (Butte et al., 2001), although there is a significant difference when the data are "adjusted" on a FFM basis. Since we do not use FFM in our models, we should assume that lactation has no impact on resting, activity, or total energy expenditure.

Table 15. Group mean estimates of DTEE, REE, & PAEE from BUTTE ET AL. (2000)

Age (months)	DTEE (kcal/d) Mean	REE (kcal/d) Mean	PAEE (Kcal/d) Mean	PAI Mean
		Females		
0 - 0.9	241	179	62	1.35
1.0 - 1.9	306	223	83	1.37
2.0 - 2.9	369	263	106	1.40
3.0 - 3.9	431	301	130	1.43
4.0 - 4.9	492	339	153	1.45
5.0 - 5.9	552	374	178	1.48
6.0 - 8.9	666	434	232	1.53
9.0 - 11.9	714	497	217	1.44
12.0 - 17.9	781	551	229	1.42
18.0 - 24.0	886	630	255	1.41

Table 15. Group mean estimates of DTEE, REE, & PAEE from BUTTE ET AL. (2000) (continued)

Age (months)	DTEE (kcal/d) Mean	REE (kcal/d) Mean	PAEE (Kcal/d) Mean	PAI Mean
		Males		
0 - 0.9	248	226	22	1.10
1.0 - 1.9	320	283	37	1.13
2.0 - 2.9	389	333	56	1.17
3.0 - 3.9	454	378	76	1.20
4.0 - 4.9	516	417	99	1.24
5.0 - 5.9	574	449	125	1.28
6.0 - 8.9	684	506	178	1.35
9.0 - 11.9	765	577	188	1.33
12.0 - 17.9	845	643	202	1.31
18.0 - 24.0	944	719	225	1.31

Abbreviations:

DTEE:	Daily Total Energy Expenditure
PAEE:	Physical Activity Energy Expenditure
PAI:	Physical Activity Index
REE:	Resting Energy Expenditure

Source:
Butte et al. (2000). "Energy requirements derived from total energy expenditure based on energy deposition during the first 2 y of life." *Amer. J. Clin. Nutr.* 72: 1558-1569.

Energy expenditure changes during and after pregnancy are pronounced. See Table 16 abstracted from Butte et al. (2004). DTEE increases through pregnancy—although not linearly--for all three BMI groups shown in the Table and decreases postpartum. REE follows the same trend, but the decrease in resting EE is relatively larger than the decrease in DTEE, resulting in an increase of PAI during pregnancy and a decrease after pregnancy, also for all three BMI groups.

After middle age, DTEE generally decreases--both on an absolute and relative mass basis--as people age. The decrease is on the order of 25-35% on an absolute basis (Wilson & Morley, 2003). REE does not decrease as much on a per-BM basis for cross-sectional samples; see, for example McMurray et al. (2014). The average cross-sectional decrease found in that report was 7.5% for both genders. The two trends together (DTEE and REE) should result in a decrease in PAI with aging over about 35 y old, which is seen in Tables 12-20 (but not so much in Table 19, especially for females). See Section 9 below for discussion of PAI.

Estimating DTEE: DLW and Other Methods

There are numerous methods that have been used over the years to estimate DTEE or some of its components (Bray, 1997; Ravussin & Rising, 1992; Y. Schutz et al., 2001; Shephard & Aoyagi, 2012). These are discussed in scores of articles and books (Acheson et al., 1980 a, b; Chen et al., 2012;; DeLany, 2012; DeLany & Lovejoy, 1996; Leonard, 2003; Leonard et al., 1997; Levine, 2005: Livingstone et al., 1992; Murgatroyd et al., 1993; Prentice, 1988; Prentice et al., 1991; Rosenbaum et al., 1996; and Westerterp et al., *1988*). Levine (2005) is a succinct summary of over 16 different approaches for measuring/estimating EE, many of which are not suitable for "free-living"/"free-ranging" conditions. A common experimental or laboratory method is the use of a room calorimeter, with a number of ways to estimate DTEE (and some of its components, such as REE using indirect calorimetry, sleeping EE, and even PAEE-with some additional information). The room calorimeter may utilize either direct or indirect methods to gather EE data (Levine, 2005). There also are non-caloric methods used to measure/estimate EE data. These include heart rate (HR) monitoring, DLW, and activity logs/factorial approach for free-living studies (Colbert & Schoeller, 2011). Other approaches require a subject to be in a defined space and so are experimental (Levine, 2005). A brief and interesting survey of EE measuring methods beginning with Lavosier's "respiratory chamber" in 1777 and ending with accelerometry in 2006 appears in Halsey (2011). A recent approach to estimating DTEE is via body-worn "calorimeters," including the Personal Calorie Monitor (Lyden et al., 2014), the Body Media armband (Lee et al., 2014), and the Energy-Monitoring Garment (shorts with textile electrodes: Tikkanen et al., 2014).

There are a number of devices of varying complexity to record HR over a day and "convert" it to oxygen consumption/EE estimates that have been applied to a wide variety of population subgroups (Levine, 2005; Livingstone et al., 1990, 2000). Problems with the HR approach are (1) the relationship between activity-specific EE and HR is non-linear; (2) there is considerable intra-individual variability between HR for most types of activity and EE; (3) affective and emotional factors affect the HR→EE relationship, and

these are not observable (Levine, 2005). Li et al. (1993) provide data that indicates HR recording has a 14-18% inter-individual COV and a 11-20% intra-individual COV in 40 30-y old females, and that there was poor agreement in EE between group and individual calibration of the HR→EE. They state that *only* individual calibration curves (HR→EE relationships) should be used with the HR approach to estimating DTEE (Li, et al., 1993). We do not provide DTEE estimates obtained using HR monitoring in this report. We similarly do not provide data from the activity log/factorial approach because of subject recording/recall issues (accuracy and precision) and the fact that activity-specific estimates of EE usually come from the Compendium (Ainsworth et al., 1993, 2000, 2011) or similar databases, and these are not specific to an individual or to the rate at which that individual is working (McCurdy et al., 2000).

Butte et al. (2010) estimated DTEE using HR and accelerometry in free-living youth 5-18 y and compared their estimates with DLW measures. A number of analytic procedures were used with the non-DLW techniques to better improve their performance in estimating DTEE. Overall, "predicted TEE values were within 11-14% of DLW-derived TEE in 75% of participants," but the remainder had larger differences (Butte et al., 2010; p. 1521). This, to me, is not very good agreement, and the non-DLW methods visually showed an increasing variance with total energy expended using Bland-Altman plots, meaning that there was heterogeneity in the method's residuals. There are numerous evaluations of different accelerometers against DLW; the interested reader is referred to the ever-expanding literature on the topic. One is RK Johnson et al. (1998), who conclude with "the main finding was that the Caltrac accelerometer was not a useful predictor of AEE (activity energy expenditure) in the sample" (p. 1050). The correlations between accelerometer-AEE and DLW-estimated AEE was a non-significant r = -0.09 (p=0.63) for a 3-d period (RK Johnson et al., 1998). Others have stated that accelerometer activity counts do "not reflect" DTEE in 4-6 y old children (Lopez-Alarcon et al., 2004). The same is true for adult activities, especially older adults with balance and gait issues. Newer accelerometers seemingly do a better job of estimating activity EE, but comparative performance is highly dependent upon the accelerometer model used (Mackey et al., 2011). A confounding problem is that the

Table 16. DTEE, REE, & PAI during pregnancy from BUTTE ET AL. (2004)

BMI Group	DTEE (kcal/d) Mean	SD	REE(kcal/d) Mean	SD	PAI Mean	SD	COV (%)	n
	Baseline (Pre-pregnant)							
Low	2348	276	1201	137	1.97	0.25	12.7	17
Normal	2434	368	1323	127	1.84	0.25	13.6	34
High	2940	421	1505	153	1.96	0.22	11.2	12
	22 Weeks Pregnant							
Low	2272	376	1330	121	1.72	0.28	16.3	17
Normal	2520	381	1413	142	1.78	0.28	15.7	34
High	2887	435	1393	210	1.72	0.25	14.5	12
	36 Weeks Pregnant							
Low	2439	485	1573	210	1.63	0.33	20.2	17
Normal	2693	372	1673	172	1.62	0.24	14.8	34
High	3020	553	2016	254	1.49	0.22	14.8	12
	27 Weeks Postpartum							
Low	2020	267	1254	169	1.68	0.30	17.9	17
Normal	2480	410	1323	136	1.88	0.29	15.4	34
High	2708	400	1505	171	1.77	0.19	10.7	12

Definitions & Abbreviations:

BMI:	Body Mass Index (kg/m**2)
COV:	Coefficient of Variation (SD/Mean)
DTEE:	Daily Total Energy Expenditure
High:	BMI >26.0 (kg/m**2)
Low:	BMI <19.8 (kg/m**2)
n:	Sample Size
Normal:	BMI 19.8-26.0 (kg/m**2)
PAI:	Physical Activity Index (DTEE/REE)

Definitions & Abbreviations:

REE:	Resting Energy Expenditure
SD:	Standard Deviation

Source:
Butte et al. (2004). "Energy requirements during pregnancy based on total energy expenditure and energy deposition." Amer. J. Clin. Nutr. 79: 1078-1087.

manufacturers change their models frequently (because of a bad evaluation?), and comparisons among a single manufacturer's different models are infrequent.

There are many algorithms in existence to convert accelerometer counts into EE estimates. Many have been developed by exercise physiologists and published in the literature so that the manufacturers' "black box" approach of doing so can be openly evaluated. In general, this work has shown that significantly different EE estimates can be obtained using different accelerometers on the same people and that these EE estimates also are significantly different than DLW measurements (Calabró et al., 2013). Newer accelerometer algorithms do a better job of replicating DLW estimates of DTEE than older algorithms (Calabró et al., 2013), but accelerometer-based estimates of PAEE still are problematic (Leenders et al., 2006). Scores of accelerometer/DLW "validation" studies are published every year. See the work by Freedson and colleagues listed in the references for a "taste" of these papers. Almost every academic exercise physiology program in the U.S. has done one or more accelerometer-EE comparisons—both *in vitro* (treadmill or other controlled-work rate method) and *in vivo* ("free-living"). This type of testing is a "growth industry" for academics. (Using "accelerometer accuracy" as a keyword phrase in a *Google Scholar* search comes up with 66,800 "hits" as of November 2013. Even if most of the hits are misidentified, redundant, or irrelevant, that leaves thousands of those types of evaluations.) See Section 10.

That leaves the doubly-labeled water (DLW) technique. Consensus of EE-measurement practitioners is that DLW is the most accurate method of estimating multi-day EE currently available (Friedman & Johnson, 2002; Racette et al., 2012; Roemmich et al., 2000; Schoeller, 1999, 2009; Schoeller & van Santen, 1982; Schoeller et al., 1980). A number of authors call it "the gold standard," "reference," or "state-of-the-art criterion method" (Eliakim et al., 1998; RK Johnson et al., 1998; Racette et al., 1995; Westerterp & Plasqui, 2004), and use it to evaluate other approaches to estimating DTEE. (Leenders et al., 2006; Schoeller et al., 1990; Schoeller & Webb, 1984; L.O. Schulz et al., 1992; S. Schulz et al., 1989; Seale et al., 1990; Singh et al., 2009; Staten et al., 2001; Trabulsi & Schoeller, 2001; Tran et al., 2000; Westerterp, 1999). A review of the use of DLW to estimate EE in ambulatory children is contained in Goran and Sun (1998), and in Leitch & Denne (2000) for low-weight infants.

DLW even has been adapted for use in estimating astronauts' EE during space flight, a unique environment posing extreme challenges on other EE-measuring devices (Gretebeck et al., 1997; Lane et al., 1997; Stein et al., 1999). However, DLW is not perfect, requiring a number of assumptions and calculations to be made about total body water volume, the isotopic elimination rates for ^{2}H and ^{18}O (see below), and other specific constants used to calculate the rate of CO_2 production given the DLW elimination rate. The analytic precision of DLW is variously stated to be ± 3% (Levine, 2005) or ± 4-5% (Black & Cole, 2000; Prentice et al., 1996; Westerterp et al., 1988). One of the foremost DLW scientists, Dr. D.A. Schoeller of the University of Chicago, states that DLW has a precision of 2-8% depending on the isotope dosage and the length of the elimination period used (Schoeller, 1988; Trabulsi et al., 2003). Goran et al. (1994) state that the "experimental variability" of DLW is 8.5% on average, and is 1- 21% over theoretical calculations for individual subjects. Discussion of the technical aspects of using the DLW technique, including the reasons for DLW imprecision, is found in Klein et al. (1984); Racette et al. (1994); Schoeller et al. (1986); Schoeller & Taylor (1987); Speakman (1990, 1995); Welle (1990); and Welle & Nair (1990).

DLW is predicated upon the fact that hydrogen and oxygen in ingested water equilibrates with body water at different rates, and this affects the turnover of water and the subsequent production of CO_2 in the body. This in turn—with some assumptions (see below)—can be used to estimate the metabolic uptake of oxygen and the production of whole-body EE (Bray, 1997). ^{2}H is "lost" as water, while ^{18}O is "lost" as both water and CO_2. (Cole & Coward, 1992; Wong, 1996). The difference in elimination rates provides an estimate of the amount of CO_2 expended over the sampling time period. The expended CO_2 is converted to EE, and divided by the elapsed time of the sampling time period. The resultant DTEE estimates are daily averages from the time of isotopes administration and whenever the urine/saliva samples are taken. Generally the elapsed period is anywhere from one week to three weeks. Re-dosing has to be undertaken for longer periods (DeLany et al., 1989). The method was developed largely by N. Lifson and colleagues in the late 1940's (Bray, 1997).

One reason for the variability in analytic precision in DLW estimates is that the conversion of CO_2 into its energy equivalents requires that the relative amount of body "fuel" type is known; the metric used for this is called the respiratory quotient (RQ). (See the Glossary for a discussion of RQ.) RQ varies by the relative amounts of carbohydrates, fats, and proteins (amino acids) "burned" to supply energy to the body. These proportions are quite different for different people (Coward & Cole, 1991). A population COV for RQ of 1.5% translates into an error of 2-3% in EE estimates for individuals (Coward & Cole, 1991). Another reason for precision error is that the disappearance of $^{2}H_2$ in body-water is nonlinear with time and this varies slightly with individuals, so the "model" (called a one-point or a two-point method) used to relate the disappearance rate is somewhat uncertain (Coward, 1988). A third reason is that the output rates (fluxes) of water and CO_2 are assumed to be constant with water pool size, which itself is assumed to be constant over time. Because eating and drinking is episodic, this assumption is frequently violated. These assumptions affect the time-dependent urine concentrations of the two isotopes, giving rise to uncertainty regarding DTEE estimates from the DLW approach (Coward, 1998). Finally, the DLW technique may be inappropriate for lactating mothers because precision of the method is compromised when water turnover is high, as is the case for lactating women (Lovelady et al., 1993). An

EE overestimation (~18%) in babies who are being weaned from a lactating mother has also been measured with the DLW method (Roberts et al., 1988).

Within-Subject Variation in DTEE

There have been a handful of free-living studies where DTEE has been repeated in individuals where body weight, activity, and physiological status remained unaltered over the two (or sometimes 3) measurement intervals. Nine of these studies are listed in Shetty et al. (1996), a very important FAO/WHO/UNU report that is available on the web. Some of the studies were undertaken in a metabolic chamber, and thus are not truly free-living, although there was no restriction on activity. The mean within-subject COV of the repeated-measurement studies are "reasonably small" with a mean COV of 8.9% for 79 individuals (Shetty et al., 1996). The study-specific mean COV's ranged from 6.8-11.0%. For just the free-living studies (n=7), I calculated the mean COV to be 9.2%.

Daily Variation of DTEE within a Week

Daily variations of DTEE within a week cannot be obtained from DLW studies unless the required urine samples are obtained daily. While possible to do so, it is very labor intensive and expensive. Thus, other EE-estimating methods are used to estimate daily differences in DTEE, mostly via heart-rate monitoring. A thorough study done in a chamber is Ribeyre et al. (2000), where French athletes and non-athletes aged 16-19 y of both genders (n=50) were monitored for a week. The mean daily range of DTEE and PAEE within the week is as follows:

	DTEE (kcal)	Daily Range (%)	Daily COV (%)	PAEE (kcal)	Percent of Day in Exercise
Female Non-Athlete	2174	-11 to + 14%	14%	550	1.1%
Female Athlete	2486	-17 to +11%	18%	574	5.0%
Male Non-Athlete	3131	-12 to +11%	12%	765	1.7%
Male Athlete	3609	-19 to +13%	17%	1052	6.4%

These data are quite interesting, even though they are from a chamber study. Female athletes expended about 14% more EE per day on average than their non-athletic counterparts, while male athletes spent about 15% more energy per day than non-athletic males. However, there is wide difference in DTEE in all four cohorts as evidenced by their daily ranges. These daily variations are rarely captured in the literature, and are not well captured in our exposure assessments that inadvertently minimizes day-to-day variability even though we have addressed daily differences in the time spent in generalized locations (Glen et al., 2008). Linking daily variability in individual activities and daily energy expenditure with daily variability in locations in our exposure models is a relatively unexplored topic, and much more work needs to be done in this area.

Seasonal Variations in DTEE

Schoeller & Hnlicka (1996) present repeated-measures data using the DLW technique on 6 employed females living in an urban area over two seasons. There were no significant seasonal differences in DTEE, REE, or PAEE estimates for the group, and the sample was equally split on whether or not winter EE > summer EE for the 3 metrics. They also provide estimates of DTEE COV's seen in a review of 16 repeated-measurement studies. The COV's ranged from 2.9% to 20.2%, with a mean of 7.8% (Schoeller & Hnlicka, 1996). Shetty et al. (1996) provide COV estimates of DTEE in weight-stable females for over a 7 months period; it was 2.0%, which seems low to me.

9.0
Physical Activity Index (PAI) & Physical Activity Level (PAL)

PAI and PAL relate to the same concept: PAI = DTEE / REE = PAL, total daily energy expended divided by basal, or resting, metabolic rate, both in kcal/d (or kJ/d). The result is a unitless metric. PAL seems to be the preferred acronym in Europe and PAI in the United States. PAI will henceforth be used in this report. McCurdy (2000) developed a "consensus" PAI classification of activity-level categories:

Sedentary	<1.55
Low Active	1.55 – 1.75
Moderately Active	1.76 – 2.00
Active	> 2.00

These categorizations are not universally accepted by exercise physiologists, and an alternative classification appears in Brooks et al. (2004). Their classes are:

Sedentary	1.00 – 1.39
Low Active	1.40 – 1.59
Active	1.60 – 1.89
Very Active	1.90 – 2.50

Note that the both of the "outer" classes are defined by a lower/upper limit; Brooks et al. (2004) do not provide a reason for this prescribed categorization, which results in uncertainty regarding PAI values outside of the bounds depicted.

The FAO/WHO/UNU (see Glossary) has developed its own PAI classifications for use in *developing countries*. Its categories are:

Light	1.40 - 1.69
Moderate	1.70 – 1.99
Vigorous	2.00 – 2.40

I could not find any defined values for categories on either side of those shown above (Dufour & Piperata, 2008). Farmers in undeveloped countries, by the way, have measured PAI's of 1.5-2.5 for males and 1.4-2.4 for females, on average. It has been estimated that the PAI value of early hominids was between 2.0-3.0 for subsistence farmers and 1.7-2.1 for hunters and gathers (Hayes et al, 2005b).

Another classification, based on both HR monitoring and accelerometers, for PAI is light <1.6; moderate 1.6-2.8; moderate 2.9-3.5; and vigorous >3.6 (Adolph et al., 2012). Obviously there is no consensus regarding the categorization of PAI in the literature.

Reasonable Boundaries of PAI

There have been several attempts to define reasonable boundaries of PAI for the general population. If a person is sleeping all day for whatever reason at an activity-specific METS ($METS_A$) of 0.80-1.10 (CHAD database, code 14400), her or his PAI would be close to 1.00. (The mean/SD for sleeping in CHAD is 0.90 ± 0.10, with a median of 0.90.) In an ambulatory population—for at least part of the day—the lowest feasible PAI is the oft-cited "Goldberg criterion" of 1.20, representing the minimally sustainable ratio of total EE-to-resting metabolism for ambulatory individuals (Black, 2000; Goldberg, 1997). However, there is disagreement concerning that value; the UNU uses 1.27 as "the survival" PAI (Goldberg, 1997), and other commentators usually round that value up to 1.30.

Inactive subjects in a calorimeter have a PA=1.21, lower that the UNU value of 1.27 (Shetty et al., 1996). Babies often have a PAI <1.3 as shown in Table 17 particularly at the younger ages. PAI increases significantly with age in babies and infants of both genders and feeding method (Butte et al., 2000).

With respect to mothers who breast-feed, lactation does not significantly affect PAI, although mothers 18-24 months post-delivery have a higher PAI than mothers that were measured within 3 months of delivery. See Table 18. Note that for both time periods, the mothers are in the "moderately active" category using the first PAI classification schema noted above.

Westerterp (2001) states that the upper bound of sustainable PAL in the general population is 2.2 - 2.5. He also states that athletes can have twice as high an upper bound due to long-term exercise training and their simultaneous consumption of carbohydrate-rich food. When athletes have a PAI >2.5, many of them "have problems maintaining energy balance" and thus lose weight (Westerterp, 2008). Shetty et al. (1996) consider a PAI of 2.4 to be "the maximum sustainable way of life for most people.

Very high PAI values have been found for participants in strenuous athletic events that can go on for several days. For instance, Cooper et al. (2011) provide data from 26 endurance-type events, and the uppermost PAI was 6.94 that was sustained over 10 days! Two other studies reported PAI's of >5.0 for a three-week and a 3-month elapsed period (Cooper et al., 2011)! S. Schulz et al. (1989) state that PAI's of 4.3-5.3 are common during the *Tour de France* races, and Westerterp et al. (1986) report PAI's of 3.6-5.2 for 3 riders in that race. A "Fitness Club" website states that

Table 17. Estimates of DTEE, SMR, & PAI for infants from BUTTE ET AL. (1990)

Feeding/ Age Group (months)	DTEE (kcal/d) Mean	SD	SMR (kcal/d) Mean	SD	PAI Mean	SD	COV (%)
	Females						
Breast							
3	394	72	330	31	1.20	0.22	18
6	554	112	428	36	1.30	0.24	18
9	645	98	497	53	1.25	0.16	13
12	736	120	598	48	1.24	0.15	12
18	820	160	645	74	1.26	0.27	21
24	951	151	684	84	1.39	0.28	20
Bottle							
3	452	93	366	29	1.20	0.19	16
6	614	91	464	33	1.32	0.15	11
9	707	98	521	43	1.36	0.19	14
12	782	129	578	65	1.40	0.29	21
18	860	174	648	69	1.38	0.28	20
24	1009	191	679	84	1.43	0.20	14
	Males						
Breast							
3	411	93	351	36	1.18	0.27	23
6	595	112	459	50	1.28	0.20	16
9	700	105	559	62	1.29	0.25	19
12	803	151	617	62	1.27	0.18	14
18	932	134	473	45	1.37	0.24	18
24	994	134	731	65	1.31	0.10	8
Bottle							
3	440	96	380	33	1.14	0.18	16
6	590	93	490	45	1.24	0.17	14
9	755	103	552	36	1.37	0.15	11
12	815	158	607	60	1.35	0.28	21
18	923	96	700	36	1.33	0.17	13
24	968	163	691	60	1.49	0.18	12

Abbreviations:	
COV:	Coefficient of Variation.
DTEE:	Daily Total Energy Expenditure.
PAI:	Physical Activity Index (DTEE/REE)
SD:	Standard Deviation.
REE:	Resting Energy Expenditure (= Sleeping EE in babies)

Source: Butte et al. (1990). "Energy utilization of breast-fed and formula-fed infants." *Amer. J. Clin. Nutr.* 51: 350-355.

Table 18. Estimates of DTEE, REE, PAEE, & PAI for lactating & non-lactating Mothers from BUTTE ET AL. (2001)

Group	DTEE (kcal/d) Mean	SD	REE (kcal/d) Mean	SD	PAEE (kcal/d) Mean	SD	Mean	PAI SD	COV (%)
Lactating	2392	351	1331	115	1061	284	1.79	0.20	11
Non-Lactating	2529	497	1350	143	1135	497	1.89	0.35	19

Abbreviations: See Table 17.

Note:
Lactating mothers were 3 months post-partum; n=24. Age at delivery=30.4 (3.2) y. Non-lactating mothers were between 18-24 months post-partum.

Source:
Butte et al. (2001). "Energy requirements of lactating women derived from doubly labeled water and milk energy output." *J. Nutr.* 131: 53-58.

"normal activity limits" (PAI) are between 1.2 and 5.5. Thus, it is physically possible to have very high PAI values for multiple days.

A review of the PAI literature provides the following general qualitative cutoffs from a summary of the literature.

Black (2000) contains generalized PAI data for all ages and both genders; her data were reprinted above as Table 13.

PAI Level	Descriptive Grouping
1.17	Obese adults
1.19	PAI measured in English females aged 91-97 (10% COV)
1.20	Goldberg Criterion (theoretical); chair-bound (Shetty et al., 1996)
1.21	Minimal sustainable (from data for ambulatory people)
1.27	FOA/WHO "survival requirement"
1.27	PAI of demented elderly
1.36	PAI measured in English males aged 91-97 (15.4% COV)
1.4 – 1.5	Seated occupation, little movement; little active leisure time (Shetty et al., 1996)
1.43-1.80	Range for retired females
1.55-1.77	Range for retired males
1.6 – 1.7	Seated work, some moving around; little active leisure activity (Shetty et al., 1996)
1.65	Mean of DLW studies used for evaluating the technique (18.2% COV)
1.75	Median PAL for the developed world (Butte et al., 2012)
1.8 – 1.9	Median value of "standing work" (Shetty et al., 1996)
2.0-2.4	Sustainable PAI's in active individuals (Shetty et al., 1996)
2.5	Very active lifestyle
3.1	Athletes in training
3.5	Nordic skiers
4.0	Maximal level of activity sustainable on a permanent basis
4.5	Dog-sledding PAI
4.7	PAI for "Tour-de-France" riders

Butte (2000) provides a table listing study-mean PAI (and DTEE, PAEE) for youth between the ages of 3.0-18.4 y old. It was developed from data from over 20 previous studies, many of which appear in this report as Table 12. There is no trend in PAI with age or gender. Using the categories presented above from McCurdy (2000), study mean PAI's from the Butte (2000) paper would be assigned to the following categories:

	Females	Frequency	Males	Frequency
<1.55	8	33.3%	5	21.7%
1.55 – 1.75	8	33.3%	8	34.8%
1.76 – 2.00	7	29.2%	6	26.1%
> 2.00	1	4.2%	4	17.4%

Thus the modal value of the studies for both genders are in the 1.55-1.75 category (low active).

Table 19. DTEE & PAI estimates from DLW studies reviewed in ROBERTS & DALLEL (2005)

Age Range (y)	Mean	DTEE (kcal/d) SD	COV (%)	Mean	PAI SD	COV (%)	Sample Size (n)
Normal-Weight Females							
20 - 29.9	3047	510	16.7	1.79	0.28	15.6	76
30 - 30.9	2964	429	14.5	1.83	0.26	14.2	59
40 - 49.9	3048	419	13.7	1.89	0.30	15.9	8
50 - 59.9	2513	401	16.0	1.75	0.22	12.6	18
60 - 69.9	2397	437	18.2	1.69	0.31	18.3	48
70 - 79.9	2407	374	15.5	1.55	0.26	16.8	14
80 - 89.9	1700	239	14.1	1.21	0.09	7.4	6
90 - 96.5	1935	156	8.1	1.17	0.13	11.1	9
Overweight Females							
20 - 29.9	2713	394	14.5	1.78	0.23	12.9	33
30 - 30.9	2794	358	12.8	1.78	0.23	12.9	41
40 - 49.9	3032	545	18.0	1.80	0.19	10.6	14
50 - 59.9	2349	368	15.7	1.68	0.26	15.5	29
60 - 69.9	2061	294	14.3	1.52	0.23	15.1	46
70 - 79.9	1868	402	21.5	1.51	0.28	18.5	19
80 - 89.9	1748	464	26.5	1.42	0.37	26.1	6
90 - 96.5	1766	292	16.5	1.33	0.22	16.5	7
Normal-Weight Males							
20 - 29.9	3047	510	16.7	1.75	0.22	12.6	48
30 - 30.9	2964	429	14.5	1.78	0.21	11.8	47
40 - 49.9	3048	419	13.7	1.84	0.23	12.5	22
50 - 59.9	2513	401	16.0	1.60	0.31	19.4	8
60 - 69.9	2397	437	18.2	1.61	0.18	11.2	14
70 - 79.9	2407	374	15.5	1.62	0.25	15.4	30
80 - 89.9	1700	239	14.1	1.17	0.15	12.8	4
90 - 96.5	1935	156	8.1	1.38	0.17	12.3	6
Overweight Males							
20 - 29.9	3224	842	26.1	1.90	0.20	10.5	10
30 - 30.9	2275	753	33.1	1.81	0.30	16.6	53
40 - 49.9	3465	588	17.0	1.88	0.24	12.8	37
50 - 59.9	3458	644	18.6	1.88	0.29	15.4	17
60 - 69.9	2851	420	14.7	1.71	0.29	17.0	30
70 - 79.9	2624	461	17.6	1.55	0.27	17.4	34
80 - 89.9	2294	357	15.6	1.47	0.16	10.9	7
90 - 96.5	1863	46	2.5	1.29	0.13	10.1	2

Abbreviations: Same as Table 17.

Source:
Roberts & Dallel (2005). “Energy requirements of aging.“ *Public Health Nutr.”* 8: 1028-1036.

Table 20. Estimates of DTEE, REE, PAEE, & PAI from SHETTY ET AL. (1996)

Age Group	DTEE (kcal/d) Mean	SD	REE (kcal/d) Mean	SD	PAEE (kcal/d) Mean	SD	PAI Mean	PAI SD	COV* (%)
Females									
18 - 29	2486	526	1482	263	1004	406	1.70	0.28	16.5
30 - 39	2390	406	1434	143	980	386	1.68	0.25	14.9
40 - 64	2342	406	1386	167	956	345	1.69	0.23	13.6
Males									
18 - 29	3298	717	1793	287	1506	598	1.85	0.33	17.8
30 - 39	3418	741	1960	430	1458	598	1.77	0.31	17.5
40 - 64	2749	406	1673	191	1076	311	1.64	0.17	10.4

Abbreviations & Symbols See Table 17.
*Calculated by the present author.

Source:
Shetty et al. (1996). "Energy requirements of adults: an update on basal metabolic rates (BMRs) and physical activity levels (PALs)." *Euro. J. Clin. Nutr.* 50(Supp. 1).

PAI and Health Issues

Mortality relative risk (RR) has been shown to be inversely related to PAI in 50-82 y old adults (Hamilton et al., 2007). While the RR numbers *per se* are not of interest in this report, data on the mean PAI levels by tertiles is of interest. The PAI for the three tertiles are (low-to-high): 1.48 ± 0.01, 1.68 ± 0.01, and 1.94 ± 0.02. The mean PAI value for the highest tertile is somewhat surprising given age of the subjects and the "high moderate"/"active" PAI classification scheme. A PAI of 1.94 certainly is higher than the mean PAI's shown in Table 12 for elderly subjects. The data in Hamilton et al. (2007) came from a 1953 English study, which is not included in this report.

Individual (Longitudinal) Variability in PAI

The multi-day COV for PAI within individuals is reported to be approximately 15% (Black, 2000), roughly comparable with most of the measured COV's seen in Table 12, but the variability in COV's seen there indicates that PAI varies considerably within similar age/gender cohorts. Shetty et al. (1996) state that Black et al. (1996) provide mean estimates *of within*-individual COV of 8.9%. Since PAI has REE as its denominator, variability in REE contributes to total variability in PAI. It is difficult to find COV estimates for REE to put that component into perspective, and the only COV estimates that I could find were for adults. Shetty et al. (1996) provide COV estimates for REE for days, weeks, and months. The daily estimates have an individual mean COV of 2.0 - 3.5% depending upon the studies (3 studies, with 43 total subjects); the weekly estimates have a mean COV of 2.2 - 4.8% (5 studies, with 47 subjects); while the one study using a monthly time span had a COV of 2.5% (Shetty et al. 1996). Shetty et al. (1996) considers the "intra-individual variations in BMR [REE], measured over a period of days, weeks, or even months or years, are small and probably not significant" (p. 2).

On the other hand, Black (2000) states that REE itself has a COV of 4-8.5%, and that the non-REE component of DTEE has even more variability. (Note that the COV for the different DLW studies itself had a multi-study cross-sectional COV of 18.1%, quite a high observation [Black, 2000].) In another report (Black & Cole, 2000), the mean within-person COV for PAI is 12.3%, while the estimated between-person variance is given as 10.7%. Obviously there is lack of agreement among researchers on the day-to-day variability of PAI within and among people.

Table 21. Estimates of PAI seen in the literature

Ages				PAI				
Mean	SD	Type	(n)	Mean	SD	COV	Citation	Comment
Females: Normal, Healthy, or Not-Specified								
a. Mean & SD statistics are provided for Age								
5.5	0.9	N	35	1.37			Nguyen et al. 1996	
5.5	0.4	H	13	1.37	0.17	12.4	Fontvielle et al. 1993	From individual data
7.6	1.7	N	25	1.41			Nagy et al. 1997	AA: Tanner 1
7.9	1.2	N	9	1.41			Nagy et al. 1997	C: Tanner 2
8.1	1.0	H	11	1.66	0.22	13.3	Dugas et al. 2008	EA

Table 21. Estimates of PAI seen in the literature (continued)

Ages				PAI				
Mean	SD	Type	(n)	Mean	SD	COV	Citation	Comment
8.1	1.7	N	24	1.44			Johnson et al. 2000	Fairly fat group; AA
8.1	1.4	N	55	1.34			Johnson et al. 2000	Fairly fat group; C
8.2	1.0	N	12	1.50	0.30	20.0	Treuth et al. 1998	
8.3	1.2	H	10	1.40	0.12	8.6	Dugas et al. 2008	MA
9.7	0.8	N	123	1.58			Bandini et al. 2002	Pre-pubertal
10.1	1.0	N	45	1.56			Craig et al. 1996	Premenarchal
10.2	1.4	N	13	1.71			Roemmich et al. 2000	Pre-pubertal
10.6	0.4	H	25	1.77	0.29	16.4	DeLany et al. 2006	C
10.7	0.7	H	28	1.74	0.32	18.4	DeLany et al. 2006	AA
10.7	0.9	N	73	1.59			Bandini et al. 2002	Pubertal
12.3	1.0	H	13	1.47			Calabro et al. 2013	Ages 11-14
12.6	0.7	M	53	1.48	0.22	14.9	DeLany et al. 2004	AA & C
12.7	2.3	N	27	1.69	0.19	11.2	Perks et al. 2000	
12.8	1.9	N	18	1.71			Roemmich et al. 2000	Pubertal
14.3	1.0	N	14	1.68	0.19	11.3	Bandini et al. 1990	
18.4	0.6	N	91	1.83			Stice et al. 2011	PAI data not reported
22.1	4.3	H	32	1.65	0.25	15.2	Hise et al. 2002	
24.1	3.5	N	10	1.90			Beidleman et al. 1995	
24.8	6.9	N	6	1.50	0.19	12.7	Casper et al. 1991	
25.2	3.5	H	10	1.77	0.32	18.1	Sawaya et al. 1995	9-day study
25.8	5.8	N	13	1.75			Leenders et al. 2006	13 accel. equations
28.0	5.7	N	33	1.70			Johannsen et al. 2008a	
31.0	6.0	N	9	1.64	0.34	20.7	Hibbert et al. 1994	PAI range: 1.34-2.15
31.3	5.0	LM	9	1.76	0.16	9.1	Lovelady et al. 1993	PAI range: 1.51-2.09
31.7	4.8	N	27	1.68	0.26	15.5	Weinsier et al. 2002	Group 1: Maintainers
32.6	13.1	NS	16	1.87			Luke et al. 2005	
33.0	6.0	N	12	1.67			Welle et al. 1992	Control group
34.0	6.1	H	83	1.56			Hunter et al. 2002	Premen.; ages 23-47 y
34.0	6.3	N	14	1.68			Amatruda et al. 1993	Ages 21-45
37.6	5.7	N	20	1.55	0.27	17.4	Weinsier et al. 2002	Group2: Gainers
38.0	8.0	H	15	1.64	0.19	11.6	Schoeller et al. 1997	Moderately active
39.6	5.9	H	10	1.75			Johannsen et al. 2008b	
48.0	14.0	H	20	1.89	0.35	18.5	Roubenoff et al. 2002	Control group
49.7	7.3	N	136	1.70	0.30	17.6	Masse et al. 2004	PAI range: 1.2-2.5
59.4	3.5	N	34	1.72			Bathalon et al. 2001	Restrain. Eaters
60.0	4.0	H	33	1.75	0.22	12.6	Hays et al. 2002	PAI range: 1.22-2.29
60.3	3.1	N	26	1.83			Bathalon et al. 2001	Unrest. Eaters
60.8	3.1	H	29	1.81	0.23	12.7	Vinken et al. 1999	Ages: 55-65
64.0	5.0	H	6	1.44	0.20	13.9	Goran & Poehlman 1992	PAI range: 1.25-1.82
64.0	7.0	NS	37	1.51	0.25	16.6	Starling et al. 1998a	Ages: 52-79; AA
65.0	8.0	H	37	1.43			Carpenter et al. 1998	AA
66.0	8.0	H	96	1.62			Brochu et al. 1999	Ages: 50-88
67.0	6.0	H	52	1.52			Carpenter et al. 1998	C

Table 21. Estimates of PAI seen in the literature (continued)

Ages				PAI				
Mean	SD	Type	(n)	Mean	SD	COV	Citation	Comment
67.6	4.1	NS	10	1.66			Roberts 1996	Meta-analysis
68.0	6.6	NS	43	1.62			Tomoyasu et al. 1999	White
69.0	5.4	N	29	1.72			Johannsen et al. 2008a	
70.0	3.9	N	15	1.80			Frisard et al. 2007	
71.5	4.8	N	21	1.56			Ades et al. 2005	
74.0	2.0	NS	10	1.62			Roberts 1996	Meta-analysis
74.0	4.4	H	10	1.59	0.19	11.9	Sawaya et al. 1995	9-day study
74.0	4.4	H	10	1.59	0.18	11.3	Vinken et al. 1999	Ages: 68-80
74.1	3.2	NS	67	1.69	0.24	14.2	Blanc et al. 2004	AA
74.5	2.8	N	40	1.68	0.19	11.3	Cooper et al. 2013	
74.8	2.8	NS	77	1.65	0.21	12.7	Blanc et al. 2004	AA
82.0	2.8	N	40	1.67	0.31	18.6	Cooper et al. 2013	
92.0	2.0	N	49	1.50			Johannsen et al. 2008a	
93.0	3.3	N	11	1.51			Frisard et al. 2007	
b. Complete age statistics are not provided								
5.0		N	43	1.35	0.14	10.4	Salbe et al. 1997	Pima Indians
5.0		N	19	1.37	0.12	8.8	Salbe et al. 1997	Whites
5 -10		H	19	1.45	0.18	12.4	Trowbridge et al. 1997	AA
5 -10		H	14	1.49	0.19	12.8	Trowbridge et al. 1997	C
8 - 9		H	27	1.59	0.21	13.2	Treuth et al. 2003a	2 lean parents
8 - 9		H	38	1.62	0.31	19.1	Treuth et al. 2003a	1 lean/1 obese parent
8 - 9		H	23	1.62	0.24	14.8	Treuth et al. 2003a	2 obese parents
8 -12		H	196	1.58			Bandini et al. 2004	Premenarchal
8 -12		NS	90	1.50			Bandini et al. 2013	Relatively low active
30-69		NS	180	1.59	0.24	15.1	Tooze et al. 2013	
49-79		NS	21	1.91			Mahabir et al. 2006	Postmenopausal
60-69		N	48	1.69	0.31	18.3	Roberts & Dallal 2005	
70-79		NS	14	1.65	0.26	15.8	Roberts & Dallal 2005	
80-89		NS	6	1.21	0.09	7.4	Roberts & Dallal 2005	
90-97		NS	9	1.17	0.13	11.1	Roberts & Dallal 2005	
Females: Active, Fit, or Athlete **a. Mean & SD are provided for Age**								
20.0	2.0	Fit	20	3.30	0.40	12.1	Castellani et al. 2006	Winter military act.
21.5	1.9	Ath	10	2.31			Beidleman et al. 1995	
23.4	4.7	At.	5	3.00	0.45	15.0	Trappe et al. 1997	Olympic trials training
25.0	1.3	Fit	9	2.50	0.50	20.0	Ruby et al. 2002	Wildfire firefighters
26.0	3.3	Ath	9	1.99	0.30	15.1	L.O. Shulz et al. 1992	Elite distance runners
40.0	7.0	Act	9	1.89	0.24	12.7	Schoeller et al. 1997	
b. Complete age statistics are not provided								
8 –12		Act	71	1,72			Bandini et al. 2013	Relatively active
Females: Sedentary, Overweight, or Obese **a. Mean & SD statistics are provided for Age**								
5.5	0.3	OW	51	1.35	0.14	10.4	Bunt et al. 2003	Pima Indians
8.5	2.0	OW	14	1.25			RK Johnson et al. 1998	Mohawk & Caucasian

Table 21. Estimates of PAI seen in the literature (continued)

Ages				PAI				
Mean	SD	Type	(n)	Mean	SD	COV	Citation	Comment
8.7	0.7	OW	12	1.60	0.20	12.5	Treuth et al. 1998	
10.5	0.3	OE	51	1.58	0.14	8.9	Bunt et al. 2003	Pima Indians
13.4	0.8	OW	20	2.02	0.41	20.3	R.Singh et al. 2009	
15.2	1.8	O	16	1.74	0.19	10.9	Bandini et al. 1990	
29.0	4.0	O	5	1.95	0.45	23.1	Hibbert et al. 1994	
31.3	13.0	O	9	1.58	0.15	9.5	L.O. Schulz et al. 1994	Pima Indians
32.0	10.0	OW	28	1.58	0.17	10.8	Tataranni et al. 2003	Pima Indians
34.6	10.6	M	172	1.75	0.20	11.4	Ebersole et al. 2008	2/3 were OW or O
35.2	7.4	Sed	17	1.61			Hunter et al. 2000	Premenopausal C
35.6	6.9	Sed	18	1.60			Hunter et al. 2000	Premenopausal AA
36.0	7.0	OW	26	1.73			Welle et al. 1992	
38.0	5.0	Sed	8	1.44	0.23	16.0	Schoeller et al. 1997	
38.5	6.1	O	10	1.56			Johannsen et al. 2008b	
38.6	8.1	OW	30	1.76	0.18	10.2	Roberts et al. 2012	CALERIE Study
38.7	6.0	O	15	1.78			Kushner et al. 1995	C
39.5	5.2	O	18	1.81			Amatruda et al. 1993	Ages 31-51
39.8	5.0	O	14	1.66			Kushner et al. 1995	AA
43.8	9.2	OW	35	2.31			Staten et al. 2001	(Question the PAI)
48.0	10.0	OW	47	1.69	0.19	11.2	Paul et al. 2004	
57.5	4.2	O	15	1.70			Rawson et al. 2002	Trp64Arg Non-Carriers
57.8	6.6	O	19	1.75			Rawson et al. 2002	Trp64Arg Carriers
64.0	8.0	OW	37	1.51	0.25	16.6	Starling et al. 1998b	AA
65.0	3.5	O	25	1.46			Nicklas et al. 1997	AA
75.5	2.8	OW	72	1.71	0.30	17.5	Manini et al. 2009	AA
75.5	2.8	OW	80	1.65	0.20	12.1	Manini et al. 2009	C
b. Complete age statistics are not provided								
40–69		OW	206	1.75	0.56	32.0	Tooze et al. 2007	62% was OW or O
49-79		OW	25	1.97			Mahabir et al. 2006	Post-menopausal
49-79		OW	19	1.73			Mahabir et al. 2006	Post-menopausal
60-69		OW	46	1.52	0.23	15.1	Roberts & Dallal 2005	
70-79		OW	19	1.51	0.28	18.5	Roberts & Dallal 2005	
80-89		OW	6	1.41	0.39	27.7	Roberts & Dallal 2005	
90-97		OW	7	1.33	0.22	16.5	Roberts & Dallal 2005	
Females: Health & Other Issues								
24.5	6.9	ANP	6	1.96	0.34	17.3	Casper et al. 1991	Amenorrheic
39.9	11.9	CP	12	1.46			RK Johnson et al. 1997	
47.0	14.0	RA	20	1.70	0.24	14.1	Roubenoff et al. 2002	Stable, with drugs
72.9	6.1	CHD	21	1.56			Ades et al. 2005	
Males: Normal, Healthy, or Not-Specified **a. Mean & SD statistics are provided for Age**								
5.2	0.7	N	36	1.27			Nguyen et al. 1996	
5.4	0.3	H	15	1.36	0.13	9.6	Fontvielle et al. 1993	From individual data
7.4	1.6	N	22	1.36			Nagy et al. 1997	AA: Tanner 1

Table 21. Estimates of PAI seen in the literature (continued)

Ages				PAI				
Mean	SD	Type	(n)	Mean	SD	COV	Citation	Comment
7.6	1.0	H	10	1.57	0.18	11.5	Dugas et al. 2008	MA
7.6	1.5	N	19	1.43			MS Johnson et al. 2006	Fairly fat group; AA
8.0	1.0	H	16	1.58	0.19	12.0	Dugas et al. 2008	EA
8.3	1.6	N	20	1.35			Nagy et al. 1997	C: Tanner 1
8.7	1.8	N	17	1.36			MS Johnson et al. 2006	Fairly fat group; C
10.9	0.6	H	29	1.69	0.23	13.6	DeLany et al. 2006	C
10.9	0.7	H	31	1.87	0.26	13.9	DeLany et al. 2006	AA
10.9	1.0	N	14	1.75			Roemmich et al. 2000	Pre-puberal
12.5	1.6	N	23	1.74	0.22	12.6	Perks et al. 2000	
12.8	0.8	M	61	1.55	0.23	14.8	DeLany et al. 2004	AA & C
12.9	2.1	H	15	1.53			Calabro et al. 2013	Ages 10-16
13.4	1.2	n	14	1.57			Roemmich et al. 2000	Pubertal
14.5	1.5	N	14	1.79	0.20	11.2	Bandini et al. 1990	
22.3	1.9	N	14	1.98	0.34	17.2	Roberts et al. 1991	Sed. Occup + active
22.7	2.5	H	17	1.97			Roberts et al. 1995	Same as above?
22.7	3.8	H	22	1.63	0.31	19.0	Hise et al. 2002	
23.1	2.4	N	24	1.94	0.31		Vinken et al. 1999	Age range: 18-28
27.0	4.4	N	20	1.89			Johannsen et al. 2008a	
35.9	13.4	NS	16	1.94			Luke et al. 2005	
41.2	9.8	NS	24	1.81	0.15	8.3	Conway et al. 2002	
42.0	16.0	H	30	1.38			Rising et al. 1994	Pima Ind.; some O
64.0	7.0	H	28	1.62			Carpenter et al. 1998	AA
64.0	8.0	NS	28	1.71	0.32	18.7	Starling et al. 1998a	Ages: 52-79
67.0	8.0	H	84	1.70			Brochu et al. 1999	Ages: 45-90
67.8	6.1	H	20	1.74	0.27	15.5	Vinken et al. 1999	Ages: 60-81
68.0	6.4	H	18	1.82			Roberts et al. 1995	
68.0	6.0	H	7	1.51	0.27	17.9	Goran & Poehlman, 1992	PAI range: 1.25-2.11
69.0	5.4	N	29	1.84			Johannsen et al. 2008a	
69.0	7.0	NS	15	1.75			Roberts 1995	Meta-analysis
70.0	6.9	H	9	1.72	0.69	40.1	Roberts et al. 1996	Same as above?
71.0	4.1	H	17	1.75			Frisard et al. 2007	
70.0	6.2	NS	39	1.74			Tomoyasu et al. 1999	White
70.0	7.0	H	47	1.63			Carpenter et al. 1998	
71.0	5.0	NS	16	1.51			Roberts 1996	
74.7	3.2	N	47	1.77	0.23	13.0	Cooper et al. 2013	
74.8	2.9	NS	72	1.71	0.22	12.9	Blanc et al. 2004	W; ages: 70-79
75.1	3.2	NS	72	1.74	0.22	12.6	Blanc et al. 2004	Ages: 70-79
82.0	3.0	NS	23	1.50	0.20	13.3	Fuller et al. 1996	Ages: 76-88
82.2	3.3	N	47	1.68	0.21	12.5	Cooper et al. 2013	
92.0	2.0	N	46	1.58			Johannsen et al. 2008a	
93.0	3.3	N	11	1.58			Frisard et al. 2007	

Table 21. Estimates of PAI seen in the literature (continued)

Ages				PAI				
Mean	SD	Type	(n)	Mean	SD	COV	Citation	Comment
b. Complete age statistics are not provided								
5.0		N	41	1.35	0.13	9.6	Salbe et al. 1997	Pima Indians
5 –10		H	12	1.44	0.17	11.8	Trowbridge et al. 1997	C
5 –10		H	17	1.41	0.17	12.1	Trowbridge et al. 1997	AA
5.0		N	24	1.33	0.13	9.8	Salbe et al. 1997	Whites
30-69		NS	189	1.69	0.25	14.8	Tooze et al. 2013	
60-69		NS	14	1.61	0.18	11.2	Roberts & Dallal 2005	
70-79		NS	30	1.62	0.25	15.4	Roberts & Dallal 2005	
80-89		NS	4	1.17	0.15	12.8	Roberts & Dallal 2005	
90-97		NS	6	1.39	0.17	12.2	Roberts & Dallal 2005	
Males: Active, Fit, or Athlete								
a. Mean & SD are provided for Age								
20.0	2.0	Fit	30	3.40	0.50	14.7	Castellani et al. 2006	Winter military act.
21.0	2.9	Act	13	1.78	0.25	14.0	Haggerty et al. 1997	Construction workers
24.5	1.8	Fit	7	2.80	0.50	17.9	Ruby et al. 2002	Wildfire firefighters
25.0	3.0	Fit	10	2.80	0.20	7.1	Hoyt et al. 2001	Cold military training
27.1	4.2	Fit	27	2.79	0.16	5.7	Hoyt et al. 1991	High-alt. military train.
31.0	4.0	Fit	6	3.14	0.19	6.1	Hoyt et al. 1994	High-alt. mitary train.
Males: Sedentary, Overweight, or Obese								
a. Mean & SD statistics are provided for Age								
8.2	1.9	OW	17	1.45			RK Johnson et al. 1998	Mohawk & Caucasian
13.7	0.7	OW	14	1.99	0.32	16.1	R. Singh et al. 2009	
14.4	1.9	O	18	1.68	0.19	11.3	Bandini et al. 1990	
35.4	13.8	O	12	1.66	0.28	16.9	Paul et al. 2004	Pima Indians
37.0	13.0	OW	64	1.58	0.20	12.7	Tataranni et al. 2003	Pima Indians
47.0	11.0	OW	44	1.64	0.19		Paul et al. 2004	
64.0	7.0	OW	28	1.71	0.32	18.7	Starling et al. 1998	AA
66.0	4.6	OW	21	1.68			Nicklas et al. 1997	AA
75.2	2.9	OW	74	1.71	0.24	14.0	Manini et al. 2009	AA
75.5	3.1	OW	76	1.73	0.21	12.1	Manini et al. 2009	
b. Complete age statistics are not provided								
40-69		OW	244	1.69	0.22	13.0	Tooze et al. 2007	75% were OW or O
60-69		OW	30	1.71	0.29	17.0	Roberts & Dallal 2005	
70-79		OW	34	1.55	0.27	17.4	Roberts & Dallal 2005	
80-89		OW	6	1.47	0.16	10.9	Roberts & Dallal 2005	
90-97		OW	2	1.29	0.13	10.1	Roberts & Dallal 2005	
Males: Health & Other Issues								
a. Mean & SD statistics are provided for Age								
35.1	11.5	CP	18	1.52			RK Johnson et al. 1997	
62.0	8.0	Park	16	1.34			Toth et al. 1997a	
72.9	7.9	Park	20	1.50			Delikanaki-Skaribas et al. 2009	

Table 21. Estimates of PAI seen in the literature (continued)

Notes & Abbreviations:	
AA	African-American
Accel.	Accelerometers
Act.	Activite
AI	American Indian
ANP	Anorexia Nervosa Patients
Astro.	Astronauts
Ath.	Athlete
C	Caucasian
CHD	Chronic Heart Disease
COV	Coefficient of Variation (SD/Mean*100)
CP	Cerebral palsy
DTEE	Daily Total Energy Expenditure (kcal/day)
EA	European-American
H	Healthy
LM	Lactating Mothers
M	Mixed lean and obese subjects

Notes & Abbreviations:	
MA	Mexican-American
(n)	Sample Size
N	Normal
NS	Not Specified
PAEE	Physical Activity Energy Expenditure
PAI	Physical Activity Index (DTEE/REE; also known as PAL: Physical Activity Level)
Park	Parkinson Disease patient
RA	Rheumatoid Arthritis
REE	Resting Energy Expenditure
RH	Rett Syndrome (a neurodevelopmental disorder)
O	Obese
OW	Overweight
SD	Standard Deviation
Sed	Sedentary

Perhaps the clearest delineation of variability in PAI is provided in Shetty et al. (1996):

> *"Thus, the 95% confidence limits on PALs [PAI's] at the individual level, assuming a measured BMR [REE] and no change in body weight or physical activity is on the order of ± 18.5%, representing about ± 0.3 PAL units on a mean PAL value of 1.65 (p. 1)."*

There have been very few studies of PAI changes in a cohort of individuals monitored over a long intervening period. One such study is contained in Cooper et al. (2013), which provides PAI data for 40 females and 47 males monitored in both 1999 and 2006. The subjects were in their mid-70s in 1999 and in their low-80s in 2006. While their data are included in Table 12, it is interesting to note that PAI in older females did not change significantly over the 7 years (1.68→1.67, on average), while the males registered a significantly lower PAI in the later year (1.77→1.68, on average) (Cooper et al., 2013).

PAI and Physical Activity at Various Levels

There is recent discussion in the exercise physiology field concerning how much moderate/vigorous physical activity contributes to PAI estimates, and the form of the relationship between PAI level and the time spent in moderate/vigorous activity (Thompson & Batterham, 2013). A parallel concern is how sedentary time affects PAI. The specific levels of activities (in terms of METS) are often called "dimensions" of PA in that literature (Thompson & Batterham, 2013). In a clinical study of 100 males over 7 days, a high correlation was found for the time spent in activities having a METS>3.0, and PAI, but that correlation decreases when the same METS criterion is held for 10 minutes or more. The association deteriorates further when >6 or >7.2 METS activities lasting >10 minutes are considered. In fact, the subjects spending the most time in >7.2 METS activities in bouts of 10 minutes or more had PAI values in the 1.65-2.05 range. Significantly less time—about 50%--was spent in those activities for subjects with PAI's >2.02 (Thompson & Batterham, 2013).

Conversely, while spending relatively more time in sedentary activities is generally negatively correlated with PAI, there is not a good correlation between relative sedentary time and time spent in activities >3 METS lasting >10 minutes (Thompson & Batterham, 2013). Because of these counter-intuitive findings, the authors make this conclusion:

> *The attainment of one threshold for a given physical activity dimension did not automatically predict how well an individual scored in another dimension…Thus, physical activity is highly heterogeneous and there is no single outcome measure that captures all the relevant information about a given individual. We propose that future studies need to capture (rather than ignore) the different physiologically-important dimensions of physical activity via generation of integrated, multidimensional physical activity 'profiles' (p. 1, e56527).*

In other words, the relationship between sedentary, moderate, and vigorous PA is not linear with daily PAI measures.

From a time use perspective, the main factor affecting PAI is the amount of time spent in moderate-intensity exercise or work level. Time spent in moderate and vigorous physical activity—MVPA—is discussed in some detail in the next Section. From Westerterp (2001):

> *In a multiple regression analysis with the fraction of time spent on activities of moderate and high intensity, only moderate activity level came out as a significant predictor of PAL (R2 = 0.51, p<0.0001). Subjects spending relatively more time on moderate-intensity activity and therefore less on low-intensity activity had a higher PAL value. There was, however, no relation between PAL value and the time spent on just high-intensity activity, presumably because this was limited by its nature to being relatively short (p. 539).*

The dominant impact of physical activity on PAI is confirmed in Westerterp & Plasqui, 2004). In addition, Westerterp (2003) states that <25% of PAEE in the "average subject" is due to high-intensity activities; the rest is due to light and moderate-level activities.

A somewhat surprising conclusion regarding the role of PAEE in DTEE, and PAI, is the statement by Westerterp & Speakman (2008) that "physical activity energy expenditure has not declined since the 1980s" in both Europe and the U.S. (even though obesity rates have increased)! Another surprising finding is that PAI is not significantly different between developed countries and "third-world" countries, or in wild terrestrial mammals (Westerterp & Speakman, 2008). I could not find confirmation for either of these statements in the more general DTEE/PAI literature(s).

Physical Activity and Physical Activity Index in Asthmatics

In 2005, about 9% of children living in the U.S. are asthmatic (Brim et al., 2008) and 3-9% of women of child-bearing age in the U.S. have asthma (Kwon et al., 2003). Thus, they are a significant part of the overall U.S. population.

Asthmatics have a slightly lower VO_2 capability than "normal" people of the same age/gender cohort and exercise level, especially for children (Counil et al., 1997, 2001; Fink et al., 1993). VO_2 can be improved in some asthmatics, however, with a sustained and high-intensity exercise program, but there is a drop-out issue associated with such a program (Counil et al., 2003; Crosbie, 2012; Dogra et al., 2011). Such improvement is not seen in youth who have exercise-induced asthma or exercised-induced bronchoconstriction (Carlsen et al., 2000; Fitch et al., 1986). Active asthmatic children can achieve $VO_{2.MAX}$ levels about 95% of non-asthmatics, but do so by lowering their respiratory frequency (f_B) and increasing their tidal volume (V_T) (Santuz et al., 1997). One of the manifestations of these changes in breathing pattern is a "shortness of breath" (Ritz et al., 2010). The net impact of these adaptations and changes is to reduce an asthmatic's "ventilatory reserve capacity" (BD.Johnson et al., 1995). One magnification of this in adults is to lose "elastic recoil" of the lung, leading to an increased cost of breathing (Johnson et al, 1991).

A logical conclusion to draw from these findings is that asthmatics would have a reduction in exercise capability, and less desire to undertake high-energy exercise (Kosmas et al., 2004; Lang et al., 2004). One author states that asthma is a "barrier" to exercise" (Glazebrook et al., 2006). This, in turn, would lead to lower fitness levels (Strunk et al., 1989), and a tendency to avoid MVPA activities (Brasholt et al., 2010; Shamoo et al., 1994). This would then result in lower PAI levels in asthmatics, especially children. These were the conclusions drawn from an early survey of asthmatics (all ages) in Los Angeles (Lichtenstein & Wyzga, 1989). They found that asthmatics spend much of their time indoors at relatively low exertion levels, and when higher levels of exertion occur, asthmatic symptoms increase (Lichtenstein & Wyzga, 1989). This survey was described in more detail in Roth Associates (1988). These general findings were also seen in a survey of 136 asthmatics in Cincinnati, with more detailed findings: <8% of asthmatics in Los Angeles and <13% of asthmatics in Cincinnati exercised strenuously in any hour (Lichtenstein et al., 1989). Overall, <80% of an asthmatic's waking time was spent at <moderate exercise level (Lichtenstein et al., 1989). (Note: this proportion would work out to be an overly-high estimate of 180 min/d at MVPA if a 15 h waking day is assumed!) The Cincinnati survey of daily asthmatic activity patterns is fully described in Roth Associates (1992). The reported findings there were only marginally different that the Lichtenstein & Wyzga (1989) paper. One interesting addition is that the authors state that 3.3% of the subject's waking hours was spent in strenuous **outdoor** activities (Roth Associates, 1992). Again, assuming a 15 h daily awake period, the 3.3% estimate works out to be 30 min/d, higher than usually seen (see Table 24), especially considering it all occurred outdoors! Their sample included 136 randomly-sampled subjects (58% female; 80% white, an average age of 26 y and a range of 1-78); see p. 2-7 of Roth Associates (1992).

