

Issue Paper on Physiological and Behavioral Changes in Pregnant and Lactating Women and Available Exposure Factors



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**Issue Paper on Physiological and Behavioral Changes
in Pregnant and Lactating Women and
Available Exposure Factors**

National Center for Environmental Assessment
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LIST OF ABBREVIATIONS AND ACRONYMS

AAP	American Academy of Pediatrics
ACOG	American College of Obstetricians and Gynecologists
ACTH	Adrenocorticotrophic hormone
ATSDR	Agency for Toxic Substances and Disease Registry
BMD	bone mineral density
BMI	body mass index
BMR	basal metabolic rate
CDC	Centers for Disease Control and Prevention
CHAPS	Canadian Human Activity Pattern Study
CI	confidence interval
CO	cardiac output
CrCl	creatinine clearance rate
CRH	corticotropin releasing hormone
CSFII	Continuing Survey of Intake by Individuals
DBP	diastolic blood pressure
EPA	U.S. Environmental Protection Agency
eRPF	effective renal plasma flow
ERV	expiratory reserve volume
FDA	U.S. Food and Drug Administration
FRC	functional residual capacity
GFR	glomerular filtration rate
GPx	glutathione peroxidase
hCG	human chorionic gonadotropin
HDL	high-density lipoprotein
HPA	hypothalamic-pituitary-adrenal axis
hPL	human placental lactogen
HR	heart rate
IC	inspiratory capacity
IOM	Institute of Medicine
IRV	inspiratory reserve volume
LDL	low-density lipoproteins
NCEA	National Center for Environmental Assessment
NHANES	National Health and Nutrition Examination Survey
NIH	National Institutes of Health

NIHCM	National Institute for Health Care Management
NSDUH	National Survey on Drug Use and Health
ORD	Office of Research and Development
PDIR	physiological daily inhalation rate
RDA	recommended daily allowance
RR	respiratory rate
RV	residual volume
SAMHSA	Substance Abuse and Mental Health Services Administration
SBP	systolic blood pressure
SD	standard deviation
SE	standard error
SIP	Share of Intake Panel
T3	triiodothyronine
T4	thyroxine
TBG	thyroid-binding globulin
TLC	total lung capacity
TSH	thyroid-stimulating hormone
TT3	total triiodothyronine
USDA	U.S. Department of Agriculture
USDHHS	U.S. Department of Health and Human Services
VC	vital capacity
VLDL	very-low-density lipoproteins
WHO	World Health Organization
WIC	Women, Infant, and Children

PREFACE

The Exposure Factors Program of the U.S. Environmental Protection Agency's (EPA's) Office of Research and Development (ORD) has three main goals: (1) provide updates to the *Exposure Factors Handbook* ([U.S. EPA, 2011](#)) and the *Child-Specific Exposure Factors Handbook* ([U.S. EPA, 2008](#)); (2) identify exposure factors data gaps and needs in consultation with clients; and (3) develop companion documents to assist clients in the use of exposure factors data. The activities under each goal are supported by and respond to the needs of the various EPA program offices and others. This issue paper provides summaries of physiological and behavioral changes cited in published literature (through December 2013) that may impact a woman's exposure or susceptibility to environmental contaminants during periods of pregnancy and lactation. Additionally, more recent targeted searches of the literature have been conducted to supplement this paper in response to peer-review comments. This paper also summarizes available exposure factors that may be used in an exposure assessment specific to pregnant and lactating women and current data gaps.

AUTHORS, CONTRIBUTORS, AND REVIEWERS

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EXECUTIVE SUMMARY

This report summarizes published literature on the physiological and behavioral changes that occur during pregnancy and subsequent lactation period. It also covers related exposure factors and includes relevant literature published through December 2013. Exposure factors are factors related to human behavior and characteristics that help determine an individual's exposure to an agent ([U.S. EPA, 2011](#)). More recent targeted searches of the literature have also been conducted to supplement the report in response to peer-review comments.

This issue paper addresses pregnancy- and lactation-related physical and behavioral changes that may affect a pregnant or lactating woman's exposure or susceptibility to environmental contaminants. The female body undergoes a variety of physiological changes to nurture the fetus and to produce milk for breastfeeding postpartum. These changes can affect a woman's body systems and behaviors in ways that differentiate her from women in the general population. Thus, pregnancy and lactation are unique lifestages in which women are potentially vulnerable to different environmental exposures. Although EPA recognizes the potential vulnerabilities of the fetus from maternal exposures, the focus of this issue paper is on the exposures to the pregnant or lactating woman.

Specific risk factors and their possible association with pregnancy outcomes or interventions are not discussed. Likewise, any potential effects that physiological or behavioral changes can have on the pregnant or lactating woman, the fetus, the infant, or later in life among individuals exposed in utero or via breastfeeding, are not the focus of this issue paper. Whenever possible, this issue paper links physiological and behavioral changes during pregnancy and lactation with the potential for experiencing differential exposures by this population. However, some of the associations between physiological or behavioral changes and exposures are not known and therefore not presented.

This issue paper is organized into four major sections: (1) physiological changes, (2) behavioral adaptations and psychological changes, (3) exposure factors, and (4) data gaps. The section on physiological factors is organized by organ system. Behavioral changes include both voluntary adaptations, such as eliminating or reducing the use of caffeine or alcohol, and psychological changes that may affect behavior (such as depression) that can occur as a result of pregnancy. The exposure factors section is organized by the various factors (e.g., water intake, dietary intake, nondietary intake). The data gaps section summarizes areas where information is limited or lacking.

Physiological changes occurring during pregnancy and lactation are summarized in Tables ES-1 and ES-2, respectively, according to organ system. They include changes in the cardiovascular, respiratory, renal, skeletal, digestive, endocrine, and integumentary systems,

where available. Some of the physiological changes during pregnancy and lactation may alter the internal dose and toxic response from environmental contaminants, resulting in health risks that are different from those of the general population. For example, the increased blood plasma volume and protein binding that occur during pregnancy can affect the volume distribution of chemicals in the pregnant woman's body ([Anderson, 2005](#)). Hormonal changes during pregnancy and lactation can affect the woman's appetite and food intake ([Gabbe et al., 2007](#)).

Table ES-1. Physiological changes during pregnancy by organ system

Measurement	Definition	Change in pregnancy
Cardiovascular system		
Cardiac output (CO)	Volume of blood being pumped by the heart per unit of time.	Increased 18–33%
Total blood volume	Combination of plasma and red blood cells.	Increased 30–45%
Oxygen consumption	The amount of oxygen consumed by the tissues of the body.	Increased 20–40% and up to 60% during labor
Heart rate	The number of heart beats per unit of time.	Increased 10–20% by week 32 of gestation
Respiratory system		
Respiratory rate (RR)	Number of breaths per minute.	Unchanged
Vital capacity (VC)	Maximum amount of air that can be forcibly expired after maximum inspiration (IC + ERV).	Unchanged
Inspiratory capacity (IC)	Maximum amount of air that can be inspired from resting expiratory level (TV + IRV).	Increased 5–10%
Tidal volume (TV)	Amount of air inspired and expired with normal breath.	Increased 30–40%
Inspiratory reserve volume (IRV)	Maximum amount of air that can be inspired at end of normal inspiration.	Unchanged
Functional residual capacity (FRC)	Amount of air in lungs at resting expiratory level (ERV + RV).	Decreased 20%
Expiratory reserve volume (ERV)	Maximum amount of air that can be expired from resting expiratory level.	Decreased 15–20%
Residual volume (RV)	Amount of air in lungs after maximum expiration.	Decreased 20–25%
Total lung capacity (TLC)	Total amount of air in lungs at maximal inspiration (VC + RV).	Decreased 5%

Table ES-1. Physiological changes during pregnancy by organ system (continued)

Measurement	Definition	Change in pregnancy
Renal system		
Glomerular filtration rates (GFR)	Flow rate of filtered fluid through the kidney.	Increased 19–40%
Creatinine clearance rate (CrCl)	Volume of blood plasma cleared of creatinine per unit time.	Increased 26–58%
Effective renal plasma flow (eRPF)	Amount of plasma flowing to the parts of the kidney that function in the production of urine.	Increased 31–50%
Skeletal system		
Bone mineral density (BMD)	The amount of minerals, such as calcium, per square centimeter of bones.	Reversible bone loss during pregnancy and lactation
Bone turnover	The continuous process of removal and replacement of bone.	Decreased during the third trimester and lactation
Neurological system		
Adrenocorticotrophic hormone (ACTH)	Produced by the pituitary gland and its key function is to stimulate the production and release of cortisol.	Increased 89% from the 7 th to the 37 th week gestation
Cortisol	Produced by the adrenal cortex. Becomes elevated in response to physical or psychological stress.	Increased 141%
Beta-endorphins	Produced by the pituitary gland in response to pain, trauma, exercise, or other forms of stress.	Decreased during pregnancy and increased during labor
Digestive/Gastrointestinal system		
Leptin	Hormone produced by fat cells and affects body weight regulation by suppressing appetite and burning fat stored in adipose tissue.	Increased from the second to the third trimester
Intestinal tone and motility	Relaxation of the muscles and transit time.	Decreased

**Table ES-1. Physiological changes during pregnancy by organ system
(continued)**

Measurement	Definition	Change in pregnancy
Digestive/Gastrointestinal system		
Appetite	Desire to satisfy the body's need for food.	Appetite may increase or decrease; generally there is an increase in caloric intake by 200 kcal/day.
Endocrine system		
Placenta volume	Volume of the temporary organ that forms in the lining of the uterus and provides nourishment to the fetus.	Increased volume with gestation from 134 ± 58 mL at 14 weeks to 659 ± 103 mL at 40 weeks
Thyroid function	Function of a gland in the neck that secretes hormones that regulate growth and metabolism. Hormones include total thyroxine (T4); total triiodothyronine (TT3); thyroid-binding globulin (TBG); thyroid-stimulating hormone (TSH).	T4 and TT3 increased during the first trimester peaking at mid gestation; TBG increased during the first trimesters and peaks at 12 to 14 weeks gestation; TSH decreased temporarily during the first trimester and remains stable through the third trimester.
Hypothalamic-Pituitary-Adrenal Axis (HPA)	Three glands of the endocrine system (i.e., pituitary, thyroid, and adrenal) that regulate body processes including energy storage and expenditures.	Increased estrogen, aldosterone, deoxycorticosterone, corticosteroid-binding globulin, cortisol (2.5 times higher than nonpregnant women), and free cortisol, testosterone, androstenedione, prolactin (10 times higher at term); decreased dehydroepiandrosterone, dehydroepiandrosterone-sulfate, follicle-stimulating hormone, luteinizing hormone, growth hormone; pituitary gland increase in size by 36% at term.

Table ES-1. Physiological changes during pregnancy by organ system (continued)

Measurement	Definition	Change in pregnancy
Endocrine system		
Glucose and carbohydrate metabolism	The process by which sugars and carbohydrates are used in the body to produce energy.	Decreased glucose levels of 10% in early pregnancy; increased insulin as well as insulin resistance; 50–80% reduction in insulin sensitivity by the third trimester.
Protein and lipid metabolism	The decomposition and synthesis of protein and lipids in the body.	Decreased protein catabolism; increased generation of glycerol, fatty acids, and ketones; increased total cholesterol (50–60%), low density lipoprotein (LDL) (50–60%), high density lipoprotein (HDL), very low density lipoproteins (VLDLS) and triglycerides; increased alpha- and gamma-tocopherols, lycopene, and beta-carotene; decreased retinol; increased total saturated fatty acids; unchanged <i>n</i> -9 fatty acids; decreased <i>n</i> -6 fatty acids.
Metabolic adjustments	Adaptive changes in the body's metabolism.	Increased energy expenditures; preferential use of carbohydrates; basal metabolic rate, sleeping metabolic rate, and minimal sleeping metabolic rate is 15–26% higher during pregnancy.
Total water metabolism	Changes in the water content in the human body.	Increased 45%
Integumentary system		
Surface area	Body surface area calculated as a function of height and weight.	Increased

Sources: [Abduljalil et al. \(2012\)](#); [Gabbe et al. \(2007\)](#); [Crapo \(1996\)](#).

Table ES-2. Physiological changes during lactation by organ system

Measurement	Change during lactation
Cardiovascular system	
Systolic blood pressure, diastolic blood pressure, and heart rate	Lower in lactating than nonlactating women
Endocrine system	
Estrogen and progesterone	Decrease levels of estrogens and progesterone that result in the onset of milk production
Parathyroid	Increase of parathyroid hormone during lactation to meet calcium demands
Prolactin	Increase of prolactin, which plays a vital role in the initiation and maintenance of lactation. It remains elevated throughout the first 12 months postpartum
Oxytocin	Oxytocin levels increase to stimulate let down of milk; low oxytocin levels are associated with mood disorders postpartum
Metabolic adjustments	Recommended increase of caloric intake by 500 kcal/day for the first 6 months of lactation and 400 kcal/day after the sixth month
Energy expenditures and sleeping metabolic rates	Levels are higher in lactating and nonlactating women
Skeletal system	
Bone turnover	Reversible bone loss to provide adequate calcium for milk production
BMD	Temporary decrease between 3–9%
Neurological system	
ACTH and cortisol	Levels are lower in lactating than nonlactating women when responding to stress
Sleep	Altered sleeping patterns to accommodate lactation schedule

Sources: [Picciano \(2003\)](#); [Stuebe et al. \(2012\)](#); [Blyton et al. \(2002\)](#); [Groer et al. \(2013\)](#); [Butte et al. \(1999\)](#).

Assessing exposures to pregnant and lactating women requires information about exposure factors for this potentially susceptible lifestage. These include water and food intake, inhalation rates, nondietary ingestion rates, time spent at various locations and activities, consumer products use, and body weight.

In general, lactating women ingest more water than pregnant women, nonpregnant women, and nonlactating women ([Forssen et al., 2009](#); [Kahn and Stralka, 2008](#); [Zender et al., 2001](#); [Ershow et al., 1991](#)). An EPA analysis of data from the 2003–2008 National Health and Nutrition Examination Survey (NHANES) found that differences in per capita food intake rates for pregnant women were statistically significant from those of nonpregnant women for some of the major food groups including: total fruits, vegetables, dairy, and grain and several individual food categories (i.e., banana, cabbage, citrus, corn, cucurbits, leafy vegetables, peaches, root and tuber vegetables, stalk and stem vegetables, stone fruits, tropic fruits, and white potatoes) ([Sarkar and Nguyen, 2013](#)) (see Appendix). On a per capita basis, there was an increase in consumption of total fruits, vegetables, dairy, and grain of 69, 13, 43, and 12%, respectively. There was also a statistically significant increase in consumption of the individual foods except for cabbage, leafy vegetables, and stalk and stem vegetables.

Some pregnant women experience cravings for nonfood substances. This behavior, known as pica, is characterized by the intentional ingestion of materials such as dirt, clay, cigarette ashes, ice, freezer frost, flour, baking soda or powder, cornstarch, powdered milk, and other materials ([Cooksey, 1995](#); [Bronstein and Dollar, 1974](#)). Pregnant and lactating women who engage in this behavior may be exposed to environmental contaminants present in soil and other materials. Data on nondietary intake among pregnant women are very limited and have focused on specific minority populations, the incidence of the behavior, and types of materials ingested, but very few authors have reported on the amounts consumed. Among the studies reporting the amounts of materials consumed (mostly clay or “dirt”), ingestion rates ranged from 1–1,650 g/day ([Kutalek et al., 2010](#); [Klitzman et al., 2002](#); [Smulian et al., 1995](#); [Ferguson and Keaton, 1950](#)). [Gavrelis et al. \(2011\)](#) found that the prevalence of pica behavior among pregnant women was twice that of nonpregnant women. There are no data on the prevalence of the behavior or on the amount ingested among lactating women.

Daily inhalation rates for normal-weight women are approximately 18–41% higher during pregnancy and 23–39% during postpartum ([Brochu et al., 2006](#)). Data on activity factors and use of consumer products are limited for pregnant and lactating women. Furthermore, the studies on activity patterns and consumer products have focused on specific geographical locations or minority populations and were based on small sample sizes. On the other hand, information on body weight gained during pregnancy and lost during the postpartum period is generally more readily available. The U.S. national mean body weight of pregnant women

averaged over the three trimesters is 75 kg. The average body weight from the same data set for all women is 71 kg ([U.S. EPA, 2011](#)). Studies during the postpartum period relate to the effects of lactation on body weight changes.

Although data are available on physiological and behavioral changes as a result of pregnancy or lactation, the direct link between these changes and the potential for experiencing differential exposures is not well understood and is a significant data gap. Most of the physiological information found relates to pregnancy, while the information on lactating women is more limited. Exposure factors data for pregnant and lactating women are also somewhat limited. Some of the studies were conducted on a small scale or on a certain geographical area or ethnic/socioeconomic group, and may not be generalizable to other pregnant and lactating women. Exposure factors for which data are available include: water and food intake (for pregnant women only), inhalation rates, and body weight. There are no data with regard to food intake by lactating women. Information regarding activity patterns and the frequency and use of consumer products is an area in which research is needed. In addition, the role that race, age, ethnicity, geographical location, and socioeconomic factors plays in the variability with regard to these exposure factors for this lifestage is not well understood. More importantly, additional analyses to understand whether the differences between exposure factors for pregnant and lactating women and those of the general population of women are significant in terms of exposure and risk have not been conducted.

The information summarized in this issue paper was obtained from various sources and presents the findings of the individual study authors. It is not intended to provide an exhaustive review of all possible physiological and behavioral changes that occur during pregnancy and lactation. Instead, it provides an introduction to the topic of pregnancy- and lactation-related changes and related exposure factors, potentially serves as a precursor to investigations into how these changes may alter environmental exposures for this potentially susceptible lifestage, and may inspire research in areas identified as data gaps.

1. INTRODUCTION

1.1. BACKGROUND

Physiological and behavioral changes occur in women during pregnancy and lactation, and these changes can lead to different environmental exposures than for those in the general population. A number of metabolic functions are altered to provide for the demands of the fetus. For instance, changes in metabolic rates cause an increase in nutritional demands during pregnancy and lactation, which may result in corresponding increases in exposures. Differences in exposure may also result from differences in food choices. Behavioral adaptations during pregnancy and lactation may also affect environmental exposures. For example, pregnant and lactating women may eliminate their use of coffee, cigarettes, and alcohol, thereby reducing their exposure to chemicals in these products.

Tables 1-1 through 1-3 provide lists of the various changes that may occur during each trimester of pregnancy, as presented in [Bonillas and Feehan \(2008\)](#). The tables illustrate the wide variety of physiological changes that occur throughout pregnancy. Not all of the changes listed in Tables 1-1 through 1-3 are relevant to potential differences in environmental exposures in this lifestage compared to nonpregnant women, and are not discussed in detail in this issue paper. There are, however, a number of changes that are potentially significant with regard to exposures or that may increase or decrease susceptibility among this population, and these changes are discussed in further detail in this issue paper.

Table 1-1. Changes during the first trimester (weeks 1–12)

Change	Explanation for change
Missed period	Hormones secreted by the blastocyst (after burrowing into the endometrial lining) take control of the menstrual cycle.
Nausea and vomiting	Due to rapidly increasing levels of the hormone, human chorionic gonadotropin (hCG); nausea tends to peak around the same time as levels of hCG.
Sensitivity to odors	Due to high levels of the hormone estrogen.
Fatigue	Occurs due to higher levels of the hormone progesterone, allowing the body to focus its energy on sustaining the pregnancy.
Breast enlargement	Due to increased levels of estrogen, the mammary glands begin to enlarge in preparation for breastfeeding.
Breast tenderness	The enlargement of the mammary glands causes the breasts to become tender.
Darkening of the areola	The pigmented areas around each breast's nipple darken due to increased levels of progesterone and estrogen (this is believed to help the newborn find the breast at birth).
Areola increases in size	Due to increased hormone levels (and believed to help the newborn find the breast at birth).
Mood swings	Partly due to surges in hormones; characterized by change in emotional stability and irritability.
Expanding uterus (womb)	The placenta produces progesterone, which relaxes the muscles of the uterus so they can stretch as the pregnancy progresses.

Source: [Bonillas and Feehan \(2008\)](#).

Table 1-2. Changes during the second trimester (weeks 13–27)

Change	Explanation for change
Slower digestion	High levels of progesterone slow down the contractions of the esophagus and intestine, thus slowing down digestion.
Constipation	Due to a slower digestion.
Hemorrhoids	Due to constipation, increased blood volume and vascular congestion.
Heartburn	The placenta produces progesterone, which relaxes the valve that separates the esophagus from the stomach, allowing gastric acids to seep back up, causing an unpleasant burning sensation.
Backaches	Due to the expanding uterus affecting posture.
Pinching of sciatic nerve	Nerve in the hip/buttock area gets pinched because of pressure exerted on it by the expanding uterus.
Facial skin changes	Dark patches appear on the face due to hormonal changes.
Increased frequency in urination	Due to increased blood flow to the kidneys and pressure from the weight of the pregnancy on the bladder.
Edema	Swelling of the ankles, hands, and face, due to fluid retention.
Expanding uterus	Due to progesterone, which in turn, relaxes the muscles of the uterus so they can stretch as the pregnancy progresses.
Abdominal enlargement	Due to the progression of the pregnancy, the uterus expands into the abdominal cavity.
Increase in blood volume	Due to the need for extra blood flow to the uterus.
Heart growth	Due to the body needing to supply more blood for the growing fetus and placenta.
Quickening	Feeling fetal movements for the first time.
Stretch marks	Due to the expanding abdomen, breasts, legs, buttocks.
Sweating	Due to hormonal changes, increased effort on physical activities due to the expanding uterus, and the fetus beginning to radiate body heat.
Difficulty in sleeping	Due to fetal movements or frequent urination at night.
Leukorrhea	Higher levels of estrogen increase blood flow to the vagina, which, in turn, increases the release of a white-colored, odorless vaginal discharge (sign of a healthy vagina).
Hair growth	Due to hormone stimulation of hair follicles on the head, arms, legs, and face.

Table 1-2. Changes during the second trimester (weeks 13–27) (continued)

Change	Explanation for change
Dry, itchy skin	Particularly on the abdomen as the skin continues to grow and stretch due to the expanding uterus.
“Linea nigra”	A dark line running from the pubic bone up the center of the abdomen to the ribs, which is caused by the increase in hormones.

Source: [Bonillas and Feehan \(2008\)](#).

Table 1-3. Changes during the third trimester (weeks 28–40)

Change	Explanation for change
Heart rotates	Takes place to make room for expanding uterus, which pushes other organs up as well.
Varicose veins	Swollen/bluish veins that bulge near the surface of the skin, usually behind the legs. As the uterus grows, it puts pressure on the large vein on the right side of the body, which in turn, increases pressure on the veins in the legs, making the veins swell from the extra pressure to return the blood from the extremities to the heart (as they work against gravity).
Heartburn	The growing fetus crowds the abdominal cavity, pushing the stomach acids back up into the esophagus.
Hemorrhoids	Due to constipation.
Leg cramps	Believed to be due to lack of calcium in the body.
Shortness of breath	Due to the expanding uterus pushing up against the diaphragm.
Braxton-Hicks contractions	Usually painless uterine contractions that help the uterus prepare for birth.
Increased frequency in urination	Due to increased blood flow to the kidneys and pressure from the weight of the pregnancy on the bladder.
Stretch marks	Due to the expanding abdomen, breasts, thighs, and buttocks.
Dry, itchy skin	Particularly on the abdomen as the skin continues to grow and stretch due to the expanding uterus.
Naval protrusion (bellybutton sticking out)	Due to the expanding abdominal cavity.
Colostrum	Yellow, watery fluid produced by the mammary glands. Colostrum contains large amounts of antibodies that help protect the mucous membranes in the throat, lungs, and intestines of the infant. White blood cells are also present in large numbers and begin protecting the infant from harmful bacteria and viruses. Beneficial bacteria are also established in the digestive tract of an infant when colostrum is ingested.
Estrogen	A pregnant woman will have more estrogen in her body during the 9 months of pregnancy than a woman who never gets pregnant will have in her entire lifetime.
Progesterone	By the end of the pregnancy, levels of this hormone will increase seven times its normal levels during pregnancy.

Source: [Bonillas and Feehan \(2008\)](#).

1.2. OBJECTIVES AND SCOPE

The primary purpose of this issue paper is to provide a summary of information from the published literature related to physiological and behavioral changes during pregnancy and lactation that may alter a woman's exposure or susceptibility to environmental contaminants. Available data on exposure factors for this lifestage and current data gaps are also summarized. Exposure factors are factors related to human behavior and characteristics that help determine an individual's exposure to an agent (e.g., water intake, food intake, inhalation rates) ([U.S. EPA, 2011](#)).

The scope of this issue paper is not on chemical-specific exposures or risk factors and their possible association with pregnancy outcomes or interventions. Therefore, potential effects that physiological or behavioral changes can have on the pregnant or lactating woman, the fetus, the infant, or later in life among individuals exposed in utero or via breastfeeding are not discussed. Not all women will experience pregnancy symptoms in the same way, and some physiological and behavioral changes may impact different women to differing degrees. For example, some women develop gestational diabetes, depression, or nausea but others do not. Other maternal factors such as gravidity, parity (number of previous childbirths), and having had previous adverse reproductive outcomes may be related to certain behavioral and physiologic changes.

Although other aspects of vulnerability may affect a pregnant/lactating woman's response to environmental exposures (e.g., access to health care, chronic health conditions), these are not the focus of this issue paper. The term vulnerability here refers to differences in risk resulting from the combination of both intrinsic differences in susceptibility and extrinsic social stress factors (e.g., low socioeconomic status, crime and violence, lack of community resources, crowding, access to health care, education, poverty, segregation, geography, etc.). Susceptibility refers to differences in risk resulting from variation in both toxicity response (sensitivity) and exposure (as a result of gender, lifestage, and behavior). The term sensitivity refers to differences in toxic response resulting from toxicodynamics differences and/or toxicokinetics differences. These differences can arise due to numerous biological factors such as lifestage (windows of enhanced sensitivity), genetic polymorphisms, gender, disease status, nutritional status, etc.

In some cases, this issue paper links physiological and behavioral changes during pregnancy and lactation with the potential for experiencing differential exposures by this population. Some associations between physiological or behavioral changes and exposures are apparent; others are not. For instance, it is known that hormonal changes are responsible for changes in the women's appetite and food intake ([Gabbe et al., 2007](#)). In contrast, as an example of a not-so-obvious association, the changes in insulin sensitivity during pregnancy have been

positively correlated with dietary intake of fat ([Chen et al., 2003](#)). However, fat intake rates for pregnant and lactating women are not available. Some changes may not be relevant with regard to susceptibility or environmental exposures (e.g., hair growth that occurs as a result of hormone stimulation of hair follicles). Therefore, this type of information is not included. Some of the physiological and behavioral changes occurring during pregnancy and lactation might not necessarily affect the exposure received, but rather such changes can increase susceptibility and alter the internal dose. For this reason, they are included in this issue paper. For example, an increase in blood volume and cardiac output due to hormonal and metabolic changes maximizes the delivery of respiratory gases to the maternal and fetal tissues ([Gabbe et al., 2007](#); [Heidemann and McClure, 2003](#); [Ciliberto and Marx, 1998](#)).

Information for various demographic groups is presented where available. Racial and ethnic categories used throughout this issue paper were the ones used by the original authors. No attempt was made to use consistent categories, since definitions may vary across studies. Information was presented by trimester where available. Inconsistencies in the presentation of data are mainly a result of data limitations.

1.3. METHODS

A targeted search of published literature was conducted through December 2013 using 19 databases via DIALOG and PubMed. The search terms included: pregnant, pregnancy (including trimesters), lactation, lactating, postpartum, physiological change, change in physiology, behavior/behaviour, environmental toxicant, environmental exposures, environmental factor, environmental risk, and activity pattern. These terms were combined with the requirement that the article also have the terms risk, expose, or exposure. Other targeted searches were conducted to supplement the report in response to external review comments. The lower limit on the years of the literature searched was determined by each individual database. Relevant articles included those that pertained to physiological or behavioral factors in any or all of the three trimesters of pregnancy or the lactation period. In addition, supplementary background information on basic obstetric science and physiology were integrated into some of the sections of this issue paper. Although studies on the U.S. population were preferred, some studies for other populations were included when data were limited or to supplement information presented. Recent articles were favored over older literature. Articles that only contained information on health effects or pregnancy outcomes and no exposure data or physiological data were not deemed relevant.

1.4. ORGANIZATION OF THE REPORT

This issue paper is organized into six main sections: (1) introduction, (2) defining the pregnant and lactating lifestage, (3) physiological changes during pregnancy and lactation, (4) behavioral adaptations and psychological changes during pregnancy and lactation, (5) exposure factors for pregnant and lactating women, and (6) exposure factors data gaps. Section 3 is organized according to the various organ systems, and Section 4 is organized according to general categories of adaptations and behavioral changes. Section 5 summarizes data for several exposure factor categories (e.g., water intake, dietary intake, inhalation rates, activity factors, consumer product use, and body weight).

1.5. INTENDED AUDIENCE

This report is intended for use by exposure and risk assessors both within and outside the EPA as a resource of information on physiological and behavioral changes that may be important to consider when assessing exposures to pregnant and lactating women. It may be used by scientists and other interested parties to inspire research in areas where data gaps have been identified.

2. DEFINING THE PREGNANT AND LACTATING LIFESTAGE

Approximately 60 million women of reproductive age live in the United States. In the Centers for Disease Control and Prevention (CDC) reports on pregnant women, reproductive age for women is most often defined as ages 15 to 44 years ([Ventura et al., 2012](#)). In reality, reproductive age begins at the onset of menses, when pregnancy can occur, and continues until menopause when menses ends and pregnancy is no longer possible. Because the age of menses onset and end can vary among women and populations, reproductive age can begin earlier than 15 years or end later than 44 years. However, since this is the historical age range used in discussions of reproductive age, this range was used in the literature search, but note that these age ranges vary among the studies cited in this issue paper.

Approximately 10% of U.S. women between the ages of 15 and 44 years become pregnant annually. In 2008 there were almost 6.6 million pregnancies (105.5 pregnancies per 1,000 women aged 15–44 years) in the United States, of which 4.2 million resulted in live births ([Ventura et al., 2012](#)). Pregnancy rates vary by age and race (see Table 2-1).

Table 2-1. U.S. pregnancy rates for 2008, by age and race (pregnancies per 1,000 women)

Population group pregnancy outcome	Total	Age (years)						
		Under 15	15–19	20–24	25–29	30–34	35–39	40–44
All races								
All pregnancies	105.5	1.4	69.8	163.0	167.9	141.2	78.5	18.8
Live births	68.1	0.6	40.2	101.8	115.0	99.4	46.8	10.6
White non-Hispanic								
All pregnancies	87.5	0.5	44.8	124.2	149.8	132.5	71.0	16.2
Live births	60.5	0.2	26.7	82.8	109.7	100.8	45.2	9.6
Black non-Hispanic								
All pregnancies	144.3	3.8	121.6	261.6	216.2	157.7	81.1	21.3
Live births	70.8	1.4	60.4	131.5	108.8	75.3	36.3	9.3
Hispanic								
All pregnancies	136.9	2.2	111.5	229.5	197.1	149.2	87.2	23.9
Live births	92.7	1.1	70.3	154.1	142.3	105.3	54.0	14.0

Source: [Ventura et al. \(2012\)](#).

The CDC reports that in 2001, approximately one-half of pregnancies in the United States were unplanned ([Finer and Henshaw, 2006](#)). This information is important in that it indicates that while physical changes in these pregnancies would proceed as in all pregnancies, changes in behaviors related to prenatal care would likely not precede pregnancy in most women, but would begin at diagnosis of the pregnancy, at the earliest. For example, consumption of prenatal vitamins or cessation of smoking may not occur prior to pregnancy, but at some point later in pregnancy or during lactation, so any associated environmental exposures may be similarly variable in the population.

Race may also be a potential factor in pregnancy- and lactation-related environmental exposures of women of reproductive age in the United States. The average U.S. woman is expected to have 3.2 pregnancies in her lifetime at current pregnancy rates; non-Hispanic black and Hispanic women are expected to have 4.3 and 4.0 pregnancies respectively, compared with 2.7 for non-Hispanic white women ([Ventura et al., 2012](#)). Therefore, if pregnancy and/or lactation carries susceptibilities to certain environmental exposures, then being pregnant more frequently increases these susceptibilities for certain racial groups than for others. For example, if African-American women have more pregnancies on average, then this population may be at increased risk for certain pregnancy- and/or lactation-related environmental exposures.

In a similar manner, age can also impact pregnancy-related exposures to environmental contaminants, since certain age groups have higher pregnancy rates. From 1990 to 2008, there was a reported 40% drop in the teenage pregnancy rate, reaching a historic low of 69.8 pregnancies per 1,000 women aged 15–19 years in 2008 ([Ventura et al., 2012](#)). Rates for younger teenagers (ages 15–17 years) declined more relative to older teenagers (ages 17–19 years). The estimated pregnancy rate for U.S. women aged 15–44 years was 105.5 pregnancies per 1,000 women. The highest pregnancy rates were for women aged 25–29 years, at 167.9 per 1,000 in 2008, followed closely by women aged 20–24 years, at 163.0 per 1,000. Pregnancy rates for women aged 30–34 years was 141.2 per 1,000 ([Ventura et al., 2012](#)).