In a telephone-based survey of parents of asthmatic (n=137) and non-asthmatic (n=106) children aged 6-12 y, Lang et al. (2004) report that "children with asthma were less active than their peers" (p. e341). This held for both duration and intensity of daily activity. The authors attribute the differences to disease severity and parental health beliefs (Lang et al., 2004).

A wrist-accelerometer study was undertaken of both "normal" and asthmatic children aged 9-11 y by Tsai et al. (2012). Its data appear in Table 24, but a few comments about the study are in order here. For one thing, the paper includes a succinct tabulation of 11 asthmatic PA studies from the US and other countries (Table 1 in their paper). Some of these studies have been mentioned here. Two of the studies found that asthmatic children were more active than non-asthmatic children ("normals"), 4 found no difference in PA, and the remainder (5 studies) found that normals participated in more PA than asthmatics (Tsai et al, 2012). Thus, there is conflicting evidence about PA in asthmatics. Tsai et al. (2012) found less MPA, VPA, and MVPA in asthmatics in their comparative study, but the differences were not significant at $\alpha<0.05$.

The US Center for Disease Control and Prevention (CDC) periodically conducts a cross-sectional survey of "Youth Risk Behavior Survey" (YRBS), one part of which focuses on physical activity. The 2003 YRBS study compared asthmatic youth's MVPA rates versus youth without asthma (SE Jones et al., 2006). There was very little difference in PA and VPA participation rates for the two groups; in fact, current asthmatics had slightly higher participation rates in both VPA (65.4% v. 63.2% for non-asthmatics) and MPA (25% v. 24.1%). While it can be argued that the actual energy expended was different for the two groups, and thus were not comparable, a marginally higher percentage of asthmatics than non-asthmatics also played on a sport team (58.8% v. 56.7%) (SE Jones et al., 2006).

Westermann et al. (2008) found less physical activity in adult asthmatic patients than in the general population using the "Paffenbarger Physical Activity and Exercise Index" derived from in-office visit questionnaires. In general, there were not significant differences among asthma severity score and PA/exercise (based on multivariate odds ratios), although there was an issue in separating out body mass (BMI) effects from asthma impacts (Westermann et al., 2008).

A number of non-USA studies have also not seen statistically significant lower exercise rates—either in duration or intensity—between asthmatic and non-asthmatic youths aged 7-16 y (Nystad, 1997; van Gent, et al., 2007). The later study used an accelerometer for 5 days, a physical activity diary, and a questionnaire-based "scale" filled out by the 7-10 y old children. Three groups of participants were included: diagnosed and undiagnosed asthmatics plus a healthy control group (van Gent et al., 2007). They conclude: "childhood asthma does not appear to be associated with a decreased level of physical activity in our study population" (p. 1018). This included both similarities in the frequency and intensity of PA. For the record, accelerometer-based overall mean min/d (range) of the MVPA data for the 3 groups were:

	Undiagnosed Asthma	Diagnosed Asthma	Healthy Controls
MVPA	86 (76-95)	78 (66-90)	78 (71-85)
VPA	22 (15-25)	21 (14-28)	20 (14-21)

These daily values were lower, but not by much, than those obtained using the diary or questionnaire.

What accelerometer data that I could find on asthmatic's MVPA appear in Table 24; there are not many entries there for asthmatics. The above findings relating to the amount of MVPA that asthmatics participate in *vis-à-vis* "normals" certainly indicate that no definitive statement can be made regarding the relative impact that asthma has on MVPA. More definitive data on that point are needed.

10.0
Time Spent Per Day in Moderate / Vigorous Physical Activity (MVPA)

Introduction

The type of physical activity (PA) undertaken during an exposure event obviously affects a person's intake dose rate during that event because of breathing rate characteristics associated with the activity ($V_{E.A}$). As depicted in Figure D-4, $V_{E.A}$ is dependent upon oxygen consumption ($VO_{2.A}$) associated with the activity and other physiological considerations that are specific to an individual. $VO_{2.A}$ is, in turn, predicated upon the activity-specific energy expenditure (EE_A) needed to undertake an activity. All of these parameters are represented in our exposure models by age/gender-specific distributions for these physiological considerations. Age and gender are the most important determinants of how much time is spent in moderate and vigorous physical activity (Arroyo, 2001, 2002). It is well documented by both objective measures and questionnaire surveys that MVPA declines rapidly during middle adolescence, especially for females, but the decline in both genders is significant (Bradley et al. 2011). Time spent in MVPA keeps declining with age, from about 35% of total non-sedentary time at age 20 y to 20% at age 90 y (Westerterp, 2000). See also Westerterp (2001, 2003).

Health-compromised individuals, including being overweight and obese, participate is less MVPA—both in total amount and less frequent participation--than healthy, normal-weight people (Brasholt et al., 2010). Persons with eating disorders—especially anorexia—on the other hand, often participate in more MVPA than "normal" control subjects (Bratland-Sanda et al., 2010). There is a strong cultural component to time spent in MVPA and its distribution over the day, week, and other time periods (Tudge et al., 2006).

There are data from the CDC and NHANES interviews that some adolescents, in particular, are active in many sports and exercise classes, much more than the "typical" teen-ager (Liu et al., 2013). There is a correlation structure, in other words, among the activities and activity level in teens that participate in organized sports. Adolescents "clump" into "natural" activity-level groupings depending upon their dominant or most prevalent sport. For males 12-19 the natural groupings are (1) basketball players and runners, (2) football players, (3) bike riders and soccer players; for females 12-19 they are (1) dancers/walkers/joggers, (2) swimmers, (3) volleyball players, and (4) soccer players (Liu et al., 2013). Athletes also participate in other sports and undertake more PA in general than sedentary individuals. The so-called natural groupings are affected by race, weight status, geographic region of the county, and season of the year—in addition to age and gender (Liu et al., 2013). Active people, in general, are more likely to have a higher intake dose rate given similar microenvironmental concentrations.

As with most biological processes and phenomena, there are four aspects, or dimensions, of physical activity that are important in delineating activity level: intensity, duration, frequency, and pattern. *Intensity* relates to the amount of energy expended in an activity. *Duration* is related to how long PA is undertaken at a specified intensity; *frequency* is how often a specified PA "bout" is repeated within a longer time period (often called an "epoch"); *pattern* refers to the time pattern of specified PA "bouts" within an epoch. Bailey et al. (1995) use the word **tempo** to account for the four dimensions of physical activity (PA). There are other temporal dimensions of MVPA associated with climate and season of the year, weekend versus weekday patterns, and time spent outdoors rather than indoors (Garcia et al., 1997; Kohl III & Hobbs, 1998; Pivarnik et al., 2003).

While all physical activities have an activity-specific energy expenditure (EE_A) associated with them, activities that are innately more energetic affect intake dose rate estimates the most. The hierarch of qualitative PA's that often is used is: resting, sedentary, light PA, moderate PA, and vigorous PA (Pate et al., 1995). Alternate descriptors exist but that is a representative listing. Often these descriptors are tied to an activity-specific METS level, with some consensus—but not unanimity—among exercise physiologists regarding appropriate METS levels for an activity. Most often moderate PA is defined to have a METS of 3.0-5.9 and vigorous PA has a METS of 6.0 or higher. Other METS levels have been used for these categories (e.g., Millward et al., 2014). In the 3/6 METS scheme, MVPA is any activity over 3 METS. Gyinhouya & Hubert (2008) state that using a METS level of 3 inflates estimates of the time spent in MPA and MVPA for most people, and especially for children. This comment highlights the difficulties of precisely defining meaningful physical activity categories. A related issue is how to accurately characterize the level of PA that is being measured. As is discussed throughout this Section, there is no good resolution of these issues: every researcher has their own way of defining and measuring MVPA (and, of course, sedentary and light activities).

An interesting description of MPA and VPA appears in Haennel & Lemire (2002) that ties together a number of exercise metrics used in this report. Data from their Table 1 (p. 67) is reproduced here:

Metric	MPA	VPA
METS	3 – 6	> 6
VO_2 & heart rate reserve (%)	40 – 59	> 60
Maximum heart rate (%)	65 – 75	> 75
Rating of perceived exertion (RPE)	"Somewhat hard"	"Hard"

The RPE metric, also known as the Borg scale, has not been discussed before in this report due to its subjective nature and rather narrow use in laboratory studies (mostly measuring VO_2 and associated metrics) or in exercise classes. RPE was first developed in the 1950s. There is an extensive literature on RPE and similar subject ratings of exertion (e.g., Borg, 1973, 1982; Herman et al., 2003). A synoptic compilation of indices of what constitutes MPA, VPA, and MVPA is contained in Table 22. It focuses on quantitative metrics involving METS, VO_2, EE, V_E, and heart rate parameters. Except where noted when discussing the "compromises" discussed in the footnotes, no attempt has been made to resolve differences among the various author's metrics.

Levels of PA lower than "moderate," however defined, are not of interest in this Report, even though most of an individual's day is spent at relatively low levels of PA. Non-MVPA activities obviously affects a person's daily *total* energy expenditure (DTEE); see Section 8. Although MVPA activities having >3 METS typically constitute less than 1-2 h/d, they often provide a significant proportion of a person's daily total energy expenditure (Butte et al., 2012). For instance, a PAL value of 1.75—which is close to the median for people living in the U.S. and in much of the developed world—means that ~33% of DTEE is represented by PAEE, and about half of that is accounted for by MVPA (Butte et al., 2012). Thus, approximately 15-17% of average daily DTEE is accounted for by MVPA activity, and this proportion is much higher in exercisers and for some occupations (see McCurdy, 2000).

MVPA data, combining MPA and VPA activities, are displayed in Table 24. A separate dataset, available from the author, provides partitioned MPA, MVPA, and VPA estimates, so it shows more detail than what is depicted in Table 24. No manner what data are provided, however, problems remain about how to measure MVPA time in free-living individuals, and how to compare the subsequent estimates using one method to one using another approach. The traditional—and subjective--methods that have been used in the past to estimate time spent in MVPA are surveys and questionnaires. More recent methods are observational studies, heart rate monitoring, and placing motion sensors on subjects. There are many different types of motion sensors that have been--and are being used to estimate MVPA time, including accelerometers and pedometers (and variants of them, including a combination of these instruments). These approaches and others are discussed later in this Section. As we shall see, it is difficult to relate MPA and VPA obtained via one method to those obtained by another approach even in narrowly-defined age/gender cohorts.

Before discussing differing approaches to estimating MVPA, we delve into officially recognized PA "standards" and "guidelines" recommended by governmental and organizational entities designed to promote healthy physical activity behaviors and practices. Doing so puts the measured data seen in Table 24 into perspective.

Alternative Recommendations for Moderate and Vigorous Physical Activity

Describing the intensity, duration, frequency, and pattern of PA is the "holy grail" of the exercise physiology field (Pate et al., 2010). Of particular importance to that discipline is to fully describe a population subgroup's moderate and vigorous physical activity due to the health benefits associated with PA (Pate et al., 2002). One reason why this is important is to evaluate a person's or a group's adherence to recommended levels of exercise and/or physical activity (Pate et al., 2002). One such recommendation is the American College of Sports Medicine (ACSM) "Quantity and Quality of Exercise for Developing and Maintaining Cardiorespiratory, Musculosketal, and Neuromotor Fitness in Apparently Healthy Adults: Guidance for Prescribing Exercise" (ACSM, 2011; Chodizo-Zajko et al., 2009). The 2011 recommendations are an update of previous ACSM guidance issued in 1975, 1978, 1995, and 1999 (Grundy et al., 1999).

Another organization having MVPA guidelines is the National association for Sport and Physical Education (Graser et al., 2009). MVPA to this group is "activity of

Table 22. Alternative quantative metrics of MVPA seen in the EXERCISE PHYSIOLOGY LITERATURE (for adults unless otherwise noted)

Metric	MPA	VPA	Source of Information (See Notes also)
METS-Based Metrics			
"Standard" METS	3.0 - 5.9	≥ 6.0	Numerous sources; see text.
Less stringent ranges	3.0 - 4.9	≥ 5.0	Sallis et al. 1993 (for grade-school children)
More stringent ranges	5.0 - 7.4	≥ 7.5	Slight mod. of Morehouse & Miller (1976)
	4.0 - 6.9	≥ 7.0	Belcher et al. 2010; Van Mechelen et al. 1997
MVPA	≥ 4		Gortmaker et al. 2012; Aaron et al. 1993
MVPA	≥ 4.5		Crespo et al. 2013; Ekelund et al. 1997;

Table 22. Alternative quantative metrics of MVPA seen in the EXERCISE PHYSIOLOGY LITERATURE (for adults unless otherwise noted)(continued)

Metric	MPA	VPA	Source of Information (See Notes also)
MVPA	≥ 4.6		Young et al. 2014 (for a 30 second epoch)
MVPA	≥ 5.5		Ekelund et al. 1997
Oxygen Consumption (VO2) Metrics			
In mL / min - kg	16.5 - 26.6	≥ 24.8	A compromise; see Note 1
Percent of VO2.Max	> 60%		Atomi et al. 1986; Janz 1994
	> 50%	≥ 70%	Livingstone et al. 1992; Maffeis et al. 1995
	30-50%	≥ 50%	Spurr & Raina 1990
	26-50%	≥ 51%	Slight mod. of Andersen et al. 1978
Energy Expenditure Metrics			
In kcal/min-kg	0.085-0.126	≥0.127	A compromise; see Note 1
In kcal/min (females)	3.5 - 5.4	≥ 5.5	Durnin 1987
In kcal/min (females)	4.0 - 5.9	≥ 6.0	Durnin 1982; see Note 2
In kcal/min (males)	5.0 - 7.4	≥ 7.5	Buskirk 1960; Durnin 1967 & 1983
Breathing Rate(VE) Metrics			
In L/min-kg	0.6 - 1.0	≥ 1.1	A compromise; see Note 3
In L/min	20 - 34	≥ 35	Buskirk 1960
Heart Rate (HR) Metrics			
Heart Rate Reserve (Note 4)	51 - 60	≥ 61	Blair & Connelly 1996; Cunningham et al. 1981
Percent of HR.Max	75 - 84%	≥ 85	Lost citation.
Multiple of HR.Rest	1.25 - 1.49	≥ 1.5	Durant et al. 1993; Welk & Corbin 1995
Heart Rate (bpm)	140 - 159	≥160	See Note 5
bpm	120 - 168	≥169	Atomi et al. 1986
bpm	≥ 150		Cunningham et al. 1981
bpm	125 - 176	≥177	Saris et al. 1977
3-5 y children: bpm	120 - 139	≥140	Freedson 1989

Notes:

1. The values show are a compromise between two different conversion factors: 1 MET= 3.30-3.65 mL O2/min-kg and 1 kcal=200-250. While close, the two methods result in different boundary values, which were (essentially) halved in the compromise. Usually a single author will provide only a single, deterministic conversion value (e.g., 1 MET=3.5 mL O2/min-kg); multiple values seen in the Table arise from showing the entire range of conversion factors seen in the physiology literature.

2. Durnin 1982 also provide estimates of MVPA for energy expenditure estimates in units of kJ/kg-min, but they are not presented here.

3. The values shown are a compromise between using two different approaches. One was based on the VE/VO2 ratio (VQ), which varies between 15-49; VQ values of 30-35 are commonly measured. The other approach is based on VE rates between 0.4-3.7 L/min-kg, and then applying commonly seen VO2.Max percentages to this range. The two methods actually produce quite different estimates; the differences were halved for the values shown in the Table.

4. By the Metobolic Chronotrophic Relationship, %HR.Reserve = %VO2.Reserve = %METS.Reserve; see Section 7.

5. Cited in: Armstrong et al. 1990, 1993, 1998; Gilliam et al. 1981; MacConnie et al. 1982; Pels & Geenen 1985; Sallis et al. 1993.

an intensity equal to brisk walking" or higher. Guidelines or recommendations proffered by individual researchers sometimes explicitly define what they mean by MVPA. One researcher defines MPA as walking for 30 min/d and VPA as any activity that "breaks or sweat" or "causes hard breathing" for 20 min/d (Kann et al. 1993, 1996). There also are Compendium-like (Ainsworth et al., 1993, 2000, 2011) definitions that simply are a listing of activities thought by exercise physiologists to elicit MVPA. These listings include domestic, occupational, and sports/recreational activities. See Wilson et al. (1986). Other groups have also promulgated physical activity recommendations, such as the President's Council on Physical Fitness, the American Heart Association (AHA), the American Cancer Society (Byers et al., 2002; Doyle et al., 2006), the US Department of health and Human Services (Brooks et al., 2004; Buchner 2014), the Centers for Disease Control (CDC), and the World Health Organization [WHO] (Chodizo-Zajko, 1997). There also have been joint recommendations made by combinations of some of the above organizations, including ACSM and AHA (Haskell et al., 2007). A discussion of international PA guidelines is contained in Oja et al. (2010). Blair et al. (1992, 2004), Blair & Connelly (1996), Physical Activity Advisory Committee (2008), and Schoenborn et al. (2011) are articles that review how physical activity recommendations have evolved over time.

PA guidelines for particular subgroups of the population exist, particularly for those with special circumstances or existing health problems. An example is PA recommendations for people with arthritis (ACR, 2000). It recommends that they participate in 30 min/d of low-to-moderate PA on 5 days/week. Guidelines for the elderly also exist (Elsawy & Higgins, 2010). PA guidelines for pregnant and post-partum females have also been proposed (Pivarnik & Mudd, 2009).

MVPA guidelines for children recommend more time in exercise than adult-oriented recommendations, as might be expected, but they are even less-precise about what constitutes MVPA! CDC guidelines state that children and adolescents 6-17 y old should undertake 60+ min/day of "age-appropriate" physical activity, and that most of it should consist of aerobic activity. The term age-appropriate is not defined. See: www.cdc.gov/physicalactivity/everyone/guidelines/children.html). Vigorous-intensity PA, otherwise not defined, should be undertaken on 3+ days/week. Likewise, muscle-strengthening PA should be undertaken 3+ days/week, as should bone-strengthening exercises. Both of these activities can be part of the 60+ min/day recommendation. There is no guidance, apparently, on what constitutes MVPA except for two examples: brisk walking (moderate) and running (vigorous). MVPA Guidelines for children have been "translated into pedometer-based steps/day criteria. These guidelines are 11,000 steps/d for girls and 15,000 for boys (Alderman et al., 2012).

Because the point here is to emphasize why adherence to PA guidelines for moderate and vigorous activity is important and not to describe alternative recommendations made by the various groups, we focus on the 2011 ACSM guidance. These recommendations involve four types of exercise: cardiorespiratory, resistance, flexibility, and neuro-motor (ACSM, 2011). Occupational PA is included in the recommendations. Only cardiorespiratory exercise is germane here. Paraphrased "basic recommendations" for it follows:

- Adults 18-64 y of age should get at least 150 min/week of moderate-intensity exercise.
- This can be met through 30-60 min of moderate-intensity exercise on 5 days/week or 20-60 min of vigorous-intensity exercise on 3 days/week.
- One continuous session or multiple sessions of at least 10 min in duration are acceptable in accumulating the 150 min/week (this is analogous to the epoch issue mentioned above).
- In addition, muscle-strengthening exercises should be undertaken on 2+ days/week (CDC, 2010).
- Gradually increasing the intensity, duration, and frequency of exercise is recommended for best adherence and least risk of injury.
- People unable to meet these minimums can still benefit from some activity.

Moderate-intensity is **not** rigorously defined in these recommendations.

CDC has its own recommendations that are a variant on the above, but its thrust is the same. See: www.cdc.gov/physicalactivity/everyone/guidelines/adults.html. CDC's recommendations for older adults (65+) have a similar scope (www.cdc.gov/physicalactivity/everyone/guidelines/olderadults.html).

It should be noted that most people in this country are ignorant of these guidelines (Morrow Jr. et al., 2004). A 2009 paid incentive survey of 10,587 people with a 65% response rate (quite high) indicated that <1% of the respondents were familiar with the 2008 year moderate+ intensity guidelines (Kay et al., 2014). Perhaps because of lack of knowledge, very few people in the United States meet recommended PA guidelines when their activity levels are measured by objective monitoring. In one study of middle-aged adults, 56% of males and only 5% of females met CDC PA guidelines using an accelerometer (Behrens et al., 2011). Adults with intellectual disabilities or overweight/obese adults meet PA guidelines even less frequently (Barnes et al., 2013; Behrens et al., 2011). Mudd et al. (2008) state that only ~23% of U.S. adults attain the CDC/ACSM guidelines.

Children also do not attain PA goals when objectively monitored (Beets et al., 2011). Pate et al. (2006) discuss the role that schools should play in attaining the CDC recommendations for children. Basically these articles allocate the overall MVPA recommendations to physical education (PE) classes, recess, and other school-based opportunities. Their recommendations for elementary school children are 150 min/week of PE, with 50% of it at MVPA for 30+ min/d including 20+ minutes/d at recess on school days (Carlson et al., 2013).

Not many children meet these standards. A pedometer study of children 6-10 y old indicated that they could accumulate more than 30 minutes of MVPA in a 3h active after-school program with a step count of about 4,600 steps. That level of activity would assure that 93% of the children would meet the guidelines (Beets et al., 2012). However, children in the study did not usually attain that number of steps. Using an accelerometer, average time spent in MVPA during the 3 h program was 18.4 ± 11.1 min for boys and 13.3 ± 8.8 min for girls. Only 16.9% of the boys and 6.1% of the girls met the 30 minute daily MVPA step or accelerometer goal in the after-school program alone (Beets et al., 2012). Thus, additional PA outside of the program is needed. Attending active PE classes increased the percentage of both girls and boys meeting PA guidelines: twice as many girls and three times as many boys meet PA goals on days with PE than on those without phys ed. Students meeting the guidelines did not increase out-of-school PA on days without PE, so PA was significantly increased overall on days with PE (Alderman et al., 2012).

As children age, they are less active and have less MVPA time, on average. The percentage of children and adolescents meeting a goal of 60 min/d or MVPA time decreases from 42% in children aged 6-11 down to only 8% of adolescents in the NHANES study (Nader et al., 2008).

The messages in the above paragraphs are that (1) "official" guidelines are generally not explicit in what is meant by MVPA in terms of VO_2, V_E, or energy expenditure (METS or kcal expended), and (2) physical education (PE) classes positively contributes to meaningful MVPA. Although not mentioned in the above material, similar to the PE/recess time difference in MVPA time in children, participating in moderate levels of occupational PA affects whether or not adults meet MVPA recommendations (increasing attainment of the guidelines by about 5-8% overall); see Boslaugh et al. (2005).

Alternative Indicators of MVPA Seen in the Literature

There have been many qualitative and quantitative (objective) indicators of MVPA that have been used over the years in the exercise physiology literature. In general, these indicators are targeted to specific age and gender groups, and sometimes to people with a particular health issue or weight problem. Probably one of the most common qualitative approach to defining MVPA activities is the METS assigned to them in Ainsworth et al. (1993, 1997, 2000, 2011). Alternative MVPA metrics are seen in Haennel & Lemire (2002) and in other papers.

Estimating Non-clinical MVPA

The rest of this Section of the report (1) defines MVPA in a manner that can be related to the CHAD database—or to an improvement thereof, and (2) provides information on how much time people spend time in MPA, VPA, or MVPA activities so that APEX/SHEDS outputs can be evaluated against objectively measured data. None of the information reported in this Section is taken from a review paper, either qualitative or meta-analytic; only data from original articles are discussed here or are included in Table 24.

We begin with an overview of the methods that have been used to estimate MVPA activities. There are many reviews and descriptions of the methods that have been used to assess PA in the general population or in specific sub-groups. The most comprehensive discussions of measuring methods commonly used are contained in textbooks, including Montoye et al. (1996) Measuring Physical Activity and Energy Expenditure and Welk [editor] (2002) Physical Activity Assessments for Health-Related Research. A number of papers contain reviews (or mini-reviews, as they are sometimes called) of PA-measurement methods. These include: Aaron et al. (1993); Armstrong & Welsman (2006) focused on youth behavior; Berlin et al. (2006); Butte et al. (2012); Cauley et al. (1987); Corder et al. (2008); Dollman et al. (2009); Dufour (1997); Going et al. (1999); Healy, 2000; Heath et al., 1993; Intille et al. (2012); Kreshel (2002); LaMonte et al. (2003); Liu et al. (2012); Matthews et al. (2012);Melanson & Freedson (1996); Montoye (1988); Norgan & Ferro-Luzzi (1978); Pate et al. (2010); Reiser & Schlenk (2009); Shepard & Aoyagi (2012); Stanish et al. (2006); Steele et al. (2010); Schutz et al. (2001); Troiano et al. (2001); Trost (2001, 2007); Tryon (2005); Tudor-Locke & Myers (2001);Valanou et al. (2006); Ward & Evans (1995); Washburn et al. (2000); Welk et al. (2000, 2012); and Wilbur et al. (1989). Pate et al. (2010) also provide an interesting discussion of the history of PA monitoring methods that have been used over the years.

General methods used to estimate MPA and VPA in free-living individuals are listed in tabular form below. The information parallels the work of Pate & Sirard (2000). The criterion approaches are considered to have high objectivity and reliability, and include Doubly Labeled Water and Direct Observation. The DLW method is thoroughly discussed in the "Estimating DTEE" subsection above (p. 82). DLW is not suitable for defining MVPA since daily DTEE estimates cannot be disaggregated into individual activity classes without using some other monitoring approach, such as accelerometry or pedometry. Indirect calorimetry that measures VO_2 consumption precisely is of course an objective measurement method, but it is impractical for field work. It therefore is not listed in the Table by Pate & Sirard (2000), nor will it be discussed here. Indirectly calorimetry basically is a criterion method against which many of the other methods (except DLW) are evaluated in the laboratory.

The remaining methods include subjective means of obtaining MVPA data, and include self-reports (mostly questionnaires), interviews, and proxy reports by a third-party in the case of young children and those who cannot provide the needed data on their own. Diaries and "cell phone-like" methods and considered to be more reliable, even though a lot of subjective user-supplied information accompanies this method. Camera-wearing techniques would be included in this category, but there was only one study where this approach was used in the exercise sciences

(mentioned below). The last general category is objective methods: heart rate monitoring, accelerometry, pedometry, and combining objective techniques to provide a more rigorous picture of MVPA activities.

Estimating the Proportion of Population Subgroups that Undertake MVPA

There is a disjunct in the literature when trying to estimate the proportion of any particular population subgroup that undertakes MVPA and at what frequency. This issue is called here the MVPA *participation rate estimation problem*. A threshold issue of course is defining exactly what is meant by MVPA. A related issue is how it is to be estimated. Before widespread use of objective methods, the usual approach to estimating MVPA participation rate was to ask people what activities they participated in during some past time period (yesterday, the last 3 days, last month, etc.), assign a METS value to them, and count the proportion of people undertaking the MVPA METS level of interest (usually 3.0 for MPA and 6.0 for VPA). Sometimes a sweating/breathing hard answer was used for the indicator of MVPA (Kann et al., 1993). In general, age-level MVPA participation rate estimates obtained by these approaches result in very high participation rates—over 50-60% or higher (Casperson et al., 2000), much higher than those obtained using an accelerometer to estimate MVPA rates. In other words, subjective estimates are unrealistically high.

Another problem arises in trying to estimate MVPA participation rates using information from objective methods papers. They rarely provide an estimate of how many subjects actually participated in MVPA, only their mean time for doing so. Some objective studies do compare their subject's MVPA times to the various Guidelines described above, but do so on a time/day basis and not on a day/epoch frequency basis. Thus, a true estimate of MVPA participation rates cannot be ascertained even using "objective data."

Methods of Estimating Physical Activity in Free-living Individuals

Pate & Sirard (2000) provide a succinct overview of common "field" PA monitoring approaches used currently and in the past; it is abstracted here from their paper, supplemented by information contained in Pate et al. (2010). Discussion of each method follows in some detail, starting with direct observation.

Direct Observation Studies

Observational studies of PA include both real-time observations by trained researchers and ex-post reviews of films/tapes made of the subjects. There have been a number of protocols and instruments used for these types of surveillance studies, usually focused on a particular cohort and/or a specific location. For instance, trained observers have used the "Observational System for Recording Physical Activity" (OSRPA) in a number of studies, especially the version used for preschool-aged children (OSRPA-P) (WH Brown et al., 2006, 2009; Hustyi et al., 2011, 2012; Kahan et al., 2013; Pate et al., 2008, 2013). Another frequently used platform is the System for Observing Play and Leisure Activity (SOPLAY) (Findholt et al., 2011; Floyd et al., 2011; Spengler et al., 2011). There is a "Behaviors of Eating and Activity for Child Health (BEACHES) protocol used by a number of researchers (Nader et al., 1995; Pate et al., 2010; Sallis et al., 1995). SOFIT, or "System for Observing Fitness Instruction Time," was originally used for investigating PA in gym classes (Capio et al., 2010; Heath et al., 2006; Keating et

Overview of PA Field Monitoring Methods

Measurement Method	Costs/Training Factors	Strengths	Limitations, Other Considerations
1. Objective (Criterion) Approaches			
Doubly Labeled Water (DLW)	Expensive; limited supply of material	A direct marker of EE: physiological processes	Provides only average multi-day estimates of energy expenditure
Direct Observation	Costly, requires extensive training	Can observe PA patterns; nuanced	Some subject reactivity; limited to small samples & only a few locations
2. Subjective Approaches: Surveys, Interviews, & Questionnaires			
Self-reported	Relatively low cost	Large sample size	Recall error; low validity; cannot capture PA patterns
Proxy Report	Moderate cost; used for children & those with cognitive issues	As above	As above; proxies do not have full knowledge
Interview (direct or by phone/computer)	Moderate cost; requires training	Moderate sized samples	Recall & validity are better; can provide verbal prompts
3. Conterminous Diary			
Paper diary or fill-in computer form	Moderate cost; need data coding/ QA	Can obtain multi- day PA patterns	High subject burden; re-activity increases with time
4. Objective Approaches (some type of motion sensing)			
Heart rate monitoring	Costly; need clinical facility/daily contact	Good at group level; small sample sizes	HR is non-linear with EE; subject compliance & Equipment issues

Overview of PA Field Monitoring Methods (continued)

Measurement Method	Costs/Training Factors	Strengths	Limitations, Other Considerations
Pedometer	Relatively low cost weekly contact needed	Large sample sizes; can get PA patterns with some models	Highly variable steps-to-EE relationships in individuals
Accelerometers	Moderate cost; weekly contact needed	Can get PA patterns	Have count-to-EE problems; subject compliance issues
5. Multiple instrument Approaches			
Smart Phone/similar device with GPS & accelerometers	Moderate-high cost; privacy issues	Can "automatically" obtain multi-day patterns/locations	Need provider cooperation & approvals; can use Apps to obtain additional info.

Abbreviations:	
EE:	Energy expenditure
GPS:	Global positioning system
PA:	Physical activity
QA:	Quality assurance

al., 1999; Levin et al., 2001; McKenzie, 2002; Scruggs, 2007; Skala et al., 2012; and Smith et al., 2014). SOFIT has been modified for estimating PA during recess (Springer et al., 2013). That version is known as SOFIT-R, but there also is a C-SOFIT, a more computerized version of the basic approach (Huang et al., 2012; Scruggs et al., 2003, 2005a, b). Another surveillance method is known as the "Systematic Observation of Play and Recreation in Communities" (SOPARC), which has been used in multiple communities over a number of seasons (Floyd et al., 2002; Kaczynski et al., 2013; Price et al., 2012; Reed et al., 2011; Ward et al., 2014). Another direct observation approach often used focuses on the Children's Activity Rating Scale (CARS); one example study is described in Puhl et al. (1990). There are other papers on CARS, but it is difficult to translate the scale into energy expenditure estimates. (Actually this is a problem with all observation studies.)

Many of these types of studies use an underlying system of assigning PA intensity to an observed behavior (e.g., Fales, 1938; Harrison & Kielhofner, 1986; Hovell et al., 1978; Kelder et al., 2005). Probably the most used system is the "Children's Activity Rating Scale" (CARS), which classifies all PA activities into 5 intensity levels, to which a METS estimate is then assigned (R. Li et al., 1995; Pate et al., 2010; Robertson et al., 1999). An analysis of one of the classification scales versus accelerometer monitoring indicates that there is a lot of overlap in the scale metrics, so they are not unique or precise (Floro et al., 2009). On occasion, observation studies of school-based PA utilize an accelerometer that is issued at the beginning of school day or PE class and collected at the end of the day or class. Carlson et al. (2014) is one such study, but there are others: e.g., Eaton (1983). MVPA data from these studies are not compiled in Table 24 as they are not collected on a full-day basis. Most studies of PE classes find that a minority of time is spent in MVPA; Skala et al. (2012), for instance, find that 38% of PE class is spent at MVPA levels. They also find that MVPA in PE class time is higher outdoors than indoors. Another study that finds low levels of MVPA in PE class is Sleap & Warburton (1996).

In general, observation studies are short in duration and confined to a single, well-delineated location (Anthbamatten et al., 2011; Berman et al., 1998; Bower et al., 2008; Brown et al., 2006, 2009; Burchfield et al., 2012; Chin & Ludwig, 2013; Chung-Do et al., 2011; A Cohen et al., 2014; DA Cohen et al., 2011; Colabianchi et al., 2011; Epstein et al., 1984; Fitzhugh et al., 2010; Hayes et al., 2008; McKenzie, 1991, 2002; McKenzie et al.,1991, 2000; Nordstrom et al., 1998; O'Hara et al., 1989; Reynolds et al., 2007; Sacheck et al., 2011; Sallis et al., 1988; Scruggs, 2007; Sirard et al., 2005). Example locations are day care centers, school yards at recess, school gyms, playgrounds, basketball courts during practice and/or games, trails in a park or urban greenway, work sites, and soccer fields during a game (Nicaise et al., 2012; Sacheck et al., 2011). A few observational studies occur at home (Eck et al., 1992; Elder et al., 1998; Nader et al., 1995). One observational study of 8 y old children that included many locations for a maximum of 4 h on 3 days is described by Bailey et al. (1995). A *post hoc* attribution of VO_2 to the observed activities was undertaken, which were then classified as low, moderate, or high PA. For both genders, 19.7% (± 3.8) of the observation time was spent in MPA and 3.1% (± 1.0) was spent in VPA (Bailey et al., 1995). The vast majority of VPA occurred in very short "bursts"; 95% of these activities lasted less than 15 sec (Bailey et al., 1995). The overall percentages of M/VPA are much higher than those seen in the Arroyo data reproduced in Table 25. Because observational studies do not include the entire day or all locations, MVPA data are not provided in Table 24 from these studies, even if an accelerometer or pedometer is used during the observation period to characterize MVPA (e.g., Bruggeman, 2006; Mukeshi et al., 1990; Sacheck et al., 2011). The Mukeshi et al. (1990) article indicates that the correlation between direct observation and Caltrac accelerometer monitoring was $r=0.62$ ($p<0.001$) in young children aged 35.1 ± 3.0 months.

One long-term (3 years) observational study investigated whether or not "tracking" of exercise occurred in children aged 4 y at the beginning of the study (Sallis et al., 1995). Subjects were observed twice at home and school every 6 months. Only 15% of home-based PA and only 8% of

school-based PA was considered to be stable over time (i.e., could be attributed to tracking). This implies that PA cannot be adequately described by single-point-in-time observations (Sallis et al., 1995).

An observation study of a different type involved putting a camera onto a subject, and having a third-party observer translate the stored pictures into PA categories. This protocol was tested on a few people using a "SenseCam" device by P.Kelly et al. (2011) in the United Kingdom, but it was found to be very expensive and burdensome.

Subjective Surveys, Questionnaires, and Interviews (Telephone or Face-to-Face)

There are five main approaches of obtaining subjective PA estimates: surveys or questionnaires, phone interviews, proxy reports concerning someone else's PA, paper or electronic diaries, and using recording devices—such as a digital assistant or "smartphone" that can provide (some) PA information "automatically" (Sternfield & Goldman-Rosas, 2012). Using a smartphone borders on objective data-gathering, and will be discussed later. The first three methods provide only retrospective (recall) information on PA, while the latter two methods can provide data on contemporary, event-based PA activity level (Garg et al., 2006). The first three approaches, like direct observation studies, require some type of subjective mapping of *activity level* onto an action or behavior to 'translate" the activity undertaken into an energy expenditure estimate (Fales, 1938). There literally are thousands of papers and reports describing studies that develop and describe subjective estimates of PA, including MPA and VPA, using one of the translation approaches. Most of them use questionnaire data for their estimates.

None of the MVPA data obtained via subjective methods involving data provided by the subjects themselves—surveys and questionnaires—will be presented in this report. One reason is that comparisons of alternative questionnaires on the same population provide wide estimates of exercise time in the population surveyed (Slater et al., 1987). For another, there simply are too many "validity" comparisons of subjective and various objective methods that show wide differences in the amount and type of PA estimated by subjective approaches versus objective monitoring, even for a recent time period (e.g., Loney et al., 2011; Másse et al., 2012; McMurray et al., 2008; Parker et al., 2008; Washburn et al., 2003; Westerterp 2009; Wickel et al., 2006; Wong et al., 2006; Yore et al., 2007; Zalewski et al., 2009). Westerterp (2009) succinctly states that questionnaire data have low reliability and validity. This finding applies even though subjective/objective comparative studies find that the subjective approach supposedly provides reliable and valid MVPA data. The statistical comparison metrics used in most comparative evaluation papers are weak and misleading (Ayen & Montoye, 1998; Ball et al., 2008; Colbert & Schoeller, 2011). Often only a Pearson correlation coefficient (r) is used to relate survey results with METS or MVPA estimates from an accelerometer or other objective technique, with r's in the 0.3-0.5 range for 1-to-7-day comparison periods (Ainsworth et al., 2006; Beyler et al., 2008; Burdette et al., 2004; Másse et al., 2012; Miller et al., 1994; Pate et al., 2003; Senso et al., 2014). Absolute differences in the PA estimates obtained by the two approaches often are ignored. This complaint generally holds true whether the metric being compared is a "count" (accelerometer / pedometer), total minutes of MPA/MVPA, or VPA, or energy expenditure metrics (Aadahl and Jørgensen, 2003; Welk et al., 2014). In addition, these "validity" studies do not normally compare estimated bout *frequencies*, and are entirely silent regarding PA *patterns*. For example, one study that concluded that a subjective approach provided valid data was Epstein et al. (1996). It included 59 children of both genders and compared self report-derived average-daily METS estimates of total PA versus that obtained wearing a Tritrac R3D accelerometer. The main finding was that self-reported daily-averaged METS was 2.26 ± 0.64 versus the 1.60 ± 0.18 actually measured, about a 30% self-reported overestimate (Epstein et al., 1996). In another evaluation study of a questionnaire—the often used 3-Day Physical Activity Recall (3DPAR)—found that only 10% of women with breast cancer met the current PA guideline using an accelerometer, but 28% did so using the 3DPAR (Johnson-Kozlow et al., 2007). Differences that large are not comparable in my estimation. In general, surveys and questionnaires consistently over-report MVPA in overweight (and especially obese) individuals (Welk et al., 2014).

The discussion and conclusion sections of many of these "validity" studies are disingenuous and often do not support their own findings. These evaluations generally conclude that that the survey/questionnaire produces reasonable **correlations** of time spent in PA as compared to some type of objective methoid (Welk et al., 2014). Infrequently, more sophisticated statistical approaches are used in these comparisons, such as plotting the data in Bland-Altman plots or using "receiver operating characteristics curves" to evaluate the subjective method (Aadahl and Jørgensen, 2003; Marshall et al., 2009). These more sophisticated approaches also show large differences in the estimates of MVPA obtained using subjective versus objective methods (Másse et al., 2005).

As an aside, Másse et al. (2005), besides evaluating a PA questionnaire and a PA diary against accelerometer data, also compared the Compendium METS estimates with accelerometer-derived activity-specific METS values. The Spearman *r* for the two approaches was only 0.31, but significant at 0.05, while the absolute difference was 1,500 METS-minutes/d on average. See also Másse et al. (2002) and Másse et al. (1999) for similar analyses and findings.

Common surveys/questionnaires used are the Three-Day Physical Activity Recall (3DPAR), the Seven-Day PAR (7DPAR) (SA Adams et al., 2005; Csizmadi et al., 2014), the Physical Activity Scale for the Elderly (PASE; Chad et al., 2005), the Godin Long-term exercise Questionnaire (Andrykowski et al., 2007), the National Children and Youth Fitness Study (NCYFS), the International Physical Activity Questionnaire (IPAQ), the Behavioral Risk Factor Surveillance System (BFRSS) (BFRSS Coordinators,

1995, 2000, 2007; Casperson & Merritt, 1995), the National College Health Risk Behavior Study (DR Brown & Blanton, 2002), the National Health Interview Survey (Casperson et al., 1986, 2000), and the Youth Risk Behavior Study (YRBS) (Bauman et al., 2009; DR Brown et al., 2007; Demissie et al., 2014; Ottenbacher et al., 2014; Pate et al., 1994). Both the BFRSS and the YRBS are nationally-applicable random-probability surveys that are undertaken every five years or so, and include other health-related factors besides the MVPA questions (DR Brown et al., 2005). The National Health and Nutrition examination Survey (NHANES) in 2003-2006 had a component focused on PA that used the ActiGraph 7164 accelerometer to measure MVPA, so this major health survey also provided objective MVPA data (Loprinzi et al., 2014a, b, c). There are many other surveys and questionnaires used to estimate MVPA time, many used only one time by a single research group (Breslow et al., 2001; Casperson et al., 1994, 1998; Prince et al., 200; Sallis et al., 2000).

Occasionally, surveys and/or questionnaires are used to estimate retrospective PA as far back as 10-20 years (Bowles et al., 2004; Dawson et al., 2003). Although most of these studies are impossible to evaluate statistically, they generally are felt to **not** produce either accurate or unbiased results (Corder et al., 2009; Corder & van Sluijs, 2010; Dawson et al., 2003; Garcia-Rio et al., 2012). Goran et al. (1998) state that only 50% of a child's PA is correctly recalled after a week. Proxy surveys, usually involving a parent or care-giver estimating the amount and type of PA undertaken by their children, have also been shown to be inaccurate (Coder et al., 2012). Parents overestimate the amount of PA undertaken by their children even when the metric simply was "active" (>60 min/d of MVPA) versus "inactive." The rate of PA was overestimated on 75% of the days that were evaluated (Corder et al., 2012).

Some important insights into PA in various groups of people can be only obtained using questionnaires. One study used the same survey 7 times over a 14 year period to estimate how PA changed in children over the years. It provides information on the partitioning of variance among and within individuals involving PA (Ridley et al., 2009). It used the MARCA (Multimedia Activity Recall for Children and Adolescents) 7-day questionnaire, and one aim of the study was to characterize the amount of intra- and inter-individual variability seen in daily PA over time, using the ICC statistic. The inter-individual COV for MVPA was 11.7%, while the intra-individual COV was 14.5%. Using PAL instead of MVPA, the inter-individual COV for PAL was 52.0% while the intra-individual COV was 83.4% (Ridley et al., 2009). Thus, intra-individual variability was greater than inter-individual variability for both PA metrics.

Questionnaire data have been gathered that confirm that physical activity declines in winter (in temperate areas) and on days with bad weather. Specifically, it was found that PA decreased 2-4% for every 10 mm of rainfall and increased 1-2% for every 10^{o}C increase (Bélanger et al., 2009). Although the specific proportions noted probably vary by climate, these results are intuitive, and are consistent with that found in the more general time use data (Graham & McCurdy, 2004).

Another reason why using data from questionnaires--especially telephone surveys--is problematic, is that response rates are dropping precipitously. People just are not returning mail surveys, and telephone surveys have very poor response rates due to cell phone use and poor participation by the general public having only a land line (Kempf & Remington, 2007). People don't answer their phone anymore when Caller ID displays an unknown number. Even without Caller ID, the high number of solicitation calls received by the average household has made people wary of answering their phone during "prime" survey times. The subsequent low response rates cause a bias in the data obtained, thus questioning the validity of data from telephone surveys (Kemp & Remington, 2007).

PA Diaries (Paper or Electronic)

Filling out a paper or electronic diary carried by a subject to record MVPA has been undertaken since at least 1965 (Bouchard et al., 1983; Huenemann et al., 1967). Exercise events are supposed to be recorded as they occur (Gleeson-Kreig, 2006; Matthews, 2002; Qian et al., 2014; Schwab et al., 1990, 1991; Sternfield et al., 2012; Washburn et al., 1990; Whitt et al., 2004; Wickel & Eisenmann, 2006), much like a conterminous time use diary. Both place a fairly high burden on the subject to compile the information, and so are used only for relatively short periods of time. Reactivity also is a problem, where the act of recording an activity affects the data quality (Matthews, 2002). Originally, only paper diaries were used, but electronic data-storing devices have been used since the 1990's, especially the Palm Digital Assistant (Yon et al., 2006). Diaries are not to be confused with a PA "log," which generally is filled out at the end of a day (or longer elapsed time span) and uses fairly broad categories of activity, such as walking, standing, and running (Buman et al., 2011; Garcia et al., 1997; Kaczynski et al., 2011, 2012; Matthews, 2002). Essentially, an activity log shares many of the issues associated with a survey or questionnaire, but with a shorter time lag between the PA and its record.

An interesting study of a PA diary is contained in Baranowski et al. (1999). Study subjects were 165 elementary school teachers who carried a PA diary for 7 days once each year for 3 years. The ICC statistic (assuming three different variance structures) was used to estimate how many days of PA data needed to be collected for a Spearman-Brown "prophesy" formula reliability coefficient of 0.7, 0.8, or 0.9. To achieve a 0.8 coefficient that EPA used in its ICC work in the past (Xue et al., 2004) requires that 2 weeks of 7 d PA activity records be collected every year (Baranowski et al., 1999).

Even though we do not provide data in this Report from PA diary surveys, a partial listing of papers describing USA studies follows if a reader wants to pursue additional information on the topic. There are a number of 7-days studies: Cummings & Vandewater (2007); Eason et al. (2000a, b); Dishman et al. (1992); Evenson & Wen (2010); Garcia et al. (1997); Katzmarzyk et al., (1998); Kerner &

Kurrant (2003); King et al. (2008); Sallis et al. (1988); and Schwab et al. (1991). Shorter 3-day USA diary studies are Bouchard et al. (1983) and Eisenmann et al. (2000).

There are a number of diary studies of MVPA undertaken in other countries that are not further analyzed. For example, see Bratteby et al. (1997) and Freene et al. (2011). We also do not include any PA data obtained from the broader "time use" field due to its lack of focus on activities associated with differing levels of energy expenditure, and especially MVPA. Most time use studies that classify physical activity use too few categories (e.g., Robinson et al., 1988, 1989, 1999; Robinson & Thomas, 1991; Wiley et al, 1991), that are then assigned into MVPA classes or to a METS estimate (Tudor-Locke et al. 2008, 2011a, b). Unhappily, EPA's CHAD database has the "too-few PA categories" problem, since it was based largely on Robinson's prior time use studies. In a few instances, however, a "traditional" time use diary survey has been analyzed to estimate physical activity participation. One such study in the United Kingdom is reported in Fisher (2002). The same approach was undertaken in this country using the American Time Use Survey (ATUS), an *ex post* sequential diary (event) study. Data from ATUS surveys from 2003-2014 are available on the web from the Bureau of Labor Statistics. A deterministic METS code was mapped to every ATUS activity code and used to identify MVPA activities (described in Tudor-Locke et al., 2009). A number of papers have used these PA codes to determine how much physical activity occurs in the general population at various intensities, including MPA and VPA. Papers describing these analyses are Dunton et al. (2008, 2012) and Tudor-Locke et al. (2010, 2011a). None of the data from these studies are included in Table 24 due to subjective nature of how the METS codes were developed and applied.

Objective Monitoring Methods: Overview

A review of the history of objective monitoring techniques used to estimate physical activity in humans is contained in Montoye (1988), a French paper with an English summary. Most of its references are in English. The article covers pedometers, force plates in shoes, and many different types of accelerometers. A version of a pedometer was first developed by Leonardo de Vinci over 500 years ago. Since then, of course, there have been many innovations in design and manufacturing of pedometers that have made them smaller and cheaper so that they are the least expensive way to estimate the number of steps taken by an individual during their waking hours. Force plates in shoes, a 1950's invention, were essentially another step-counter; this technique is not currently mentioned in the literature. Early accelerometers include (1) a long-term movement integrator (LSI) developed in the 1950's and located on a subject's wrist (FG Foster et al., 1978); (2) a 1970's "biometer," no longer seen; (3) single-plane accelerometers developed in the early 1960's and still used, sometimes called an "actometer" (Buss et al., 1980); and (4) a tri-axial accelerometer, which was first developed in the 1970's (Halverson & Waldrop, 1973). The main improvements in accelerometers over the years relates to their reduction in size and weight, and their ability to store ever-increasing amounts of data for longer periods of time (Freedson & Miller, 2000; Montoye, 1988). Another technique that Montoye (1988) mentioned was a radar detector that transmitted data back to a central station. An other review of objective monitoring over the years was published by Butte et al. (2012). It mentions "six main categories" of PA objective methods, including load transducers and foot-contact monitors, which are not mentioned in this report. Other reviews of objective monitoring in general include Chen et al. (2002), Prince et al. (2008), and Schuna Jr., et al. (2013a).

We divide objective monitoring methods into 4 major categories: heart rate monitors, accelerometers, pedometers, and multiple-instruments. We briefly discuss each in turn. Intille et al. (2012) provide insightful information regarding the temporal trend of PA sensor development, and speculate about what changes in sensors may be anticipated. Freedson et al. (2012) make recommendations regarding how PA monitors should be calibrated and used in the field, as does Bassett Jr. et al. (2012). Chen et al. (2012) develop recommendations regarding what information regarding sensors should be obtained by PA researchers before making a decision regarding what type of monitor should be used in a study. Heil et al. (2012) do much the same thing.

Heart Rate Monitoring

HR monitoring has long been used to assess PA (Achten and Jeukendrup, 2003; Gilliam et al., 1981; Glagov et al., 1970; Goldsmith & Hale, 1971). Benedict proposed the use of HR monitoring to provide an indirect estimate of EE in the early 1900s, and monitors to do so were developed in the 1950s (Janz, 2002). In the past, HR monitoring was done using a portable electrocardiogram that stored HR data, but this approach was replaced by a chest strap monitor that transmitted data to a nearby receiver, oftentimes located on the subject's wrist (Janz, 2002; Pate et al., 1996). There are a number of HR monitors available commercially.

No data using the HR monitoring method are provided in Table 24 because of problems with subject compliance issues, ambient interferences, and equipment failures. Also, HR is non-linearly related to VO_2 at different PA levels and activities, so reliability is an issue (RB Andrews, 1971; Christensen et al., 1983). Thus, HR monitoring is non-linearly related to energy expenditure, V_E, and MVPA levels (and time spent at different levels).

Many studies comparing HR estimates of EE with those measuring VO_2 directly found large differences in group mean EE estimates—and even larger differences in individual estimates of EE. These studies include Allor & Pivarnik (2000), Daucey & James (1979), Emons et al. (1992), Eston et al. (1997), Livingstone et al. (1992), Lovelady et al. (1993), Luke et al. (1997), Morio et al. (1997), Rachette et al. (1995), Schulz et al. (1989), and Spurr et al. (1988). Other studies that compared HR monitoring to VO_2 measurements for specific activities found that HR estimates were within 10% or so of the group-mean VO_2 values (Bradfield et al., 1969; Ceesay et al., 1989; Maffeis et al., 1995; McCrory et al., 1997; Moore et al., 1997; Strath et al., 2000; and Treiber et al., 1989). In most cases, the correlation between HR and

VO_2 was high, even when the absolute differences were quite large. It depends upon the intensity level of the PA, personal fitness level of the subject, and—problematically—their emotional state at the time. These factors negatively affect reliability and validity of heart rate monitoring. For an evaluation of HR monitoring versus the Caltrac accelerometer, see Allor & Pivarnik (2001). There are other comparisons and reviews of HR monitoring versus other objective methods that might be of interest: Cole & Miller (1973); Corder et al. (2007); Davis et al. (1971); Drenowatz & Eisenmann (2011); Dugas (2005); Edmunds et al. (2010); and Epstein et al. (2001). For additional information on HR monitoring, see Appendix B.

An associated problem with HR monitoring—which actually affects most objective monitoring approaches—is to determine what constitutes a threshold for moderate or active PA. Often a HR value of 160 bpm is used for "strenuous" activities (Armstrong et al., 1991; Harro, 1997), but this value is greatly affected by age, gender, and HR reserve of an individual. A HR of 160 bpm corresponds approximately to 60-70% of HR reserve in "normal" children aged 7-12 (Al-Hazzaa et al., 1994). Using a percentage of HR_{MAX} of 65-75% as an indicator of MPA and >75% for VPA in children aged 4.3 ± 0.7 y, Benham-Deal (2005) found that about 20% of their time was spent at MVPA on average. There were no statistically significant differences in this percentage between weekdays and weekends or among the morning, afternoon or evening time periods (Benham-Deal, 2005). Several weekdays and weekends should be monitored using a HR method to obtain a representative daily average PA estimate (Gretebeck et al., 1991).

Accelerometers

Principles and Overview

Acclerometers utilize a coupling seismic mass suspended on one or more levers that deflects upon movement. Piezoresistors on each bridge respond to this deflection and a current proportional to the displacement is induced and processed. The signals are filtered to limit the sensor to frequencies that are associated with actual body motion. In general there are uniaxial and triaxial accelerometers. A good discussion of the principles behind accelerometry is contained in Servais et al. (1984) and Welk (2005).

The first article that I found that used the term accelerometer is Smidt et al. (1971). It cites 8 previously-published articles that used accelerometry to estimate walking "kinematic and kinetic information," but I did not follow up on them given the narrow activity that was being monitored, all in a clinical setting. Other early articles on accelerometry are Kupfer et al. (1972), Johnson (1971), and MacCoby et al. (1965). Kupfer et al. (1972) discusses a uniaxial accelerometer that transmits movement data to a receiver having a 100 foot range; it was used to monitor mental patients. Morris (1973) discusses how using 6 well-placed uni-axial accelerometer can be used "to completely define a person's movement in space." Thus, in general, accelerometry was being discussed in the early 1970's. So, the idea of accelerometry has been around for a rather long time.

Protocols have been developed and used to calibrate and "validate" accelerometers to provide "best practice" approaches to undertaking research in exercise science (Bassett Jr., et al., 2012; Evenson et al., 2008; Freedson et al., 2012). See also: Bassett, Jr. (2000); Bray et al. (1992, 1994); Chen et al., (2005); Eslinger et al. (2005); John & Freedson (2012); Kelly et al. (2013); Khan et al. (2010); Labyak & Bouguignon (2002); McClain et al. (2009); Meijer et al. (1991); Redmond & Hegge, 1985; Schaefer et al. (2014); and Troiano (2006 & 2007), Troiano & Freedson (2010), Troiano et al. (2012). P.J. Trost is the first author on many overview and application articles related to accelerometers (and pedometers); see for example, Trost (2001), and Trost et al. (2000, 2001, 2005).

The literature on using accelerometers to estimate physical activity levels is vast and growing. See Table 23 for an example of the increase in publications for the ActiPAL accelerometer, which is not the most frequently used one in the exercise physiology literature. There are hundreds of papers on accelerometers in general, many "validating" different accelerometers against other objective methods or doubly labeled water (e.g., Hageman et al., 2004). A few

Table 23. Number of publication for the ActiPAL ACCELEROMETER by year of publication

Year	Journal Papers	Reports / Theses	Conference Presentations
2004	1	0	8
2005	0	1	11
2006	4	1	4
2007	11	1	15
2008	8	0	1
2009	12	1	7
2010	25	14	28
2011	31	3	58
2012	38	3	57
2013	27	4	60

Source: ActiPAL website: *www.paltechnologies.com/bibliography*

accelerometers have even been used as a "criterion method" against which other accelerometers or pedometers have been compared.

The most frequently used accelerometer in exercise studies of "free-living" individuals in this country is the ActiGraph monitor. There are various models of this instrument, including the 7164, GT2M and GT3X. Before it was known as the ActiGraph 7164, it was called the MTI 7164, and before that it was known as the AM-7164 or the CSA 7164. There also is an ActiGraph 71256 model used infrequently (Buman et al., 2010). The most recent version of the ActiGraph series of accelerometers is the GT3X and GT3X+ (Feito et al., 2012; Rowlands et al., 2015). To confuse matters more, the ActiGraph accelerometer is available in a uniaxial and triaxial version (Oftedal et al., 2014).

The second most widely used accelerometer in the U.S. is the Mini Mitter Company's Actical (Evenson et al., 2008). The Actical accelerometer is not the same as the ActiGraph or any of the other "Acti-"models; similar names are a problem with distinguishing among accelerometers! Additional accelerometers (or hybrid accelerometer/pedometer units) mentioned in the literature include the Actibelt, ActiPAL, ActiPed, Actiwatch, ActiReg, ActivTracer, Actimarker, Biotrainer-Pre, Calcount, IDEEA, DynaPort, CSA1, the IMS (Integrated PA Monitoring System); Kenz, Tracmore, GENEA, GENEActiv, PASE (a PA-sensing earpiece [Manohar et al., 2009]), Polar Activity Watch, SenseWear (SWA & Mini) and TRACMORE accelerometers (Balogun et al., 1988; Barreira et al., 2013; Bassett Jr. et al., 2000; Bassett Jr. & Strath, 2002; Benito et al., 2011; Bjornson, 2005; Bonomi et al., 2010; Bornstein et al., 2011a; Brazeau et al. 2011a,b; Brugniaux et al., 2010; Busser et al., 1997; Calabró et al., 2009; Conn et al., 2000; Egger et al., 2001; Eslinger et al., 2007, 2011; Hayden-Wade et al., 2003; Hikihara et al., 2012; Hildebrand et al., 2014; Huberty et al. 2011a; Hustvedt et al., 2008; Jerrett et al. 2013; John et al., 2011; Kavanaugh & Menz, 2008; McCrorie et al., 2014; Motl et al. 2012; Nightingale et al., 2014; Patel et al., 2007; Roemmich et al., 2007; Welch et al., 2014; Welk et al., 2014).

There are a number of comparative studies of accelerometer performance. They have been compared against different models of the same brand, different brands, against VO_2 and HR monitoring, and against EE (via indirect calorimetry. These comparison studies have found a number of issues of accelerometer performance, not the least of which are big differences found in accelerometer performance seen within the same manufacturer's model. Accelerometers frequently have high inter-instrument variability, in other words (Hollowell et al., 2009). Instrument continuity over time also is a problem as accelerometer manufacturers change their models often (perhaps after a bad review of their performance?), and subsequent models sometimes compare favorably with their predecessors from the same manufacturer but other times do not! For instance, a comparative study of performance in three Actigraph accelerometers (the GT1M, GT3X, and the GT3X+, found close agreement both in the lab and in the field in their total counts of activity (Robusto & Trost, 2012). On the other hand, a direct comparative study of two ActiGraph models, the 7164 and the GT3X, researchers found that the two versions do not produce comparable step counts or estimates of MVPA time (Cain et al., 2013a). In fact, the same researchers reviewed 273 articles that used ActiGraph accelerometers to estimate physical activity in youth and concluded with this disheartening note:

> Studies using [the ActiGraph] accelerometer more than doubled from 2005-2010. Two accelerometer models were used, as was 6 epoch lengths, 6 nonwear definitions, 13 valid day definitions, 8 minimum wear day thresholds, 12 moderate-intensity physical activity cut points, and 11 sedentary cut points…The increasing diversity of methods used to process and store accelerometer data for youth precludes comparison of results across studies. Decision rule reporting is inconsistent, and trends indicate declining standardization of methods [Cain et al., 2013b; p. 437].

Some studies have also compared accelerometer counts/minute from different body locations on the same people, or compared the step-counting function of some accelerometers versus using a pedometer on the same people. Usually these studies are laboratory experiments, but some are field-based in free-living subjects. For studies that evaluate the validity and reliability of accelerometers, see Ayen & Montoye (1998), Balogun et al. (1998, 1989), Barriera et al. (2009, 2013), Bassett Jr. et al. (2000), Bouten et al. (1994, 1996), Eston et al. (1998), Fehling et al. (1999), Feito et al. (2012), Haymes & Brynes (1993), Janz (1994), John et al. (2011), Kilanowski et al. (1999), Leenders et al. (2000); Louie et al. (1999), Maliszewski et al. (1991), Matthews et al. (2000), Melanson & Freedson (1995), Pambianco et al. (1990), Sallis et al. (1990), Schutz et al. (1987), Swartz et al. (2000), Trost et al. (1997, 1998), and Welk et al. (1998, 2000). This is just a sampling of validity/reliability articles; many more could be cited.

Comparative studies rarely provide MVPA data of the type that we need in free-living people, so they are not often included in Table 24. Additional comparisons of accelerometers are contained in Beets et al. (2011), Bouchard & Trudeau (2007), Cliff & Okely (2007), Fischer et al. (2012), and Lee et al. (2014). Those studies that present MPA/VPA data only as a % of total valid wear time, and said wear time could not be determined, are also not included in Table 24 (e.g., Epstein et al., 2005).

Accuracy of different accelerometer models and brands vary with the type of activity chosen and the level of effort expended. Some are more accurate at moderate or vigorous activities, while others are more accurate at low intensity tasks. In other words, accelerometer estimates of PA are not linear over the entire gamut of human activities that are undertaken, and neither are the various regression equations developed to translate movement counts to energy expenditure, VO_2, or METS (Bassett Jr., et al., 2000; Freedson et al., 2011JAP; Másse et al., 2005). Walking at different speeds is often the activity of choice in many accelerometer model evaluations since it spans the light PA-to moderate PA spectrum and is easy to monitor (Bassett

Table 24. Estimates of moderate & vigorous (MVPA) physical activity (PA)

PUBLISHED IN THE U.S. LITERATURE							
Age Range (years)			MVPA (min/day)				
Mean	SD	(n)	Mean	SD	Citation	Accelerometer Used (Make/Model #/Protocol)¥	CPM
Females: Normal, Healthy, or Not Specified							
3.7	0.5	21	12.7		Kelly et al. 2007	AG 3d	3200
5.2	0.4	205	31.5	15.9	Francis et al. 2011	AG 7164; 3-4 d	2296
7.1	1.9	64	55.0	28.4	Sarzynski et al. 2010	AG GT1M; 7 d	2172
7.3	0.9	48	206.0		Pate et al. 2013	CSA 7164; 7d	
7.5	0.6		72.4	27.7	Willis et al. 2015	AG GT3x+; 4 d	
7.7	1.1	79	60.4	40.8	HM Hayes et al. 2011	MTI-7164; 7 d	2172
8.0	0.2	35	106.3	41.2	Sherwood et al. 2004	AG 7164; 3 d;1200-1800	
8.1	0.7	63	54.1	19.7	NC Crespo et al. 2013	AG GT1M; 7 d	
8.7	0.6	229	38.6	21.2	Francis et al. 2011	AG 7164; 3-4 d	2296
8.7	1.8	60	28.7	16.8	Beets et al. 2011	AG GT1M ; ≥1d, 5.7h	
8.7	1.8	60	39.6	20.0	Beets et al. 2011	AG GT1M; ≥1d, 5.7h	1952
8.7	1.8	60	14.7	11.0	Beets et al. 2011	AG GT1M ; ≥1d, 5.7h	
8.7	1.8	60	17.6	12.4	Beets et al. 2011	AG GT1M; ≥1d, 5.7h	
8.7	1.8	60	19.6	13.4	Beets et al. 2011	AG GT1M ;≥1d, 5.7h	
8.8	0.9	52	101.0	47.0	Atkins et al. 2004	CSA; 3d, 1200-1800	3200
8.9	0.8	60	83.0	42.9	Atkins et al. 2004	AG 7164; 3 d;1200-1800	
9.1	0.8	61	125.5	61.7	Atkins et al. 2004	AG 7164; 3 d;1200-1800	
9.2	0.9	198	66.5	30.9	Jago et al. 2004	AG 3 d; 24 h	1952
9.2	2.1	423	55.9	26.7	MW Long et al. 2013	AG 7164; 7 d	
9.4	0.9	48	88.0		Hsu et al. 2014	AG GT1M	
9.9	1.1	23	64.3	23.9	Olvera et al. 2010	AG GT1M	1500
10.4	1.1	23	35.4	21.9	Olvera et al. 2010	AG GT1M	1500
10.4	1.0	46	111.0		Pate et al. 2012	CSA 7164; 7d	
10.6	0.6	22	128.7	45.5	Fuemmeler et al. 2011	MTI-7164; 4 d; 24 h	
10.6	0.6	22	138.5	60.1	Fuemmeler et al. 2011	MTI-7164; 4 d; 24 h	
10.7	4.3	230	79.0	57.0	Butte et al. 2007	Actiwatch; 3 d; 24 h	
11.2	0.3	171	26.4	10.9	Francis et al. 2011	AG 7164; 3-4 d	2296
11.2	0.3	184	22.1	13.5	Janz et al. 2007	AG 7164 d	3000
11.7	0.4	229	22.5	16.6	Pate et al. 2006	MTI 7164; 7d	3000
11.7	0.4	204	136.9	83.3	CC.Johnson et al. 2008	ActiGraph	
11.8	0.4	267	167.8	74.1	CC.Johnson et al. 2008	ActiGraph	
11.8	0.4	291	27.1	17.1	Pate et al. 2006	MTI 7164; 7d	3000
11.8	0.5	1559	23.6	11.8	Taber et al. 2011	Actigraph; 7 d	1500
11.9	0.4	984	23.5	11.6	J.Stevens et al. 2007	AG 7164; 6 d	

Table 24. Estimates of moderate & vigorous (MVPA) physical activity (PA) (continued)

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Age Range (years)			MVPA (min/day)				
Mean	SD	(n)	Mean	SD	Citation	Accelerometer Used (Make/Model #/Protocol)¥	CPM
11.9	0.4	216	174.8	96.8	CC.Johnson et al. 2008	ActiGraph	
11.9	0.5	1043	26.9	32.3	Treuth et al. 2007	AG 7165; 7 d	1500
11.9	0.5	1043	19.2	16.1	Treuth et al. 2007	AG 7165; 7 d	1500
12.0	0.3	229	27.7	16.6	Pate et al. 2006	MTI 7164; 7d	3000
12.0	0.4	269	22.9	18.0	Pate et al. 2006	MTI 7164; 7d	3000
12.0	0.4	262	134.6	63.7	CC.Johnson et al. 2008	ActiGraph	
12.0	0.4	175	25.6	77.9	CC.Johnson et al. 2008	ActiGraph	
12.0	0.5	645	25.3	1.1	McMurray et al. 2008	AG; 6 d; 30 sec epoch	
12.0	0.5	1576	23.7	11.7	Young et al. 2014	MTI 7164; 7d	1500
12.0	0.5	1162	99.2	99.9	Saksvig et al. 2007	AG 7164; 6 d	1500
12.0	0.5	112	113.0	41.6	Saksvig et al. 2007	AG 7164; 6 d	1500
12.1	0.5	274	20.8	18.2	Pate et al. 2006	MTI 7164; 7d	3000
12.2	0.7	273	135.7	82.4	CC.Johnson et al. 2008	ActiGraph	
12.3	0.3	65	17.1	20.1	Tucker et al. 2011	Biotrainer; d	
12.3	0.7	286	21.5	18.6	Pate et al. 2007	MTI 7164; 7d	3000
12.6	2.8	1235	18.6	16.8	Mark & Janssen 2009	AG 7124; 7 d	3000
12.8	1.0	48	75.0		Pate et al. 2012	CSA 7164; 7d	
12.8	1.3	471	50.0	24.2	Sanchez et al. 2007	CSA 7164; 7d	
13.3	0.4	168	31.6	18.1	Francis et al. 2011	AG 7164; 3-4 d	2296
13.3	0.6	43	46.9	28.3	Anderson et al. 2005	MTI 7164; 4d 0600-2300	1399
13.3	0.6	43	9.2	6.5	Anderson et al. 2005	MTI 7164; 4d 0600-2301	3200
13.9	0.4	984	22.0	10.8	J.Stevens et al. 2007	AG 7164; 6 d	
14.0	0.9	70	49.4	23.3	Pate et al. 2003	CSA 7164; 7d;0700-2400	
14.0	0.5	3085	22.2	11.2	Young et al. 2014	MTI 7164; 7d	1500
14.1	0.5	1140	21.9	10.9	Lohman et al. 2008	AG 7164; 6 d	
14.5	1.8	289	37.5	21.4	Sirard et al. 2010	AG 7164; 5 d	
14.6	1.8	360	26.7	13.9	Hearst et al. 2012	MTI 7164; 7d	
14.9	1.9	149	16.4	1.5	O'Neill et al. 2011	AG 7164; 8 d	1500
14.9	1.9	149	29.7	1.4	O'Neill et al. 2011	AG 7164; 8 d	1500
15.0	2.9	859	20.6	35.2	MW Long et al. 2013	AG 7164; 7 d	2020
15.5	1.1	48	55.0		Pate et al. 2012	CSA 7164; 7d	
16.2	1.1	104	34.2	19.8	Gutin et al. 2005	MTI 7164; 7d	
16.3	1.2	121	35.7	25.3	Gutin et al. 2005	MTI 7164; 7d	
16.7	1.2	27	38.0	18.6	Sirard et al. 2008	AG GT1M; 7 d	940
31.9	8.7	55	31.9	18.0	Whitt et al. 2003	Actisplit	

Table 24. Estimates of moderate & vigorous (MVPA) physical activity (PA) (continued)

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Age Range (years)			MVPA (min/day)				
Mean	SD	(n)	Mean	SD	Citation	Accelerometer Used (Make/Model #/Protocol)¥	CPM
36.2	7.3	51	20.8	18.1	Olvera et al. 2011	AG GT1M ; 2 d; 8 h/d	
36.5	9.2	57	52.0	32.0	Buchowski et al. 2009	TriTrac R3D; 7 d	
36.5	9.2	57	40.0	39.0	Buchowski et al. 2009	TriTrac R3D; 7 d	
36.5	9.2	57	63.0	30.0	Buchowski et al. 2009	TriTrac R3D; 7 d	
36.5	9.2	57	45.0	36.0	Buchowski et al. 2009	TriTrac R3D; 7 d	
36.5	9.2	57	58.0	30.0	Buchowski et al. 2009	TriTrac R3D; 7 d	
36.5	9.2	57	49.0	34.0	Buchowski et al. 2009	TriTrac R3D; 7 d	
40.6	5.6	45	33.8	25.2	Fuemmeler et al. 2011	MTI-7164; 4 d; 24 h	
40.6	5.6	45	26.3	20.4	Fuemmeler et al. 2011	MTI-7164; 4 d; 24 h	
42.9	10.7	504	27.5		Candelaria et al. 2012	Actigraph; 7 d	
47.0	14.0	786	95.6	117.7	Welk et al. 2014	SenseWear Mini; 1 d	
47.0	9.0	1150	26.0	10.0	Glazer et al. 2013	Actical; 7 d	1486
47.7	31.0	1963	16.8		Camhi et al. 2011	AG AN-7164; 7 d	
48.1	17.1	1594	78.0	40.4	Strath et al. 2008	AG 7164; 7 d	
48.1	23.5	2208	18.3	23.5	Loprinzi & Pariser 2014	AG 7164; 7 d	2020
49.3	20.8	535	18.0	13.9	Loprinzi PM 2012	AG 7164; 7 d	2020
53.3	6.9	184	112.8	59.0	Jilcott et al. 2011	Actigraph; 7 d	574
65.0	5.0	19	85.0	36.0	Gonzales et al. 2011	AG GT1M; 4 d	1041
73.2	1.7	148	61.8	4.8	Gabriel et al. 2013	AG GT1M; 7 d	760
Females, as above; complete are statistics are not provided							
2-	3.0	124	5.4		Grzywacz et al. 2014	Actical; 7 d	715
3-	4.0	26	6.7	6.5	Shen et al. 2013	AG GT1M; 5 d	615
3-	4.0	26	8.8	7.1	Shen et al. 2013	AG GT1M; 5 d	615
3-	5.0	192	7.0	2.0	Dowda et al. 2011	AG 7164; 14d	420
5.3		184	24.0		Kwon et al. 2011	AG 7164; 4 d	3000
6-	11.0	288	75.0	37.9	Gortmaker et al. 2012	AG 7164; 7 d	
6-	11.0	325	78.0	36.0	Gortmaker et al. 2012	AG 7164; 7 d	
8.7		184	25.0		Kwon et al. 2011	AG 7164; 4 d	3000
9-	9.0	431	173.3	46.4	Nader et al. 2008	Actigraph; 7 d	
9-	9.0	431	173.3	64.3	Nader et al. 2008	Actigraph; 7d	
9-	9.0	90	44.4		Trost et al. 2013	AG GT3X+; 7 d	
10-	10.0	93	40.2		Trost et al. 2013	AG GT3X+; 7 d	
11-	11.0	85	32.5		Trost et al. 2013	AG GT3X+; 7 d	
11-	11.0	434	115.3	36.3	Nader et al. 2008	Actigraph; 7d	
11-	11.0	434	112.6	53.2	Nader et al. 2008	Actigraph; 7d	

Table 24. Estimates of moderate & vigorous (MVPA) physical activity (PA) (continued)

PUBLISHED IN THE U.S. LITERATURE							
Age Range (years)			MVPA (min/day)				
Mean	SD	(n)	Mean	SD	Citation	Accelerometer Used (Make/Model #/Protocol)¥	CPM
11.3		184	23.0		Kwon et al. 2011	ActiGraph 7164; 4 d	3000
11-	13	43	62.2	38.6	Cradock et al. 2004	TriTrac R3D; 2d, 24 h	
11 -	11.0	143	30.8	11.6	Barker et al. 2003	AG 7164; 7 d	
11 -	15.0	15	98.0	62.0	Kozub & Farmer 2011	ActiGraph	
11 -	15.0	23	149.0	108.0	Kozub & Farmer 2011	ActiGraph	
12-	12.0	351	86.0	32.5	Nader et al. 2008	Actigraph; 7d	
12-	12.0	351	73.9	45.8	Nader et al. 2008	Actigraph; 7d	
12-	19.0	535	21.9	34.7	Gortmaker et al. 2012	AG 7164; 7 d	
13-	13.0	149	35.9	14.8	Barker et al. 2011	AG 7164; 7 d	
13.2		202	30.6		Kwon et al. 2013	Actigraph 7164; 4d	3000
15-	15.0	280	38.7	23.6	Nader et al. 2008	Actigraph; 7d	
15-	15.0	280	25.5	23.3	Nader et al. 2008	Actigraph; 7d	
15.3		134	28.1		Kwon et al. 2013	Actigraph 7164; 4d	3000
20-	39	920	111.4	59.6	Martin et al. 2014	AG AM-7164; 7d	760
20-	65	837	21.6	25.1	Luke et al. BMC 2011	AG 7164; 7 d	2020
20-	65	375	18.5	25.2	Luke et al. BMC 2011	AG 7164; 7 d	2020
20-	65	383	19.1	17.6	Luke et al. BMC 2011	AG 7164; 7 d	2020
22-	41	10	91.0	16.7	Calabro et al. 2009	IDEEA; 1 d	
22-	41	10	150.0	16.7	Calabro et al. 2009	SenseWear; 1 d	
26.2		359	12.3	16.7	Evenson & Wen 2011	AG 7164; 4 d	2020
26.2		359	111.8	57.4	Evenson & Wen 2011	AG 7164; 4 d	574
40-	59	903	104.4	65.6	Martin et al. 2014	AG AM-7164; 7d	760
60-	69	522	79.5	62.0	Martin et al. 2014	AG AM-7164; 7d	760
66-	69	106	15.2		Buman et al. 2010	AG 7164/71256; 7d	1952
66-	69	106	14.4		Buman et al. 2010	AG 7164/71256; 7d	1952
70-	79	229	10.4		Buman et al. 2010	AG 7164/71256; 7d	1952
70-	79	229	9.8		Buman et al. 2010	AG 7164/71256; 7d	1952
70+		602	44.9	46.8	Martin et al. 2014	AG AM-7164; 7d	760
80+		147	4.0		Buman et al. 2010	AG AM-7164; 7d	1952
80+		147	4.0		Buman et al. 2010	AG AM-7164; 7d	1952
Females: Health Considerations or Being Overweight/Obese							
8.4	0.9	***23***	32.8	17.1	DuBose & McK 2014	ActiGraph GT1M	
9.3	1.1	7	47.0		Hsu et al. 2014	ActiGraph GT1M	
10.9	3.6	226	74.0	46.0	Butte et al. 2007	Actiwatch; 3 d; 24 h	
11.9	0.5	534	24.1	41.6	Treuth et al. 2007	AG 7165; 7 d	1500

Table 24. Estimates of moderate & vigorous (MVPA) physical activity (PA) (continued)

PUBLISHED IN THE U.S. LITERATURE							
Age Range (years)			MVPA (min/day)				
Mean	SD	(n)	Mean	SD	Citation	Accelerometer Used (Make/Model #/Protocol)¥	CPM
11.9	0.5	534	16.7	16.2	Treuth et al. 2007	AG 7165; 7 d	1500
12.0	0.6	378	20.8	0.5	McMurray et al. 2008	AG 6 d; 30 s epoch	
15.7	1.1	37	27.9	17.8	Gyllen. et al. 2013	AG GT1M; 7 d	2020
27.8	6.5	27	8.8	10.3	Loprinzi et al. 2012b	AG 7164; 7 d	2020
28.9	5.6	114	14.3	43.6	Loprinzi et al. 2012b	AG 7164; 7 d	2020
41.1	15.9	21	12.6	12.2	Behrens et al. 2011	AG GT1M; 7d; 10 h/d	
53.0	9.0	41	113.4		Rogers et al. 2009	AG GT1M; 7 d	1953
54.5	6.9	196	22.2	17.4	Farr et al. 2008	MTI-7164; 7 d	2225
61.1	16.5	337	8.6	11.0	Loprinzi & Pariser 2014	AG 7164; 7 d	
Males: Normal, Healthy, or Not Specified							
3.8	0.4	21	19.4		Kelly et al. 2007	ActiGraph; 3d	3200
7.2	0.9	42	243.0		Pate et al. 2012	CSA 7164; 7d	
7.2	2.0	68	70.8	31.4	Sarzynski et al. 2010	AG GT1M; 7 d	2172
7.6	0.6		90.6	34.7	Willis et al. 2015	AG GT3x+; 4 d	
7.7	1.3	78	69.8	30.8	HM Hayes et al. 2011	MTI-7164; 7 d	2172
8.1	0.7	50	65.2	28.0	NC Crespo et al. 2013	AG GT1M; 7 d	
9.1	2.0	393	71.7	61.5	MW Long et al. 2013	AG 7164; 7 d	
10.1	1.0	51	146.0		Pate et al. 2013	CSA 7164; 7d	
10.6	0.8	23	168.7	59.9	Fuemmeler et al. 2011	MTI-7164; 4 d; 24 h	
10.6	0.8	23	145.0	51.9	Fuemmeler et al. 2011	MTI-7164; 4 d; 24 h	
10.7	4.0	194	96.0	57.0	Butte et al. 2007	Actiwatch; 3 d; 24 h	
11.2	0.3	184	41.6	22.0	Janz et al. 2007	ActiGraph 7164; 5 d	3000
12.0	1.0	48	88.0		Pate et al. 2013	CSA 7164; 7d	
12.3	0.4	56	42.1	35.1	Tucker et al. 2011	Biotrainer; 3 d	
12.5	2.4	19	109.7	32.7	Holmes et al. 2008	MTI; 4 d	
12.7	1.4	407	67.6	30.8	Sanchez et al. 2007	CSA 7164; 9 d	
12.8	1.1	210	24.8	17.6	Jago et al. 2005	MTI; 3 d; 24 h	
12.8	2.7	1263	34.3	23.5	Mark & Janssen 2009	AG 7164; 7 d	3000
13.4	0.5	37	86.5	47.9	Anderson et al. 2005	MTI 7164; 4d, 0600-2300	1399
13.4	0.5	37	27.1	22.9	Anderson et al. 2005	MTI 7164; 4d, 0600-2300	3200
14.5	1.8	286	49.7	28.6	Sirard et al. 2010	AG 7164; 5 d	
14.6	1.8	340	35.0	18.3	Hearst et al. 2012	MTI 7164; 7 d	
14.7	3.0	873	36.5	50.2	MW Long et al. 2013	AG 7164; 7 d	2020
15.7	1.0	44	61.0		Pate et al. 2015	CSA 7164; 7d	
16.7	1.4	37	60.0	25.8	Sirard et al. 2008	AG GT1M; 7 d	940

Table 24. Estimates of moderate & vigorous (MVPA) physical activity (PA) (continued)

PUBLISHED IN THE U.S. LITERATURE							
Age Range (years)			MVPA (min/day)				
Mean	SD	(n)	Mean	SD	Citation	Accelerometer Used (Make/Model #/Protocol)¥	CPM
42.8	6.2	45	30.5	23.2	Fuemmeler et al. 2011	MTI-7164; 4 d; 24 h	
42.8	6.2	45	29.5	18.8	Fuemmeler et al. 2011	MTI-7164; 4 d; 24 h	
43.0	10.2	547	38.4		Candelaria et al. 2012	Actigraph; 7 d	
46.2	16.8	1678	102.7	53.1	Strath et al. 2008	AG 7164; 7 d	
45.4	16.6	561	168.0	113.3	Welk et al. 2014	SenseWear Mini; 1 d	
46.0	24.3	2364	31.9	34.0	Loprinzi & Pariser 2014	AG 7164; 7 d	
46.5	38.0	1781	29.5		Camhi et al. 2011	AG AN-7164; 7 d	
47.0	8.0	959	30.0	22.0	Glazer et al. 2013	Actical; 7 d	1486
47.5	24.7	611	30.4	34.6	Loprinzi PM 2012	AG 7164; 7 d	2020
65.0	5.0	19	109.0	49.0	Gonzales et al. 2011	AG GT1M; 4 d	1041
78.5	4.8	2157	90.8	60.7	Cawthon et al. 2013	SenseWear Pro; 5 d	
Males, as above; complete age statistics not provided							
2-	3.0	118	6.6		Grzywacz et al. 2014	Actical; 7 d	715
3-	4.0	20	9.3	15.7	Shen et al. 2013	AG GT1M; 5 d	615
3-	4.0	20	10.4	9.1	Shen et al. 2013	AG GT1M; 5 d	615
3-	5.0	177	8.1	2.1	Dowda et al. 2011	AG 7164; 14d	420
5.2		142	31.0		Kwon et al. 2011	AG 7164; 4 d	3000
6-	11.0	265	96.5	78.1	Gortmaker et al. 2012	AG 7164; 7 d	
6-	11.0	319	101.2	72.1	Gortmaker et al. 2012	AG 7164; 7 d	
8.7		184	40.0		Kwon et al. 2011	AG 7164; 4 d	3000
9-	9.0	555	190.8	53.2	Nader et al. 2008	Actigraph; 7d;	
9-	9.0	555	184.3	68.6	Nader et al. 2008	Actigraph; 7d	
9-	9.0	68	59.9		Trost et al. 2013	AG GT3X+; 7 d	
10-	10.0	70	51.7		Trost et al. 2013	AG GT3X+; 7 d	
9-	9.0	64	57.5		Trost et al. 2013	AG GT3X+; 7 d	
11-	11.0	544	133.0	42.9	Nader et al. 2008	Actigraph; 7d	
11-	11.0	544	127.0	59.5	Nader et al. 2008	Actigraph; 7d	
11.3		184	41.0		Kwon et al. 2011	AG 7164; 4 d	3000
11-	13	43	82.3	42.8	Cradock et al. 2004	TriTrac R3D; 2d; 24 h	
11 -	15.0	9	181.0	103.0	Kozub & Farmer 2011	ActiGraph	
11 -	15.0	21	242.0	77.0	Kozub & Farmer 2011	ActiGraph	
12-	12.0	416	105.3	40.2	Nader et al. 2008	Actigraph; 7d	
12-	12.0	416	93.4	55.3	Nader et al. 2008	Actigraph; 7d	
12-	19.0	577	39.9	55.2	Gortmaker et al. 2012	AG 7164; 7 d	
12-	19.0	549	36.9	44.5	Gortmaker et al. 2012	AG 7164; 7 d	

Table 24. Estimates of moderate & vigorous (MVPA) physical activity (PA) (continued)

PUBLISHED IN THE U.S. LITERATURE							
Age Range (years)			MVPA (min/day)				
Mean	SD	(n)	Mean	SD	Citation	Accelerometer Used (Make/Model #/Protocol)¥	CPM
13.3		199	51.5		Kwon et al. 2013	AG 7164; 4 d	3000
15-	15.0	324	58.2	31.8	Nader et al. 2008	Actigraph; 7d	
15-	15.0	324	43.2	38.0	Nader et al. 2008	Actigraph; 7d	
15.3		133	40.1		Kwon et al. 2013	AG 7164; 4 d	3000
20-	39	795	151.2	88.8	Martin et al. 2014	AG AM-7164; 7d	760
20-	65	926	34.7	27.4	Luke et al. BMC 2011	AG 7164; 7 d	2020
20-	65	386	33.4	27.5	Luke et al. BMC 2011	AG 7164; 7 d	2020
20-	65	463	42.1	32.3	Luke et al. BMC 2011	AG 7164; 7 d	2020
22-	41	10	89.0	16.7	Calabro et al. 2009	IDEEA; 1 d	
22-	41	10	112.0	16.7	Calabro et al. 2009	SenseWear; 1 d	
40-	59	899	135.1	66.7	Martin et al. 2014	AG AM-7164; 7d	760
60-	69	501	98.4	54.5	Martin et al. 2014	AG AM-7164; 7d	760
66-	69	109	21.3		Buman et al. 2010	AG 7164/71256; 7d	1952
66-	69	109	22.3		Buman et al. 2010	AG 7164/71256; 7d	1952
70-	79	182	15.3		Buman et al. 2010	AG 7164/71256; 7d	1952
70-	79	182	14.2		Buman et al. 2010	AG 7164/71256; 7d	1952
70+		646	56.8	48.1	Martin et al. 2014	AG AM-7164; 7d	760
80+		89	11.6		Buman et al. 2010	AG 7164/71256; 7d	1952
80+		89	10.7		Buman et al. 2010	AG 7164/71256; 7d	1952
Males: Health Issues, or Overweight/Obese							
11.1	3.5	247	88.0	50.0	Butte et al. 2007	Actiwatch; 3 d; 24 h	
15.5	2.1	18	46.2	15.9	Holmes et al. 2008	MTI; 4 d	
37.8	14.0	9	34.2	22.5	Behrens et al. 2011	AG GT1M; 7d; 10 h/d	
55.3	8.0	59	32.4	22.1	Farr et al. 2008	MTI-7164; 7 d	2225
57.9	19.9	396	15.8	25.9	Loprinzi & Pariser 2014	AG 7164; 7 d	
80.6	5.6	743	58.6	53.2	Cawthon et al. 2013	SenseWear Pro; 5 d	
Both genders							
3 -		80	96.7	32.0	HG Williams et al. 2008	AG 7164; 7 d	1680
3-	3.0		85.0	38.0	Edwards et al. 2013	RT-3; 3 d	1400
3.5	1.1	337	14.9	9.5	Dolinsky et al. 2011	Actical; 7 d	715
4-		118	96.0	24.4	HG Williams et al. 2008	AG 7164; 7 d	1680
4-	4.0		90.0	37.0	Edwards et al. 2013	RT-3; 3 d	1400
5-	5.0		94.0	37.0	Edwards et al. 2013	RT-3; 3 d	1400
6-	6.0		87.0	33.0	Edwards et al. 2013	RT-3; 3 d	1400
7.0	1.9	63	90.7	22.4	Eisenmann et al. 2010	MTI-7164; 4 d	2172

Table 24. Estimates of moderate & vigorous (MVPA) physical activity (PA) (continued)

PUBLISHED IN THE U.S. LITERATURE							
Age Range (years)			MVPA (min/day)				
Mean	SD	(n)	Mean	SD	Citation	Accelerometer Used (Make/Model #/Protocol)¥	CPM
7.5	2.0	61	38.1	12.0	Eisenmann et al. 2010	MTI-7164; 4 d	2172
7-	7.0		80.0	40.0	Edwards et al. 2013	RT-3; 3 d	1400
8-		282	66.0	67.2	Butte et al. 2014	Actical; 3 d	
8-	14.0	291	19.5	15.5	Dunton et al. 2012	AG GT2M; 7d	2020
9-		282	55.0	33.6	Butte et al. 2014	Actical; 3 d	
9.1	1.5	76	113.4	37.0	Hennessy et al. 2010	AG 7164; 7 d	
9.1	1.6	682	144.0	52.0	Kneeshaw et al. 2013	AG GT1M; 7 d	
9.2	1.6	713	62.2	36.5	Tandon et al. 2014	AG GT1M; 7 d	
9.6	0.8	65	25.8	6.3	Wrontniak et al. 2007	AG 7164; 7 d; 10 h/d	3200
9.9	0.7	27	307.8	96.6	Tsai et al. 2012	Actiwatch 64; 7d	700
9.9	0.9	27	265.2	82.8	Tsai et al. 2012	Actiwatch 64; 7d	700
10-		282	53.0	33.6	Butte et al. 2014	Actical ;3 d	
10.0	0.7	51	49.1	26.9	Olvera et al. 2011	AG GT1M; 2 d; 8 h	
11.4	0.7	198	47.5	2.0	Wilson et al. 2011	Mini-Mitter; 7 d	1500
13.3	2.1	181	27.6	21.2	Lawman & Wilson 2014	Actical; 7 d	1500
14.7	1.7	91	42.0	26.4	Matthews et al. 2013	AG GT3X; 7 d	1952
14.7	2.0		39.5	26.6	AC Long et al. 2008	Actiwatch 64; 7 d	1500
14.8	0.5	130	44.2	27.1	DJ Graham et al. 2011	AG 7164; 7 d	1952
15.1	1.4		20.2	18.1	AC Long et al. 2008	Actiwatch 64; 7 d	1500
21.3	2.3	34	85.7	37.0	Sisson & Tudor-L. 2008	AG 7164; 2 d	
21.7	4.0	35	50.3	23.8	Sisson & Tudor-L. 2008	AG 7164; 2 d	
32.3	8.4	45	21.0	18.6	Dixon-Ibarra et al. 2013	GT1M; 4 d	2020
42.1	14.8	88	36.0	24.0	Matthews et al. 2013	AG GT3X; 7 d	1952
43.4	11.6	135	122.3	75.2	Warner et al. 2012	Actical; 7 d; 10 h/d	
43.6	10.7	946	30.3		Rovniak et al. 2010;	AG 7164/71256; 7 d	
46.0	10.6	655	23.8		Rovniak et al. 2010;	AG 7164/71256; 7 d	
51.1	15.4	30	64.7		Pugh et al. 2012	AG GT3X; 7 d	
53.7	14.3	20	7.5		Pugh et al. 2012	AG GT3X; 7 d	
57.4	9.9	71	35.4	24.2	Banda et al. 2010	Actical; 7d	
57.9	6.9	31	10.2	13.8	Dixon-Ibarra et al. 2013	GT1M; 4 d	2020
58.3	10.3	139	11.8	28.8	Sloane et al. 2009	RT3; 7 d	
62.0	9.0	519	14.0		J Song et al. 2010	AG GT1M; 7 d	
63.5	8.3	200	14.0	19.0	Hutto et al. 2013	Actical; 4 d	1065
64.3	6.9	232	22.9	22.0	Swartz et al. 2012	AG 7164; 7 d	1952
65.6	13.2	22	11.5	11.0	Stevenson et al. 2009	AG GT1M; 7 d	

Table 24. Estimates of moderate & vigorous (MVPA) physical activity (PA) (continued)

PUBLISHED IN THE U.S. LITERATURE							
Age Range (years)			MVPA (min/day)				
Mean	SD	(n)	Mean	SD	Citation	Accelerometer Used (Make/Model #/Protocol)¥	CPM
70.5	12.6	13	41.6	30.9	Erikson et al. 2013	SenseWear; 3 d; NS	
71.3	8.4	84	79.7	46.7	Parker et al. 2008	AG 7164; 7 d	760
73.2	5.9	33	21.6	13.8	Dixon-Ibarra et al. 2013	GT1M; 4 d	2020
75.0	7.5	28	86.2	118.5	Erikson et al. 2013	SenseWear; 3 d; NS	
76.8	9.3	26	40.7	43.4	Erikson et al. 2013	SenseWear; 3 d; NS	
78.8	4.2	121	14.8	17.0	J.Kerr et al. 2013	AG GT3X+; 4 d	1952
89.3	3.8	94	5.3	9.3	J.Kerr et al. 2013	AG GT3X+; 4 d	1952

¥ All studies require that the accelerometer not be worn during “wet” events (bathing, swimming, or other); 24h studies allow it to be worn during sleeping; duration is “awake hours” unless otherwise noted.

AG=ActiGraph.

CPM=Counts per minute

d = Day(s)

NS=Not specified length of time

Jr. et al., 2008; Lyden et al., 2011). However, Welk et al. (2000) caution that regression equations developed in a laboratory or clinical setting to relate accelerometer counts to activity-specific energy expenditure estimates probably are not applicable to field conditions. They also think that certain accelerometers over-estimate EE at all levels of activity (Welk et al., 2000).

Accelerometers have been evaluated against the "gold-standard" doubly-labelled water (DLW) method of obtaining EE on a daily basis. Most evaluations indicate that there is "poor agreement" between physical activity measured by accelerometers (at any level of activity) and PAEE evaluated by the DLW technique (N.Campbell et al., 2012; J. Carter et al., 2008). This is a near-universal finding of these types of aggregate EE evaluation studies (e.g., Leenders et al., 2006). Accelerometers frequently have been evaluated against VO_2 monitoring (indirect calorimeter): an interesting one is Adolph et al. (2012); there are many others. With respect to accelerometer "validity" (evaluation) studies, some researchers make a distinction between "convergent validity" (represented by a correlation coefficient) and "criterion validity" (represented by absolute estimates). Convergent validity also is called "concurrent validity," especially when multiple monitors are being compared (Welk et al., 2000).

Accelerometers were used in two two-year "waves" (2003-2004 & 2005-2006) of the National Health and Nutrition examination Surveys (NHANES). There are 20-30 papers in the literature analyzing data from these waves, differing mostly in which subgroups are the cohort of interest in a particular paper (youth of different age groupings, people with COPD, etc.). One first author, Paul D. Loprinzi of the University of Kentucky has over ten papers on the 2003-2006 NHANES papers himself focused on different cohorts! Table 24 contains data from some of these papers and other NHANES analyses, but I tried to not provide "redundant" MVPA data in the Table (MVPA data for essentially the same subgroup) from the NHANES papers; doing so would bias variability estimates included in the database if some type of meta-analysis would be done on the information. I'm sure that some NHANES redundancy has crept into Table 24, because of the multiple authors/institutions involved in publishing data from the same study. Oftentimes the only difference among the studies is the accelerometer counts used for the MPA and VPA cutoffs. None of the *combined* female/male data from NHANES studies are reproduced in Table 24 (e.g., Loprinzi et al., 2011a, 2014b).

The most sophisticated analysis of the NHANES data that exists is Metzger et al. (2008). They succinctly describe how the MPA and VPA count cutoffs for ActiGraph 7164 accelerometer used in NHANES affected subsequent estimates of MVPA. The NHANES study cutoffs used a sample-size weighted average of cutoffs published in Brage et al. (2003), Freedson et al. (1998), Leenders et al. (2003), and Yngve et al. (2003). The count cutoff averages were 2020 CPM for MPA and 5999 for VPA. Many other count cutpoints have been developed and used as can be seen in Table 24. Accelerometer counts used for MPA and VPA in various studies vary greatly, even for the same instrument. More on that below.

Please note that the Table 24 presented in this report is an abbreviated version of one that is available from the author. The original table has separate estimates for MPA and VPA, where available, and relates the CPM cutoffs to METS classes, also where available. Some of the articles that provide separate MPA and VPA estimates in min/d do not provide MVPA estimates also, so there are more data available on time spent per day in moderate and/or vigorous physical activity than shown in this Report. Finally, it should be noted that Table 24 does not contain MVPA data that were presented only (1) graphically, such as Song, et al. (2010, or (2) as quartiles or quintiles, etc., such as Thiese et al. (2011).

Systemic Issues with Accelerometry

Since accelerometers are an electro-mechanical device, they cannot come in contact with water. That obviates their use for estimating energy expenditure or oxygen consumption during water-contact activities or water sports. This may significantly underestimate daily MVPA for 2% of so of all adults whose exercise consists solely of swimming or pool-based exercise classes (Sidney et al., 1991). Accelerometers have also been shown to be very inaccurate for some activities, like cycling (where hip-mounted devices do not pick up on exercise that does not change the vertical plane much) which leads to a systematic under-estimate of MVPA in individuals that participate in those types of activities (Miller et al., 2006). We have experiential knowledge of this in an in-house experiment: a subject wore an accelerometer on his hip while riding a bike on roads between northern Durham and RTP and it recorded hardly any activity, whereas when he drove a car with bad shocks on the same roads, the accelerometer recorded very high levels of activity! We certainly got a very misleading indication of activity-specific energy expenditure in that trial. Swimming, water sports, and biking are all high-METS activities, generally >7 METS, and fairly popular in the general population. A lot of MVPA min/d can be missed during these activities solely due to accelerometer limitations.

Besides these problems, there are four universal issues with accelerometers: (1) how to handle non-wear time, also known as zero count data; (2) what temporal "epoch" should be used to aggregate count data (recorded between 1-15 seconds normally) before storing the information and how "bouts" of PA are defined; (3) how to translate mechanical movement of a lever into an estimate of energy expenditure, which then has to be related to oxygen consumption; and (4) how many hours per day are enough to accurately estimate total daily physical activity (Bornstein et al., 2011a; Gabriel et al., 2010; Herrmann et al., 2013; King et al., 2011; Troiano & Freedson, 2010; Troiano et al., 2012). These issues will be briefly addressed below in order.

Where on the body an accelerometer is placed has a major effect on its estimates (Abel et al., 2009b). Most of the studies place it on or near the "non-dominant" hip, but sometimes it is placed on an ankle or wrist. Table 24 data are

supposed to be exclusively from hip-placed accelerometers, but a few non-hip study results may have slipped into the Table. Accelerometer placement on the body also affects estimates of non-wear time, an issue discussed next, even if the subject is expending energy. Subject differences in BMI, stride length, gait speed and pattern, and activities undertaken all affect MVPA estimates (Storti et al., 2008). In addition, certain health considerations also affect accelerometer count-to-energy expenditure rate metrics compared to "normals," another source of uncertainty in the results (Agiovlasitis et al., 2012; Almeida et al., 2011). Of course, age and gender are always important considerations leading to different accelerometer results. These considerations give rise to the large counts/steps differences for similar activities that are discussed below.

Often accelerometer studies involve placing the subjects into a typology of cohorts, such as age groupings, gender, health condition, etc. For MPA/VPA studies the cohort aggregate data include subjects engaging in no MVPA time/d being combined with people participating in significant exercise time. This leads to the well-recognized problem that the mean estimate (of MVPA time/d) is a compromise statistic, overestimating non-doer time (which is zero minutes) and systematically underestimating those that undertake significant amounts of PA. For instance, Strauss et al. (2001) found that 16% of their 13 y old subjects participated in MVPA and only 7% undertook VPA. These are MVPA doers. If the mean MPA time was 43.8 min/d (using Anderson et al., 2005 data; see Table 24), then 84% of the sample had 0 min/d, while doers had 274 min/d in MVPA exercise. Quite a different MVPA picture is provided for doers and non-doers than might be obtained by looking at just the mean. The same holds true, even more so, for VPA. Unhappily, however, rarely does an exercise paper include both the participation rate and the mean time/d data to address this data issue.

The COV's associated with data in Table 24 indicate that there is a lot of variability in the samples taken. The total number of studies for which a COV for MVPA time can be calculated is 226. The median COV for these studies is 60% (mean=57.8%). For "normal, healthy, or not specified" samples having "complete" age statistics available, the median COV for MPA time for females is 56% (n=79; range=4-304%) and 58% for males (n=28; range=36-138%). For samples containing people with complete age statistics having health problems or being overweight/obese, the median COV for female MPA time is 97% (n=11; range=2-305% and 67% for males (n=5; range=34-164%). These data confirm what Saris & Binkhorst (1977) stated in 1977: "it is well known that daily physical activity varies enormously" within the general population. The wide range in individual COV's just presented in the parentheses confirms this statement.

Within a reasonably compact age group there is a wide variability in the number of minutes per day of MVPA undertaken. For instance, in a study of elders (mean age = 71.3 ± 8.4; range 55-87), the mean MVPA estimate for the both-gender cohort over 7+ days was 79.9 ± 46.7, with a range of 9.6 – 220.3 min/d for individual subjects. Daily MVPA values were of course wider than that, but were not provided (Parker et al., 2008). The difference in the mean range of 210 min/d is very large for the cohort.

Non-Wear Time.

How to handle zero- or near-zero count data from accelerometers for a specified epoch time to determine non-wear time by a subject is a major issue with accelerometers (Choi et al., 2011, 2012; Evenson & Terry, 2009; Herrmann et al., 2014; Semamik et al., 2010; Tudor-Locke et al., 2011e). There are a plethora of ways that zero counts are treated in accelerometer studies. Usually a criterion is established that considers zero counts to be non-wear time if they exceed 60 consecutive minutes in duration (Brown. BB & Werner, 2007; Loprinzi et al., 2014d; HG Williams et al., 2008). Other less conservative decision rules have been developed, such as excluding any zero count lasting 10 minutes or longer (Beets et al., 2011). On the other hand, Hutto et al. (2013) state that using 120 min of consecutive zero counts provides "dependable population-based estimates of wear and nonwear time, and time spent being sedentary and active in older adults wearing the Actical™ activity monitor" (p. 120). Because accelerometers can be—and frequently are—worn while sleeping, there also are decision rules that relate to what is an "effective" zero-count, such as no non-zero readings for <10 minutes or other defined time period (Cradock et al., 2004). Thus, a zero-count may not really be zero per se, but some small count number that is thought to be improbable if the subject is awake and wearing the monitor. See Crouter et al. (2013) for more information on these points. Non-wear time is subtracted from the daily span of time that the accelerometer is supposed to be worn, generally the total awake time of the subject, except for bathing or swimming time.

Another issue is that the impact of non-wear time is not spread out evenly over the day.

> At either end of the day, nonwear time appears to distort population estimates of all accelerometer time and physical activity volume indicators [e.g., MVPA], but its effects are particularly clear on population estimates of time spend in sedentary behavior (Tudor-Locke et al., 2011; p. 693).

Rarely, however, are time-of-day specific rules used to define non-wear time in a study.

The different decision rules used for non-wear time greatly affect the estimates of MVPA and other levels of activity. Non-wear time definitions affect both the absolute and relative (% of time in the day) min/d estimates of MVPA. An interesting simulation evaluation of elderly accelerometer data obtained during the 2005-2006 NHANES health survey that included accelerometer monitoring, indicates that using different criteria of what constitutes non-wear time causes MPA estimates to vary from 7-26 min/d and VPA from 14-40 min/d (Herrmann et al., 2013, 2014). These are large ranges in an elderly cohort. On the other hand, in another study of older adults with knee osteoarthritis who did not have a lot of physical activity to begin with, different non-wear decision

rules did not significantly change the observed MVPA of 14 min/d (Song et al., 2010). Three different thresholds of minimum consecutive zero counts of 60, 90, and 120 min were tested in that study.

Because of the uncertainty of whether or not a zero count for a defined epoch is "real" or not, there have been a number of accelerometer count **imputation** schemes devised to substitute non-zero counts when researchers think that physical activity was occurring but not recorded. Imputation is a complex topic, involving a number of "nested" decision rules and criteria. Imputation cannot really be treated in this Report to do the topic justice. For additional information, see, for instance, Dowda et al. (2007); Morris et al. (2006); and Paul et al. (2008). The Morris et al. (2006) paper describes a sophisticated approach to imputing missing data via the modeling of wavelet-based functional mixed models; it is an interesting paper. Perhaps the most rigorous analysis of non-wear algorithms focused on MVPA estimates is contained in Crespo et al. (2012).

Metzger et al. (2008) describe how imputation of missing daily accelerometer data affects estimates of MPA and VPA, and how imputation also affects the number of "valid" days of data used for further analysis. (See below for more information on the number-of-days issue.) They then use latent class analysis to determine if "natural groupings" of age-weighted cohorts existed in the sample with respect to the number of MPA/VPA accumulated per day given the missing day problem. Five classes of people were identified, whose mean daily MVPA ranged from 134 min/d in a miniscule 0.9% of the population to the lowest class only averaging 5.3 min/d for 33.6% of the population. The authors label this last group as "inactive" (Metzger et al., 2008). That group and the next lowest, averaging about 20 min/d MVPA, constitute about 79% of the weighted population. When daily MVPA time/d is analyzed with respect to 10 min "bouts," the proportion of the weighted population MVPA "bout-minutes" indicates that the highest class had about 90 min/d in bouts but were only 0.6% of the population; the lowest two classes, averaging <10 min/d of MVPA constituted 93.5% of the weighted population (Metzger et al., 2008).

Affected even more by zero count considerations are those results reported only in *percent of time* above any count cutpoint, as we would have to know wear time and epoch length to calculate the minutes/day in MVPA. We do not generally report percentage of time data in this Report. One study that provided estimates of the percent of daily time spent in MPA and VPA states that a mixed sample of 92 females/males aged 13.3 ± 2.0 y spent a mean of 16 ± 3.7% of their 14h day in MPA and another 7.1 ± 2.4% in VPA (Strauss et al., 2001). Multiplying these percentages by monitoring time works out to be about 138 min/d for MPA and 58 min/d for VPA. These are relatively high estimates.

Even though decision rules used to calculate non-wear time are important in understanding accelerometer results, we do not report them in Table 24 or in the text, as we would get bogged down in a lot of tedious detail. Suffice to say that the data in Table 24 are implicitly based upon whatever non-wear and valid day decision rules that authors of the papers themselves applied in their study of daily MVPA time and participation rates. These rules undoubtedly affect the MVPA estimates.

Epoch Time and Activity "Bouts."

The range used for epochs in reported studies is between 5 and 60 seconds. The most commonly used epoch by far is 60 seconds. Obviously more data have to be stored for short epoch times given a fixed daily monitoring period. This leads to instrument storage and battery-life problems. This issue is related to the criteria used to identify MVPA bouts, such as x counts per 10 minutes, 20 minutes, etc. It is not to be confused with the even longer length of time over which the monitoring is undertaken, such as 10 h/d over 4 d or 7 days, etc. that also affects storage capacity and battery life. Papers by Edwardson & Gorley (2010), Kuffel et al. (2011), McClain et al. (2008), Rowlands et al. (2006), and Vale et al. (2009) discuss the epoch length/bout issues, including providing alternative estimates of MVPA depending upon different algorithms used to define bout and epoch length. Edwardson & Gorley (2010) recommend that a short epoch length be used to realistically estimate MPA and VPA, especially in children, whose PA occurs in "bursts." Epochs <60 sec result in significantly higher estimates of MVPA time/d, but with smaller bias relative to direct observation using the Children's Activity Rating Scale (CARS) (Hislop et al., 2012a,b). The same is true for shorter bout length.

An example from Kang et al. (2010) of how epoch length, accelerometer counts, and bout length all interact in an estimate of MVPA time/d:

> PA bouts were defined as time intervals having accelerometer counts >500 counts per 30-s epoch (cpe) for at least 7 min, allowing for up to 2 min of epoch below that threshold during the 7-min interval. Multiple time intervals with breaks ≤2 min were considered as one bout if the entire sequence of counts satisfied the count criteria (p. 1420).

Explaining all of this in Table 24 would be a herculean task, even if the authors of each study provided it in their papers, which many do not.

Since epoch length directly affects MVPA count cutpoints, reproduced here is a table from a paper by McClain et al. (2008) that clearly relates these two parameters in a study of 5th grade students. Four different decision criteria were used to derive epoch duration/accelerometer cutpoint. These are identified by the papers where they were first published (first author names only). Count cutpoints increase by epoch length are non-linear for three of the four studies, with an apparent inflection point at a 30 sec epoch time. (Actually the data plot as two splines with 30 seconds as the inflection point. Whether or not the relationship between epoch length and counts truly is a continuous non-linear function cannot be ascertained with the data provided.) Note also that for the last two papers (by the same first author), age of the child affects the count/epoch length relationship.

MVPA Count Cutpoint for Different Epoch Lengths

		Length (in seconds)				
Epoch Derivation	**Age**	**5**	**10**	**20**	**30**	**60**
Treuth (2004)	N/A	250	500	1000	1500	3000
Mattocks (2007a)	N/A	298	596	1193	1790	3851
Freedson (2005)	10 y	159	318	636	955	1910
Freedson (1997)	11 y	171	343	686	1030	2060

None of the MVPA data from McClain et al. (2008) appears in Table 24 due to the authors only presenting MVPA data for a 30 minute PE class for 5th graders. Free-living MVPA information for an entire day was not provided in the paper. McClain et al. (2008) also discuss the epoch/bout issue in previously-published papers (McClain et al., 2007a, b). Data from McClain et al. (2007a) were not presented in Table 24 because the authors did not provide age information by gender for their subjects. Data on adults from McClain et al. (2007b) were not reproduced because they mixed genders and did not provide a mean age value for them.

A number of studies aggregate counts into 10-minute (or other length) bouts and utilize cutpoint thresholds for only that time period (Glazer et al., 2013; Thompson et al., 2009). One study considers moderate intensity activities to be those between 30,000-49,999 CPM per 10 min bout; vigorous activities are those with 50,000 counts per 10/min bout (Bailey & McInnis, 2011). Data from this study are not included in Table 24 because the authors only provided the number of days having one or more bouts over the cutpoints; total minutes per day spent at moderate/vigorous level were not reported. MVPA estimates drop drastically when a longer time period is used to define a bout. If bout length is raised to 10 minutes from 1 minute and the cutpoint criterion is not changed, MVPA estimates drop by 25-50% on average (Glazer et al., 2013). Trost et al. (2002) evaluate how many 5-, 10-, or 20-minute bouts of MVPA (≥3 METS) occur/week in 1-12 grade students using an Actigraph 7164 accelerometer. The number of weekly MVPA bouts drops greatly as bout-length increases at all grade levels in both genders. They also drop with grade level (Trost et al., 2002). For example the number of weekly 5-min bouts in boys drops from 85 in grades 1-3 to 22 in grades 10-12. The drop in weekly bouts for grade 1-3 boys is from 85 for 5-min bout lengths to 8 for 20-min bout lengths (Trost et al., 2002). Similar decreases in both the number of bouts/grade and bouts/bout-length occurs in females.

A correspondingly wide span of estimates for MVPA bouts of 10 minutes or longer and total minutes/day is depicted in Ham et al. (2007). These authors also provide 10-minute bout estimates by four heart rate reserve categories (<25%; 25-44%; 45-59%; and ≥60%) and found that most MPA accelerometer estimates fell into the light HR category of <45% heart rate reserve.

Daily Monitoring Period.

Related issues to non-wear time and data imputation are how many hours/day constitutes a "valid day" and how many valid days out of the overall monitoring period are required to provide an unbiased estimate of MVPA for a sample of subjects. The criterion used by researchers for these related topics vary widely. The criterion for having a valid day of data often is ≥10 h (Cook et al., 2012; Glazer et al., 2013; Loprinzi et al., 2014b; Rowlands et al., 2015), but other periods are used also: 24 h is used by Cook et al. (2012). Barnes et al. (2013) and Cradock et al. (2004) use 8 h; Trost et al. (2013) use 9 h. Some studies use a minimum of 4 days of valid data to be called a valid sample, with at least 8 h of complete information per day (Francis et al., 2011; Hayes et al., 2013). A very loose criterion for a 7 d study is that each individual has to have ≥ 1 h of valid data on ≥ 3 days for their data to be included in subsequent analyses (Pate et al., 2004). How a criterion like this can be considered to provide a valid estimate of daily time spent in MVPA is questionable. An alternative—and more sophisticated--criterion of non-wear time that has been used is that if 2 of the 3 vectors in a triaxial accelerometer records a zero count for **any** epoch length of time ≤ 60 sec, the data are not considered to be valid (Burdette et al., 2004). That is a stringent definition; usually single axis analysis of triaxial data is not undertaken due to the increased storage capacity needed to maintain single axis information. Sometimes a valid day only includes a specific part of the day—say 1000-1400 and all other hours are ignored; a valid day in that case is not necessarily even daily awake time. How a valid day is defined using accelerometer data involves many factors and the possibilities are many.

A simulation study of 40 days of accelerometer data having 14 h/d of valid data is described in Herrmann et al. (2013, 2014). The 40 days were randomly sampled from a base study of 1,200 days, not all of which had a complete 14 h/d of valid data. From these 40 days, they repeatedly sampled between 10 and 13 h/d from the valid days of data and compared the absolute and "absolute percentage error" (APE) for each sample versus the original 14 h/d data set. For MPA and VPA, the absolute estimate of time/d fell as the number of monitoring hours/day decreased, while the APE increased greatly. For 10 h/d, the APE was 29.2 ± 25.9% for MPA and 41.7 ± 75.8% for VPA, while for a 13 h/d sample it was 6.4 ± 11.2% for MPA and 5.6 ± 24,2% (Herrmann et al., 2013). The other h/d simulations were in between these values. The absolute differences were about 10 min/d for MPA and only 2 min/d for VPA; which works out to 21.2% of the original 14 h/d data for MPA and 36.5% for VPA: quite large differences. Thus, the number of hours per day considered to be valid non-wear time makes a large difference in subsequent estimates of the time spent in MPA and VPA (and by extension to MVPA).

In an interesting analysis of valid versus invalid accelerometer data in children and adolescents, Loprinzi et al. (2014c) found that overweight or obese youth are far more likely (60% more so) to have invalid data than normal weight youth, thus introducing another bias in accelerometer data and limiting its generalizability (Loprinzi et al. 2014c).

Translating Accelerometer Counts into Activity Estimates.

The "translation" process is most often accomplished using the manufacturer's proprietary software to categorize energy expended (as VO_2 or METS) associated with accelerometer counts. Manufacturers usually do not provide their algorithms used to make that conversion, so the method used is a "black box" for users. Exercise physiologists employing accelerometers quickly found that there was wide variability in their PA estimates due to these unknown translation algorithms (Calabró et al., 2009; Cliff & Okely, 2007; Freedson et al., 1997, 1998; Mark & Janssen, 2011; Montoye et al., 1996). This has led to a "mini-industry" for academic researchers in devising regression-based algorithms to translate accelerometer counts into better EE, METS, or categorical estimates, such as "moderate" and "vigorous" PA levels (Griffiths et al., 2012; Leenders et al., 2006; Lyden et al., 2011; Matthews, 2005; Trost et al, 2011; Trumpeter et al., 2012; Troiano et al., 2006; Tucker et al., 2003). In doing so, researchers themselves are forced to operationally define what constitutes MVPA levels, and then delineate what accelerometer count limits are used for the MVPA levels. Most use 3-5.9 METS as moderate PA and ≥6 METS as vigorous PA. As we have seen in Section 6, METS limits themselves are problematic, being based upon an inflated REE value (3.5 mL/kg-min). The accelerometer count bases used for the different study MVPA estimates are provided in Table 24 where available. Many authors do not supply these data, however, so we have to assume that they use the manufacturer's translating algorithms.

Another dimension of the translation issue is the difference among subjects in step frequency, acceleration rate, gait, and type of locomotion, all of which affect the counts-to-energy expenditure relationships implicit in the manufacturer's unexplained accelerometer algorithms. These considerations certainly affect variability in individual results for similar activities (Brage et al., 2003a, b). Non-linear relationships between counts and forward acceleration exist in many accelerometers, resulting in significant differences in inter-instrument readings—about a 20% COV—for specific motions in a mechanical test not involving humans wearing an accelerometer (Brage et al., 2003c). There are numerous studies that develop regression equations to "correct" for acceleration and other differences in specific accelerometers; for example, Brandes et al. (2012) and Calabró et al. (2009). There are scores of these regression papers, "correcting" the manufacturer's built-in algorithms to better predict energy expenditure for a set of activities. Calabró et al. (2009) developed their own algorithms that dropped the overall average error of the SenseWear Pro accelerometer estimates versus treadmill results from 32% to 1.7%; however, error improvement was activity-specific. It was -20.7% for rest, -4.0% for coloring, -4.9% for playing computer games, between -0.9% and +3.5% for walking on a treadmill at 3 different speeds, and -25.7% for biking (Calabró et al., 2009). However, only the new biking algorithm produced statistically significantly different results compared with the built-in algorithms.

Because of the translation issue, there are a number of clinical studies testing the various algorithms that have been devised to translate accelerometer counts/minute (CPM) into estimates of EE, VO_2, and PA categories. We are only interested in the PA categories issue here. Some of these studies follow.

One study developed its own unique counts/min algorithms based on observing 7 y old subjects undertake MPA activities and then averaged the cutpoints over their sample (Sarzynski et al., 2010). In general, the relationship between accelerometer counts and energy expenditure (and/or) METS is specific to each individual and is non-linear; for this reason, mean count values are used for age/gender-specific cohorts (Tudor-Locke et al., 2011). It has to be realized, however, that any cutpoint designation is a compromise for any activity over a set of subjects.

An analysis of six alternative cutpoints used to define MPA and VPA for a single accelerometer—an ActiGraph GT1M—was undertaken by Crouter et al. (2013). The lower bound cutpoints for four studies, two of which used alternative algorithms from a single first author group are reproduced below.

These cutpoints are highly variable for a single accelerometer model.

	Lower-Bound Cutpoints (Counts / min)	
Citation/Algorithm	**MPA**	**VPA**
Crouter (2006)		
Walking/running algorithm	1,588	6,774
Lifestyle algorithm	388	2,826
Crouter (2010)		
Walking/running algorithm	297	1,126
Lifestyle algorithm	61	445
NHANES	2,020	5,999
Mathews (3 papers)	760	5,725

Freedson et al. (2005) reported an earlier evaluation of alternative cutpoints for the ActiGraph 7164, as well as the RT3 and Actical acccelerometers. They provide the following alternative cutpoints for "youth" (children and teens) MPA and VPA criteria for the ActiGraph 7164 monitor based on either VO_2 or EE criteria.

Paper	Sample Size (n)	Age Range	Criterion	MPA (CPM)	VPA (CPM)
Treuth et al.2004	74	13-14	VO_2	3000	5200
Puyua et al. 2002	26	6-16	Energy Exp.	3200	8200
Eston et al. 1998	30	8-10	VO_2	500	4000

As can be seen from the above two tables, there is a lot of variability in cutpoints for both the Crouter et al. (2006, 2010) and Freedson et al. (2005) papers, even for narrow age ranges. Wide variability is also seen in Crouter et al. (2013). Guinhouya et al. (2006) state that the various accelerometer cutpoints used by exercise physiologists are inconsistent and biased. Loprinzi et al. (2012c) provide an excellent overview of the cutpoint differences used by researchers and how they affect estimates of MVPA in children and adults. It probably is the most comprehensive study of its type in the literature. Another good review of how epoch length affects MPA and VPA estimates is contained in Sirard et al. (2011). They investigated 5 different "data reduction algorithms" (24 different possible combinations), and found that only a handful would *not* produce statistically significant differences in MPA estimates. They repeated the analysis for VPA for a different study time period, using a repeated measures general linear modeling approach, and found a similar result (Sirard et al., 2011).

In another analysis, four academic research groups devised accelerometer count "cutpoints" for MPA and VPA for the Actigraph GT1M uniaxial model (Actigraph LLC. Pensacola FL) using regression analysis of treadmill and indirect calorimetry data. For a discussion of cutpoint differences for the GT1M and a comparison of them with output from another accelerometer (the Kenz Lifecorder), see Abel et al. (2009a). Data from this paper are not contained in Table 24 since results were not provided by gender, but they are instructive concerning how much variability is seen in accelerometer performance by the academic community. One-minute accelerometer cutpoints—"translated" into CPM--varied by an order-of-magnitude among the four groups for the same model accelerometer! Data from the Abel (2009a) paper are reproduced here.