Provided that there are no health concerns after delivery, an increasing number of mothers breastfeed. The American Academy of Pediatrics (AAP) reaffirms their recommendation of exclusive breastfeeding for approximately the first 6 months of life and supports the continuation of breastfeeding for the first year and beyond if desired by the mother and child ([AAP, 2012, 2005](#)). Breastfeeding rates increased between 2008 and 2010; 74.6% of mothers breastfed their infants in the early postpartum timeframe in 2008 and 76.5% of mothers breastfed their infants over a similar timeframe in 2010 ([CDC, 2013](#)). At 6 and 12 months postpartum, 49% and 27%, respectively, continued to breastfeed ([CDC, 2013](#)). Breastfeeding rates vary with the mother's age and other sociodemographic factors. Breastfeeding data

stratified by sociodemographic characteristics for 2008 or later have not been released by CDC. The percentage of mothers that breastfeed varies with age, race, and postpartum time (see Table 2-2). The highest percentages of breastfeeding are for mothers 30 years and older and for non-Hispanic white mothers ([CDC, 2012](#)).

Table 2-2. Percentage of women breastfeeding in 2007 by maternal age and race

Age or race	Breastfeeding ever	Breastfeeding at 6 months postpartum	Breastfeeding at 12 months postpartum
Age (years)			
<20	59.7	22.2	10.7
20–29	69.7	33.4	16.1
≥30	79.3	50.5	27.1
Race			
Non-Hispanic white	76.2	44.7	23.3
Non-Hispanic black	58.1	27.5	12.5
Hispanic	72.8	41.9	21.5

Source: [CDC \(2012\)](#).

3. PHYSIOLOGICAL CHANGES DURING PREGNANCY AND LACTATION

The following sections present a summary of physiological changes women can experience during pregnancy and lactation. Most of the information available and presented in this section relates to changes during pregnancy. Very limited data have been found on physiological changes in lactating women. Information is organized according to the human body organ systems.

3.1. CARDIOVASCULAR SYSTEM AND HEMATOLOGICAL SYSTEMS

As early as 5 weeks gestation, significant physiological changes and adaptive mechanisms occur in the maternal cardiovascular and hematological systems. These changes, such as increased cardiac output and increased blood volume, are a result of hormonal and metabolic changes that maximize delivery of respiratory gases, nutrients, and metabolites to maternal and fetal tissues ([Gabbe et al., 2007](#); [Heidemann and McClure, 2003](#); [Ciliberto and Marx, 1998](#)). Many of these changes influence susceptibility to environmental contaminants in pregnant women due to the impacts that these changes can have on physiological activities such as duration of action of exogenous chemicals in the blood stream, blood flow patterns, and other pharmacokinetic factors. The mean \pm standard deviation (SD) cardiac output in liters/hour increases from the prepregnancy value of 301 ± 65 to 354 ± 76 , 386 ± 75 , 400 ± 79 , and 391 ± 79 at 10, 20, 36, and 38 weeks of gestation, respectively ([Abduljalil et al., 2012](#)). This represents an increase of 18–33% from weeks 10–38. Total blood volume, which is a combination of plasma and red blood cells, increases 30–45% during pregnancy. This increase occurs rapidly until mid-pregnancy, more slowly during the latter half, and plateaus or decreases slightly to term ([Blackburn, 2007](#)). During pregnancy, there is also a progressive increase in resting oxygen consumption, which is a reflection of the metabolic needs of the mother and the fetus. Oxygen consumption reaches its peak increase of 20–30% by term ([Blackburn, 2007](#)). In addition, maternal heart rate (HR) increases progressively during pregnancy, by an average of 10–20 beats per minute (10–20% increase) by 32 weeks gestation ([Blackburn, 2007](#)).

In a study of 45 postpartum women (22 breastfeeding; 23 formula feeding), there was a statistically significant decline in systolic blood pressure (SBP) and HR. This decline was statistically significantly lower in the breastfeeding group than in the formula-feeding group, even after adjusting for body mass index (BMI) ([Groer et al., 2013](#)). Diastolic blood pressure (DBP) was also lower for the breastfeeding group.

3.2. RESPIRATORY SYSTEM

Due to a combination of hormonal fluctuations and mechanical factors that affect the physical configuration of the thoracic cage, both anatomical and physiological changes occur in the maternal respiratory system during pregnancy. These changes, which may or may not affect airborne exposures, include changes in pulmonary function, lung volume and capacities, and respiration. Inhalation rates and oxygen consumption increase during gestation to meet the metabolic demands during pregnancy. Inhalation rates by pregnant and lactating women are discussed in Section 5.4.

3.2.1. Pulmonary Function, Lung Volume, and Capacities

Respiratory parameter changes during pregnancy reported in the literature include increases in tidal volume, minute ventilation, respiratory frequency, inspiratory drive, inspiratory capacity, respiratory resistance, and occlusion pressure and decreases in respiratory tract conductance, peak expiratory flow rates, and expiratory reserve volume ([Harirah et al., 2005](#); [Kolarzyk et al., 2005](#); [Chhabra et al., 1988](#)). The mechanical pressure from the enlarging uterus causes an upward displacement of the diaphragm by up to 4 cm as gestation progresses. Total lung capacity, however, is reduced only slightly (about 5%) because of compensating increases that occur in chest diameter and a flaring of the ribs from hormone-induced relaxation of the ligaments between the ribs and sternum ([Gabbe et al., 2007](#); [Ciliberto and Marx, 1998](#)).

Despite observed changes in certain lung capacity measures, overall pulmonary muscle function and respiratory rates are not generally observed to be affected by pregnancy (e.g., maximum inspiratory and expiratory pressures are unchanged). Also, spirometric measurements assessing bronchial flow (e.g., forced vital capacity) are generally reported to be unaltered, suggesting compensations that facilitate stability of airway function during pregnancy ([Gabbe et al., 2007](#); [Kolarzyk et al., 2005](#); [Brancazio et al., 1997](#); [Weinberger et al., 1980](#)). However, spirometric measurements have been observed to vary in pregnant women when taking into account additional factors such as trimester, position (e.g., sitting, standing), and body mass. The fact that some values (e.g., respiratory resistance) increase during pregnancy, while others (e.g., vital capacity) remain the same is thought to reflect the effect of the autonomous nervous system on the respiratory tract ([Kolarzyk et al., 2005](#)).

Peak expiratory flow rate, expiratory reserve volume, and vital capacity have been shown to be affected by maternal position (e.g., sitting, standing) at the time the measurement is made ([Harirah et al., 2005](#); [Chhabra et al., 1988](#)). Correlations have been observed between BMI (measured before pregnancy) and the magnitude of increases in minute ventilation, inspiratory drive, and occlusion pressure across all trimesters ([Kolarzyk et al., 2005](#)). These alterations in the pulmonary function and lung volume of pregnant women may affect the disposition of air pollutants in the respiratory tract.

3.2.2. Respiration

The amount of air breathed in or out during normal respiration (i.e., tidal volume) is influenced by hormonal changes in pregnant women. Increasing levels of progesterone during pregnancy drive a state of chronic hyperventilation, which has been observed to increase tidal volume by up to 30–40% at 8 weeks gestation ([Gabbe et al., 2007](#)) (see Table 3-1).

Table 3-1. Changes in lung volumes and capacities during pregnancy

Measurement	Definition	Change in pregnancy
Respiratory rate (RR)	Number of breaths per minute.	Unchanged
Vital capacity (VC)	Maximum amount of air that can be forcibly expired after maximum inspiration (IC + ERV).	Unchanged
Inspiratory capacity (IC)	Maximum amount of air that can be inspired from resting expiratory level (TV + IRV).	Increased 5–10%
Tidal volume (TV)	Amount of air inspired and expired with normal breath.	Increased 30–40%
Inspiratory reserve volume (IRV)	Maximum amount of air that can be inspired at end of normal inspiration.	Unchanged
Functional residual capacity (FRC)	Amount of air in lungs at resting expiratory level (ERV + RV).	Decreased 20%
Expiratory reserve volume (ERV)	Maximum amount of air that can be expired from resting expiratory level.	Decreased 15–20%
Residual volume (RV)	Amount of air in lungs after maximum expiration.	Decreased 20–25%
Total lung capacity (TLC)	Total amount of air in lungs at maximal inspiration (VC + RV).	Decreased 5%

Source: [Crapo \(1996\)](#).

Progesterone-induced hyperventilation and concurrent increases in tidal volume also lead to an overall parallel rise in minute ventilation, despite a stable respiratory rate (Minute ventilation = Tidal volume × Respiratory rate). As the minute volume increases, a concurrent increase in oxygen uptake and consumption occurs, with maternal oxygen consumption typically observed to be 20–40% greater in pregnant women due to the oxygen requirements of the fetus, the placenta, and maternal organs and up to 60% greater during labor due to the exaggerated cardiac and respiratory work load ([Ciliberto and Marx, 1998](#)). This rise in minute volume also ultimately increases alveolar oxygen ([Gabbe et al., 2007](#)).

Oxygen consumption at rest in pregnant women ranges from 249–331 mL/minute and from 191–254 mL/minute in nonpregnant women ([Abduljalil et al., 2012](#)). Early in pregnancy, the arterial oxygen increases (106–108 mmHg); however, due to the enlarging uterus, a slight decrease in arterial oxygen (101–104 mmHg) is observed by the third trimester ([Gabbe et al.,](#)

[2007](#)). Oxygen depletion has also been cited as a possible physiological cause of the fatigue frequently observed in the first trimester of pregnancy ([Poole, 1986](#)). Inhalation rates for pregnant and lactating women are presented in Section 5.4.

3.3. RENAL SYSTEM

Several changes occur in the renal system during pregnancy. The kidneys enlarge in size and their weight increases by approximately 30% due to increased renal vasculature, interstitial volume, and urinary dead space ([Abduljalil et al., 2012](#); [Gabbe et al., 2007](#)). Changes are also observed as the maternal anatomy to accommodate the growing fetus. These changes decrease the capacity of the bladder and increase the frequency of urinary incontinence. Frequent urination is also caused by increased blood flow to the kidneys and increased pressure on the bladder from the weight of the pregnancy ([Bonillas and Feehan, 2008](#)). The increased urination may have affect the elimination of chemicals from the body. Nocturia, or excessive urination at night, is also common during pregnancy as water retained during the day is excreted at night when the woman is in the recumbent position ([Chesley and Sloan, 1964](#)).

The glomerular filtration rates (GFR), defined as the flow rate of filtered fluid through the kidneys, and creatinine clearance rate (CrCl), the volume of blood plasma cleared of creatinine per unit time, increase throughout the pregnancy ([Abduljalil et al., 2012](#); [Gabbe et al., 2007](#)). The increase in glomerular filtration rates and creatinine clearance can affect the elimination of chemicals from the body ([Hebert, 2013](#)). Effective renal plasma flow (eRPF), the amount of plasma flowing to the parts of the kidney that function in the production of urine, increases during early pregnancy, but decreases towards term ([Abduljalil et al., 2012](#)). The mean \pm SD in mL/minute of glomerular filtration rates increases from a prepregnancy value of 114 ± 28 to 136 ± 32 , 156 ± 26 , 160 ± 26 , and 156 ± 42 at 10, 16, 26, and 36 weeks of gestation, respectively ([Abduljalil et al., 2012](#)) and represents an increase ranging from 19% to 40% in early pregnancy. The mean \pm SD in mL/minute of creatinine clearance increases from a prepregnancy value of 98.3 ± 14.4 to 126 ± 20 , 155 ± 28 , 152 ± 39 , and 124 ± 34 at 12, 26, 33, and 37 weeks of gestation, respectively ([Abduljalil et al., 2012](#)), representing an increase ranging from 26% to 58%. The mean \pm SD in L/hour of effective renal plasma flow increases from a prepregnancy value of 32.3 ± 6.4 to 44.5 ± 6.1 , 48.4 ± 8.8 , 47.8 ± 12.5 , and 42.3 ± 11.2 at 7, 16, 26, and 36 weeks of gestation, respectively ([Abduljalil et al., 2012](#)), representing an increase ranging from 31% to 50%. These changes along with many other physiological changes are attributed to causing the energy depletion and fatigue during the first trimester ([Poole, 1986](#)).

3.4. SKELETAL SYSTEM

There are two primary measures of the skeletal state. The first is bone mineral density (BMD) and the other is bone turnover. BMD is a point measurement taken at different bones of the skeleton. Bone turnover is a flux measurement incorporating both calcium absorption and depletion in bone. BMD is measured as the amount of minerals, such as calcium, per square centimeter of bone. Both pregnancy and lactation result in reversible bone loss, caused by the need to provide the adequate calcium for the developing fetus and for milk production ([Møller et al., 2012](#); [Gabbe et al., 2007](#); [Ritchie et al., 1998](#); [Krebs et al., 1997](#)). Hormone levels inherently affect bone turnover while a woman is supporting a fetus ([Holmberg-Marttila et al., 2003](#)). These changes in bone depletion and absorption may permit the release to the blood system of any pollutants (e.g., lead) that may have been deposited in the bones. The majority of the literature addresses BMD during and after lactation. More specifically, while lactating, women experience an increase in bone absorption with a larger decrease in bone deposition resulting in a net loss or higher bone turnover ([Osterloh and Kelly, 1999](#)).

The process of calcium absorption in the small intestine and bone turnover in the skeleton both affect the overall BMD throughout pregnancy and into the postpartum period. Therefore, calcium metabolism and its relationship with BMD during pregnancy and lactation is also a highly studied area. It is generally accepted and supported in the literature that absorption and urinary excretion of calcium increase during the second trimester, whereas bone turnover increases during the third trimester and lactation ([O'Brien et al., 2006](#); [Silva et al., 2005](#); [Yoon et al., 2000](#); [Kolthoff et al., 1998](#); [Cross et al., 1995](#); [Cole et al., 1987](#)). During the third trimester, the fetal demand on calcium is at its peak due to bone calcification ([More et al., 2001](#)). For active women, there is some evidence to indicate that changes in BMD during pregnancy and lactation may represent changes in mechanical stress as a result of weight gain, changes in posture and/or activity, or some other factor specific to this population ([Drinkwater and Chesnut, 1991](#)).

The majority of BMD loss is during the first 5 months of lactation. Between 5–12 months postpartum, there is no further BMD loss ([Karlsson et al., 2001](#)). Once menstruation resumes, BMD recovers. Bone recovery back to prepregnancy level appears to be modulated slightly by lactation habits and hormonal status ([Holmberg-Marttila et al., 2003](#); [Holmberg-Marttila et al., 2000](#); [Laskey and Prentice, 1999](#)). The length of lactation, maternal age ([Holmberg-Marttila et al., 2003](#)), and ovarian dysfunction ([Kalkwarf, 2004](#); [Honda et al., 1998](#)) are positively correlated with increased bone turnover during lactation. Higher parity, longer history of previous lactation ([Holmberg-Marttila et al., 2003](#)), and resumption of menstruation ([Holmberg-Marttila et al., 2000](#); [Laskey and Prentice, 1999](#)) are related to bone density recovery.

Further evidence indicates that when a woman has dual demands of lactation and a subsequent pregnancy, she is not at risk for failure to recover the bone loss ([Sowers et al., 1995b](#)). By 12 to 24 months postpartum, regardless of lactation practice, most women have regained their prepregnancy BMD ([Polatti et al., 1999](#); [Kalkwarf and Specker, 1995](#); [Matsumoto et al., 1995](#); [Sowers et al., 1995a](#); [Sowers et al., 1993](#)). No associations have been detected between bone loss and calcium intake, physical activity, body size ([Sowers et al., 1995a](#); [Sowers et al., 1993](#)), weight changes, or initial bone density ([Kolthoff et al., 1998](#)).

Pregnant women may be at risk years after exposure to lead due to calcium mobilization from bone when calcium demand increases in pregnancy ([Alba et al., 2012](#)). A case study of a woman exposed to lead at levels of 145 µg/dL for 7 years prior to pregnancy, showed that measured blood lead levels tripled to 81 µg/dL within 5 months after giving birth ([Riess and Halm, 2007](#)). Two studies found limited evidence supporting the hypothesis of lead mobilization from bone during lactation ([Moline et al., 2000](#); [Osterloh and Kelly, 1999](#)), while others found that breastfeeding practices and maternal bone lead were good predictors of blood lead levels ([Tellez-Rojo et al., 2002](#)). The blood lead concentration is shown to be highest 3–6 months after parturition ([Gulson et al., 2004](#); [Gulson et al., 2003](#)). This potential risk of lead exposure to the woman and the breastfeeding infant is associated with very low (one-half to two-thirds the daily recommended requirements) calcium intakes ([Gulson et al., 2004](#); [Gulson et al., 1999](#)). It has also been shown that foods high in calcium may have a protective effect against the accumulation of lead in bone ([Hernandez-Avila et al., 1996](#)). Calcium supplementation has a limited benefit inhibiting lead mobilization from bone during lactation ([Gulson et al., 2004](#)), but low calcium dietary intake is an indicator for higher bone lead mobilization.

3.5. NEUROLOGICAL SYSTEM

Neuroendocrine processes are significantly altered during pregnancy. Associations have been made between prenatal psychosocial stress, social support, and personality variables with neuroendocrine parameters (plasma levels of adrenocorticotrophic hormone [ACTH], beta-endorphin, and cortisol) ([Wadhwa et al., 1996](#)). ACTH is produced by the pituitary gland and the hormone's primary function is to stimulate the production and release of cortisol. More information about ACTH and pituitary function is found in Section 3.7.4. Cortisol is produced by the adrenal cortex in response to physical or psychological stress. Mean cortisol levels also increase during pregnancy from 12.5 ± 1.3 µg/dL in nonpregnant women to 30.1 ± 6.6 µg/dL ([Gabbe et al., 2007](#)), a 141% increase. Levels of beta-endorphin, a neurochemical produced in the pituitary gland in response to pain, trauma, exercise, or stress, are statistically significantly lower during pregnancy than in the nonpregnant state ([Goebelsmann et al., 1984](#)). The levels are at their lowest during the second trimester of pregnancy. Beta-endorphin levels rise dramatically

during early and advanced labor. The sympathetic nervous system, which is responsible for regulating an individual's "fight or flight" response is dampened due to diminished blood pressure responses while female reproductive hormones are elevated ([Matthews and Rodin, 1992](#)). Responses to stress in postpartum women who did not lactate indicated increased sympathetic and decreased parasympathetic nervous system activity. However, the lactating counterparts did not have attenuated physiological or anxiety responses ([Altemus et al., 2001](#)). These changes may alter a woman's behavior (e.g., eating patterns, time spent at various activities at different locations) and her chances for exposure to environmental chemicals. Additional information on stress and anxiety is found in Section 4.3.

Cholinesterase is one of many important enzymes needed for the proper functioning of the nervous system. Some studies have shown that serum cholinesterase activity changes during pregnancy. A study conducted by [Evans et al. \(1988\)](#) examined serum cholinesterase activity in 44 women before, during, and after pregnancy. Some women showed a decline in cholinesterase activity after conception, with no return towards preconception values before delivery. Other women exhibited a decline in cholinesterase activity accompanied by a partial or complete return to preconception values before delivery. A few women displayed either no discernible decline or increased cholinesterase activity during gestation. The differences are potentially age-related as the continuous decrease in cholinesterase activity occurred in the youngest group of women, the decrease followed by an increase occurred in the intermediate age group, and no decrease at all was seen in the oldest group, although none of the age-related differences were statistically significant.

3.6. DIGESTIVE/GASTROINTESTINAL SYSTEM

It is common during pregnancy and lactation for changes to occur in the amount, frequency, and choices of food consumed. Increased or decreased appetite during pregnancy and changes in taste may be induced by estrogen and progesterone, which are present at elevated levels during pregnancy ([Faas et al., 2010](#)). Generally, appetite increases during pregnancy result in the average consumption of an additional 200 kcal/day. Fat storage and appetite are regulated by free and bound leptin, respectively. Leptin is a pregnancy-related hormone that regulates appetite and metabolism, and it is usually produced in adipose tissue. Since pregnancy is generally associated with increased appetite, it is likely that a leptin-resistant state develops during pregnancy allowing for an increase in food intake ([Augustine et al., 2008](#)). Leptin has also been found to control energy expenditures and body mass accumulations (see Section 3.7.6).

In general, leptin has been observed to be higher in pregnant women than nonpregnant women and may increase progressively through pregnancy or after delivery ([Teppa et al., 2000](#); [Lage et al., 1999](#); [Lin, 1999](#); [Butte et al., 1997](#)). Free leptin tends to increase from the first to the

second trimester and then remains the same for the rest of the pregnancy ([Gabbe et al., 2007](#)). Bound leptin that affects metabolism, and therefore appetite, increases from the second to the third trimester ([Widjaja et al., 2000](#)). Leptin serum level concentrations in pregnant women during the first trimester were reported to range from 14.3 ± 1.4 – 14.7 ± 0.7 ng/mL ([Lage et al., 1999](#); [Lin, 1999](#)). Second trimester leptin levels ranged between 16.3 ± 1.3 and 18.3 ± 0.6 ng/mL ([Lin, 1999](#)). The highest leptin levels were observed during the third trimester, with values ranging from 21.1 ± 1.4 to 33.8 ± 4.1 ng/mL ([Teppa et al., 2000](#); [Lin, 1999](#)). Leptin plasma levels reach a peak at gestation weeks 35–41 ([Lin, 1999](#)). [Lin \(1999\)](#) reported a positive correlation of these levels with a BMI in 65 women ($r = 0.65$, $p < 0.001$). Leptin values in nonpregnant women have been reported to range from 9.1 ± 0.6 – 16.5 ± 0.9 ng/mL, with the highest values observed in women with higher BMIs. Leptin serum level concentrations were 48.1 ± 5.6 ng/mL in patients with preeclampsia in a study of 18 healthy, 18 preeclamptic, and 18 never-pregnant women ([Teppa et al., 2000](#)). Preeclampsia is a pregnancy-related condition that usually develops after the 20th week of pregnancy and is marked by high blood pressure, edema in the hands and feet, and protein in the urine.

Obese pregnant women have been found to have statistically significant changes in several gastrointestinal hormones affecting food intake, such as acylated ghrelin, peptide YY, and cholecystokinin, a possible explanation for the enhanced appetite and increased food intake in these individuals ([Sodowski et al., 2007](#)). Ghrelin concentrations may change with increased adiposity and may play a role in body weight postpartum ([Larson-Meyer et al., 2010](#)). However, neither ghrelin nor peptide YY were shown to affect appetite and body weight regulation during lactation in the cohort of women in the study.

A common digestive complaint during pregnancy is nausea and vomiting, also commonly referred to as “morning sickness.” Approximately 70% of pregnant women suffer from nausea in the first trimester, about 10–25% report it continuing into the second trimester, and 1–3% develop into severe cases that persist throughout pregnancy. The most severe form can lead to significant weight loss, excess ketones in the blood, or electrolyte imbalances. Food eating patterns may change in order to cope with this condition. For example, women may eat more frequent and smaller meals throughout the day, eat bland foods that are easier to digest, consume foods that are high in protein, drink more fluids, and avoid high-fat foods ([March of Dimes, 2013a](#); [Gabbe et al., 2007](#)). Data on dietary intake by pregnant women are presented in Section 5.2.

The stomach also changes in tone and motility, likely due to progesterone- and estrogen-induced relaxation of the smooth muscle ([Shah et al., 2001](#)). Anywhere from 30–50% of women report an increase in gastric reflux and indigestion due to increased hormone levels and the physical compression of the stomach from the enlarging uterus ([Gabbe et al., 2007](#)).

During pregnancy, there is also an uncommon symptom called ptyalism, which is popularly believed to be the inability of the nauseated woman to swallow saliva and can result in a loss of 1 to 2 L of saliva per day ([Gabbe et al., 2007](#)).

Pregnancy is credited with causing slower digestion due to a slowdown in esophageal and intestinal contractions ([Bonillas and Feehan, 2008](#)). The increase in progesterone levels during pregnancy produces a relaxation of the muscles that results in a decrease in intestinal tone and motility ([Blackburn, 2007](#)). The decrease in intestinal motility leads to an increase in the absorption of nutrients such as calcium and iron, as well as other substances ([Blackburn, 2007](#)). The small intestines and colon have a higher rate of water and sodium absorption and a slower rate of mobility, which can lead to constipation ([Parry et al., 1970](#)). Slowed digestion may result in longer residence time of contaminated food in pregnant women, and therefore, increased uptake of ingested environmental contaminants.

3.7. ENDOCRINE SYSTEM

A pregnant woman experiences a multitude of hormonal changes throughout pregnancy. These hormones direct various changes in the woman's body systems and processes that primarily function to support the fetus during its different stages of development. The following discussion of pregnancy- and lactation-related changes in the endocrine system is divided into separate sections that include: the placenta, thyroid function, parathyroid function, hypothalamic-pituitary-adrenal axis, glucose and carbohydrate metabolism, protein and lipid metabolism, metabolic adjustments, and total body water metabolism.

3.7.1. Placenta

The placenta is a temporary endocrine organ developed during pregnancy that has the primary functions of nourishing the fetus as well as eliminating fetal waste materials. The placenta becomes the main source of progesterone during the second and third trimester ([Abduljalil et al., 2012](#)). Other hormones produced by the placenta include human chorionic gonadotropin, human placental lactogen, estrogen, and leptin ([Blackburn, 2008](#); [Lin, 1999](#)). These hormones play a critical role in many metabolic and endocrine changes during pregnancy. For example, human chorionic gonadotropin alters maternal protein, carbohydrate, and fat metabolism ([Blackburn, 2008](#)). During pregnancy, the placenta also synthesizes corticotropin-releasing hormone (CRH), which may modulate important physiological aspects of labor, glucose transport to the placenta and the fetus, and the psychological mood of the mother ([Thomson, 2013](#)). CRH has been found to be stimulated by cortisol ([Sirianni et al., 2004](#)) (see Section 3.5).

The placenta also secretes leptin, which is a pregnancy-related hormone that regulates appetite and metabolism, as discussed in Section 3.6. Comparisons to age- and BMI-matched nonpregnant women, suggest that placental production of leptin is one of the major sources of leptin to maternal circulation ([Butte et al., 1997](#)).

The size of the placenta increases with gestational time. The mean \pm SD placenta volumes in mL are 134 ± 58 , 254 ± 62 , 460 ± 173 , 593 ± 90 , and 659 ± 103 at 14, 20, 30, 36, and 40 weeks of gestation ([Abduljalil et al., 2012](#)).

3.7.2. Thyroid Function

During pregnancy, there are alterations in maternal thyroid morphology, histology, and laboratory indices, although pregnant women generally retain normal thyroid function. The thyroid is a gland in the neck that secretes hormones that regulate growth and metabolism. The thyroid gland may increase in size, but if adequate iodine intake is maintained the size changes may be extremely small to none. The thyroid continues to synthesize and secrete thyroid hormone actively during pregnancy (see Figure 3-1). During the first trimester, total thyroxine (T4) and total triiodothyronine (TT3) levels begin to increase. However, T4 and TT3 peak at the end of the second trimester due to the increased production of thyroid-binding globulin (TBG), which also begins in the first trimester and plateaus at 12 to 14 weeks. Thyroid-stimulating hormone (TSH) concentrations decrease temporarily in the first trimester, but then return to prepregnancy levels by the end of the first trimester and remain stable through the second and third trimester ([Burrow et al., 1994](#)). The temporary decrease in TSH and increase in T4 during the first trimester are attributed to the thyrotropic effects of human chorionic gonadotropin (hCG; higher hCG levels suppress more TSH). TSH and hCG are structurally very similar, but the exact role of hCG in maternal thyroid function is not well understood ([Gabbe et al., 2007](#)).

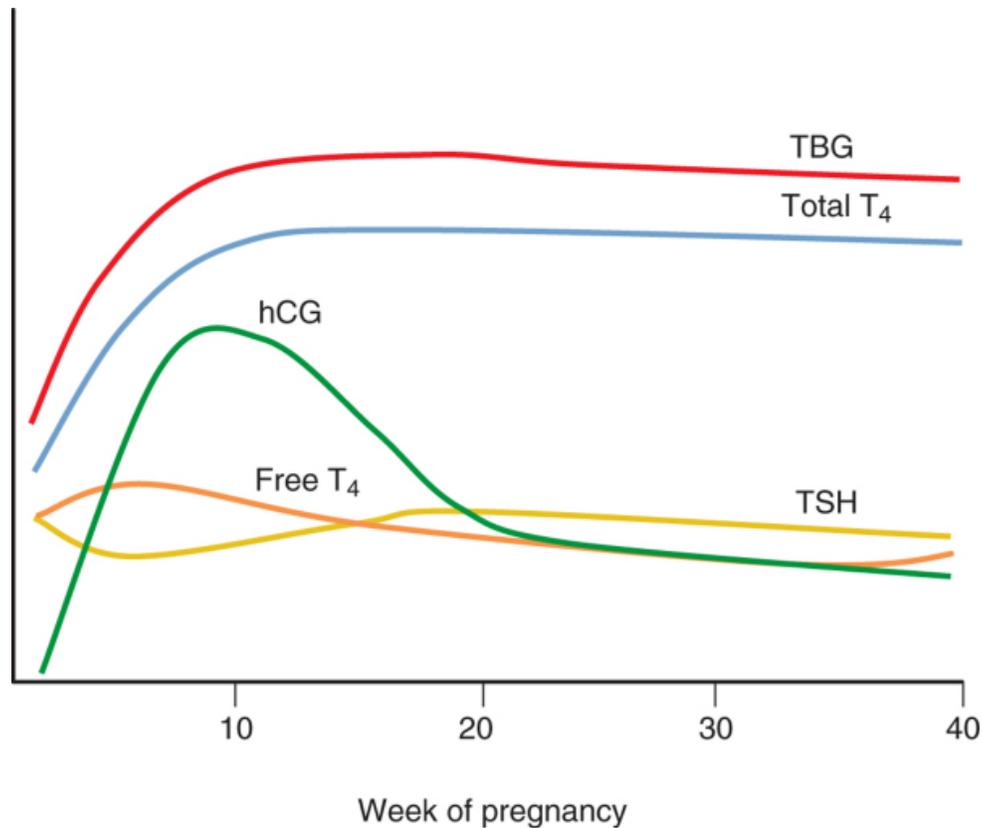


Figure 3-1. Relative changes in maternal thyroid function during pregnancy.

Source: [Burrow et al. \(1994\)](#). From The New England Journal of Medicine, Burrow et al., Maternal and Fetal Thyroid Function, 331, 1072. Copyright © 1994 Massachusetts Medical Society. Reprinted with permission from Massachusetts Medical Society.

The effect of gestation on women with hypothyroidism (insufficient production of thyroid hormone) has been investigated. In a retrospective study of data from 167 pregnancies on women with hypothyroidism, the median T₄ dose (μg) used to supplement low thyroid status was observed to increase significantly during pregnancy (first trimester: 100; second trimester: 125, $p < 0.001$; and third trimester: 150, $p < 0.001$) ([Idris et al., 2005](#)). Exposure to certain exogenous chemicals may further inhibit iodine uptake (e.g., perchlorate) ([Leung et al., 2010](#)).

Studies have suggested that women with hormone concentrations even in the lower euthyroid range (i.e., normal thyroid function) may be at greater risk of developing postpartum depressive symptoms ([Pedersen et al., 2007](#)). Both statistically significantly higher T₃-resin uptake and marginally lower total T₄ concentrations have been observed at 38 weeks of pregnancy in women with higher postpartum depression ratings. Further, mean antenatal T₄

concentrations and free T4 indices were statistically significantly and negatively correlated with mean depression scores during postpartum weeks 2–6, 14–18, and 20–24 in a study of 31 women ([Pedersen et al., 2007](#)).

3.7.3. Parathyroid Function

Changes in parathyroid hormone levels (produced by the parathyroid glands to maintain the body's calcium level) during pregnancy have also been studied. Parathyroid hormone promotes the absorption of calcium from the bones. Research has suggested that parathyroid hormone-related protein produced in the fetoplacental unit, the breast, or both, can reach the maternal circulation. Parathyroid hormone levels increase during pregnancy and lactation to meet the calcium demands of the mother and the growing fetus ([Ardawi et al., 1997](#)). Higher demands for calcium may impact the mother's skeletal system (see Section 3.4). In a study of 40 healthy nonpregnant women, 90 healthy pregnant women (30 in each trimester), and 140 postpartum women (74 breastfeeding, 33 mixed feeding, 33 bottle feeding) plasma and umbilical cord (in 24 women) levels of parathyroid hormone-related protein were measured ([Hirota et al., 1997](#)). Mean plasma level of parathyroid hormone-related protein increased throughout pregnancy and was statistically significantly higher in the third trimester (increasing from 1.06 ± 0.19 pmol/L in the first trimester to 1.17 ± 0.16 pmol/L in the third trimester), and was closely associated with the degree of breastfeeding at 1 month postpartum. The umbilical venous blood also contained statistically significantly higher levels of parathyroid hormone-related protein than was in maternal circulation.

3.7.4. Hypothalamic-Pituitary-Adrenal Axis

Hypothalamic-pituitary-adrenal axis (HPA) changes in pregnant and lactating women affect a number of body systems. HPA consist of three glands (hypothalamus, pituitary, adrenal) of the endocrine system that regulate body processes including energy storage and expenditures. Although the adrenal glands themselves do not increase in size significantly during pregnancy, the area within the glands that produces glucocorticoids (zona fasciculata) expands. Changes in maternal adrenocortical function during pregnancy include increased serum levels of aldosterone, deoxycorticosterone, corticosteroid-binding globulin, cortisol, and free cortisol. Specifically, the plasma corticosteroid-binding globulin concentration doubles by the end of the 6th month of gestation and there is an exponential increase in corticotropin-releasing hormone, which is produced by the placenta and fetal membranes, during the third trimester. Corticotropin-releasing hormone, in turn, triggers the production of ACTH in the pituitary. Levels of ACTH increase during pregnancy from 12.1 ± 5.8 pg/mL during the 7th to 9th week of gestation to 22.9 ± 1.0 pg/mL by the 36th to the 37th week, representing an increase of 89%

([Klimek, 2005](#)). These increases together stimulate elevations in cortisol (see Section 3.5) ([Gabbe et al., 2007](#)). Deoxycorticosterone levels also increase by mid-gestation and peak in the third trimester. Regarding the androgenic steroids, dehydroepiandrosterone and dehydroepiandrosterone-sulfate levels are decreased due to increased metabolic processing. In addition, maternal concentrations of testosterone are slightly higher during pregnancy due to an elevation in sex-binding hormone, and androstenedione is higher due to increased synthesis ([Gabbe et al., 2007](#)).

The alteration of maternal neuroendocrine processes during pregnancy also has implications for neuroendocrine responsivity to exogenous conditions and psychosocial factors ([Wadhwa et al., 1996](#)) (see Section 3.5). A study of 54 pregnant women was conducted to assess the association between prenatal psychosocial factors and stress-related neuroendocrine parameters ([Wadhwa et al., 1996](#)). The psychosocial factors were strongly associated with the maternal-placental-fetal neuroendocrine parameters, both in magnitude and specificity. In addition, a combination of the maternal psychosocial and sociodemographic factors measured during pregnancy accounted for 36% of the variance in ACTH, 22% of the variance in the ACTH-beta-endorphin dysregulation index, 13% of the variance in cortisol, and 3% of the variance in beta-endorphin ([Wadhwa et al., 1996](#)). More information about stress and anxiety during pregnancy is found in Section 4.3.

The pituitary gland also undergoes changes during pregnancy, increasing in size by up to 36% at term due to a proliferation of prolactin-producing (lactotroph) cells in the anterior pituitary. Lactotroph proliferation results in increases in serum prolactin production, which functions to prepare the breast for lactation. Prolactin levels begin to increase at 5 to 8 weeks gestation and continue to increase until reaching levels up to 10 times higher at term. Postpartum, the prolactin levels return to normal within 3 months in nonlactating women, while it may take several months (with intermittent episodes of hyperprolactinemia) in women who are nursing. Other pituitary hormone levels that change dramatically throughout gestation include follicle-stimulating hormone, luteinizing hormone, and growth hormone. Maternal follicle-stimulating hormone and luteinizing hormone decrease to undetectable levels as a result of feedback inhibition from the elevated levels of estrogen, progesterone, and inhibin. Growth hormone levels are also suppressed because of the action of placental growth hormone variant on the hypothalamus and pituitary ([Gabbe et al., 2007](#)).

HPA responses to stress may also be suppressed in lactating women. For instance, in a study of 10 lactating and 10 nonlactating women between 7 and 18 weeks postpartum, plasma ACTH, cortisol, glucose, and basal norepinephrine responses to physical exercise (20 minutes on treadmill) were considerably attenuated in lactating women ([Altemus et al., 1995](#)). In another

study, however, lactation was observed to have little effect on the HPA responses to psychological stress ([Altemus et al., 2001](#)).

In 24 lactating women, 13 postpartum nonlactating women, and 14 healthy control women in the early follicular phase of the menstrual cycle, ACTH, cortisol, heart rate, diastolic blood pressure, systolic blood pressure, and subjective anxiety ratings were all statistically significantly elevated in response to psychological stress (Trier Social Stress Test). However, there were no differences among the three groups in any of these responses to the stress. The only difference observed in the postpartum lactating women was a persistently lower systolic blood pressure and higher cardiac vagal tone than the nonlactating women in response to stress, suggesting enhanced vagal control of cardiac reactivity in lactating women ([Altemus et al., 2001](#)). For more information regarding stress and anxiety during pregnancy and lactation see Section 4.3.

3.7.5. Glucose and Carbohydrate Metabolism

Substantial physiologic changes in carbohydrate metabolism, which is the process by which sugars and carbohydrates are used in the body to produce energy, occur during pregnancy to allow for the continuous transport of glucose to the fetus and placenta. In early pregnancy, the release of insulin is increased, causing a 10% reduction of glucose levels and enhanced lipogenesis (fat storage) in pregnant women.

As pregnancy progresses, hyperinsulinemia (insulin resistance) develops after the first trimester, resulting in a 50–80% reduction in insulin sensitivity by the third trimester ([Gabbe et al., 2007](#); [Paramsothy and Knopp, 2005](#)). Insulin resistance functions to allow for glucose competition between the maternal tissues to favor the fetus ([Paramsothy and Knopp, 2005](#)). Insulin resistance causes further changes in maternal regulation of blood glucose levels, including hypoglycemia (low blood glucose) during fasting and hyperglycemia (excessive blood glucose) after meals. The average fasting glucose levels in pregnant versus nonpregnant women was 73.1 mg/dL and 79.7 mg/dL, respectively ([O'Sullivan and Mahan, 1966](#)). The release of insulin also increases progressively through pregnancy, peaking in the third trimester. In healthy pregnancies, the exaggerated response in insulin production and greater glucose fluctuations from the fasting to the postfeeding state glucose generally function to maintain glucose homeostasis ([Gabbe et al., 2007](#)). Although the physiological causes of the insulin resistance are not well understood, they may be influenced by hormonal factors such as human placental lactogen, cortisol, progesterone, and estrogen ([Paramsothy and Knopp, 2005](#)).

Longitudinal changes in various measures of carbohydrate metabolism have been observed in association with pregnancy. Changes in pancreatic beta function and metabolic clearance rates of insulin were evaluated in seven women with normal glucose tolerance and nine

women with abnormal glucose tolerance prior to conception, and in early (12–14 weeks) and late (34–36 weeks) gestation ([Catalano et al., 1998a](#)). There were progressive alterations in insulin kinetics with advancing gestation, including statistically significant increases in basal insulin, C-peptide concentrations and the metabolic clearance rate of insulin. No statistically significant differences between the women with normal and abnormal glucose tolerance were observed. These changes in insulin kinetics are partly responsible for pregnancy-related hyperinsulinemia and support the unique role of pregnancy on maternal carbohydrate metabolism. Changes in energy expenditure and body composition as a function of altered carbohydrate metabolism during pregnancy were also investigated in 6 women with normal glucose tolerance and 10 women with abnormal glucose tolerance before conception, and in early (12 to 14 weeks) and late (34 to 36 weeks) gestation ([Catalano et al., 1998b](#)). Increases in basal oxygen utilization, resting energy expenditure, and carbohydrate oxidation with increasing gestational age were observed. Overall, observed changes in maternal fat mass and basal oxygen consumption correlated inversely with changes in insulin sensitivity during pregnancy ([Catalano et al., 1998b](#)).

Normal pregnancy is also associated with increased antioxidant enzyme activity, and there appear to be ethnic differences in antioxidant responses and dietary fat intake ([Chen et al., 2003](#)). Glutathione peroxidase (GPx) activity (one of the most important antioxidant enzymes in humans), measures of insulin resistance (fasting serum insulin, plasma glucose, and C-peptide), and dietary fat intake were measured in 408 normotensive nondiabetic pregnant women at 16 weeks and during the third trimester of pregnancy ([Chen et al., 2003](#)). Increases in GPx activity and insulin resistance were observed between the first and third trimesters, with overall GPx activity also being positively correlated with the dietary intake of fat and polyunsaturated fatty acids, suggesting a potential link between antioxidant defenses, insulin resistance, and dietary fat intake. In addition, African-Americans had statistically significant higher GPx activity, dietary fat, and polyunsaturated fatty acid intake than Hispanics and Caucasians ([Chen et al., 2003](#)). In contrast to these findings of decreased glucose tolerance in the developed world, in a study of 58 nondiabetic pregnant African women in Tanzania, [Lutale et al. \(1993\)](#) found that women in an urban African setting showed little change in glucose tolerance during pregnancy.

3.7.6. Protein and Lipid Metabolism

Protein and lipid metabolism is the decomposition and synthesis of protein and lipids in the body. By the third trimester of pregnancy, the increase in glucose and amino acid uptake by the fetus results in a metabolic shift from predominantly carbohydrate to predominantly fat utilization ([Butte et al., 1999](#)). Protein catabolism is also decreased during pregnancy, resulting in the preferential use of fat stores to fuel metabolism. This lipolysis in turn results in increased generation of glycerol, fatty acids, and ketones for gluconeogenesis and fuel metabolism (i.e.,

hyperlipidemia and hyperketonemia) ([Gabbe et al., 2007](#)). Changes in protein and lipid metabolism may result in changes in appetite.

Increases in maternal plasma cholesterol and triglycerides from the first to the third trimester of gestation, in conjunction with free fatty acids increases from the first trimester through the third trimester to delivery, suggest an enhanced lipolytic activity during pregnancy ([Herrera et al., 2004](#)). In addition, plasma levels of alpha- and gamma-tocopherols, lycopene, and beta-carotene also increase with gestation while retinol levels decline with gestational time. Finally, the proportion of total saturated fatty acids increases with gestation and total *n*-9 fatty acids remain stable throughout pregnancy whereas total *n*-6 fatty acids decline ([Herrera et al., 2004](#)).

Blood plasma levels of lipids (fats/triglycerides, fatty acids, cholesterol) and lipoproteins (i.e., low-density lipoproteins [LDLs], high-density lipoproteins [HDLs], and very-low-density lipoproteins [VLDLs]) increase in pregnancy in a study of 19 pregnant women in Sweden. By full term, triglyceride levels may increase by up to two to three times (levels of 200 to 300 mg/dL are considered normal), and total cholesterol and LDLs may increase by 50–60% ([Salameh and Mastrogiannis, 1994](#)). After temporarily rising in the first 28 weeks of pregnancy, HDL levels decrease in late gestation, reaching levels that are generally about 15% higher than nonpregnant levels by term. By 8 weeks postpartum, triglyceride levels return to prepregnancy levels (including during lactation). In contrast, cholesterol and LDL levels remain elevated. Mechanisms causing the pregnancy-induced changes in lipids are hypothesized to be related to the elevated levels of estrogen, progesterone, and human placental lactogen (hPL) ([Gabbe et al., 2007](#)).

Pregnancy-associated changes in blood concentrations of lipids, lipoproteins, and apolipoproteins have been investigated in several studies. [Desoye et al. \(1987\)](#) conducted a longitudinal study to investigate correlations between hormones and lipid/lipoprotein levels during pregnancy and postpartum. Concentrations of plasma lipids and lipo- and apolipoproteins were measured in 24 nonpregnant and 42 pregnant women. Insulin concentrations were constant during pregnancy until week 25 and then increased for the duration of the pregnancy. Plasma, beta-estradiol, progesterone, and hPL as well as plasma lipid levels rose continuously during gestation. Apolipoproteins AI, AII, and B concentrations increased until weeks 25, 28, and 32, respectively, and then remained constant until term. LDL cholesterol reached maximum levels at week 36. HDL cholesterol exhibited a triphasic behavior, with maximum levels at week 25, a fall until week 32, and then maintenance of the level until term. Time series analysis revealed positive correlations with beta-estradiol, progesterone, and hPL ([Desoye et al., 1987](#)).

[Mazurkiewicz et al. \(1994\)](#) measured fasting serum concentrations of total cholesterol, triglyceride, LDL cholesterol, HDL cholesterol, apolipoproteins AI, AII, and B, and

lipoprotein(a). These parameters were measured in 178 women with normal glucose tolerance in the second and third trimesters of pregnancy and in a control group of 58 nonpregnant women of similar age. Pregnant women had statistically significantly higher concentrations of total cholesterol, triglyceride, LDL cholesterol, HDL cholesterol, and apolipoproteins (AI, AII, and B) than the control women. Also, the ratio of apolipoprotein B to apolipoprotein AI was statistically significantly higher in the pregnant women than in the controls, but the total cholesterol–HDL cholesterol ratio was not statistically significantly different.

The relationship between recreational physical activity and plasma lipid concentrations in early pregnancy was investigated in 925 normotensive, nondiabetic pregnant women averaging 32 years of age from Washington State ([Butler et al., 2004](#)). Mean triglyceride concentration was 12.7 mg/dL lower in women performing any physical activity versus none. Mean total cholesterol was also reduced in women with the highest levels of physical activity, although there was no association observed between physical activity and HDL cholesterol. There was also a consistent linear relationship across levels of physical activity measures for triglyceride and total cholesterol, suggesting that habitual physical activity may attenuate disruptions in blood lipid levels (dyslipidemia) frequently observed during pregnancy ([Butler et al., 2004](#)).

3.7.7. Metabolic Adjustments

In general, metabolic adjustments characterized by increased energy expenditure and preferential use of carbohydrates are observed during pregnancy and lactation to support fetal growth and milk synthesis ([Butte and King, 2005](#); [Butte et al., 2004](#); [Butte et al., 1999](#); [Blackburn and Calloway, 1976](#)). These are adaptive changes in the body's metabolism and may result in altered appetite. Energy expenditure also, in part, increases during pregnancy because of the metabolic contribution of the uterus and fetus and the increased work by the maternal heart and lungs after adjusting for free fat mass, fat mass, and energy balance ([Butte et al., 1999](#)). Related to these metabolic changes, maternal fat stores increase to a peak late in the second trimester and then decline for the remainder of gestation as a result of mobilization to support the rapidly growing fetus ([Widjaja et al., 2000](#)).

In a study of energy expenditures in 76 women (40 lactating, 36 nonlactating) at 37 weeks gestation and 3 and 6 months postpartum, total energy expenditure and its components (basal metabolic rate, sleeping metabolic rate, and minimal sleeping metabolic rate) were 15–26% higher during pregnancy than postpartum ([Butte et al., 1999](#)). During the postpartum period, total energy expenditure and sleeping metabolic rate were higher in lactating than in nonlactating women. [Butte et al. \(1999\)](#) suggested that total energy expenditure and its components (basal metabolic rate, sleeping metabolic rate, and minimal sleeping metabolic rate) correlated positively with fasting serum insulin, insulin-like growth factor I, fatty acids, leptin,

norepinephrine, epinephrine, and dopamine. In addition, elevated respiratory quotients and preferential utilization of carbohydrates were observed to occur during pregnancy and continue through lactation, which was considered to be consistent with the preferential use of glucose by the fetus and mammary glands ([Butte et al., 1999](#)).

Optimal energy requirements of pregnant and lactating women are not fully known. In part, some of this uncertainty is due to variability in energy use possibly related to pregnancy-associated fat deposition and reductions in physical activity ([Butte et al., 2004](#)). Pregnancy-related energy adaptations of 63 women (17 underweight with a low BMI, 34 with a normal BMI, and 12 overweight with a high BMI) were estimated at 0, 9, 22, and 36 weeks of pregnancy and at 27 weeks postpartum. Basal metabolic rate (BMR) generally increased gradually throughout pregnancy at a mean rate of 10.7 ± 5.4 kcal/gestational-week, although there was notable variability between individuals in the study (e.g., some individuals had decreased BMR initially before increases began). The recommended increase in energy intake for lactating women is 500 kcal/day during the first six months of lactation and 400 kcal/day after the sixth month ([Picciano, 2003](#)).

In the normal BMI group, energy requirements increased negligibly in the first trimester, by 350 kcal/day in the second trimester, and by 500 kcal/day in the third trimester. In addition, because there was a slight decrease in activity energy expenditure, total energy expenditure increased by only 5.2 ± 12.8 kcal/gestational week. There were also statistically significant differences in pregnancy-associated metabolic responses associated with BMI. For example, in the normal BMI group, BMR increased by 2% in the first trimester, 7% in the second trimester, and 28% in the third trimester, whereas in the high-BMI group, the increase in BMR was greater (7, 16, and 38% in the first, second, and third trimesters, respectively), consistent with that group's greater gestational weight gain ([Butte et al., 2004](#)).

[Butte and King \(2005\)](#) calculated that the estimated total energy cost of pregnancy for women with a mean gestational weight gain of 12.0 kg, was 76,670–77,625 kcal. This energy is distributed as 90; 287; and 466 kcal/day, for the first, second, and third trimesters, respectively. Other research has suggested that energy expenditure in pregnant women may be seriously underestimated if energy cost figures do not take into account level of fitness and rate of recovery/oxygen uptake in the calculations ([Blackburn and Calloway, 1976](#)).

Several studies have specifically examined energy requirements and expenditures during lactation, which in general causes substantial energy stress for the woman ([Spaaij et al., 1994](#)). A study of 24 Dutch women before pregnancy and 2 months postdelivery was conducted to investigate whether any of the three components of energy metabolism (metabolic rate at rest, following a meal, and following exercise) show signs of metabolic adaptation in the lactating women. The women were from the middle to upper socioeconomic stratum, nonsmokers with an

average age of 29.8 years. The women in the study showed signs of metabolic adaptation during lactation ([Spaaij et al., 1994](#)). Resting metabolic rate and postprandial metabolic rate increased similarly during lactation relative to prepregnancy, and metabolic rates measured after cycling exercise did not change significantly. Accordingly, the researchers suggest that there are no statistically significant changes in diet- and work-induced thermogenesis or metabolic efficiency during lactation ([Spaaij et al., 1994](#)).

In a population of 40 healthy, but rural and poor, lactating Filipino women, energy intake, energy expenditure, and body composition were measured throughout the first 6 months of lactation ([Guillermo-Tuazon et al., 1992](#)). Energy intakes at 6 and 30 weeks of lactation were 244 and 185 kcal/day, respectively. These values were significantly higher ($p < 0.05$) than in early pregnancy. Energy intakes decreased slightly between 6 weeks and 30 weeks of lactation from $2,213 \pm 489$ to $2,073 \pm 566$ kcal/day. Basal metabolic rates also remained unchanged throughout lactation in this study.

Leptin (see Section 3.6), an adipose-derived hormone that plays a key role in regulating energy intake and energy expenditure (as well as hypothesized roles in maternal and fetal fat mass changes during pregnancy), has received particular attention in studies of metabolic changes during pregnancy. Leptin inhibits appetite by acting on receptors in the hypothalamus.

3.7.8. Total Body Water Metabolism

Total body water increases gradually during pregnancy. The total body water content increase by the end of pregnancy is considered one of the most major changes in pregnancy ([Gabbe et al., 2007](#)). This increase is the result of water in the fetus, the placenta, amniotic fluid, enlargement of reproductive organs, increased blood volume, expanded adipose tissue, and increase in inter- and extracellular water ([Gabbe et al., 2007](#); [Hyttén et al., 1966](#)). [Abduljalil et al. \(2012\)](#) conducted a meta-analysis of available total body water data. The analysis shows that the mean \pm SD of total body water in liters increases from 31.67 ± 4.60 before pregnancy to 35.22 ± 1.65 , 40.14 ± 7.55 , and 46.00 ± 5.50 at 12, 25, and 40 weeks of gestation, respectively, an overall increase of 45% from prepregnancy to the 40th week of gestation ([Abduljalil et al., 2012](#)). Approximately 3.5 L is accounted for by the water content of the fetus, placenta, and amniotic fluid at term ([Gabbe et al., 2007](#)). The expansions of the maternal blood volume by 1,500 to 1,600 mL, plasma volume by 1,200 to 1,300 mL, and red blood cells by 300 to 400 mL account for additional water ([Theunissen and Parer, 1994](#)). The remainder is attributed to extravascular fluid, intracellular fluid in the uterus and breasts, and expanded adipose tissue ([Gabbe et al., 2007](#)).

3.8. INTEGUMENTARY SYSTEM

The integumentary system is the body system consisting of the skin and its associated structures, such as the hair, nails, sweat glands, and sebaceous glands. Blood flow to the maternal skin increases during pregnancy, particularly in the extremities ([Abduljalil et al., 2012](#)). Increased blood flow during pregnancy may not affect percutaneous absorption through normal skin, but may affect the absorption rate of the skin that has been stripped of its outside layer (stratum corneum) ([U.S. EPA, 1992](#)). During pregnancy, the combined effect of endocrine, metabolic, mechanical, and blood flow alterations in the body cause a woman's skin to undergo substantial changes. Most of the changes are cosmetic in nature and are therefore not harmful or associated with risks to the mother or developing fetus ([Gabbe et al., 2007](#)). Most of the complaints of pregnancy-related skin changes can be expected to resolve or improve postpartum ([Gabbe et al., 2007](#)). Surface area of the skin, which is calculated as a function of height and weight, increases during pregnancy due to weight gain. Increased surface area and increased permeability may be important considerations for exposure to waterborne contaminants.

3.9. WEIGHT CHANGES

Differences in the quantity of food consumed by pregnant or lactating women may lead to weight loss or gain. Resistance to free leptin, a pregnancy-related hormone (see Section 3.6), is related to an increase in BMI during the middle of the pregnancy ([Widjaja et al., 2000](#)). In a study of 630 women from Galicia, Spain, study researchers concluded that leptin increases may be responsible for the postpartum weight gain observed in some women ([Lage et al., 1999](#)).

The differences in body weight can affect the potential dose received by the pregnant or lactating mother. While weight gain is a topic largely discussed and monitored in the three trimesters of pregnancy, weight loss is more closely associated with the postpartum lactation phase. However, while there is a large body of published research on weight gain during pregnancy, there is considerably less published literature on the issue of weight loss during pregnancy or the postpartum period. Morning sickness and associated nausea may lead to weight loss during pregnancy (see Section 4.4.3), and lactation does promote weight loss during the first year postpartum, particularly if breastfeeding continues for at least 6 months ([Dewey et al., 1993](#)).

[Cohen and Kim \(2009\)](#) used data from the Behavioral Risk Factor Surveillance System collected by the CDC to study sociodemographic and behavioral factors associated with attempting to lose weight during pregnancy. Using data collected from 1996–2003 on 8,036 pregnant women aged 18 to 44, [Cohen and Kim \(2009\)](#) reported that 8.1% of pregnant women intentionally try to lose weight during pregnancy, and that this behavior was associated

with maternal age (35–44 years), Hispanic ethnicity, obesity, lower income levels, alcohol consumption, and mental distress.

The recommended weight gain during pregnancy is between 25 and 35 pounds for normal weight women (BMI = 19.8 to 26 kg/m²) ([Brochu et al., 2006](#)), which amounts to the consumption of an additional 200 kcal/day ([Cox, 2003](#)). Recommendations for weight gain are slightly higher (28–40 pounds) in underweight women (BMI < 19.8 kg/m²) ([Brochu et al., 2006](#)) and slightly lower (15–25 pounds) in obese women (BMI > 26 kg/m²) ([Brochu et al., 2006](#)). As the fetus gains most of its weight during the last 2 months of pregnancy, it is recommended that women regulate their weight gain accordingly ([Cox, 2003](#)). Increased consumption to adequately support a pregnancy may affect exposures to environmental contaminants found in drinking water and food.

Reports indicate that only 30–40% of women actually gain weight within recommended ranges, while most women have inadequate prenatal weight gain ([Hickey, 2000](#)). A review of the literature to examine demographic, sociocultural, and behavioral factors associated with the risk of low prenatal weight gain among adult women with low and normal BMIs found that ethnicity, socioeconomic status, age, education, pregnancy intendedness or wantedness, prenatal advice, and psychosocial characteristics such as attitude toward weight gain, social support, depression, stress, anxiety, and self-efficacy may have an impact ([Hickey, 2000](#)). However, Hickey (2000) concluded that further identification and characterization of sociocultural and behavioral risk factors, along with reproductive and nutritional characteristics, are needed to help predict which women are most likely to have inadequate prenatal weight gain.

In adolescent pregnant women who are still growing and maturing, it has long been thought that any maternal statural growth occurring during pregnancy would be insignificant and have little impact on fetal growth. A study investigating growth and weight gain in this group of pregnant women found that maternal growth is prevalent and is associated with increased weight gain during pregnancy ([Scholl et al., 1993](#)). Postpartum measures indicated that growth and weight gain occurred in the pregnant adolescents even when caloric intakes were equivalent to pregnant, nongrowing adolescents or mature women. The infant birth weights of growing pregnant teens were reduced, an indication that fat reserves in growing pregnant adolescents support the mother's development rather than fetal growth.

Efforts to determine whether there are modifiable behavioral factors that can predict inadequate and excessive gestational weight gain in the U.S. population found that there are valid and easily implemented measures of change. Prepregnancy food intake, physical activity, and cigarette smoking during pregnancy were each statistically significantly and independently related to gestational weight gain ([Olson and Strawderman, 2003](#)). Women who consumed more or less food during pregnancy than prior to pregnancy had statistically significantly greater and

less gestational weight gain, respectively, compared to women who maintained similar food intake patterns during pregnancy as in prepregnancy. Decreased physical activity during pregnancy was associated with statistically significant greater gestational weight gain. Consumption of more than one and a half packs of cigarettes per day was associated with significantly lower gestational weight gain ([Olson and Strawderman, 2003](#)).

Major weight gain in pregnancy and subsequent weight retention 1 year postpartum were associated with the factors of gestational weight gain, postpartum exercise frequency, and food intake. Economic status also had an impact because lower income women gained more weight in pregnancy than the recommended amount and were at higher risk for weight retention ([Olson et al., 2003](#)). In another study investigating the patterns of maternal weight gain in pregnancy, factors that were associated with statistically significant differences in average weekly weight gain were parity, BMI, smoking habit, and raised blood pressure ([Dawes and Grudzinskas, 1991](#)). Additional research into the factors associated with maternal weight gain patterns found they vary according to trimester of pregnancy ([Abrams et al., 1995](#)). In a study of 10,418 women in California, the most important predictors of maternal weight gain were found to be Asian race-ethnicity and age in the first trimester; prepregnancy BMI, parity, and height in the second trimester; and hypertension, age, and parity in the third trimester ([Abrams et al., 1995](#)). Pregnant women, who successfully practiced dietary restraint to maintain a proper weight prior to pregnancy, reported lower levels of dietary restraint, were less dissatisfied with their body shape, and showed higher eating self-efficacy than nonpregnant women ([Clark and Ogden, 1999](#)).

4. BEHAVIORAL ADAPTATIONS AND PSYCHOLOGICAL CHANGES DURING PREGNANCY AND LACTATION

Behavioral changes in pregnant and lactating women differ from physiological changes in that the physiological changes passively happen to a woman, whereas the behavioral changes usually involve an active response from the woman. In other words, a woman may have more control over a behavioral change than she may have over a physiological change that occurs during pregnancy or lactation. However, the level of control a woman has may vary for different behavioral factors, with some being more controllable than others. Also, some women may make more of an effort to actively alter these behaviors than others. For instance, a pregnant woman may elect to stop smoking, reduce caffeine consumption, or eat a healthier diet to benefit the health of the fetus. On the other hand, a woman may experience uncontrollable feelings of depression, stress, fatigue, or irritability during pregnancy and modify her behavior as a result. These changes in behavior may impact her exposure to environmental contaminants.

While it is possible that a woman will experience most, if not all, of the physiological changes noted in the previous sections, it is possible that a woman may not undergo many, or even any, of the possible behavioral changes noted in the following sections. Many of the behavioral changes noted include those to benefit the health of the pregnant woman and her child. Recommended behavioral changes are associated with documented risk factors for adverse pregnancy outcomes; such risks include smoking, low prepregnancy weight, and inadequate weight gain during pregnancy ([Savitz et al., 2012](#)). There is some overlap between physiological and behavioral changes, since modifications in behavior may result from any of the physiological changes described in the preceding section.

Pregnancy or breastfeeding can be strong motivators for changing a woman's behavior ([Rayburn and Phelan, 2008](#)). The following sections describe the various behavioral changes or recommendations for change found in the literature. It is worth noting that many of the published articles do not describe a direct link between behavioral changes during pregnancy or lactation and the potential for exposures to environmental contaminants. However, the behavioral factors identified are presented as evidence of behavioral modifications commonly found in pregnant and lactating women. These behavioral modifications may result in exposures to environmental contaminants that are different in pregnant and lactating women from those of non-pregnant, non-lactating women. For that reason, the potential behavioral changes common to this lifestage are worth presenting.

4.1. ADAPTATIONS

During pregnancy and in the lactation period that follows, women may be advised to reduce or avoid a number of behaviors that may harm the health of the mother or the developing fetus. Among the key behaviors to be avoided are smoking, the consumption of caffeine and alcohol, and illicit drug use.

4.1.1. Smoking

Active smoking and secondhand exposure to smoke while pregnant and after is a concern because of linkages to premature births, fetal growth restrictions, low birth weight babies, pregnancy complications, and sudden infant death syndrome, among other health concerns. Tobacco smoke contains nicotine, carbon monoxide, and thousands of other compounds ([Cal/EPA, 2006](#)). Nicotine and carbon monoxide can cross the placenta and enter fetal tissues ([Jacqz-Aigrain et al., 2002](#); [Jauniaux et al., 1999](#)). Quitting smoking and reducing exposure to second-hand smoke during pregnancy can eliminate exposure to environmental contaminants contained in the inhaled cigarette smoke as well as reduce the risk of adverse pregnancy outcomes ([Mund et al., 2013](#)).

In a study designed primarily to assess alcohol use among pregnant women, respondents were also asked about their current smoking and intended smoking in future pregnancies. When asked about their smoking behavior, 16% of the respondents indicated that they had smoked during their last pregnancy, 5% intended to continue smoking if they were planning to become pregnant, and 4% intended to smoke if they became pregnant in the future ([Peadon et al., 2011](#)). Based on data from the 2009/2010 National Survey on Drug Use and Health (NSDUH), 16.3% of pregnant women aged 15–44 years smoked cigarettes compared to 26.7% of nonpregnant women in the same age group ([Behnke and Smith, 2013](#); [SAMHSA, 2011](#)) (see Table 4-1). The NSDUH is an annual survey of the about 67,000 civilian, noninstitutionalized people in the United States ages 12 years or older.

Table 4-1. Comparison of cigarette, alcohol, and illicit drug use among pregnant and nonpregnant women, aged 15–44 years, based on the 2009/2010 NSDUH

Behavior	Pregnant Women	Nonpregnant Women
Cigarette use	16.3%	26.7%
Alcohol use	10.8%	54.7%
Binge drinking	3.7%	24.6%
Heavy alcohol use	1.0%	5.4%
Illicit drug use	4.4%	10.9%

Source: [SAMHSA \(2011\)](#).

A recent study examining psychosocial factors found that women who were less educated and unmarried, who were living below the poverty level, whose partners smoked or suggested an abortion, or who had mental health problems were identified as more likely to be smokers while pregnant or postpartum ([Page et al., 2012](#)). In a study examining smoking and smoking cessation behaviors among U.S. pregnant women, sociodemographic correlates of smoking cessation in pregnancy were investigated ([Yu et al., 2002](#)). Four categories of smoking behavior were analyzed: nonsmoking at last pregnancy, persistent smoking throughout pregnancy, attempting unsuccessfully to quit during pregnancy, and successfully quitting during pregnancy. Results of this study showed that the factors most strongly associated with attempts to quit smoking were Hispanic ethnicity and the combined effect of age and smoking duration. Race was shown not to impact smoking cessation in a study of prenatal smoking cessation among U.S. women that found a similar level of spontaneous cessation for black (46.8%) and white (43.3%) women who quit smoking when they learned they were pregnant ([Orr et al., 2007](#)).

In another study, Hispanic mothers were identified as being more likely to smoke postpartum than to smoke throughout their pregnancies ([Page et al., 2012](#)). In addition, socioeconomically disadvantaged women from ethnic minority groups were found to be more likely to smoke before pregnancy and postpartum ([Hawkins et al., 2010](#)). Urban minority pregnant women were significantly more likely to continue smoking during pregnancy when they also reported symptoms of depression ([Tan et al., 2011](#)).

A study of U.K. women ([Morris et al., 2008](#)) investigated whether women in their second or subsequent pregnancy (multigravid) were more or less likely than women pregnant for the first time (primigravidae) to change their smoking behavior. While in general, women who reported smoking before pregnancy showed a decreasing trend in continuing to smoke the same

amount after recognizing their pregnancy, the multigravid smokers were almost 75% more likely than primigravid smokers to continue to smoke while pregnant with no change in consumption. However, women who were breastfeeding smoked less than nonbreastfeeding women, and prolonged breastfeeding was found to reduce the risk of smoking relapse ([Lauria et al., 2012](#)).

4.1.2. Caffeine Consumption

Although there is no general agreement among researchers, some studies concerning caffeine consumption during pregnancy suggest that reducing caffeine consumption improves fetal outcome. These studies advise that caffeine consumption should be reduced or avoided during pregnancy because of a concern with increased pregnancy loss or fetal growth retardation ([Weng et al., 2008](#); [Klebanoff et al., 1999](#); [Mills et al., 1993](#)). However, there are others that found no evidence to indicate that moderate caffeine use increased the risk of spontaneous abortion, intrauterine growth retardation, or microcephaly after accounting for other risk factors ([Mills et al., 1993](#)). Although caffeine consumption itself may or may not affect fetal outcome, exposure to contaminants may occur if they are present in the source water used to make coffee or tea (e.g., furan, PAHs, ochratoxin A, cadmium, cobalt) ([Guenther et al., 2010](#); [Houessou et al., 2007](#); [Vargas et al., 2005](#); [Horwitz and van der Linden, 1974](#)). Thus, reducing or avoiding the consumption of coffee or tea may reduce a pregnant or lactating woman's exposure to those environmental contaminants.

The [March of Dimes \(2012\)](#) recommends that women who are pregnant or trying to get pregnant limit their caffeine intake to 200 mg/day. [Knight et al. \(2004\)](#) used data from the 1999 Share of Intake Panel (SIP), a marketing research program, which contained data for more than 10,000 caffeinated beverage consumers to estimate caffeine consumption in pregnant and nonpregnant women. Caffeine consumption among pregnant women averaged 58 mg/day compared to 91 and 109 mg/day for 20–24 and 25–34-year-old nonpregnant women, respectively. Coffee was the major source of caffeine consumption. For comparison purposes, using data from the 1994–96 Continuing Survey of Intake by Individuals (CSFII), [USDA \(2000a\)](#) estimated caffeine intake to be 143, 209, and 250 mg/day for 20–29- ($N = 720$), 30–39- ($N = 816$), and 40–49- ($N = 902$) year-old women, respectively (data were not reported for pregnant or lactating women).