Accelerometer: Algorithm	MPA CPM Cutpoint	Time (Min/d)	VPA CPM Cut-point	Time (Min/d)
GT1M: Hendelman et al. (All)	191-7525	245±106	≥ 7526	260±102
Swartz et al.	574-4944	129± 70	≥ 4945	155±68
Freedson et al.	1952-5724	40± 24	≥ 5725	60±31
Hendelman et al (Walk)	2191-6892	39± 27	≥ 6893	55±31
Nichols et al	3285-5676	39± 24	≥ 5677	60±31
Kenz Lifecorder		29± 22		52±34

Another study that compared accelerometer counts to activity-specific oxygen consumption measures depicts the same wide range in the relationships as noted above. The study is Evenson et al. (2008) using 5-8 y old subjects. A table in their paper includes data from other studies that developed MPA and VPA cutpoints for children aged 3-16 y old. Two accelerometers were used: the ActiGraph 7164 (predecessor of the GT1M) and the Actical, which despite the similar name, is made by another company. A version of their table follows; in the "activities included" column, play is P, other is O, R is run, and walk is W; CPM is counts per minute.

	Accelerometer/ Algorithm	Activities Included	MPA CPM Cutpoint	VPA CPM Cutpoint	Subject Ages (y)	Sample Size
7164:	Puyua et al. (2002)	W, R, O	3200-8199	≥ 8200	6 - 16	26
	Sirard et al. (2005)	Sit, P, W, R	615-1230	≥ 1231	3	5
		As above	812-1234	≥ 1235	4	5
		As above	891-1254	≥ 1255	5	6
	Pate et al. (2006)	W, R	420 - 841	≥ 841	3 - 5	29
	Evenson et al. (2008)	W, R, O	574-1002	≥ 1002	5 – 8	33
Actical:	Puyua et al. (2004)	W, R, O	1500-6499	≥ 6500	7 – 18	32
	Pfeiffer et al. (2006)	Sit, P, W, R	715-1410	≥ 1415	3 – 5	18
	Evenson et al. (2008)	W, R, O	508- 718	≥ 719	5 – 8	33

A wide difference in the estimated number of MPA, MVPA, and VPA minutes/day is seen depending upon the count cutpoints used in a mixed-gender sample that also carried a portable VO_2 indirect calorimeter. The study period was between 5-6 h in a field setting (Strath et al., 2003a). The following results were obtained for activities classified as MPA and VPA according to the "standard" 3.0-3.59 / >6.0 METS categories using 5 different cutpoints from the literature.

Monitor/ Cutpoint Distinctions	Min of MPA	Ratio to Indir. Calor.	Min. of VPA	Ratio to Indir. Calor.
Indirect Calorimetry	64 ± 41		6 ± 8	
Freedson (1998)	26 ± 19	0.41	10 ± 18	1.67
Hendelman#1 (2000)	141 ± 48	2.19	6 ± 14	1.00
Hendelman#2 (2000)	26 ± 15	0.41	7 ± 14	1.17
Nichols (2000)	29 ± 17	0.45	10 ± 18	1.67
Swartz (2000)	82 ± 39	1.28	11 ± 18	1.83

None of the count cutpoint approaches are particularly accurate for either MVPA category compared with indirect calorimeter measures (the ratio of the accelerometer min/d to indirect calorimetry is shown as "Ratio to Indir.Calor.).

Another interesting comparison of cutpoint algorithms is contained in Alhassan et al. (2012). They looked at both the GT1M Actigraph and Actical accelerometers in a clinical, treadmill study of children 8-16 y. Four algorithms were evaluated for the GT1M and 3 for the Actical. None of them were used in the Abel et al. (2009a) paper discussed above. Their emphasis was on comparing EE estimates using the algorithms/models and not on the cutpoints for MPA and VPA per se. However, Alhassan et al. (2012) provided cutpoint estimates in counts/min for three situations:

	MPA (CPM)	VPA (CPM)
Actigraph GT1M: Treuth et al.	3200-8199	≥8200
Trost et al.	3000-5199	≥5200
Actical: Puyua et al.	1500-6499	≥6500

Alhassan et al. (2012) also provide regression equations to relate METS (in kcal/min) to accelerometer counts/min for the seven model/algorithm cases studied. Most of them are linear, but one is non-linear (the Puyua et al., 2002 algorithm); one of the equations includes body mass as an independent variable along with counts, and another is based on age and an age/CPM "interactive" term. Thus, there is variety in what the both the form and content of the regression equations evaluated. A similar finding has been reported by Balogun et al. (1989). Alhassan et al. (2012) calculated the Kappa statistic to quantify the statistical agreement between observed METS estimates and treadmill kcal. The Kappa coefficient (*K*) represents the precision of two categorizations; it has the same form as a Spearman correlation coefficient and may be interpreted as the percent of agreement attained given the amount of agreement obtained by chance alone (Viera & Garrett, 2005). For moderate PA, *K* varied from -0.01-0.34 for the four Actigraph/algorithm combinations; *K's* of this magnitude are considered to have poor-to-slight agreement. The *K's* using the same four combinations for vigorous PA were 0.03-0.55, and three of the combinations were considered to have slight-to-fair agreement. The Actical *K* analysis showed better agreement for both moderate and vigorous PA. The *K's* for the three Actical model/algorithms ranged from 0.14-0.26 for MPA and 0.27-0.34 for VPA. These agreements can be considered to be slight-to-fair (Viera & Garrett, 2005).

Bornstein et al. (2011a, b) explicitly discuss the problems of different accelerometer count cutpoints used to identify MVPA levels in pre-school children. In a secondary data analysis of 5 different studies focused on children aged 3-5 y using the ActiGraph 7164 monitor, the authors applied the various cutpoints and obtained estimates of MVPA that ranged from 40-269 min/d (Bornstein et al., 2011b). They then developed regression equations to relate count data for the ActiGraph accelerometer from one study to the other 4 using linear regression equations: 50 different equations in all! (There were so many because there were 2-3 equations for each study depending on the independent variables included in the regressions. All included the MVPA cutpoint for each study—either directly or as $MVPA^2$ or $MVPA^{0.5}$—and also may have included age of the subject and accelerometer wear time/day as independent variables. Even so, the absolute percent error of the equations when taken in pairs varied between 6.4% and 38.4%, with the median error being 15.8% (Bornstein et al., 2011b).

With count differences like those shown above for the same model accelerometer, a user has to wonder what really is being measured. When accelerometer counts are translated into minutes/day of MPA and VPA, very large differences are seen in MPA (but smaller differences for VPA) using the various algorithms. The potential for wide differences in time spent in moderate and vigorous PA can be large. It is results like these that led to my decision to only provide accelerometer model-specific results in Table 24 for daily time spent in MVPA. Other than undertaking the type of analyses done by Bornstein et al. (2011b), there simply is no rational way to reconcile the various cutpoints used to

estimate time spent in PA at any level. The user, therefore, has to be skeptical about some of the estimates presented in Table 24. Many of the MVPA estimates in the Table are considerably higher than those recorded in the United Kingdom, for example, for similar age/gender samples using the same accelerometer model (e.g., Davis & Fox, 2007).

Besides time spent in MPA and VPA, accelerometer data often are provided for METS intervals. Abel et al. (2011) relate accelerometer-based step rates to METS using a "best fit" non-linear regression approach. Their equation for females is:

$$METS = 0.00008048 * (Step\ Rate)^{2.288} \qquad R^2 = 0.91$$

And for males it is:

$$METS = 0.00004325 * (Step\ Rate)^{2.4528} \qquad R^2 = 0.79$$

The METS estimates were obtained from 19 subjects who were young (29 ± 7 y) and recreationally active. Individual resting VO_2 was used to calculate the step-specific METS estimates, so the problematic use of the 3.5 ml kg^{-1} min^{-1} usually used by exercise physiologists as basal metabolic rate—and the basis for the Compendium's METS estimates--is not an issue with their work. (The measured resting VO_2 was not too different than 3.5 ml kg^{-1} min^{-1}, by the way. It was 3.6 ml kg^{-1} min^{-1} for females and 3.3 ml kg^{-1} min^{-1} for males. That is usually not the case; see McMurray et al., 2014.) Abel et al. (2011) did not provide estimates of the daily time spent in MPA and/or VPA so their data also do not appear in Table 24.

Intra-Day Patterns of MVPA

Accelerometers can provide time-of-day PA data if the time pattern of counts/min is preserved when the data are downloaded. The information is there, but often is not analyzed. A few intra-day analyses have been published; one is for preschool children attending daycare (Van Dauwenberghe et al., 2012). They found that for both genders, MVPA is generally highest during the mid-afternoon to early evening time period. MVPA is generally quite low at other times. For school-age children, significant amounts of MVPA occurs during outdoor recess and lunch periods where the students can go outdoors, but it is still less than that occurring after school. In a study of children in grades 6-8 in two different types of schools (rural public and private), the percent of daily MVPA occurring at school varied from an average of 13.3% for males in public school up to 35.1% for males in private school. For females, the proportions were 20.0% for rural public schools and 18.8% for private schools. Thus, the pattern for the percent of MVPA by school location is quite variable by gender and type of school. The actual minutes/d recorded in this study appears in Table 24. A non-USA study looking at intra-day variability of objectively-measured PA is Verbestel et al. (2011); that paper is worthy of review. Additional intra-day information on MVPA using accelerometry appears below under locational considerations and sports participation information.

The next few subsections of this report provide information on using accelerometers to address various aspects of participating in MVPA activities, such as day-of-the week and seasonal effects, the impact of sports on MVPA levels, and the locations where MVPA activities occur. While these are all important factors to consider in trying to model MVPA, there is surprising little information on many of them. One study that does so is an EPA-funded analyses of data obtained by Harvard University (Arroyo, 2000). The TriTrac R3D accelerometer was used to estimate (among other things) the *percentage* of time during a 4-day (maximum) monitoring period in the Winter/Spring season that youth aged 11-13 spent in moderate (3.0-6.0 METS) and vigorous PA (>6.0 METS). Subjects were 251 students randomly selected from 10 schools in the Boston area (Arroyo, 2000). Only the data for the 0700-2200 time period were analyzed, and only for those days with <40% zero accelerometer counts. This study provides detailed information for a number of variables, such as day-of-the week, obesity status, and ethnicity. An abstract of these data appeared below in Table 25. The gender, season (February/March v. April/May), weekday v. weekend, and day-of-the-week differences in percentage of time spent in MPA were all statistically different (using the F-test at α=0.05) with p vales all <0.0025. Most articles are not as synoptic as the Arroyo (2000) article, providing data on some of these issues but not others.

Longitudinal (multi-day) and Day-of-the-Week Effects on MVPA Estimates.

One study that provided individual daily data was Almeida et al. (2011). They were interested in determining how many days of data were needed to adequately describe weekly average energy expenditure in a sample of women with rheumatoid arthritis (aged 50-60 y). It turns out that four days of accelerometer monitoring were needed to predict with 95% confidence 84% of the weekly variability in PAEE >3 METS, which most exercise scientists use as the low end of moderate PA. The authors also present data on the percentage of DTEE accounted for by MVPA ≥3 METS. The daily percentages varied from 9.6% (Monday) to 13.0% (Thursday). The weekly average DTEE accounted for by ≥3 METS activities was 11.4% (Almeida et al., 2011). If we assume that a MPA METS of 3 would occur only during an 8 h period of the day, it works out to be about 27 min/d, which does not seem to be too bad of an estimate.

Cook et al. (2012) also report daily MPA and VPA measurements for a 7-day period; there was little difference between weekday and weekend time spent in these levels of physical activity for adult minority females. Shen et al. (2013) found the same result in pre-school children across two seasons of the year. Their two-day accelerometer study found that the COV was 21% for time spent in MPA (Finn et al., 2002). A third multi-day study that reported daily MVPA frequency (but not min/d) data is Janz et al. (1995). They found little daily variation in MVPA frequency in individuals over the 6 days. Coleman & Epstein (1998) state that 3-4 days of accelerometer monitoring is needed in sedentary males to capture weekday/weekend differences.

Buchowski et al. (2004) is a study that reported aggregated MVPA data on a weekday/weekend basis; adult males expend more time in MVPA on weekends than on weekdays (about 3.4% more), but there is little weekend/weekday difference in adult females: only 0.2% fewer minutes/d of MVPA for them on weekends (Buchowski et al., 2004; graphical data). Farr et al. (2008) reported a similar finding. As usual, however, there is contrary evidence in PA studies. Corder et al. (2013) found a statistically significant difference in adult MPA for both genders between weekdays and weekends. So did Treuth et al. (2007), which reported statistically significant MVPA differences between weekdays and weekends for both normal-weight and overweight 11-12 y old girls. Weekdays had more MPA and VPA minutes than weekends. The reduction in weekend MPA time was ~36% and ~25% for VPA for the group as a whole, and for each weight cohort singly. The biggest differences in the weekday/weekend MVPA times occurred in the morning (6:30-10:00 am) and early afternoon (2:00-4:00 pm) periods (Treuth et al., 2007). The data from this study appears in Table 24.

Long et al. (2013) analyzed data from the accelerometer monitoring portion of the 2003-2005 NHANES survey for those years. Their paper only describes analyses undertaken on weekdays, but distinguishes between schooldays and not, and by time-of-day on schooldays. Summary data from their paper appear in Table 24. Males and females of both age groups evaluated (6-11y and 12-19y) participated in MVPA activities more on school days than on a non-school weekday: the percentage differences were between 11.2-19.8% among the four age/gender groups (Long et al., 2013). A difference between school- and non-school days during the week has also been found by Panter et al. (2011) and Ridgers et al. (2006).

One of best analyses of daily differences in MVPA time that I came across is Metzger et al. (2008). In their classification of the weighted adult population into 5 "natural classes," there were distinct daily patterns seen in the NHANES data of 3,462 people aged 20 y or more. When total mean daily MVPA is considered, there was very little daily difference in time/d for the two lowest classes totaling 88.7% of the

Table 25. Time spent in MVPA categories from ARROYO (2000)

Category	Sample Size (n)	% of Time Spent in MPA 3.0-6.0 METS		Estimated Time (min/d)		Estimated in VPA >6 METS		Time (min/d)	
		Mean	SD	Mean	SD	Mean	SD	Mean	SD
Females	107	3.41	2.31	30.7	20.8	0.09	0.21	0.8	1.9
Males	144	6.21	3.91	55.9	35.2	0.23	3.91	2.1	35.2
Weekday	183	5.16	3.87	46.4	34.8	0.18	0.53	1.6	4.8
Weekend	73	4.61	2.70	41.5	24.3	0.16	0.29	1.4	2.6
Monday	19	4.96	3.53	44.6	31.8	0.10	0.19	0.9	1.7
Tuesday	3	6.23	2.52	56.1	22.7	0.36	0.40	3.2	3.6
Wednesday	5	4.46	3.94	40.1	35.5	0.23	0.19	2.1	1.7
Thursday	45	5.24	5.05	47.2	45.5	0.33	0.95	3.0	8.6
Friday	111	5.17	3.43	46.5	30.9	0.12	0.28	1.1	2.5
Saturday	73	4.61	2.70	41.5	24.3	0.16	0.29	1.4	2.6
Obese	62	4.85	2.56	43.7	23.0	0.14	0.26	1.3	2.3
Non-obese	177	4.8	3.24	43.2	29.2	0.54	0.04	4.9	0.4
February	43	4.97	2.99	44.7	26.9	0.16	0.03	1.4	0.2
March	119	5.03	4.02	45.3	36.2	0.18	0.60	1.6	5.4
April	54	4.51	2.95	40.6	26.6	0.16	0.33	1.4	3.0
May	40	5.64	3.57	50.8	32.1	0.17	0.37	1.5	3.3

Source: Arroyo (2000). "A preliminary analysis of children's physical activity data." (Project report). Cambridge MA: Harvard University.

population. The next two highest classes, constituting about 20.3% of the weighted population, had a moderate drop-off of MVPA on the weekend. The highest class, however, had a major drop-off of MVPA on the weekend, especially on Sunday. They constituted only 0.9% of the weighted population. Thus, very active people during weekdays had a major reduction in MVPA time on weekends.

When MVPA time was classified into 10-minutes "bouts," an interesting change in the activity patterns occurs. While MVPA time/d estimates fall for all population classes due to the longer bout period (consecutive minutes of MVPA time), there appears to be a "weekend warrior" pattern in the third-lowest population class, the only one to increase MVPA time/d on both Saturday and Sunday, particularly Sunday (Metzger et al., 2008). They represent about 1.8% of the total weighted population. This finding indicates that there are complex patterns with respect to MVPA time/d in the population on a daily basis and by subgroup. These patterns will be difficult to replicate in our exposure models, although the "D & A" approach used in our recent models could possibly mimic some of them.

Mean Daily Estimates of MVPA and VPA by Day-of-week

	MVPA (min/d)		VPA (min/d)	
Day of Week	Mean	SD	Mean	SD
Monday	25.6	27.3	0.8	3.81
Tuesday	25.4	27.9	0.8	3.62
Wednesday	25.1	28.1	0.8	5.48
Thursday	25.5	28.0	0.8	3.21
Friday	24.6	26.5	0.7	3.51
Saturday	21.4	25.0	0.7	4.51
Sunday	19.4	23.5	0.7	4.22

Data from the Metzger et al. (2008) paper follows for the entire sample of 3,802 people; the weighted daily mean min/d for both MPA and VPA are the metrics of concern. All 5 "natural classes" of the population are included in these estimates. Note the very high standard deviations for the daily data, especially for VPA (about 570%)! The mean estimates do not reflect the "class" differences noted above, or the "weekend warrior" phenomenon either.

Another analysis of MVPA participation indicates that there was a weekday/weekend difference in the percentage of the sample undertaking ≥30 minutes of MVPA (Patnode et al., 2011). The 720 subjects with an average age of 14.7 y and in grades 6-8 were found to participate in ≥30 min of MVPA more on weekdays than weekends in both genders (♀: 30.9 v. 14.8%; ♂: 46.1 v. 31.2%).

An interesting study of MVPA participation by parent/child pairs on non-school days found low levels of MVPA in both groups and that only 16% of MVPA occurred when the child and parent were in the same location (Dunton et al., 2012).

Rarely does a study provide the participation rate (percent of the population studied) for MPA or VPA. One study that does is Marquez et al. (2011). Using a GT1M accelerometer, they evaluated MVPA in 148 Latino adults living in Chicago. One-hundred percent of their sample participated at least once in MPA during the 7 day study; however, that percentage decreases to 88% of males and 63% of females for VPA (Marquez et al., 2011). Data from this paper appears in Table 24. In a study of adults aged 70-89 y, 19-22% of the sample participated in MVPA at least once per week (Pruitt et al., 2008).

It should be noted that pedometer studies also generally show a higher number of steps on weekdays than on weekends, so there is consistency between the two types of PA monitors (Pelclová et al., 2010). There has been found a difference among days whether or not MVPA goals are achieved, so day-of-the-week is an important consideration in MVPA compliance (Moore et al., 2014).

Seasonal and Weather (Temperature and Precipitation) Impacts on MVPA

There obviously is a correlation between where MVPA occurs and weather/seasonal considerations (Suminski et al., 2008). Reviews of seasonality in PA, using accelerometer, pedometer, and questionnaire data indicate that PA (and MVPA) vary with seasons (Shephard and Aoyagi, 2009; Taveras et al., 2005). Obtaining precise estimates of these differences using accelerometer data, however, is difficult to find. Buchowski et al. (2009) evaluate accelerometer-derived time in MVPA for middle-aged females using a TriTrac R3D model and state that there are "larger seasonal differences during weekends than weekdays" (p. 258). They also found more variability in MVPA among the seasons on weekends compared to weekdays. They state that their results confirm Pivarnik et al. (2003)'s findings (Buchowski et al., 2009). A United Kingdom study found that overall PA increased as daylight time increased after adjusting for rainfall; out-of-home play accounted for 50% of this increase (Goodman et al., 2012a). Daylight time MVPA also was investigated in Winkler et al. (2005).

Fisher et al. (2005) described a two-season study (spring & summer) that found relatively small—but statistically significant—differences in MVPA behavior for the two seasons. Using an Actigraph GT1M accelerometer, Hopkins et al. (2011) found that all levels of PA decreased by 17 min/d from summer to fall. In another longitudinal study of MVPA in 294 youth aged 10-17 y living in Portland, average monthly temperature explained the most variability in MVPA time for males, but not females (Patnode et al., 2010). Female MVPA apparently was more affected than male MVPA by both temperature *and* precipitation. This pattern was also seen by Brodersen et al. (2005) in England. Shen et al. (2013) found that there were statistically significant differences in pre-school children's MPA after-school time between fall and

winter for both girls and boys, but only on weekends. They did not find a seasonality effect—or a location effect--on VPA either at school or afterward. MVPA time for all locations and day-of-the-week were less in winter than in the fall (Shen et al., 2013).

Pedometer studies of PA also indicate a seasonal and monthly effect on the number of steps taken by adolescents, both in this country and in Europe (Pelclová et al., 2010). However, Rich et al. (2012) state that there currently is insufficient evidence from accelerometry studies to conclude that significant seasonal differences in PA is occurring.

Locational Aspects of MVPA

Only a few accelerometer studies disaggregate MVPA by location. Those that do have shown that MVPA is generally higher outdoors than indoors (Burdette et al., 2004; Sallis et al., 2000; Wheeler et al., 2010), and higher during daylight hours, on days with mean daily temperature between 20-75 ^{o}F, and on days with little or no rain (Feinglass et al., 2011; Harrison et al., 2011). Welk et al. (2000) state that time spent outdoors is strongly predictive of MVPA (and overall physical activity) in children. Tran et al. (2013) provide pedometer data that support this observation. They state that outdoor recess resulted in more MVPA steps/period than indoor recess, and the differences are statistically significant at $p<0.0001$ (Tran et al., 2013).

A study of children aged 6-11 y found that the outdoor and neighborhood locations had the highest average percent of time at MVPA than any other location (Kneeshaw-Price et al., 2013). Boys had slightly higher percentages of time spent outdoors than girls, and children of both genders aged 6-8 y had higher percentages than children 9-11 y. Around 43-49% of time spent outdoors or in the neighborhood by children (both genders) was spent in MVPA, while 31-41% of time spent in the neighborhood was at that level (Kneeshaw-Price et al., 2013). In a study of pre-school children in Malmo, Sweden and Raleigh NC, the Swedish kids spent more time outdoors (47% v. 10%), but MVPA counts were significantly higher outdoors than indoors in both locations (Raustorp et al., 2012). Children aged 10-12 had higher activity counts outdoors than indoors (Stone & Faulkner, 2014). In middle-aged adults participating in an accelerometer/GIS study, the percent of time spent in MVPA activities away-from-home is 5.6 times higher than that incurred at home, and about 35 times **higher** [sic.] on weekends than on weekdays (Ramulu et al., 2012; n=35, mean age=38 y, both genders).

An interesting study that looks at the locational aspects of MVPA, as well as seasonal effects, is Oreskovic et al. (2012). The study involved 24 middle-school children in Massachusetts for 3 seasons, and included 5 weekday and 2 weekend days per season. MVPA data were monitored with an Actigraph GT1M accelerometer and a Forerunner 201 GPS mapping device, measuring locations at a 1 minute interval. Results of their MVPA monitoring follow; there was no MVPA time recorded in "car," another specific location that was included (thank goodness!).

Percent of Total Daily MVPA Time by Location and Season

Location	Winter	Spring	Summer
Home	43.1	33.8	12.3
School	12.3	8.8	-
Indoor Other	15.4	5.1	10.2
Park/Playground	-	8.5	57.4
Street/Walking	19.2	43.8	11.1
Outdoor other & unknown	11.0	0.0	9.0

Note: - is <0.1%

The locational impacts on MVPA in school or out seems to depend upon gender; after-school MVPA is higher than in-school MVPA is females, but not in males (Panter et al., 2011). Recess time spent in MVPA seems to be highly variable, both within an individual school and among a set of schools in the same general area. In a "Ready for Recess" study of 393 children aged 8-11y attending 12 schools that emphasized active recesses, individuals spent between 15-63% of recess time at MVPA levels, but this was highly dependent upon the school itself; the mean time spend at MVPA at the various schools varied between 12-33% (Saint-Maurice et al., 2014). Wheeler et al. (2010) found that children aged 10-11y in Bristol, UK spent 13% of their time outdoors, which accounted for 35% of their total MVPA time. Most of the time spent outdoors (85%) was not in a "greenspace" (park or other location).

Using American Time Use Survey data, Dunton et al. (2012) report that there is a gender difference in where MPA and VPA occurs: MVPA occurs more frequently and for a longer duration at "someone else's house" (55%) and school (64%) for boys, and "outdoors" (54%) and at home (49%) for girls.

Participation in Sports and Recreational Activities

We know from observational and clinical studies that accelerometer counts are correlated with both heart rate monitoring and VO_2 consumption (Coe & Pivarnik, 2001). Thus daily accelerometry counts should be higher in people who participate in sports and high-intensity recreational activities than those who do not. That is seen in the data. People who participate in high-intensity sports and physical activities have many more min/d of MVPA than does the rest of the population regardless of gender or age. Between 92-97% of youth (both genders) aged 7-14 that participate on soccer teams have more than 30 min/d in MVPA just in that activity (Leek et al., 2011). A goodly percentage even reaches 60 min/d of MVPA just in that sport: around 30% (Leek et

al., 2011). The percentage drops rapidly, however, for less intense sports; only 50-75% of youth playing organized baseball get 30 min/d of MVPA in that sport. There is not much difference in these percentages when the samples are disaggregated into two age groups: 7-10y and 11-14y. The importance of organized sports contributing to MVPA in males, but not in females, is also discussed in Patnode et al. (2010) and Sallis et al. (2000).

A study that evaluated the percentage of time spent in two sports activities designed to elicit cardiovascular fitness gains in middle-school girls using the TriTrac R3D accelerometer indicates that MVPA was attained about 56% of the time spent participating in sports activities (Arnett & Lutz, 2003). Specifically, the 95th CI of time spent at MVPA for soccer was 52.6-61.0% and 51.2-59.6% for field hockey regardless of skill level of the girls involved. There was a definite monotonic trend in this CI by skill level, with the highest-ability level girls spending more time in the MVPA interval than those of lower ability levels (Arnett & Lutz, 2003).

Wickel & Eisenmann (2007) looked at the relative and absolute impact of organized sport on total daily PA and MVPA in 113 6-12 y boys. The GT1M accelerometer was used, so swimming activities were excluded from the study. The average monitoring period was 811 min/d. Sports played included soccer, basketball, and flag football. Participants averaged 110 min/d in MVPA, 40 min/d of it in VPA. There were differences in MVPA time among the different sports. When PA time was disaggregated into time spent in organized sports, PE class, and recess, sports accounted for 26% of MVPA, while PE class and recess accounted for 11% and 16%, respectively. The remainder of MVPA time (47%) was attributed to "unstructured activities" (Wickel & Eisenmann, 2007). There was a significant difference in MVPA time between a "sport day" and a "non-sport day," as might be expected: 125 min/d versus 95 min/d.

A study whose MVPA data do not fit into the Table 24 format is Davison & Jago (2009). It is a longitudinal study of 96 girls who have MPA and VPA on a min/d basis when they were 13 and 15 y old. The reason why their data do not fit is that it applies only to girls who were active at age 13 y, meeting at least 30 min/d of MVPA originally. (We have called these people "doers" in the past.) They were disaggregated into two groups: those that maintained at least that level of MVPA (n=24; 25% of the original sample) and those that did not (n=72; 75%). The time spent per day (in minutes) in MVPA for these groups of girls appears below:

	Maintained PA (n=24)		Decreased PA (n=72)	
Age	MVPA	VPA	MVPA	VPA
13	45.7 ± 13.0	5.9 ± 3.7	32.2 ± 12.9	3.6 ± 3.6
15	44.1 ± 12.9	5.0 ± 4.3	20.3 ± 10.5	1.5 ± 1.9

The differences between the two groups are statistically different at $p<0.01$ for both ages and PA levels. Obviously, the maintainers were more active at age 13, and did not reduce MVPA much over the two year period. Questionnaire studies of longitudinal "tracking" of MVPA show much the same pattern: there is gradual fall off in time spent in MVPA in most everyone, but it is less in the more committed individuals (DeBourdeaudhuij et al., 2002). Another analysis of MVPA tracking in children aged 10-12 also showed moderate correlations of MVPA over the years (Dencker et al., 2013).

Health and Other Impacts on MVPA

People having health problems, intellectual disabilities, or are obese usually have lower accelerometer counts than the general population (Coleman et al., 1997, 1999; Cook et al., 2012). MVPA has been shown in accelerometer studies to be inversely proportional to adverse health conditions (COPD: Loprinzi et al., 2014d; diabetes: Loprinzi et al., 2014b; and the hearing-impaired elderly: Gispen et al., 2014). MVPA is inversely related to a person's weight status (Kitzman-Ulrich et al., 2010; Treuth et al., 2007). A study of adults with intellectual disabilities indicates that MVPA is lower for them than for normal-functioning individuals; the study also shows that MVPA is less in overweight/obese (BMI > 25 kg/m^2) than non-overweight adults (Barnes et al., 2013; Dixon-Ibarra et al., 2013). Ethnicity and type of house that the subjects resided in were not important correlates of MVPA. Valid data for this study was defined to be >8 h/d of non-zero count data for 4 or more of the 7 d monitoring period; MVPA was defined using the Freedson et al. (1998)/Matthews et al. (2002) count criteria. Only 23.7% of the sample with "valid" accelerometry data met the recommended PA Guidelines (Barnes et al., 2013).

A study of adolescents with fibromyalgia (Kashikar-Zuck et al., 2010, 2013) provides unique data for the ratio of 5-min peak-to-mean activity levels for the sample of 104 boys & girls aged 14.9 ± 1.8 y. The ratios were 19.0 for girls and 16.0 for boys, indicating that there is a fair amount of temporal variability in accelerometer count measures for 5 minute averages in the adolescent population. This ratio is probably even higher in healthy youth as fibromyalgia is a chronic pain condition associated with impaired physical functioning (Kasikar-Zuck et al., 2010). Note that ratios of this magnitude are similar to $METS_{MAX}$-to-$METS_{SITTING}$ ratios seen in the literature.

An analysis of 2003-2006 NHANES accelerometer data for pregnant women indicates that the trimester of pregnancy did not significantly affect time spent in MPA, MVPA, or VPA activity levels using "Swartz MVPA count cutpoints", but so did for the 2nd/3rd trimesters using the "Triono cutpoints" (Evenson & Wen, 2011). This finding highlights the importance of count cutoffs in affecting findings of a study (see above).

A number of studies have investigated the effects that menopause has on time spent in MVPA. If data from these studies are compatible with the format of Table 24, their information appears in it. However, many of these studies have data that do not fit into the Table's format. According to Sternfeld et al. (2005), over 60% of women coming into or finishing menopause have >60 min/d or MPA. Since their ages were in the range of 48-55 y, these proportions are quite high. Average minutes of MVPA estimates were not provided.

Readers interested in additional information on the impact that health-related conditions has on MVPA should consult the work of Sternfield et al. (1999, 2002, 2005), Bonen et al. (1981), Harlow & Matanoski (1991), Kushi et al. (1997), Melzer et al. (2010), and Stevenson (1997). An interesting overview of physical activity during pregnancy is contained in Artal (1992).

Ethnicity does not seem to affect MVPA levels significantly. A 7 d accelerometer study found that black/while ethnicity did not affect time/d spent in MVPA (Dong et al., 2010).

Number of days needed to adequately characterize MVPA

Variability in MVPA over days leads to the related issue of ascertaining how many days of data are needed to reliably characterize a cohort's mean/SD MVPA statistics given the inherent variability in individual daily MVPA time. Addressing this issue depends largely on the pattern of daily PA in a group of subjects, but it also is affected by systematic and random sampling error in the cohort sample itself (Baranowski et al., 2008) and by which monitoring method is used for determining MVPA. The Baranowski article discusses inherent variability in MVPA and how many days of data are needed to describe (with a known degree of confidence) grouped physical activity levels given both intra- and inter-individual variability in participation rates.The largest source of MVPA variance estimates in healthy adults as a whole is due to inter-individual variability (55-60% of total variance), but intra-individual variability accounts for 30-45% of total variance (Matthews et al., 2002). In their sample, only 1-8% of total variance is attributed to day-of-the-week variability.

Multi-day accelerometer data rarely are reported on a daily basis; only MPA and/or VPA time/d averaged over the entire monitoring period is reported. Papers that analyze how many days of data are needed to "adequately" estimate the mean time spent in various categories of physical activity usually use the ICC statistic and the Spearman-Brown Prophecy formula to estimate the number of days to attain a reliability coefficient of 0.8, a common "reliability target" (Ridley et al., 2009). (We have done so in the past; see Xue et al., 2004.) When MVPA time is evaluated, the number of monitoring days needed, for either objective monitors or for questionnaires, turns out to be between 3-9 days (Ridley et al., 2009). There are, however, a number of issues associated with the ICC-based procedure involving addressing inherent variability versus bias that usually are not addressed well, so these analyses have to be used with caution (Ridley et al., 2009). Janz et al. (1995) state that 5 d of accelerometer data is needed to attain a reliability coefficient of approximately 0.8 for a population-average estimate of MPA and VPA; one fewer day was needed to attain the same reliability coefficient with respect to sedentary activity.

At least 5-6 days of accelerometer monitoring is needed to "minimize the intra-individual variance [in estimates of MPA and VPA] to a reasonable degree" (Gretebeck & Montoyne, 1992; p. 1167). Hart et al. (2011) state that 3 days of accelerometer data are needed to "accurately predict" PA levels in elderly people aged 55-86 over any 21 day period using a Spearman-Brown Prophecy formula with a 0.80 reliability coefficient. Using the same reliability criterion, Kim (2006) states that at least 4 days of monitoring was required to account for weekday/weekend activity patterns, as do Kim & Kim (2009) and S-Y Kim & Yun (2009). If only average daily time spent in MVPA is of interest, then 2 days of accelerometer data are required (using the ActiGraph-7164 model). One study of variability in PAEE indicates that 4 days of complete accelerometer data would be needed to predict variability in METS≥3 activities with a probability >84% Almeida et al., 2011).

These findings are different from those reported in Herrmann et al. (2014), which states that their review of a study by Jerome et al. (2006) that applied the Spearman-Brown prophecy (S-B.P) formula to obese subjects having at least 6 h/day of valid data, indicates that 5-6 days of data are required to adequately address variability in the sample. A similar theme is struck by Levin et al. (1999), who state that 6 days of accelerometer data are needed to obtain a 0.80 reliability. According to Penpraze et al. (2006) seven days of accelerometer data (using the 7164) are required to attain a reliability of 80%. Hinkley et al. (2012) may provide the key to understanding these differences in their study of the number of days of accelerometer data needed to reliably determine time spent in MVPA; their study focused on preschool children and also used the S-B.P formula at 3 different reliability levels (0.7, 0.8, and 0.9). They found that fewer days of accelerometer data were required when the number of h/d was increased from 8 to 10 (Hinkley et al., 2010). This is a logical finding.

The papers by Baranowski et al. (1993), Coleman & Epstein, 1998; Janz et al. (1995), Levin et al. (1999), Matthews et al. (2001), Patterson (2000), Trost et al. (2000), Tudor-Locke et al. (2005), and Ugrinowitsch et al. (2004) are worthy of review concerning the question of how many days of data are needed to reliability characterize physical activity in the population.

Pedometers

Pedometers are an accelerometer-like step counter that are used mostly to estimate the amount of walking that a subject undertakes. They can be used to measure steps and distances undertaken, and generally are worn on or around the waist (Bassett & Strath, 2002). Apparently the first

reported use—in one person—of a pedometer to measure free-living activity occurred in 1926 (Stunkard, 1960). The first multi-subject research effort to use modern miniaturized pedometers to estimate physical activity in infants and children that I could find is discussed in a 1968 article that describes a 1959 study of a modified wrist watch to record movement (RQ Bell, 1968). The wrist-watch pedometer was placed on the feet of children aged 2 y in a pilot study of 37 children. Another early pedometer study of activity in obese girls occurred in 1960, and found that they walked as far as normal-weight girls during a two-week period (Stunkard & Pestka, 1962).

Some early studies of pedometers and physical activity used an "actometer" (Eaton & Duroski, 1986; Eaton et al, 1988; Redmond & Hegge, 1985) which does not seem to be currently available. Studies that compared pedometer estimates versus oxygen consumption or other measure of energy expenditure include Eston et al. (1998) and Hendelman et al. (2000). Reviews of pedometer studies are contained in Clemes & Bidddle (2013) and Melanson et al. (2004). Theoretical shortcomings of using pedometers to estimate daily PA in children (at all activity levels) are discussed in Eisenmann & Wickel (2005). A "steps/minute cadence criterion" has been developed to be able to relate pedometer data to moderate intensity physical activity but data are rarely reported using it (Harrington et al., 2011, 2012). Reactivity to wearing a pedometer and thereby biasing its use does not seem to be a problem (Matevey et al., 2006), although their study may not have been able to test all aspects of the issue (Beets, 2006).

Pedometers generally are relatively inexpensive and a number of models exist from a number of manufacturers; seemingly the preferred model for exercise physiologists is the Yamax Digiwalker SW-200 due to its reliability (Bassett & Strath, 2000; Brusseau et al., 2011; Gaydos et al., 2011; Montoye et al., 1996). Other commercial pedometers that have been evaluated by U.S. exercise physiologists are the "Freestyle Pacer," "Eddie Bauer," "L.L. Bean," the New Lifestyles NL2000, the "Stepwatch", the Omron HJ-112, the "Stepping Meter," the Sport Brain iStepX1, and the "Accusplit" (Bassett Jr., et al., 1996; Beets et al., 2007; Bjornson et al., 2007; Busse et al., 2009; Cadmus-Bertram et al., 2014; Cavanaugh et al., 2007; Clemes et al., 2010; Crouter et al., 2003; Dauenhauser & Keating, 2011; DeCrocker et al.,2006; Dueker et al., 2012; Foster,RC et al., 2005; McKee et al., 2012; Nunez-Gaunaurd et al., 2013; Oh et al., 2012; Pettee et al., 2008; Raustorp, et al., 2007; AM Swartz et al., 2009; Swift et al., 2012; Tudor-Locke et al., 2004). C. Tudor-Locke and colleagues have probably written the most articles about pedometer applications on various age-gender cohorts. See, for example, Tudor-Locke (2001a & b, 2002, 2005, 2006, 2008). A "free" pedometer in the iPhone—called iPedometer—exists, but has been found to be inaccurate (Bergman et al., 2012). There even are pedometers for the blind that provide information via voice announcements; these brands/models are: Centrious, TALKiNG, and Sportline Talking (Beets et al., 2007). A review and meta-analysis of 26 pedometer studies appears in Bravata et al. (2007).

While the SW-200 is used a lot (e.g., Bennett et al., 2006; Fuller, 2000; Kang et al., 2009, 2012), it only counts the number of total steps taken during an elapsed time period. Other pedometers can record steps, distances, and an estimate of EE expended, and save these values by time of day over a 7-d period (Montoye et al., 1996). These attributes allow the pedometer to estimate and store steps/minute (SPM) data which have been shown to be reasonably correlated with oxygen consumption/energy expenditure level in subjects (Graser et al., 2009). Thus, the newer and more sophisticated pedometers purportedly provide essentially the same type of information as an accelerometer at a far lower cost. Pedometers have the same "black box" problem of estimating EE from steps/distance taken as does the accelerometer's counts-to-EE (Leiper & Cralk, 1991). I did not find any pedometer study that reported time spent in MPA or VPA, however defined; therefore, there are no pedometer data listed in Table 24. If daily step rates are high, some of them occurred at higher PA levels, but the specific time spent in MPA and/or VPA is not provided in pedometer studies to date.

In general, there are significant differences found among pedometers in estimating both the distances traveled and the steps taken; there also is a significant amount of inter-instrument variability among pedometer units for the same model unit (Bassett Jr., et al., 1996; Beets et al., 2007). Some of the inter-instrumental variability problem is due to weak quality control during the manufacturing process, differences in exactly where the pedometer is placed on the body, tilt changes during the study, and differential sensitivity to the type of activity undertaken (Beets et al., 2007). Gait problems in the elderly hamper the use of pedometers and cause high error rates when compared to oxygen consumption estimates of energy expenditure (Cyarto et al., 2004).

There are numerous articles in the literature that recommend a specific number of steps/d to attain and maintain a "healthy lifestyle." They are promulgated by both governmental and non-governmental organizations. Since these recommendations are not tied *directly* to time spent in MVPA, they are not reviewed in detail here. Some are specific to children (Duncan et al., 2006), to adults (Tudor-Locke & Bassett, 2004), or to people with various disabilities. Duncan et al. (2007) state that these recommendations should be based on percent body fat measurements—a surrogate for fitness—to be more realistic and apropos. I have never seen such a distinction made in articles making pedometer recommendations.

The number of steps/d needed for "healthy body composition" in 6-12 y children has been found to be 12,000 in girls and 15,000 in boys (Tudor-Locke et al., 2004). Colley et al. (2012) indicate that children in that age bracket in Canada recorded between 11,200-15,212 steps/d on average,

not quite meeting the recommended levels. Canadian step guidance for children aged 3-5 y is 6,000 steps/d (Gabel et al., 2013).

In a study of elementary school children in 2nd-5th grades, a SW-200 was worn during classroom periods, recess, and PE classes (Barfield et al., 2004). Even though recess had the shortest duration of the three periods, its mean steps/minute (SPM) estimates were the highest: 43-45 SPM on average, slightly more than PE classes, which were ~40 SPM. Classroom time only had 7-8 steps/min (Barfield et al., 2004). Analyses of the data were not reported to determine if the observed differences were statistically significant or not. Steps/d were higher in elementary school children on days with PE periods of 60 min in duration than those having 30 minutes of PE or on non-PE days (Dauenhauer & Keating, 2011). However, about half of PE class time is spent in fairly sedentary activities (Fuller et al., 2009) as measured by a SW-200 monitor. Erwin et al. (2012) state that a 15-minute recess accounted for 17-44% of all school steps in elementary school aged children. The larger the school campus, the more MVPA occurred during outdoor recess (Cradock et al., 2007). In another study of grade school children, steps-out-of-school were about the same as those during the shorter school time period (Cox et al., 2006).

Seasonal differences in the steps taken by 1st-5th graders using the Walk4Life pedometer are discussed in Beighle et al. (2008). Steps taken in school versus out-of-school was not distinguished. They found that steps/day were significantly higher in the spring (May) than in winter (February) for both genders. There were seasonal difference in step counts between seasons for 3-4 y old children also (McKee et al., 2012). In an interesting study of the number of steps taken/day during two seasons of the school year, it was found that there were no significant differences between fall and winter seasons in steps taken in and on school grounds, but there was in out-of-school steps (Beighle et al. 2012). The pedometer used was the Walk4Life MLS 2505, and the subjects were 112 students in grades 3-5; 4 days of monitoring were collected with between 10-12 h/d of valid recorded data. The fall period was in October 2007 and the winter period was in February 2008; no snow accumulation occurred during the February session, so severe weather was not an issue. Another study of weather (as reflected in seasonal differences), there was a statistically-significant interaction among weather, month-of-the-year, and day-type that affected PA levels (Chen & Mao, 2006; Clemes et al., 2011). Less MVPA is undertaken during the "cold months," but this seasonal affect is moderated by day-of-the-week effects.

Pedometer data were analyzed to distinguish between the number of steps/min taken during recess vrs. out-of-school time (Beighle et al., 2006). Steps/min taken were not too different for the 9 y old boys and girls during these two periods. Girls had 98 SPM during recess and 90 SPM out-of-school; boys had 108 SPM during recess and 93 SPM out-of-school (Beighle et al., 2006). However, since the out-of-school time was 6.6-6.8 times longer in duration compared to recess time, out-of-school steps cumulatively were much greater: 5,754-7,136 steps vrs. 918-1,262 steps for recess (Beighle et al., 2006). These differences were statistically significant. Girls spent 63% of their recess time engaged in PA, while boys spent 78% of their time doing so (Beighle et al., 2006). Note that none of these results are focused on MPA, MVPA, or VPA cutpoints; they include total cumulative steps taken for the time periods of interest.

A comparative pedometer study of children aged 10-13 y with cerebral palsy (CP) and those developing normally indicates that the number of steps taken in a day is inversely proportional to severity of CP, as measured by gross motor skills (Bjornson et al., 2007). The difference between normal children and the most severe class of CP children was statistically significant (6,739 steps/d versus 4,222, on average), with a large difference in the range of steps/d (SPD) seen in the two cohorts: 6.123-7,355 SPD for normally developing as opposed to 3,739-4,749 SPD for youth with cerebral palsy (Bjornson et al., 2007).

Kang et al. (2009, 2012) describe interesting simulations of pedometer data from 23 adults, mostly female, that had a full-year of pedometer monitoring using a Yamax SW-200. In their first analysis, they found that 6 randomly-sampled days were needed to describe the daily-average steps taken in a year with an ICC of 0.8 (Kang et al., 2009). If the goal was to reduce the mean absolute percentage error (MAPE) to 10% of the daily mean, then 14 randomly-selected days were required. If non-random consecutive days were used, then 30 days of data were needed for the same MAPE goal (Kang et al., 2009). In the second analysis, they looked at monthly patterns within the same dataset, which was obtained in the Knoxville TN area. April through October mean steps/d were consistently higher than those during the other months: about 1,000 steps/day on average (~10% higher) (Kang et al., 2012). The number of pedometer monitoring days needed per year to reduce MAPE varied somewhat by season, from 5 days in the spring to 7 days in the summer and fall; 6 days provided the lowest MAPE is winter (Kang et al., 2012). Generally 4-7 days of pedometer monitoring in "free-ranging" individuals is undertaken, but at least 7 d are required to reliability capture daily variability in MVPA (Clemes & Griffith, 2008).

In a study of the OMRON HJ-112 pedometer, Kim (2006) and Kim & Kim (2009) found that to correctly account for intra- and individual variability in personal activity patterns and instrument variability (at a reliability coefficient of 0.8), requires that monitoring be performed for 4 weekdays, 6 weekend days, and for 8 weekday/weekend days combined. The population mean daily coefficient of variation (COV) for pedometer step counts in 6-12 y old males observed in 3 countries (US, Sweden, and Australia) is 22% (Wickel et al., 2007). Individual COV's varied between 2-88%, quite a range. In addition, the authors found significant differences in steps taken by day-of-the-week (weekdays>weekends) and

seasons (summer>spring>winter). The authors state that they found very similar patterns in college-aged students using a total daily energy expenditure monitoring approach.

Multiple Methods to Capture MVPA

One type of multiple methods study is to apply the same monitor to different part of the body and integrate the measures into a composite metric (Choi et al., 2010). That has been done frequently in monitoring method evaluation studies and will not be considered further here. More interesting are those applications that involve combining two or more objective methods to estimate energy expended by an exercising individual. Combining methods has been shown to be more accurate in the laboratory when compared to indirect calorimetry (Tikkanen et al., 2014). Combining accelerometers and pedometers is relatively common (Kilanowski et al., 1999), and some manufacturers combine both in one instrument (Dijkstra et al., 2008). Others combine heart rate monitoring with accelerometry (Castiglioni et al., 2007). One interesting study combined heart rate monitoring, accelerometry, and an energy-measuring garment that estimated energy expended by leg muscles using textile electrodes (Tikkanen et al., 2014). To date, combining monitoring approaches to estimate PA expended seems to be confined to laboratory or relatively small-scale investigations only.

The following combinations of non-questionnaire monitoring methods have been reported in the peer review literature; not all of them are U.S. studies:

1. Accelerometer and GPS: Almanza et al., 2012; Chaix et al., 2014; Colby et al., 2014; Dunton et al., 2012; Durand et al., 2011a,b; Evenson et al., 2009, 2013; Frank et al., 2005; Gell & Wadsworth, 2014; Hermann et al., 2011; Hongu et al., 2013; Jerrett et al., 2013; AP.Jones et al., 2009; Kang et al., 2013; Klinker et al., 2014; Maddison et al., 2010; Nguyen et al. 2013; Norland et al., 2014; Oliver et al. 2010; Oreskovic et al., 2012; Quigg et al., 2010; Rainham et al., 2012; Ramulu et al., 2012; Rodriguez et al., 2005, 2011; Scheck et al., 2011; Southward et al., 2012; Troped et al., 2007, 2010; Wheeler et al., 2010; Zenk et al., 2011.
2. Accelerometer and the use of *post*-hoc GIS information: McCorrie et al., 2014; Patnode et al., 2010; Scott et al., 2007; Wheeler et al., 2010; Wieters et al., 2012.
3. Accelerometer with GPS and heart rate monitoring: Panter, 2014.
4. Accelerometer and a heat flux/temperature monitor (the SenseWear monitor): Almeida et al., 2011.
5. Accelerometer with a heart rate monitor: Calabró et al., 2014; Brage et al., 2004, 2005, 2007; Fudge et al., 2007; Strath et al., 2001, 2003a,b.
6. Accelerometer with a light-level sensor to determine if the subject is indoors or outdoors: Flynn et al., 2014; Gehrman et al., 2004.
7. Accelerometer and direct observation in a single location: Gao et al., 2011; Huberty et al., 2011a,b; Mukeshi et al., 1990; Nelson et al., 2011; Pate et al., 2004; Sacheck et al., 2011; Saint-Maurice et al., 2011, 2014; Sarkin et al., 1997; Schuna Jr. et al., 2013b, c; Trost et al. (2008).
8. Accelerometer with a PA diary: Goodman et al., 2011, 2012b; Murphy et al., 2012; AD Stein et al., 2003.
9. Accelerometer and a "SenseCam" (an automated picture-taking camera): J.Kerr et al., 2013.
10. Pedometer with a mobile phone- or paper-based activity diary: Fukuoka et al., 2011; Strycker et al., 2007
11. Pedometer and a heart rate monitor: Graser et al., 2009; Scruggs et al., 2005.
12. Pedometer with direct observation in a single location: Hustyi et al. 2011; Scruggs, 2007, 2013; Scruggs et al., 2013.
13. Heart rate monitoring and GPS: Duncan, JS et al., 2009; Fjørtoft et al., 2009; Panter et al., 2014; Worringham et al., 2011.
14. Heart rate monitoring and activity diary: Campbell et al., 2010; Kalkwarf et al., 1989.
15. Heart rate monitoring with direct observation: Horvat & Franklin, 2001; O'Hara et al., 1989.
16. Observation and GIS: Suminski et al., 2008.

While not a PA-monitoring method *per se*, GPS units have been attached to individuals to distinguish if a subject is outdoors or not, and to record movement in space, mostly to distinguish among walking, running, bicycling, and motor vehicle travel (Cho et al., 2011; Dueker et al., 2014; Duncan, MJ et al., 2007; Evenson et al., 2009; Maddison et al., 2009; Rainham et al., 2008; Wiehe et al., 2008a, b). Along this line, a gyroscope has been combined with a microphone and a camera to record MVPA and other activities (Clarkson et al., 2000). The microphone is used to record the specific activity being undertaken. Apropos, EPA funded RTI to record activities via a voice-activated recorder in 9 people who were simultaneously hooked up to a heart rate monitor. The study was unsuccessful for a number of technical reasons related to failure of the HR monitoring equipment and problems with the voice recognition software used.

There are scores of papers that evaluate accelerometers and pedometers against self-reported estimates of MVPA, either from questionnaires, diaries, or perceived estimates of the "scale of work" involved in an activity (RPE). They mostly involve method evaluation (or "validation") studies of a particular instrument. None of them is included in the list noted above or in Table 24.

11.0
Activity-Specific Energy Expenditure Estimates

Papers contained in file cabinets located in E-253 contain over 250 articles providing activity-specific energy expenditure (EE_{ACT}) estimates. These papers can be used to compare selected EE_{ACT} estimates with those appearing in the three Compendium articles (Ainsworth 1993, 2000, 2011) that only provide a single METS value for each listed activity. Some of the papers in the cabinets are incomplete, in that only their data and first page were copied without the rest of the article. (In the old days copying a paper was a time-consuming and expensive, manual process so to save time only important portions of a paper were copied.) More papers, however, and most of the newer articles, are stored as PDFs located on the computer in Room E253. Papers containing EE_{ACT} estimates are noted with an "EEa" on its entry in my "working" bibliography, so articles containing activity-specific EE data can be searched by using "EEa" in the "Find" function of Microsoft's Word program. These papers include both U.S. and non-US studies, since the relative nature of work needed to accomplish a specific task is not necessarily (or probably) culturally-dependent. The country of origin of most papers also is provided in the working bibliography.

I call my bibliography a working one because the list of authors has been truncated to (generally) only two names and the rest are cited as "et al." This was done to save space (lines) while still being able to find an article by the first author's name. In fact, the second author's name is provided only to distinguish between/among numerous articles published in the same year by the same author. And that happens frequently.

It would be a yeoman's job to abstract EE_{ACT} data from these ~250-300 articles and list them in a table like Table 1 for $VO_{2\,MAX}$ or Table 7 for V_E. Since many of the articles provide EE_{ACT} estimates for a number of specific activities—and differing experimental rates of doing a single activity, organized by age/gender cohorts--such an EE_{ACT} table might be longer than Tables 1 and 7 combined. For instance, Agiovlasitis et al. (2012) provide EE_{ACT} information for 6 different walking speeds for 2 groups: 12 different EE_{ACT} estimates from one article! I just don't have time at present to undertake a systematic review needed to compile a complete EE_{ACT} dataset from the information currently on hand. (And there may be additional articles "out there" that have not been systematically evaluated.)

The Compendium's single METS estimate for each activity, which also has been carried over to the ATUS and related databases (Tudor-Locke et al., 2009, 2010, 2011a, b), is obviously a problem since it ignores both intra- and inter-individual variability in EE_{ACT}. This shortcoming is surprising given that early energy expenditure articles depict a wide range in EE_{ACT} for individuals undertaking common activities; see, for instance, Figure 13-1 in Åstrand & Rodahl's 1986 *Textbook of Work Physiology*. Other early compilations of EE_{ACT} showing a range of measured energy expenditure across subjects undertaking specific activities is Durnin & Passmore *Energy, Work and Leisure* (1967) and Durnin & Namyslowski "Individual variations in the energy expenditure of standardized activities" (1958). Thus, there was ample information available to the authors of the Compendium in 1993 on variability in EE_{ACT} among individuals.

Sallis (1991) states that "standard lists" of METS are inaccurate, biased toward adults, and inapplicable to children. EE is underestimated in children if adult values are used due to the prevailing over-estimate of REE. The "standard" value of REE of 3.5 mL O_2 kg^{-1} min^{-1}, which is especially incorrect for children, is partly responsible for the $METS_{ACT}$ under-estimates (McMurray et al., 2014). If nothing else, Sallis (1991) posits that METS estimates for the same activity decrease with age due to changes in gait, even though cellular metabolic considerations associated with undertaking common activities are not very different by age.

It was for these reasons that my 1998-9 work on fitting METS distributions to the Compendium values involved reviewing what EE_{ACT} data that I could find and "mapping" them onto the highly aggregated time use codes used in CHAD. The resultant statistical distributions of the mapped values were then fitted and analyzed. See McCurdy (2000) for a brief discussion of the procedure used. I had considered then that this approach was a temporary one, and stated so in the article. Unhappily, however, we in EPA have never gone back with a synoptic "hard look" at the METS data for different activities to update and improve upon what was done earlier. Given the rather crude CHAD codes that had (and still has) to be used relating to physical activities, it may not really be worth the effort to redo CHAD METS codes, but at least the issue should be investigated to determine its impact on exposure-modeling results. One promising effort might be to make EE_{ACT} relative to both $METS_{MAX}$ limits as well as the METS=1 basal rate (the "metabolic chromotropic relationship, in other words). See Sections 6, 7, and Appendix C.

At any rate, instead of developing a synoptic table of EE_{ACT} to depict inherent population variability in $METS_{ACT}$, I will simply discuss some of the general findings regarding METS (and/or VO_2 or EE_{ACT}) COV's seen in the literature

for specific activities. The COV's presented, however, are themselves problematic given that they *assume* a normal distribution and other sampling assumptions that rarely are discussed in the exercise physiology literature. Probably a log-normal distribution would generally be a more appropriate sampling distribution for EE_{ACT}, as Dr. Kristin Isaacs has found (personal communication, 2014).

Selected EE_{ACT} COV's seen in the literature include data presented by Agiovlasitis et al. (2012) regarding activity-specific energy expenditure of adults with and without Down syndrome (DS). The subjects walked at 5 different speeds plus another that was preferred by each individual. The COV's for DS subjects were marginally greater than for non-DS subjects for every speed tested except the fastest (1.5 m/s); the difference in COV's were 1-3% larger in DS subjects. (All of the speed-specific METS were statistically significantly higher for DS subjects than for non-DS subjects also.) The range of the COV's was 17-24% for non-DS subjects and 18-26% for DS subjects (Agiovlasitis et al., 2012).

Brooks.AG et al. (2004) provided EE_{ACT} estimates for 4 different household activities using 3 different metrics: (1) absolute energy expenditure in kJ/kg-h; (2) a METS estimate using the "standard" resting metabolic rate of 3.5 mL/kg-min (called $METS_{3.5}$); and (3) a METS estimate based on the individuals' own measured REE ($METS_{IND}$) . The sample consisted of 36 "representative females" (?) aged 35-45 y old. EE_{ACT} was measured in both the laboratory and in a subject's own home. The home-based data follow, plus the "walking" activity, which was measured only in the lab.

Activity	EE_{ACT} (kJ/kg-h)	COV (%)	$METS_{IND}$	$METS_{3.5}$
Window Cleaning	14.0 ± 2.5	17.9	3.8 ± 0.5	3.3 ± 0.6
Vacuuming	15.8 ± 2.6	16.5	4.3 ± 0.7	3.7 ± 0.6
Sweeping	17.3 ± 2.9	16.8	4.7 ± 0.6	4.0 ± 0.7
Lawn Mowing	22.8 ± 4.6	20.2	6.2 ± 1.0	5.3 ± 1.0
Walking	17.4 ± 3.0	17.2	4.8 ± 0.7	4.1 ± 0.7

Note the fact that $METS_{IND} > METS_{3.5}$ for all activities, which conforms to the criticism leveled against the use of 3.5 mL/kg-min as the basis for a general population basal metabolic rate. In addition, the COV's for $METS_{IND}$ estimates are lower than those for absolute EE_{ACT} (kJ/kg-h) measures (data not shown), as expected because there is less variability in relative metrics than in absolute measures (McCurdy, 1997). The COV's for $METS_{3.5}$ are sometimes higher or lower than those for the EE_{ACT} measure itself. This, too, indicates another problem, generally unrecognized, with using "standard" METS values from the Compendium: by ignoring individual-specific REE, additional non-linear variability is added to the relative METS concept itself. Again, using the metabolic chronotropic relationship would improve EE_{ACT} estimates, and make them biologically more relevant in our exposure and intake dose rate models. It also would provide a better theoretical basis for estimating task-specific energy expenditure metrics for exercise physiologists.

Benden et al. (2011) provide interesting data for two activities that you think would not have a lot of intra- and inter-individual variability: sitting and standing. The absolute EE_{ACT} for sitting in 21 children aged 7.5 y (0.9 SD) was 0.63 kcal/min ± 0.18 for a COV of 28.6% for the sample! The COV for standing was lower (26.4%) even though the absolute EE_{ACT} was slightly higher: 0.72 kcal/min (0.19 SD). What is remarkable about their data, however, is the wide differences in individual COV's seen in the children over 10 repeat measurements for each activity. While the subject-specific COV data are presented only graphically, it is clear that the sitting data have much less individual variability than the standing data. Three of the 31 children had a sitting COV > 40% and it was 10% or less for the remainder. Individual standing COV showed much more variability. Four children had an $EE_{STANDING}$ COV <15%, 8 had a COV between 50-100%, and another 8 had an $EE_{STANDING}$ COV >100% (Benden et al., 2011). These are high individual coefficients of variation for such a low energy-expenditure activity. Intra-individual variability of EE_{ACT} is a real problem from the perspective of using a single METS value for an individual, let alone a collection of similar age/gender individuals. See also Benden et al. (2012) for a discussion of within-subject (intra-individual) variability of using a standing desk versus a sitting desk, obviously a follow up study of the work discussed above.