4.1.3. Alcohol Use

There are a variety of contaminants in alcohol, including aluminum, cadmium, and ochratoxin A ([Battilani et al., 2006](#); [Lopez et al., 1998](#); [Mena et al., 1996](#)). Alcohol consumption during pregnancy is a concern because of the possible teratogenic effects on the offspring as well as multiple congenital abnormalities, developmental delays, and behavioral changes ([Ornoy and](#)

[Ergaz, 2010](#); [Grisso et al., 1984](#)). Many women who drink alcoholic beverages regularly before becoming pregnant either reduce their alcohol intake or stop drinking during pregnancy ([Takimoto et al., 2003](#)), thereby reducing their exposure to contaminants in alcohol. In a study of drinking behavior, 4.6% of women reported drinking an average of one alcoholic drink per day by the end of the third trimester of pregnancy, compared with 44% before pregnancy ([Day et al., 1989](#)). Similar results were found in a U.S. government survey, which indicated that about 13% of pregnant women drink alcohol during pregnancy and about 3% of pregnant women report binge drinking (five or more drinks on any one occasion) or frequent drinking (seven or more drinks per week) ([CDC, 2004](#)). Based on data from the 2009/2010 NSDUH, 10.8% of pregnant women aged 15–44 years used alcohol compared to 54.7% of nonpregnant women in the same age group ([Behnke and Smith, 2013](#); [SAMHSA, 2011](#)) (see Table 4-1). Only 3.7% of pregnant women reported binge drinking compared to 24.6% of nonpregnant women. Heavy alcohol use was reported by 1.0% of pregnant women compared to 5.4% of nonpregnant women.

In a study of pregnancy-related changes in alcohol consumption between black and white women, white women were more likely to reduce both drinking and binge drinking behavior during their pregnancies ([Morris et al., 2008](#)). The study population was comprised of 280,126 non-Hispanic white or black women, aged 18–44, selected for the years 2001–2005 from the CDC’s Behavioral Risk Factor Surveillance System, a national telephone survey. Results of the survey showed that pregnant white women averaged 38% fewer drinks and had a 33% greater reduction in binge drinking than pregnant black women in the study. Groups also seen to reduce their drinking or binge drinking were pregnant younger women (aged 18–33) and pregnant women with more than a high school education. Smoking status was the greatest predictor of drinking behavior for both pregnant and nonpregnant women; pregnant smokers were more than 2.5 times more likely to drink and more than 4 times as likely to binge drink as pregnant nonsmokers.

To examine the attitudes and behavior of women regarding alcohol use during pregnancy, 1,103 nonpregnant Australian women of childbearing age were interviewed by telephone ([Peadon et al., 2011, 2010](#)). The majority of respondents (93%) agreed that alcohol can affect the fetus, but a small percentage (16%) believed that the effects on the fetus were transient. Women with higher education levels were more likely to know the effects of alcohol consumption on the fetus. Of those respondents who had been pregnant in the past, 34% drank alcohol while they were pregnant and 31% intended to consume alcohol in a future pregnancy. Education level and knowledge about the effects of alcohol consumption were not associated with the respondent’s attitudes regarding alcohol consumption in pregnancy.

4.1.4. Other Adaptations

In addition to the key avoidances of smoking, caffeine, and alcohol, pregnant and lactating women are also cautioned to make other behavioral changes to benefit their health and the health of their child. Many of these adaptations would serve to reduce a pregnant or lactating woman's exposure to environmental contaminants. For example, the March of Dimes recommends that pregnant women avoid marijuana, ecstasy, methamphetamines, physical abuse, foods that may contain mercury (e.g., certain fish), rodents, lead, solvents, paints, pesticides, benzene, formaldehyde, and carbon monoxide. They also caution against the use of street drugs, over-the-counter drugs, prescription drugs, certain dietary supplements, herbal preparations, and other medications that have not been approved by a doctor who is aware of the impact of exposure of pregnant women to potentially harmful substances ([March of Dimes, 2013b](#)). [SAMHSA \(2011\)](#) found that illicit drug use was lower among pregnant women than nonpregnant women of reproductive age. During 2009/2010 an estimated 4.4% of pregnant women aged 15–44 years used illicit drugs compared to 10.9% of nonpregnant women in the same age group. Illicit drug use was highest (16.2%) among pregnant women aged 15–17 years and lowest (1.9%) for pregnant women aged 26–44 years ([Behnke and Smith, 2013](#); [SAMHSA, 2011](#)) (see Table 4-1).

In addition to avoidances, the March of Dimes also recommends other adaptations pregnant women should actively practice to foster a healthy environment for the mother and child while pregnant and lactating, including the consumption of prenatal vitamins and minerals, the addition of calcium in the diet, a healthy diet, and exercise.

4.2. DEPRESSION

Depression is not only a disorder in which one feels sad, or depressed, but also is a condition that can manifest itself in a host of additional symptoms and behavioral changes that can include appetite disturbance or significant weight change, sleep loss or excessive sleep, psychomotor agitation/retardation, fatigue or energy loss, feelings of worthlessness or guilt, impaired thinking or concentration, and suicidal ideation. These symptoms cover a wide range of behaviors and depression can therefore look vastly different from person to person ([APA, 2000](#)). Changes in appetite or behavior as a result of depression may result in either reduced or increased exposure to environmental contaminants in pregnant and lactating women.

4.2.1. Prevalence of Depression

Each year approximately twice as many women (12.0%) as men (6.6%) suffer from a depressive disorder ([Regier et al., 1993](#)). According to the American College of Obstetricians and Gynecologists (ACOG), reproductive-age women have the highest prevalence of major

depressive disorders; with approximately 1 in 10 women at risk for having major or minor depression sometime during pregnancy and in the postpartum period ([ACOG, 2006](#)). Women between the ages of 25 and 44 years are at the greatest risk for a major depressive episode, which is the primary age bracket for childbearing. Approximately, 10–20% of women suffer from depression during pregnancy or in the first 12 months postpartum ([NIHCM, 2010](#)). Hormonal changes during pregnancy, genetics, and psychosocial factors can trigger depression ([NIHCM, 2010](#)). Depressive disorders are the leading cause of disease burden for women aged 15–44 years ([WHO, 2008](#)). Unfortunately, major depressive episodes during pregnancy are often undiagnosed and untreated as illustrated in a large sample ($N = 3,472$) of pregnant women screened in obstetric settings, in which 20% had significant symptoms and only 13.8% were receiving treatment ([Marcus et al., 2003](#)).

4.2.2. Drug Treatment for Depression

Drug treatment during pregnancy and lactation has been shown to be effective in treating depression. In a study of the prevalence of medication use among pregnant women in Boston and Philadelphia, [Mitchell et al. \(2011\)](#) found that the use of antidepressants increased from <1% in 1976–1990 to 7.5% in 2006–2008. [Hayes et al. \(2012\)](#) suggests that the prevalence of medication therapy to treat depression during pregnancy is about 4–10% in the United States and Canada. Selective serotonin reuptake inhibitors are medications commonly used to treat depression ([ACOG, 2006](#)), and they accounted for the majority of the antidepressants taken by pregnant women in recent years ([Mitchell et al., 2011](#)). However, given the potential for harm to the developing fetus, women previously diagnosed with depression prior to pregnancy often choose to discontinue the use of this medication during pregnancy ([NIHCM, 2010](#)).

4.2.3. Factors Impacting Depression in Pregnancy and Lactation

This section provides a summary of the additional studies related to depression that were found in the literature and that provide information on factors that have been shown to make some women more prone to depression and the subsequent behavioral changes that occur. There is a large body of relevant research that addresses changes in maternal health from depression in pregnant and lactating women. Race/ethnicity, age, and socioeconomic status are found to be good predictors of maternal depression ([NIHCM, 2010](#)). One study found that education, material deprivation, and subjective social standing were independently associated with all health measures ([Stewart et al., 2007](#)). After adjusting for all socioeconomic status variables, there were racial/ethnic disparities remaining in depression rates for all minority groups, and disparities in self-rated health for Asian/Pacific Islanders. In another socioeconomic-related study, changes in health status experienced by a multiethnic cohort of women during and after

pregnancy were characterized ([Haas et al., 2005](#)). Insufficient money for food or housing and lack of exercise were associated with prevalence of depressive symptoms before, during, and after pregnancy. The study supports the common finding that depressive symptoms may be more prevalent during the postpartum period than during pregnancy.

In an investigation of hormones and metabolism on depression in women, a study of neuroactive ring A-reduced metabolites of progesterone in human plasma during pregnancy measured elevated levels of 5 α -dihydroprogesterone in depressed patients during the latter half of pregnancy. Mean levels of progesterone metabolites tended to be higher in depressed patients compared with controls, and this difference reached statistical significance for 5 α -dihydroprogesterone both at 27 weeks and at 37 weeks of gestation. A marked rise in all of the progesterone metabolites was found during pregnancy suggesting that these metabolites may be involved in the mood changes of pregnancy and early postpartum period ([Pearson Murphy et al., 2001](#)).

In another study investigating the impact of metabolism, the relationship between thyroid status during late pregnancy and antenatal and postpartum depression scores was studied ([Pedersen et al., 2007](#)). Thyroid measures were obtained at 32–35, 36, and 37 weeks of pregnancy in women with normal range thyroid hormone levels. Pregnant women with antenatal total and free T4 concentrations in the lower euthyroid range may be at greater risk of developing postpartum depressive symptoms.

Finally, a longitudinal study of women's mental and physical health from pregnancy through 6 months postpartum was conducted to determine whether health was related to length of maternity leave by investigating changes in women's mental and physical health around the time of childbirth ([Gjerdingen et al., 1991](#)). The study of first-time mothers revealed that while many physiological symptoms resolve soon after delivery, there are lingering physical and emotional symptoms that persist. There was an increase in depressive symptoms for new mothers from pregnancy to the 6th week postpartum, with a subsequent decline thereafter. In addition, from pregnancy to the 6th postpartum month, the number of days that mothers were ill due to infections steadily increased. A significant decline in depressive symptoms was observed from the prenatal period through the 6th postpartum month for women who did not return to work during the period of the study.

4.3. STRESS, ANXIETY, IRRITABILITY, SLEEP, AND FATIGUE

The terms “anxiety” and “stress” are sometimes used interchangeably by laymen, but in the medical community the terms have distinct definitions. Clinically, anxiety is defined as a feeling of apprehension or fear. In extreme cases, anxiety can manifest itself in behaviors such as a phobia, avoidance, posttraumatic stress disorder, or obsessive-compulsive disorder ([Gabbe](#)

[et al., 2007](#)). On the other hand, stress can arise from any situation or thought that causes feelings of frustration, anger, nervousness, or even anxiousness and produces the “fight or flight” response that can lead to positive adaptive behaviors or negative behaviors such as social withdrawal, drug or alcohol abuse, overeating, undereating, or angry outbursts. Changes in appetite or behavior in pregnant and lactating women as a result of anxiety, stress, irritability, lack of sleep, and fatigue may result in either reduced or increased exposure to environmental contaminants.

Anxiety can have a number of impacts on pregnant or lactating women. Anxiety is cited as one of many factors that may potentially influence maternal weight gain. [Hickey \(2000\)](#) suggested that stress may result in neuroendocrine-mediated alterations in prenatal energy metabolism that may be responsible, in part, for low weight gain. Lactating women suffer less anxiety over regulation of food and fluid intake as they tend to be “significantly more calm,” both before and after meals, than either nonpregnant or nonlactating women, based on subjective self-ratings ([Heck and de Castro, 1993](#)).

4.4. CHANGES IN DIETARY BEHAVIORS

Dietary behaviors may change during pregnancy and lactation as a result of the nutritional needs, energy requirements, or cravings or aversions of the mother. These dietary changes may influence environmental exposures for pregnant and lactating women.

4.4.1. Nutritional Needs

Pregnant and lactating women require a wide range of nutrients to support the health of both the mother and the infant. The U.S. Department of Agriculture (USDA) and U.S. Department of Health and Human Services (USDHHS) published 2010 Dietary Guidelines for Americans to assist consumers in selecting the types and amounts of foods that are appropriate for their age, gender, and activity levels ([USDA, 2010](#)). Pregnant or lactating women have special nutritional needs. An online daily food plan tool was created by USDA to provide guidance to pregnant and lactating women on specific nutritional needs based on their age, height, weight, physical activity level, and stage of pregnancy or breastfeeding status (www.choosemyplate.gov/supertracker-tools/daily-food-plans/moms.html). Data available on intake rates of various food items by pregnant/lactating women are provided in Section 5.

Generally, nutritional research efforts focus on understanding nutrient intake and the resulting impacts on women during periods of pregnancy and lactation. A popular nutrient of interest is calcium. Calcium is used by various systems throughout the body. When a woman does not get enough calcium from her diet the body mobilizes it from her bones (see Section 3.4). Over time, this loss may weaken bone and lead to osteoporosis. Because fetal

growth requires extra calcium to build healthy teeth and bones, pregnancy also places added calcium demands on women that can increase the leaching of calcium and environmental pollutants (e.g., lead, cadmium) from bones ([WHO, 2013](#); [Alba et al., 2012](#)). Average calcium intake during pregnancy has been measured at 1,526 mg/day, 1,622 mg/day during lactation, and 1,756 mg/day in nonpregnant women ([Drinkwater and Chesnut, 1991](#)). Nonpregnant women in the study were counseled by a registered dietician to maintain a calcium intake of 1,500 mg/day, either by increasing intake of dairy products or by adding supplements. Pregnant women in the study initially followed the same protocol as nonpregnant women, but then followed their physician's advice regarding calcium intake. Increased dietary calcium intakes have been shown to improve calcium balance and may minimize bone loss across pregnancy and lactation in women with habitual intakes of <500 mg calcium/day ([O'Brien et al., 2006](#)).

An adequate diet rich in calcium can also provide protection for women in cases of exposure to lead ([Hernandez-Avila et al., 1996](#)). A study of bone lead levels in recently postpartum Mexico City women found that consumption of foods with high calcium content may protect against the accumulation of lead in bone ([Hernandez-Avila et al., 1996](#)). Low consumption of milk and cheese, as compared to the highest consumption category (every day), was associated with an increase in tibia bone lead of 9.7 µg of lead/g of bone mineral ([Hernandez-Avila et al., 1996](#)). Because there is some evidence that mobilization of lead from bone may be markedly enhanced during the increased bone turnover of pregnancy and lactation—potentially resulting in lead exposure to the fetus and the breastfed infant—the potential for delayed toxicity from bone lead stores remains a significant public health concern.

Vitamin D promotes calcium absorption and is needed for bone growth and remodeling ([NIH, 2014](#)). During pregnancy, adequate Vitamin D levels are needed to meet the demands of the growing fetus ([Specker, 2004](#)). Sufficient levels of Vitamin D in lactating women are needed to prevent rickets in breastfeeding children ([CDC, 2015](#)). The Institute of Medicine's (IOM) recommended daily allowance (RDA) of Vitamin D for all women, including those who are pregnant and lactating, is 600 International Units (IU) (15 µg/day). For pregnant and lactating women ages 19–50 years who are at risk of Vitamin D deficiency, the RDA is 1,500–2000 IU (37.5–50 µg/day) ([Holick et al., 2011](#)). Using data from the 2001–2006 National Health and Nutrition Examination Survey (NHANES), [Looker et al. \(2011\)](#) evaluated the Vitamin D status of the U.S. population. Approximately 24% were “at risk of inadequacy,” 8% were “at risk of deficiency,” and 1% had levels that could possibly be harmful. However, [Looker et al. \(2011\)](#) also found that pregnant and lactating women were less likely to be Vitamin D deficient than nonpregnant women. Selenium is another important dietary mineral during pregnancy. According to [ATSDR \(2003\)](#), “selenium is a biologically active part of a number of important proteins, particularly enzymes involved in antioxidant defense mechanisms (e.g., glutathione

peroxidases), thyroid hormone metabolism (e.g., deiodinase enzymes), and redox control of intracellular reactions (e.g., thioredoxin reductase).” Deficiency in selenium has been associated with adverse outcomes of pregnancy ([Kantola et al., 2004](#)). In a study of pregnant Finnish and Estonian women, selenium concentrations were 10–30% lower at term relative to preconception, regardless of the significant differences in selenium status among the different mothers ([Kantola et al., 2004](#)). Based on this decline and observations of higher cord-blood selenium concentrations than maternal whole blood levels, active transportation of selenium to the fetus is inferred. Evidence suggests that selenium has an active role in the mother’s defense systems against the toxicity of environmental pollutants and chemical stress, including the constituents of cigarette smoke ([Kantola et al., 2004](#)).

Iron demand increases during pregnancy as a result of the expanded blood volume and growth of the fetus, placenta, and other maternal tissues ([Mei et al., 2011](#)). The recommended daily allowance for iron is 27 mg/day for pregnant women and 10 mg/day for lactating women ([NIH, 2015](#)). [Mei et al. \(2011\)](#) analyzed data from the 2001–2006 NHANES to assess the iron status of pregnant women in the United States, and found that 18% of pregnant women were iron deficient, with iron deficiency increasing over the course of pregnancy from approximately 7% in the first trimester to 30% in the third trimester. Dietary intake of folic acid is also recommended for women of childbearing age and pregnant women in order to reduce the infant’s risk of spina bifida or other neural tube defects ([CDC, 1992](#)). [Branum et al. \(2013\)](#) analyzed folic acid and iron supplement intake data from 1,296 pregnant women who participated in the NHANES, 1999–2006. Results indicated that approximately 55–60% of women in their first trimester took a folic acid- or iron-containing supplements compared with 76–78% in their second trimester and 89% in their third trimester. Among all pregnant women in the survey that were taking folic acid supplements ($N = 761$), the mean supplemental folic acid intake was 817 ± 27.6 $\mu\text{g/day}$ ([Branum et al., 2013](#)). Among those taking iron supplements, supplemental iron intake was 47.7 ± 4.2 mg/day ([Branum et al., 2013](#)). In an earlier study, using data from the 1988–1994 NHANES, [Cogswell et al. \(2003\)](#) found that 72% of pregnant women and 60% of lactating women consumed supplements containing iron, compared to 23% of nonpregnant, nonlactating women aged 19–50 years.

4.4.2. Energy Requirements

There is a sizable amount of research on the topic of energy requirements during pregnancy and lactation. During pregnancy, more calories, protein, and other nutrients are required for the growth of the fetus, placenta, and uterus ([Landau, 1983](#)). According to [Fowles \(2006\)](#), the USDA recommends no increase of caloric intake during the first trimester, an increase of 340 kcal/day during the second trimester, and 450 kcal/day during the third trimester.

In 2000, the USDA recommended the number of servings for each of the food groups in the food pyramid for three caloric levels (i.e., 1,600 kcal; 2,200 kcal; 2,800 kcal) for pregnant women ([USDA, 2000b](#)) (see Table 4-2). More recently, the USDA replaced the food pyramid with an interactive tool that allows the user to determine the adequate number of servings of each food group that the pregnant mother should eat based on a personal profile, which includes information on age, weight, height, and level of physical activity (<http://www.choosemyplate.gov/pregnancy-breastfeeding/pregnancy-nutritional-needs.html>).

Table 4-2. Recommended number of servings for three caloric intake levels for pregnant women

Food group	Calorie level (kcal)		
	1,600	2,200	2,800
Bread (grain group)	6	9	11
Vegetable group	3	4	5
Fruit group	2	3	4
Milk group	3	3	3
Meat group (ounces)	5	6	7

Source: [USDA \(2000b\)](#).

A U.S. study by [Rifas-Shiman et al. \(2006\)](#) assessed changes in food and nutrient intake from the first to second trimester of pregnancy. Whereas diet in the first trimester may be more important to fetal development and differentiation of various organs, maternal diet later in pregnancy may be important for overall fetal growth as well as for brain development. The study authors examined individual-level changes in food and nutrient intake from the first to second trimester of pregnancy. The mean energy intake reported for the first trimester, 2,046 kcal, was similar to the mean intake reported during the second trimester, 2,137 kcal, but the food and nutrient intakes changed. The foods and energy-adjusted nutrients from foods for which overall mean intakes increased more than 5% from the first to second trimester were skim or 1% dairy foods (22%), whole-fat dairy foods (15%), red and processed meat (11%), saturated fat (6%) and vitamin D (7%). On the other hand, intake of caffeinated beverages decreased by about 30% and alcoholic beverages decreased about 88%. Mean multivitamin intake increased by 35% from the first to second trimester, thereby increasing the total micronutrient intake ([Rifas-Shiman et al., 2006](#)).

Dietary evaluations of calories and energy intake have also been performed on women during pregnancy and lactation. In a study of U.S. pregnant women, the average daily energy intake was reported as 1,955 kcal or 28.5 kcal/kg for the latter half of gestation ([Blackburn and Calloway, 1976](#)). Pregnant women had a mean protein intake of 1.17 g/kg-day, which represented 17% of gross energy consumed. Energy intake in lactating women was 30 kcal/kg, which represented 74% of need when adjusted for milk production. Average energy intake of nonlactating women was 19 kcal/kg, with protein intake representing 19% of energy consumed for both groups of lactating and nonlactating women ([Blackburn and Calloway, 1976](#)).

A comparison of dietary intake in U.S. women during lactation at 6 weeks postpartum to intake in two groups of nonpregnant, nonlactating women was performed to determine the regulation of food and fluid intake in lactating women ([Heck and de Castro, 1993](#)). Lactating women did not differ from body weight-matched, nonlactating controls in their total daily intakes or their meal patterns, but they did consume a significantly smaller percentage of the recommended dietary allowances per day than did their nonlactating counterparts. The lack of compensation in intake for lactating women to meet the caloric demands of lactation either indicates that the lactating women catabolize weight gained during pregnancy faster than accounted for in the recommended dietary allowance, or that lactating women increase their metabolic efficiency ([Heck and de Castro, 1993](#)).

4.4.3. Cravings and Aversions

Food cravings and aversions that occur during periods of pregnancy and lactation can greatly influence the types and amounts of food consumed. One functional hypothesis, known as the maternal-embryo protection hypothesis, proposes that pregnant women may avoid certain foods that can contain toxins or pathogens in order to protect themselves or the developing fetus ([Steinmetz et al., 2012](#)). Alternatively, food cravings during pregnancy have been suggested to promote maternal intake of beneficial foods containing needed nutrients, or that the foods that are craved relieve the nausea and vomiting associated with morning sickness ([Weigel et al., 2011](#)).

A study of taste changes across pregnancy evaluated reactions to salty, sweet (sucrose), sour (citric acid), and bitter (quinine hydrochloride) stimuli in both pregnant women and controls ([Duffy et al., 1998](#)). Stimuli were evaluated on intensity (scale ranging from “nothing” to “extremely”) and hedonistic value (pleasant or unpleasant). The study authors indicate the taste intensity and hedonistic changes across pregnancy could serve to support healthy pregnancy outcomes: increases in bitter intensity in first trimester to protect against ingesting poisons; changes in salty, sour, and bitter preference later in pregnancy to support ingesting a varied diet ([Duffy et al., 1998](#)).

A laboratory study on the taste and specific food consumption changes across the course of pregnancy found that women in the second trimester had an increased preference for sweet food, but not salty or nonsweet/nonsalty food, as compared with women during other points in pregnancy ([Bowen, 1992](#)). However, the pregnant women who participated in the study did not exhibit excessive weight gain. One reason for this may have been that these women were generally classified as “restrained eaters” and so they may have refrained from daily consumption of excess sweet foods ([Bowen, 1992](#)).

The patterns in food choices from prepregnancy through midpregnancy and 2 years postpartum were investigated along with other factors, including breastfeeding behavior ([Olson, 2005](#)). The food choice behaviors evaluated were consuming ≥ 2 cups of milk per day, consuming ≥ 3 fruits and vegetables per day, and eating a daily breakfast. The results showed a significant increase in the proportion of women engaging in these three behaviors during pregnancy compared to prepregnancy. Approximately 66% of the women breastfed for any length of time and over 33% of the women breastfed for 1 year or more.

Pregnancy may result in dietary changes among women who diet and/or experience eating disorders. The impact of pregnancy on eating disorders, dietary habits, and body image perception was studied in a population of pregnant women with positive and negative histories of dieting and/or an eating disorder diagnosis ([Rocco et al., 2005](#)). Pregnancy had a protective effect in the groups of women as their concerns with shape, body attitude, thinness, and daily worries were reduced in comparison with the obligations to the child ([Rocco et al., 2005](#); [Baker et al., 1999](#)). Another study observed that symptoms of eating disorders (anorexia nervosa, bulimia nervosa, or binge eating) that lead to weight loss diminish during pregnancy, but return to baseline levels postpartum ([Crow et al., 2008](#)). Factors significantly associated with binge eating during pregnancy include sexual and physical abuse, anxiety and depression, low self-esteem and low life satisfaction, smoking, alcohol use, and lack of social support ([Knoph Berg et al., 2011](#)).

Morning sickness during pregnancy can also impact food cravings and aversions. In a study of the association between morning sickness symptoms and dietary preferences, cravings, and aversions in pregnant women, [Crystal et al. \(1999\)](#) found that women reported more aversions during pregnancy than before pregnancy. Also, women with more severe pregnancy symptoms reported a greater number of aversions both before and during pregnancy than women with less severe morning sickness.

Some pregnant women may also crave and ingest nonfood substances. This is known as pica behavior. The term “pica” generally refers to behavior associated with the intentional ingestion of foreign (i.e., nonfood or nonnutritive) substances ([Bronstein and Dollar, 1974](#)). The

types of materials ingested can include: dirt, clay, cigarette ashes, ice, freezer frost, flour, baking soda or powder, cornstarch, powdered milk ([Cooksey, 1995](#)), or other materials.

Studies have indicated that pica behavior may be more prevalent among pregnant women than among nonpregnant women, and some researchers have theorized that this behavior may result from the desire to satisfy cravings or hunger due to poor nutrition, the need to supplement minerals (e.g., calcium or iron) in the diet, cultural practices, or other physiological needs or behaviors ([Bronstein and Dollar, 1974](#)). Others have suggested that geophagy (a specific type of pica in which soil or clay are ingested) ([ATSDR, 2001](#)) among pregnant women is best explained as protection against symptoms of gastrointestinal distress and the effects of harmful chemicals, parasites, and pathogens ([Young et al., 2011](#); [Young, 2010](#)). The behavior has been more commonly found among socioeconomically disadvantaged women in rural and immigrant communities, and in women of African heritage ([Kim and Nelson, 2012](#)). Pregnant or lactating women who engage in pica behavior may be exposing themselves to environmental contaminants present in soil or other nonfood substances that they ingest. Information about prevalence of this behavior and the amounts of substances ingested is discussed in Section 5.

5. EXPOSURE FACTORS FOR PREGNANT/LACTATING WOMEN

Exposure factors are factors related to human behavior and characteristics that help determine an individual's exposure to an agent ([U.S. EPA, 2011](#)). For example, these include water and food intake, inhalation rates, nondietary (e.g., soil) ingestion rates, time spent at various microenvironments and activities, body weight, and use of consumer products. Due to the physiological and behavioral changes that occur during pregnancy and lactation, exposure factors for pregnant and lactating women may be different than those of the general population of women and they may, in turn, impact the fetus or newborn. The following sections summarize the available exposure factor data for women during this lifestage. Data for pregnant women are presented by trimester where available.

5.1. WATER INTAKE

Pregnant and lactating women tend to increase their consumption of water to support the physiological requirements of the growing fetus and to produce milk. [Ershow et al. \(1991\)](#) used data from a 1977–1978 national dietary survey to evaluate drinking water intake rates for pregnant and lactating women. In general, lactating women ingested more water than pregnant women and pregnant women ingested more water than control women. The 3-day average total fluid intake rates (mean \pm SD) were $1,940 \pm 686$ mL/day for control women, $2,076 \pm 743$ mL/day for pregnant women, and $2,242 \pm 658$ mL/day for lactating women (see Table 5-1). Tapwater intake rates were also calculated. Mean \pm SD tapwater intake rates were estimated to be $1,157 \pm 635$ mL/day for control women; $1,189 \pm 699$ mL/day for pregnant women; and $1,310 \pm 591$ mL/day for lactating women ([Ershow et al., 1991](#)). Because these rates are based on data that were collected more than three decades ago, they may not reflect current tapwater intake rates. For example, because the consumption of bottled water has increased in the United States since the 1977–1978 survey was conducted, the results are likely to overstate current consumption patterns for tapwater ([Burmester, 1998](#)).

Table 5-1. Tapwater and total fluid intake among pregnant, lactating, and control women, based on a 1977–1978 dietary survey (mL/day)

	Pregnant N = 188	Lactating N = 77	Control N = 6,201
Tapwater			
Mean ± SD	1,189 ± 699	1,310 ± 591	1,157 ± 635
Median	1,063	1,330	1,065
95 th percentile	2,424	2,191	2,310
Total Fluid			
Mean ± SD	2,076 ± 743	2,242 ± 658	1,940 ± 686
Median	2,164	1,928	1,835
95 th percentile	3,475	3,353	3,186

N = Number of observations.
SD = Standard deviation.

Source: [Ershow et al. \(1991\)](#).

[Zender et al. \(2001\)](#) conducted a study in Colorado in 1996 and 1997 to compare tapwater intake among pregnant and nonpregnant women. A total of 71 pregnant and 43 nonpregnant women were recruited from Women, Infant, and Children (WIC) clinics. Nearly one-half of the pregnant women were in their second trimester, and one-quarter were in each of the first and third trimesters. Total tapwater intake included tapwater consumed directly as a beverage and tapwater-based cold and hot beverages. Information on the sources of the water consumed (e.g., tapwater, bottled water, or filtered water) were also collected. Total tapwater intake was slightly higher for pregnant women (3.4 L/day) than nonpregnant women (3.0 L/day) (see Table 5-2). The proportions of each principal source of drinking water to total water intake for pregnant and for nonpregnant women (see Table 5-3) were similar for bottled water (14% and 12%), filtered water (11% and 16%) and tapwater (75% and 72%). Seventeen percent of pregnant women reported altering their source of drinking water after they became pregnant.

Table 5-2. Water ingestion rates by pregnancy status (L/day) for a population of women in Colorado

Characteristic		Mean	SD	Percentiles			Mean	SD	Percentiles		
				25 th	50 th	75 th			25 th	50 th	75 th
		Pregnant (N = 71)					Nonpregnant (N = 43)				
Home	Cold tapwater	1.8	1.4	0.9	1.4	2.3	1.3	1.0	0.5	0.9	2.0
	Cold tapwater-based beverages	1.0	0.8	0.7	0.9	1.4	0.9	0.6	0.4	0.7	1.2
	Hot tapwater-based beverages	0.1	0.2	0.0	0.0	0.2	0.2	0.5	0.0	0.0	0.2
	Total tapwater intake	2.9	1.8	1.8	2.3	3.7	2.4	1.2	1.5	2.3	2.9
		Pregnant (N = 36)					Nonpregnant (N = 23)				
Work	Cold tapwater	0.7	0.6	0.2	0.4	1.3	1.0	1.2	0.4	0.7	1.4
	Cold tapwater-based beverages	0.1	0.3	0.0	0.0	0.0	0.1	0.3	0.0	0.0	0.0
	Hot tapwater-based beverages	0.1	0.2	0.0	0.0	0.0	0.1	0.1	0.0	0.0	0.0
	Total tapwater intake	0.9	0.7	0.4	0.7	1.4	1.2	1.2	0.5	0.9	1.4
		Pregnant (N = 71)					Nonpregnant (N = 43)				
TOTAL	Cold tapwater	2.1	1.5	1.1	1.8	2.8	1.8	1.6	0.7	1.5	2.7
	Cold tapwater-based beverages	1.1	0.8	0.7	0.9	1.4	0.9	0.6	0.4	0.9	1.4
	Hot tapwater-based beverages	0.2	0.3	0.0	0.0	0.2	0.3	0.5	0.0	0.0	0.4
	Total tapwater intake	3.4	1.8	2.0	3.0	4.3	3.0	1.7	1.8	2.7	4.1

SD = Standard deviation.
 N = Number of observations.

Source: [Zender et al. \(2001\)](#).

Table 5-3. Principal sources of drinking water at home for a population of women in Colorado (%)

Source of water	Pregnant N = 71	Nonpregnant N = 43
Tapwater	74.6	72.1
Bottled water	14.1	11.6
Filtered water	11.3	16.3

N = Number of observations.

Source: [Zender et al. \(2001\)](#).

[Kahn and Stralka \(2008\)](#) used data from USDA's 1994–1996 and 1998 Continuing Survey of Food Intakes by Individuals to estimate drinking water intake. The data were collected from a total of 70 pregnant women, 41 lactating women, and 2,221 nonpregnant and nonlactating women aged 15–44 years ([USDA, 1998](#)). Consumer-only and per capita water ingestion rates were estimated for both community water only and for all sources of water. The percentage of consumers was approximately 93% and 83% for pregnant and lactating women, respectively. Community water was defined as tapwater from a community or municipal water supply, and all sources as tapwater from the community water supply plus bottled water, water obtained from wells, springs, and cisterns, and other sources that could not be identified. Estimates of drinking water intake included direct water ingestion (i.e., as a beverage) and indirect water ingestion (i.e., water added to foods and beverages during final preparation), but commercial water added by a manufacturer (i.e., water contained in soda or beer) and intrinsic water in foods and liquids (i.e., milk and natural undiluted juice) were not included.

[Kahn and Stralka \(2008\)](#) estimated mean and upper percentile intake rates (mL/day and indexed by body weight in mL/kg-day) for the four groups of women: (1) pregnant, (2) lactating, (3) nonpregnant and nonlactating, aged 15–44 years, and (4) all women, aged 15–44 years (see Table 5-4). The mean total water intake was lowest among nonpregnant and nonlactating women and highest among lactating women. For community water source only, the mean was lowest among pregnant women and highest among lactating women. Per capita mean and 95th percentile values for drinking water ingestion among pregnant women were 819 mL/day and 2,503 mL/day, respectively. Per capita mean and 95th percentile values for lactating women were 1,379 mL/day and 3,434 mL/day, respectively.