Graves et al. (2008a) provide energy expenditure data for 11-17 y old adolescents playing 3 active video games (Nintendo Wii) and a sedentary video game (XBOX 360). Thirteen male and female adolescents were involved in the study, and each game was played for 15 minutes, in random order, with a 5-min seated rest between games. Two EE_{ACT} indices were measured, oxygen consumption (VO_2) and heart rate (HR). As mentioned in Appendix B and Section 3, there is a non-linear relationship between VO_2 and HR so we do not normally use that metric to estimate EE_{ACT}, but HR data are included here to make a point about using HR as an indicator of group COV's. The VO_2 data were converted into EE metrics using 1 litre of VO_2 = 4.9 kcal and 1 kJ = 0.239 kcal (Graves et al., 2008a). (Incidentally, while the kJ to kcal conversion is identical to has been used in the past, we use 1 L VO_2 = 4.85 kcal, citing Erb [1981]. Since this conversion depends upon an assumed RQ, there is a range of VO_2-to-kcal values seen in the exercise physiology literature, anywhere from 1 L =4.71 to 1 L = 5.01 kcal. 1 L VO_2 = 4.85 or 4.90 are compromises for a difficult-to-measure metric. For purposes of this illustration, the conversion simply changes the "scale" of the COV's, but does not affect their relative magnitudes.)

Data—means and standard deviations--from the Graves et al. (2008a) paper follow for the activities tested. I calculated the rounded-off COV's.

Activity	VO2 (mL/min)	COV	EE(J/kg-min)	COV	HR(beats/min)	COV
Rest	250 ± 60	24.0%	84.0 ± 14.6	17.4%	70.1 ± 12.1	17.3%
XBOX 360	350 ± 70	20.0	115.8 ± 18.3	15.8	85.0 ± 11.7	13.8
Wii Bowling	550 ± 170	30.1	182.1 ± 41.3	22.7	103.2 ±16.7	16.2
Wii Tennis	610 ± 190	31.1	200.5 ± 54.0	26.9	107.0 ±15.2	14.2
Wii Boxing	820 ± 400	48.8	267.2 ± 115.8	43.3	136.7 ±24.5	17.9

A number of interesting findings from these data can be observed. One is that the COV's for EE_{ACT} are not identical to those for $VO_{2.ACT}$ even those the former is a linear transformation of the latter. That is somewhat of a surprise and it may be due to differences in the subject's RQ, which varies with the type of foodstuffs ingested by an individual. See "Weir's Equation" in the Glossary of Terms (Appendix E). In general, there is less variability in EE than in VO_2. Finally, the COV's for HR vary non-monotonically compared to COV's for VO_2 and EE, highlighting again the problems with using HR as an indicator of activity level. The approximate METS levels for the activities, based on the mean resting values, are—in the order listed: 1.5, 2.3, 2.5, and 2.4 METS (Graves et al., 2008b), so the activities investigated are low-EE_{ACT} undertakings.

Graves (2010) expands upon their previous work by having a wider age range of subjects (of both genders) undertake Wii and treadmill activities. They present mean/SD data for VO_2, EE, HR, and METS metrics, where 1 MET=the VO_2 resting value. Their data are presented below; the EE and HR data will not be discussed further. Age statistics for the three cohorts are: adolescents: 15.8 ± 1.3 y (n=14), young adults: 28.2 ± 4.6 y (n=15), and older adults: 57.6 ± 6.7 y (n=13). I calculated the COV's (as percentages) from their data. Older adults did not try the treadmill jogging task. The differences between VO_2 COV's and METS COV's are striking, but there is less variability in COV metrics among the three age groups (except for "treadmill jogging"). Note that using the "standard" breakdown of METS into PA categories, Wii aerobics is a MPA task, as is treadmill walking. Treadmill jogging would be considered to be a VPA task.

While it is interesting to review the exercise physiology literature and provide explicit data on activity-specific COV's for METS and/or EE_{ACT} (or $VO_{2.ACT}$) sample statistics, I feel that the above information is sufficient to make the point that there is considerable population variability in the amount of work needed to undertake even fairly narrowly-defined tasks. This variability is ignored using a single-point estimate of activity-specific METS (or VO_2, HR, and EE_{ACT}). Sample-specific COV's addresses inter-individual (among individuals) variability. Except for the Benden et al. (2011) paper discussed above, I did not uncover any paper that explicitly addressed within-subject or intra-individual variability in undertaking a specific task, but my review of the literature on that topic was not synoptic. Longitudinal variability in EE_{ACT} within an individual certainly exists, as anyone knows from past personal experience, but it is rarely reported by exercise physiologists, even if measured.

	VO_2 (L/min)					
	Adolescents		Young Adults		Older Adults	
Activity	Mean (SD)	COV	Mean (SD)	COV	Mean (SD)	COV
Rest *Handheld*	0.35 (0.07)	20.0%	0.31 (0.05)	16.1%	0.32 (0.07)	21.9%
Gaming Wii	0.36 (0.09)	25.0	0.34 (0.06)	17.6	0.32 (0.07)	21.9
Balance *Wii*	0.59 (0.11)	18.6	0.58 (0.13)	22.4	0.57 (0.17)	29.8
Yoga Wii Muscle	0.60 (0.10)	16.7	0.57 (0.14)	24.6	0.57 (0.16)	28.1
Conditioning	0.74 (0.12)	16.2	0.73 (0.17)	23.3	0.68 (0.22)	32.4
Wii Aerobics Treadmill	1.09 (0.17)	15.6	1.09 (0.22)	20.2	0.96 (0.29)	30.2
Walking *Treadmill*	1.20 (0.26)	21.7	1.35 (0.26)	19.3	1.17 (0.47)	40.2
Jogging	2.21 (0.43)	19.5	2.44 (0.35)	14.3		

Not only is there considerable variability in activity-specific oxygen consumption required for each task, their COV's are also quite different by both activity and age group. (COV's for each task would probably be reduced if both gender- and age-specific data were provided.) The METS data provided in Graves et al. (2010) follows; again, I calculated the COV's.

	METS (Unitless)					
	Adolescents		Young Adult		Older Adult	
Activity	**Mean/SD**	**COV**	**Mean/SD**	**COV**	**Mean/SD**	**COV**
Handheld Gaming	1.0 ± 0.1	10.0	1.1 ± 0.1	10.0	1.1 ± 0.3	30.0
Wii Balance	1.7 ± 0.4	23.5	1.9 ± 0.5	26.3	1.9 ± 0.5	26.3
Wii Yoga	1.7 ± 0.3	17.6	1.9 ± 0.4	21.1	1.9 ± 0.4	21.0
Wii Muscle Conditioning	2.2 ± 0.4	18.1	2.4 ± 0.4	16.7	2.3 ± 0.6	26.1
Wii Aerobics	3.2 ± 0.7	21.8	3.6 ± 0.8	22.2	3.2 ± 0.8	25.0
Treadmill Walking	3.5 ± 0.5	14.3	4.5 ± 1.0	22.2	4.0 ± 1.5	37.5
Treadmill Jogging	6.5 ± 1.5	23.1	8.0 ± 1.2	15.0		

12.0
Human Exposure Modeling Research Needs

Although the current APEX and SHEDS exposure models are quite sophisticated in their general approach and procedures, there are a number of areas that could be improved or expanded upon. Besides better addressing both inter-and intra-individual variability (uncertainty) for most physiological parameters and other deterministic "constants" used in the overall modeling effort—which are not addressed here except where noted--the biggest areas of needed improvements in my view are those involving human activity/physiology data inputs to the models. The main focus here is on the air route of exposure/intake dose rate, but many of the comments affect the modeling of all routes of exposure.

In rough order of priority, they are:

1. Expansion of the longitudinal time-activity database, especially coordinated with locational and activity-specific measurements data, is needed. At the current time, this information can best be obtained via "smart-phones" having built-in GPS and accelerometry monitoring capabilities. As the review in Section 11 on accelerometers indicates, however, there are (surmountable) problems with accelerometer information (and, to lesser extent) GPS data-gathering that also need to be addressed. Perhaps by the time that EPA will be able to undertake such a data-gathering effort, these essentially technical (engineering) problems will have been solved. In general, using a protocol where smart-phone subjects are "automatically" queried when "unusual" changes in GPS/accelerometer data occur using real-time algorithms that "monitor" the smart-phone—including getting no signal for a specified interval--will obviate many missing data problems. The subjects would be "automatically" queried, asking them to text what is going on in order to provide a usable signal on a real-time basis. Receiving location/activity data on essentially a contemporaneous basis can obviate some of the problems with the technology. It is recommended that at least 7 consecutive days of complete 24 h data be collected from each study individual at least 4 times per year to obtain adequate longitudinal human activity coverage (Xue et al., 2004). Doing so also expands data on the correlation structure of time spent in selective locations that would improve the D & A procedure used to develop cohort-specific longitudinal activity patterns (Glen et al., 2008).
2. Especially important is obtaining better time-activity data for "susceptible groups of individuals" with pre-existing health conditions that make them particularly vulnerable to airborne insults. Examples are asthmatics, people with COPD, people with cardiovascular disease, overweight and obese people (especially children), and other health-compromised groups (that may have pollutant-specific issues). For instance, the prevalence of overweight/obese children and adolescents more than doubled during the 1990-2000 time period (Jolliffee, 2004). Their metabolic and physiological makeup is sufficiently different from the general population to warrant treating them as a separate cohort in EPA's exposure/intake dose models. Another important group of individuals that should be focused on are active people, especially children and adolescents, and outdoor workers: these cohorts are important with respect to setting many of the NAAQS standards, especially ozone. Their activity pattern data are under-represented in CHAD.
3. Rigorous characterization of the "feedback loop" that certainly exists between microenvironment-specific pollutant concentrations and respiratory parameters is needed. These parameters include breathing rate ($\mathbf{f}_B$), dead-space and tidal volume (V_D & V_T), ventilation rate (V_E), and alveolar ventilation (V_A), which is a function of these parameters (Valcke & Krishnan, 2011). Doing so means that a modeled subject's physiological parameters will have to be "dynamic" in that they potentially will be altered "on the fly," so to speak, to reflect exogenous impacts on a receptor. While conceptually important, it is not expected that this feedback-loop capability will affect intake dose rate metrics significantly except in very highly polluted locations frequented by highly active people.
4. One aspect of this feedback loop improvement is to develop a systematic procedure to differentiate between nasal and oral routes of intake dose rate as inhalation rates increase due to increasing activity-specific energy expenditure. These different routes obviously affect how much material gets into the lung and other organs given the same amount of exogenous material in the environment. To date, our exposure models have ignored this issue.
5. Addressing mitigating behavior should become a priority. This issue may have a lot of impact on exposure estimates, and is focused on better addressing the "mitigating behavior" issue, where

a person may alter her or his time use patterns due to identified environmental problems existing in a microenvironment or larger geographic location. One dimension of the issue is EPA's Air Quality Index" (AQI) used to provide the public with timely information on potential "code red", etc. days that may affect (some) people's health status. The AQI is designed to inform the public and affect change in sensitive people's near-term activity/location (behavior) choices. There is a lot of uncertainty regarding the real impact of these types of programs (Mansfield et al., 2003, 2004, 2006a b), so more information is needed to characterize what people do differently on "bad air" days. Probably this shortcoming affects mostly ozone and particulate matter exposure assessments. This type of information probably could most effectively be obtained via expansion of the longitudinal time-activity database (Need #1), especially focused on asthmatics, people with COPD, and/or people with cardiovascular disease issues.

6. Characterizing the systematic variability in physiological parameters due to circadian and other "rhythms of life" in humansis is needed. These rhythms can have a significant impact on intake dose rates. This phenomenon has been recognized since the 1860's (E. Smith, 1861). Linn (1991) states that lung function and symptoms in asthmatics show a significant circadian variation, and this variation increases with asthma severity. The magnitude of the circadian variability differs among individuals (Linn, 1991). There also is a circadian rhythm associated with resting metabolic rate (REE), VO_{2MAX}, $V_{E.MAX}$ in many individuals on (mostly) a daily basis, which causes—among other impacts—daily variability in total daily energy expenditure (DTEE) seen in longitudinal studies of energy expenditure (Reilly et al., 1997, 2000). Circadian rhythms might affect the temporal timing of physiological peak characteristics, such as $V_{E.MAX}$. A peak V_E value coinciding with a peak environmental concentration would result in a significantly larger intake dose rate than might be expected if daily average physiologic estimates were used. In addition, total daily dose-received estimates are affected by these rather short-term alterations in a person's basic metabolic states.
7. There also is a weekly pattern to human activities that significantly affects DTEE in some individuals; see Section 8. There also are longer-term rhythmic patterns in people that could affect intake dose rate over time. Some obvious examples are pregnant or lactating females, and even fecund females going through their monthly cycles (Reilly et al., 2000). Other similar longer-term patterns in physiology (and metabolism) parameters should be investigated and incorporated into exposure model algorithms where appropriate.
8. Better characterizing activity-specific energy expenditure (EE_{ACT}) definitely is needed, as the material in Section 11 indicates. In essence, this means that we need better $METS_{ACT}$, or equivalent, distributions, including estimates that are valid for a longer period of time than normally used by exercise physiologists in their testing protocols. In this manner, the fatigue/EPOC procedure used in our exposure models can be evaluated and improved, if needed (Isaacs et al., 2007). The new METS (or oxygen consumption) estimates should be based on a person's individual basal metabolic rate instead of the 3.5 mL/kg-min "standard" factor (see McMurray et al., 2014). These individual $METS_{ACT}$ estimates should be analyzed in such a manner that population distributions of $METS_{ACT}$ can be developed. Combined with individual $METS_{ACT}$ data over multiple measurements will allow the characterization of intra-individual variability in the $METS_{ACT}$ distributions as well as characterization of inter-individual variability in them. The ICC statistic should be useful in this endeavor.
9. Actually, new EE_{ACT} estimates should be based upon the metabolic chronotropic relationship and reserve physiological measurements and concepts as discussed in Section 7. Doing so would considerably reduce uncertainty about $METS_{ACT}$ estimates currently provided in the literature, including (1) age/gender-specific basal energy expenditure values (and their predictive equations), (2) $VO_{2.MAX}$ estimates, and (3) $METS_{MAX}$ measures. Using reserve physiological parameters reduces the impact that age/gender/health status has on most of the physiological processes that we currently address in the APEX and SHEDS models.
10. Providing a linked "micro-activity" and "macro-activity" (time use) database for comprehensive multi-media exposure assessment is needed. This means that locational and time-of-day data needs to be developed for such events as hand-to-mouth frequencies in children, water ingestion rates by location and time-of-day, and better temporal and locational data on babies crawling behavior, and so forth (Xue et al., 2007, 2009). In that matter, micro-activities could be tied directly to EPA's CHAD database, as was recommended in 1998 by an expert panel (Pechan Associates, 2001). The few papers that provide information that could become the basis for micro- and macro-activity diary estimates include Kissel et al. (1996) and Shepherd-Banigan et al (2014). There may be more papers on this topic, but identifying them would require a separate literature search and compilation. Time constraints do not permit such a search.

APPENDIX A
Physiological Testing Protocols with an Emphasis on $VO_{2.MAX}$

Much of the information contained in this report comes from clinical studies of exercise and respiratory physiology from four general classes of subjects. These are: (1) athletes or "trained" individuals, usually with the aim of improving performance; (2) participants in a clinical study oriented toward a lifestyle modification or other type of intervention, especially getting people to exercise more and/or eating a more healthy diet; (3) people with some type of health problem; and (4) the general population in order to establish relevant fitness or other physiological benchmarks. Additional physiological data comes from (1) work-place studies of activity-specific work rates, mostly focused on employee fitness to do the job and programs to improve it; (2) studies of energy expended by participants undertaking a particular type of recreational activity or exercise regime; and (3) dietary and nutrition studies, where basal and daily total energy expenditure data are collected. Usually some type of data on HR, VO_2, and V_E is provided for study participants in these research efforts. Rarely, however, does a single study report data for all of these parameters, even if they were all collected. Thus, obtaining a complete picture of physiological parameters used in our exposure models requires the analyst to combine information from disparate studies. That is what is done in this report.

Exercise Testing Fundamentals

There appear to be three phases during an exercise physiology test as workload increases from low to maximum intensity, which is the usual progression: I-Aerobic metabolism, II-Transition, III-Anaerobic metabolism. As work increases in **Phase I**, an increasing amount of O_2 is extracted by muscle and other tissues. This produces more CO_2 than is expired. VO_2, V_E, and HR all increase linearly with workload. Blood lactate (lactic acid: LA) remains level, and the respiratory quotient (R: VCO_2/VO_2) remains steady at about 0.7-0.8. **Phase II** begins in individuals around 40-60% of VO_{2MAX} depending upon their fitness level. VO_2 and HR continue to rise linearly, but LA doubles (to around 4 mmol/L, on average) and CO_2 increases above linearity. The "respiratory center" is stimulated to increase V_E and the combination of increased V_E and CO_2 greatly increases VCO_2; thus R increases greatly. At this level, the rise in V_E and VCO_2 is greater than the increase in VO_2.

The ratio of V_E/VO_2 is called the respiratory quotient, or RQ, and it increases non-linearly above the point of inflection between V_E and VO_2. HR also increases non-linearly. The threshold at which the non-linear rise in RQ occurs is called the "aerobic threshold" in Skinner & McLellan (1980), and seems to be called the "ventilatory threshold" in more recent articles. See below for more on this.

Phase III occurs at the "anaerobic threshold," around 65-70% of VO_{2MAX} in most people, but it can be as high as 90% in very fit individuals, who can sustain high work levels for longer periods of time, mostly because they have low LA levels above the anaerobic threshold, contrary to the general population. In general, LA levels increase above the anaerobic threshold, VO_2 increases to maintain the respiratory muscles, and V_E increases even more rapidly to support the increased "metabolic cost of breathing." Thus, VQ increases sharply above the anaerobic threshold, as does HR.

Of course, a number of other physiological changes occur during the workload transition, such as: muscle fiber composition and oxidation (removal of electrons in the mitochondria, lactate dehydrogenase (the "Krebs cycle"), anaerobiosis, and blood flow from the heart to exercising muscles (i.e., cardiopulmonary system functioning, etc.). These factors are not explicitly considered in our exposure models, and so are slighted here.

Anaerobic Threshold / Ventilatory Threshold

As one author states: "despite the popularity of the concept of 'anaerobic threshold (AT), the noninvasive detection criteria [to determine it] remain subjective, and invasive validations of AT ignore differences in lactate concentrations in arterial, mixed venous, venous and capillary blood samples" (Yeh et al. 1983). There have been scores of subjective criteria developed for defining the AT and Yeh et al. (1983) lists 10 of them. After applying a number of subjective and objective criteria of AT, including looking at concentrations of lactate from both venous and arterial blood samples, the authors conclude:

> This study shows that while anaerobic metabolism does occur during exercise, a threshold phenomenon is not detectable with the invasive methods. In addition, when current noninvasive methods to determine AT are used, there is an unacceptably large range of AT values for individual subjects when determined by different reviewers. Without the support of reliable invasive methods for assessing AT, the development of computerized noninvasive assessment techniques are on a unstable foundation (Yeh et al., 1983; p. 1185).

The average difference among the four reviews of the subjective AT data was about 50% of the between-subject range of values, which Yeh et al. (1983) considers to be too large.

There is a specifically-named anaerobic threshold test called the Friel FATT test (Yuen et al., 2011). The Friel test is based upon a HR deflection point **and** a rating of perceived exertion (RPE) of 17 on the Borg Scale (Borg, 1973, 1986). This test is a good example of the difficulties in using exercise physiology information. Yuen et al., 2011) states that the Fiell FATT test is described differently in a number of publications, even by Dr. Friel himself, so they used one particular version appearing in Friel 2004 (Yuen et al., 2011; p. 173.) There were significant differences in estimating AT and $VO_{2.MAX}$ depending upon the protocol used.

It also is interesting to note that although the AT is normally stated to be around 60-70% of $VO_{2.MAX}$ in "normal," healthy subjects, and higher in trained individuals, studies have been done which measured "end-exercise oxygen cost" in both children and adults at 125% of their estimated AT (Mahon & Cheatham, 2001; Zanconato et al., 1991). Multiplying that by the AT range estimate, means that people were exercising at 75-88% of their $VO_{2.MAX}$, quite a high—but possible—level, at least for short periods of time.

In more recent articles, ventilatory threshold (V_T or VT) is substituted for anaerobic threshold (McArdle et al., 2001). Again, there is difficulty determining a precise point for this threshold, so there is uncertainty concerning the concept and its relationship to lactic acid accumulation. V_T decreases with age in children and adolescents both on an absolute and relative sense. As a percent of $VO_{2.MAX}$, V_T decreased from about 75% in male youth to around 65%; the relative change in female youth was from 70% to 55% (Mahon & Cheatham, 2002). However, that finding apparently is not universal, as other researchers find no difference in relative V_T between children and adults (Mahon & Cheatham, 2002), while others find no trend by age and gender (Washington et al., 1988).

Most studies seem to agree that endurance training results in an increased relative V_T; increases in V_T as a percent of $VO_{2.MAX}$ on the order of 20% are not uncommon due to training (Mahon & Cheatham, 2001).

$VO_{2.MAX}$ testing

Whole-body $VO_{2.MAX}$ testing has been used as a marker of fitness since A.V. Hill developed it in 1923 (Akalan et al., 2008). A brief history of $VO_{2.MAX}$ testing is contained in Yoon et al. (2007). There probably are as many exercise testing protocols used to ascertain $VO_{2.MAX}$ as there are laboratories doing the testing. For a *review* of fitness tests, see Burke (1976), Lamb (1984), JN Myers (1994), Nieman (1990), Robergs & Burnett (2003), and Rowland (1996). Most of them use either a treadmill or bicycle ergometer to elicit $VO_{2.MAX}$, but arm-cranking (JA Davis et al., 1976; Kang et al., 1997; Washburn & Seals, 1984), bench-stepping (Olson et al., 1995), aerobic dancing (Olson et al., 1995), stair climbing, wheelchair ergometers (Keyser et al., 1999), running in water (McComb et al., 2006), repetitive lifting of heavy boxes (Nindl et al. 1998), lifting handweights (Robertson et al., 1990), one-mile walking time (Weiglein et al., 2011). and rowing protocols (Carey et al., 1974) have also been used. Activity-specific, sub-maximal, VO_2 measurements also are obtained using the same protocols.

$VO_{2.MAX}$ itself often is predicted using non-maximal testing, either estimating oxygen consumption directly, or by measuring sub-maximal HR, which then is "extrapolated" or extended to obtain an estimate of maximal work rate, which in turn is used as an estimate of $VO_{2.MAX}$. One common approach is based on regression analyses of occupationally-specific activities or sub-maximal treadmill work rates, especially in heart failure patients and the elderly (e.g., Pescatello et al., 1994; Ribisl & Kachadorian, 1969). We do not provide $VO_{2.MAX}$ estimates obtained in this matter due to the unstated uncertainty attending these procedures. For examples of this type of prediction equation, see George et al. (1993, 1997), Kline et al. (1987), Morris et al. (2009), and Peate et al. (2002),. Malek et al. (2004) provides a detailed review of eight $VO_{2.MAX}$ prediction equations for active, trained individuals. These extrapolating techniques are different than the $VO_{2.MAX}$-predicting equation discussed later, which generally are based on anthropogenic characteristics divorced from an exercise protocol.

There also are multiple protocols that have been developed for each method or machine, such as a treadmill (Wilkoff & Miller, 1992). Each has its own name, such as "Fox running protocol," the "Åstrand protocol," "Balke protocol," " Bruce protocol," "Costill/Fox protocol," the "modified Naughton protocol," etc. (Diaz et al., 1978; Falls & Humphrey, 1973; Kang et al., 2001; Lockwood et al., 1997; Panton et al., 1996). Even then, there are variants of these protocols used by different labs, so there are a wide variety of ways that have been used to measure $VO_{2.MAX}$ over the years. $VO_{2.MAX}$ differences of 10-13% have been seen in the same set of subjects using alternative exercise protocols (McArdle et al., 2001), and walking versus running on a treadmill with everything else being equal the "Bruce protocol" has been shown to result in statistically significant different $VO_{2.MAX}$ estimates for the same sample (Ward et al.,1998). Another discussion of statistically significant differences among $VO_{2.MAX}$ estimates using the various protocols appears in Kang et al. (2001). Other investigations have shown that the differences are not significant at α=0.05 (McArdle et al., 2001). Investigations into the reliability of duplicate testing of the same subjects a number of times indicates that the SE_{EST} of at least some of the measurement protocols is fairly small: about 2.4% (Taylor et al., 1955). A study of 3 different treadmill protocols used on 144 children aged 6-15 indicated no statistically significant differences among the three tests on a gender-specific basis (Skinner et al. 1971). Apparently, the data are inconsistent with respect to the impact of different protocols on estimated maximal oxygen consumption measurements.

Treadmill testing generally provides higher $VO_{2.MAX}$ estimates than the cycle ergometer. This is true for youth (Boileau et al., 1977; Davis et al., 1997; FI Katch et al., 1974; Lukaski et al., 1989) as well as adults (Kamon & Pandolf, 1972; Robertson et al. 1990; AC Snyder et al., 1993). In adults, the differences in $VO_{2.MAX}$ obtained by these two procedures has been measured to be +11% (range: 4-22%) for males and +7% (range: -1% to +17%) of varying fitness levels (Kamon & Pandolf, 1972).

Reproducibility of treadmill measures of $VO_{2.MAX}$ over relatively short periods of time using the same protocol is relatively high (using a reliability coefficient, which apparently is a standard Pearson product-moment correlation coefficient): 0.52-0.99, and averaging about 0.90 (Freedson & Goodman, 1993). These test/retest *r's* are similar to those seen in Kirkeberg et al. (2011), who also provide a COV in the 2-3% range for $VO_{2.MAX}$ estimates for 3 different exercise durations.

An example of a well-described $VO_{2.MAX}$ procedure comes from Iwamoto et al. (1994). They call it a "standard open circuit" treadmill protocol where VO_2 is measured by a mouthpiece and a nose clip to minimize nasal breathing. The treadmill grade starts at 0% and increases 2.5% in slope every 2 minutes. Data are collected in the last 20 seconds of each condition to assure a "steady state" estimate. Treadmill speed is adjusted for "pre-determined" (by questionnaire) fitness level of the subject: 3.5 mph for low fit people—a walking speed—and 6.0 mph for higher fit people—running speed. Subjects are exercised until voluntary exhaustion (Iwamoto et al., 1994). It should be noted that most labs do not vary treadmill speed by fitness level; everyone runs at the same treadmill speed (Kang et al., 2001).

One of the clearest descriptions of successful attainment of $VO_{2.MAX}$ used for both "normal, healthy" and overweight/obese subjects of both genders is contained in Wood et al. (2010). They declare a measurement of $VO_{2.MAX}$ to be valid if at least **3** of the following 5 criteria are achieved during the last 30 s of the last completed graded treadmill test:

1. <50% increase in VO_2 of that expected for the change in mechanical work (this is based upon past experience)
2. HR is within ± 11 bpm of the subject's age-predicted maximum (220-Age)
3. RER ≥ 1.15
4. Peak blood lactate ≥ 8 mmol L^{-1}
5. RPE (Rating of Perceived Exertion) ≥ 18 (see the Glossary for a discussion of RPE)

There also are "pre-conditions" associated with a valid $VO_{2.MAX}$ testing protocol that have been followed since the mid-1970's, such as no strenuous work on the prior day, testing in the morning after a good night's sleep, and testing in a "low-anxiety environment," etc. (Shephard, 1987). A number of these attributes have currently been relaxed, especially the hour rest period in a thermally-neutral environment prior to the test.

There is one aspect of $VO_{2.MAX}$ testing that is quite subjective and which influences the resulting estimate: verbal encouragement by testing staff for the subject to "keep going and work harder." Oftentimes the nature and extent of this encouragement is not fully described in the protocol, and so is not well documented. The presence or absence of encouragement has been shown to result in significantly higher $VO_{2.MAX}$ values for untrained subjects but not for competitive runners (Moffatt et al., 1994). One cardiologist states that RER (CO_2 as a volume / O_2 as a volume) is "the most definitive and objective clinically available measure of physiological level of effort during exercise" (Brubaker & Kitzman, 2011; p. 1012). The range in RER is <0.85 at rest and >1.20 during intense, exhaustive exercise; a value <1.05 indicates that a peak work load was not obtained (Brukaker & Kitzman, 2001).

In general, no manner how measured, the main criterion of whether or not $VO_{2.MAX}$ is attained is that the measured oxygen consumption shows no further rise with increasing work load (Kemper & Verschuur, 1980; Nieman, 1990; Robergs, 2001; Sanchez-Otero et al., 2014; Taylor et al., 1955), but that criterion is not universally defined. Some labs use <10% difference in VO_2 between workloads; others use a tighter definition: < 50 mL/min increase with a 1% increase in treadmill grade (Ardestani et al., 2011). The Wood et al. (2010) criterion mentioned above is another example. Since some people—and especially children—never see a leveling off of VO_2 with increasing work load, secondary attainment criteria are often used in those cases (McArdle et al., 2001).

One secondary criterion is that HR of the exercising person is >95% of HR_{MAX}—measured or predicted, and there are other relative levels also seen, such as attaining ± 10 bpm of age-predicted maximal heart rate (Ardestani et al., 2011). A third criterion is that the respiratory gas exchange ratio is >1.00, (or, alternatively: 1.10), while a fourth is that $V_{E/BM}$ be >1.6 (Kemper & Verschuur, 1980). If a leveling off of VO_2 does not occur during the exercise test, some physiologists state that the performance is limited by (1) local muscular factors rather than central circulatory dynamics, (2) anaerobic substrate metabolism. In fact, the presence or absence of a plateau may be associated with anaerobic capacity level itself, so this criterion may be "circular" in practice. In many cases, when a plateau is not seen, the term $VO_{2.PEAK}$ is used instead of $VO_{2.MAX}$ (McArdle et al., 2001). That distinction is not universally made either, and many contemporary authors use $VO_{2.PEAK}$ even if a plateau in VO_2 is attained. That is why we use the two terms interchangeably in this report. Howley et al. (1995) is a succinct review of the criteria used by exercise physiologists to ascertain if $VO_{2.PEAK/MAX}$ is attained or not. A similar paper by Huggett et al. (2005), focused on the elderly, also is informative. The interested reader is referred to those papers, but there are many other papers and books that discuss the maximum oxygen consumption testing process.

Given all of the nuances of the tests and the criteria used to ascertain attainment of $VO_{2.MAX}$, we take the pragmatic viewpoint that the measured and reported $VO_{2.MAX}$ value is an indicator of "true" maximal oxygen capacity of an individual, even though uncertainty exists regarding what exactly is being measured. If we have a choice, I present treadmill $VO_{2.MAX}$ instead of bicycle ergometry data. The data in Table 1 certainly indicates a wide variety in the mean and standard deviation of $VO_{2.MAX}$ estimates for relatively similar age/gender groups. These differences are most certainly at least partly due to different subjects being tested, but also to the protocol used to assess the parameter.

There are a number of $VO_{2.MAX}$ prediction equations in the literature using only age, gender, and/or body mass as independent variables. Additional prediction equations have been formulated using some of these anthropometric measures plus sub-maximal exercise data (see, for instance: Magaria et al., 1965 and McAuley et al., 2011). These types of prediction equations also are not reviewed in this report. The reader is directed to Armstrong & Welsman (1994, 1997), Armstrong et al. (1999), Bonen et al. (1979), Mahar et al. (2011), and McMurray et al. (1998), for a discussion of $VO_{2.MAX}$ prediction equations in children and adolescents. $VO_{2.MAX}$ prediction equations for older groups appears in Bradsford and Howley (1977), Darby & Pohlman (1999), Dolenger et al. (1994); Fleg (1994), Fleg et al. (2005), and Peterson et al. (2003). Other citations could be provided regarding $VO_{2.MAX}$ prediction equations using anthropometric inputs, but these should suffice to indicate the number of citations available in the exercise physiology literature.

We focused on VO_2 and $VO_{2.MAX}$ measuring methods and protocols in this Appendix, but alternative metrics of energy expenditure are used in the clinical nutrition and exercise physiology fields. There are a number of books and articles describing these alternatives besides the oft-cited Exercise Physiology (5^{th} Ed.) book by McArdle et al. (2001). Some of these are:

T.A. Baumgartner and A.S. Jackson (1999). Measurement for Evaluation in Physical Education and Exercise Science (6^{th} Ed.).

H.J. Montoye, et al. (1996). Measuring Physical Activity and Energy Expenditure.

M.J. Safrit and T.M. Wood (1995). Introduction to Measurement in Physical Education and Exercise Science.

G.J. Welk [ed.] (2002). Physical Activity Assessments for Health-Related Research.

For additional information regarding oxygen consumption measurement, please see one of these books.

APPENDIX B
Examples of the Three Types of General Metrics with a Focus on Heart Rate

Most physiological studies include some type of heart rate (HR) measurement, for one or more of these conditions: basal or resting state: HR_R , an absolute metric; activity-specific heart rate, also an absolute metric: HR_A (or as $\%HR_{MAX}$, which is a one-sided relative metric); or maximal HR, an absolute metric: HR_{MAX}. Occasionally, data on a percentage of HR reserve (HRR) are presented, and this is a two-sided relative metric. Non-relative HR measures have units of beats per minute (bpm or b min^{-1}).

There is a problem with using absolute levels of HR with respect to understanding oxygen consumption impacts associated with any HR level. That is due to deviation from linearity in the absolute HR→ VO_2 association at both low- and high-intensity work rates (Acten & Jeukendrup, 2003; Shakerian et al., 2012). Since it is VO_2 that more closely approximates energy expenditure, non-linearity with HR is problematic with respect to its use in predicting activity-specific or maximal EE (and consequently, METS). For example, $VO_{2.MAX}$ predicted from submaximal HR estimates are 10-20% higher than actual measured $VO_{2.MAX}$ (Acten & Jeukendrup, 2003), probably too high an "error rate" for our models.

The non-linearity is due to many factors. One is that the hydration status of the individual affects the distribution of blood to working muscles as VO_2 as HR increases, causing a disassociation of the HR/VO_2 relationship (Åstrand & Rodahl, 1986). There are also non-linear relationships between cardiac output and (1) VO_2, (2) arterial/venous oxygen differences, and (3) stroke volume (Åstrand, 1980). The change in the cardiac output/stroke volume relationship is called "cardiovascular drift" (Acton & Jeukendrup, 2003). In addition, HR→ VO_2 is quite individualistic, and is inconsistently reproducible over time even in the same individual (Acten & Jeukendrup, 2003). The HR→VO_2 relationship is greatly affected by the type of work performed, the relative amounts of arm versus leg (large muscle mass) movement involved, and the subject's emotional state at the time of exercise (Armstrong, 1998; Louhevaara et al., 1990). Thus, absolute values of HR have a lot of uncertainty concerning relationships of interest to us.

The first relative HR metric: a "one-sided" characterization of $\%HR_{MAX}$, shows better associations and more stability vis-à-vis other physiological parameters than HR alone. With respect to $\%VO_{2.MAX}$ and $\%HR_{MAX}$, an association sometimes seen in the literature. A regression between these two metrics in elderly subjects had a R^2 of 0.71 and a 10% SE (Panton et al., 1996). A good relationship between $\%HR_{MAX}$ and $\%VO_{2.MAX}$ was also found in obese males (Eizadi, et al., 2011); the regression equation developed from their study of 34 middle-aged obese males (BMI ≥30) was $\%VO_{2.MAX}$ = -58.4 +[1.61* $\%HR_{MAX}$] with an R^2 of 0.81 (no SE provided). A third "good regression" relationship was found for patients with spinal cord injury (Jacobs et al., 1997). Their equation is $\%HR_{MAX}$ = 0.28 + [0.72 * $\%VO_{2.MAX}$] (R^2=0.85, no SE provided). On the other hand, a study of motorcross riders—working at a vigorous rate—indicates that a HR of ≥ 90% HR_{MAX} can elicit anywhere from 70-95% of $VO_{2.MAX}$ (Burr et al., 2010), a fairly wide range. Davis and Covertino (1975) equate a 70% HR_{MAX} work load to a 55-60% $VO_{2.MAX}$ response. Stated more generally, a $\%HR_{MAX}$ estimate is between 5-10% greater than the $\%VO_{2.MAX}$ value for the same relative work rates (Kohrt et al., 1998; Simmons et al., 2000). Londeree & Ames (1976) and Londeree et al. (1995) provide an overview of $\%VO_{2.MAX}$ → $\%HR_{MAX}$ regressions seen in the exercise literature.

It is difficult to succinctly summarize the relationship between the $\%HR_{MAX}$ and $\%VO_{2.MAX}$ metrics based on these findings. They track closely for some cohorts and protocols but not for others (Meyer et al., 1999). Using these two metrics in an exposure model could produce quite unstable oxygen consumption estimates.

Infrequently, the relationship between $\%VO_{2.MAX}$, a one-sided metric, and HRR, a two-sided metric, is addressed Scharff-Olsen et al., 1992). Interestingly, Brawner et al. (2002) state that %HRR is a better estimator of $\%VO_{2.MAX}$ than it is of $\%VO_{2.RES}$, but that finding is contrary to a number of other studies (Dalleck & Kravitz, 2006; Swain et al., 1998; Swain & Franklin, 2000a,b; Swain & Leutholz, 1997). Jakcic et al. (1995) also found excellent agreement between %HRR and **%**$VO_{2.MAX}$, but only between workloads of 40-70% of $VO_{2.MAX}$. Contrarily, in a study of young female gymnasts, %HRR could *not* be used to accurately estimate $\%VO_{2.MAX}$, even though the two metrics were correlated (Guidetti et al., 1999). In a cross-section exercise study of breast cancer survivors, Kirham (2010) found that there was a 13% COV for $\%VO_{2.MAX}$ at the same %HRR percentage. In a study of asthmatic patients, Molanorouzi & Mojtaba (2011) regressed the two metrics for HRR values ranging between 10% and 80% of HRR and found that the regression line was $\%VO_{2.MAX}$ = 0.66 + [14.8 * %HRR] with an R^2=0.55 (no S.E. provided). Given the lower R^2, there is considerable scatter in the data (no statistics provided).

There are only a few studies that relate $VO_{2.RES}$, a bounded reserve metric, to the one-sided HR_{PEAK} metric. Swain & Franklin (2002a) review a number of aerobic training studies in cardiac patients and show rather large variability in HR_{PEAK}

(and HRR) to $VO_{2.RES}$ percentages. They also provide a regression equation for the HR_{PEAK}-to-$\%VO_{2.}$ relationship using both REE-corrected and uncorrected measures, citing another 2002 Swain & Franklin paper (2002b) for it. Their regression equation was $\%VO_{2.RES} = (1.667 * \% HR_{PEAK}) – 70\%$ (no statistics reported). However when Swain & Franklin (2002b) was read, the equation's basis was not provided, only a citation to Swain et al. (1994) as the source of the relation. Swain et al. (1994) did not include the regression cited! Thus, since I could not find another paper using the formula, or anything like it, its validity and usefulness is not verified.

The statistical connection between the two-sided HRR and $VO_{2.RES}$ metrics were discussed in Section 7 of the main report under the metabolic chronotropic relationship. The relationship is fundamental in associating the various reserve forms of important physiological parameters used in our exposure models. What follows is a discussion of the role that HRR plays in exercise prescription programs, especially for people with cardiovascular problems.

Even with the non-linearity problem between heart rate and VO_2 reserve metrics, cardiologists and other medical disciplines use HRR in their work on improving physical fitness of patients with health problems. To operationalize the concept, they have developed the "Kavonen approach" to prescribing exercise rates in diabetics, subjects with past heart failure, and the elderly (Azarbal et al., 2004; Madden et al., 2009; Skidmore et al., 2008). This approach is based on the "Kavonen formula" (Karvonen et al., 1957):

Prescribed exercise level desired = $(HR_{EX} – HR_R) / HRR$

Where: HR_{EX} = HR of the exercise undertaken

HR_R = Resting HR

$HRR = HR_{MAX} - HR_R$

The formula usually is used in this form: $HR_{EX} = HR_R + [$ % exercise level desired * HRR] (McArdle et al., 2001). It has been "institutionalized" by the American College of Sports Medicine in its guidance to practitioners on setting exercise testing limits for individuals (ACSM, 2001). Thus, it enjoys wide use (Geddes et al., 2009; Hepple et al., 1997). It has been found to be equally applicable in overweight and obese people and normal weight individuals (Miller et al., 1993).

The ACSM Guidelines are increasing being used in exercise prescription programs for healthy individuals also. Athletes normally train at a relatively high proportion of their HRR, e.g., 85%, for a specified period of time (Patterson et al., 2005). Lower relative rates are prescribed for improving or maintaining fitness in sedentary people: 50%, for example (Patterson et al., 2005). For older persons, exercising at 30-45% of HRR is a sufficient training stimulus (Badenhop et al., 1983). In fact an exercise program at 35% of HRR for a moderate period of time provided similar improvements in aerobic capacity in sedentary people aged 65-75 in an 85% HRR program for shorter periods of time (Belman & Glasser, 1991). However, questions have been raised about how the Guidelines are being used to prescribe appropriate exercise levels in health-compromised people. Dalleck and Kravitz (2006) state that the Guidelines are often misapplied and misinterpreted, and cite specific studies. (None of the papers cited above are on their misapplication/misinterpreted list, however.) The ACSM Guidelines are based on an assumed RMR of 3.5 mL kg^{-1} min^{-1} (Hultgren & Burke, 1980) which we know is not universally applicable.

One major problem with the Karoven formula is that HRR often is not explicitly measured, but is *estimated*, particularly in health-compromised groups. HR_{MAX} is not measured in these people due to potential adverse consequences, but is estimated by one of a number of formulae developed by regressing HR on age. One such formula is: $HR_{MAX} = 210 – [0.8 * Age]$ (Suurnäkki et al., 1991). A more commonly used one is $HR_{MAX} = 220 – Age$ formula (ACSM, 2001; Carvalho et al., 2008; Lui et al., 2011; Robergs & Landwehr, 2002). Carvalho et al. (2008) have shown that predicted versus measured HR_{MAX} estimates are good for healthy, young adult subjects of both genders using the 220-Age equation (98.6 ± 2.2%), but are inaccurate for similar subjects with pre-existing heart failure conditions (65.4 ± 11.1%). Note the wide disparity between the COV's in these groups: 2.2% in healthy people versus 17.0% in heart failure people. This raises the issue of differential uncertainty among cohorts regarding predicted HR_{MAX} estimates. Graves et al. (2012) state that using 220-Age significantly underestimates HR_{MAX} in the healthy elderly; this is confirmed by Whaley et al. (1994). Nelson et al. (2010) state that the prediction equation is inaccurate for all 10-year age cohorts between the ages of 30 and 69. Gulati et al. (2010) state that the formula systematically overestimates HR_{MAX} for females in general, and older females in particular. Finally, Robergs & Landwehr (2002) state "research spanning more than two decades reveals the large error inherent in the estimation of HR_{MAX}. The formula HR_{MAX}=220-Age has no scientific merit for use in exercise physiology and related fields" (ISSN 1097-9751).

There are a number of other HR_{MAX}-estimating formulae in existence, as noted below, but they will not be discussed further here.

A positive consideration for using the HRR metric in exercise studies is that it is relatively stable as people age. A study of 15,247 males aged 40-59 had HRR values between 101 ± 9 and 138 ± 5 in four HRR quartiles, while the range was 103 ± 7 to 139 ± 6 for 12,212 males aged 20-39. Decreases in HR_{MAX} with age apparently are matched by decreases in HR_R. Their COV's for the two age groups are similar also: 4.1% for younger males and 4.5% for older individuals (Cheng et al., 2002). Another positive is that there is not a gender difference in the %HRR and $\%VO_{2.RES}$ relationship, at least for adolescents (Eklund et al., 2001).

Other cardiologists disagree that HR_R decreases with age; see Brubaker & Kitzman (2011.) With "an inexorable" (and highly predictable) decrease in HR_{MAX}, they believe that HRR will also decease with age (Brubaker & Kitzman, 2011).

Because of the above concerns about the HRR metric, how it varies with age, and its relationship with $VO_{2.RES}$, no tabular HRR data will be presented in this report. A list of papers containing *measured* HRR, or its component parameters follows for specific population groups. See the list of References for a complete citation for the papers cited below. Papers that present measured HR_{RES} data are:

Cheng et al. (2002) Med. Sci. Sports Exer.

Kasser & Bruce (1969) Circulation

Papers that present measured (rather than estimated using one of the formulas) HR_{MAX} ***and*** resting heart rate (HR_R), but not HRR data *per se*, for a set of subjects are listed below. In a pinch, these data could be used to estimate group-means and an approximate SD for the HRR metric by subtraction, or more rigorously, by calculating those statistics using a formal meta-analysis approach. Neither option is explored in this report.

Billinger et al., 2012

Blanksby & Reidy, 1988

Dalleck & Kravitz, 2006

Davis & Shephard, 1988

Detollenaere et al., 1993

Dunn et al., 1999

Edwards, 1974

Iwamoto et al., 1994

Nikolai et al., 2009

Noah et al., 2011

Pettitt et al., 2008

Robinson, 1938

Sidney et al., 1992; 1998

Skidmore et al., 2008

Szymanski & Satin, 2012

Finally, HRR papers that estimate HR_{MAX} in their subjects using a formula but measure HR_R follow.

HR_{MAX} = 200 (a constant for youth aged 6-18 y):

Stratton, 1996

HR_{MAX} using a 220 - Age formula:

ACSM, 2001

Azarbal et al., 2004

Carvalho et al., 2008

Graves et al., 2012

Hui & Chan, 2006

Mahon et al., 2010

Miller et al., 1993

*HR_{MAX} using a 210 – (0.8 * Age) formula:*

Suurnäkki et al., 1991

*HR_{MAX} using a 208 – (0.7 * Age) formula*

Mahon et al., 2010

Tanaka et al. (2001)

*HR_{MAX} using a 206 – (0.88 * Age) formula*

Gulati et al. (2010): for females only

Tanaka et al. (2001)

Miller et al. (1993) provide information on the performance of 6 different HR_{MAX} formulae, some in a format never seen in any other paper, three of which apply solely to obese individuals. See that paper for more information on alternative HR_{MAX} formulae. The most widely-used formula, HR_{MAX} = 220 – Age, "often leads to an underestimation [of HR_{MAX}] for ages <40 y and overestimation for ages over 40" (Kirham, 2010: p. 23).

We have discussed heart rate at some length because there is a lot of information on all three types of metrics for that parameter, and because its associations with VO_2 have been fairly well studied. We are more interested in reserve metrics, however, for VO_2, V_E, and METS. Discussion of those metrics are contained in the main body of this report.

APPENDIX C
Background on Reserve Metrics

There seems to be three main sources of the reserve concept used over the years by different disciplines, with little communication among them. (Scientific balkanization?) The earliest work that we found using the reserve concept seems to be by exercise physiologists in Finland (Karvonen et al., 1957). That paper cites studies in the 1950's, but they apparently do not use *reserve* terminology, so genesis of the concept seems to be Karvonen et al. (1957) itself. In their paper, the authors state that HR of an activity (HR_A, in this case, exercise) should be expressed as a *percentage* of HRR: $HR_A/(HR_{MAX} - HR_R) * 100$. There is a short discussion in Karvonen et al. (1957) that HR and VO_2 are highly correlated based on work reported by others, but there is no information given in the paper on the explicit functional relationship between HRR and $VO_{2.RES}$. Swain (2000) states that Davis & Covertino (1975) made the case that $\%HRR = \%VO_{2.RES}$, which they call net VO_2. Thus, the reserve concept was identified and evaluated many years ago.

Current use of the oxygen consumption reserve by exercise physiologists is discussed thoroughly by Swain and colleagues in a series of articles. One of the most succinct is Swain (2000). An excerpt from that paper follows.

> Recent research has resulted in a number of recommended changes in how fitness professionals should prescribe target workloads and calculate the energy cost of exercise. The principal changes are in the use of oxygen consumption reserve (VO_2R) as an alternative to percentage of maximal oxygen consumption (VO_{2MAX}) for prescribing exercise intensity, the use of net VO_2 rather than gross VO_2 for the calculation of caloric expenditure during exercise …Several recent studies have shown that there is a discrepancy between the exercise intensity at given percentages of HRR and VO_{2MAX}, but that HRR and VO_2R yield equivalent exercise intensities. The use of VO_2R in exercise prescription provides more accurate target workloads, especially for individuals with a low fitness level. (Swain, 2000; p. 17).

Work by Swain et al. that analytically evaluated problems with using either the absolute or relative maximal values of HR (HR_{MAX}, $\%HR_{MAX}$) and VO_2 ($VO_{2.MAX}$, $\% VO_{2.MAX}$) are discussed in Swain & Leutholz (1997); Swain & Franklin (2002a, 2002b); and Swain et al. (1994, 1998). Other discussions of the reserve concept are found in Brawner et al. (2002), Franklin et al. (2000), and Pollock et al. (1997).

Swain (2000) states that equivalency between %HRR and %Vo2max is not expected on "theoretical grounds" because the concepts are not consistent at resting conditions. The discrepancy is smaller for highly fit people (Belman & Gaesser, 1991; Panton et al., 1996).

Hypothesis testing that %HRR = %VO2R was conducted by Swain & Leutholz (1997). The sample involved 33 ♂ (mean VO2max=3.33 ± 0.12 L/min) and 30 ♀ subjects (mean VO2max=2.02 ± 0.08) who were 18-40 y old. Regressions of %HRR on %VO2R were not significantly different than 0.0 for the intercept (-0.1 ±0.6 %HHR units) and 1.0 for the slope (1.0 ± 0.01). The mean correlation between the two measures was 0.991 ± 0.001. The regressions and other statistics *seem* to be averages of individually-based regressions. Swain et al. (1998) is very similar to the Swain & Leutholz 1997 paper, with a slightly smaller sample size. The averaged regression (explicitly this time) was %HRR = (1.03*%VO2R) +1.5 with an R^2=0.990 ± 0.009. The mean intercept was 1.5 ± 0.6 and the mean slope was 1.03 ± 0.01.

The disparity between %HRR and %VO2R is greater at low intensities than high, and in fit versus non-fit individuals (Brawner et al (2002). They investigated 3 groups of health-compromised subjects: people with myocardial infarction (MF; n=65); patients having a previous heart failure (HF; n=72); and subjects only having suspected risk factors for heart problems but with no overt symptoms at the time of the study (RF; n=42). Subject ages were in the 53-62 range (means) and included both genders. In regressing %HRR on %VO2R for the 3 groups, none had a statistically different slope from 1.00 (at $p<0.05$). The intercepts were significantly different than 0.0 for the HF and RF groups. The regressions, without SE's being provided) are: MI group--%HRR=(0.96*%VO2R)-1.9; R^2=0.95); HF group--%HRR=(0.97*%VO2R)-5.9; R^2=0.90); RF group--%HRR=(1.01*%VO2R)-4.7; R^2=0.95). The difference in the intercepts "suggests that %HRR is not equal to %VO2R" (p. 420). The authors did not have measured HR at rest, and it was assumed to be 3.5 mL/min-kg.

The second use of the reserve logic first involves occupational work physiologists in Finland (Ilmarinen, 1980, 1984; Karpansalo et al., 2002, 2003; Suurnäkki et al., 1991) and other European countries. These researchers focus on "stress" (work load) and "strain" of occupational activities (Oja et al., 1977). Strain is the impact of the work load on the cardiovascular and/or respiratory systems. Percent HRR is often used to estimate job-related strain (Suurnäkki et al., 1991). Prior to using HRR, strain was estimated by using

percentage of *maximal* VO_2, HR, or (even) ventilation rate (V_E) (Åstrand, 1960, 1967). The "%Max" approach is still being used, as we shall see.

The third historic users of the reserve concept are cardiologists, who want to improve their patients' cardiovascular system performance without exceeding their exercise capacity (Renlund et al., 1996; Wilkoff & Miller, 1992). Doing so, of course, could result in serious complications and even death. These patients include cardiac transplant recipients, people with chronic heart failure, coronary artery disease, hypertension and/or vascular stiffing (Renlund and Gerstenblith, 1987). The intent generally is to increase cardiac output, including heart rate, at increasing work rates. Often the patients can only perform at 62-68% of $VO_{2.MAX}$ levels seen in similar, but healthy, age/gender cohorts (Renlund et al., 1996).

The METS approach can easily be placed on a reserve logic basis. METS have been discussed since World War II, although origin of the idea is attributed to Dill (1936) and use of the term is attributed to Gagge et al. (1941), who predicated it upon body-surface area (BSA) heat loss. They defined 1 *met* to be the metabolism (thermal activity) of a subject resting in a **sitting** position on a kilocalories m^{-2} h^{-1} basis (Gagge et al., 1941). Since then a MET is defined to be RMR in a prone position.

The *concept* of making activity-specific energy expenditure relative to a person's resting (lying down) rate actually was discussed by E. Smith in 1861! METS-like values were presented by Smith for 29 activities, many involving tasks that are no longer common, but some that are still undertaken (Smith, 1861). His values are lower than those presented in the METS Compendium for similar activities (Ainsworth et al., 1991).

Less frequently seen in the literature is the METS reserve ($METS_{RES}$) metric (McCurdy & Graham, 2004; Wilkoff et al., 1989). $METS_{RES}$ is the difference between a person's $METS_{MAX}$ and their resting METS, which is equal to 1. Thus, $METS_{RES} = METS_{MAX} - 1$. Thus, it is easy to calculate $METS_{RES}$ when $METS_{MAX}$ is known.

The Wilkoff et al. (1989) paper contains the first use of the $METS_{RES}$ that I could find in the literature, although it is called the "metabolic reserve." However, that paper does not describe how $METS_{REST}$, as they term it, was measured. It does not appear that VO_2 at rest was actually measured anytime during their multi-day exercise protocol, but was *estimated* from HR itself using the ACSM formula; thus the depicted relationship in Wilkoff et al. (1989) between HRR and $METS_{RES}$ is tautological. The paper also does not describe how activity-specific METS were defined—by using the 3.5 mL kg^{-1} min^{-1} value or by using a "measured" basal rate. I give the authors credit for using the term "$METS_{RES}$" first, but it is unclear how they operationally defined it.

Rarely is there information available on the population distribution of $METS_{MAX}$, but Kokkinos et al. (2010) provides one for a large sample of male patients participating in a Veterans Administration study. There is no information available on how representative is the distribution in the overall population, but perhaps the age/ $METS_{MAX}$ categories are of some interest. The authors had previously shown that survivors over an 8.1 y time period had a higher $METS_{MAX}$ than non-survivors; 6.3 ± 2.4 METS to 5.3 ± 2.0 (Kokkinos et al., 2010). Using the 3.5 mL kg^{-1} min^{-1} factor for 1 MET, the population "baseline" breakdown of $METS_{MAX}$ is:

Fitness Category ($METS_{MAX}$ Class)	Age (Mean SD)	Sample Size (n)	Percent Mean of Total $METS_{MAX}$ Group (%)	Mean $METS_{MAX}$ (Unitless)
≤ 4.0	72.4 ± 5.3	1,083	20.3	3.2 ± 0.7
4.1 - 5.0	72.4 ± 5.3	1,226	22.9	4.7 ± 0.3
5.1 - 6.0	71.6 ± 5.0	886	16.6	5.6 ± 0.3
6.1 – 7.0	70.8 ± 4.5	835	15.7	6.6 ± 0.3
7.1 – 8.0	70.7 ± 5.0	486	9.1	7.6 ± 0.3
8.1 – 9.0	70.4 ± 4.7	355	6.7	8.6 ± 0.3
≥ 9.1	69.4 ± 4.0	463	8.7	11.0± 1.8

There is a clear decrease in $METS_{MAX}$ with age in the sample, and a pretty good decrease in the proportion of the study population in the higher $METS_{MAX}$ categories. The sample size is large: 5,334 males (Kokkinos et al., 2010).

A related reserve-like approach was developed in the 1960's by Bink (1962) and Bonjer (1962). The concept is that the amount of energy expenditure that can be maintained by an individual can be estimated if the aerobic capacity (as measured by $VO_{2.MAX}$) and elapsed time of the activity itself is known. This concept was called the physical working capacity of an individual. We actually used that approach in our early exposure models, citing Bink (1962) and Erb (1981). While not a reserve metric *per se*, it approaches it as well as factoring in the diminished work capacity over time. This limitation is also used in our current models in a method developed by Issacs et al. (2007) that has been successfully tested against the Bink/Erb approach.

The reserve concept is also used in non-human genera, where it is called "aerobic scope." A brief literature search of that term indicates that thousands of articles discuss aerobic scope in species as diverse as birds, lizards, snakes, fish, and non-human mammals. See, for example, Bishop (1999): "The maxima oxygen consumption and aerobic scope of birds and mammals: getting to the heart of the matter." I did not find any article that applied the term to humans.

APPENDIX D
Background Material on Exposure Modeling

This is a slightly modified reprint of Section 1 of *Data Sources Available for Modeling Environmental exposures in Older Adults)*

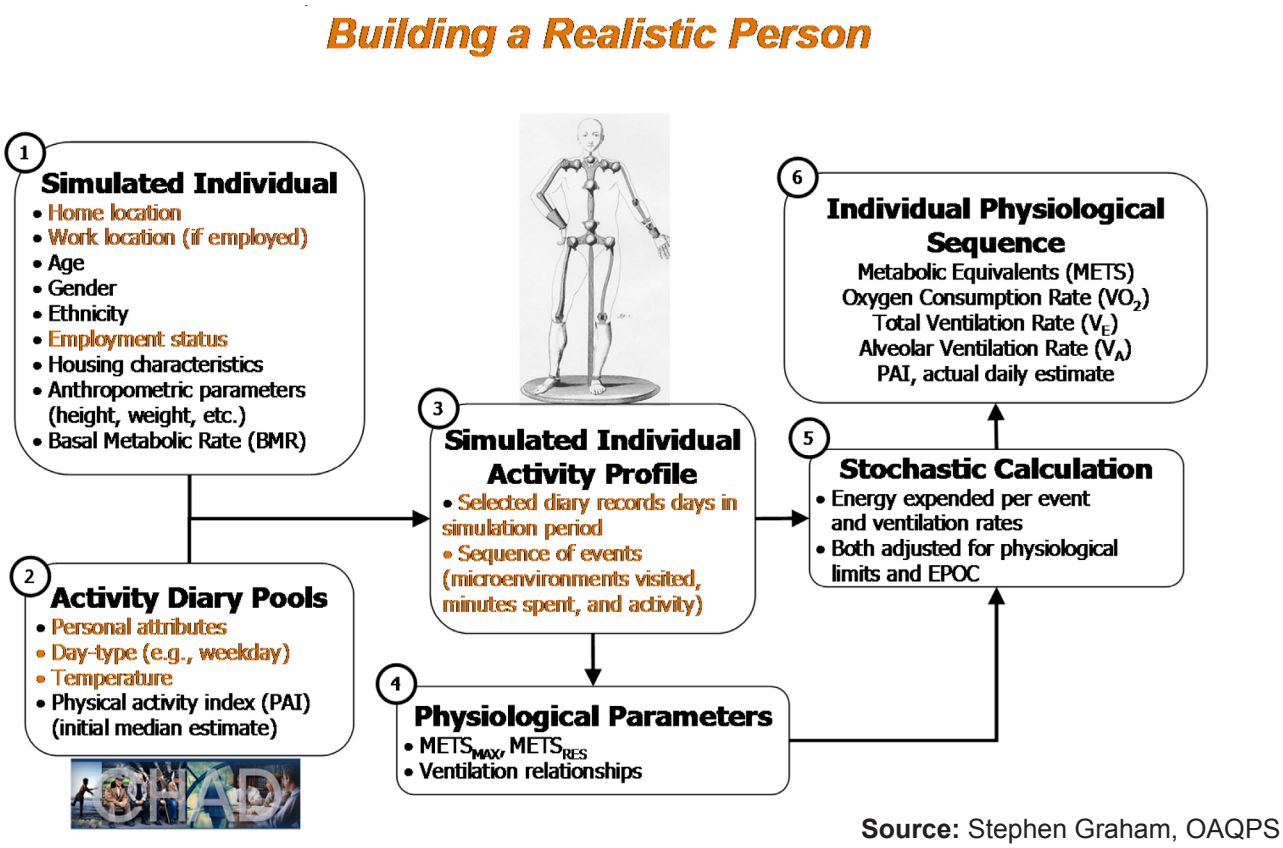
D.1 Exposure Modeling Overview and Principles

This report is focused on time use, physical activity, and physiological inputs needed for modeling inhalation exposures and intake dose rates, such as the APEX and SHEDS models. This subsection describes, in general terms, the approach, algorithms, and important variables used in both models. APEX is the primary air exposure model used by EPA's Office of Air Quality and Standards (OAQPS) to evaluate existing and proposed alternative National Ambient Air Quality Standards (NAAQS). APEX is also part of OAQPS's TRIM (Total Risk Integrated Methodology) program (U.S. EPA, 2008a, b), along with EPA's Hazardous Air Pollutant Exposure Model (HAPEM). HAPEM is a longer term exposure model that uses many of the same activity and physiological inputs as does APEX and SHEDS (Palma et al., 1999) but functions primarily to evaluate exposures to hazardous air pollutants from mobile and stationary sources of air toxics. The SHEDS model is an umbrella term for EPA's Stochastic Human Exposure and Dose Simulation model (Burke et al., 2001; Zartarian et al., 2000), of which there are a series of route-specific versions (dietary/nondietary, pesticides, etc.). It was developed by EPA staff in NERL's Human Exposure and Atmospheric Sciences Division (*HEASD*) and staff of Alion Science and Technology, Inc. The SHEDS model discussed here is oriented toward modeling exposures and intake dose rates for airborne pollutants (SHEDS-Air), but because the activity/time use and physiological concepts are similar in all of the SHEDS models, the findings reported here are more widely applicable to the modeling of all routes of exposure.