Table 5-4. Water ingestion rates of pregnant, lactating, and nonpregnant nonlactating U.S. women aged 15–44 years, community water^a and (total water from all sources), based on 1994–1996 and 1998 CSFII data

Group	N	Mean		95 th percentile	
		mL/day	mL/kg-day	mL/day	mL/kg-day
Per capita^b					
Pregnant women	70 (70)	819 ^c (1,318 ^c)	13 ^c (21 ^c)	2,503 ^c (2,674 ^c)	43 ^c (44 ^c)
Lactating women	41 (41)	1,379 ^c (1,806 ^c)	21 ^c (21 ^c)	3,434 ^c (3,767 ^c)	55 ^c (55 ^c)
Nonpregnant and nonlactating women, aged 15 to 44 years	2,221 (2,221)	916 (1,243)	14 (19)	2,575 (2,937)	38 (46)
All women, aged 15 to 44 years	2,332 (2,332)	922 (1,256)	14 (19)	2,605 (2,949)	39 (46)
Consumer-only^d					
Pregnant women	65 (70)	872 ^c (1,318 ^c)	14 ^c (21 ^c)	2,589 ^c (2,674 ^c)	43 ^c (44 ^c)
Lactating women	34 (41)	1,665 ^c (1,806 ^c)	26 ^c (28 ^c)	3,588 ^c (3,767 ^c)	55 ^c (57 ^c)
Nonpregnant and nonlactating women, aged 15 to 44 years	2,077 (2,203)	976 (1,252)	15 (19)	2,614 (2,941)	38 (46)
All women, aged 15 to 44 years	2,176 (2,314)	985 (1,265)	15 (20)	2,732 (2,953)	39 (46)

^aIngestion rates for combined direct and indirect water from community water supply.

^bPer capita intake rates are generated by averaging consumer-only intakes over the entire population (including those individuals that reported no intake).

^cEstimates are less statistically reliable based on guidance published in the *Joint Policy on Variance Estimation and Statistical Reporting Standards on NHANES III and CSFII Reports: NHIS/NCHS Analytical Working Group Recommendations* (NCHS, 1993).

^dConsumer-only intake represents the quantity of water consumed only by individuals that reported consuming water during the survey period.

N = Sample size.

Source: [Kahn and Stralka \(2008\)](#).

In a study of 1,990 pregnant women from three southern cities in the United States, mean cold tapwater intake increased from prepregnancy (1.5 L/day) through early pregnancy (1.7 L/day) to mid-pregnancy (1.8 L/day). Mean hot tapwater intake decreased slightly from prepregnancy (0.18 L/day) to early and mid-pregnancy (0.16 L/day). Bottled water consumption was essentially the same during early and mid-pregnancy (0.57 and 0.59 L/day, respectively). The greatest changes in water consumption were reported for cold tapwater for which 80% of the women reported either increases or decreases in consumption. Thirty-three percent reported changes (increases or decreases) equal to or greater than 1.0 L/day ([Forssen et al., 2009](#)).

A few studies were identified that investigated differences in water intake by pregnant women in relation to age, employment, income, and ethnicity. [Smith et al. \(2009\)](#) estimated the amount of water ingested by 39 pregnant women in northern England. There were no differences in water intake with regard to age, employment status, or income level. However, the results suggested that pregnant women of South Asian origin ($N = 16$; including Pakistani and Indian women) may consume more tapwater than other ethnic groups. Mean tapwater consumption for pregnant women in the study ($N = 39$) was 1.8 L/day, and represented 84% of all fluid intake.

In a study of 34 pregnant women in North Carolina, daily intake of cold tapwater at home was 1.7 times higher for women employed part-time or less than for those employed full-time ([Shimokura et al., 1998](#)). Considerably higher levels of cold tapwater consumption were reported at home versus work in other studies ([Forssén et al., 2007](#); [Zender et al., 2001](#)). In a study of 2,297 pregnant women in three geographical locations of the southern United States, [Forssén et al., \(2007\)](#) reported similar daily intake levels of cold tapwater (1.7 L/day) and bottled water (0.5–0.6 L/day). Among this population, non-Hispanic white women drank 0.4 L/day more cold tapwater than Hispanic women and 0.3 L/day more than non-Hispanic black women. Increases in cold tapwater intake during pregnancy were also associated with non-Hispanic women older than 35 years of age and income level less than \$40,000 per year ([Forssen et al., 2009](#)).

The treatment (i.e., filtered or unfiltered) and sources of the water (i.e., bottled water) consumed by pregnant women have also been studied. Daily intake of cold filtered tap water by pregnant women increased for those older in age, those who had higher income and education, and those who were unemployed ([Forssén et al., 2007](#)). A higher proportion of the water consumed by Hispanic women was bottled. Black and non-Hispanic women drank more of the water as unfiltered tapwater. Mean bottled water consumption among pregnant women has been reported as 0.6 L/day ([Forssén et al., 2007](#)) and 0.94 L/day ([Kaur et al., 2004](#)).

5.2. DIETARY INTAKE

EPA's Office of Pesticide Programs estimated food intake rates for pregnant and nonpregnant women of child-bearing age (13 to 49 years) using data from the NHANES for the years 2003 to 2008 ([Sarkar and Nguyen, 2013](#)) (see Appendix). It should be noted that no editing was performed to the appendix. A select number of tables and figures from the appendix were extracted and edited for format and in response to peer review comments before inclusion in the main report.

NHANES collects data on dietary recall of foods eaten over the previous 24-hour period on two nonconsecutive days. Two-day data were available for 612 pregnant women and 4,321 women of child-bearing age that were not pregnant during the 2003 to 2008 survey years. EPA's Food Commodity Intake Database was used to convert the NHANES "as eaten" food consumption data into consumption of individual food commodities, and the data were weighted according to sampling weights provided for the years 2003 to 2008 ([Sarkar and Nguyen, 2013](#)) (see Appendix). Food commodities and food groupings were selected consistent with previous consumption analyses presented in EPA's *Exposure Factors Handbook* ([U.S. EPA, 2011](#)). Two-day average intake rates were calculated for each survey respondent for the major food groups (total fruits, total vegetables, total meats, total dairy, total fish, and total grains) and for a variety of individual food items/groups, and summary statistics were calculated for the pregnant and nonpregnant women on both a consumer-only and on a per capita basis. Consumer-only intake is defined as the quantity of foods consumed by the women during the survey period. Per capita intake represents an average across the entire population of women surveyed, regardless of whether those individuals reported consumption or not.

Table 5-5 provides summary statistics for per capita intake and Table 5-6 provides data for consumers only. Mean, standard error, 95th percentile per capita, and consumer-only intake rates for a variety of individual foods and food groups are provided in Table 5-7. Figure 5-1 depicts the ratios of intake for pregnant women to those of nonpregnant women, based on mean consumer-only intake rates for these two groups of women in rank order from low to high. For graphical convenience, these have been split arbitrarily into two groups: those with lower pregnant to nonpregnant consumption ratios (List 1) and those with higher ratios (List 2). As shown in Figure 5-1, ratio >1 indicates higher consumption for pregnant females. Statistical comparisons of mean consumption for pregnant and nonpregnant women were also evaluated using an alpha level of 0.05. A two sample (unpaired) t-test adapted for complex survey procedure accounting for sampling weight, stratification, and multistage sampling was used. As noted in Tables 5-5, 5-6, and 5-7, and Figure 5-1, intake rates for pregnant women were found to be significantly different from those of nonpregnant women for some of the major food groups

(i.e., total fruits, total vegetables, total dairy, and total grain) and several individual food categories.¹

¹Note that multiple tests have been performed in EPA's analysis of these data, and any putative statistical differences have not been corrected to account for these. Many of these differences may not be statistically significant if such adjustments had been made. In addition, differences in mean consumption between pregnant and not pregnant females of child-bearing age do not necessarily imply differences in overall exposure estimates or potential risk. Statistical tests for the difference in consumption between pregnant and nonpregnant females were performed for the mean, and not for the percentiles. In many cases at the upper (and lower) percentiles of the consumers-only distribution (and particularly for less commonly consumed food commodities or commodity groupings), there are not adequate numbers of individuals to produce reliable estimates of consumption.

Table 5-5. Per capita intake of major food groups: U.S. pregnant and nonpregnant women of child-bearing age (13 to 49 years), based on NHANES 2003–2008 (g/kg-day)

Food group	N	% Cons.	Mean	SE	Min	Percentiles									Max
						1 st	5 th	10 th	25 th	50 th	75 th	90 th	95 th	99 th	
Total fruits															
Pregnant	612	92.5	1.66 ^a	0.13	— ^b	— ^b	—	—	0.07	0.97	2.65	4.34	5.02	8.11 ^b	11.01 ^b
Nonpregnant	4,321	83.6	0.98	0.04	— ^b	—	—	—	0.00	0.39	1.41	2.83	3.75	6.19	16.67 ^b
Total vegetables															
Pregnant	612	100	2.74 ^a	0.14	0.01 ^b	0.23 ^b	0.42	0.95	1.47	2.31	3.52	5.00	6.26	9.57 ^b	18.30 ^b
Nonpregnant	4,321	99.8	2.43	0.06	— ^b	0.05	0.36	0.62	1.16	2.03	3.25	4.69	5.93	8.79	17.06 ^b
Total meats															
Pregnant	612	99.3	1.59	0.05	— ^b	0.00 ^b	0.29	0.5	0.89	1.47	2.15	2.94	3.34	4.54 ^b	5.91 ^b
Nonpregnant	4,321	98.0	1.53	0.03	— ^b	—	0.14	0.36	0.79	1.30	2.01	2.98	3.54	4.97	12.23 ^b
Total dairy															
Pregnant	612	100	5.04 ^a	0.28	0.00 ^b	0.09 ^b	0.43	0.82	2.23	3.88	6.91	10.05	12.52	22.54 ^b	52.68 ^b
Nonpregnant	4,321	99.6	3.53	0.13	— ^b	0.02	0.21	0.43	1.02	2.41	4.90	7.94	10.48	17.16	52.07 ^b
Total fish															
Pregnant	612	26.5	0.19	0.03	— ^b	— ^b	—	—	—	—	0.00	0.55	1.28	2.40 ^b	5.32 ^b
Nonpregnant	4,321	28.7	0.19	0.01	— ^b	—	—	—	—	—	0.03	0.68	1.10	2.24	8.64 ^b
Total grains															
Pregnant	612	100	2.12 ^a	0.07	0.21 ^b	0.57 ^b	0.79	1.03	1.38	1.94	2.65	3.44	3.94	4.88 ^b	7.76 ^b
Nonpregnant	4,321	99.8	1.90	0.04	— ^b	0.20	0.53	0.75	1.12	1.68	2.44	3.36	3.94	5.66	9.79 ^b

Table 5-5. Per capita intake of major food groups: U.S. pregnant and nonpregnant women of child-bearing age (13 to 49 years), based on NHANES 2003–2008 (g/kg-day) (continued)

^aMean of pregnant female is statistically significantly different from the nonpregnant female; alpha = 0.05 level. Significant differences were NOT evaluated for percentiles values.

^bEstimates are less statistically reliable based on $np < 8$ “Design Effect” guidance published in the *Joint Policy on Variance Estimation and Statistical Reporting Standards on NHANES III and CSFII* ([NCHS, 1993](#)); where n refers to the sample size and p is the percentile expressed as a fraction.

— = Either no reported per capita consumption at this percentile, or per capita consumption is < 0.0001 g/kg body weight.

N = Sample size.

% Cons. = Number of individuals who consumed the food item during the survey period divided by the total number of individuals surveyed $\times 100$.

SE = Standard error.

Source: [Sarkar and Nguyen \(2013\)](#) (see Appendix).

Table 5-6. Consumer-only intake of major food groups: U.S. pregnant and nonpregnant women of child-bearing age (13 to 49 years), based on NHANES 2003–2008 (g/kg-day)

Food group	N	Mean	SE	Min	Percentiles									Max
					1 st	5 th	10 th	25 th	50 th	75 th	90 th	95 th	99 th	
Total fruits														
Pregnant	558	1.79 ^a	0.14	0.00 ^b	0.00 ^b	0.00 ^b	0.01	0.15	1.10	2.82	4.64	5.37 ^b	8.11 ^b	11.01 ^b
Nonpregnant	3,640	1.18	0.05	0.00 ^b	0.00	0.00	0.00	0.09	0.68	1.67	3.16	4.06	6.45	16.67 ^b
Total vegetables														
Pregnant	612	2.74 ^a	0.14	0.01 ^b	0.23 ^b	0.42	0.95	1.47	2.31	3.52	5.00	6.26	9.57 ^b	18.30 ^b
Nonpregnant	4,318	2.43	0.06	0.00 ^b	0.05	0.37	0.63	1.16	2.03	3.25	4.69	5.93	8.79	17.06 ^b
Total meats														
Pregnant	607	1.60	0.05	0.00 ^b	0.03 ^b	0.32	0.51	0.93	1.48	2.15	2.94	3.34	4.54 ^b	5.91 ^b
Nonpregnant	4,259	1.56	0.03	0.00 ^b	0.01	0.25	0.43	0.82	1.32	2.03	3.01	3.56	4.97	12.23 ^b
Total dairy														
Pregnant	612	5.04 ^a	0.28	0.00 ^b	0.09 ^b	0.43	0.82	2.23	3.88	6.91	10.05	12.52	22.52 ^b	52.68 ^b
Nonpregnant	4,310	3.54	0.12	0.00 ^b	0.03	0.22	0.44	1.03	2.41	4.91	7.97	10.51	17.16	52.07 ^b
Total fish														
Pregnant	153	0.71	0.12	0.00 ^b	0.00 ^b	0.00 ^b	0.02 ^b	0.18	0.41	1.00	1.59 ^b	2.40 ^b	3.79 ^b	5.32 ^b
Nonpregnant	1,204	0.65	0.04	0.00 ^b	0.00 ^b	0.00	0.00	0.18	0.43	0.86	1.44	1.93	3.45 ^b	8.64 ^b
Total grains														
Pregnant	612	2.12 ^a	0.07	0.21 ^b	0.57 ^b	0.79	1.03	1.38	1.94	2.65	3.44	3.94	4.88 ^b	7.76 ^b
Nonpregnant	4,318	1.90	0.04	0.00 ^b	0.20	0.53	0.75	1.12	1.68	2.44	3.36	3.94	5.66	9.79 ^b

^aMean of pregnant female is statistically significantly different from the nonpregnant female; alpha = 0.05 level. Significant differences were NOT evaluated for percentiles values.

^bEstimates are less statistically reliable based on $np < 8$ “Design Effect” guidance published in the *Joint Policy on Variance Estimation and Statistical Reporting Standards on NHANES III and CSFII* (NCHS, 1993); where n refers to sample size and p is the percentile expressed as a fraction.

N = Sample size.

SE = Standard error.

Source: [Sarkar and Nguyen \(2013\)](#) (see Appendix).

Table 5-7. Per capita and consumer-only intake of individual foods: U.S. pregnant and nonpregnant women of child-bearing age (13 to 49 years), based on NHANES 2003–2008 (g/kg-day)

Food	Per capita					Consumer-only			
	% Cons.	N	Mean	SE	95 th	N	Mean	SE	95 th
Fruits and vegetables									
Apples									
Pregnant	38.3	612	0.33	0.05	2.02	245	0.86	0.12	2.75 ^b
Nonpregnant	28.1	4,321	0.24	0.02	1.61	1,181	0.87	0.05	2.70
Bananas									
Pregnant	63.4	612	0.34 ^a	0.04	1.78	383	0.53 ^a	0.06	1.97 ^b
Nonpregnant	49.4	4,321	0.20	0.01	1.27	2,259	0.41	0.02	1.75
Beans									
Pregnant	46.6	612	0.20	0.02	0.80	319	0.44	0.04	1.16 ^b
Nonpregnant	45.7	4,321	0.18	0.01	0.85	1,964	0.39	0.02	1.21
Berries and small fruits									
Pregnant	75.5	612	0.25	0.03	1.25	429	0.33	0.04	1.29 ^b
Nonpregnant	65.7	4,321	0.22	0.01	1.13	2,821	0.33	0.02	1.37
Broccoli									
Pregnant	14.6	612	0.08	0.02	0.74	95	0.58	0.08	1.45 ^b
Nonpregnant	17.1	4,321	0.09	0.01	0.67	610	0.54	0.04	1.55
Bulb vegetables									
Pregnant	97.5	612	0.19	0.02	0.62	596	0.20	0.02	0.62
Nonpregnant	97.3	4,321	0.16	0.01	0.53	4,206	0.17	0.01	0.54
Cabbage									
Pregnant	8.0	612	0.02 ^a	0.00	0.06	79	0.24 ^a	0.04	0.80 ^b
Nonpregnant	12.0	4,321	0.05	0.01	0.26	497	0.39	0.04	1.29
Carrot									
Pregnant	46.1	612	0.10	0.01	0.46	292	0.21	0.02	0.65 ^b
Nonpregnant	45.7	4,321	0.11	0.01	0.59	1,850	0.25	0.01	0.91

Table 5-7. Per capita and consumer-only intake of individual foods: U.S. pregnant and nonpregnant women of child-bearing age (13 to 49 years), based on NHANES 2003–2008 (g/kg-day) (continued)

Food	Per capita					Consumer-only			
	% Cons.	N	Mean	SE	95 th	N	Mean	SE	95 th
Fruits and vegetables									
Citrus									
Pregnant	23.7	612	0.29 ^a	0.06	1.80	156	1.21 ^a	0.20	3.23 ^b
Nonpregnant	20.5	4,321	0.10	0.01	0.80	877	0.50	0.05	2.05
Corn									
Pregnant	99.6	612	0.42 ^a	0.03	1.42	604	0.42 ^a	0.03	1.42
Nonpregnant	95.8	4,321	0.31	0.01	1.13	4,157	0.32	0.01	1.15
Cucumbers									
Pregnant	46.4	612	0.14	0.05	0.65	249	0.29	0.10	1.16 ^b
Nonpregnant	43.0	4,321	0.09	0.01	0.50	1,679	0.22	0.01	0.83
Cucurbits									
Pregnant	55.6	612	0.48 ^a	0.09	2.93	309	0.87 ^a	0.14	3.84 ^b
Nonpregnant	50.6	4,321	0.27	0.04	1.43	1,990	0.54	0.07	2.27
Fruiting vegetables									
Pregnant	99.1	612	0.77	0.04	2.08	601	0.78	0.04	2.08
Nonpregnant	96.0	4,321	0.72	0.02	2.33	4,134	0.75	0.02	2.42
Leafy vegetables									
Pregnant	93.4	612	0.46 ^a	0.03	1.58	576	0.49 ^a	0.03	1.64
Nonpregnant	92.8	4,321	0.57	0.02	1.97	3,978	0.61	0.03	2.01
Legume vegetables									
Pregnant	95.1	612	0.38	0.07	1.39	597	0.40	0.08	1.47
Nonpregnant	95.2	4,321	0.33	0.02	1.37	4,100	0.35	0.02	1.42
Lettuce									
Pregnant	64.2	612	0.24	0.02	0.89	381	0.37 ^a	0.03	1.14 ^b
Nonpregnant	60.0	4,321	0.27	0.01	1.15	2,492	0.46	0.02	1.47

Table 5-7. Per capita and consumer-only intake of individual foods: U.S. pregnant and nonpregnant women of child-bearing age (13 to 49 years), based on NHANES 2003–2008 (g/kg-day) (continued)

Food	Per capita					Consumer-only			
	% Cons.	N	Mean	SE	95 th	N	Mean	SE	95 th
Fruits and vegetables									
Onions									
Pregnant	97.4	612	0.19	0.02	0.59	595	0.19	0.02	0.60
Nonpregnant	96.5	4,321	0.16	0.01	0.52	4,175	0.16	0.01	0.52
Peaches									
Pregnant	54.6	612	0.09 ^a	0.02	0.76	317	0.17	0.04	1.18 ^b
Nonpregnant	44.4	4,321	0.04	0.01	0.14	2,034	0.10	0.01	0.59
Pears									
Pregnant	8.9	612	0.05	0.01	0.22	56	0.52	0.11	1.72 ^b
Nonpregnant	7.0	4,321	0.04	0.01	0.11	318	0.60	0.07	2.35 ^b
Peas									
Pregnant	13.4	612	0.04	0.01	0.37	101	0.29	0.03	0.80 ^b
Nonpregnant	18.4	4,321	0.05	0.01	0.34	743	0.28	0.02	0.87
Pome fruit									
Pregnant	40.9	612	0.37	0.06	2.02	265	0.91	0.11	2.75 ^b
Nonpregnant	31.1	4,321	0.29	0.02	1.86	1,354	0.92	0.04	2.84
Root and tuber vegetables									
Pregnant	100	612	1.03 ^a	0.08	2.67	612	1.03 ^a	0.80	2.67
Nonpregnant	99.8	4,321	0.84	0.02	2.36	4,315	0.84	0.02	2.36
Stalk and stem vegetables									
Pregnant	23.5	612	0.04	0.01	0.23	131	0.15 ^a	0.03	0.52 ^b
Nonpregnant	21.3	4,321	0.05	0.00	0.26	764	0.21	0.02	0.74
Stone fruits									
Pregnant	60.4	612	0.17 ^a	0.04	1.22	340	0.28	0.06	1.33 ^b
Nonpregnant	47.2	4,321	0.08	0.01	0.53	2,137	0.17	0.02	0.97

Table 5-7. Per capita and consumer-only intake of individual foods: U.S. pregnant and nonpregnant women of child-bearing age (13 to 49 years), based on NHANES 2003–2008 (g/kg-day) (continued)

Food	Per capita					Consumer-only			
	% Cons.	N	Mean	SE	95 th	N	Mean	SE	95 th
Fruits and vegetables									
Strawberries									
Pregnant	49.2	612	0.09	0.02	0.63	261	0.18	0.04	0.81 ^b
Nonpregnant	38.2	4,321	0.08	0.01	0.60	1,532	0.21	0.02	1.09
Tomatoes									
Pregnant	96.0	612	0.70	0.04	1.88	578	0.73	0.04	1.93
Nonpregnant	87.5	4,321	0.63	0.02	2.02	3,830	0.72	0.02	2.17
Tropical fruits									
Pregnant	72.0	612	0.52 ^a	0.07	2.35	447	0.73 ^a	0.08	3.26 ^b
Nonpregnant	59.4	4,321	0.27	0.02	1.53	2,700	0.45	0.02	1.85
White potatoes									
Pregnant	93.0	612	0.65 ^a	0.07	2.04	563	0.70 ^a	0.07	2.04 ^b
Nonpregnant	90.2	4,321	0.48	0.02	1.92	3,868	0.53	0.02	1.98
Meat, fish, and grains									
Beef									
Pregnant	89.9	612	0.64	0.05	1.78	540	0.71	0.05	2.09 ^b
Nonpregnant	85.7	4,321	0.58	0.02	1.98	3,744	0.67	0.02	2.13
Poultry									
Pregnant	80.9	612	0.67	0.05	1.99	488	0.82	0.06	2.05 ^b
Nonpregnant	76.8	4,321	0.67	0.02	2.17	3,414	0.87	0.03	2.40
Pork									
Pregnant	85.5	612	0.29	0.02	1.01	529	0.33	0.03	1.12 ^b
Nonpregnant	77.9	4,321	0.28	0.01	1.07	3,397	0.36	0.01	1.19
Finfish									
Pregnant	20.9	612	0.12	0.02	0.94	108	0.59	0.10	1.80 ^b
Nonpregnant	22.9	4,321	0.13	0.01	0.90	882	0.59	0.03	1.75

Table 5-7. Per capita and consumer-only intake of individual foods: U.S. pregnant and nonpregnant women of child-bearing age (13 to 49 years), based on NHANES 2003–2008 (g/kg-day) (continued)

Food	Per capita					Consumer-only			
	% Cons.	N	Mean	SE	95 th	N	Mean	SE	95 th
Meat, fish, and grains									
Shellfish									
Pregnant	12.9	612	0.07	0.02	0.48	75	0.51	0.09	1.79 ^b
Nonpregnant	10.6	4,321	0.05	0.01	0.34	492	0.48	0.05	1.79
Rice									
Pregnant	90.1	612	0.22	0.04	0.98	555	0.24	0.04	0.98 ^b
Nonpregnant	86.7	4,321	0.20	0.01	0.86	3,690	0.23	0.01	0.95
Total cereal									
Pregnant	100	612	3.02 ^a	0.08	5.71	612	3.02 ^a	0.08	5.71
Nonpregnant	100	4,321	2.80	0.04	5.77	4,320	2.80	0.04	5.77

^aMean of pregnant female is statistically significantly different from the nonpregnant female; alpha = 0.05 level. Significant differences were NOT evaluated for percentiles values.

^bEstimates are less statistically reliable based on $np < 8$ “Design Effect” guidance published in the *Joint Policy on Variance Estimation and Statistical Reporting Standards on NHANES III and CSFII* (NCHS, 1993); where n refers to sample size and p is the percentile expressed as a fraction.

N = Sample size.

% Cons. = Number of individuals who consumed the food item during the survey period divided by the total number of individuals surveyed $\times 100$.

SE = Standard error.

Source: [Sarkar and Nguyen \(2013\)](#) (see Appendix).

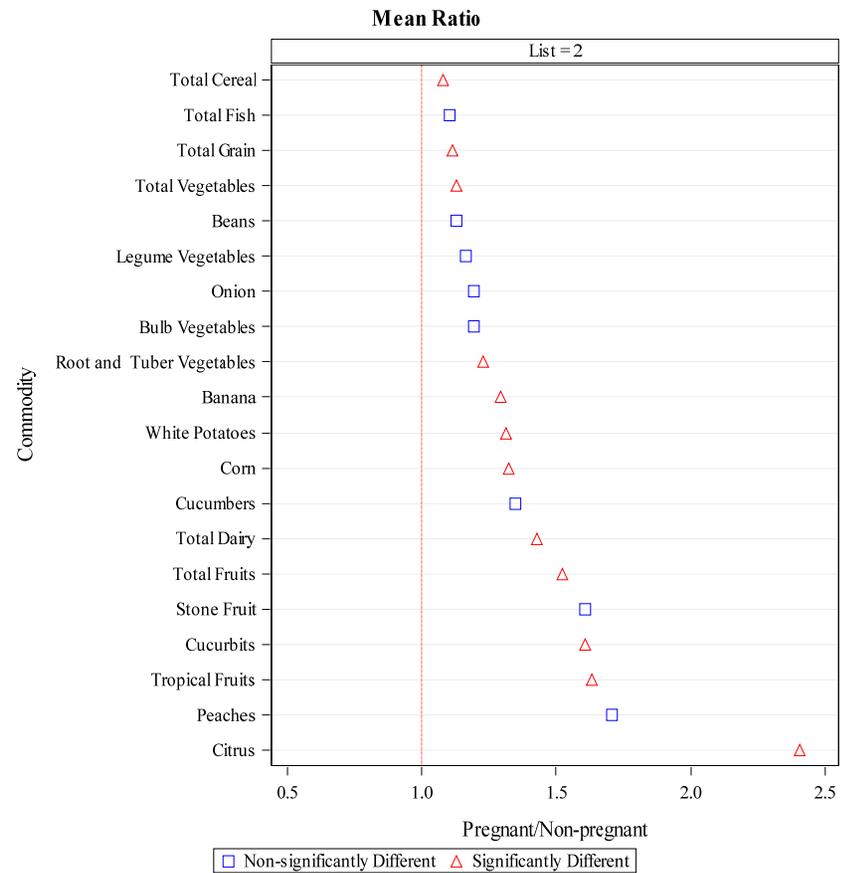
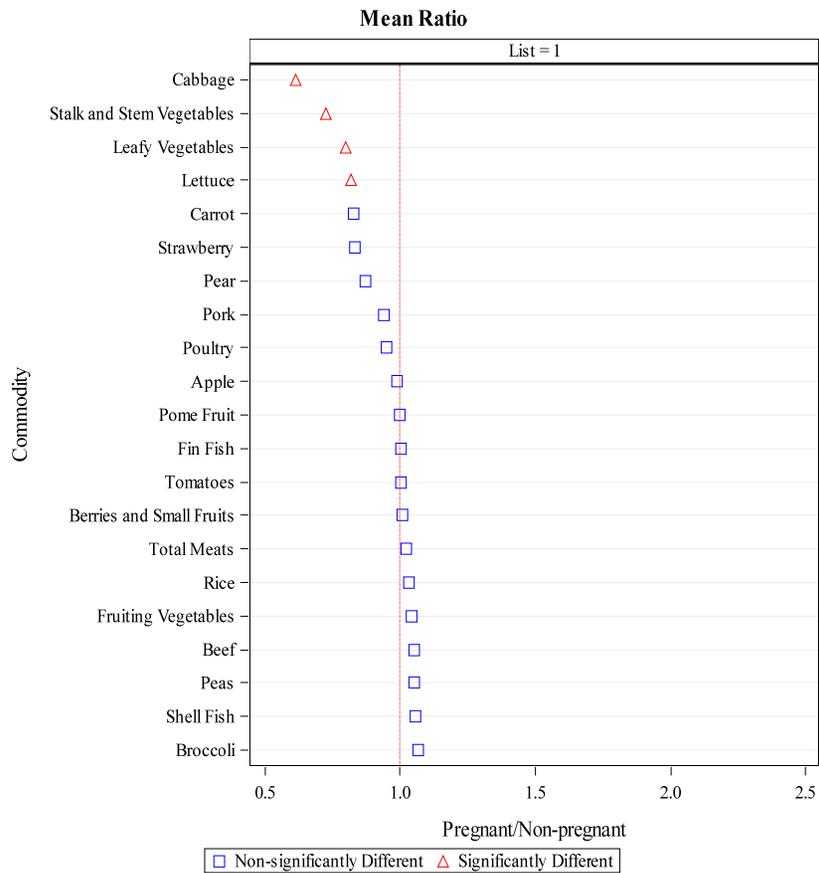


Figure 5-1. Ratios of mean consumer-only intake for pregnant women to that of nonpregnant women in the United States, based on NHANES 2003–2008. Note: List 1 contains the commodities that have lower mean ratio; List 2 contains the commodities that have slightly higher mean ratio. Food commodities were presented in two graphs for clarity.

Source: [Sarkar and Nguyen \(2013\)](#) (see Appendix).

As mentioned in Section 4, U.S. research related to dietary intake during pregnancy has focused primarily on nutrient intake (e.g., calcium, folic acid), changes in eating patterns, or associations between contaminant intake (e.g., mercury in fish) and pregnancy outcomes ([Bloomingdale et al., 2010](#); [Mirel et al., 2009](#); [Xue et al., 2007](#); [Buck et al., 1997](#)). Thus, limited data are available on the difference in food intake rates between pregnant and nonpregnant women. In addition, limited research was found with regard to the role that race and ethnicity, as well as other demographic variables, play in food choices and amounts of foods consumed. Data on food consumption by lactating women is extremely limited. [Dubowitz et al. \(2007\)](#) conducted a study in a multiethnic sample of 662 low-income, postpartum women in the Boston, MA metropolitan area and found that foreign-born women ate 2.5 more servings of fruits and vegetables than women born in the United States. [Leslie et al. \(2012\)](#) observed differences in fruits and vegetables servings between low and high socioeconomic position among breastfeeding mothers in Melbourne, Australia.

[Crozier et al. \(2009\)](#) collected dietary data using a food frequency questionnaire for 2,270 women in early pregnancy (about 12 weeks); 2,649 women in late pregnancy (about 35 weeks); and 12,572 nonpregnant women in Southampton, U.K. Data on the consumption of 48 foods or food groups were collected. During early pregnancy, intake of 21 foods or food groups increased, including: white bread, breakfast cereals, cakes and biscuits (cookies), processed meat, crisps (potato chips), fruits and fruit juices, dried fruit, sweet spreads, confectionery, and hot chocolate drinks. During late pregnancy, intake of puddings, cream, milk, cheese, full-fat spread, cooking fats and salad oils, red meat, and soft drinks increased. Intake of 10 foods or food groups, including: rice and pasta, liver and kidney, salad vegetables, other vegetables, vegetable dishes, nuts, diet cola, tea, and coffee decreased during pregnancy ([Crozier et al., 2009](#)). These results indicate that there may be differences in food intake rates between pregnant and nonpregnant women and that they may change across each trimester. However, the consumption patterns or food choices observed in this study may not be representative of pregnant women in the United States.

Adequate protein consumption, including consumption of fish, is essential during periods of pregnancy and lactation. Fish consumption during pregnancy and lactation is particularly of interest to health officials because fish may contain contaminants that are harmful to the developing fetus, and the benefits of fish as a healthy choice and a good source for omega-3 fatty acids and other nutrients essential for fetal neurodevelopment must be weighed against the potential risks associated with the contaminants. Limited data are available on fish consumption rates for pregnant and lactating women. Many of the studies found relate to knowledge of fish advisories by pregnant and lactating women and effectiveness of intervention techniques ([Teisl et al., 2011](#); [Karouna-Renier et al., 2008](#); [Scherer et al., 2008](#)).

A study at a WIC clinic was performed to characterize commercial and sport fish consumption patterns and advisory awareness among 500 ethnically diverse women living in California's Sacramento-San Joaquin Delta ([Silver et al., 2007](#)). The study included 66 pregnant women (13%) and 105 breastfeeding mothers (21%). The area was under a state health advisory limiting consumption of certain Delta fish, to be followed in conjunction with a federal advisory for commercial and sport fish. Among all women surveyed, 95% consumed commercial fish and 32% consumed sport fish. Commercial fish are those caught for profit and may be a more widely distributed food source than sport fish. Sport fish are those caught as part of a sporting or recreational activity and not for the purpose of providing a primary source of food ([U.S. EPA, 2011](#)). Pregnant women ate less fish overall (geometric mean = 16.8 g/day) than nonpregnant women (geometric mean = 30.0 g/day). The geometric mean fish consumption rate among breastfeeding mothers was 31.1 g/day. Sport fish consumption among pregnant and lactating women was 12.8 and 10.2 g/day, respectively (geometric mean). These data may not be generalizable to the U.S. population because the study was limited to women in WIC clinics in California.