APEX and SHEDS now have similar features and input needs. Both use EPA's CHAD for their time use input data (McCurdy et al., 2000). CHAD, therefore, is discussed in some detail in this report.

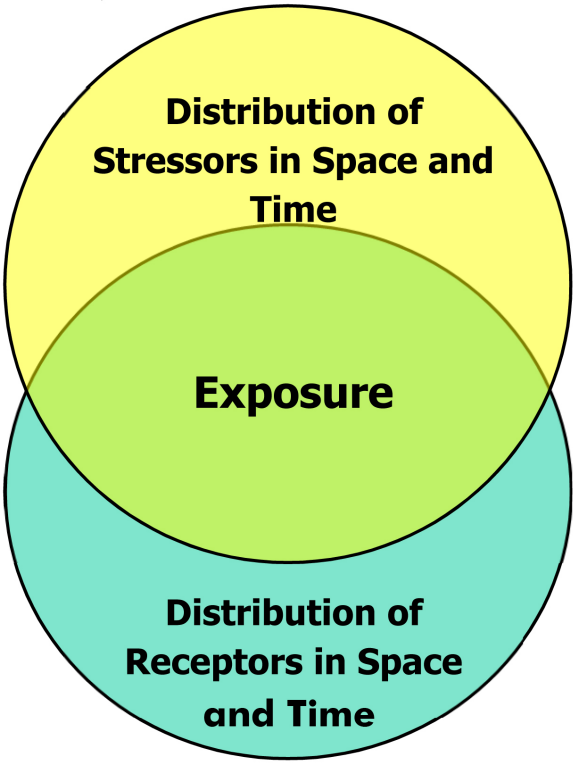
There are a number of important principles that have guided exposure and intake dose modeling since 1980 (Johnson, 1995; McCurdy, 1995, 1997). In general, these principles (15 in number and described just below) apply to all groups and not just to older adults.

1. An individual is the unit of analysis (Figure D-1). Each individual has a unique dose-response (D/R) relationship (National Research Council, 2009), which often is called a dose-effect (D/E) curve to distinguish it from the population-level D/R association. D/E uniqueness results from genetic factors; preexisting disease considerations; age/gender differences in biology, physiology, and time use patterns (location and activities); and lifestage and lifestyle differences among people (Dörre, 1997; McCurdy, 2000). EPA's exposure models are designed to reproduce such uniqueness. Being older can influence greatly D/E relationships in individuals both directly and indirectly because of physiological changes, immune system challenges, neurological



Source: Stephen Graham, OAQPS

Figure D-1. The individual is the unit of analysis. APEX and SHEDS construct simulated populations based on the above characteristics.



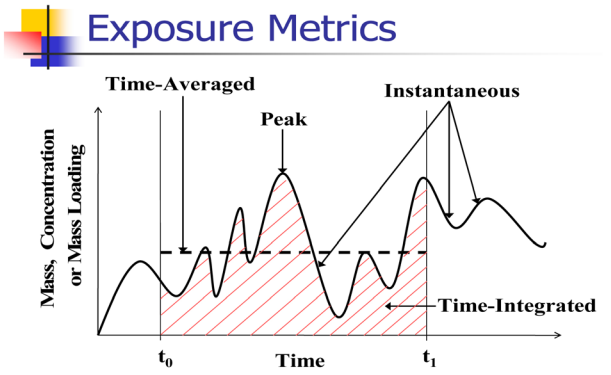
Source: Adapted from NERL Framework for Exposure Science

Figure D-2. A Venn diagram of exposure.

impairment (cognitive decline), and other physical alterations (Hertzog et al., 2008; Jette, 2006; Kiely et al., 2009).

2. Location is critical to evaluating an exposure to an environmental pollutant (often termed a "stressor") because, by definition, exposure is the "contact between an agent [substance or pollutant] and a receptor [a person in our case]" (Figure D-2). Contact takes place at an exposure surface over an "exposure period" (Zartarian et al., 2005),1 directly implying a specific location. It should be noted that there is a correlation structure to location patterns in an individual, both within and among days; locations that a person inhabits cannot be modeled using a "random-walk" process. On the other hand, there is day-to-day variability in locations that any individual frequents (unless confined to bed or an institution), so using "averaged" data does not capture daily variability in this important exposure variable either (Glen et al., 2008). This point is discussed further in principles 12 and 13.
3. An individual is not averaged over time or space; a person can be in only one location at any particular time.
4. A location having a constant concentration (C_T) for a specified period of time is called a "microenvironment" (μE). Microenvironmental data are crucial inputs to an exposure model (locations and concentrations),

[1] From the "Official Glossary" of the International Society of Exposure Science



Source: Duan et al., 1990, as modified by Thomas McCurdy (1996)

Figure D-3. Exposure metrics available from an exposure time-series.

and time spent in the various μEs vary greatly with age, gender, and lifestyle. In the APEX and SHEDS models, locational data come from CHAD, whereas μE concentration data are derived from ambient measurement data or route/pathway-specific model algorithms.

5. An exposure event is the smallest unit of time used in the two models and is characterized by a person being in a unique μE, undertaking a single type of activity and, therefore, experiencing a specific activity-level (see below.) By definition, an event does not cross a clock hour; longer activities are subdivided into two or more exposure events in that case (McCurdy et al., 2000). If any of these factors change, a new event occurs.
6. The event-based time pattern of concentrations experienced by an individual is called the exposure profile, or the exposure time-series. An example of an exposure profile is depicted in Figure D-3. A number of alternative exposure metrics may be derived from this profile, such as the number of peak exposures over a specified concentration level, the mean exposure level, and the time integral of exposures over some important value.
7. Activity level is the amount of energy expended (EE) by an individual to complete the activity undertaken (expressed in kcal or kJ/min/kg). Other metrics performing the same function were used in the past in EPA's exposure models.2 Activity level affects how much dose is received given an exposure. Activity levels are correlated over time in an individual, because prior physiological circumstances affect subsequent ones when EE reaches individually specific limits (Isaacs et al., 2008). These limits are determined, in

[2] Activity level generally was defined to be the breathing rate (L/min) associated with the activity. The EE metric is a more generalized approach to modeling activity level and accommodates non-air exposure modeling (McCurdy, 2000).

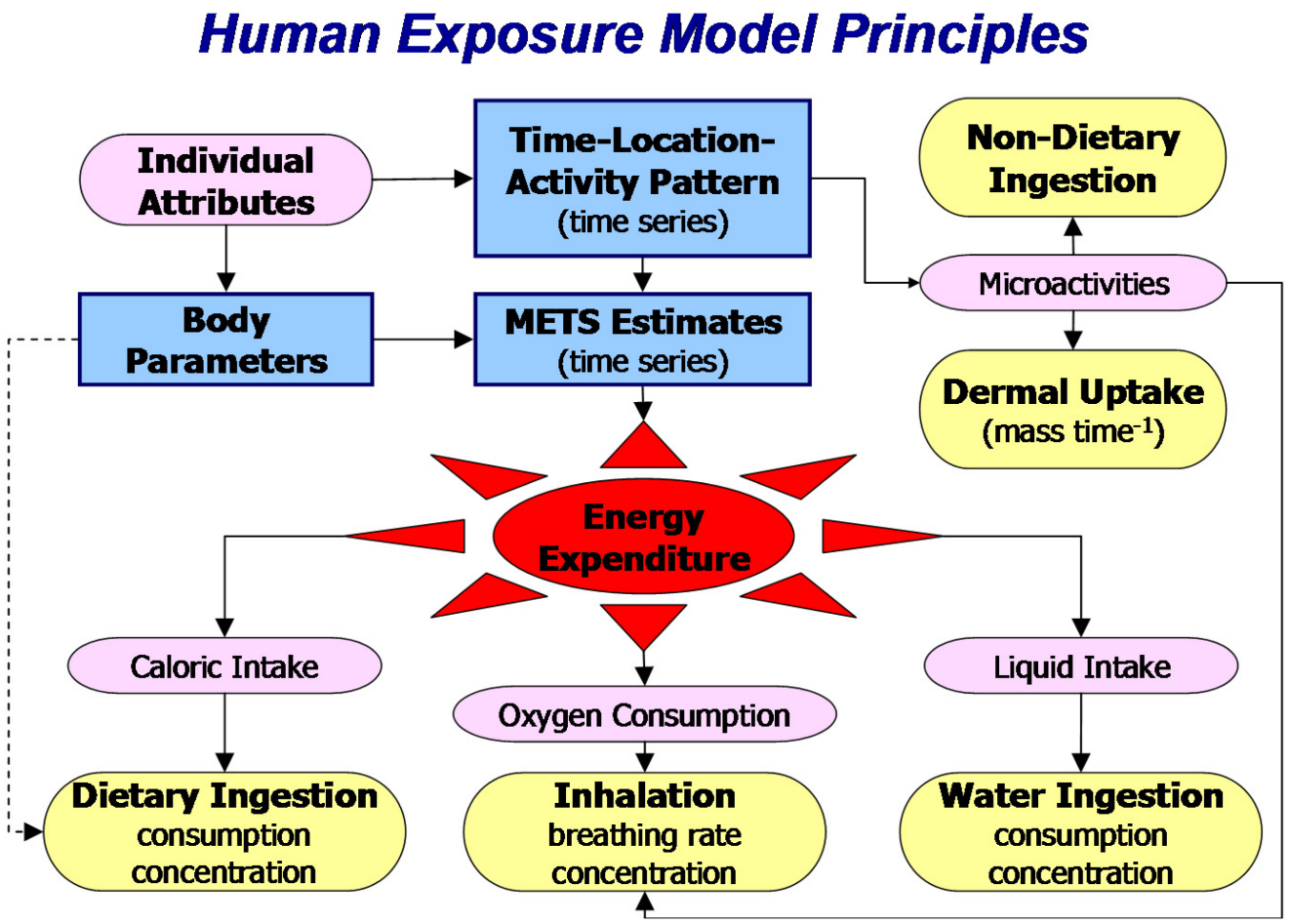


Figure D-4. Human exposure model principles. This schematic diagram illustrates the relationship among activity level, energy expenditure, and the intakes needed to maintain that activity level.

part, by an individual's age, gender, fitness level, and functional (health) limitations that may exist (Figure F-4).

8. Work is defined to be activity-specific energy expenditure. In the APEX and SHEDS models, activity-level-specific energy expenditure (EEa) by an individual i (EEai) is estimated by multiplying an activity-specific relative energy value in metabolic equivalents of work (METSa) sampled from a literature-derived distribution by the modeled person's basal metabolic rate (BMRi)—EEai = BMRi * METSa. See Ainsworth et al. (1993) and McArdle et al. (2001) for a discussion of the METS concept. A person's BMR is dependent on age, gender, health conditions, and lifestyle factors. Numerous equations exist in the nutrition literature for estimating BMRi using a multitude of independent variables (Froehle, 2008; Müller et al., 2004; Schofield, 1985; Speakman, 2005). It is important to note that BMR in older individuals is quite different than that in younger adults; see Section 2.B.
9. Given a µE exposure concentration, activity level ultimately determines a person's intake dose rate, the amount of material inhaled, ingested, or absorbed into an individual (Figure F-4). For inhalation exposures, intake dose rate is a function of the amount of air breathed per unit time multiplied by the µE concentration; its units ideally are in moles/min, but alternative metrics sometimes are used. The magnitude of intake dose rate is affected greatly by the amount of work being undertaken by an exposed person at the time of exposure. The pattern of intake dose rate experienced over time often is called the intake dose profile, and is similar in appearance to the exposure profile depicted in Figure F-3.
10. A relevant dose metric must be utilized to properly address individual dose-effect (D/E) or population dose-response (D/R) relationships (Lorenzana et al., 2005; National Research Council, 2009). However, in general, health effects are associated with the time pattern of dose rate received (Lippmann, 1989; McCurdy, 1997). Knowing this specific pattern (abbreviated as DT/dt) enables any longer term dose metric to be calculated, including dose levels

exceeding selected levels one or more times in a year, the mean dose rate, and other metrics of interest. For example, an exposure assessment conducted for the most recent ozone (O3) NAAQS review (U.S. EPA, 2007a) focused on 8-h peak exposures coincident with moderate or greater exercise levels occurring within a year. Multiple, short-term peak dose metrics like these cannot be uniquely determined from an aggregated, time-averaged dose metric. They only can be modeled using an intake dose rate simulation approach that calculates the time series of exposures such as those produced by the APEX and SHEDS models.

11. Multiple-route intake/uptake dose rates are correlated in an individual because of the bioenergetics of human metabolism. Basically, this principle derives from conservation of mass and energy (McArdle et al., 2001). In contrast, "micro-activity" dose rate uptakes, such as nondietary ingestion associated with hand-to-mouth or hand-to-surface activity—of concern with respect to environmental exposures of children—are not directly associated with bioenergetics but are related instead to age/gender differences in behavioral characteristics of children inhabiting a particular location. Thus, there is a correlation among pathways, and it is maintained in SHEDS-Multimedia by basing dietary and water consumption, as well as ventilation rate, on activity level considerations. Microactivity intake dose rate modeling will not be considered further in this paper. See Tulve et al. (2002) or Xue et al. (2007) for a discussion of microactivity exposure modeling. For modeling air route exposures to older individuals, we assume that there is no nondietary (or dietary for that matter) ingestion resulting from hand-to-mouth activity in that population. This assumption can be evaluated if data on nondietary mouthing behavior become available for older people.

12. There are seasonal, day-of-week (or workday/nonworkday), and meteorological (temperature and precipitation) differences in time use within and among individuals (Fisher et al., 2005; Hill, 1985). EPA exposure models maintain the time use patterns via targeted selection of appropriate CHAD diaries for each day of the simulated year for each individual. This is another reason why average time use data are deficient in capturing and interpreting what people do in time and space.

13. There are day-to-day similarities and differences in locations inhabited and activities undertaken by an individual and among individuals within a larger population cohort (Xue et al., 2004; Glen et al., 2008). These similarities and differences are affected by the contextual culture of a society, habits, and technology. Viewed over time, there is a structure to these effects, resulting in longitudinal patterns of locations visited and activities performed in a population (Echols et al., 1999, 2001; Frazier et al., 2008; Glen et al., 2008). Ramifications of this observation are that both intra- and interindividual variability have to be addressed in an exposure modeling effort, as well as day-to-day correlations within an individual.

14. There are long-term patterns to a person's use of time—called "tracking"—that can be addressed analytically to some extent in multiyear exposure modeling (Elgethun et al., 2003, 2007). Tracking is affected greatly by changing physiological and functional limitations and housing pattern changes in the aged. It is difficult to obtain information on this subject, except in the physical activity literature; see Section 5.

15. Because of the inherent nature of the risk assessment process where judgments have to be made regarding uncertain future events, including intake dose rates associated with inhaling a pollutant by population subgroups undertaking multiple activities in many locations, said assessments often use a stochastic simulation modeling approach (Jordan et al., 1983; Ott et al., 1988). A simulation model facilitates evaluation of variability and uncertainty in parameters of the model, often ignored in many exposure modeling efforts. Uncertainty in the model structure itself, however, only can be addressed by using a different model and comparing output estimates with measured data. This rarely is done because of resource limitations.

D.2 Functional Structure of the APEX Model

How these principles are implemented in the APEX and SHEDS-Air models is shown in Figure D-5. Those symbols and abbreviations not already described above are defined in the List of Abbreviations, Symbols, and Acronyms. Figure D-5 depicts the event-based exposure and intake dose rate simulation logic frequently used in the two models. Specific applications of them may differ in the details depicted. Major model inputs are shown outside of the dashed-line portion of the Figure; they are (1) environmental concentration data, (2) U.S. Census population data, (3) CHAD time use data, and (4) daily meteorological data for the geographical area being modeled. This review focuses on the model processes inside the dashed line portion. Because some of the inputs differ between the APEX and SHEDS models, as well as among different applications of either of the models, it would be tediousfor the reader to continually distinguish among the versions. The following discussion is oriented toward a generalized ideal APEX model.

Area of analysis and population groups of concern. APEX usually is applied at the community- or urban-scale level for three specified air quality conditions, generally described by a period of time: (1) some past time period having measured (or modeled) ambient concentration field data, (2) current (or as is) air quality conditions also using either measured or modeled concentrations, and (3) some indefinite future time when environmental concentrations just meet one or more alternative standards being evaluated. Comparing outputs for these three scenarios provides a quantitative

estimate of the "effectiveness" of each scenario modeled. An example is New York City for as is conditions in 2007 versus just attaining a specified standard level occurring at some future time. (This approach is called a *standards objective* analysis. If a specific control scenario is evaluated, usually compared with an alternative control approach, it is called a *standards impact* assessment [Feagans, 1986]). The population groups of concern may be the entire population or a specific portion of it; exercising children (a small subset of U.S. children) was the focus of EPA's recent O_3 NAAQS exposure analyses (U.S. EPA, 2007a, b). Older adults with compromised cardiovascular systems (chronic obstructive pulmonary disease, angina, etc.) likely will be an important subpopulation to consider for modeling exposures in the next PM NAAQS review.

Environmental concentration field. An environmental concentration field, or profile, is estimated for all outdoor locations in the selected geographic area, often referred to as the modeling domain. This concentration field may be measured (monitored) and/or modeled ambient data; the latter data usually are used for future-time air quality scenarios. The output of this step typically is a time series of hourly concentrations for every hour of the day during the modeling period, usually for an entire year. See "Sequence of Hourly Environmental Concentrations" depicted inside of the dashed lines in Figure D-5.

Microenvironmental-specific concentration estimates are developed from these hourly concentration profiles. If a person is outdoors, the hourly environmental concentration ($C_{OUT.h}$) value itself often, but not always, is equivalent to the ambient concentration and used for this μE for the duration of the exposure event. In other words, a C_t may be the same as an hourly $C_{OUT.h}$ value. Note that, if there is within-hour variability in C_{OUT}, then $C_{OUT.t}$ would be based on the sub-hourly time period of concern, such as 5 min used in the sulfur dioxide NAAQS review.

If a person is indoors or inside a motor vehicle, the concentration within that μE depends on a variety of chemical/physical factors, such as chemical deposition and removal rates, air exchange rate, and indoor source strengths. There have been a number of approaches used to model these factors over the years, but three are most commonly used: (1) solving a mass-balance equation for the specific location; (2) sampling from literature-derived "indoor/outdoor" ratios specific to the μE being modeled (McCurdy, 1995); and (3) using a linear-regression-based algorithm that relates outdoor-to-indoor concentrations (the regression slope) with an additive term (the regression intercept) for indoor sources. The number of indoor locations used in EPA's exposure models range varies with the pollutant being analyzed, but is generally between 7 and 27 specific locations. Usually <10 locations are used. Some examples are home, work, school, retail establishments, motorways, retail stores, and a "residual" location ("other indoors"). Outdoor locations also are subdivided, but the concentration assigned to them may simply be the ambient concentration estimate noted above. The output of these steps is a time series of μE concentration estimates $\{C_1, C_2, C_3 \ldots C_T\}$ for all outdoor and indoor locations that the simulated population may inhabit (see Figure D-5).

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It is possible to model more μEs than the 7 to 27 locations noted above, but input data to calculate the μE concentration are limited for many locations. Most time use studies use a hierarchical locational coding scheme, some down to individual rooms in a home, but rarely do subjects provide data on time spent in them, even for contemporaneous diary studies, for which subjects are supposed to record in some manner where they were at the time, with a new entry for every location inhabited. Remembering specific locations in the commonly used ex post time use recall surveys done over the phone (e.g., "What did you do yesterday?") is almost impossible. Misleading modeling results would occur for specific locations using most recall survey data for exposures in detailed μEs, as there would be a lot of false negatives ("0 time") spent in isolated locations of interest. Thus, only a handful of general microenvironments are considered in most exposure modeling efforts.

There is a lively literature on the diary versus recall protocols used to gather time use data; see Ås, 1978; Collopy, 1996; Fenstermaker, 1996; Geurts and De Ree, 1993; Harvey, 1993; Nickols and Ayieko, 1996; Niemi, 1993; and Stinson, 1999, among others. CHAD contains both recall and contemporaneous diary time use information. See Section 4 for a more detailed discussion of time use data.

Census data. U.S. Census data are a major input to EPA's exposure models. The data are used to define how many people are within the modeling domain, along with their age, gender, employment, housing, and commuting characteristics. The proportion of people in each 1-year age category by gender for the population groups of interest is derived from the Census data and governs the number of simulations undertaken. The Census also provides frequency distributions of work commuting trips among every census tract in the United States (centroid to centroid distances). These data provide an estimate of commuting trips between any pair of census tracts in the area being modeled (e.g., U.S. EPA, 2007a, b).

After characterizing the simulated population, development of an actual pool of simulated persons begins. Suppose that we are interested in modeling the exposures to 45- to 65-year-old workers of both genders. A single person within that age range is selected randomly, say, a 65-year-old female. That person has some probability (using the Census data) of living in a single family residence having gas heating and cooking. A random draw from this probability distribution will assign the person to a single housing type based on the Census probability. Work (paid) or nonwork status is determined from Census probabilities for the subject's age/gender combination. If a worker, the subject will be assigned to a work district (Census tract) location based on Census commuting probabilities. Thus,

the simulated example person is characterized by a specific age, gender, housing type, and home and work locations. Additional characteristics are sometimes used if warranted. This could include variables, such as health status, body mass index, etc., all defined by population probabilities that exist in additionally provided external data, but not in the Census. For example, additional information is needed to determine the proportion of asthmatics aged 65 to 69 years relative to the total population residing within the modeling domain. Activity patterns explicit for people having specific health conditions are uncommon, thus judgments are used to determine the appropriateness of available diary data for use in the assessment (typically not available for the health compromised). If the existing activity data do not reflect what people having a health condition do in time and space, then selected attributes of the diary information have to be adjusted to better represent time use patterns of the modeled group. Sensitivity analyses can then be implemented to evaluate the implications of making these modifications.

This process is repeated until the simulated population has proportionally the same characteristics of the Census-derived population data.

Physiological profile generator. Physiological characteristics are needed for every simulated person in the population pool. The main inputs required to do so are derived from the person's anthropogenic data, such as age, gender, weight (body mass [BM]), height (HT), body mass index (BMI), and health status variables that might affect a person's physiology (e.g., asthma, cardiovascular problems, poor fitness, etc.). BMR is a very important bioenergetic parameter, as we shall see, and it is derived from the age, gender, BM, and HT data for each person. Although a number of equations are available for estimating BMR, the APEX and SHEDS models currently use the Schofield (1985) set of equations that account for variability in age, gender, and BM. Because of criticisms that the Schofield (1985)-derived equations may not reflect current population characteristics, such as the higher BM and larger BMI[3] seen in the current population (Frankenfield et al., 2005; Livingston and Kohlstadt, 2005), the BMR equations used in APEX and SHEDS will change in the near future.

The variables mentioned above also affect a person's maximal oxygen consumption rate ($VO_{2.Max[i]}$), which, in turn, places an upper limit on the amount of air that a person can breathe at maximal exercise ($V_{E.Max[i]}$) (see Blomstrand et al., 1997). Using commonly available physiological relationships (McArdle et al., 2001), $VO_{2.Max[i]}$ can be related directly to a person's $METS_{Max[i]}$. As noted above, METS are activity-specific metabolic equivalents of work based on the ratio of energy expenditure (EE) needed to undertake an activity (EE_A) to a person's BMR_i (Ainsworth et al., 1993, 2006). Activity-specific VO_2 is a function of a person's $VO_{2.Max[i]}$ and prior event work rates (EE) undertaken (Isaacs et al., 2008).

Activity-specific METS, EE, VO_2, and breathing rate (V_E) all are related to each other via well-accepted physiological principles (Isaacs et al., 2008). However, there is still a lot of uncertainty regarding applications of the known principles to actual cases, with limited knowledge concerning the relationship among fitness level, lifestyle, and the physiological parameters mentioned. Many of these uncertainties are amenable to sensitivity analyses, so that implications of the assumptions and relationships used can be addressed quantitatively. If needed for a particular standard assessment, alveolar ventilation (V_A) can be derived from the V_E estimates; EPA staff currently are working on defining new $V_E \rightarrow V_A$ functional relationships for use in the APEX and SHEDS models.

CHAD diary selection criteria. CHAD has 34,773 person-days of diary data available for use in the APEX and SHEDS models. About 41% of them (14,249) are single-day (cross-sectional) diaries. The remainder has between 2 and 369 days of data per person (see Table D-1). To simulate year-long activity patterns requires that single-day diaries be sampled multiple times—a problem that exists with every exposure model because of the dearth of longitudinal time use data. We have developed a method (called the "D&A" approach) of simulating longitudinal activity patterns based on maintaining the intra- and interindividual variability in time use seen in the few repeated-measures analyses of variance that have been undertaken on multiday surveys and replicating the day-to-day correlations within individuals in the time spent in selected, important locations. The method is quite complex but is logically straight-forward and runs fast in the simulations (see Glen et al. [2008]). In essence, the method imposes only as much habitual behavior on individuals and the population (as a whole) that is described in the literature. See Section 4.E for additional discussion of the method and metrics used to implement it.

Conflating CHAD diaries/time use data with the physiological profiles.

The crux of APEX and SHEDS is combining simulated individually specific time use data (activity/location) and concentration patterns with simulated activity-specific breathing rates (VE.A) to obtain intake dose rates. The first step in doing so is to match simulated people with their appropriate diary pool, including seasonal and daily meteorological constraints on human activities. Day-specific National Climatic Center (NCC) data are used to classify every day into one of eight seasonal and meteorological categories (four temperature classes and two precipitation categories: "none/trace" and ">0.5" per day). These become "diary day bins" for the model simulations. Bin definitions are not fixed but are defined according to the simulation objectives.

[3] BMI = BM (kg)/HT2 (m), a widely used index of relative fatness

Table D-1. Summary of the CHAD Database

Study Name	Year*	Diaries	Number of Days of Data per Person		Sponsor
			Range	Median	
Denver MSA	1983	805	1	1	EPA
Washington, DC, MSA	1983	699	1	1	EPA
Cincinnati MSA	1986	2,614	1-3	3	EPRI
California - adolescents	1988	183	1	1	CARB
California - adults	1988	1,579	1	1	CARB
Los Angeles - elementary	1989	51	3	3	API
Los Angeles - high school	1990	43	2-3	3	API
California - children	1990	1,200	1	1	CARB
Valdez, AK	1991	397	1	1	Oil companies
NHAPS - A	1994	4,723	1	1	EPA
NHAPS - B	1994	4,663	1	1	EPA
PSID (CDS) I	1997	5,616	1-2	2	NICHHD
Baltimore Elderly	1998	391	1-24	14	EPA
EPA # 1	2000	367	367	367	EPA
RTP Unhealthy	2001	1,000	8-33	32	EPA
Seattle MSA	2002	1,693	5-10	10	EPA
EPA # 2	2002	197	197	197	EPA
PSID (CDS) II	2003	4,782	1-2	2	NICHHD
RTI Averting Behavior	2003	2,907	1-6	4	EPA
Internal EPA	2007	432	35-69	54	EPA
EPA #1	2007	369	369	369	EPA
Mother and Child	2008	62	31	31	EPA
Totals		34,773			

Notes and Abbreviations:

* The last year of a multiyear study is used.

Number (of days)

API = American Petroleum Institute

CARB = California Air Resources Board

CDS = Child Development Supplement

EPA = Environmental Protection Agency

MSA = Metropolitan Statistical Area

NICHHD = National Institute of Child Health and Human Development

PSID = Population Study of Income Dynamics

RTI = Research Triangle Institute

RTP = Research Triangle Park

The simulations are undertaken on an event-by-event basis, beginning at midnight on the first day of the analysis period. For each person, a diary is selected from the appropriate bin, and a breathing rate is modeled for each event undertaken. This is repeated for the daily sequence of activities, and the output is a string of hourly averaged VE estimates developed from event-specific EE estimates. A daily physical activity index (PAI) is calculated from the time-weighted average of the sum of all the event-specific EE estimates for the day. PAI can be used to provide a check on the physiological modeling procedure used in APEX and SHEDS (McCurdy and Xue, 2004) and as a surrogate for a person's lifestyle and fitness level. In fact, each person's median PAI can be calculated directly from the CHAD data and could be one of the physiological metrics used to develop the diary pools in the first place (see above).

All of these steps use stochastic processes. The Ct estimates are partly the result of sampling from known or approximated distributions of mass-balance equation parameters (or from indoor/outdoor μE relationship data). Monte Carlo techniques are used for this sampling. The same is true for most of the physiological parameters needed to estimate energy

expenditure, oxygen consumption, ventilation (breathing) rate, and alveolar ventilation rate, if needed. This stochastic approach is used to ensure that population variability is addressed regarding the parameters of interest.

Modeling intake or uptake dose. The second major step in estimating exposure and dose patterns is to combine the μE-specific concentration field with the physiological profiles described above. The simulated person goes through her or his day, comes in contact with a concentration (or not) on an event-by-event basis, and receives a dose based on the estimated activity level. When the day is completed, the next day is modeled for the person, continuing for every day in the simulation period, usually a year. The entire process is repeated for every individual in the simulated population.

Intermediate model outputs (for inhalation exposure analyses) are strings of 1-h averaged exposure estimates, 1-h averaged VE estimates, and 1-h dose estimates (e.g., E * VE) for each person, plus any aggregation of them for whatever time period is of interest. This is the dose profile mentioned earlier. For O3, for example, the main APEX output of interest is the number of 8-h daily maximum (the highest 8 h in each day) incidences of exposures when people, especially children, were exercising at ≥27 L min-1 m-2 (this is a body surface area normalized ventilation metric). An illustration of this type of model output appears as Figure F-6; it depicts the 8-h daily maximum exposure estimates for three population groups in 12 Metropolitan Statistical Areas for one air quality scenario, with 2002 air quality just meeting the current O3 8-h daily maximum standard. Five other scenarios also were evaluated (not shown). Separate sensitivity analyses of many of the model parameters were simulated in this assessment, giving an estimate of confidence intervals about the percentage values depicted in Figure F-6, (although not shown in the figure). A more thorough discussion of this sensitivity analysis is presented in U.S. EPA (2007b).

Modeling Response to a Dose

The next step after modeling the dose profile is estimating a response—adverse or not—from the time pattern of dose rate received. The loci of the response eventually will be at the cellular level but, currently, is at the organ level or at a whole-body systems level, using some type of toxicokinetic modeling approach. EPA has funded a number of reports describing how this approach can be used to model adverse health effects to older adults associated with exposures to xenobiotic substances. See Hattis and Russ (2003), Ginsberg et al. (2005), and Krishnan and Hattis (2005) for example risk assessment documents focused on older people. Although dose-response and toxicokinetic modeling are needed to explicitly define health effects associated with intake dose rates, the topics are discussed extensively in the scientific literature and really are one step removed from the exposure/intake dose modeling focus of this report.

D.3 Exposure Model Evaluation

The APEX and SHEDS models have received only a limited amount of evaluation against measured personal monitoring data over the years. In general, OAQPS compares some of their exposure estimates against personal monitoring data, but usually the latter are for longer averaging times than those of interest in the exposure assessment. For instance, OAQPS compared O3 exposure estimates for children against weekly average personal monitoring data obtained for a few weeks in 1995-1996 in two separate areas of San Bernardino County: (1) urban Upland, CA, and (2) two small mountain towns (Langstaff, 2006; U.S. EPA, 2007a). That was the only dataset available to the Agency for such a comparison, even though it was relatively old and based on a longer averaging time (6 to 7 days) than of interest in the assessment (1- or 8-h daily exposures). The APEX model performed reasonably well in the mid-range of the cumulative distribution of weekly exposure estimates (20th to 70th percentiles) but systematically overestimated the low end of the exposure distribution and systematically underestimated the high end (U.S. EPA, 2007a). This phenomenon has been found in all synoptic short- to mid-term model evaluation efforts of which the author is aware: Burke et al. (2001), Law et al. (1997), Ott et al. (1988), and Zartarian et al. (2000, 2006). The overestimate of low-end exposures is not of much interest, because health risks associated with low-end exposures generally are not of regulatory concern (McCurdy, 1995). The probable cause of systematically underestimating high-end exposures results from the models' inability to mimic repeated daily activity patterns that lead to high exposures seen in the measured data (Law et al., 1997). Thus, the main reason for model underestimation is basically a longitudinal time use issue, although the current D&A procedure may reduce activity variability over time and improve model performance. The impact of using the D&A approach has not been evaluated thoroughly with respect to exposure model output distributions.

The impact that time use data per se have on APEX exposure modeling results has received a limited amount of sensitivity analyses (Nysewander et al., 2009). These analyses consisted of 5,000 simulations of seven time use variables in two urban areas, Atlanta and Boston, using the APEX model. The locational codes used in CHAD were collapsed to 12 aggregated locations that accounted for all places visited by every individual in the simulations (all 24 h were accounted for, in other words.) A number of "impact" indices were used to describe sensitivity: time spent in each microenvironment, daily average and 1-hour maximum O_3 exposure estimates, and distributional tests. The seven variables included the following.180

1. Selection of the appropriate intra- and interindividual statistics to combine diary days into longitudinal patterns
2. Choice of the "key location" used to sort the above statistics (e.g., in vehicles versus outdoor time)
3. Differences in start and stop times for the diary day (All events were shifted forward and backward 1 h.)

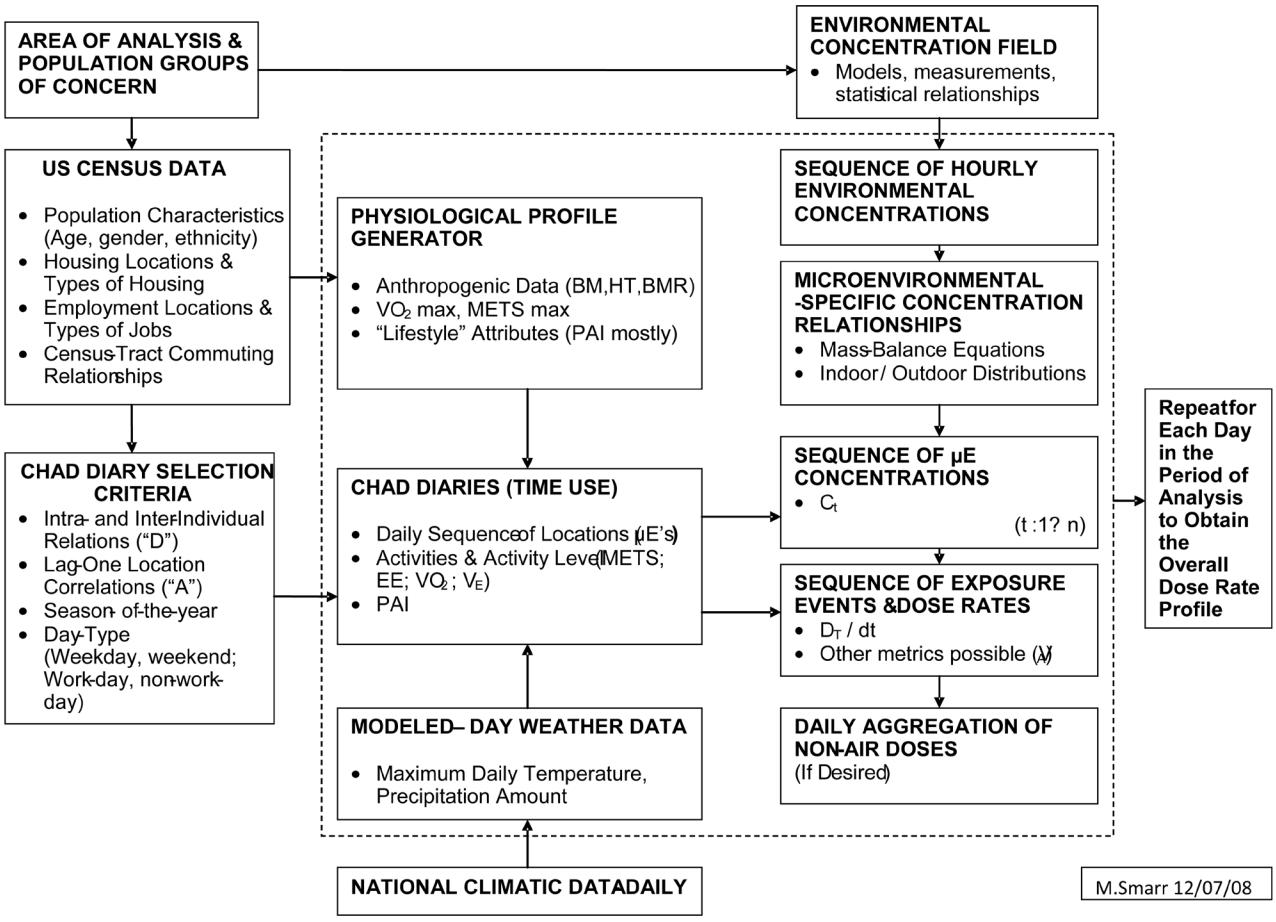

Figure D-5. Logic flowchart of the APEX model.

4. Using diaries from different years to test changes in time spent outdoors by children (There was a 5.2-min decrease per year in this time for CHAD diaries from the 1980s to 2007.)
5. Alternative assignments of "ambiguous location codes" to either indoors or outdoors (e.g., travel by boat—indoors or outdoors?)
6. Modifying the diary "weights" used in the National Human Activity Pattern Survey
7. Level of detail in the diaries (Short events were collapsed into longer durations of 2-, 5-, 10-, and 15-min durations.)

Using the exposure impact indices, differences among the various simulations were greater than simply selecting diaries at random, but the differences were small: ~1% to 2% versus ~0.2% to 0.5%. The one exception was age of the diary data itself (the year that the data were obtained). Using the older diaries *increased* exposure estimates by ~1.5% to 21.8% (Nysewander et al., 2009), mostly because high-end O_3 exposures were associated with time spent outdoors, which has decreased over the years. However, this finding may be a result of how the diaries themselves were coded for the different μEs, rather than a function of age of the diary per se. More work on understanding the impacts of age of diary data is needed before a definitive conclusion can be made about the topic.

It should be noted that obtaining longitudinal personal exposure data is extremely expensive, especially when using "active" short-term monitors (as opposed to passive long-term "diffusion tubes" that are based on Brownian movement). Active personal monitoring involves attaching a monitor having a small pump to each individual on a daily basis, usually at the subject's home at a preselected time. Active monitoring requires a field staff, multiple (expensive) monitors, and detailed logistics. These types of studies also involve collecting time use data. Needless to say, these are invasive protocols, and it is difficult to retain subjects for periods longer than a week at a time. A monitoring study—passive or active—reflects "the state of nature" at the time of the study, including the unique societal and environmental conditions present at that time. Because these conditions generally will not be present at some future time when environmental control scenarios being modeled are implemented, there is uncertainty concerning applicability of exposure/dose relations found in the past in one area being applicable in another area at a different time. From the modeling perspective, the best use of monitoring data is to "ground-truth" performance of the model itself.

A concerted sensitivity/uncertainty evaluation of EPA's time series exposure models following the principles advocated in Saltelli et al. (2000) would be useful and provide insights into those variables and parameters that significantly affect their performance.

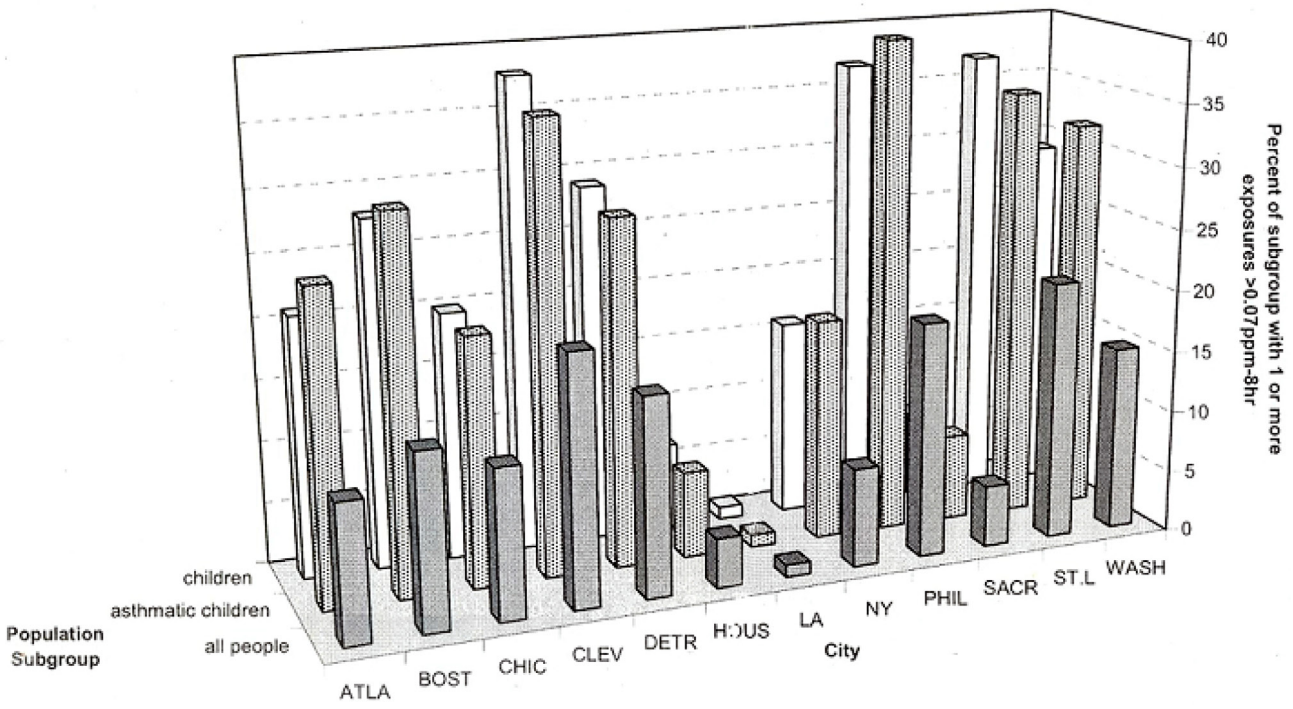

Figure D-6. Percent of people in three groups—(1) all children, (2) asthmatic children, and (3) all persons—estimated to experience 1+ days with an 8-h daily maximum O3 exposure >0.07 ppm while at moderate exercise when the current 8-h daily maximum NAAQS of 0.08 ppm is just met.

APPENDIX E
Supplemental Material

This Appendix consists of three independent supplements focused on abbreviations and symbols used in this report; a glossary of terms used; and a table of common conversion factors used in the exercise physiology and clinical nutrition literatures.

E-1. Abbreviations & Symbols Used in this Synthesis

Symbol	Definition	Units	Alternate Units
♀	Female(s)		
♂	Male(s)		
→	For relationships between two variables:		
≈	Approximately equal to		
≥	Greater than or equal to		
≤	Less than or equal to		
α	Alpha (level of significance: probability of rejecting a true H_O)		
β	Beta (power of the test: probability of rejecting a false H_O when it is false)		
σ^2	Variance		
μ	Mu; a prefix=10^{-6}; in exposure assessment it means “micro”		
μE	Microenvironment: a location with C_t for a specified time period		
ACSM	American Council of Sports Medicine		
ADL	Activities of daily living		
AIHC	American Industrial Health Council		
ANOVA	Analysis of variance		
APEX	Air Pollution Exposure Model (an OAQPS exposure model)		
AT	Anaerobic threshold	L/min	L min^{-1}
ATP	ATP: Adenosine triphosphate		
ATPD	Ambient temperature and pressure, dry.		
ATPS	Ambient temperature and pressure, saturated with water vapor		
atm	Standard atmospheric pressure	Pascals (Pa)	bar
a-vO_2 Diff	Difference in oxygen content between arterial and mixed venous blood.		
BF	Body fat	kg	
BLSA	Baltimore Longitudinal Study of Aging		
BM	Body mass [commonly: “weight”]	kg	
BMI	Body mass index [BM/HT ^{2}]	kg/m^2	kg m^{-2}
BMR	Basal metabolic rate (functionally identical to REE or RMR)	kcal/d	kcal kg^{-1} d^{-1}
bpm	Heart rate	beats/min	beats min^{-1}
BRFSS	Behavioral Risk Factor Surveillance Survey		
BSA	Body Surface Area	m^2	
BTPS	Body temperature and ambient barometric pressure, saturated with water vapor		
C	Calorie [English units]	1,000 kcal	Calorie
°C	Degrees Celisius (Centigrade)		
cal	Calorie, a measure of work		

E-1. Abbreviations & Symbols Used in this Synthesis (continued)

Symbol	Definition	Units	Alternate Units
CASAC	Chemical Abstracts Service		
CASAC	Clean Air Scientific Advisory Committee		
cc	Cubic centimeter		
CC	Closing capacity		
CDC	Centers for Disease Control and Prevention		
C_{dyn}	Dynamic compliance		
cfd	Cumulative frequency distribution		
CHAD	Consolidated Human Activity Database (www.epa.gov/chadnet1)		
CHD	Coronary heart disease		
CI	Confidence Interval		
cm	Centimeter		
CNS	Central nervous system		
CO	Carbon monoxide		
CO_2	Carbon dioxide		
COLD	Chronic obstructive lung disease		
COPD	Chronic obstructive pulmonary disease		
COV	Coefficient of variation (= standard deviation/mean)	unitless	
C-S	Cross-sectional [a study type focused on a single time period]		
C_t	Concentration at time period "t" [air medium]	$\mu g/m^3$	$\mu g\ m^{-3}$
CV	Coefficient of variation (= standard deviation/mean) [CV=COV]	unitless	
d	day		
D	Dose (various units and time periods)		
D_I	Intake dose		
D_L	Diffusing capacity of the lung	mL/min	$mL\ min^{-1}$
D_I	Intake dose rate [air media]	moles/min	$moles\ min^{-1}$
D&A	"Diversity & Autocorrelation" [an approach to developing exposure cohorts]		
DIT	Dietary induced thermogenesis (EE expended to digest food)	kcal	
DLW	Doubly labeled water [having a chemical composition of $^2H_2{}^{18}O$]	L	
D/E	Dose-effect relationships [for an individual]		
D/R	Dose-response relationship [for a population]		
D_{It}/dt	Time pattern of intake dose rate	$moles/min^2$	$moles\ min^{-2}$
E	Exposure [various units and averaging times]	$\mu g\text{-}min/m^3$	ppm min
ECG	Electrocardiogram		
EE	Energy expenditure [various units and averaging times]	kcal	$kcal\ d^{-1}$
EE_A	Activity-specific energy expenditure	kcal	$kcal\ d^{-1}$
$EE_{/BM}$	EE per body mass	kcal/kg-min	$kcal\ kg\text{-}min^{-1}$
$EE_{/FFM}$	EE per fat-free body mass	$kcal/kg_{FFM}$-min	$kcal\ kg_{FFM}\text{-}min^{-1}$
$EE_{/LBM}$	EE per lean body mass	$kcal/kg_{LBM}$-min	$kcal\ kg_{LBM}\text{-}min^{-1}$
EELV	End-expiratory lung volume	%TLC	
EFH	EPA's Exposure Factors Handbook		
EI	Energy intake		
ELV	Effective lung volume		
EMRB	Exposure Modeling Research Branch (a part of HEASD)		
EPA	US Environmental Protection Agency		
EPOC	Excess post-oxygen consumption	kcal	
ERV	Expiratory reserve volume	L/min-m2	
est.	Estimate, estimated		
EVR	Equivalent ventilation (breathing) rate [V_E/BSA]	$L/min\text{-}m^2$	$L\ min^{-1}\ m^{-2}$
Ex	Exercise: planned, structured, & purposeful physical activity		
◦F	Degrees Fahrenheit		

E-1. Abbreviations & Symbols Used in this Synthesis (continued)

Symbol	Definition	Units	Alternate Units
f_C	Heart rate	bpm	
FEF	Forced expiratory flow	L	
FEF_t	FEF for a specified "t" time		
FEV_t	Forced expiratory volume in "t" time		
FEV_1	Forced expiratory volume in one second		
FFM	Fat-free mass	kg	
f_R	Breathing (ventilation) rate	L/min	$L\ min^{-1}$
FRC	Functional residual capacity	L	
ft	Foot: a measure of length=12 inches		
FVC	Forced vital capacity		
Gaw	Airway conductance		
h	Hour		
^2H	An isotope of hydrogen: deuterium		
$^2H_2{}^{18}O$	Chemical formula for DLW		
HbO_2	Oxyhemoglobin		
H_O	A hypothesis subjected to statistical testing		
HEASD	Human Exposure and Atmos. Sci. Division (a part of NERL)		
HDL	High-density lipoprotein cholesterol		
HR	Heart rate	bpm	$beats\ min^{-1}$
HR_A	Activity-specific heart rate		
HR_{MAX}	Maximal heart rate	bpm	beats/min
HR_{PEAK}	Peak heart rate (functionally identical to HR_{MAX})		
HR_R	Resting heart rate	bpm	$beats\ min^{-1}$
HRR	Heart rate reserve [HR_{MAX} - HR_R]	bpm	beats/min
HT	Height	m	cm
IADL	Indep. Act. of daily living [min. ADL for non-institutional living]		
IC	Inspiratory capacity		
ICC	Intraclass correlation coefficient		
IEEE-MBS	Inter. Elect. & Electron. Engineers; Med. & Biol. Section		
IRV	Inspiratory reserve volume	L	
IVC	Inspiratory vital capacity		
J	Joule, a unit of work or mechanical energy		
◦K	Degrees Kelvin (1K=273 ◦C)		
kcal	Kilocalorie	cal	
kg	Kilogram; a measure of mass		
km	Kilometer; a measure of distance		
K-S	Kolmorgov-Smirnoff "non-parametric" test of two distributions		
L	Liter		
LAT	Lactic acid threshold		
LBM	Lean body mass [=fat-free mass]	kg	
LPA	Light physical activity [a category of activity]		
m	Meter: measure of length		
$_{MAX}$	A subscript denoting "maximum" or "maximal"		
METS	Metabolic Equivalents of work (unitless) [METS at rest=1.0]		
$METS_A$	Activity-specific METS (unitless)		
$METS_{MAX}$	Maximal achievable or measured METS (unitless)		
$METS_{RES}$	METS reserve [METSMAX - 1.0] (unitless)		
min	Minute		
mo	Month		
MPA	Moderate physical activity		
MVPA	Moderate & vigorous physical activity		
MVR	Minute ventilation rate [V_E]		

E-1. Abbreviations & Symbols Used in this Synthesis (continued)

Symbol	Definition	Units	Alternate Units
MVV	Maximal voluntary ventilation		
n	Sample size		
N	Newton		
NAAQS	National Ambient Air Quality Standard		
NAPAP	National Acid Precipitation Assessment Program		
NCEA	National Center for Environmental Assessment (a part of EPA)		
NCHS	National Center for Health Statistics (a part of NIH)		
NEAT	Non-exercise activity thermogenesis		
NEM	NAAQPS Exposure Model		
NERL	National Exposure research Laboratory (a part of EPA)		
NHANES	National Health and Nutrition Examination Study		
NHAPS	National Human Activity Pattern Survey		
NHEERL	National Health and Environmental Effects Lab. (a part of EPA)		
NHIS	National Health Interview Survey		
NHLBI	National Heart, Lung, and Blood Institute		
NIA	National Institute on Aging		
NICHHD	National Institute of Child Health and Human Development		
NIH	National Institute of Health		
NO	Nitric oxide		
NO_2	Nitrogen dioxide		
NO_X	Nitrogen oxides		
ns	Not significant: associated with a statistical test at some specified α level		
O_2	Molecular oxygen		
O_3	Ozone		
OAQPS	Office of Air Planning and Standards (a part of OAR/EPA)		
OAR	Office of Air and Radiation (a part of EPA)		
OEL	Occupational Exposure Limit		
ORD	Office of Research and Development (a part of EPA)		
OSHA	Occupational Safety and Health Administration		
p	Probability		
P	Pressure		
PA	Physical activity		
PAEE	Physical activity energy expenditure	kcal	
PAEE	Physical Activity Index [various definitions; generally TDEE/BMR]	unitless	
PAI	Physical Activity Level (identical to PAL)		
PAL	Physical Activity Level (identical to PAI)	L/min	$L\ min^{-1}$
PAO_2	Arterial partial pressure of oxygen		
PaO_2	Alveolar partial pressure of oxygen		
PE	Physical education; generally as in “class” of structured PA		
PEFR	Peak expiratory flow rate	L/min	$L\ min^{-1}$
PEFV	Peak expiratory flow volume	L	
PEL	Permissible Exposure Level		
PEM	Personal exposure monitor		
PFI	Personal Fitness Index		
PM	Particulate matter (particles or aerosols of varying sizes)		
$PM_{2.5}$	PM with a mean aerodynamic diameter of 2.5 microns or less		
pNEM	Probabilistic NAAQS Exposure Model		
PO_2	Pratial oxygen pressure		
ppb	Parts per billion		
pphm	Parts per hundred million		
ppm	Parts per million		
PWC	Physical working capacity		
Q	Cardiac output (blood flow)	mL/min	$mL\ min^{-1}$

E-1. Abbreviations & Symbols Used in this Synthesis (continued)

Symbol	Definition	Units	Alternate Units
Q_C	Capillary perfusion		
r	Pearson "product-moment" correlation coefficient		
r_S	Spearman rank-order correlation coefficient		
R	Gas exchange ratio (also known as RQ)	unitless	
Raw	Airway resistance		
REE	Resting energy expenditure (functionally identical to BMR)	kcal/d	kcal kg^{-1} d^{-1}
REL	Recommended Exposure Limit		
RER	Respiratory exchange ratio	unitless	
$_{RES}$	A subscript denoting "reserve" [$_{MAX}$ - $_{MIN\ (or\ REST)}$]		
RH	Relative humidity		
RMR	Resting metabolic rate (functionally identical to BMR)	kcal/d	kcal kg^{-1} d^{-1}
RQ	Respiratory quotient [VCO/VO2, as volumes]	unitless	
RR	Respiratory rate [V_I]	L/ min	L min^{-1}
RV	Residual volume	L	
SD	Standard deviation of the mean		
SE	Standard error of the estimate [SE=SD/√n]		
sec	Second		
SEE	Sleeping energy expenditure	kcal	
Sgaw	Specific airway conductance		
SHEDS	Stochastic Human Exposure and Dose Simulation model		
SI	Systéme Internationale d'Unités (international system of scientific units)		
STP	Standard Temperature and Pressure		
STPD	Standard Temperature and Pressure, Dry		
SV	Stroke volume	mL	
$_t$	An index of time, used as a subscript generally		
TDEE	Total daily energy expenditure	kcal	
T_E	Time for one exhaled breath	sec	
T_I	Time for one inhaled breath	sec	
T_{TOTAL}	Time it takes for one complete breathing cycle (T_{TOTAL}= T_I + T_E	sec	
TLC	Total lung capacity	L	
TLV	Threshold limit value		
™	Trade Mark		
TRIM	Total Risk Integrated Method (an OAQPS risk modeling approach)		
TSP	Total suspended particulates		
T_V	Total volume	L	
TWA	Time-weighted average concentration	µg/min	ppm
U	Conversion factor between EE and VO_2 (kcal↔L/min)		
V_A	Alveolar ventilation rate	L/min	L min^{-1}
VAT	Ventilatory anerobic threshold	L/min	L min^{-1}
VC	Vital Lung capacity	L	
VCO_2	Carbon dioxide ventilation rate produced during respiration	mL/min	mL min^{-1}
V_D	Dead-space volume	L	
V_E	Ventilation (breathing) rate = minute ventilation rate	L/min	L min^{-1}
$V_{E/BM}$	Ventilation rate per body mass	L /kg_{BM}-min	L kg_{BM}^{-1} min^{-1}
$V_{E/LBM}$	Ventilation rate per lean body mass	L /kg_{LBM}-min	L kg_{LBM}^{-1} min^{-1}
$V_{E.A}$	Activity-specific ventilation rate	L/min	L min^{-1}
$V_{E.A/BM}$	Activity-specific ventilation rate per body mass	L /kg_{BM}-min	L kg_{BM}^{-1} min^{-1}
$V_{E.MAX}$	Maximal ventilation rate (defined by an exercise protocol)	L/min	L min^{-1}
$V_{E.MAX/BM}$	Maximal ventilation rate on a per body mass basis	L /kg_{BM}-min	L kg_{BM}^{-1} min^{-1}
$V_{E.R}$	Resting ventilation rate ("basal" or resting conditions)	L/min	L min^{-1}

E-1. Abbreviations & Symbols Used in this Synthesis (continued)

Symbol	Definition	Units	Alternate Units
$V_{E.R/BM}$	Resting ventilation rate on a body mass basis	L /kg_{BM}-min	L kg_{BM}^{-1} min^{-1}
$V_{E.RES}$	Ventilation rate reserve [$V_{E.MAX}$ - $V_{E.R}$]	L/min	L min^{-1}
$V_{E.RES/BM}$	Ventilation rate reserve on a per body mass basis[$V_{E.MAX}$ - $V_{E.R}$]	L /kg_{BM}-min	L kg_{BM}^{-1} min^{-1}
V_I	Inspired ventilation rate	L/ min	L min^{-1}
V_L	Lung volume	L	
V_T	Total volume of the pulmonary system (lungs & conducting airways)	L	
VO_2	Oxygen uptake or consumption rate	mL/min	mL min^{-1}
$VO_{2/BM}$	Oxygen consumption per body mass	mL /kg_{BM}-min	mL kg_{BM}^{-1} min^{-1}
$VO_{2/LBM}$	Oxygen consumption per lean body mass	mL /kg_{LBM}-min	mL kg_{LBM}^{-1} min^{-1}
$VO_{2.A}$	Activity-specific oxygen consumption rate	mL/min	mL min^{-1}
$VO_{2.A/BM}$	Activity-specific oxygen consumption rate per body mass	mL /kg_{BM}-min	mL kg_{BM}^{-1} min^{-1}
$VO_{2.MAX}$	Maximal oxygen consumption rate (defined by an exercise protocol)	mL/min	mL min^{-1}
$VO_{2.MAX/BM}$	Maximal oxygen consumption rate per body mass	mL /kg_{BM}-min	mL kg_{BM}^{-1} min^{-1}
$VO_{2.PEAK}$	Peak oxygen consumption rate (functionally = $VO_{2.MAX}$)	mL/min	mL min^{-1}
$VO_{2MAX/BM}$	Peak oxygen consumption rate per body mass (functionally = $VO_{2.MAX/BM}$)	mL /kg_{BM}-min	mL kg_{BM}^{-1} min^{-1}
$VO_{2.R}$	Resting oxygen consumption rate	mL/min	mL min^{-1}
$VO_{2.R/BM}$	Resting oxygen consumption rate per body mass	mL /kg_{BM}-min	mL kg_{BM}^{-1} min^{-1}
$VO_{2.RES}$	Oxygen consumption rate reserve [$VO_{2.MAX}$ - $VO_{2.R}$]	mL/min	mL min^{-1}
$VO_{2.RES/BM}$	Oxygen consumption rate reserve per body mass [$VO_{2.MAX/BM}$ - $VO_{2.R/BM}$]	mL /kg_{BM}-min	mL kg_{BM}^{-1} min^{-1}
VPA	Vigorous physical activity [a category of activity]		
VQ	Ventilatory equivalent [V_E / VO_2]	unitless	
V_T	Tidal volume of the lungs per breath	L/breath	L $breath^{-1}$
V_T/V_I	Mean inspiratory flow; an index of “respiratory drive”	L/sec	L sec^{-1}
VT	Ventilatory (anaerobic) threshold	L/ min	L min^{-1}
w	Week		
W	Watt		
W_{170}	Work performed at a heart rate of 170 bpm	W	J/sec
WHO	World Health Organization (part of the United Nations)		
y	Year		

E-2 Glossary of Terms Used in this Synthesis

A

Absorbed Dose: The amount of a chemical or substance penetrating an absorption barrier--the exchange boundaries of the skin, lung, or digestive tract--through an uptake process via either a physical or biological process per specified time period; that process often is diffusion through a resisting boundary layer (IPCS, 2000).