[Xue et al. \(2007\)](#) estimated mean total fish consumption among 1,024 pregnant mothers in five Michigan communities. During the first 6 months of pregnancy, fish intake was estimated to be 19.6 meals/6 months (3.3 meals/month). Similar results were obtained by [Gliori et al. \(2006\)](#) who surveyed 726 postpartum women in Wisconsin in 2003 to obtain information about the number of types of fish that women consumed while pregnant and their knowledge about outreach materials regarding fish consumption during their pregnancy. Eighty-five percent of the women had consumed fish during the year prior to giving birth. The average consumption among pregnant women was 3 meals/month. The types of fish most frequently consumed were tuna and commercially purchased frozen fish, followed by shellfish and sport-caught fish ([Gliori et al., 2006](#)). In another study, [Oken et al. \(2003\)](#) used food frequency questionnaires completed by 2,235 pregnant women in eastern Massachusetts, to estimate the number of fish meals consumed in a month in order to assess changes in consumption habits after implementation of the 2001 National Mercury Advisory. A decline in fish consumption among pregnant women was observed following the advisory. The mean number of fish meals consumed per month was 7.7 before the mercury advisory and 6.4 after the mercury advisory ([Oken et al., 2003](#)). A study of 22 pregnant women in the Boston area assessed women's knowledge of the health effects of fish consumption during pregnancy and changes in consumption during pregnancy ([Bloomingdale et al., 2010](#)). One-half of the women (11 of 22) reported eliminating consumption of sushi during pregnancy ([Bloomingdale et al., 2010](#)), and others eliminated or reduced certain fish species from their diets during pregnancy ([Bloomingdale et al., 2010](#)). The

data from these studies are not based on nationally representative samples and may not be generalizable to the U.S. population.

The Food and Drug Administration (FDA) and the CDC conducted a study of pregnant women and new mothers to assess awareness of mercury contamination in food and examined fish consumption levels across groups ([Lando et al., 2012](#)). The study was conducted from May through December of 2005. It included 4,902 “single birth” (not expecting twins or other multiples) pregnant women age 18 or older who were recruited nationally in the third trimester. The women were also followed postpartum if their gestational time was greater than 35 weeks, both mother and infant were healthy at birth, and the infant’s birth weight was greater than 2.3 kg (5 lbs). The study included a control group of women who were between the ages of 18 and 40 years, were not pregnant, had not had a baby within the past year, and had not already participated in the study. A food frequency questionnaire developed by the National Cancer Institute was used to collect intake data using a recall period of 1 month. A total of 1,286 pregnant, 522 postpartum, and 1,349 control group women completed the dietary intake component of the study. Table 5-8 presents the percentage of women in each group who ate fish and their daily fish intake levels. Mean fish consumption rates were 12.7, 14.6, and 17.0 g/day for pregnant, postpartum, and control women, respectively. Awareness about mercury in food was reported to be 73.3, 74.0, and 58.9% among pregnant, postpartum, and control women, respectively.

Table 5-8. Percentage of women who ate each type of fish during the prior month and their weekly intake, based on a FDA/CDC study

Type of fish	Percentage that ate each type of fish during the prior month			Daily intake (consumers only) (g) ^a								
				Pregnant N = 1,018			Postpartum N = 412			Control N = 1,121		
	Pregnant N = 1,286	Postpartum N = 522	Control N = 1,349	25 th	50 th	75 th	25 th	50 th	75 th	25 th	50 th	75 th
Total fish	79.2	78.9	83.1	4.8	7.4	16.4	5.4	10.2	17.0	5.4	12.2	19.3
All canned tuna	53.3	53.3	57.6	2.6	5.4	9.6	2.6	5.4	8.5	2.6	4.8	9.6
Albacore	33.4	33.9	41.1	1.7	3.7	6.2	1.7	2.8	5.7	1.7	3.1	7.4
Not albacore	30.3	32.4	32.1	2.0	4.0	8.2	2.0	4.3	8.2	1.7	3.7	6.5
Fresh tuna	2.4	5.6	8.5	0.9	1.7	4.3	0.9	1.4	3.1	0.9	1.8	3.7
Salmon	16.8	19.5	23.9	1.1	2.3	5.7	2.0	3.3	5.7	1.1	2.6	5.7
Shark, swordfish, tilefish, king mackerel	1.1	4.0	4.5	0.9	1.7	2.6	0.9	1.4	2.6	0.6	1.1	2.6
All shellfish	41.7	40.6	52.3	1.7	3.1	6.7	2.0	3.7	8.5	2.3	5.4	11.1
Shrimp	38.3	36.4	48.9	1.4	2.8	6.0	1.7	3.1	6.2	1.7	3.4	6.8
Not shrimp	18.4	19.4	30.1	1.1	2.3	5.4	1.1	2.6	4.8	1.4	3.1	6.5
All other fish (includes fish sticks)	46.6	46.0	51.9	2.0	4.0	6.2	2.0	4.4	10.1	2.0	4.0	7.1

^aDaily intake is based on the number of women who ate each type of fish, and therefore sample sizes vary by the type of fish. Weekly intake from [Lando et al. \(2012\)](#) was converted to daily intake by dividing by 7.

N = Sample size.

Source: [Lando et al. \(2012\)](#).

Limited information is available on fish intake among pregnant Native American women. The New York State Department of Health ([Fitzgerald et al., 1995](#)) conducted a study among breastfeeding Mohawk women residing near three industrial sites between 1986 and 1992. A total of 97 breastfeeding Mohawk women living on the Akwesasne Reservation and 154 breastfeeding white controls living in Warren and Schoharie Counties in NY were included in the study. Data were collected on fish intake during pregnancy, the year before the pregnancy, and more than 1 year before the pregnancy ([Fitzgerald et al., 1995](#)). Table 5-9 presents the mean number of local fish meals consumed by Mohawk and control women per year by time period for all respondents. Of the 97 Mohawk mothers and 154 control mothers, local fish were consumed by 82 Mohawk mothers (85%) and 72 control mothers (47%). Annual consumption of local fish dramatically declined over time for all Mohawk respondents, from 23.4 (over 1 year before pregnancy) to 9.2 (less than 1 year before pregnancy) to 3.9 (during pregnancy). Data on the mean number of fish meals consumed per year by time period and selected characteristics (age, education, cigarette smoking, and alcohol consumption) are also provided in Table 5-9. The trend of decreased fish consumption during pregnancy was more notable among Mohawk women 25 to 29 years old and those with some college education.

Table 5-9. Mean number of local fish meals consumed per year by time period and selected characteristics for all respondents^a (Mohawk, *N* = 97; Control, *N* = 154), based on a New York Health Department study

Variable	Time period					
	During pregnancy		≤1 year before pregnancy		>1 year before pregnancy	
	Mohawk	Control	Mohawk	Control	Mohawk	Control
All	3.9	7.3	9.2	10.7	23.4^b	10.9
Age (years)						
<20	7.7	0.8	13.5	13.9	27.4	10.4
20 to 24	1.3	5.9	5.7	14.5	20.4	15.9
25 to 29	3.9	9.9	15.5	6.2	25.1	5.4
30 to 34	12.0	7.6	9.5	2.9	12.0	5.6
>34	1.8	11.2	1.8	26.2	52.3	22.1 ^c
Education (years)						
<12	6.3	7.9	14.8	12.4	24.7	8.6
12	7.3	5.4	8.1	8.4	15.3	11.4
13 to 15	1.7	10.1	8.0	15.4	29.2	13.3
>15	0.9	6.8	10.7	0.8	18.7	2.1
Cigarette smoking						
Yes	3.8	8.8	10.4	13.0	31.6	10.9
No	3.9	6.4	8.4	8.3	18.1	10.8
Alcohol consumption						
Yes	4.2	9.9	6.8	13.8	18.0	14.8
No	3.8	6.3 ^d	12.1	4.7 ^e	29.8	2.9 ^f

^aLocal fish were consumed by 82 Mohawk mothers (85%) and 72 Control mothers (47%).

^b*p* = <0.001 for Mohawks vs. Controls.

^c*F* (4, 149) = 2.66, *p* = 0.035 for Age Among Controls.

^d*F* (1, 152) = 3.77, *p* = 0.054 for Alcohol Among Controls.

^e*F* (1, 152) = 5.20, *p* = 0.024 for Alcohol Among Controls.

^f*F* (1, 152) = 6.42, *p* = 0.012 for Alcohol Among Controls.

Note: *F* (*r*1, *r*2) = *F* statistic with *r*1 and *r*2 degrees of freedom.

N = Sample size.

Source: [Fitzgerald et al. \(1995\)](#)

5.3. NONDIETARY INTAKE (PICA)

Several researchers have attempted to estimate the prevalence and nature of pica behavior among pregnant women. As noted previously, pica generally refers to behavior associated with the intentional ingestion of foreign (i.e., nonfood or nonnutritive) substances ([Bronstein and Dollar, 1974](#)). The types of materials ingested can include: dirt, clay, cigarette ashes, ice, freezer frost, flour, baking soda or powder, cornstarch, powdered milk, or other materials ([Cooksey, 1995](#)). Soil pica has been used to refer to the recurrent ingestion of large quantities of soil ([ATSDR, 2001](#)). Geophagy, a special type of pica, involves the deliberate ingestion of earth materials such as clay.

The practice of geophagy is still widespread in many parts of the world including Asia, Africa, South America, North America, and parts of Europe ([Al-Rmalli et al., 2010](#)). During pregnancy, some cultures encourage geophagy as means to obtain essential elements. The prevalence of geophagy among pregnant and lactating women in Africa has been reported to range from 29–73% ([Kutalek et al., 2010](#); [Kawai et al., 2009](#); [Nyaruhucha, 2009](#); [Luoba et al., 2005](#); [Prince et al., 1999](#)). Amounts reported among these African populations have ranged from 1–100 g/day ([Kutalek et al., 2010](#)).

In the United States, studies on the practice of pica and geophagy among pregnant and lactating women are limited, and the available data on this topic are more than three decades old. [Ferguson and Keaton \(1950\)](#) surveyed 361 pregnant, predominantly black, low-income women in Mississippi. Clay eating was reported to be 27% among the black women and 7% among white women, and ranged from 1 tablespoon/day–1 cup/day (~15–240g/day). Starch eating was reported to be 41% among the black women and 10% among white women, and the amounts consumed ranged from 2–3 small lumps–3 boxes (24 ounces) per day (~680 g/day). [Hook \(1978\)](#) interviewed 250 new mothers in New York about any cravings or aversions for other foods or nonfood items that may have developed at any time during their pregnancy. Three women reported eating ice and one woman reported eating chalk from a river clay bank, but no quantitative data were provided. [Bronstein and Dollar \(1974\)](#) studied 410 pregnant, low-income women from both urban ($N = 201$) and rural ($N = 209$) areas in Georgia. Women were interviewed during their initial prenatal visit about food frequency, social and dietary history, and the presence of pica. A total of 65 women (16%) indicated that they practiced pica (see Table 5-10). Laundry starch was the substance most commonly ingested, but specific information on the amount ingested was not provided. [Vermeer and Frate \(1979\)](#) conducted a similar survey among 142 pregnant black females in rural Mississippi. Forty of these women (28%) reported geophagy, and another 27 respondents (19%) reported ingesting other types of substances including laundry starch, dry powdered milk, and baking soda.

Table 5-10. Frequency of pica behavior among low-income women in Georgia (N = 410)

	Rural		Urban		Total	
	Number	%	Number	%	Number	%
Nonpica	172	82	172	86	345	84
Pica	37	18	28	14	65	16
Total	209	100	201	100	410	100
Chalk	16	42	8	27	24	35
Starch	14	37	17	57	31	46
Clay	3	8	3	10	6	9
Other	5	13	2	7	7	10

N = Sample size.

Source: [Bronstein and Dollar \(1974\)](#). Reprinted with permission.

[Cooksey \(1995\)](#) asked 300 postpartum women at a midwestern hospital about cravings to eat ice or other nonfood items during their pregnancies. The majority of women in the study were low-income blacks. Sixty-five percent reported that they had ingested one or more pica substances during their pregnancy. Freezer ice was one of the items most commonly consumed. The largest quantities of items ingested on a daily basis were reported to be three to four 8-pound bags of ice, two to three boxes of cornstarch, two cans of baking powder, one cereal bowl of dirt, five quarts of freezer frost, and one large can of powdered cleanser.

[Smulian et al. \(1995\)](#) conducted a survey among 125 pregnant women in Muscogee County, Georgia. Of the 18 women (14%) who reported practicing pica, four acknowledged eating “white dirt” or “red dirt” (0.5 to 1.0 pounds of dirt or clay per week-roughly ~30–70 g/day). Of the nine women who reported the amounts of substances ingested, six stated that their ingestion occurred daily and three stated that it occurred three times per week. The prevalence of the overall pica, was 17.8% among black women, 10.6% among white women, and 0% among the Asian and Hispanic women in the sample, with no significant differences between pica and nonpica groups with respect to age distribution and race.

[Simpson et al. \(2000\)](#) interviewed 225 Mexican-born women residing in low-income areas of Ensenada, Mexico (N = 75), and Santa Ana, Bakersfield, and East Los Angeles, California (N = 150) who were pregnant or had been pregnant within the previous year. Among the women interviewed in California, 46 (31%) reported pica behavior with ice being the item

eaten by the highest percentage of women. Excluding ice, 34 (23%) of the women interviewed in California reported pica behavior. The items ingested and the number of women reporting pica for each type of item is shown in Table 5-11. The reasons given for consuming nonfood items included the following: because of the taste, smell, or texture of the items, for medicinal purposes, or because of advice from someone, and one woman reported eating clay for religious reasons. Except for magnesium carbonate, which was reported to have been consumed in amounts ranging from a quarter of a block to five blocks per day, no specific quantities of pica substances ingested were provided. [Simpson et al. \(2000\)](#) compared the blocks to approximately the size of a 35-mm film box (i.e., about 2 × 1 × 1 inches).

Table 5-11. Items ingested by low-income Mexican-born women who practiced pica during pregnancy in California (N = 46)

Item ingested	Number (%) ingesting items
Dirt	11 (24)
Bean stones ^a	17 (37)
Magnesium carbonate	8 (17)
Ashes	5 (11)
Clay	4 (9)
Ice	18 (39)
Other ^b	17 (37)

^aLittle clods of dirt found among unwashed beans.

^bIncluding eggshells, starch, paper, lipstick, pieces of clay pot, and adobe.

N = Number of individuals reporting pica behavior.

Source: [Simpson et al. \(2000\)](#).

[Klitzman et al. \(2002\)](#) interviewed 33 pregnant women with elevated blood lead levels (i.e., >20 µg/dL) in New York City. Thirteen of the 33 women (39%) reported pica behavior during their pregnancy; 10 reported eating soil, dirt or clay, 2 reported pulverizing and eating pottery, and 1 reported eating soap. Except for one of the women who reported eating approximately one quart of dirt daily (~1,650 g/day assuming a soil bulk density of 1.5 g/cm³) ([U.S. EPA, 2002](#)) from her backyard over a 3-month period, no other quantity data were reported. Using NHANES data for the years 1971–1975 (NHANES I) and 1976–1980 (NHANES II), [Gavrelis et al. \(2011\)](#) conducted an analysis of the prevalence of nondietary intake among the U.S. population. The prevalence of ingestion of nonfood substances among

pregnant females 12 years and older was found to be more than twice (2.5%; 95% confidence interval [CI]: 0.0–5.6%) that of nonpregnant women (1.0%; 95% CI: 0.7–1.4%) in both NHANES I and NHANES II.

5.4. INHALATION RATES

Inhalation rates among pregnant women may differ from those of nonpregnant females as a result of changes in hormone levels, anatomy, activity levels, and body weight. [Brochu et al. \(2006\)](#) developed physiological daily inhalation rates (PDIRs) for pregnant and lactating females aged 11 to 55 years. Published data on total daily energy expenditures, and energy costs for growth, pregnancy, and lactation (breast-energy output and maternal milk-energy synthesis) were used to estimate rates for underweight, normal-weight, and overweight/obese females in prepregnancy, at weeks 9, 22, and 36 during pregnancy, and weeks 6 and 27 postpartum. “Underweight, normal-weight, and overweight/obese [were] defined as those having [body mass indices] BMIs lower than 19.8 kg/m², between 19.8 and 26 kg/m², and greater than 26 kg/m², respectively” ([Brochu et al., 2006](#)). [Brochu et al. \(2006\)](#) used data for 357 nonpregnant and nonlactating females and 91 pregnant and breastfeeding females. Monte Carlo simulations were used to integrate total daily energy requirements of nonpregnant and nonlactating females into energy costs and weight changes at the 9th, 22nd, and 36th weeks of pregnancy and at the 6th and 27th postpartum weeks. Energetic values in kcal/day and kcal/kg-day were converted into PDIRs in m³/day and m³/kg-day by using the equation developed by [Layton \(1993\)](#). Tables 5-12, 5-13, and 5-14 present the mean and 95th percentile PDIRs in m³/day and m³/kg-day for underweight, normal-weight, and overweight/obese females, respectively. Daily inhalation rates for normal weight women are approximately 18–41% higher during pregnancy and 23–39% higher during postpartum ([Brochu et al., 2006](#)). For all weight groups, inhalation rates were estimated to be higher during pregnancy and postpregnancy than before pregnancy, with decreases only evident among the 27th postpartum weeks time period.

Table 5-12. Simulated inhalation rates of prepregnant, pregnant, and postpartum lactating underweight women, aged 11 to 55 years^a

Group	Mean		95 th percentile	
	m ³ /day	m ³ /kg-day	m ³ /day	m ³ /kg-day
11 to <23 years	12.18	0.277	15.60	0.352
Prepregnancy	12.27	0.276	15.48	0.345
Pregnant, 9 th week	17.83	0.385	23.13	0.504
Pregnant, 22 nd week	17.98	0.343	23.90	0.455
Pregnant, 36 th week	18.68	0.323	25.59	0.452
Postpartum, lactating, 6 th week	20.39	0.368	24.82	0.548
Postpartum, lactating, 27 th week	20.21	0.383	24.61	0.584
23 to <50 years	13.93	0.264	17.65	0.342
Prepregnancy	13.91	0.264	17.81	0.361
Pregnant, 9 th week	20.03	0.366	26.94	0.501
Pregnant, 22 nd week	20.15	0.332	27.46	0.452
Pregnant, 36 th week	20.91	0.317	28.95	0.439
Postpartum, lactating, 6 th week	22.45	0.352	27.68	0.518
Postpartum, lactating, 27 th week	22.25	0.364	27.44	0.545
50 to <55 years	12.89	0.249	15.20	0.293
Prepregnancy	12.91	0.249	15.13	0.294
Pregnant, 9 th week	18.68	0.347	22.69	0.431
Pregnant, 22 nd week	18.84	0.315	23.20	0.401
Pregnant, 36 th week	19.60	0.301	25.58	0.404
Postpartum, lactating, 6 th week	21.19	0.337	24.53	0.457
Postpartum, lactating, 27 th week	21.01	0.349	24.31	0.483

^aBased on data for 81 underweight women. Number of simulated women = 5,000.

Source: [Brochu et al. \(2006\)](#).

Table 5-13. Simulated inhalation rates of prepregnant, pregnant, and postpartum lactating normal-weight women, aged 11 to 55 years^a

Group	Mean		95 th percentile	
	m ³ /day	m ³ /kg-day	m ³ /day	m ³ /kg-day
11 to <23 years				
Prepregnancy	14.55	0.252	18.71	0.339
Pregnant, 9 th week	19.99	0.344	25.89	0.468
Pregnant, 22 nd week	22.59	0.360	30.75	0.500
Pregnant, 36 th week	23.27	0.329	31.07	0.453
Postpartum, lactating, 6 th week	23.28	0.342	28.98	0.499
Postpartum, lactating, 27 th week	23.08	0.352	28.73	0.527
23 to <50 years				
Prepregnancy	13.66	0.222	17.87	0.285
Pregnant, 9 th week	19.00	0.308	24.49	0.395
Pregnant, 22 nd week	21.36	0.321	28.43	0.433
Pregnant, 36 th week	22.14	0.297	29.27	0.399
Postpartum, lactating, 6 th week	22.15	0.309	27.53	0.425
Postpartum, lactating, 27 th week	21.96	0.317	27.29	0.441
50 to <55 years				
Prepregnancy	13.79	0.229	17.02	0.287
Pregnant, 9 th week	19.02	0.314	23.38	0.400
Pregnant, 22 nd week	21.53	0.330	28.30	0.439
Pregnant, 36 th week	22.20	0.303	28.53	0.401
Postpartum, lactating, 6 th week	22.31	0.316	26.70	0.434
Postpartum, lactating, 27 th week	22.12	0.325	26.47	0.453

^aBased on data for 172 normal weight women. Number of simulated women = 5,000.

Source: [Brochu et al. \(2006\)](#).

Table 5-14. Simulated inhalation rates of prepregnant, pregnant, and postpartum lactating overweight/obese women, aged 11 to 55 years^a

Group	Mean		95 th percentile	
	m ³ /day	m ³ /kg-day	m ³ /day	m ³ /kg-day
11 to <23 years	16.62	0.206	21.41	0.261
Prepregnancy	16.64	0.207	20.06	0.253
Pregnant, 9 th week	25.51	0.302	33.32	0.401
Pregnant, 22 nd week	26.10	0.287	34.93	0.391
Pregnant, 36 th week	25.71	0.270	34.95	0.377
Postpartum, lactating, 6 th week	25.93	0.280	30.53	0.395
Postpartum, lactating, 27 th week	25.71	0.285	30.26	0.409
23 to <50 years	15.45	0.186	19.27	0.227
Prepregnancy	15.47	0.186	19.46	0.233
Pregnant, 9 th week	23.93	0.274	31.77	0.374
Pregnant, 22 nd week	24.44	0.261	33.49	0.360
Pregnant, 36 th week	24.15	0.245	34.18	0.360
Postpartum, lactating, 6 th week	24.47	0.256	29.43	0.360
Postpartum, lactating, 27 th week	24.25	0.260	29.17	0.372
50 to <55 years	15.87	0.184	20.01	0.235
Prepregnancy	15.83	0.184	19.47	0.226
Pregnant, 9 th week	24.47	0.272	33.08	0.378
Pregnant, 22 nd week	25.02	0.259	35.01	0.363
Pregnant, 36 th week	24.46	0.242	34.27	0.351
Postpartum, lactating, 6 th week	24.91	0.253	29.75	0.364
Postpartum, lactating, 27 th week	24.70	0.257	29.50	0.374

^aBased on data for 104 overweight/obese women. Number of simulated women = 5,000.

Source: [Brochu et al. \(2006\)](#).

5.5. ACTIVITY FACTORS AND CONSUMER PRODUCT USE

Most of the activity pattern studies located in the literature addressed relationships between physical activity (e.g., exercise) and birth weight or pregnancy outcome ([Evenson and Wen, 2010](#); [Borodulin et al., 2009](#); [Borodulin et al., 2008](#); [Mottola and Campbell, 2003](#)). Few data are available on activity factors that can be used to evaluate the relationship between time use and exposure to environmental agents among pregnant and lactating women.

[Nethery et al. \(2009\)](#) compared the time-activity patterns among a nonrandom sample of 62 pregnant Canadian women and a comparison group of 103 women in the Canadian Human Activity Pattern Study (CHAPS). The data were collected in 2005–2006. Changes in location-based activity patterns (i.e., at or near home, work, other indoor locations, outdoors, car, bus, walk, bike) were measured over the course of pregnancy. The mean and 95% CI for the pregnancy cohort and the CHAPS comparison group are provide in Table 5-15.

Table 5-15. Mean (95% CI) time spent in various activities (hours/day), by trimester among a population of Canadian women

Activity/ location	Pregnant cohort N = 62	Pregnant cohort by trimester			CHAPS comparison group N = 103
		1 st trimester N = 11	2 nd trimester N = 62	3 rd trimester N = 54	
At/near home	16.2 (15.7–16.8)	14.4 (13.3–15.4)	16.1 (15.3–17.0)	16.9 (16.0–17.8)	15.5 (14.7–16.3)
Work	4.2 (3.6–4.7)	5.6 (4.4–6.7)	4.3 (3.5–5.1)	3.7 (2.8–4.6)	3.8 (3.0–4.6)
Indoors, other	1.6 (1.3–1.8)	2.2 (0.9–3.6)	1.6 (1.3–1.9)	1.4 (1.1–1.7)	2.5 (1.9–3.0)
Outdoors	0.3 (0.2–0.4)	0.0 (0.0–0.0)	0.2 (0.07–0.2)	0.4 (0.2–0.6)	0.6 (0.3–0.8)
Car	0.9 (0.7–1.0)	1.1 (0.5–1.7)	0.9 (0.7–1.1)	0.8 (0.6–1.0)	1.4 (1.1–1.7)
Bus	0.2 (0.2–0.3)	0.3 (0.01–0.5)	0.2 (0.1–0.4)	0.2 (0.08–0.3)	0.1 (0.1–0.2)
Walk	0.7 (0.5–0.8)	0.4 (0.2–0.7)	0.7 (0.6–0.9)	0.6 (0.4–0.8)	0.2 (0.1–0.2)
Bike	0.1 (0.0–0.1)	0.02 (0.0–0.07)	0.06 (0.0–0.1)	0.06 (0.0–0.1)	0.0 (0.0–0.1)

CI = Confidence interval.

N = Sample size.

Source: [Nethery et al. \(2009\)](#).

In 1996 and 1997, [Zender et al. \(2001\)](#) conducted a study in Colorado to compare tapwater intake among pregnant and nonpregnant women. Data were also collected on activities resulting in dermal contact with tapwater (e.g., showering, bathing, swimming, cleaning, etc.). A total of 71 pregnant and 43 nonpregnant women were recruited from WIC clinics; nearly one-half of the pregnant women were in their second trimester, and one-quarter were in each of the first and third trimesters. Most of the women were white and had fewer than 13 years of education. The average ages were 24 years for pregnant women and 27 years for nonpregnant women. Approximately one-half of the women worked outside the home, and nearly all the women used municipal water source in their home. The women were interviewed in person or by phone and responded to questions about tapwater usage and activities such as frequency and duration of showering, bathing, and swimming. Table 5-16 shows the statistics for these activities and others including bathing children or pets and washing dishes, clothes, or cars. The frequency and duration of showering was similar for pregnant and nonpregnant women, but pregnant women spent more time bathing than nonpregnant women.

Table 5-16. Activities associated with exposure to water, by pregnancy status in a population of women in Colorado

Characteristic	Pregnant (N = 71)	Not pregnant (N = 43)
Showering at home (%)	97.2	100.0
Showers per week ^a (mean ± SD)	7.1 ± 2.8	7.2 ± 2.4
Duration of showers (minutes)	13.9 ± 5.2	13.9 ± 6.0
Bathing at home (%)	50.7	37.2
Baths per week ^b (mean ± SD)	3.0 ± 3.5	1.4 ± 1.1
Duration of baths (minutes)	28.8 ± 12.9	41.3 ± 30.3
Swimming (%)	25.4	27.9
Swimming per week ^c (mean ± SD)	1.3 ± 1.6	0.6 ± 0.5
Duration of swimming (minutes)	73.9 ± 46.1	80.8 ± 79.3
Bathing children (%)	43.7	81.4
Washing dishes (%)	66.2	76.2
Washing clothes (%)	14.1	21.4
Washing cars (%)	18.3	31.0
Bathing pets (%)	18.3	11.9

^aAmong women showering at home.

^bAmong women bathing at home.

^cAmong women swimming.

N = Number of observations.

SD = Standard deviation.

Source: [Zender et al. \(2001\)](#).

[Bell and Belanger \(2012\)](#) studied women's residential mobility (i.e., change of residence) during pregnancy and the potential implications for environmental exposures during pregnancy. Data from 14 studies on residential mobility among pregnant women were examined for overall mobility rates and distances moved. Of the 14 studies, 7 were based in the United States, and the remaining 7 were based in the United Kingdom, Canada, The Netherlands, Norway, and Australia. The percentage of women who moved during pregnancy ranged from 9–32% (median = 20%). [Bell and Belanger \(2012\)](#) reported that more moves occurred during the second trimester of pregnancy, based on the studies that presented data by trimester. Several other factors were found to affect mothers' residential mobility including age (the probability of moving generally declined with age), socioeconomic status (mobility was generally higher

among women with lower income), marital status (married women were less likely to move), and parity (rates were generally higher in women with fewer pregnancies). Relationships with factors such as race, smoking, and alcohol use were more variable. Of the studies that reported on the distance moved, most distances were short, with median values typically <10 km.

Consumer product use data for pregnant women are also limited. No consumer products use data were found for lactating women. [Just et al. \(2010\)](#) conducted a survey among 186 women, 18–35 years old, who self-identified as either African-American or Dominican and had resided in Northern Manhattan or the South Bronx for at least 1 year prior to pregnancy. The women were part of the Mothers and Newborns cohort study of the Columbia Center for Children’s Environmental Health. The primary objective of the study was to explore relationships between the use of personal care products and exposure to phthalates. Participants were included in this study if phthalates were “measured within a week in either a personal air and/or urine sample collected during the third trimester of pregnancy.” Consumer product use questionnaires were administered to study participants in the third trimester of pregnancy. The questionnaire was designed to gather information on the total number of uses over the previous 48 hours and the frequency of use during each trimester of pregnancy (>1/day, 1/day, 2–3/week, 1/week, <1/week–1/month, <1/month), based on recall. Data were collected for seven product categories: deodorant, lotion or mist (spray application), perfume, liquid soap or body wash, hair gel, hair spray, and nail polish or nail polish remover.

[Just et al. \(2010\)](#) summarized the percentage of participants using the personal care products over a 48-hour survey period and throughout the individual trimesters of pregnancy in 2003–2006 (see Table 5-17). Participants used an average of three product types of the seven product categories. The median number of times that women in the study used any product was seven (range = 1–26; $N = 180$). The product with the most prevalence use was deodorant (98%). Liquid soap was the most frequently used product category with a mean of 3.4 uses in 48 hours, followed by lotion and deodorant. Perfume use was reported by 41% of participants in the 48 hours of the study and was slightly higher among African-Americans (45%) than among Dominicans (40%); although the difference in proportions was not significant. The median number of perfume uses over the 48-hour period was two. Overall, 84% of the women in the study reported using perfume at some point throughout their pregnancy. Among the women who reported information about frequency of perfume use during the third trimester ($N = 166$), 37% reported using it at least daily. Nail product use was reported by 10% of participants (18 of $N = 186$) over the 48-hour period. Among those women who reported nail product use, all reported a single use over the 48-hour period. Overall, 69% of the women reported using nail products at some point during their pregnancy.

Table 5-17. Percentage of a population of pregnant minority women residing in New York ($N = 186$) who reported use of selected personal care products over a 48-hour survey period

Personal care product	%
Deodorant	98
Lotion	82
Perfume	41
Liquid soap	29
Hair gel	25
Hair spray	10
Nail polish or polish remover	10

$N =$ Sample size.

Source: [Just et al. \(2010\)](#).

5.6. BODY WEIGHT

[Janney et al. \(1997\)](#) evaluated body weight among a sample of women in Ann Arbor, Michigan area. Prepregnancy body weights of 110 women were compared to postpartum weights at 0.5, 2, 4, 6, 12, and 18 months after parturition. The women ranged in age from 20–40 years, and most were white (106 whites, 1 Asian-American, and 3 African-Americans). Data on weight gained during pregnancy, pre- and postpregnancy body weights for the women, as well as information on weight retained after pregnancy, are provided in Table 5-18. Mean body weight declined from 67.2 kg at 0.5 months after parturition to 62.4 kg at 12 months after parturition (see Table 5-18).

Table 5-18. Weight gained during pregnancy, and pre- and postpregnancy body weight (kg), among a population of Michigan women (N = 110)

Variable	Mean ± SD (range)
Weight before pregnancy	59.7 ± 9.7 (43.1–93.0)
Weight gained during pregnancy	
First trimester	3.1 ± 2.9 (–4.5–11.3)
Second trimester	6.4 ± 3.0 (–6.8–13.6)
Third trimester	6.5 ± 2.9 (1.4–18.1)
Total	16.2 ± 5.2 (1.4–35.8)
Postpregnancy weight	
0.5 month	67.2 ± 1.0 (47.9–96.4)
2 months	65.5 ± 1.0 (49.3–94.9)
4 months	64.3 ± 1.0 (48.6–3.2)
6 months	63.6 ± 1.0 (47.2–94.2)
12 months	62.4 ± 1.1 (44.4–96.0)
18 months	63.8 ± 1.3 (47.8–98.4)
Retained weight, postpregnancy	
0.5 month	7.4 ± 0.5 (–6.5–20.8)
2 months	5.8 ± 0.4 (–5.1–16.8)
4 months	4.7 ± 0.4 (–5.8–15.5)
6 months	3.9 ± 0.4 (–7.5–15.1)
12 months	2.5 ± 0.5 (–8.3–13.5)
18 months	3.0 ± 0.5 (10.1–14.5)

N = Sample size.
SD = standard deviation.

Source: [Janney et al. \(1997\)](#).

[Carmichael et al. \(1997\)](#) conducted a study in 4,218 California women who had good pregnancy outcomes between 1980 and 1990 to obtain the distribution of maternal weight gain by trimester. A good pregnancy outcome was defined as a “vaginal, term (37 or more completed weeks’ gestation) delivery of a live infant of average size for gestational age (i.e., between the 10th and 90th percentiles of gestation specific birthweight, based on data from more than

2 million California births) to a mother without diabetes or hypertension.” The average age of the women was 27.7 years. The mean prepregnancy weight for these women was 57.6 kg; 29% were underweight, 61% were of normal weight, 5% were overweight, and 4% were obese, based on BMI calculations. The difference between the self-reported prepregnancy weight and the last measured weight was used to estimate weight gain. Table 5-19 presents the estimated mean \pm SD weight gain for underweight, normal-weight, overweight, and obese women during the first, second, and third trimesters of pregnancy. The average weight gains for the first, second, and third trimesters, calculated by averaging the weight gains for the four groups (i.e., underweight, normal weight, overweight, and obese), were 1.98 kg, 6.73 kg, and 6.37 kg, respectively. Based on the prepregnancy weight of 57.6 kg, total body weights for the first, second, and third trimesters would be 59.6 kg, 66.3 kg, and 72.7 kg, respectively (i.e., calculated by adding the average weight gain for each trimester to the prepregnancy weight and the previous trimester weight).

Table 5-19. Weight gained during pregnancy (kg), for populations of underweight, normal-weight, overweight, and obese women in California who had good pregnancy outcomes (N = 4,218)

Variable	Mean ± SD
first trimester	
Underweight	1.92 ± 3.06
Normal weight	2.19 ± 3.47
Overweight	2.16 ± 3.95
Obese	1.65 ± 3.94
second trimester	
Underweight	7.41 ± 2.60
Normal weight	7.54 ± 2.86
Overweight	6.63 ± 3.12
Obese	5.33 ± 3.51
third trimester	
Underweight	6.24 ± 2.47
Normal weight	6.63 ± 2.73
Overweight	6.37 ± 2.86
Obese	6.11 ± 3.12

N = Sample size
SD = standard deviation.

Source: [Carmichael et al. \(1997\)](#).

In 2010, EPA analyzed body weight data for 1,248 pregnant women from the 1999–2006 NHANES. After removing a few very large and improbable body weights (i.e., outliers), the statistically weighted average body weight of all pregnant women was 75 kg (see Table 5-20) ([U.S. EPA, 2011](#)). The same data showed the average body weight for all women aged 16–40 years old is 71 kg ([U.S. EPA, 2011](#)).

Table 5-20. Estimated body weight (kg) of pregnant women—NHANES (1999–2006)^a

Trimester	N	Mean	SD	Percentiles								
				5 th	10 th	15 th	25 th	50 th	75 th	85 th	90 th	95 th
1	204	76	3	48	50	55	60	74	91	98	106	108
2	430	73	1	50	53	57	61	72	83	93	95	98
3	402	80	1	60	63	65	69	77	88	99	104	108
Ref/Dk ^b	186	69	2	46	52	55	60	65	77	84	87	108
All	1,222	75	1	50	55	59	63	73	85	94	99	107

^aDue to a few large weight (>90 kg) respondents with very large sample weights (>18,000 kg), the weighted mean body weight of first trimester women (76 kg) is larger than that of second trimester women (73 kg).

^bRefers to pregnant women who either refused to tell which trimester they were in, or did not know, or when data were missing.

N = Sample size.

SD = standard deviation.

Source: [U.S. EPA \(2011\)](#).

Lactation can also have an effect on body weight. One study conducted in a cohort of 405 Brazilian women suggested that breastfeeding for longer periods of time might contribute to decreases in postpartum weight retention ([Kac et al., 2004](#)). [Brewer et al. \(1989\)](#) examined postpartum weight changes in 56 Louisiana women. The women were over 18 years of age (mean age = 27 years) and were predominantly white, well educated, and middle to upper-middle income. The women were assigned to one of three groups according to the method of feeding the infants during the first 6 months of life: exclusive breastfeeding, exclusive formula feeding, or a combination of breastfeeding and formula feeding. The mothers were weighed in the hospital 1 to 2 days postpartum and at home at 3 and 6 months after delivery. Although responses from individual participants varied, overall, there was a steady, significant decline in weight for all three groups, and the most weight loss occurred during the first 3 months (see Table 5-21). With the exception of one woman who showed essentially no change in weight and two women who gained weight, total weight losses ranged from 1.9–20.9 kg over the 6-month study period for all participants. Weight losses averaged 8.30 kg for the breastfeeding group, 8.19 kg for the formula feeding group, and 7.23 kg for the combination of breastfeeding and formula. Weight loss after pregnancy may have implications with regard to lipid mobilization (e.g., contaminants stored in lipid).

Table 5-21. Mean \pm SD prepregnancy weight, pregnancy weight gain, and postpartum weight loss (kg) among a population of Louisiana women ($N = 56$)

Lifestage group	Pregpregnancy weight	Pregnancy weight gain	Postpartum weight loss ^a		
			0 to 3 months	3 to 6 months	0 to 6 months
Breastfeeding	59.8 \pm 13.1	14.6 \pm 5.8	6.75 \pm 0.53 ^b	1.29 \pm 0.64 ^c	8.30 \pm 0.74 ^b
Formula feeding	54.9 \pm 6.0	16.3 \pm 5.7	8.14 \pm 0.68 ^b	0.16 \pm 0.85	8.19 \pm 0.96 ^b
Combination	57.3 \pm 7.5	14.6 \pm 5.0	6.39 \pm 0.53 ^b	0.82 \pm 0.65	7.23 \pm 0.73 ^b

^aPostpartum weight loss measured in reference to the last weight recorded before delivery.

^bRepresents a statistically significant change ($p < 0.001$).

^cRepresents a statistically significant change ($p < 0.05$).

N = Sample size.

SD = Standard deviation.

Source: [Brewer et al. \(1989\)](#).

Weight loss during lactation was studied in California women participating in the Davis Area Research on Lactation Infant Nutrition and Growth study by [Dewey et al. \(1993\)](#). A total of 46 mothers who breastfed their infants and 39 mothers who fed their infants formula were included in the study. The mean ages, education levels, pregnancy weight gains, and prepregnancy weights of the women in the two groups were similar. Infants in the breastfeeding group received <120 mL/day of other milks until at least 12 months of age, and more than one-third of the infants were breastfed for longer than 18 months. The mothers' weights were measured monthly from 1 to 18 months, and at 21 and 24 months postpartum. The results indicated that weight loss among the two groups of women was similar at 1 month postpartum, but the breastfeeding women weight losses were statistically significantly greater than that of the formula-feeding group in subsequent months. At 6 months postpartum, the breastfeeding group had an average body weight that was approximately 2.8 kg lower than that of the formula-feeding group (see Figure 5-2). At 12 months the breastfeeding group had a mean body weight that was 3.2 kg lower than the formula-feeding group. Table 5-22 presents data showing the differences in weight loss for the two groups of mothers. Over the first 12 months postpartum, breastfeeding mothers lost 4.4 kg compared to 2.4 kg for the formula-feeding mothers. Other factors that contributed to greater weight loss were parity and mothers' height.

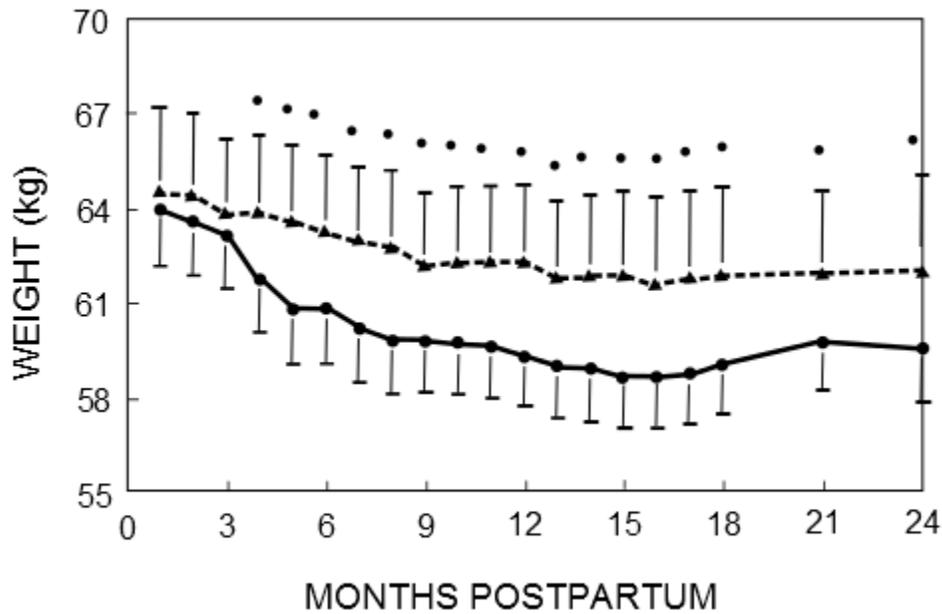


Figure 5-2. Weight of breastfeeding (●, *N* = 26) and formula-feeding (▲, *N* = 27) women during the first 24 months postpartum.

From Am. J. Clin. Nutri. (1993; 58; 162-166), American Society for Nutrition. Reprinted with permission. Source: [Dewey et al. \(1993\)](#).

Table 5-22. Mean ± SD changes in maternal body weight in populations of breastfeeding and formula feeding California women

Postpartum Timeframe	Breastfeeding		Formula feeding	
	<i>N</i>	Body weight change (kg)	<i>N</i>	Body weight change (kg)
1 to 3 months	37	-1.2 ± 2.1	27	-1.0 ± 2.3
3 to 6 months	42	-2.2 ± 2.2	31	-0.3 ± 1.9
6 to 9 months	40	-0.6 ± 1.5	30	-0.9 ± 1.8
9 to 12 months	35	-0.3 ± 1.5	33	-0.0 ± 1.6
Total (1 to 12 months)	29	-4.4 ± 3.4	21	-2.4 ± 5.0

N = Sample size.
SD = Standard deviation.

Source: [Dewey et al. \(1993\)](#).

[Janney et al. \(1997\)](#) also evaluated weight loss according to how the women fed their infants. The women in the study were categorized as fully breastfeeding, partly breastfeeding, or bottle feeding. [Janney et al. \(1997\)](#) found that women who bottle fed their infants retained more weight over time than women who breastfed their infants. Lactating women retained less of their body weight gained during pregnancy than did nonlactating women. The duration of lactation practice was found to be a significant predictor of postpartum weight retention over time ([Janney et al., 1997](#)). Figure 5-3 depicts weight losses according to breastfeeding practices.

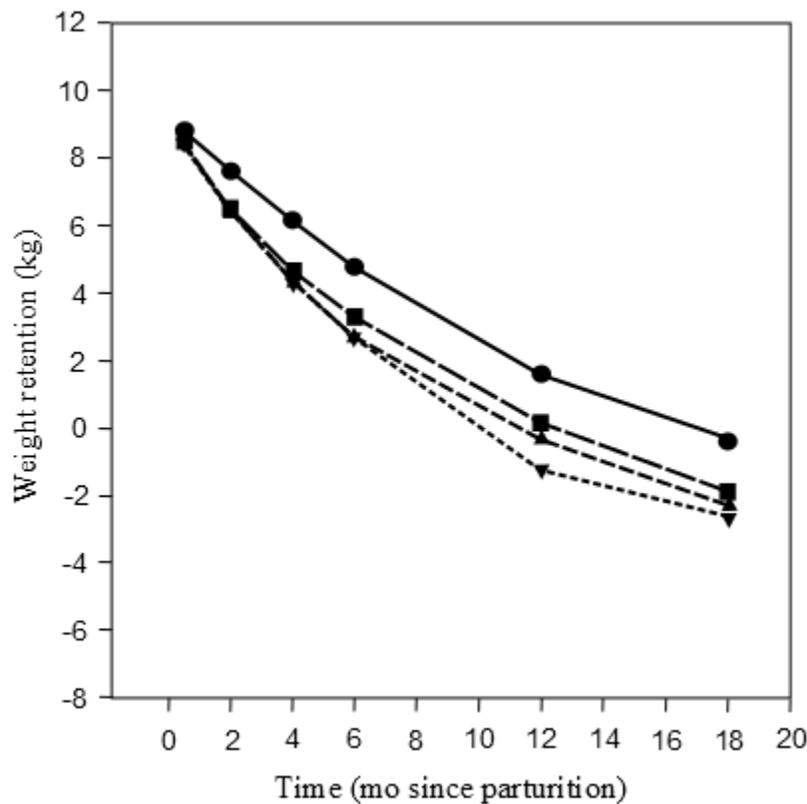


Figure 5-3. Predicted weight-retention curves over time for four lactation practices. Bottle-feeding only (●); fully breastfeeding at 2 weeks, partly breastfeeding at 2 months, and bottle feeding or infant weaned at 4, 6, 12, and 18 months (■); fully breastfeeding for 6 months and bottle-feeding or infant weaned at 12 and 18 months (▲); and fully breastfeeding for 6 months, partly breastfeeding for 12 months, and bottle feeding or infant weaned at 18 months (▼).

From Am. J. Clin. Nutr. (1997; 66; 1116-1124), American Society for Nutrition. Reprinted with permission.

Source: [Janney et al. \(1997\)](#).

Another study investigated the effect of protein intake during pregnancy and postpartum body weight changes ([Castro et al., 2010](#)). Dietary intake information was obtained using a food frequency questionnaire from a cohort of 421 women in Rio de Janeiro, Brazil at 15 days, 2, 6, and 9 months postpartum. The study found a positive association between postpartum weight loss and protein intake during pregnancy. The average postpartum weight loss was 0.409 kg/month (± 0.12). Women with adequate protein intake (≥ 1.2 g/kg body weight) lost an additional 0.094 kg/month (± 0.04) than women with inadequate protein intake (< 1.2 g/kg body weight) ([Castro et al., 2010](#)).

6. DATA GAPS

This issue paper provides an overview of the physiological and behavioral changes that occur during pregnancy and lactation, and exposure factors for pregnant and lactating women. It is not meant to provide a comprehensive review of every aspect of pregnancy and lactation, but is meant to introduce topics related to women's potential susceptibility to environmental contaminants as a result of differences in physiology and behavior, summarize key areas that may be relevant to exposure, and highlight areas where data gaps exist. Not all changes during pregnancy and lactation would be likely to result in new or different environmental exposures, but some may increase or decrease exposures to environmental contaminants.

Sections 3 and 4 of this paper present the available literature with respect to physiological and behavioral changes during pregnancy and lactation. However, the direct link between these changes and the potential for experiencing differential exposures is not well understood and is a significant data gap. Most of the physiological information found in the literature relates to pregnancy, while the information on lactating women is more limited. This may be because most of the physiological changes occurring during lactation relate to hormonal changes necessary for the production of milk and the resulting mobilization of calcium from the bones. Other organ systems may not undergo significant changes. The remainder of this section (see Section 6) summarizes some of the data gaps pertaining to exposure factors for pregnant and lactating women and highlights areas where additional information would be useful for estimating exposures among this potentially susceptible lifestage.

Most of the research on pregnant and lactating women has been conducted for clinical purposes and is aimed at providing recommendations for pregnant and lactating women in support of a good pregnancy outcome, or to support the health of the nursing infant. Research to develop exposure factor data specifically for pregnant women is somewhat limited, but data for some factors are available (see Section 5). Exposure factor data for lactating women is even more limited. For factors where data are available, some are based on studies conducted on a specific geographical location or for a specific demographic group. These data may not be representative of pregnant and lactating women in other areas of the United States or within other demographic groups. For instance, several studies are available regarding fish consumption during pregnancy, but these are limited to specific geographical locations or tribal groups. The results of these studies may not be generalizable to other populations. Fish consumption rates may be highly dependent on geographical location, cultural practices, and other factors. These studies were also primarily conducted to assess knowledge and compliance with fish advisories rather than intake.

Some data are available at the national level, but these data are sometimes limited by sample size. For example, data on consumption of community water are available on a national scale, but the sample size for pregnant and lactating women is small. The national-scale analysis of food intake by pregnant women also suffers from small sample size limitations, and data are not available for lactating women. Some studies report on adaptive behaviors with regard to food consumption, but do not provide information on amounts of food consumed. Also, although research is available on the adequacy of the pregnant woman's diet with regard to the intake of certain nutrients (e.g., calcium, folic acid, iron, vitamin D) needed for a good pregnancy outcome, data on the quantities of foods is more limited. In addition, the role that race, age, ethnicity, geographical location, and socioeconomic factors plays in the variability with regard to these exposure factors for this lifestyle is not well understood.

Activity pattern data are limited for pregnant women in the U.S. population, and are entirely lacking for lactating women. Many of the studies available examined the relationship between physical activity and pregnancy outcomes, but did not provide information about the duration and frequency of activities. One study provided information on time spent in a limited number of activities for a population of pregnant Canadian women. Another study conducted in the United States reported on activities related to water exposures in a small population of pregnant women in Colorado. Another study on residential mobility examined the data from seven studies conducted in the United States that reported on the percentage of pregnant women who move during pregnancy and the distance moved, but provided no information on residence time. The use of consumer products by pregnant and lactating women is another area where research may be warranted. There is some information regarding the percentage of pregnant women using selected personal products, but no information about the amount of product use and the frequency of use.

For some behaviors, there are data with regard to the prevalence of the behavior, but quantitative estimates on the frequency and duration of the activity are not available. For example, there is some information about the prevalence of pica behavior among pregnant women, but limited data on the amount of material ingested. In addition, the data on the prevalence of pica are primarily based on older studies and may not reflect current behaviors. No data on pica behavior during lactation were found.

For some exposure factors, no data for pregnant and lactating women were available. For example, no data on skin surface area were found, but these data could be generated from height and weight data for these pregnant women. While there are some exposure factors data for pregnant women, data for lactating women are lacking. Factors for which no data are available for lactating women include: food intake, soil intake, prevalence of pica or geophagy, activity patterns, and consumer product use.

More importantly, additional analyses are needed to understand whether differences between the exposure factors for pregnant and lactating women and those of the general population of women are significant in terms of exposure and risk. Some of the questions that need to be answered include:

- Are differences in exposure factor values statistically different (i.e., at the mean or upper end of the distribution)?
- Would such differences result in statistically relevant differences in exposure and risk?
- Would risk management decisions differ if based on the pregnant and lactating women instead of women in the general population?
- When (e.g., for what types of chemicals) would it be appropriate to base exposure/risk assessments on pregnant and lactating women?
- Are current risk assessment practices and guidances properly accounting for variations in this potentially susceptible lifestage?

These and other questions remain in terms of how the information in this issue paper may be applied in the context of the assessment and management of risk.

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**APPENDIX. COMPARISON OF COMMODITY
CONSUMPTION PATTERNS OF PREGNANT AND
NONPREGNANT WOMEN OF CHILDBEARING AGE.**



UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
WASHINGTON, D.C. 20460

OFFICE OF CHEMICAL SAFETY AND
POLLUTION PREVENTION

MEMORANDUM

DATE: April 3, 2013

SUBJECT: Comparison of Commodity Consumption Patterns of Pregnant and Non-Pregnant Women of Childbearing Age, WWEIA-FCID 2003-08

PC Code: NA
Decision No.: 476479
Petition No.: NA
Risk Assessment Type: NA
TXR No.: NA
MRID No.: NA

DP Barcode: 410152
Registration No.: GP-33396
Regulatory Action: NA
Case No.: NA
CAS No.: NA
40 CFR: NA

Ver. Apr. 08

FROM: Bayazid H. Sarkar, Mathematical Statistician
and
James Nguyen, Mathematical Statistician
Chemistry and Exposure Branch,
Health Effects Division (7509P)

THROUGH: David J. Miller, Chief
Chemistry and Exposure Branch,
Health Effects Division (7509P)

TO: Jacqueline Moya, Environmental Engineer
National Center for Environmental Assessment (8623P)
Office of Research and Development

SUMMARY

CEB was requested by ORD's National Center for Environmental Assessment (NCEA) to examine and compare food commodity consumption patterns of pregnant women and non-pregnant women of childbearing age. NCEA intends to use this information to update the *Exposure Factors Handbook* (EFH) and also intends to release an Issue Paper on exposure factors for pregnant women later this year. CEB had previously provided updated food consumption data (based on the NHANES/WWEIA 2003-2006 survey) to NCEA for various demographic subpopulations, and this data was incorporated into the 2011 EFH issued during the

fall, 2011. In order to be consistent with our previous analysis and the 2011 EFH data and previous versions to the EFH and as requested by NCEA, the summary statistics here represent 2-day averages of those survey respondents who report both days of consumption. Thus, the mean and various percentile amounts are average values over the two non-consecutive days of the survey.

At NCEA's request, CEB's new analysis compared dietary consumption patterns for pregnant vs. non-pregnant females of child bearing age (here, assumed to be ages 13-49). With respect to overall consumption of fruits and vegetables, we found that pregnant females have a slightly higher per capita mean intake of total vegetables (2.74 g/kg-day) than non-pregnant females (2.43 g/kg-day), which is statistically significant. The 95th percentile of per capita consumption of total vegetables for pregnant females (6.26 g/kg-day) is also higher than that of non-pregnant females (5.93 g/kg-day). With respect to total fruit consumption, pregnant females on average consume more total fruits (1.79 g/kg-day consumers only, 1.66 g/kg-day per capita basis) than non-pregnant females (1.18 g/kg-day consumers only, 0.98 g/kg-day per capita basis), and this difference is statistically significant. The 95th percentile of per capita consumption of total fruits for pregnant females (5.02 g/kg-day) is also higher than that of non-pregnant females (3.75 g/kg-day).

In addition, we found that pregnant females in general in the years examined consumed greater amounts of certain commodities, including (in particular) citrus, peaches, cucurbits, and tropical fruits. We also found that pregnant females tended to consume lesser amounts of other commodities, including less cabbage, stalk and stem vegetables, leafy vegetables, and lettuce.

Note that multiple tests have been performed in our analysis, and any putative statistical differences have not been corrected to account for this. Many of these differences may not have been found to be statistically significant if such adjustments had been made. Thus, it is left to the data user to determine if differences in mean consumption between pregnant and not pregnant females of child-bearing age are substantive from an exposure assessment viewpoint. It is also important for the user to understand that differences in consumption in an exposure or risk assessment context may or may not produce differences in exposure or risk in that increased

consumption of one commodity or type of commodity may result in reduced consumption of other commodities/commodity types.

DETAILED ANALYSIS

Overview and Methods

Since OPP has updated the NHANES/WWEIA 2003-2006 data we originally provided to NCEA to include the 2007-2008 consumption data, our current analysis is based on the NHANES 2003-2008 data that is now available at www.fcid.foodrisk.org. At NCEA's request, our analysis focuses on the 2-day average consumption of food commodities (e.g., apples, bananas) and associated commodity groupings (e.g., citrus, leafy vegetables, fruiting vegetables) that had been defined previously in the EFH (available at <http://cfpub.epa.gov/ncea/risk/recordisplay.cfm?deid=236252#tab-3>). This was done to be consistent with previous analyses and with previous versions of the EFH. CEB compared consumption of pregnant women and not-pregnant women, aged 13 – 49 years, where pregnancy status was defined in the NHANES survey by 'ridexprg²' variable in the NHANES demographic file.

NHANES/WWEIA collects two days of 24-hour dietary recall on foods using WWEIA standardized food vocabulary. Because the WWEIA food vocabulary is based on foods as reported eaten, U.S. EPA's Food Commodity Intake Database (FCID) was used to convert WWEIA food consumption into consumption of individual food commodities. The intake rates we calculated represent 2-day averages of intake of all forms of the commodities for pregnant and non-pregnant women of childbearing age (13 – 49 years old) who provided two complete days of 24-hour dietary recall. Two-day average intake rates of the commodities and commodity groups of interest were first calculated for women of childbearing age. After calculating two-day average intake for each survey respondent, summary statistics were calculated on both a

² Ridexprg=1 indicates Yes, positive lab pregnancy test or self-reported pregnant at exam; Ridexprg=2 indicates SP not pregnant at exam.

consumer-only and on a per capita basis using six-year survey weights and NHANES survey design variables (i.e., strata and primary sampling unit). See the appendix-3 for the detailed summary statistics of pregnant and non-pregnant females of child bearing age (13-49 years).

Summary statistics that were calculated for each commodity and commodity group included:

- Number of consumers,
- Percentage of the population reported consumption,
- Mean consumption,
- Standard error,
- Minimum and maximum, and,
- Various selected percentiles ranging from the 1st to the 99th.

SAS version 9.3 was used for statistical analysis. Statistical comparisons of mean consumption between pregnant and non-pregnant females (each aged 13-49 years old) were also evaluated using an alpha level of 0.05. Table 1 and Table 2 in Appendix 3 provide the detailed summary statistics on both consumers only and per capita basis. Appendices 1 and 2 present the plot of ratios³ of mean to different percentiles for consumption between pregnant and non-pregnant females within child bearing age (13-49 years old). EPA uses the recommendation based on Appendix-B of “Analytic And Reporting Guidelines: The Third National Health and Nutrition Examination Survey, NHANES III (1988-94)” (available at : <http://www.cdc.gov/nchs/data/nhanes/nhanes3/nh3gui.pdf>) and “Healthy People 2010 Criteria for Data Suppression (2002)” (available at: <http://www.cdc.gov/nchs/data/statnt/statnt24.pdf>) for determining reliability of tail percentiles.

³ Ratio = statistic of consumption of pregnant female / statistic of consumption of non-pregnant female; ratio > 1 indicates higher consumption for pregnant female.

RESULTS AND DISCUSSION

This section presents some of the highlights of the dietary consumption of pregnant and non-pregnant females. Details of the result can be found in Appendix 3.

Pregnant females have a slightly higher per capita mean intake of total vegetables (2.74 g/kg-day) than non-pregnant female (2.43 g/kg-day), which is statistically significant (see Table 2 in Appendix 3)⁴. The 95th percentile of per capita consumption of total vegetables for pregnant females (6.26 g/kg-day) is also higher than that of non-pregnant females (5.93 g/kg-day) (Table 2, Appendix 3). On average, pregnant females consume more total fruits (1.79 g/kg-day consumers only, 1.66 g/kg-day per capita basis) than non-pregnant females (1.18 g/kg-day consumers only, 0.98 g/kg-day per capita basis) (See Table 1 and and Table 2). The 95th percentile of per capita consumption of total fruits for pregnant females (5.02 g/kg-day) is also higher than that of non-pregnant females (3.75 g/kg-day) (Table 2).

For some of the individual fruit categories, pregnant women were also found to have higher commodity consumption than non-pregnant women. As an example, pregnant women have significantly higher average citrus fruit consumption than non-pregnant women on both a consumers only (1.21 g/kg-day and 0.50 g/kg-day, respectively) and a per capita basis (0.29 g/kg-day and 0.10 g/kg-day) (Table 1 and Table 2). Figure 9 demonstrates that 95th percentiles of per capita consumption of citrus and peaches among pregnant females are more than twice than that of non-pregnant females. In contrast, pregnant females consume less cabbage, stalk and stem vegetables, leafy vegetables, and lettuce than non-pregnant females at both mean and upper-end percentile consumptions (i.e., ratio of means and 95th percentiles are less than 1; see Figure 1, Figure 5, and Figure 9).

⁴ Multiple testing, meaning simultaneous testing of several hypotheses, can be a concern due to inflating the combined type I error (http://ies.ed.gov/ncee/pubs/20084018/app_b.asp). Note that multiple tests have been performed and the statistical differences cited here have not been corrected for this. Many of these differences may not have been found to be statistically significant if such adjustments had been made. Thus, it is left to the data user to determine if differences in mean consumption between pregnant and not pregnant females of child-bearing age are substantive from an exposure assessment viewpoint.

Although pregnant females were found to have statistically significantly higher mean consumption than non-pregnant females for total grains (2.12 g/kg-day vs. 1.90 g/kg-day on a per capita basis, respectively) (see Table 1 and and Table 2), the ratios of the mean and upper percentiles are all close to one (Figures 1, 5, 6, 9) which may indicate that the difference may not be substantial. The average per capita consumption of total dairy for pregnant females is higher than that of non-pregnant females (5.04 g/Kg-day vs. 3.53 g/Kg-day from Table 2). There was no statistically significant difference found in total fish and total meat consumption between pregnant and non-pregnant women.

It is important to consider a number of issues while evaluating consumption by pregnant and non-pregnant females. For example: whether there are sufficient numbers of individuals in each group; whether there is a statistically significant difference in the means; whether there are differences in the upper percentiles (e.g., 75th, 90th, and 95th); how much of a difference is important from a substantive (exposure assessment) point of view; and how these should be considered on a combined/synthesized basis. It should be noted that statistical tests for the difference in consumption between pregnant and non-pregnant females was performed for the mean, and NOT for the percentiles. In many cases at the upper (and lower) percentiles of the consumers only distribution (and particularly for less commonly consumed food commodities or commodity groupings), there are not adequate numbers of individuals to produce reliable estimates of consumption. While there may be statistically significant consumption differences between the two groups, this does not necessarily imply there would be differences in exposure or risk.