Absorption Barrier: Any of the exchange barriers of the body that allow differential diffusion across a boundary (e.g., the lung).

Activity: From an exposure perspective, a specific action related to a human behavior or task that may result in an exposure to the substance of interest. Recently, activities have been further differentiated into "macro-activities" and "micro-activities." Data on activities often are developed from a time-use, or human-activity, study.

Activity Pattern: A series of events describing what a person does (the activity), her or his activity "level" associated with the activity (the estimated energy expended), and where the activity occurs (location, or the "microenvironment"). This information is gathered via a time/activity study using various techniques.

Acute Exposure: A vague term that loosely relates to a short-term, high-peak exposure even, usually < 24h in duration (IPCS, 2000).

Adiposity: The amount of body fat, presented either as a weight (mass) or as a percentage of total body mass. Adiposity is estimated by skinfold measurements, bioimpedance analysis, underwater densitometry, and body scanning by x-rays or computerized tomography. "Excess" adiposity is a measure of obesity.

Adolescence: (1) The state or process of growing up. (2) The period of life from puberty to maturity terminating legally at the age of majority (Webster's, 1984).

Adolescent: (1) A human that is between 11-21 years old (Bar-Or & Baranowski, 1994).

Aerobic: (1) Using or requiring oxygen, said of an organism (International). (2) Denoting an environment in which free oxygen is present (International, 1986).

Aerobic Exercise: Activity in which the amount of oxygen consumed increases directly with the amount of physical exertion (Solomon, 1984). Aerobic activity involves an associated increase in respiratory rate, heart rate, stroke volume, systolic blood pressure, and coronary blood flow (Leon, 1989).

Aerobic Fitness: The capacity to accomplish endurance activities that depend largely on aerobic metabolism (Legér, 1996).

Aerobic Power: A synonym for maximal oxygen consumption/uptake [see].

Aerobic Training: Training that improves the efficiency of the aerobic energy-producing systems and which often improves cardiorespiratory endurance (Nieman, 1999).

Aerobic Scope: The ratio of $VO_{2\,MAX}$ to $VO_{2\,R}$, which is approximately equal to $VO_{2\,MAX}$ / $VO_{2\,BASAL}$ and HR_{MAX} / HR_{R}. It is also known as the "metabolic scope" (Rowland, 1989). This term is rarely seen in the newer literature; the term used now is $VO_{2.RESERVE}$.

Agent: A chemical, physical, mineralogical, or biological entity that may cause deleterious effects in an exposed organism (IPCS, 2000).

Age-Predicted HR_{MAX} (A/P HR_{MAX}): A formula that is widely used in the exercising physiology literature to estimate HR_{MAX} at maximal voluntary exercise (exertion). The most common formula used is: A-P HR_{MAX} = (220 - Age), in units of bpm. One alternative version uses 226 in lieu of 220; see, for example, Dishman (1994). There are other definitions used.

Air Exchange Rate: The rate at which outside air replaces indoor air in a given space (IPCS, 2000).

Airways: Air passages in the "respiratory tree" that includes the pharynx, larynx, trachea, and the lung.

Allometric Equation: The equation used to "describe" the relationship between a physiological attribute and body mass (BM) or other body parameter (Rowland, 1996). It takes the general form: Y = a * BMb

Where: Y = An attribute of interest (e.g., basal metabolism)

a = a constant of proportionality

b = the power parameter needed to make "a" a constant

Taking logs of both sides gives the linear-transformed equation:

Log Y = log a + (b * log BM)

There is regularity in the value of exponent b for classes of physiological attributes, especially those dealing with flow rates in the body: cardiac output, minute volume (ventilation rate), basal metabolic rate, oxygen consumption, glomerular filtration rate, and food/water consumption (IPG, 1992). Many of these rates are directly associated with the amount of energy expended by the organism. See: "Energy Expenditure" and "Total Daily Energy Expenditure." Biological rates tend to maintain proportionality with $BM^{0.75}$ (IPG, 1992), but using lean body mass (LBM) often shows less variability in the relationship.

The BM exponent "b" for volumes and capacities of the body, such as blood volume, organ sizes, and lung volume, tend to maintain proportionality with BM^{1} (BM) in large and small mammals (IPG, 1992). A lively literature exists on cross-species scaling, on both empirical and theoretical grounds. $BM^{0.67}$ ($BM^{2/3}$) is another metric often used and analyzed. See Rowland (1996) for a succinct discussion of scaling in humans.

Allometry: (1) The measure and study of relative growth of a part with respect to the entire organism (Webster's, 1984). (2) The study of the regular variation in features of anatomy and physiology as a function of overall body size (IPG, 1992). (3) The study of the variation in physiological "attribute" of mammals--and the consequences of that variation--as function of body mass or other body parameter [body surface area is sometimes used] (IPG, 1992). Some of the "attributes" that are investigated include heart rate, basal metabolism, and blood flow.

Alveolar: Pertaining to an alveolus, which is an air cell, one of the terminal saclike dilations of the alveolar ducts in the lung (Stedman's, 1982).

Alveolar Air: Literally, air present in the pulmonary alveoli that participates in gas exchange with blood in the pulmonary capillaries. This air cannot be obtained for analyses, so the last portion of air expelled in a deep expiration approximates it in composition--however, this expelled air comes from the respiratory bronchioles and alveolar ducts, as well as the alveoli (Morehouse & Miller, 1976). Thus, deeply expired air does not equal alveolar air.

Alveolar Ventilation: The process in which gas exchange with blood occurs (Dorland's, 1988).

Alveolar Ventilation Rate (V_A): The rate at which the total ventilation volume is involved in gas exchange with the blood. Alveolar ventilation is < total ventilation because when a tidal volume of gas leaves the alveolar spaces, the last part does not get expelled from the body but occupies the dead space, to be re-inspired with the next inspiration. Thus, the volume of alveolar gas actually expelled completely is equal to tidal volume minus dead space volume (EPA, 1989).

Aveoli: Tiny air sacs in the lungs through whose walls gases such as oxygen and carbon dioxide diffuse in an out of blood (Fahey et al., 2007).

Alveolus: One of the numerous thin-walled polyhedral formations that line the walls of the alveolar sacs that open into the alveolar ducts at the termination of a respiratory bronchiole in the lung; it is the ultimate respiratory unit where gas exchange takes place (International, 1986).

Ambient Air: Air external to a structure, building, or other air-flow barrier to which the public has legal access.

Anaerobic: Denoting an oxygen-free environment (International, 1986); occurring in the absence of oxygen.

Anaerobic Activities: Physical activity where energy is provided for muscular function without oxygen. This results in an increase in muscle lactic acid formation from breakdown of stored carbohydrates (glycogen). Anaerobic metabolism accounts for most of the energy expended during the first few minutes of prolonged dynamic activity, during short-duration high-intensity dynamic activity, during static activity, and is progressively stimulated when intensity of dynamic exercise exceeds ~ 70% of maximal aerobic capacity [(VO_{2MAX}) (Leon, 1989).

Anaerobic Energy System: See "non-oxidative energy system."

Anaerobic Threshold (AT): (1) The level of exercise at which anaerobic production of energy through glycolysis leads to the rapid accumulation of lactic acid in the blood (Lamb, 1984). (2) The AT for an individual represents the maximal workload where production and elimination of lactate are in equilibrium; it is the upper limit of an almost exclusively aerobic metabolism that permits exercise lasting for hours at a lactate level of approximately 2 mmol L min^{-1} (Guidetti et al. 2008). (3) The fraction of $VO_{2.MAX}$ that can be maintained during an endurance event (Luks et al., 2012). It also is known as the ventilatory threshold (VT) [see] or the lactate threshold [see], but recent work distinguished among these terms.

Anaerobic Training: Training that improves the efficiency of the anaerobic energy-producing systems and which often increases muscular strength and tolerance for acid-base imbalances produced during high-intensity efforts (Nieman, 1999).

Asthma: (1) A condition marked by recurrent attacks of paroxysmal dyspnea, with wheezing due to spasmodic contraction of the bronchi. Some cases of asthma are allergic manifestations in sensitized persons (atopic allergy), while others are provoked by a variety of factors, including rigorous exercise (exercise-induced asthma), irritants, and psychological stress (Dorland's, 1988). See "Bronchial Asthma," "Bronchitic Asthma," "Essential Asthma", "Extrinsic Asthma," and "Intrinsic Asthma." (2) A chronic inflammatory disorder of the airways, characterized by recurrent episodes of wheezing, breathlessness, chest tightness, and coughing. These episodes are usually associated with widespread, but variable airflow obstruction. (Strunk, 2002).

Asthmatic Bronchitis: A condition characterized by the clinical features of both asthma and bronchitis (International, 1986).

Atopic: (1) Pertaining to atopy, which is a genetic predisposition toward developing immediate hyper-sensitivity reactions against common environmental antigens or substances. It occurs in about 10% of the general population (Dorland's, 1988). (2) Clinical hyperreactivity of the airways associated with asthma and allergies (EPA,1989).

ATPD: Ambient temperature and pressure, dry.

ATPS: Ambient temperature and pressure, saturated with water vapor. These are conditions existing in a water spirometer, used for lung functional testing. Lung volume measures at ATPS will be approximately 8-10% smaller than when measured at BTPS (Shephard, 1967).

Average Exposure: Instantaneous exposures averaged over a time period (Duan ,et al.,1990).

Averaging Time: The time period over which any function is measured, often yielding a time-weighted average.

a-O_2 Diff.: Difference in oxygen content between arterial and mixed venous blood.

B

Basal Metabolism: (1) State of minimal metabolic activity (energy expenditure) associated with maintenance of body function at its normal temperature and at mental and physical rest (International, 1986). (2) Alternatively, the caloric requirements of the fasting body, at physical and mental rest, and at room temperature (20 ◦ C). It corresponds to the unavoidable loss of heat due to cell metabolism and the energy expended in maintaining minimal bodily functions: circulation, respiration, digestion, and involuntary muscle tone (Diem & Lentner, 1970). Basal metabolism varies with age, gender, and body size, and is highly correlated with fat-free body mass (Andersen et al., 1978).

Basal Metabolic Rate (BMR): The rate at which BMR occurs, in units of L min^{-1} when oxygen consumption is measured, or in kcal min^{-1} (or kJ min^{-1}) when energy is measured directly. Generally BMR is measured as oxygen consumption using an indirect calorimeter. It often is "normalized" on a body mass (BM) basis (kcal kg^{-1} min^{-1}) or—in past decades—on a body surface area basis in units of either kcal min^{-1} m^{-2} or kJ min^{-1} m^{-2}. It is *functionally*-- but not mathematically-- equivalent to the "resting metabolic rate" (RMR) or the "resting energy expenditure" (REE), but there are differences in the standard protocols used to ascertain these metrics. Consequently, the measurements are not identical in terms of energy expended.

Bias: Any systematic departure from "true values" (IPCS, 2000). Also known as "systematic error." Bias can take many forms: instrumental error, measurement error, faulty assumptions, "publication bias," "interpretation bias," "sampling bias," "selection bias" (also known as Berkson's error), etc.

Bimodal distribution: A distribution of values having two modes of high frequency separated by a region of lower values (Last, 1983).

Bioavailability: State of being absorbed and available to interact with metabolic processes of an organism (IPCS, 2000).

Biological marker (biomarker): A measureable posterior indicator of exposure to an exogenous chemical, metabolite, or product of biological interaction between the chemical and some target molecule or cell (IPCS, 2000).

Biologically effective dose: The amount of a contaminant that reaches cells or target site where an interaction with a membrane surface or an adverse effect occurs (IPCS, 2000).

Body Build: A combination of body weight and fat content of a person.

Body Composition: A health-related component of physical fitness that relates to the relative amount of muscle, fat, bone, and other vital tissues in a person (Nieman, 1999).

Body Mass Index (BMI): One of the anthropometric measures of body mass. BMI = weight/$height^{2.}$ The units usually seen are in kg m^{-2}. BMI also is known as Quetelet's Index (Last, 1983), but this term is rarely seen in the current literature. For the general adult population, overweight is defined to be a BMI≥25 and <30.0. For adult Asians, overweight is ≥23.0 an <25.0; for adult Pacific Islanders overweight is defined to be ≥26.0 and <32.0. Obese adults are defined to be those people with a BMI ≥30.0 for the general population; ≥ 25.0 for Asian adults; and ≥32.0 for Pacific Islanders.

Overweight and obesity for youth (children, adolescents, and those <20 y of age) is defined by BMI values for age/gender-specific percentiles; overweight is ≥ 85th percentile and obese is ≥95th percentile of the BMI distributions. (This approach alters the BMI benchmark over time, as the BMI distributions change—mostly increasing in the general population (Kuczmarski & Flegal, 2000).

Body Surface Area (BSA): One of the anthropometric factors used to normalize" or scale ventilatory measures. There are a number of alternative measures of BSA that have been developed over the years; for example, see Dubois & Dubois, (1916): BSA = $Mass^{0.425}$ * $Height^{0.725}$ * 71.84. The units are cm^2, obtained from: kg * cm * cm/kg). BSA is *roughly* proportional to BMR, and the ratio of BSA-to-BMR is approximately constant across species. BSA also is approximately proportional to an animal's body weight (mass) raised to the ⅔ power.

bpm: Beats per minute (a heart rate metric).

brpm: Breaths per minute (a breathing rate metric).

Breath-by-Breath – A method for measurement of respiratory gas exchange in a breath during which expired gas volume and simultaneously measured expired gas concentration are collected, integrated and reported.

Breathing Pattern: A general term designating characteristics of ventilatory activity, such as frequency of breathing (f_R), tidal volume (V_T), and shape of the volume-time curve associated with specific human activities (OAQPS Staff, 1988).

Breathing Rate (f_R): The number of breaths taken per minute (bpm, breaths min^{-1}).

Breathing Zone: Air within the vicinity of an organism from which inspired air is drawn, generally the area around the nose and mouth (EPA, 1992).

Bronchi: The first subdivisions of the trachea, which conduct air to and from the bronchioles of the lungs.

Bronchial: Pertaining to the airways of the lung below the larynx that lead to the alveolar region of the lungs (EPA, 1989).

Bronchial Asthma: Asthma associated with an allergy in persons with a constricted airway (International, 1986).

Bronchiole: One of the finer subdivisions of the bronchial (trachea) tubes, less than 1 millimeter in diameter, and having no cartilage in its wall.

Bronchitic Asthma: An asthmatic disorder accompanying bronchitis (International, 1986).

BTPS: Body temperature and ambient barometric pressure, saturated with water vapor. This is the "reference condition" for most pulmonary functional tests associated with clinical status of exercise and/or exposure to air pollutants. For humans, the normal temperature is 37 ^{o}C, the pressure is based on the barometric pressure, and the partial pressure of water vapor is 47 torr.

C

C x T: Pollutant concentration multiplied by time; an index or metric of exposure that often is incorrectly used as an index or metric of dose. Since most effects are better described by the time profile of intake dose received, C x T has limited usefulness in explaining exposure-response relationships. See "Exposure Profile" and "Dose Profile." C * T is used with "Area Under the Curve" logic (deleted here), and does not explain adverse health effects in a target organ for most chemicals.

Calorie: A unit of heat energy; the amount of heat required to raise temperature of 1 gram of water at 15 ^{o}C by 1 ^{o}C. 1 cal = 4.186 kJ = 3.968 x 10^{-3} BTU, but other conversions are seen in the literature.

Calorimetry: Methods use to estimate the rate of energy expenditure in a person undertaking work or at rest.

> **Direct Calorimetry**: A method that estimates energy expenditure by a direct measure of total body heat production.
>
> **Indirect Calorimetry:** A method of estimating energy expenditure by measuring inspired and exhaled respiratory gases (O_2 and CO_2).

Capillary: (1) The smallest type of vessel; it distributes blood to all parts of the body. Usually used in reference to a blood or lymphatic capillary vessel. (2) The tiny, thin-walled blood vessels interposed between the arteries and veins in which materials are exchanged between blood and tissues or, in the lung, between blood and alveolar air (Morehouse & Miller, 1976).

Carbon Dioxide Output (VCO2): The amount of CO2 exhaled from the body into the atmosphere per unit time, expressed in milliliters (mL) or liters (L) per minute. A normal adult value at rest is 200 ml/min, and increases with exercise (Luks et al., 2012). It is not the same as CO2 production by metabolic processes, but are the same in the steady state and low energy expenditure.

Carbon Monoxide: An odorless, colorless, toxic gas with a strong affinity for hemoglobin and cytochrome; it reduces oxygen absorption capacity, transport, and utilization in the blood stream (EPA, 1989).

Carboxyhemoglobin: A fairly stable union of carbon monoxide with hemoglobin that interferes with the normal transfer of carbon dioxide and oxygen during circulation of blood. Increasing levels of carboxyhemoglobin result in various degrees of asphyxiation, including death (EPA, 1993).

Cardiac Index: Heart rate divided by body surface area (HR/BSA) in units of beats min^{-1} m^{-2} (Rowland, 1989). This index is not often used currently by exercise physiologists.

Cardiac Output (Q): The blood flow or volume of blood passing through the heart per unit time measured in liters min^{-1}. It is estimated as heart rate (HR) * stroke volume (SV) (Lamb, 1984). Because it is a rate, a "dot" should be placed over Q to distinguish it from blood volume in L, also denoted as Q in some papers (Diem & Lentner, 1970).

Cardiac Reserve: Ability of the heart to increase its blood output by increasing heart rate (HR) or stroke volume (SV), or both (Morehouse & Miller,1976).

Cardiorespiratory Endurance Capacity: A synonym for maximal oxygen consumption/uptake [see].

Cardiorespiratory Fitness (CRF): (1) One aspect of "physical fitness" that, in practice, is defined only by the recommended levels of activity (exercise) needed to maintain it (it is a tautology, in other words). Attributes included in CRF recommendations are: (a) frequency, duration, and intensity of training, and (b) the mode of activity that should be pursued (ACSM, 1990). CRF is also known as "aerobic fitness" [see]. (2) A health-related component of physical fitness that relates to ability of the circulatory and respiratory systems to supply oxygen during sustained physical activity (Nieman, 1990).

Cardiovascular: Pertaining to the heart and blood vessels (Lamb 1984).

Cell: The smallest membrane-bound protoplasmic body, consisting of a nucleus and its surrounding cytoplasm, capable of independent reproduction (OTA 1986).

Cellular permeability: Ability of gases to enter and leave cells; a sensitive indicator of injury to deep-lung cells.

Central Nervous System (CNS): The brain and the spinal cord.

CHAMPS: Children's Activity and Movement in Preschool Study.

Chronic: (1) Of or characterized by an extended duration, and typically by slow development or a pattern of recurrence (International, 1986). (2) Referring to a health-related state lasting a long time (Last 1983). (3) Referring to prolonged or long-term exposure, often with reference to low-intensity concentration levels (Last, 1983).

Chronic Bronchitis: (1) Chronic inflammation of bronchi resulting in cough, sputum production, and progressive dyspnea (International, 1986). (2) A long-continued form of bronchitis, often with a tendency to reoccur after a quiescent period. It is due to repeated attacks of acute bronchitis or to a chronic general disease (Dorland's, 1988).

Chronic Disease: A disorder or disease of long duration or frequent recurrence that often is characterized by slowly progressing seriousness.

Chronic Exposure: (1) A vague term that loosely relates to a low-level, long-term exposure profile. Used by some analysts to represent exposures lasting greater than six months to a lifetime (IPCS, 2000). (2) Multiple exposures—over some specified level—occurring over a long period of time (IPCS, 2000). A persistent, recurring, or long-term exposure. Chronic exposure [dose really] to a substance is thought to result in a health effect--such as cancer--that is delayed in its onset, often occurring long after exposure has ceased (CMA,n.d.). However, see "Dose Profile."

Chronic Intake: A vague term relating to a long time period over which a substance crosses the outer exchange boundary of an organism (IPCS, 2000). See "Dose Profile."

Chronic Obstructive Pulmonary Disease (COPD): A general term for a pulmonary condition of uncertain etiology characterized by persistent slowing of airflow during forced expiration; also known as COLD, where lung is substituted for pulmonary. A more specific term should be used, such as chronic obstructive bronchitis or chronic obstructive emphysema (OAQPS Staff, 1988).

Cilia: Motile, often hair-like extensions of a cell surface.

Ciliary Action: Movements of cilia in the upper respiratory tract, which move mucus and foreign material upward (EPA, 1993).

Ciliated Epithelial Cell: A cell with cilia that lines the tracheobronchial region of the lung. The beating of the cilia moves mucus and substances (such as inhaled particles trapped on/in the mucus) upwards and out of the lung, thereby contributing significantly to lung clearance.

Circadian Rhythm: Fluctuation in biological variables that are repetitive and cyclical over a solar or 24 h day. The most prominent rhythms from a performance and/or energy expenditure viewpoint are those of body temperature and wakefulness. Isometric strength of back and leg muscles, anaerobic power, and exercise performance also seem to follow a circadian rhythm. $VO_{2.MAX}$, however, in general, does not (Reilly & Garrett, 1998).

Citric Acid (Krebs) Cycle: A major biochemical pathway in cells, involving terminal oxidation of fatty acids and carbohydrates. It yields a major portion of energy needed for essential body functions and is the major source of carbon dioxide. It also serves to regulate the synthesis of a number of compounds required by a cell.

Clara Cell: A nonciliated cell in the epithelium of the respiratory tract.

Clearance: (1) Removal of a solute or substance from a specific volume of blood per unit of time (Dorland's, 1988). (2) Removal of insoluble particles or other substances that are deposited on epithelial surfaces of the lung (Lippmann, 1989).

Clinical: Of or pertaining to direct observation or experimentation on human subjects. In our context, it means direct and controlled (1) physiological or metabolic experiments, or (2) exposure-effect observations on humans in a laboratory or experimental chamber.

Coefficient of Variation (CV, COV): Ratio of the standard deviation of a sample to its mean, when the sample is measured on a ratio scale, randomly sampled, and is normally distributed.

Congenial: A condition that is present at birth (OTA, 1986).

Cohort: (1) A group of individuals sharing a statistical characteristic for a epidemiologic or other study of disease (Dorland's, 1988). (2) A taxonomic category approximately equivalent to a division order, or suborder in a population classification (Dorland's,1988). (3) A group of people within a population who are assumed to have similar exposures.

Cohort Study: A study of a group of persons sharing a common experience (e.g., exposure to a substance) within a defined time period; this experiment is used to determine if an increased risk of a health effect (disease) is associated with that exposure (EPA,1989).

Chronic obstructive lung disease (COLD): see "Chronic Obstructive Pulmonary Disease."

Community Exposure: A general term to depict the situation in which people in a sizeable area are subjected to ambient pollutant concentrations; this term is ambiguous.

Compartments: (1) Representation in a model of a particular tissue or organ group with anatomical significance. (2) All tissues, organs, cells, and/or fluids for which the rate of uptake and loss of a substance (chemical) is sufficiently similar as to preclude further kinetic resolution (Dietz et al., 1983).

Compound: A substance with its own distinct properties, formed by the chemical combination of two or more elements in fixed proportion (EPA, 1993).

Concentration: The amount of a substance of interest that is contained or dissolved in a specified amount/ quantity of another substance. The amount of material in air (IPCS, 2000).

Concentration-Effect Relationship (Curve): A mathematical or graphical association or causal relationship between an ambient concentration of a contaminant or substance and a specified biological effect in an individual (Duffus, 2000).

Concentration-Response Relationship (Curve): A mathematical or graphical association or causal relationship between an ambient concentration of a contaminant or substance and a specified biological effect in a population (Duffus, 2000).

Concentration Ratio: The ratio of concentration of a substance in a tissue or organ compared with that which is found in surrounding tissue/organ(s) under equilibrium or steady-state conditions (IPCS, 2000).

Confidence Interval (CI): A range of values from a sample that bracket a *point estimate* assuming a random sample, normal distribution, and a ratio measurement (IPCS, 2000). For a *95%* CI, it means that there is a 95% probability that the true value is contained in the range (with an $\alpha = 0.05$).

Confidence Limit: The lower and upper specific values for the confidence interval.

Congenital Abnormality: Any abnormality, genetic or non-genetic, that is present at birth (OTA, 1986).

Concentration: (1) Quantity of a substance per unit volume or weight (Stedman's, 1982). (2) Ratio of the mass or volume of a solute to the mass or volume of a solution or solvent (Dorland's, 1988). Usually designated as "c".

Confounder (Confounding variable): A variable that can cause or prevent an outcome of interest which is not an intermediate variable, and is not associated with the factor under investigation (Last, 1983).

Consistent, Consistency: The property of a measurement or estimate that conforms to themselves over time or repeated trials; see "reliability."

Consolidated Human Activity Database (CHAD): A data base of daily human activity patterns (time-sequenced activity information) for U.S. residents that was developed for, and is maintained on, EPA's web site (www.epa.gov/chadnet1/). As of 2012, CHAD contains 34,773 person-days of data, about half of which is a single diary-day of information for an individual. Additional data are included on an *ad hoc* basis as they become available.

Contact Rate: The rate per unit time that a boundary of an organism comes into contact with a medium.

Continuous Exposure: An exposure profile in which no concentration level falls to zero or to some value below a level of interest (for a specified time period).

Control Group: A group of subjects observed in the absence of a condition or exposure agent for comparison with subjects having the condition or exposure (EPA, 1989).

Coronary Blood Vessels: Blood vessels that supply blood to the heart muscle (Morehouse & Miller, 1976).

Correlation: A *change* association between two or more entities (variables) characterized by a linear relationship.

Correlation Coefficient (r): A measure of association indicating the degree to which two of more samples fit a linear association, assuming random probability sampling, a normal distribution, and an α=0.05. Also called a Person product-moment correlation coefficient. See also "Spearman Rank Correlation Coefficient."

Critical Pathways: Environmental or other pathways by which a significant amount of a substance moves from a source to a receptor of concern.

Critical Receptor: A specified, or identified, receptor of a substance that is most adversely affected by receiving a dose of the substance.

Critical Tissue: Tissue that shows adverse effects at the lowest dose, with no reference to severity of the effects (International, 1986).

Cross-sectional Study: A study or analysis having samples for only one point in time.

Cumulative Frequency Distribution: A statistical distribution where sampled values are ranked in a specific order, generally lowest-to-highest.

D

Daily Energy Expenditure: A measure of the amount of energy expended by a person (or living organism) on a daily basis to support basic metabolism and dietary processes, and to undertake all other physical activities, including work. An alternative term for "Total Daily Energy Expenditure."

Dead Space Fraction (V_D/V_T): A unitless measure of the physiological dead space of the lungs and represents the fraction of inspired air that does not exchange gas with capillary blood. In normal individuals, the value is generally between 0.25-0.35 and decreases with progressive exercise. (If it does not, it is a marker of either pulmonary vascular or interstitial lung disease.) Luks, et al. (2012).

Dead Space Volume (V_D): The combined volume of all air passages in which no gas exchange occurs; these include the trachea, bronchi, and bronchioles down to--but not including--respiratory bronchioles (Morehouse & Miller, 1976). About a third of every resting breath, or about 150 mL, is exhaled exactly as it came into the body. Because of dead space, taking deep breaths more slowly (e.g., ten 500 mL breaths per minute) is more effective than taking shallow breaths quickly (e.g., twenty 250 mL breaths per minute). Although the amount of gas per minute is the same (5 L/min), a large proportion of the shallow breaths is dead space, and does not allow oxygen to get into the blood.

There are several components that go into dead space. These include anatomical dead space (gas in the conducting areas of the respiratory system, such as the mouth and trachea, where the air doesn't come to the alveoli of the lungs), physiological dead space (the anatomical dead space plus the alveolar dead space), and alveolar dead space (the area in the alveoli that does not exchange air because there is not enough blood flowing through the capillaries for exchange to be effective). Alveolar dead space is normally very small (less than 5 mL) in healthy individuals, but can increase dramatically in heart or lung disease.

Demographic Group: A group of people within a population that share one or more defined demographic characteristics, such as gender, age, ethnicity, household income, working status, health impairment, or housing type. These groups usually are defined differently depending upon the health end point of interest, the pollutant, and the time or spatial area of interest. Often it is called simply as a "cohort."

Dermal adsorption: The process by which materials come in contact with the skin surface and are then retained and adhered to the epithelial epidermis without being taken into the body. (EPA, 1992).

Dermal Exposure: The contact of an organism's external membrane, generally the skin, with a chemical substance or physical agent via any medium.

Dermally absorbed dose: The amount of substance from a dermal exposure that is absorbed into the body.

Deterministic: In statistics and modeling, a variable taking on a single, unchanging value.

Deterministic Model: A mathematical model in which its parameters and variables are not subject to random processes. The underlying system defined by the model is entirely defined by its initial conditions (IPCS, 2000).

Diary Study: A "field" study in which subjects are asked to record general of specific activities, such as foods consumed, time uses undertaken, locations frequented, etc. as they are done. Compare with "Recall Survey."

Dietary Induced Thermogenesis (DIT): The amount of energy needed to process the digestion of food above that needed for basal metabolism (Nieman, 1990). Often it is simply defined to be some proportion of DTEE, usually 10%, but that is a questionable assumption due to individual differences and the type of food ingested (Nieman, 1990). It also is known as: the "Thermic effect of food" (TEF).

Diffusion: (1) Movement of a chemical substance from areas of high concentration to areas of low concentration. Biologically, diffusion is an important means for toxicant deposition for gases and very small particles in the pulmonary region of the lungs (EPA, 1989). (2) The process by which molecules or other particles intermingle as a result of their random thermal motion (EPA, 1993).

Direct Exposure: Exposure to a receptor or subject who comes into contact with a chemical or substance in the same medium in which it was released into the environment (IPCS, 2000).

Disease: Any deviation from, or interruption of, the normal structure or function of any part, organ, or system of the body that is manifested by a set of symptoms or signs and woes etiology, pathology, and prognosis may be known or unknown (Dorland's, 1988).

Distribution: In biology and toxicology: the transport of a substance through the body by a physical means, such as active transport or diffusion. It is dependent upon the chemical properties of the toxicant or its metabolites and--to some extent--on the route of exposure and physiological state of the body (EPA, 1988).

In statistics: A set of sampled values or measurements derived from a specific population that represents the range and array of data for the measured quantity (EPA, 1995).

Distribution-free: A method of statistical testing a hypothesis, or establishing a confidence interval, that does not depend upon form of the underlying distribution. Generally applied to variables not following a normal distribution (Last, 1983).

Diurnal: Having a repeating pattern or cycle 24 hours long.

Doer: A person who participates in a specific type of time use or activity.

Dosage: "Dose Rate" [see].

Dose: (1) Inspired air concentration per unit time. (2) Presence of a pollutant [substance] inside a target (Duan, et al., 1990). (3) Quantity of a substance (contaminant) absorbed across an exchange boundary of a receptor organ and available for metabolic interactions (EPA, 1992). See also: "Applied Dose," "Biologically Effective Dose," "Intake Dose," and "Internal Dose." (4). The quantity of energy or xenobiotic substance available for interaction with metabolic processes or biological receptors after it crosses the outer boundary of an organism (RPA, 2003). (5). The amount of agent that enters a target during a specified time interval by crossing a contact boundary (Zartarian et al., 1997). (6) In pharmacology, the quantity of a drug or other material to be administered at one time (IPCS, 2000).

Dose-Effect Relationship (D/E): A correlative relationship between a dose of a substance or agent and the biological response (effect) in an individual (not a population). This often is confused with "Dose-Response Relationship," which should only be applied to a population. A linear dose-effect relationship between dose and biological response follows a straight line. In other words, the rate of change (slope) in the effect is the same at any dose. A linear dose response is written mathematically as follows: if E represents the expected, or average, effect and D represents dose, then $E = a * D$, where a is the slope, also called the linear coefficient.

Dose Membrane: A barrier that resists the flow of an agent after it crosses a contact boundary (Zartarian, et al., 1997).

Dose Metric: A specific description of the dose received by a receptor or target organ during a specified time period. A fully-specified dose metric includes an estimate of the magnitude or intensity of the substance, an averaging period, and a "profile" of the dose received over time. The time pattern of dose rate received is a fully-specified dose metric.

Dose Profile: The time pattern of intake dose received by a target organ or system. For inhaled substances, it is the sequential pattern of concentration for a specified time period times the intake, or inhalation, rate (e.g., $c * t * V_E$).

Dose Rate: (1) Dose per unit time (and, sometimes, per body mass), sometimes called "dosage." Often dose rate is expressed on a body-weight basis, such as mg kg^{-1} day^{-1}; dose rate also is expressed as an average over a time period (EPA, 1992). (2) The quantity of material absorbed across a unit area of an exchange boundary per unit time.

Dose-Response Curve: A curve on a graph based on responses occurring in a population as a result of a series of stimuli intensities or doses. A visual representation of a D/R relationship.

Dose-Response Relationship (D/R): (1) A correlative relationship between a dose of a substance or agent and the proportion of a population that experiences a specified effect. It is developed by integrating across individual dose-effect relationships for a specified effect level. (2)

The relationship between magnitude of applied or internal dose and a specified biological response. [In an individual, this is called "dose-effect"; see the above.] Response can be expressed as measured or observed incidence ["effect" in an individual], percent response in populations, or the probability of occurrence of a response in a population (EPA, 1992). A linear dose-response relationship between dose and biological response for a population follows a straight line. In other words, the rate of change (slope) in the population response is the same at any dose level. A general linear dose response is written mathematically as follows: if R represents the expected, or average, population response and D represents dose, then R = a * D, where a is the slope, also called the linear coefficient.

Dosimetry: (1) Accurate determination of dose (Stedman's, 1982). (2) Determination of absorbed dose in a substance by measuring chemical reactions (International 1986). (3) Estimating the amount of substance delivered to or absorbed by a specific target site (Miller et al., 1989). (4) The modeling of the amount, rate, and distribution of a substance in the body, especially as it pertains to producing a particular (specified) biological effect (EPA, 1989).

Doubly Labeled Water (DLW): The "gold standard" for measuring Total Daily Energy Expenditure [see]. It is based upon the following relationships: CO_2 production is estimated from the difference in turnover rates of two tracer isotopes--^{2}H and ^{18}O--in the body's water pool. ^{2}H is lost from the body entirely as water (urine, sweating, insensible water loss, etc.) while ^{18}O is lost both as water and CO_2. The difference between the elimination rates of the two isotopes is therefore an estimate of CO_2 production rate. That, plus an assumed (or measured) respiratory quotient (RQ) allows one to predict total daily oxygen consumption. Daily total energy expenditure is estimated from CO_2 by using the Weir Equation [see].

Duration: A measure of the length of time associated with a specified event of interest; e.g.: an exposure, dose intake, exercise period.

E

Effective Dose: A dose metric that simply is the product of concentration, exposure duration, and ventilation rate (Adams, Savin, and Christo, 1981). $ED = c * t * V_E$. Its units often are ppm-liters per elapsed time period (min, hour, day) or could simply be a concentration metric (µg) for the applicable time period.

Energy: The capacity to perform work, produce force, or generate heat (McArdle et al., 1991).

Energy Cost of Breathing: The oxygen consumption requirements of breathing itself needed to generate sufficient pressure (force) in the respiratory system to move blood to the locomotor muscles (McArdle et al., 2001). This is also known as the "Oxygen Cost of Breathing." At maximal exercise, it may be as high as 15% of total oxygen consumption needed for the workload. This cost is lower relative to $VO_{2.MAX}$ in fit individuals than in "normals."

Energy Expenditure: (1) The amount of energy needed to maintain life and perform work (undertake physical activity). Energy is expended in humans in three general ways: (a) to maintain body temperature and those involuntary muscular contractions needed for circulation and respiration--this is resting [but not basal] metabolism; (b) to digest and assimilate food--also known as dietary induced thermogenesis; and (c) to support muscular activity (Montoye et al.,1996). There are a number of ways to measure energy expenditure in humans. The unit of energy used in nutritional studies generally is the kilocalorie (kcal). It is equivalent to 4.185 kJ "and corresponds to the consumption of about 239 mL of oxygen" (Andersen et al., 1978).

Energy Metabolism: Metabolic activity associated with energy production or utilization (International, 1986).

Epidemiology: Study of the distribution and associations of health-related states or events in specified populations (Last, 1983).

EPOC: Excess post-exposure O_2 consumption; it also is known as recovery O_2. See also "Oxygen Debt."

Ergometer: An apparatus for measuring the amount of work performed by a subject, generally as oxygen consumed; the stationary bike ergometer is one example (Morehouse & Miller, 1976).

Essential Asthma: Asthma of unknown or not apparent cause; also known as "true asthma." (Dorland's, 1988).

Event: (1) In exposure modeling, a time period ≤1 clock h characterized by a specific activity, "activity-level" (energy expenditure or breathing level), exposure level (intensity), and location. If any of these characteristics change, the event changes, even if a person stays in the same location and undertakes the same general activity. (2) An observed state of activity (action) having a specific time duration of measureable discrete units (t) within a larger temporal period T. An event has a measurable intensity on some property of interest that occurs. (3) Frequency of events is the number of times a specified event occurs with a specified T. Pattern of events occur if a series of similar event types are seen in T. If t between events is regular, the events have periodicity; otherwise the events have an irregular pattern. Periodic event that occur over multiple T's have rhythm. Adapted from McGrath & Tschan (2004).

Excess Metabolism of Exercise: (1) Increase in metabolic activity during exercise and recovery from it, over that used during sleep (International, 1986). (2) Amount by which the oxygen consumed (or CO_2 eliminated) during exercise and recovery exceeds the corresponding rates during sleep (Dorland's, 1986).

Excess Post-Oxygen Consumption (EPOC): The oxygen consumption to "make up" oxygen—the "oxygen deficit"--needed for anaerobic process associated with moderate-to-high physical activity. See also "oxygen debt."

Exercise: (1) A subset of physical activity that is planned, structured, and repetitive and has as an objective to improve or maintain a person's physical fitness (Kohl et al., 2000). (2) Any and all activity involving generation of force by activated muscles which results in a disruption of a homeostatic state (ACSM, 1990).

Exercise Conditioning: Repeated exercise with sufficient intensity and duration to increase a participant's strength and endurance (Morehouse & Miller, 1976).

Exercise Intensity: A specific level of maintenance of muscular activity that can be quantified in terms of power, isometric force sustained, or velocity (American College of Sports Medicine as cited in McArdle, et al., 2001).

Exercise Training: Repeated exercise that improves economy of movement that increases performance (Morehouse & Miller, 1976).

Exergonic: Characterized or accompanied by the release of energy; said of chemical reactions that release free energy so that their products have lower free energy than the reactants (Dorland's, 1988).

Exogenous: Produced or otherwise originating outside of an organism (International, 1986).

Expected Value (E[x]): The mean value of a cumulative normal distribution random sample.

Expiration: Act of breathing out, or expelling air from the lungs (Dorland's, 1988). The time it takes for one expiration is known as T_E.

Expiratory Reserve Volume (ERV): The maximal volume that can be exhaled from the lung's resting end-tidal expiratory position. See also "functional residual capacity."

Expired Ventilation (VE): The amount of air in the lungs that is expired per breathing event.

Expired Ventilation Rate (V_E): The rate at which expired ventilation occurs; it also is known as pulmonary ventilation in L min^{-1} BTPS. V_E approximates V_I, and by definition $V_E = f_R * V_T$.

Exposure: (1) Contact between a target and a pollutant [substance/agent] at an exposure boundary (Duan et al., 1990). (2) Proximity and/or contact with a disease agent [substance] in such a manner that transmission of the agent to the organism of interest may occur (Last, 1983). (3) Exposure is quantified as the amount of agent/substance available at the exchange boundary of the receptor organism per specified time period (EPA, 1989).

Exposure Assessment: Measurement or estimation of the magnitude, frequency, duration, pattern and route of exposure of a target--an individual or a population--to substances in the environment for a specified time period. An exposure assessment also describes the nature of exposure and the size and nature of the exposed populations (EPA, 1989).

Exposure Concentration: Concentration of a chemical or pollutant in a transport or carrier medium at the point of contact with a receptor of interest (EPA, 1992).

Exposure Duration: Length of time that contact with a chemical or pollutant occurs; total time that an individual is exposed to a chemical being evaluated (EPA, 1997).

Exposure-Effect Relationship: Exposure-Response Relationship: The association between a fully-specified exposure metric and the distribution of adverse effects in a person (receptor of interest).

Exposure Event: (1) The joint set of occurrences in which the contact boundary of a receptor of interest intersects a medium having agent concentrations of interest during a time interval of interest (Zartarian et al., 1997). (2) In the APEX, pNEM, and NEM series of exposure models, it is a varying period of time between 1 minute and the next clock hour where a subject is located in a single microenvironment that is characterized by a constant concentration level and an activity-specific activity level (energy expenditure level). If any of these parameters change, a new exposure event occurs.

Exposure Factor: A "point estimate" or a distribution of values for any unknown quantity of interest used to undertake an exposure assessment. Generally these factors appear in a "sanctioned" handbook, such as EPA's Exposure Factors Handbook or AIHC's Exposure Factors Sourcebook.

Exposure Frequency: The number of times and exposure of interest occurs in a specified time period.

Exposure Level: Concentration of a contaminant to which an individual or a population is exposed.

Exposure Limit: Suggested or mandatory limit, standard, or restriction implemented by some authority to ensure that possible receptors are not exposed to concentrations of a substance, usually in a specified location, that can cause some unwanted effect (CMA, n.d.). It is thought that the limit will result in minimal or no adverse (health or other) effects. A fully specified limit will describe the level (magnitude or intensity), duration, frequency, and pattern of exposure that should be avoided.

Exposure Medium: See "Media" / "Medium."

Exposure Metric: A specific description of the exposure experienced by a receptor or organism during a specified time period. A fully specified exposure metric includes an estimate of the magnitude or intensity of the substance, an averaging period, and a "profile" of the exposure experienced over a specified time period. The "Exposure Profile" is an example of a fully-specified exposure metric.

Exposure Monitoring: The actual measuring or monitoring of substances in microenvironments and/or at or near individuals as they undertake personal activities in various microenvironments. See "Microenvironmental Monitoring" and "Personal Monitoring."

Exposure Pattern: See "Exposure Profile."

Exposure Pathway: The physical course that a substance takes between its source and an exposed receptor [organism] (CMA, n.d.).

Exposure Profile: The record of instantaneous exposures over a time period; a function of time (Duan et al., 1990). Some authors label it the "time course of exposure." A fully described exposure profile provides information concerning the intensity (magnitude), duration, and time pattern of exposure experienced by a receptor.

Exposure-Response Relationship: The association between a fully-specified exposure metric and the probability of an adverse effect in a population, which is estimated by integrating across individual exposure-effect relationships.

Exposure Route: The manner in which a substance or chemical enters into, or is absorbed by, an exposed receptor or organism upon first contact; the main routes of exposure include inhalation, ingestion, and dermal absorption (CMA, n.d.). A substance may enter by all three routes within some specified time period of analysis.

Exposure Scenario: A set of facts, assumptions, and/or inferences about how a particular exposure event occurs that assists an exposure assessor in evaluating, estimating, modeling, and/or otherwise quantifying exposure(s) to a specified receptor or population (CMA, n.d.).

Exposure Surface: A target surface where an agent is present. Examples include a/an: stomach wall lining, lung surface, exterior of an eyeball, skin surface, leaf, wall, and a conceptual surface over an open mouth (IPCS, 2000).

External Dose: A vague term that approximates "exposure." It is confusing and should not be used.

Extrinsic Asthma: Asthma caused by an environmental factor (Dorland's, 1988).

F

Factorial Method: An approach used by clinical nutritionists and exercise physiologists to estimate the energy expended by a specific activity (EE_A) or a set of activities. Basically it is simply the multiplication of time spent in an activity by an estimate of the oxygen consumption or energy expenditure associated with that activity—usually generalized from clinical measurement studies. An example is time in activity A times $METS_A$. The basis of EPA exposure models using the METS distributions in CHAD is the factorial approach of method.

FAO: Food and Agricultural Organization (part of the United Nations).

Fat-free Mass (FM): Body mass devoid of all extractable fat (FFM=BM-FM) (McArdle et al., 2001). It differs from lean body mass (LBM) in that the latter does not include essential fat, which is about 3% of total body mass.

Fatigue: Diminished capacity for work caused by previous work; usually used for subjective sensations (Morehouse & Miller, 1976).

Fick Equation: An equation developed by German physiologist Adolph Fick in 1870 that describes the relationship among cardiac output (stroke volume), the difference between arterial and venous blood, and oxygen consumption (McArdle et al., 2001). O_2 = HR × SV × (a-O_2 Diff.).

Fit(ness): See "Physical Fitness".

Flow Volume Curve: Graph of instantaneous forced expiratory flow recorded at the mouth, against corresponding lung volume. When recorded over the full vital capacity, the curve includes maximum expiratory flow rates at all lung volumes in the vital capacity range and is called a maximum expiratory flow-volume curve (MEFV). A partial expiratory flow-volume curve (PEFV) is one which describes maximum expiratory flow rate over a portion of the vital capacity only (EPA, 1989).

Frailty: A medical syndrome with multiple causes and contributers that is characterized by diminished strength, endurance, and reduced physiologic function that increases an individual's vulnerability for developing increased depency and/or death. (Gordon et al., 2013; p. 8.)

Frequency of Exposure: The number of times some specified exposure event occurs within a specified time period (CMA). The term usually is used when the specified exposure event [of some specified magnitude or duration] occurs on an intermittent basis. A relevant example is EPA's O_3 NAAQS standard; it is designed to reduce the number of daily 8h exposures to 0.08 ppm or higher O_3 concentrations--i.e., the frequency of exposure to the O_3 level specified--during the "ozone season" [generally April-September].

G

Gas Exchange: As regards the lung, it is the exchange of gases between alveoli and capillaries; often used to denote movement of O_2 into pulmonary capillary blood as CO_2 enters the alveoli from blood (OAQPS Staff, 1988).

Gas Exchange Ratio (R): See "Respiratory Quotient."

Gas Exchange Ventilatory Threshold (GET): An indirect and non-invasive index of the transition from aerobic to anaerobic metabolism (Lind et al., 2005).

Gastrointestinal: Pertaining to the intestines and stomach.

Gender: A person's self-representation as male or female; it is shaped by environmental factors and by experience. The term refers to socially-influenced behaviors (Arbuckle, 2005), but is used in this report to represent "sex" to distinguish it from sex as a type of activity.

Geometric Mean: An estimate of the average of a log-normal distribution. Specifically, it is the nth root of the product of n observations.

Geometric Standard Deviation: A measure of variability of a log-normal distribution. It is the antilogarithm of the standard deviation of the logarithms of the observations.

H

Habitué: A person who enters into, or is within, a particular or specific location or microenvironment.

Haldane Transformation: A relationship between inspired and expired air developed in the 1920's and used to "correct" $VO_{2.E}$ expired air flow rates—which is what is measured—to estimate $VO_{2.I}$ inspired air flow rates, which is what is desired, but not readily measureable. Subsequent analyses by others have shown that the average difference between measured and estimated $VO_{2.E}$ using the Transformation is about 0.8%, with a higher bias at higher work rates (McArdle et al., 2001).

Health: Physical, mental, and social well-being; not just the absence of disease (McArdle, et al., 2001).

Heart Rate (HR, f_C): The number of heart beats (complete pulsations) per time specified period, e.g., beats per minute. The f_C term is used most often by exercise physiologists (Norgan, 1996).

> **HR_{MAX}**: Maximum HR that can be sustained for a specified activity and time period, following a defined exercise protocol. Some definitions link it to the heart rate at $VO_{2.MAX}$ or $VO_{.PEAK}$.
>
> **HR_R:** Heart rate at rest. Also denoted as HR_{REST} (in bpm).
>
> **HR_{RES}**: Heart rate reserve (in bpm); it is equal to: (HR_{MAX} - HR_{REST}).
>
> **HR_{REST}**: Heart rate at rest (in bpm).

Heart Rate Recovery ($HR_{RECOVERY}$): – HRR is defined as the decay in heart rate over the first one minute of exercise recovery and it relates to the degree of sympathetic and parasympathetic neuronal control. The average normal Heart Rate Recovery is 28 beats per minute, and a HRR of less than 12 beats per minute is indicative of patient risk. This parameter is useful in assessing patients with congestive heart failure, coronary artery disease and angina. It is effectively used in evaluating the physiological response to cardiac rehabilitation and pharmaceutical or medical device intervention.

Heart Rate Reserve (HRR): The difference between maximum (or peak) heart rate and resting heart rate. Often a theoretical HR_{MAX} is used (HR_{MAX} = 220-Age is the most common estimated, but others are seen in the literature) instead of a measured rate (Luks et al., 2012).

Heart Rate Reserve Percent (%HRR): The percent or ratio of the actual heart rate at a level of work to the maximum heart rate. (How close an individual is to achieving their max heart rate). The percent heart rate reserve is calculated as follows: [(HRstage – HRrest)/(HRpeak – HRrest)] X 100, where HRstage is the observed heart rate at any point in exercise, HRpeak is the actual observed HR at the peak level of exercise performed (i.e., not a theoretical value), and HRrest is the observed resting heart rate. In other words, percent heart rate reserve is the difference between the heart rate at any point in exercise and the heart rate at rest divided by the difference between the maximally observed heart rate and the heart rate at rest with the result multiplied by 100 to equal percent.

The exception to this formula is in maximal exercise stress testing, where HRpeak is a theoretical value based on the formula 220 minus the patient's age in years (220 – age in years).

Hemoglobin (Hb): The red, respiratory protein of the red blood cells, hemoglobin transports oxygen from the lungs to the tissues as oxyhemoglobin (HbO_2) and returns carbon dioxide to the lungs as hemoglobin carbamate, completing the respiratory cycle (EPA, 1989). Hemoglobin's affinity for CO is 200 times greater than that of O_2 (Haymes & Wells, 1986).

Homeostasis: (1) State of equilibrium in the body or organ with respect to various functions and chemical composition of fluids and tissues (Stedman's, 1982). (2) The process by which body or organ equilibrium is maintained (Stedman's, 1982), which generally is achieved by negative feedback mechanisms (Dorland's,1988).

Hormesis: A toxic substance that causes a "stimulation" of a bodily reaction or process at low doses, but inhibits responses at subsequent higher doses (Calabrese & Baldwin, 1998). Hormesis is an inappropriate concept when discussing a population dose-response relationship, by definition.

Hormetic Agent: An agent or condition that causes toxicity at high doses but shows net "beneficial effects" at very low doses (Hart and Frame, 1996).

Human Population Biology: The study of human variety at every level of organization within, between, and among populations. Emphasis is placed on understanding the development, causes, and evolution of that variety and the biosocial effects of it (Harrison, 1996). It is associated with population genetics, environmental physiology, bio-demography, and sociobiology.

Human physiology: Study of phenomena associated with the functioning of humans (International, 1986).

Hyperventilation: Over-ventilation; increased rate of air exchange relative to metabolic carbon dioxide production so that alveolar carbon dioxide pressure tends to fall below normal (EPA, 1989). Pulmonary ventilation that is increased out of proportion to metabolic requirements (Morehouse & Miller, 1976).

Hypoxia: Any state in which oxygen in the lungs, blood, and/or tissues is abnormally low relative to that of a normal man resting at sea level (OAQPS Staff, 1988).

I

ICC: Intraclass correlation coefficient [see].

Index (Indices) of Exposure: See: "Average Exposure," "Exposure Profile," "Peak Exposure," "Integrated Exposure."

Indirect Exposure: Exposure not resulting from direct contact with a substance in the environmental medium into which it is first emitted. Eating food that is contaminated by a chemical substance originally emitted into the air or water in an example of an indirect exposure.

Indirect Exposure Assessment: An approach used to model or estimate human exposures by combining data on microenvironmental concentrations and human activity information for the same time period; the joint set of these two data bases results in an exposure estimate for a particular time period.

Individual (Intra-individual) Variation: Variation of biological variables within an individual (Last, 1983).

Indoor Air: Air inside of a structure, building, or other space that often can be regulated or "conditioned" by a mechanical means.

Inhalation: (1) Drawing of air and other substances into the lungs via the nasal or oral respiratory route (Dorland's, 1988). (2) Inspiration (Stedman's, 1982).

Inhalation Exposure: An exposure to a substance of interest associated with inhalation or respiration (the intake of air into the oral/nasal boundary).

Inhaled Dose: The amount of an inhaled substance that is available for interaction with metabolic processes or biologically significant receptors after crossing over the outer boundary of an organism (EPA, 1997).

Intake: Process by which a substance penetrates the outer boundary of an organism without passing an absorption barrier; examples are: ingestion and inhalation (EPA, 1992).

Intake Dose: (1) Amount of a substance or material that is inhaled, ingested, or absorbed by an organism (EPA, 1992). (2) The dose resulting from an agent crossing a contact boundary without subsequently diffusing through a resisting boundary layer (Zartarian et al., 1997).

Intake Dose Rate: Intake dose per unit time period.

Intake Rate: The rate at which a carrier medium crosses a contact boundary (Zartarian et al., 1997). For ingestion, the intake rate is simply the amount of food containing the contaminant of interest that an individual ingests during some specific time period (units of mass/time). For inhalation, the intake rate is the rate at which contaminated air is inhaled. Factors that affect dermal exposure are the amount of material that comes into contact with the skin, and the rate at which the contaminant is absorbed (EPA, 1997).

Integrated Dose: The amount of a substance entering the target during a specified time period; it is the integral of instantaneous dose over time.

Integrated Exposure: The integral of instantaneous exposures time (Duan et al., 1990).

Intensity: As often used in exposure assessment, it is a synonym for concentration or energy level ("magnitude") for a specified time period.

Interindividual Variation: Variation of biological parameters among individuals in a population (Last, 1983).

Intermediate Variable: A variable that occurs in a causal pathway from an independent to a dependent variable, and that is statistically associated with both variables (Last, 1983).

Intermittent Exposures: An exposure profile that includes "gaps" or respites in which concentration of the substance of interest goes to zero or to some value below a level of interest (for a specified time period).

Internal Dose: (1) The amount of a substance penetrating across an absorption or exchange boundary of an organism (EPA, 1992). It approximates "Intake Dose." (2) In exposure assessments, the amount of a substance penetrating the absorption barriers (e.g., skin,, lung tissue, gastrointestinal tract) of an organism through either physical or biological processes: "absorbed dose" [see] (EPA,1997).

Intraclass Correlation Coefficient (ICC): The ratio of between-group variability to the total amount of variability (between + within) variability "explained" by a statistical procedure. ICC = $\sigma_B / (\sigma_B + \sigma_W)$. The same concept holds for between-individual variability and within-individual variability. The statistical procedures used include one- and two-way (or repeated-measures) AVOVA, and even a three-way ANOVA (Safrit & Wood, 1989). An ICC varies between 0 and 1, with a low value indicating a lot of within-individual (group) variance relative to between-group variance.

The statistic is often used in estimating the reliability of repeated observations for an instrument (or person) in a sample containing multiple instruments (or persons); it takes the design of *k*-measures from *n* subjects (Safrit & Wood, 1989).

Intrinsic Asthma: Asthma attributed to pathophysiologic disturbances and not to environmental factors (Dorland's, 1988).

K

Kilocalorie: The amount of heat required to raise the temperature of 1 kilogram of water 1℃ (Morehouse & Miller, 1976). This is sometimes known as a "large calorie." 1 kcal = 1,000 Cal = 4,186 J = 4.186 kJ = 3.968 BTU. These equivalencies vary in the literature.

Krebs Cycle: See "Citric Acid Cycle."

K-S test: The Kolmogorov-Smirnov "non-parametric" statistical test of two distributions where sample values are ordered as a cumulative frequency distribution [see]. The sample assumes random-sampling from an identical population, but does not assume that the data, which can be interval, ordinal, or ratio observations, are normally distributed.

L

Lactate: The anionic (containing an anion, or negatively charged ion) form of lactic acid in the blood (Dorland's, 1988).

Lactate Threshold (L_T): The highest oxygen consumption (exercise or work intensity) with <1.0 mM / L increase in blood lactate concentration above the pre-exercise level (McArdle et al., 2001). L_T is also expressed as mg dL^{-1} of whole blood, and sometimes as a volume percent of whole blood (vol%). 1.0 mM L^{-1} = 9.0% vol%. By convention, blood lactate concentration usually is expressed simply as millimoles (mM) (McArdle et al., 2001).

L_T varies significantly with respect to physical fitness. Training increases the VO_2 level at which L_T occurs. In untrained persons, it is about 67% of $VO_{2.MAX}$. In Trained "normals," L_T is on the order of 80% of $VO_{2.MAX}$, and in people with coronary artery disease, L_T may be 100% of $VO_{2.MAX}$ (McArdle et al., 2001).

Lactic Acid: (1) A metabolic intermediate involved in many biochemical processes (Dorland's, 1988). (2) End product of glycolysis, which provides energy anaerobically in skeletal muscle during heavy exercise and which can be oxidized aerobically in the heart for energy production (Dorland's,1988).

Lactic Acid Threshold (LAT): The O_2 uptake level above which lactate acid accumulates [see L_T]. It used to be thought that it was the point in the oxygen consumption/work rate relationship where there is hyperventilation relative to VO_2 but not to CO_2 elimination. That is now known as the Ventilatory Threshold [se].

Lazarev & Brusilouskaza's Rule: A "rule" of exposure where $c * t^b$ = constant toxic effect = k.

Lean Body Mass (LBM): Technically, LBM refers to the mass of muscle, water, bone, and "essential fat" in the body. Essential fat is the small percentage of non-sex-specific fat contained in the central nervous system, bone marrow, and internal organs. Essential fat approximates 3% of total body mass (McArdle et al., 2001). It often is used interchangeably with fat-free mass (FFM), but they differ in the amount of essential fat in the body.

Leisure: Any activity chosen primarily for its own sake; thus, freedom of choice and intrinsic meaning to the individual are the defining factors (Kelly et al., 1986).

Lifestage: A period of time in a person's life associated with the life course of development as related to life cycle of the family. With respect to the former, it includes such stages as neonate, baby (infant), toddler, pre-schooler, school child, teenager, young adult, etc. Viewed in that context, the lifestage progression is one from absolute dependence on a caregiver to relative independence and autonomy. Conditions of aging can, of course reverse this pattern; frailty and sickness can lead back to complete dependence on caregivers for existence. The family role dimension relates to such socio-cultural attributes as intra-familial relationships, child care responsibilities, household and individual consumption patterns (economic dimension), residential mobility and "space consumption" needs (residential housing needs), career stage (work-family relationships), sexual roles and relationships, and transgenerational roles. Sometimes the emphasis is placed on periods of transitions between relatively stable lifestages, which often result in stress and role identification problems.

Lifestyle: (1) Those components of daily behavior that are systematic and regular over a specified period of time. The term encompasses behavioral factors, such as time use (work, leisure, sleeping patterns), dietary intake (nutrition), personal habits (smoking, drug, and alcohol use), *patterns* of physical activity and exercise (exercise habits, physical fitness), health-promoting behaviors in general (including taking action to prevent or detect disease, or for improving health and well-being), and psychological and social considerations (the form/intensity of social interaction, psychological health, sexual health). Other attributes that sometimes are included are locational considerations of where a person chooses to live (condominiums, single-family subdivisions, rural locations, isolated areas, etc.). Thus, the concept has a number of dimensions and complex attributes. See: Harrison, 1996). (2). The pattern of living as expressed in a person's activities, interests and opinions

Light Physical Activity: A phrase with many meanings, in that the energy expenditure levels associated with it are defined in highly variable ways (heart rate, % of heart rate reserve, VO_2 consumption, and % of VO_2 reserve, % of $VO_{2\ Peak}$, METS, etc.). Most of the definitions are laboratory or investigator-dependent, with little attempt at standardization of the metrics used. Perhaps the most rigorous definition might be the lower third of VO_2 reserve.

Linear Dose-Response Relationship: A relationship between dose of a substance and the frequency or severity of biological response in a population that varies proportionately with the amount of dose (IRIS, 1999).

Linear Model: A mathematical or statistical model where the dependent variable Y varies as a linear function of one or more independent variables or factors. For one independent variable (x), the most reduced form of the equation is Y = b*x. For a statistical version of this simple model, Y = a + b*x + e, where: a = an "intercept constant," and e represents random variation, or error (Last, 1983).

Location: With respect to human activity modeling or monitoring, a three-dimensional space that is occupied for some known period of time by a habitué. When it has a constant environmental concentration for a specified period of time, it is known as a microenvironment.

Log-normal Distribution: A distribution of data such that Y=log X is normally distributed. It is a "skewed" distribution having regular parameters in log space.

Log-Transformation: Taking a logarithm of a sampled quantity in order, generally, to makes its association with one or more other sampled quantities linear so that usual (linear) statistical tests can be used on the data. They generally are used when one variable takes on a wide range of values, but with diminishing association with one or more other variables as the sampled values increase or decrease. The

assumption of random-probability sampling still holds, however. The data are usually characterized by a geometric mean and geometric standard deviation.

Long-term: A vague term that relates to a relatively long time period.

Longitudinal Study: A study or analysis with observations or samples taken at multiple time points.

Lower Respiratory Tract: That part of the respiratory tract below the larynx (EPA, 1989).

Lung: (1) One of a pair of highly elastic cone-shaped organs of respiration occupying the thoracic cavity and surrounded by a pleural sac (International, 1986). (2) Either of the pair of organs that aerate blood. The right lung has a superior, middle, and inferior lobes; while the left lung only has the first and third lobes just mentioned. Each lobe is subdivided into 2-5 bronchopulmonary segments. The lung consists of an external serous coat (visceral layer of the pleura), subserous areolar tissue, and lung parenchyma. The latter is made up of lobules, which are bound together by connective tissue. A primary lobule consists of terminal bronchiole, respiratory bronchioles, and alveolar ducts, which communicate with many alveoli. Each alveolus is surrounded by a network of capillary blood vessels at the interface of which gas exchange occurs (Dorland's,1988). The average lung surface area in a normal adult human is 70-90 m^2 (Åstrand & Rodahl, 1986).

Lung Volume (V_L): Volume of the lung, including volume of the conducting airways.

M

Macro-activity: With respect to human activity modeling or monitoring, the general activity or pursuit that an individual is engaged in for a specified period of time. See also: Micro-activity.

Macro-Activity Data: Information on where a person is and what she/he is doing for a specified period of time. This information includes an identification of the microenvironment being occupied, the general activity being undertaken, and the energy expenditure level (or heart rate, breathing rate, etc.) of the activity being engaged in (or relative energy level, such as METS).

MAX (max): Maximum; often used as a subscript.

Maximal Aerobic Capacity ($VO_{2.MAX}$): The maximal oxygen consumption rate recorded for an individual following a, generally more progressive, protocol.

Maximal Expiratory Flow: See Maximal Ventilation Rate.

Maximal Heart Rate (HR_{MAX}): The highest heart rate value measured during an all-out effort to the point of exhaustion (Nieman, 1999).

Maximal Heart Rate Reserve (HRR): The difference between maximal and resting heart rates (Nieman, 1999).

Maximal Oxygen Uptake/Consumption ($VO_{2.MAX}$): The maximal capacity for oxygen consumption during maximal exertion; also known as aerobic power and cardiorespiratory endurance capacity (Nieman, 1999). See $VO_{2.MAX}$. It is associated with the peak rate of oxygen delivery to the working muscle, which in turn is dependent upon capacity of the lungs and the cardiovascular system to transfer oxygen in the body (Blomqvist, 1978).

For short periods of time, $VO_{2.MAX}$ is a relatively stable and reproducible individual characteristic although it does change over the years and is affected by health status, body size age, sex, and habitual level of physical activity of the individual (Blomqvist, 1978). There is not much difference in $VO_{2.MAX}$, on either an absolute or relative (body mass) basis, in prepubertal children, but there is for subsequent ages due to body composition changes at puberty; level of physical activity also decreases in most females at that time (Blomqvist, 1978). The COV for $VO_{2.MAX}$ for healthy, similar age/gender cohorts is 10-15%, but larger relative variations have been measured.

Maximal Ventilation Rate ($V_{E.MAX}$): A surrogate for the maximal (inspired) breathing rate needed to sustain a person's maximal oxygen consumption rate.

Maximal Voluntary Ventilation (MVV): The volume of air breathed by a subject during voluntary maximum hyperventilation [rapid deep breathing] for some specified time period. Its units are in liters at BTPS. MVV is also known as maximal breathing capacity, now an obsolete term. Individuals vary greatly with respect to MVV, partly due to motivational factors. MVV for college aged subjects are 70-120 L/min ♀ and 100-180 ♂ (Åstrand & Rodahl, 1986).

Media / Medium: One of the "basic categories" of material surrounding or containing a receptor organism through which a chemical or pollutant can reach it. Usually applied to ambient air, beverages, drinking water, food, indoor air, sediments, soil, and surface or underground water.

Metabolic Equivalent of Work: See METS. Also called a "metabolic unit" (Cowan et al., 2009).

Metabolite: (1) A substance taking part in or produced by metabolic activity (International, 1988). (2) One of the intermediate or final products in the metabolic breakdown of foodstuffs in the body (Morehouse & Miller, 1976).

Metaboic Chronotropic Relationship (MCR): The difference between maximal VO_2 and resting VO_2 in an individual, also called VO_2 reserve. The relative amount of activity-specific VO_2 to VO_2 reserve approximates that of energy expenditure metrics and heart rate metrics.

Metabolic Rate: The rate at which the body uses energy.

Metabolic reserve (percent) – percent metabolic reserve is the difference between the METS level at any point in exercise and METS level achieved at peak exercise. It reflects the level of work during any stage of exercise. The percent metabolic reserve is calculated as follows: $[(METS_{ACT} - MET_R)/(METS_{MAX} - MET_R)] * 100$, where $METS_A$ is the observed metabolic equivalents for activity A (an exercise level in METS), $METS_{MAX}$ is the actual observed METS at the peak level of exercise performed (i.e.,

not a theoretical peak value), and MET_R is the MET value while the patient is at rest (MET=1). This is simplified to: $METS_{RESERVE} = [(METS_{ACT} -1)(METS_{MAX} – 1)] * 100$.

Metabolism: (1) Totality of chemical processes occurring in a living organ, especially those associated with exchange of matter and energy between a cell and its environment (International, 1986). (2) Physical and chemical processes by which a living organized substance is produced and maintained (Dorland's, 1988). (3) Aerobic metabolism is dependent upon the presence of oxygen; also called respiratory metabolism (International, 1986). Anaerobic metabolism occurs in the absence of molecular oxygen.

MET(S): Metabolic equivalents of work. It is the ratio of an activity-specific metabolic rate to a person's resting (or basal) metabolic rate. (1) One MET *approximates* 1 kcal kg^{-1} $hour^{-1}$ of energy expended in an adult, but this equivalency masks important age and gender differences. It *approximates* 3.3-3.5 ml of O_2 uptake kg^{-1} min^{-1}, which is often used as the resting state oxygen consumption rate for humans, but is not really justified by measurement data. There are comprehensive lists of the METS associated with common human activities, including Durnin & Passmore (1967), Ainsworth et al. (1993), and Montoye et al. (1996). Most of the data are for young adults, and generally underestimate *relative* energy expended by children and overestimate energy expended by the elderly to accomplish a task.

Micro-activity: Skin-surface--including the mouth--contact with an object [toys, furniture, materials, surfaces, liquids, another part of the subject's skin itself, another person's skin surface, etc.] by an individual that occurs as part of engaging in a more general activity (macro-activity).

Micro-activity Data: Quantified information on the frequency, duration, intensity, and pattern of skin surface contact with the hand or object. This information should include the nature of the skin-to-object contact [pressure, motion, area and location of the body surface touched] as well as characteristics of the surface area itself [surface type, texture, and absorbing capacity].

Microenvironment: A physical 3-dimensional space that is treated as a well-characterized, relatively homogenous *location* with respect to a chemical or substance concentration for a specified time period (adapted from EPA, 1992).

Microenvironmental Model/Method: A predictive exposure assessment approach to estimating sequential exposures experience by an individual passing through a series of microenvironments, as defined by the individual's "actual" or estimated human activity information. Usually, the time period of interest is an entire day, or a series of days that are "strung together" using daily activity information. See "Activity Pattern," "Microenvironment," and "Indirect Exposure."

Microenvironmental Monitoring: (1) The monitoring, or measuring, of one or more specified substances in a micro-environment via some type of media-specific sampling device. The device may be "active" [flow through the sampling train is mechanically regulated] or "passive" [flow rate is not controlled, and the sampling rate is greatly affected by deposition and Brownian movement]. Microenvironmental monitoring procedures are independent of whether or not a potential receptor inhabits the space that is being investigated.

Minute Ventilation: The volume of air expired per minute (International, 1988).

Minute Ventilation Rate: See "V_I."

Minute Volume (MV): The minute volume of breathing (MV); a product of tidal volume (V_T) times the respiratory frequency (f_R) in one minute; synonymous with minute ventilation.

Mitochondria: Intracellular structures containing enzymes used in the chemical reactions that convert food energy to a form that the body can utilize (Fahey et al., 2007).

Model: (1) Theoretical propositions on a domain of reality (Becker, 1989). (2) An abstract representation of the relationship among logical, analytical, or empirical components of a system (Last, 1983). A model usually consists of the mathematical structure and particular constants or parameters associated with the structure. A model may be deterministic or stochastic (Last, 1988). (3) A representation or simulation of an actual situation or natural system. The output (end result) of a model is an estimate or prediction resulting from entering a set of input quantities into and "running" (exercising) the mathematical relationships that constitute the model's structure.

Moderate Physical Activity: A phrase with many meanings, in that the energy expenditure levels associated with it are defined in highly variable ways (heart rate, % of heart rate reserve, VO_2 consumption, and % of VO_2 reserve, % of $VO_{2\ Peak}$, METS, etc.). Most of the definitions are laboratory or investigator-dependent, with little attempt at standardization of the metrics used. One rigorous definition might be the middle third of VO_2 reserve (McCurdy & Graham, 2004).

Moderate and Vigorous Physical Activity (MVPA): Moderate (MPA) **and** vigorous physical activity (VPA) combined.

N

National Ambient Air Quality (NAAQS): Federal and nationally-applicable air standards that are established by the EPA under Section 109 of the Clean Air Act after a lengthy review and comment period involving Agency and independent scientists, the general public, interested parties [generally, environmentalists and industrialists], State air regulatory agencies, and the political administration currently in office.

Nasopharyngeal: Relating to the nose, nasal cavity, and the pharynx.

National Exposure Research Laboratory (NERL): A group within EPA's Office of Research and Development (ORD) that is focused on the measurement and monitoring of human exposures, including ambient processes that lead to these exposures.

National Human Activity Pattern Survey (NHAPS): A random-probability telephone survey of "yesterday's" activity pattern of continental US residents that was conducted by the University of Maryland's Social Research Center under contract to NERL in 1992-1994. NHAPS contains 9,386 individual person-days of activity data, and it is part of the CHAD database.

Neonatal: Newly born. In humans, it is considered to be up to 6 weeks of age (EPA, 1989).

Non-Exercise Activity Thermogenesis (NEAT): All non-exercise physical activity, such as fidgeting and squirming (Levine et al., 2000).

Non-oxidative Energy System: The anaerobic system that supplies energy to muscle cells through the breakdown of muscle stores of glucose and glycogen. This is also known as the anaerobic system or the lactic acid system because chemical reactions take place without oxygen and produce lactic acid (Fahey et al., 2007).

Non-parametric: Data that are not necessarily normally distributed; said to be distribution-free. The term often is used to denote a large category of statistical tests which do not require an assumption of normally-distributed data or a population (Blalock, 1960).

Normal Workload: A light or moderate load in which a person's oxygen intake is adequate to supply the needs of the body (Morehouse & Miller, 1976).

Null Hypothesis (H_0): An precisely-stated hypothesis the truth of which is examined by a statistical test having a specified level of significance, generally an α of 0.05, which is the probability of rejecting $\mathbf{H_0}$ if it is true (also called Type I error).

O

Obesity: (1) A bodily-state above normal adiposity at which health problems are likely to occur. Various criteria have been used to identify obesity, such as "excess" body mass on a per-age, height, or BMI bases, or on "excess" adiposity (Bar-Or & Baranowski, 1994). See "Adiposity" and "BMI". (2) An excess accumulation of body fat. Alternative measures from Summerfield (1990) are: (a) for male children: body mass is >25% fat, as estimated by the skinfold method, (b) for female children: body mass is >32% fat, and (c) for others: weight-for-height is >20% of the ideal.

Objective (Monitoring) Method: A means of obtaining interval or ratio data directly from a subject by some type of methods that does not involve a subjective decision, or observation, by the subject regarding the measurement quantity.

Occupational Exposure Limit (OEL): A generic term denoting a variety of values and standards, some of them enforceable by law. They generally are time-weighted average concentrations--most often for airborne substances--to which a worker can be exposed during one or more defined time periods [e.g., 15 min, 1 h, 8 h] (EPA,1989).

Onset of Blood Lactate Accumulation (OBLA): The point in the blood lactate concentration that shows a systematic increase = 4.0 mM (McArdle et al., 2001). They also state that researchers of equate the OBLA with the lactate threshold (L_T), but the two terms represent operationally different (and precise) points in blood lactate/exercise intensity levels. See Figure 14.5 in McArdle et al. (2001) .

Oronasal: Breathing through the nose and mouth simultaneously. This is the typical human breathing pattern at moderate-to-high (heavy) levels of exercise or work. Nasal-only breathing is the norm at rest or at low levels of exercise or work, although some people are predominately nasal breathers even at high exertion rates. (However, other people are predominately oral breathers at any exertion rate.) (Samet, et al., 1993).

Outcome: All possible results that stem from exposure to a causal factor or to an intervention (Last, 1983).

Overload: (1) Impairment of lung clearance of a pollutant [substance] at high lung burdens (Vincent & Donaldson, 1990). (2) A heavy workload in which oxygen uptake is inadequate to meet the requirement (Morehouse & Miller, 1976).

Oxidant: A chemical compound that has the ability to remove electrons from another chemical species, thereby oxidizing it; also, a substance containing oxygen that reacts in air to produce a new substance, or one formed by the action of sunlight on oxides of nitrogen and hydrocarbons (EPA, 1992).

Oxidation: (1) An ion or molecule undergoes oxidation by donating electrons. (2) The removal of hydrogen or electrons from a compound. In biological oxidation, oxygen does not directly combine with the substance "being oxidized," but combines with hydrogen to form water (Morehouse & Miller, 1976).

Oxygen Cost (of Breathing): The amount of O2 needed to sustain breathing itself. At rest (~6 L min-1) the oxygen cost is ~2%. As ventilation increases, the energy cost per liter ventilation increases rapidly, as does the oxygen cost--up to ~10% at 50 L min-1 or so (Åstrand & Rodahl,1986).

Oxygen Debt: (1) Delayed return of oxygen uptake (VO_2) to a resting level after the cessation of exercise [work] (Åstrand & Rodahl, 1986). (2) Oxygen consumed in excess of the resting (post-exercise) O_2 requirement (McArdle et al., 2001).

Oxygen Consumption (VO_2):. Oxygen taken into the body and used in tissues. In the physiology literature, it is a volume if shown without an overstrike over the "V", and as a rate (per minute) with an overstrike. The terms "Oxygen

Intake," "Oxygen Utilization," and "Oxygen Uptake," generally are used as synonyms for oxygen consumption (Montoye et al, 1996).

Oxygen Deficit: (1) The difference between the oxygen requirement and the oxygen intake during performance of the task (Morehouse & Miller, 1976). (2) The difference between the total O_2 consumed during exercise (work) and the total that would have been consumed had a steady rate of aerobic metabolism been reached immediately at the start of the exercise (McArdle et al., 1991).

Oxygen Consumption Reserve ($VO_{2.RES}$): The difference between maximal oxygen consumption ($VO_{2.MAX}$) and oxygen consumption at rest in an individual ($VO_{2.R}$): $VO_{2.RES} = VO_{2.MAX} - VO_{2.R}$. $VO_{2.MAX}$ is about 10-15 times higher than $VO_{2.R}$ is normally-active and fit individuals on a group mean basis, and even greater is specific persons (Blomqvist, 1978).

Oxygen Intake: See "Oxygen consumption."

Oxygen Pulse: Oxygen pulse is an indirect measurement of stroke volume. It is defined as the oxygen uptake per heart beat and is measured by dividing the oxygen uptake in one minute over heart rate (VO_2/HR). As stroke volume increase, so does O2 pulse. It is the amount of oxygen extracted by the tissues of the body from the O2 carried by the blood pumped from the heart in each stroke. The term is derived from the Fick equation (Luks et al., 2012).

Oxygen Pulse/Oxygen Saturation: The amount of O_2 combined with hemoglobin, expressed as a percentage of the O_2 capacity of that hemoglobin. **Oxygen Uptake**: See "oxygen consumption."

Ozone (O_3): A reactive oxidant gas produced naturally in trace amounts in the earth's atmosphere; it is composed of three oxygen atoms. Most of the earth's atmospheric O_3 is found in the stratosphere.

P

PaO_2: Arterial partial pressure of oxygen.

PAO_2: Alveolar partial pressure of oxygen.

Parameter: In mathematics, a constant; in statistics and epidemiology, a measurable characteristic of a population that may take on varying values (Last, 1983).

Pathway: See "Exposure Pathway."

PE: Physical education: generally, a period of time during a school or college day in which students undertake one or more physical activities in a relatively structured way. The activities often are moderate-to-vigorous in nature, involving individual or group sports events.

Peak Exposure: The maximum instantaneous exposure for a specified time period (Duan et al., 1990).

Peak Oxygen Consumption ($VO_{2.PEAK}$ or $VO_{2.MAX}$): As a rate—overstrike over the "V"—it is the maximal VO_2 rate of an individual at the point where the individual stops an exercise fitness test. Occasionally, physiologists define $VO_{2.PEAK}$ as the above when "objective" criteria for attaining $VO_{2.MAX}$ has not been attained. Most generally, this distinction is not made, and the two terms are treated synonymously.

Percent Body Fat (%BF): The percentage of body weight (mass) that is fat, estimated by skin-fold measurements, bioelectrical impedance, or by displacement of water or air by immersion in a tank of water or sealed air chamber.

Perfusion: Passage of blood or other fluid through blood or lymph vessels or any part of body (International, 1986).

Personal Exposure Measurement: A concentration measurement collected from an individual's immediate environment using active or passive devices ((IPCS, 2000).

Personal Exposure Monitor (PEM): A personal exposure measurement device worn on or near a contact boundary (Zartarian et al., 1997).

Personal Monitoring: (1) Monitoring, or the measurement of one or more specific substances in, on, or immediately near a specified living receptor via some type of media-specific sampling device. The device may be "active" [flow rate through the sampler is mechanically regulated] or "passive" [flow rate is not controlled, and the sampling rate is greatly affected by the physics of deposition and Browning movement]. Compare with "Microenvironmental Monitoring;" personal monitoring moves with the receptor as he/she/it enters and leaves the various microenvironments that are encountered over the sampling period.

Pharynx: The irregularly-shaped cavity into which the nose and mouth open. The larynx is below. Pharynx is the medical term for throat. Air and food passages cross in the pharynx.

Photochemical oxidants: Primary ozone, nitrogen dioxide, and peroxyacetyl nitrate, with lesser amounts of other compounds, formed as products of atmospheric reactions involving organic pollutants, nitrogen oxides, oxygen, and sunlight (EPA, 1993).

Photochemical smog: Air pollution caused by chemical reaction of various airborne chemicals in sunlight (EPA, 1993).

Physical Activity (PA): (1) Any bodily movement produced by skeletal muscles that results in an expenditure of energy above the resting level (Baranowski et al., 1992; Kohl et al., 1988). Exercise is a major component of physical activity. (2) Naturally occurring body movement (Bar-Or & Baranowski, 1994). (3) Dynamic or static skeletal muscle exertion that increases the body's energy expenditure and results in cardiorespiratory adjustments. Dynamic PA involves body movements through rhythmic contraction and relaxation of large skeletal muscle groups. Static [isometric] activity consists of increased muscular tension against a fixed resistance with no change in fiber length (Leon, 1989). It has both physiological and behavioral aspects.

Often physical activity is further described by the level of activity, such as light, moderate, and vigorous. It also is divided into source or type of physical activity, such as occupational, domestic, leisure-time, physical educational, or recreational.

Physical Activity Index (PAI): Daily Total Energy Expenditure (in kcal/kg) / Basal / Resting Metabolic Rate (in kcal/kg). Thus is a unitless metric that is used to characterize a person relative daily energy expenditure vis-à-vis normative rates for sedentary, low-active, active, etc. individuals.

Physical Activity Level (PAL): Identical to PAI, used mostly by European exercise physiologists.

Physical Fitness: (1) The ability to carry out daily tasks with vigor and alertness, without undue fatigue and with ample energy to undertake leisure time activities and to meet "energy emergencies" (Kohl et al., 1988). (2) The ability to do physical activity or to perform physical work; a measure of a person's "functional capacity" (Solomon, 1984). (3) The ability to perform moderate-to-vigorous levels of physical activity without undue fatigue, and the capability of maintaining such activity throughout life (ACSM, 1990). (4) The ability to maintain internal equilibria as closely as possible to the resting state during strenuous exercise and to quickly restore any disturbed equilibriums (Åstrand, 1956). There are alternative definitions; see: Pate, 1988). $VO_{2.MAX}$ is considered by many to be an objective measure of fitness, and timed distance runs are used as a surrogate for fitness in field settings (Pate, 1991; Pate et al., 1990). Again, there are alternative field "measures" of fitness.

Sometimes physical fitness is disaggregated into "health-related fitness" and "skill-related fitness." The former includes cardio-respiratory endurance, body composition, and musculosketal considerations (flexibility, strength, and muscular endurance). Skill-related fitness includes activity-specific factors, such as agility, balance, coordination, speed, power, and reaction time (Nieman, 1990).

Physical Fitness Index (PFI): A measure of O_2 consumption per body weight (mL/kg).

Physical Working Capacity (PWC): It is the maximal rate of oxygen utilization in aerobic metabolic processes. It also is known as "functional capacity," "cardiorespiratory fitness," or "maximal aerobic power" (Simons-Norton et al., 1988).

PWC-170 (or PWC_{170}): Physical working capacity of an individual, in units of kilopond-meters min^{-1} (kp/min), or body mass-adjusted kp/min, at a heart rate of 170 beats per minute (bpm).

Physiology: Study of or the normal functioning of a living organism (International, 1986).

Point-of-Contact Exposure: An exposure estimate expressed as the product of concentration of a substance in an exposure medium, the duration of contact, and body surface area of the receptor in contact with the substance; a typical unit for this metric is mg m^{-2} h^{-1} (CMA, n.d.). It is a surrogate estimate of dose received for those substances that produce toxicity directly at the point of contact with the body [skin or mouth].

Point-of-contact Exposure Measurement: An approach to quantifying exposure by taking measurements of concentration over time at or near the point of contact between the substance and the receptor surface of interest while the exposure is occurring (IPCS, 2000).

Pollutant: (1) Substance in a medium to which the target is exposed (Duan et al., 1990). (2) An undesirable modification of a medium by a substance that is toxic, results in an adverse effect on health, or is offensive (Last, 1983).

Population: The complete set from which a sample is drawn.

Population Variability: The concept of differences in susceptibility of individuals within a population to toxicants due to variations such as genetic differences in metabolism and response of biological tissue to chemicals (EPA, 1989).

Portal-of-Entry Effect: A biological response to a toxicant at its site of entry into the body (EPA, 1989).

Post-Exposure Period: The time period subsequent to the last exposure to a substance but within the period of analysis (Vincent & Donaldson, 1990*mod*).

Potential (Human) Exposure: A potential exposure situation arises when two conditions are present: (1) valid information, usually analytical environmental data, indicates that a contaminant of public health concern exists in one or more environmental media [i.e., air, water, soil, food]; and, (2) that there is an identified route of exposure between the medium/media and human receptors: i.e., drinking contaminated water, breathing contaminated air, having contact with contaminated soil/pesticides/etc., or eating contaminated food (ATSDR,1999).

Power: (1) Work performed per unit time (Lamb, 1984). (2) The rate of performing work; the derivative of work with respect to time; the product of force and velocity (McArdle et al., 2001). See also "statistical power."

Precision: The quality of being exactly or sharply defined (Webster's Ninth, 1974).

Probability: (1) Limit of the relative frequency of an event in a sequence of $\underline{n}$ random trials as $\underline{n}$ approaches infinity; the limit of: [number of occurrences of an event]/$\underline{n}$ (Last, 1983). (2) A measure, ranging from 0 to 1, of the degree of belief in a hypothesis or statement (Last, 1983).

Probability Density Function: (1). A function whose value at a particular point describes the relative probability that an uncertain value will be near that point (Feagans & Biller, 1981). (2).The derivative of a cumulative distribution function.

Probability Distribution: A distribution giving the probability of any value *x* as a function of *x* (Kendall & Buckland, 1971).

Probability Encoding: An explicit, precise, and formal technique for quantifying expert judgments on well-defined, but uncertain quantities (Feagans & Biller, 1981).

Probability Sampling: Any method of selection of a sample based on probability theory.

Probabilistic Analysis: A general term; one definition is the calculation and expression of health risk using one or more of a number of possible risk metrics to estimate the likelihood of an (adverse) effect of interest. Probabilistic risk results delineate a range of possible outcomes and their likelihood; they are often presented as a frequency distribution that quantitatively depicts variability of the estimate. The uncertainty regarding this distribution may also be depicted. See "Uncertainty" and "Variability."

Probit Model: A dose-response model that can be derived under the assumption that individual tolerance is a random variable following a log-normal distribution (EPA, 1989).

Pulmonary: Pertaining to the lungs (Dorland's, 1988). Often used with function, as in pulmonary function.

Pulmonary Compliance: The volume change per unit of pressure change for the lungs, thorax, or the lung-thorax system. The distensibility of the lungs or thorax (EPA, 1989).

Pulmonary Edema: An accumulation of excessive amounts of fluid in the lung extravascular tissue and air spaces.

Pulmonary Measurements: Measurements of the volume of air moved during a normal or forced inspiration or expiration. Specific lung volume measurements are defined independently.

Pulmonary Region: The area of the respiratory system consisting of the respiratory bronchioles and alveoli where gas exchange occurs (EPA, 1989).

Pulmonary Ventilation: Total exchange of air and gas between the lungs and air needed for aerobic energy metabolism, usually measured in liters per minute (Dorland's, 1988). It is measured by V_I or by V_E, which are not exactly equal (Åstrand & Rodahl, 1986).

Q

Quetelet Index: See "Body Mass Index."

R

Random Sample: A sample that is arrived at by selecting sample units such that each possible unit has a fixed and determinate probability of selection (Last, 1983).

Rating of Perceived Exertion (RPE): The subjective effect, discomfort, strain, and fatigue during exercise of other physical activity (Robertson & Noble, 1997). The most common format of the RPE is as a categorical scalar; 5 different versions are listed in the citation. The researcher most associated with the concept is Gunnar Borg, a Swedish exercise psychologists (Borg, 1973). There are 3 different Borg Scales, with the 15-point RPE Scale probably being the one most used.

Reaction: (1) See "Response." (2) A process in which a substance is changed chemically (International, 1986).

Reactivity: (1) Tendency of a substance to undergo chemical change. (2) In a human study, it is a "change in behavior due to being monitored" (Beets (2006).

Recall Survey: A study design that asks subjects to "subjectively" recall some type of past event or activity. There are many forms of this survey, using different time period, activities participated in, locations visited, foods eaten, pollutants encountered, etc.

Receptor: Any living organism or non-living entity, substance, or material that is exposed to a pollutant of interest.

Reliable, Reliability: (1) A quantity that is sound and dependable (stable) over repeated measurements. (2) Consistency of response across (a) multiple trials of a single administration of a test or instrument [this is internal consistency], or (b) across multiple administrations [test/re-test stability or reliability] (Patterson, 2000). It should be estimated via an intra-class correlation coefficient from an analysis of variance, however, and **not** by **r**. (3) Repeatable and reproducible are synonyms when used as a noun, but not in their verb form: repeatability and reproducibility. Note that the term does not refer to the quality of the measurement or estimate, but to the *process* of performing something more than once (IPCS, 2000).

Relative Aerobic Strain (RAS): The unitless ratio of the oxygen consumption needed to perform a specific task to a person's maximum oxygen consumption, usually multiplied by 100 to change it into a percent ((Oja et al., 1977). It is used by industrial physiologists as a measure of "strain," or activity-long work rates.

Reserve: A quantity available beyond what normally is needed; a surplus of potential use in extra-ordinary circumstances (International, 1986). In a number of "reserve" physiological metrics, it is the difference between the maximal measurement and that occurring at rest, or basal conditions.

Residual volume (RV): that volume of air remaining in the lungs after maximal exhalation. The method of measurement should be indicated in the text or, when necessary, by appropriate qualifying symbols. RV = FRC - ERV. RV also is used to denote "total lung capacity ratio," equal to RV/TLC. RV used this way expresses the percentage of total lung capacity occupied by residual volume; this varies somewhat with age, but ordinarily should be no more than 20 to 30%.

Resistance Training: Training designed to increase strength, power, and muscle endurance (Nieman, 1999).

Respiration: (1) The totality of the processes of gaseous exchange between tissues of the body and its environment; the process of breathing (International, 1986). (2) Exchange of O_2, and CO_2 between atmosphere and cells, including inspiration and expiration [ventilation], the diffusion of oxygen from pulmonary alveoli to the blood, and the transport of O_2 to and CO_2 from body cells (Dorland's, 1988). (3) The exergonic metabolic processes in living cells by which molecular O_2 is taken in, organic substances are oxidized, free energy is released, and oxidized products [CO_2, H_2O, etc.] are given off by cells (Dorland's, 1988).

Respiratory Cycle: See "Respiration Rate."

Respiratory Frequency (f_R): Breathing rate in breaths min^{-1}. At rest, f_R ~ 10-20 breaths min^{-1}, but it can be between 6-31 bpm in adults (Bendcertit, 2000). Also known as breathing frequency (f_s).

Respiratory Quotient (RQ, R): Ratio of the volume of carbon dioxide produced (CO_2) divided by the volume of oxygen consumed (O_2) by an organism, an organ, or a tissue during a given period of time (CO_2 O_2^{-1}). Respiratory quotients are measured by comparing the composition of an incoming and an outgoing medium, such as inspired and expired gas, inspired gas and alveolar gas, or arterial and venous blood. This ratio reflects the metabolic exchange of the gases in the body's tissues and is dictated by the percentage of carbohydrate, fat, and amino acids used in energy production by the cells. Carbohydrate metabolism yields an RQ of 1, whereas proteins and fats yield RQs of 0.8 - 0.9 and 0.7, respectively. A normal mixture of fat and carbohydrate metabolism yields an RQ of around 0.8. Except in malnourishment, protein is seldom used for energy metabolism.

See McArdle et al. (2001) for calculation formula for RQ based on CO_2 and O_2 flow rates (p. 1120). RQ changes with the degree of work (physical activity) undertaken. At rest, RQ usually is 0.75-0.81 but increases close to 1.00 when only carbohydrates—the "preferred fuel" for heavy exercise--are used (Nieman, 1990). RQ actually can go above 1.00 during recovery due to the buffering of lactic acid; RQ's above 1.15 indicate that maximal exertion has occurred.

Sometimes the phrase "respiratory exchange ratio" (RER) is used to designate the ratio of carbon dioxide output to the oxygen uptake by the lungs, with "respiratory quotient" being restricted to the actual metabolic carbon dioxide output and oxygen uptake by the tissues. Using this definition, respiratory quotient and respiratory exchange ratio are identical only in the steady state, a condition which implies constancy of the oxygen and carbon dioxide stores.

Respiratory Rate (RR): The frequency of a complete cycle of a breath; includes inhalation and exhalation [in L min^{-1}]. See: "f_R." The time it takes for one breathing cycle is T_{TOTAL}, which equals $T_I + T_E$. In general, $T_E > T_I$ (Benchetit, 2000).

Respiratory System: The lungs, air passages, and breathing muscles that supply oxygen to the body and carries off carbon dioxide (Fahey et al., 2007).

Response: (1) An action or movement due to a stimulus (Dorland's, 1988). (2) Any organic process elicited by a stimulus, either muscular, glandular, biochemical, or immunochemical reaction (International, 1986).

Rest: Repose, inactivity (Dorland's, 1988).

Resting Energy Expenditure (REE): Assumed to be functionally identical to basal metabolic rate [see]

Resting Metabolic Rate (RMR): Assumed to be functionally identical to basal metabolic rate [see].

Retention: Used to refer to the amount of an inhaled material that remains in the lung [pulmonary retention] or to the amount of a toxicant dose that remains in the body or body compartment for a specified period of time (EPA,1989).

Route of Entry: The means by which a substance enters the body: ingestion, inhalation, dermal. See "Exposure Route" and "Route of Exposure."

Route of Exposure: (1) The mechanism by which the medium reaches a target (Duan et al., 1990). (2) The means by which a toxic substance (agent) gains access to an organism: ingestion; inhalation; dermal absorption; intravenous, subcutaneous, intramuscular, and intraperitoneal administration.

S

Sarcopenia: The involuntary loss of skeletal muscle that occurs with advancing age (Cesari et al., 2005).

Saturation: (1) Having all chemical affinities satisfied (Dorland's, 1988). (2) Unable to hold in solution any more of a given substance (Dorland's, 1988). (3) State of a solution in which a specified substance cannot dissolve or vaporize because it is in equilibrium (adapted from International, 1986).

Sensitivity Analysis: A technique that evaluates the sensitivity of an output variable to possible variation in the input variables of a given model. The main purposes of sensitivity analysis are to (a) quantify the influence of input variables on the outputs variable, and (b) understand the "bounds" of the model output. Sensitivity of the output variable of a given mathematical model depends on the model's mathematical relationships and on plausible values of its input variables. For a given model, sensitivity of the output variable with respect to each input variable is computed and compared, usually in a sequential manner by changing one variable at a time and keeping all other variables held fixed at their nominal vales (correlated input variables, however, must be varied together in a logical fashion. Varying several input parameters at the same time often highlights interaction effects in a model which are not obvious during "one at a time" variation (IPCS, 2000).

Screening Study (Analysis, Assessment): A [risk] assessment using tentative or preliminary data. The results of such an assessment are not viewed as an absolute indicator of risk, but are viewed as an indicator of the relative importance of the various factors that give rise to risk: such as pollution sources, source-receptor geometry, the nature of the substances involved, and the patterns of exposure experienced. Most urban air toxic risk assessments to date are considered to be screening--or "scoping"--studies, useful mostly to point out where additional scientific and analytical work is needed before a definitive risk assessment can be undertaken (adapted loosely from EPA, 1989). Obviously "screening study" is a vague term that should be used with caution.

Scoping Study: See "Screening Study"

Sedentary / Sedentarism / Sedentary Person: A person who expends <10% of his or her waking daily total energy in moderate or vigorous activities [≥ 4 METS] (Bernstein et al. (1999).

Sensitive: (1) Able to respond to stimuli; often used to mean unusually responsive or responding quickly or acutely (Dorland's, 1988). (2) Quality or state of possessing a low threshold to a stimulus (International, 1986).

Sensitive Person/Population: A person/people who respond--often hyper-respond--to a pollutant exposure that would not affect most other people; a pre-existing illness often affects a person's sensitivity to an exposure (Lebowitz, 1991). Compare with "Susceptible Persons/Populations."

Sensitization: A condition in which response to later stimuli is greater than response to an original stimuli (International, 1986).

Sex: The classification of living things into generally two categories (female or male) according to the reproductive organs and functions associated with the subject's chromosomal complement. (Arbuckle, 2005). An activity undertaken for physical gratification, enjoyment, and/or procreation. See "Gender" also.

Short-Term: A vague term that relates to a relatively short time period.

Short-Term Exposure: Multiple or continuous exposure to a substance for a short period of time, usually one week (IRIS, 1999).

SI: Système Internationale d'Unités: the International System of scientific units, adopted by the World Health Organization as the official units of measurement for phenomenon inherent in the physiological, medical, and other health sciences.

Solubility: Quality or fact of being soluble, which means the susceptibility of being dissolved in the matrix in which the substance is located (adapted from Dorland's, 1988).

Spearman Rank-Order Correlation Coefficient (r_S): A measure of linear association between the rank-order of two or more sampled variables that can be used for nominal and/or interval scaled-data with $\alpha = 0.05$. The metric still assumes random probability sampling, but this assumption often is violated.

Spirometry: The measurement of air volumes of the lungs; examples: tidal volume and reserve volume (EPA, 1989). It usually involves the timed collection of exhaled air during the forced vital capacity (FVC) maneuver.

Spirometer: A mechanical device, including bellows or other sealed, moving parts, that collects and stores gases to provide a graphical or electronic record of lung volume changes over time (EPA, 1989). It usually is used to collect a timed sample of exhaled air during the forced vital capacity (FVC) maneuver.

Standard Deviation (SD): An index of dispersion around a mean of measurements in a sample, equal to √Variance. The positive square root of the sample variance.

Standard Error (SE): Standard deviation of the sampling distribution of a statistic for random samples of *n* size, equal to SD / √n.

Standard Temperature and Pressure (STP): Defined to be O ^{o}C, 760 millimeters of mercury (760 torr). Formula are presented in McArdle et al. (2001) to convert atmospheric temperature and pressures to STP based on Charles' and Boyle's laws (p. 1117).

Standard Temperature and Pressure Dry (STPD) conditions: These are the conditions of a volume of gas at O ^{o}C and 760 torr, without water vapor. An STPD volume of a given gas contains a known number of moles of that gas.

Statistic: (1) A function of one or more random variable that does not depend upon any unknown parameters. (2) A summary value calculated from a sample of observations (Kendall & Buckland, 1971).

Statistical Power (1-β): (1) The probability of being able to detect an effect is there is one (IPCS, 2000); (2) the probability of rejecting the tested hypothesis when it is false (when the alternative hypothesis—H_A—is true); (3) the probability of correctly rejecting $\mathbf{H_O}$ when it is false; it equals 1- the probability of rejecting $\mathbf{H_O}$.

Statistically Significant Effect: In the analysis of data, an effect that results in a difference between a study group sample and a control group population that is unlikely to arise by chance alone--the "chance" usually is specified in the statistical test used to test the null hypothesis of no effect, such as $\alpha = 0.05$, or a 5% probability of being wrong (EPA, 1989).

Steady State Exercise – Steady state exercise is a characteristic of physiological systems in which its functional demands are being met such that its output per unit time becomes constant. It is a level of exercise intensity at which the patient is in steady state. To reach that exercise intensity, the subject must first pass through a period of dynamic exercise to reach the steady state level.

Steady-State Exposure: Exposure to air pollutants whose concentration remains constant for a period of time; generally this is an unrealistic exposure profile.

Stochastic: The property of varying in some manner that can be described with a statistical function [i.e., follows some type of known probability function; a narrow sense is that the variability is random in nature, such as a normal probability distribution].

Stochastic Model: A mathematical model which includes one or more stochastic variables or parameters. Estimates made using this type of model therefore do not give single-point estimates, but a distribution of possible estimates [with some specified probability].

Stoichiometry: (1) The application of the laws of (a) definite proportion and (b) conservation of matter and energy to chemical activity. (2) A quantitative relationship among constituents in a substance, especially those undergoing physical or chemical change.

Stratification: The division of a population into two or more subgroups for sampling or analysis purposes.

Strength: The ability of muscle to exert force (Nieman, 1999).

Stroke Volume (SV): the amount of blood pumped per heart beat, in liters or milliliters beat^{-1}.

Subchronic: A period of time that is intermediate between acute and chronic (CMA, n.d.). This term is vague and should not be used.

Subchronic Exposure: (1) A vague term used by some cancer risk assessors to denote an exposure to a substance that spans no more than ten percent of the exposed organism's lifetime (EPA, 1992). (2) An exposure of intermediate duration between acute and chronic (IPCS, 2000). (3) Multiple or continuous exposures lasting for approximately ten percent of an experimental species lifetime, usually over a three-month period (EPA, 1997).

Substance: Any material of a specified nature but of no shape or dimension, as a chemical or tissue (International, 1986).

Subjective Interpretation of Probability: The view that probability is a measure of the degree of belief--or quantified judgement--of an individual, where that individual is willing to make choices in a well-defined situation (Feagans & Biller, 1981). See also: "Frequency Interpretation of Probability" and "Probability Encoding."

Susceptibility: (1) Condition of being susceptible, or liable to the effects of substances, toxins, or other influences; lacking capacity to respond effectively to pathogens (International, 1986). (2) Preexisting biological characteristics that lead to an enhanced response to a dose or exposure. Susceptible individuals, when sufficiently dosed (exposed) become sensitive to further doses [exposures]; susceptibility may be specific or non-specific (Lebowitz, 1991).

Susceptible Person/Population: A person with a pre-existing disease that makes them susceptible [see].

Synergistic Effect: (1) Any effect of two chemicals [substances] acting together which is greater than the simple sum of their effects when acting alone (Duffus, 2000). (2) Joint effects of two or more agents, such as drugs that increase each other's effectiveness when taken together (SRA, 1999).

System: (1) A complex of anatomically-related structures that perform a specific function (International, 1986). (2) A method of arrangement whereby separate parts or functions work together as a unit (International, 1986).

Systemic: Pertaining to or affecting the body as a whole or acting in a portion of the body other than the site of entry, used to refer generally to non-cancer effects.

Systematic Error: A reproducible inaccuracy caused by faulty, equipment, calibration, or measuring technique (IPCS, 2000).

T

Tachypnea: Very rapid breathing.

Target Heart Rate (THR): The heart rate for an individual undertaking an exercise test that is estimated to attain a specific exercise intensity. That intensity itself is defined in a number of ways (oxygen consumption, %maximal oxygen consumption, etc.), as is the method used to convert this intensity into heart rate. There are a number of methods used to do so, but probably the most rigorous is to equate $\%VO_{2\,RES}$ that is desired with $\%HR_{RES}$ and translate that into THR (Kirham, 2008).

Target Organ Dose: The amount of a potentially toxic substance reaching the organ chiefly affected by that substance (Duffus, 2000).

Target Population: (1) The collection of individuals, items, measurements, etc., about which we want to make inferences. The term is sometimes used to indicate the population from which a sample is drawn and sometimes to denote any "reference" population about which inferences are required. (2) The group of persons for whom an intervention is planned (Last, 1983).

Temporally-Averaged Exposure: The temporally-integrated exposure divided by duration of the time interval of interest (Zartarian et al., 1997).

Temporally-Integrated Exposure: The integral of instantaneous "point" exposures over a specified time period (Zartarian et al., 1997).

Thermic Effect of Food: See "dietary induced thermogenesis."

Thorax: Part of the human body between the neck and diaphragm, partially enclosed by ribs; the chest.

Threshold: (1) The minimum amount of stimulus (concentration level) required to elicit a particular response (adapted from International, 1986). (2) The level at which a physiological or psychological effect begins to be produced (EPA, 1989).

Threshold Dose: The lowest dose level at which a specified (measurable) biological effect is observed and below which it is not observed.

Tidal Volume (V_T): The volume of air inhaled or exhaled with each breath during breathing; usually defined for a state of quiet breathing.

Time-Activity Pattern: The phrase used in the exposure measurement and modeling field for "time use data." The daily sequential pattern of activities in which an individual engages in, including: the length of time spent performing each activity, the location (microenvironment) where the activity occurs, and some type of "activity-level indicator" indicating how much energy is being expended in the activity (e.g., breathing rate, oxygen consumption, heart rate, accelerometer counts). EPA's CHAD database contains 22,968 person-days of time-activity pattern information. Frequently, these data are aggregated to the proportion of a

day spent doing activity *x* in microenvironment *y* at activity level *z*, but doing so destroys the correlated nature of human activity and exposure events.

Time-of-Life: The stage of life that a living receptor is in, which may affect its' response to dose received; an obvious example is that teratogenic effects can only occur in receptors that are in utero (CMA, n.d.).

Time Pattern/Time Profile: A continuous record of the time series of instantaneous point exposures/doses/intakes estimates for a specified time period (Zartarian et al., 1997). An example is the "time pattern of dose rate received".

Time Use Data: Data on what a subject does in time and space for a specified time period, i.e., their time-activity pattern.

Time-Weighted Average: The average of a quantity over a specified time period.

Tissue: An aggregation of cells and intercellular matter that subserves a united function (International, 1986).

Total Daily Energy Expenditure (DTEE): The total amount of energy expended by a living organism on a daily basis. It is the sum of metabolic, dietary, physical activity (work, movement, fidgeting/shivering, etc.)-related energy expenditures over some specified time period (Bar-Or & Baranowski, 1994).

Total Exposure: See "Total Human Exposure."

Total Fluid Intake: Consumption of all types of fluids including tap water, milk, soft drinks, alcoholic beverages, and water intrinsic to purchased foods (EPA, 1997).

Total Human Exposure: An exposure assessment--monitoring or modeling--that accounts for all exposures a person has to a specific substance, regardless of the environmental medium or route of entry [inhalation, ingestion, and dermal absorption]. Sometimes total exposure is used incorrectly to refer to exposure to all pollutants in an environment; total exposure to more than one pollutant should be stated explicitly as such (IPCS, 2000).

Total Suspended Particulates (TSP): Solid and liquid particles present in the atmosphere.

Total Ventilation: The total volume of air breathed in a specified time period (International, 1986).

Trachea: A cartilaginous air tube extending from the larynx into the thorax, where it divides into two branches.

Tracheobronchial Region: The area of the lungs including the trachea--windpipe--and conducting airways--bronchi, bronchioles, and terminal bronchioles (EPA, 1989).

Tracking: A person's stability over time in undertaking physical activity, often measured by ranked relative categories of exercise level (e.g., the top 25% quartile) (Anderssen et al., 2005).

Training: A regime in which people undergo a structured, often supervised, set of exercises over weeks or months (Bar-Or & Baranowski 1994).

Transfer (Media): The movement of an agent or chemical substance from one environmental media to another.

Transformation: (1) Change of chemical state, form, or structure (Dorland's, 1988). (2) The conversion--through chemical or physical processes--of one or more compounds into other compounds. These transformations may occur in many media [ambient air; water; soil; etc.].

Transport: The movement of an agent or chemical substance within a medium, either the environment [e.g., air] or within the body [e.g., blood].

T_{Total}: The time that it takes for one breathing cycle. $T_{Total} = T_I + T_E$.

Type I Error: The probability of rejecting H_o when it is true (α).

Type II Error: The probability of accepting H_o when it is false (β). $\beta = 1$ - power.

U

µ: Mu, a prefixused as "micro"; see "µ" in the M Section.

Uncertain: Indefinite; indeterminate; not certain to occur; problematical; not known beyond doubt; not clearly defined; variable (Webster's). Lack of knowledge (Bogen, 1995).

Uncertainty: (1) The quality or state of being uncertain (Webster's). (2) In cancer risk assessments, the lack of precise scientific data regarding a phenomenon, relationship, or endpoint. This lack requires that assumptions and "best" scientific judgments be used in critical portions of the risk assessment [e.g., hazard identification, dose-response relationships], resulting in a [high] degree of uncertainty regarding risk estimates (EPA,1989 (3) A probability estimate of the statistical confidence limits associated with an estimated or measured value. (4) A lack of confidence in the prediction of a risk assessment that may result from natural variability in natural processes, imperfect or incomplete knowledge, or errors in conducting an assessment (IPCS, 2000).

Uncertainty Analysis: A process in which the sources of uncertainty in an estimate are identified, and an estimate made of the magnitude and direction of the resulting error: (a) **qualitative**--utilizes descriptive methods; (b) **semi-quantitative**--uses simple mathematical techniques such as sensitivity analyses; (c) **quantitative**--uses more complex mathematical techniques such as Monte Carlo analysis (AIHA, 2000). (2) A detailed examination of the systematic and random errors of a measurement or estimate; an analytical process to provide information regarding uncertainty (SRA, 1999).

UNU: United Nations University.

Upper Bound: A plausible upper limit to the "true value" of a quantity; it usually is not a true statistical confidence limit (IRIS, 1999).

Upper Respiratory Tract: The structures that conduct air into the lungs, including the nasal cavity, mouth, pharynx, and larynx (EPA, 1989).

Uptake: (1) Absorption and assimilation of a substance by an organ or tissue (International, 1986). (2) Process by which a chemical [substance] crosses an absorption boundary and is absorbed (EPA, 1992).

V

Valid, Validity: (1) Supported by objective truth or accepted authority; sound and sufficient. A test or experimental procedure that measures what it purports to: it is sufficient. (Last, 1983 via IPCS, 2000). (2) Validity has two components: relevance and reliability; objectivity is a component of reliability (Safrit & Wood, 1989). (3) There are 4 aspects of validity (Baumgartner & Jackson, 1999):

> Logical validity: the instrument measures the capacities that it is intended to measure.
>
> Concurrent validity: a measure of an instrument's correlation with a specified criterion (generally using **r**).
>
> Predictive validity: the value of an instrument to predict its performance on a criterion measure.
>
> Construct validity: used in an abstract sense; the instrument measures what is desired, but that cannot be directly measured itself but can be addressed statistically via hypothesis tests.

Other authors break down essentially the same concepts but use different words; Morrow et al. (2000), for instance, distinguishes among "content-related," "criterion-related," and construct-related validity.

Variable: Any quantity that varies, taking on different numerical values (Last, 1983).

Variance: An indicator of the variability inherent in a set of observations—a sample—equal to the sum of squared deviations from the mean divided by the degrees of freedom in the sample (IPCS, 2000).

Variation: A divergence in a developing organism from the usual or normal range of structural constitution that may not adversely affect organ health or survival (EPA, 1989).

Variability: Heterogeneity in a population parameter or variable.

Vascular: Pertaining to blood vessels.

VCO_2: CO_2 production during respiration [in mL min^{-1}].

Ventilation: In respiratory physiology, the process of gaseous exchange between the blood and environment via the lungs (International, 1986). See "Pulmonary Ventilation," "Alveolar Ventilation," "Total Ventilation," "Minute Ventilation," "Respiration," and "Expired Ventilation."

In indoor air pollution, the exchange of air in a room or structure with ambient ["fresh"] air [or air from another room or structure]; in a general sense, it also means the circulation of air.

Ventilation, Dead Space (V_D): Ventilation per minute of the physiologic dead space [volume of gas not involved in gas exchange with the blood], at body temperature and pressure, saturated conditions. It is defined by the following equation:

$$V_D(PaCO_2 - P_ECO_2)/(PaCO_2 - P_ICO_2)$$

Ventilation Perfusion Ratio (V_A/Q): Ratio of the alveolar ventilation rate to the blood perfusion volume flow through the pulmonary parenchyma, such as pulmonary blood flow or right heart cardia output; this ratio is a fundamental determinant of the oxygen and carbon dioxide pressure of the alveoli gas and of the end-capillary blood. Throughout the lungs, the local ventilation/perfusion ratios vary, and, consequently, the local alveolar gas and end-capillary blood compositions also vary (EPA, 1993).

Ventilation Rate (V_E): The" breathing rate" (in L/min) needed to support oxygen consumed for a particular activity. It actually is defined to be the breathing rate (f_R) times Tidal Volume (V_T).

> $V_{E.MAX}$: Maximum V_E for a person undergoing a strenuous (for them) exercise protocol [in L min^{-1}].

Ventilatory Anaerobic Threshold (VAT): A point in an incremental exercise test where V_E increases out of proportion to VO_2. It also is known as the ventilatory threshold [V_T]. See Hebestreit et al. (2000). There are a number of different indicators of VAT now used; it is a marker of physiological fitness. It often is defined to be the point on the VO_2"curve" where VQ (V_E/VO_2), R, and $PETO_2$ increase while VO_2/VCO_2 decreases or remains constant (Hansen et al. [1984]). McArdle et al. (2001) state: "the term ventilator threshold (VT) describes the point at which pulmonary ventilation increases disproportionally with oxygen consumption during graded exercise" [p. 291]. At this exercise intensity, pulmonary ventilation on longer links tightly to oxygen demand at the cellular level. It often is defined to be identical to the lactate threshold and the anaerobic threshold per se. There is no universal method of estimating VAT; three different methods are often used, and they provide similar—but not exact –estimates, within 7% of one another or less, around 71% of $VO_{2.MAX}$ (Fleg et al., 2000). However, other cardiologists state VAT in healthy individuals is approximately 40-60% of $VO_{2.MAX}$, and in trained endurance athletes it can be as high as 80% (Mezzani et al., 2009).

Ventilatory Equivalent: The ratio of minute ventilation (MV) to oxygen consumption, defined as V_E/VO_2 [V_E VO_2^{-1}]. This ratio in healthy people is on the order of 20-32 [L min^{-1}/L min^{-1}--unitless] at moderate exercise levels (McArdle, et al., 1991). It is higher at more extreme exercise levels, and values in the 40's are possible for short periods of time <5 min (Åstrand & Rodahl, 1986). High values are a marker of inefficient ventilation due to hyperventilation, increased dead space, and/or the "oxygen cost of breathing." Subject with heart failure or other problems have a high VQ (Luks et al., 2012).

Ventilatory Reserve (VR): The difference between the maximum minute ventilation reached by a subject at peak exercise ($V_{E.MAX}$) and her or his maximum voluntary ventilation (MVV); it is also known as the breathing reserve (Luks et al., 2012). V_E, for instance, is only 60-85% of MVV at $VO_{2.MAX}$ (McArdle et al., 2001).

Ventilatory Scope: The ratio of $V_{E.MAX}$ to $V_{E.BASAL}$. It is approximately equal to $V_{E.MAX}$ /$V_{E.REST}$ (Rowland, 1989).

Ventilatory Threshold: The point in a progressive exercise test where lactic acidosis begins to develop; it is also known as the "anaerobic threshold" (Luks et al, 2012) and "ventilatory anaerobic threshold." It is about 2/3 of the way through a good maximal effort, and minute ventilation increases at a higher rate than VO_2 at that point (Luks et al., 2012). It approximates—but is not identical to--the lactate threshold [see]. It also known as the anaerobic threshold (Barstow & Mole, 1991).

Vigorous Physical Activity (VPA): A phrase with many meanings, in that the energy expenditure levels associated with it are defined in highly variable ways (heart rate, % of heart rate reserve, VO_2 consumption, and % of VO_2 reserve, % of $VO_{2\ Peak}$, METS, etc.). Most of the definitions are laboratory or investigator-dependent, with little attempt at standardization of the metrics used. Perhaps the most rigorous definition might be to the lactate threshold, the ventilator threshold, etc. McCurdy & Graham (2004) defined it to be the highest third of the VO_2 reserve distribution.

VO_2: Oxygen uptake (a rate) during respiration [in mL min^{-1}]. $VO_2 = V_I * \%O_2$, where $\%O_2 = 20.93\%$, by definition, in the normal case. VO_2, as a volume—not used in this report—is the amount of oxygen consumed in a fixed time period.

$VO_{2\ MAX}$: Maximal VO_2 needed to complete a specified physical activity [in L min^{-1}]. Also known as the maximal aerobic capacity, maximal O_2 consumption, and maximal O_2 intake. There are many functional definitions of $VO_{2\ .MAX}$. Typical definitions are: (a) the amount of O_2 consumption associated with a plateau in the VO_2 uptake [in L kg^{-1} min^{-1} or mL min^{-1}] curve as workload (exercise) is increased, (b) "the point at which VO_2 shows no further increase [or increases only slightly] with additional workload," and (c) a quantitative measure of the person's maximum capacity for the aerobic resynthesis of ATP (McArdle et al., 1991). Because of problems with defining the term and investigator-specific protocols used to determine $VO_{2.MAX}$, many exercise physiologists are now using the "peak" term $VO_{2.PEAK}$ instead of the time-honored $VO_{2.MAX}$.

1. The VO_2 measure that is associated with a RER >1.0 (Whaley et al., 1995).
2. The highest VO_2 that is observed during the final minute of a stress test at voluntary exertion, with a HR>90% of the age-predicted maximum, and a RER>1.0 (Jackson et al., 1995). The terms often are used interchangeably, although some authors state that $VO_{2.PEAK}$ is lower than $VO_{2.MAX}$ due to a more liberal allowance of test cessation of the fitness test before criteria for $VO_{2.MAX}$ has been reached (Cowan et al., 2009).

VO_2 Reserve ($VO_{2.RES}$): The difference (in consistent units) between $VO_{2.MAX}$ and $VO_{2.REST}$, which itself is VO_2 at "basal" (or resting) metabolic conditions.

W

W_{170}: Work accomplished at a heart rate of 170 beats per minute.

Weir's Equation: The formula used to estimate energy expenditure (EE in kcal/min) from measures of pulmonary ventilation and expired oxygen percentage, developed in 1949 by J.B. Weir. It is accurate to within ± 1% of the traditional Respiratory Quotient (RQ) method (McArdle et al., 2001). The formula actually assumes that protein breakdown accounts for a fixed 12.5% of energy produced by a person, which is a reasonable assumption (but rather inflexible). Observations of relative protein consumption from around the world indicates that it accounts for 10-14% by weight (Weir, 1949). Weir's basic equation is:

$$EE = V_{E\ (STPD)} * (1.044 - [0.0499 * \%O_{2.E}])$$

$V_{E\ (STPD)}$= Expired ventilation rate in L/min

$\%O_{2.E}$ = Oxygen percentage in the expired air (the remainder generally is CO_2)

The term in parentheses is called the "Weir Factor" and lookup tables exist that to easily convert it to a dimensionless value for various values of $\%O_{2.E}$. See p. 184 of McArdle et al. (2001).

Weir also developed another equation that estimates EE in kcal/min from respiratory quotient (RQ) [see] and oxygen consumption (VO_2) observations (McArdle et al., 2001). It is:

$$EE = ([1.1 * RQ] + 3.9) * VO_2$$

RQ is dimensionless

VO_2 has units of L/min (usually its units are mL/min).

WHO: World Health Organization.

Work: (1) The transfer of energy from one physical system to another, especially by the application of force. (2) Physical or mental effort or activity expended to accomplish a task. (3) The product of a force and distance through space that a force is applied. (4) Force expressed through a distance, but with no limitation on time. Work is **not** synonymous with muscular exercise (McArdle et al., 1991).

Workload: The amount or intensity of work expended in a specified time period.

X

Xenobiotic: (1) A chemical foreign to the biologic system (Dorland's, 1988). (2) Not occurring in nature, used especially of certain synthetic chemical compounds that do not biodegrade readily (International, 1986). (3) A foreign compound that is metabolized in the body (Last, 1983).

E-3. Table of Conversion Factors Used in this Synthesis

"Basis"	Chosen Factor	Citation	Alternatives	Citations
1 L $O_{2=}$	4.85 kcal	Erb (1981)	4.69 - 5.01	Stegemann (1981)
			4.69 - 5.05	Freedson & Goodman (1993)
			4.71	Daly et al. (1985)
			4.74-4.95	Cotes (1975)
			4.78-4.94	Solomon et al. (1982)
			4.825	Leger et al. (1980); Sinclair (1971)
			4.83	Brown (1973)
			4.87	Schulz et al. (1989)
			4.8735	Park et al. (2008)
			4.90	Christensen et al. (1983); Croonen & Binkhorst (1974)
			4.84	Weir equation direct, using a RQ=0.855
			4.86	McArdle et al. (2001); RQ=0.85
			4.69	As above; RQ=0.71
			5.05	As above; RQ=1.00
1 L $O_{2=}$	20.5 kJ	Emons et al. 1992)	20.19	Lovelady et al. (1993); McCrory et al. (1997)
			20.35	Brage et al. (2004)
			20.92	Cunningham et al. (1981)
1 kcal	4.184 kJ	Handbook of Physics[1]	4.175	Brun et al. (1985)
			4.186	Åstrand & Rodahl (1986); Diem & Lentner (1970)
			4.192	Lee & Paffenbarger (2000)
1 kcal	210 mL O_2		200	Females; range: 190-210
			210	Males; range: 200-220
			206	Reciprocal of Erb (1981)
1 kJ	0.2389 kcal	Montoye (1975)	0.239	Montoye et al. (1996)
			0.2395	Brun et al. (1985)
1 MJ	239 kcal	Durnin (1987)		
1 MJ/d	0.6944 kJ/min	Using Durnin (1987)		
	694.44 J/min	Using Durnin (1987)		
	41.667 kJ/h	Using Durnin (1987)		
1 MJ/d	9.958 kcal/h	Using Durnin (1987)		
	0.166 cal/min	Using Durnin (1987)		

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