APPENDICES

Appendix 1: Plots of ratio of Consumption between Pregnant and Non-pregnant Females (Consumers Only)

Appendix 2: Plots of ratio of Consumption between Pregnant and Non-pregnant Females (Per Capita)

Appendix 3: Tables of Consumption

Appendix 4: SAS codes

Appendix 5: Reference

Appendix 1

Plots of ratio of consumption between Pregnant and Non- Pregnant Females (Consumers Only)

Figure 1: Plot of the Mean Ratio in rank order (Consumers only)

A-10

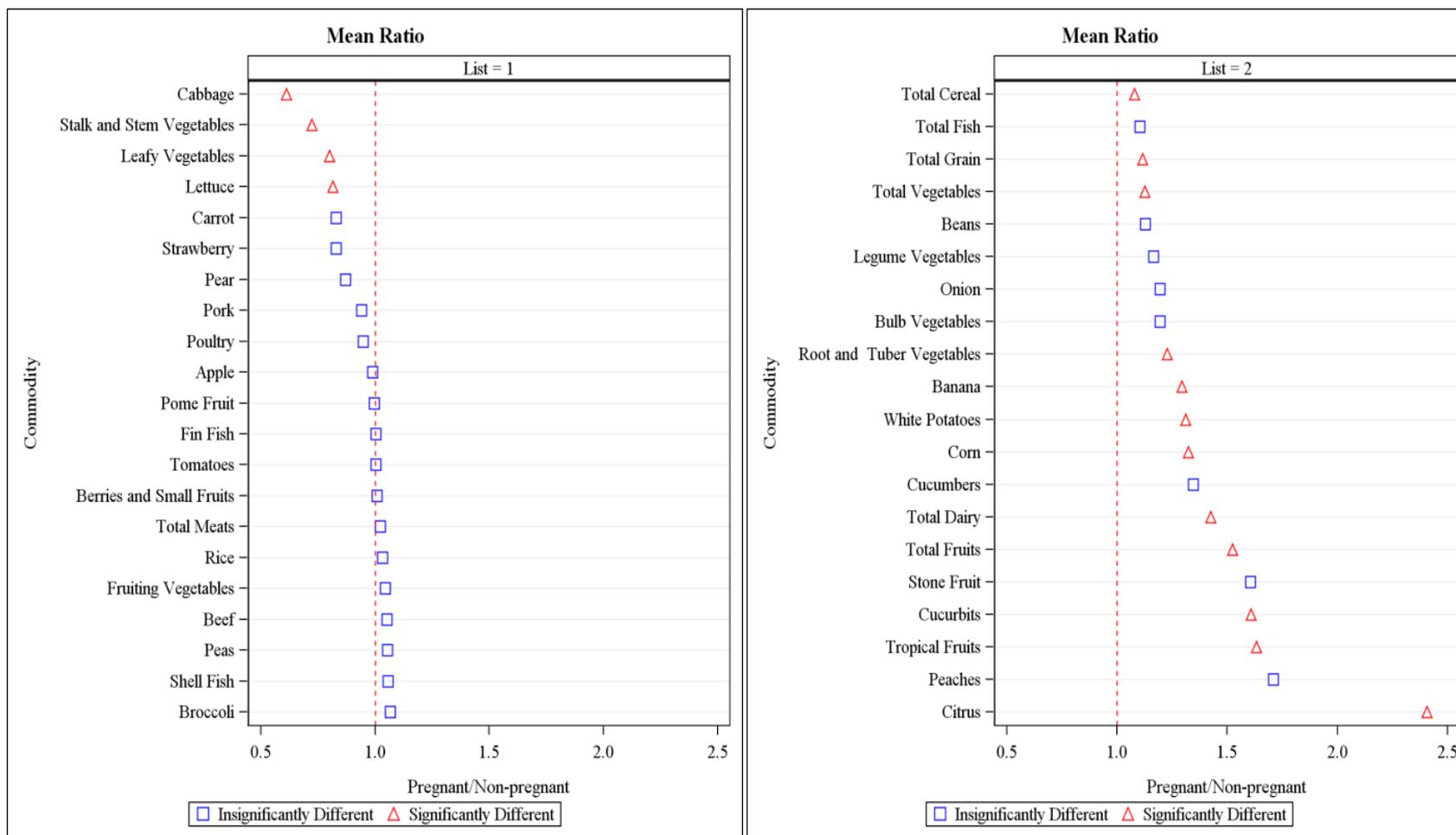


Figure 2: Plot of the ratio of 50th percentile in rank order (Consumers only)

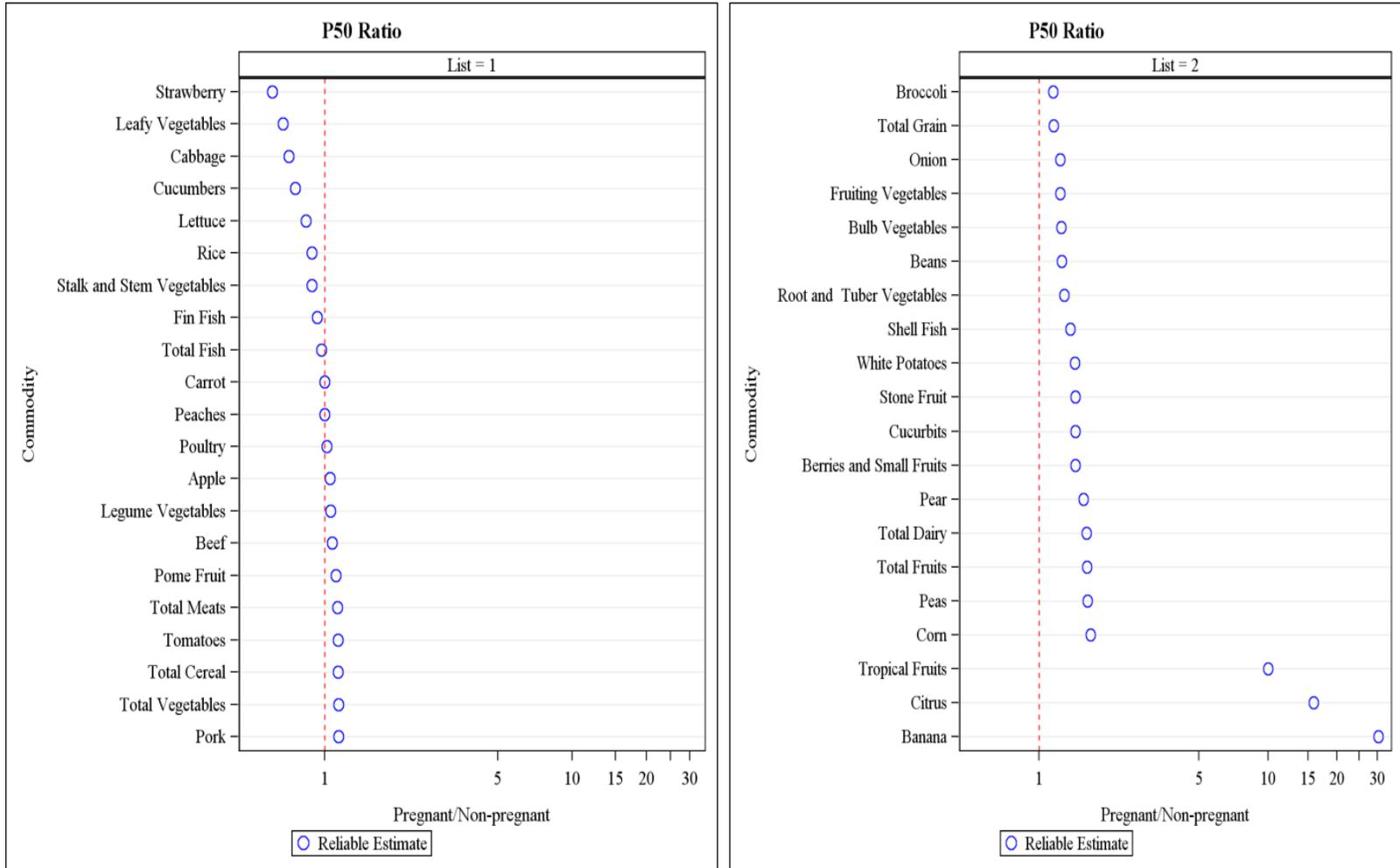


Figure 3: Plot of the ratio of 75th percentile in rank order (Consumers only)

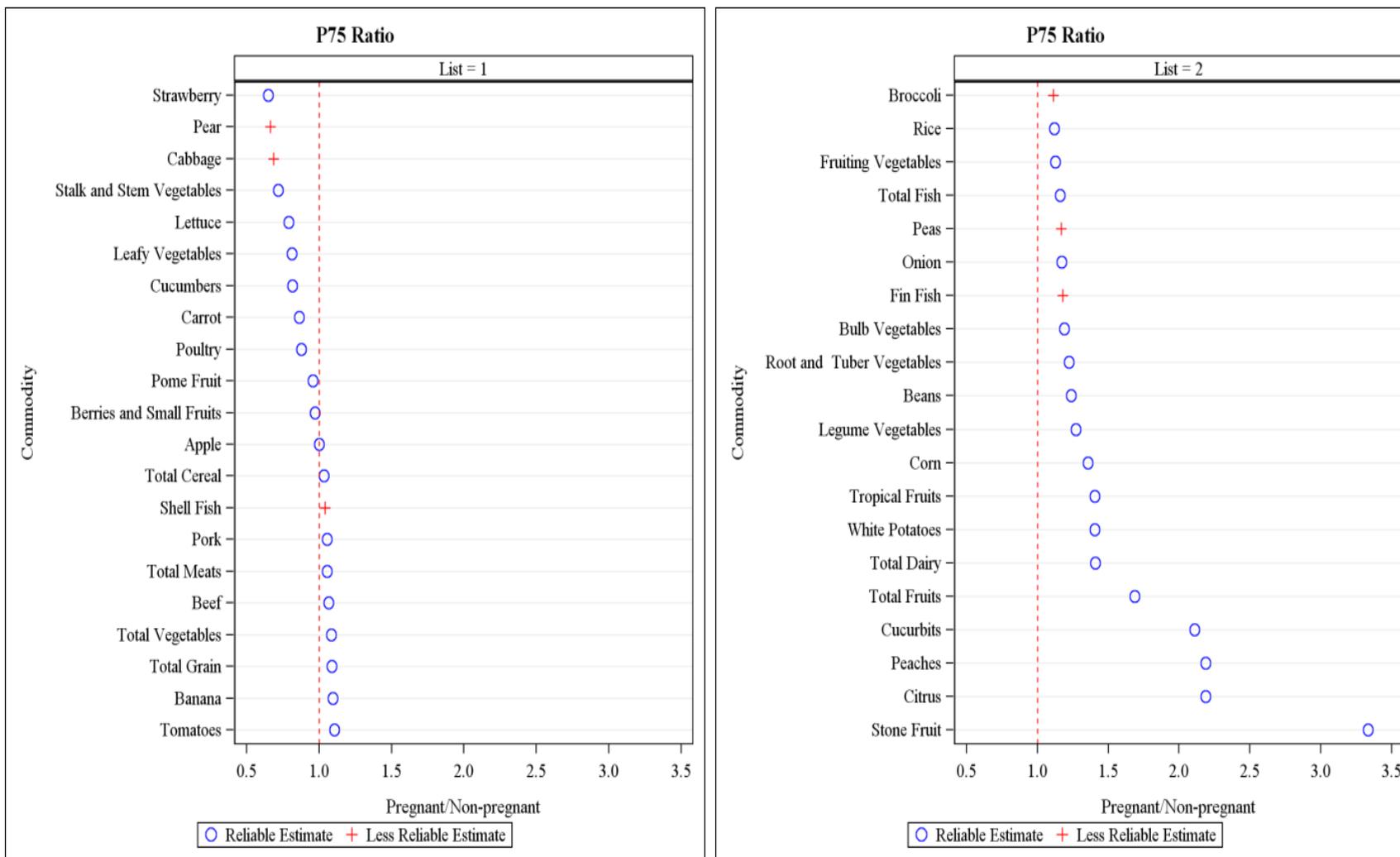


Figure 4: Plot of the ratio of 90th percentile in rank order (Consumers only)

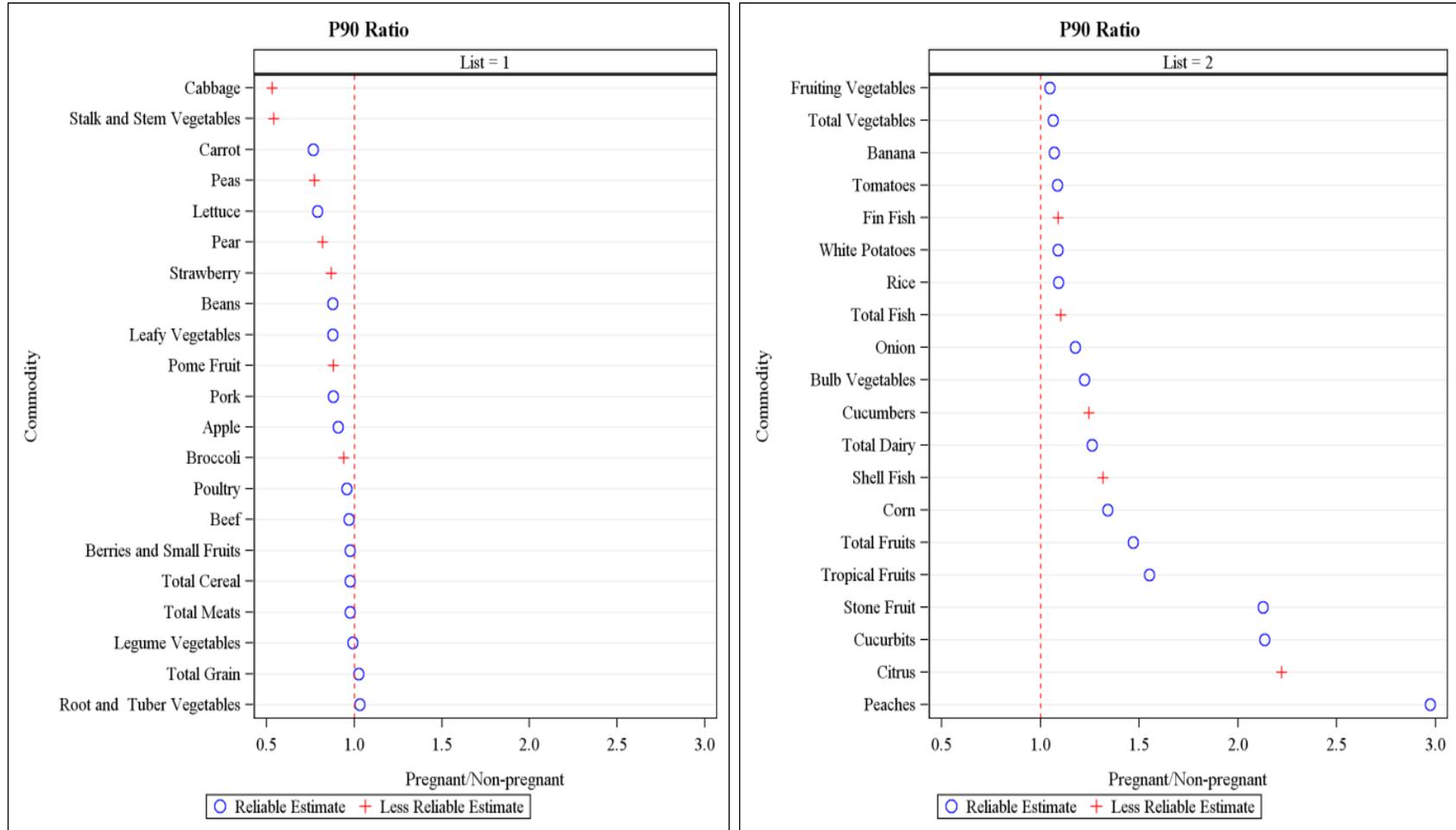
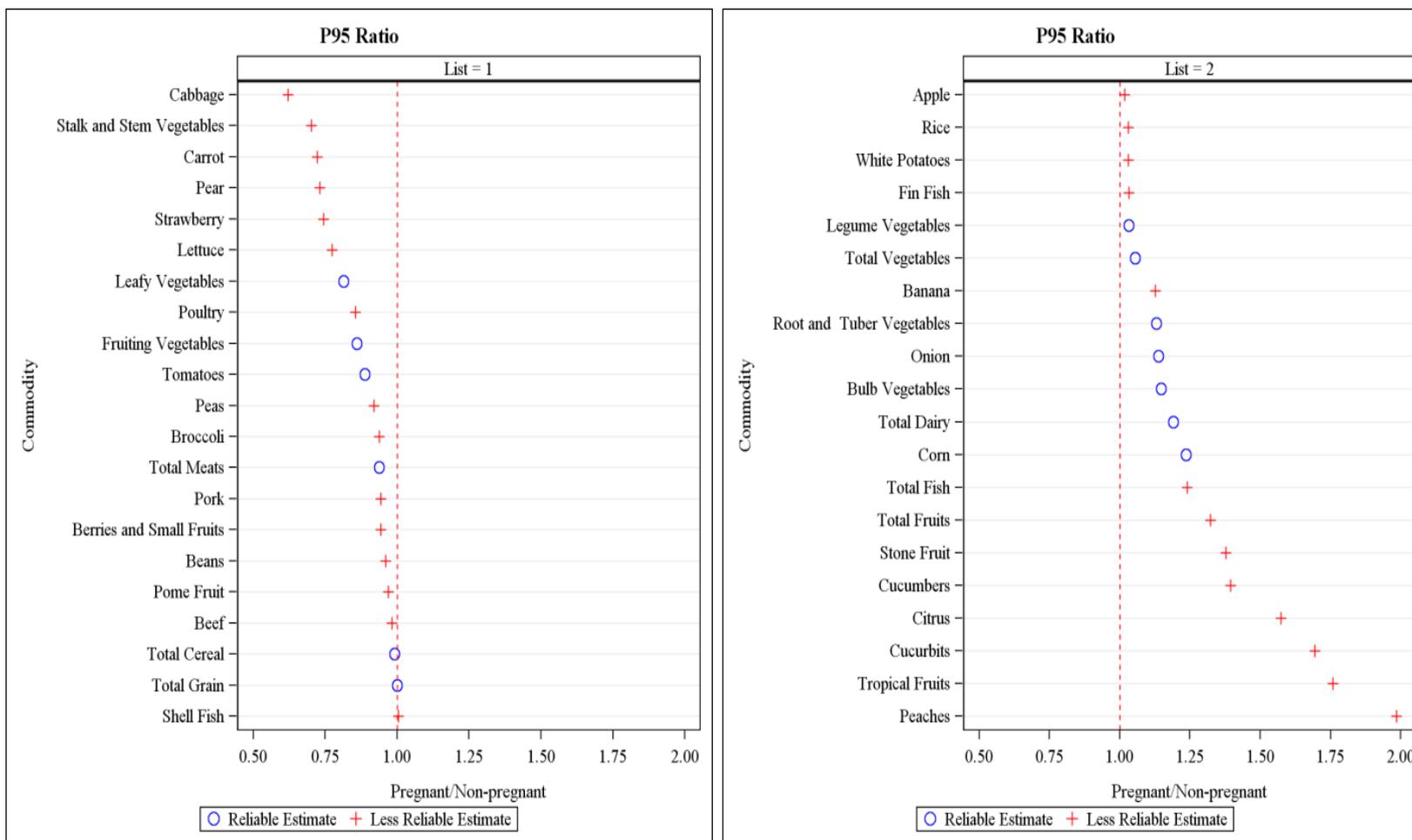


Figure 5: Plot of the ratio of 95th percentile in rank order (Consumers only)



Appendix 2

Plots of ratio of consumption between Pregnant and Non-Pregnant Females (Per Capita)

Notes: Plots of ratios of the 50th percentile for per capita were not provided since the values were zero for several groups of commodities. For the same reason, the ratios for other percentiles were not plotted for some commodities.

Figure 6: Plot of the mean ratio in rank order (per capita)

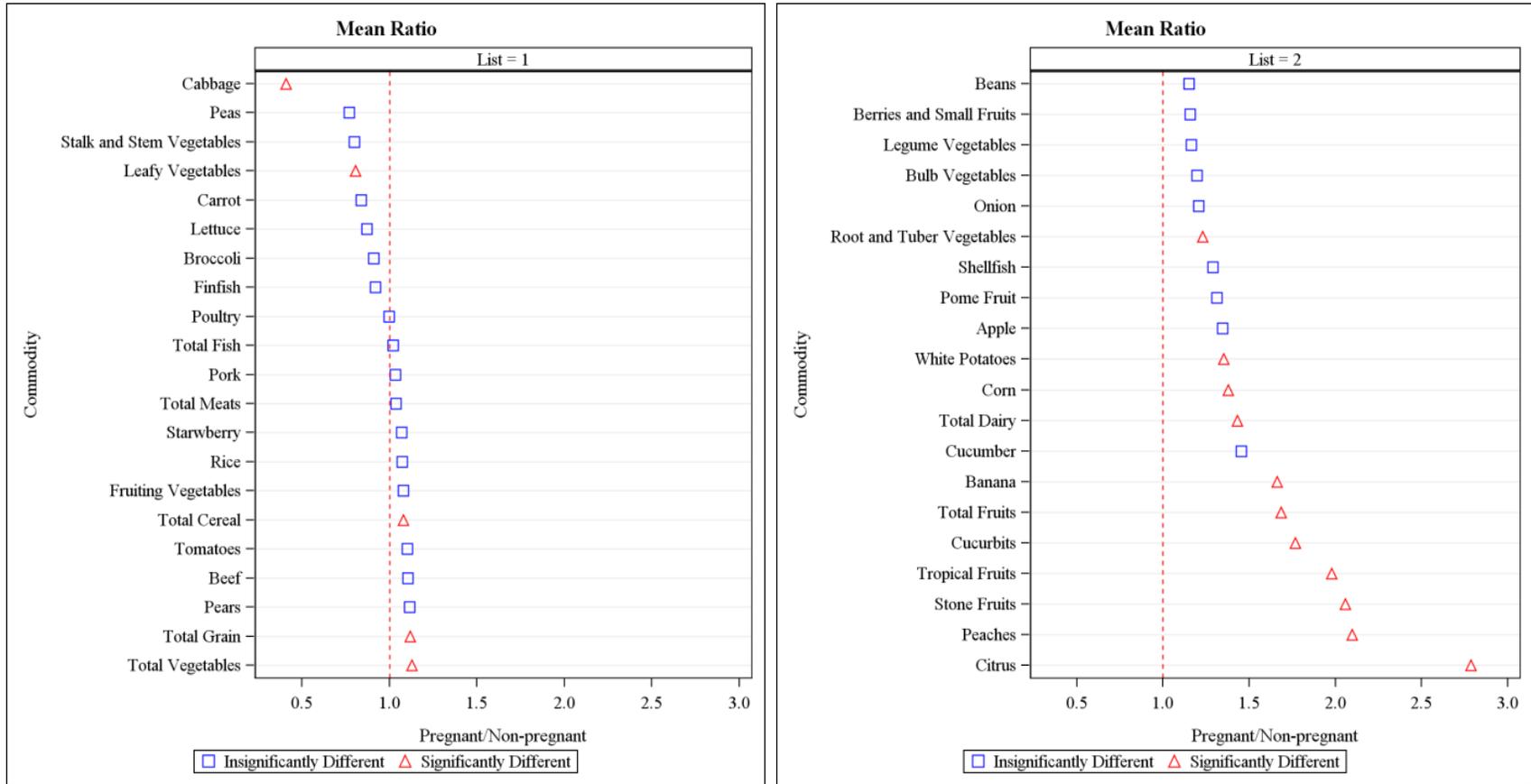


Figure 7: Plot of the ratio of 75th percentile in rank order (per capita)

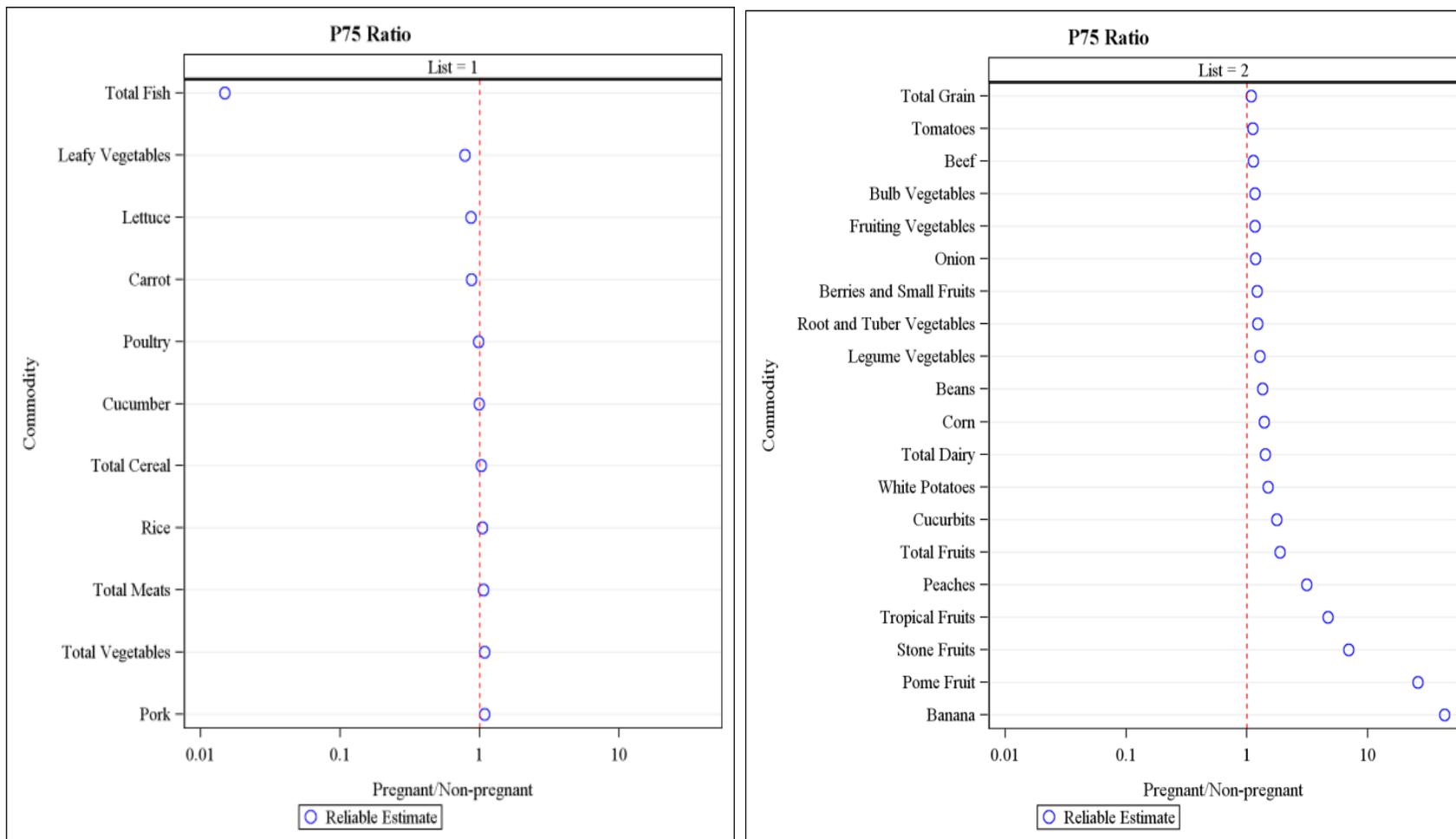


Figure 8: Plot of the ratio of 90th percentile in rank order (per capita)

81-V

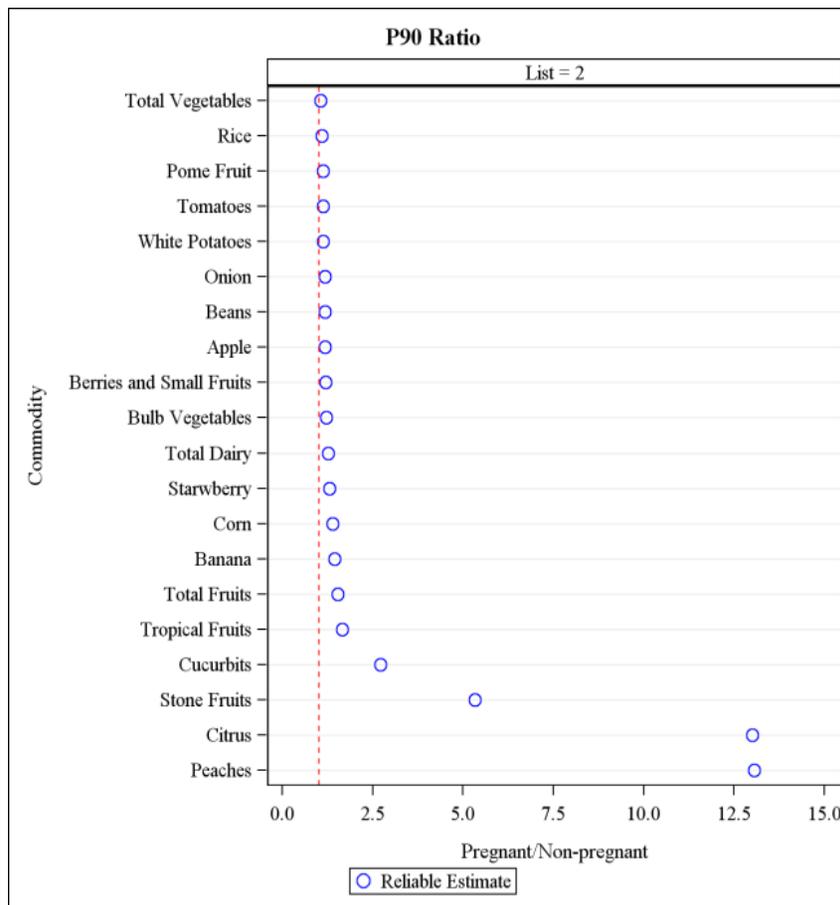
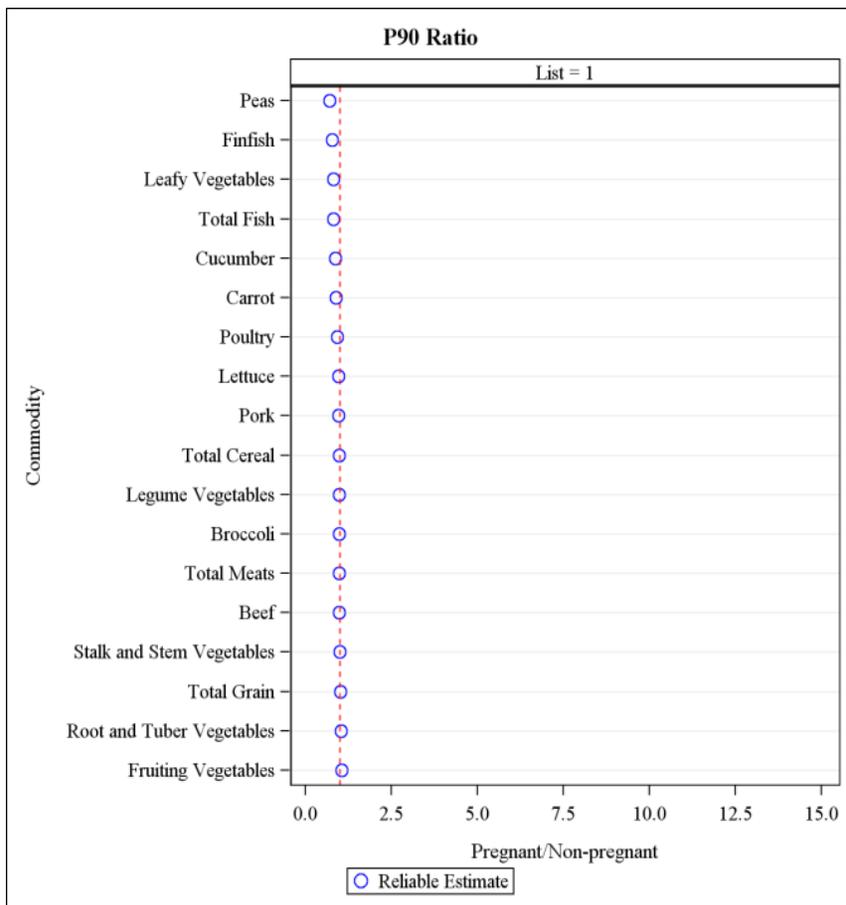
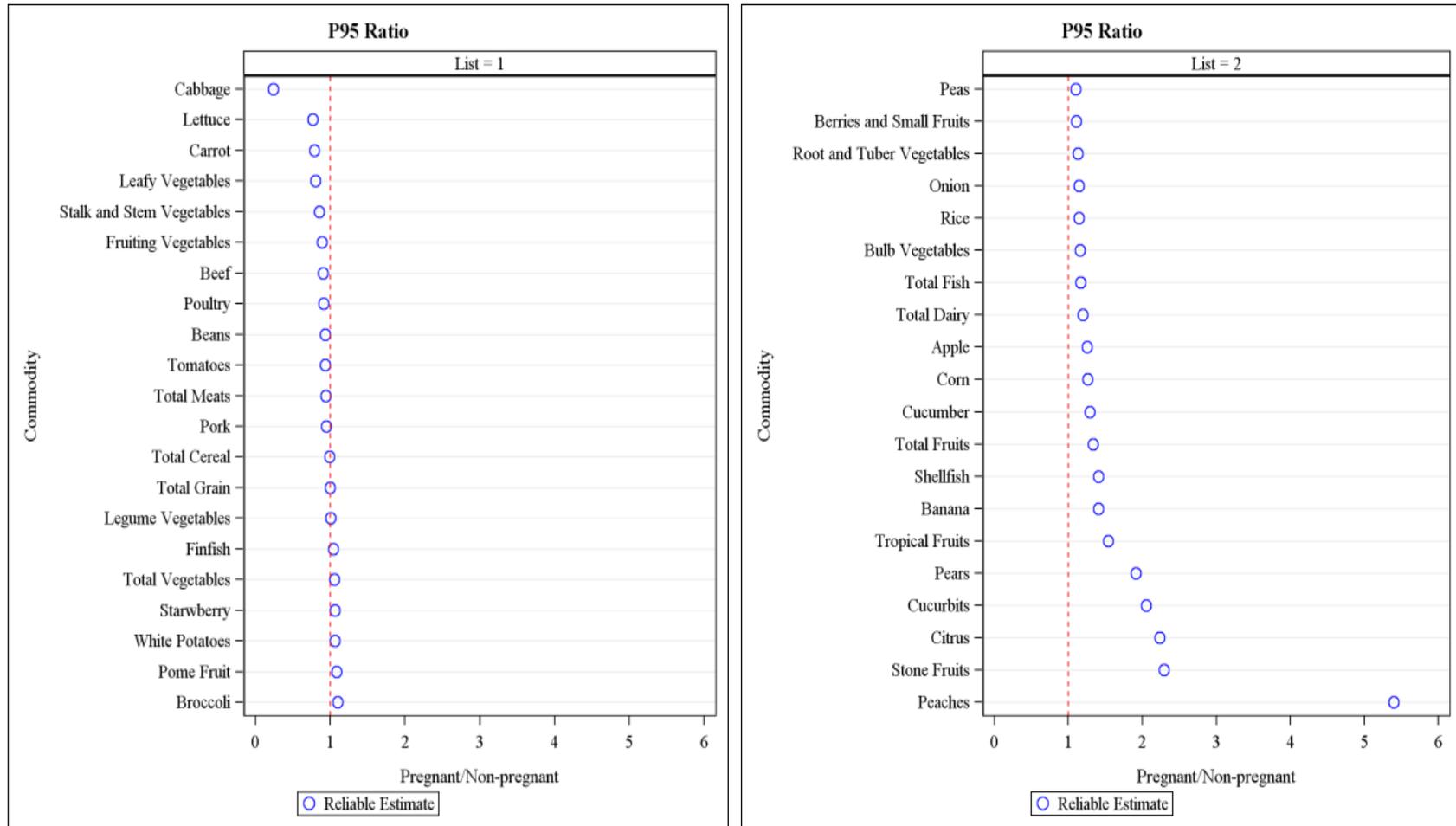


Figure 9: Plot of the ratio of the 95th percentile in rank order (per capita)



Appendix 3: Tables of consumption

Table 1: Table of Consumption for Consumers Only (g/kg-day)

Commodity	Pregnancy ^a	N	Mean	SE	max	P99	p95	p90	p75	p50	p25	p10	p5	p1	min
Total Vegetables	PREGNANT	612	2.7409*	0.1362	18.3032†	9.5662†	6.2623	4.9957	3.5249	2.3098	1.4698	0.9457	0.4169	0.2254†	0.0144†
	NOT-PREGNANT	4318	2.4342	0.0633	17.0561†	8.7948	5.9334	4.6928	3.2537	2.034	1.1609	0.6292	0.3716	0.0524	0.0000†
Total Fruits	PREGNANT	558	1.7900*	0.1424	11.0089†	8.1086†	5.3692†	4.6415	2.818	1.0951	0.1481	0.0057	0.0002†	0.0000†	0.0000†
	NOT-PREGNANT	3640	1.1751	0.0482	16.6742†	6.4516	4.0614	3.1614	1.67	0.6769	0.0873	0.0039	0.0001	0.0000	0.0000†
Apple	PREGNANT	245	0.8555	0.1174	3.6472†	3.1992†	2.7473†	2.0214	1.3295	0.7267	0.0669	0.0000	0.0000†	0.0000†	0.0000†
	NOT-PREGNANT	1181	0.8652	0.0471	6.9416†	3.6275†	2.7035	2.2258	1.3295	0.6922	0.0405	0.0000	0.0000	0.0000†	0.0000†
Banana	PREGNANT	383	0.5289*	0.0554	3.2114†	2.7927 †	1.9699†	1.3603	0.8206	0.4039	0.0006	0.0000	0.0000†	0.0000†	0.0000†
	NOT-PREGNANT	2259	0.4089	0.0243	6.7417†	2.4066	1.7481	1.2702	0.7504	0.0133	0.0000	0.0000	0.0000	0.0000†	0.0000†
Beans	PREGNANT	319	0.4348	0.039	4.9616†	2.2083†	1.1644†	0.7963	0.6541	0.3176	0.1294	0.0387	0.0061†	0.0021†	0.0000†
	NOT-PREGNANT	1964	0.3853	0.0148	4.0313†	2.1228†	1.2136	0.9092	0.5279	0.2523	0.0953	0.019	0.0076	0.0014†	0.0000†
Berries and Small Fruits	PREGNANT	429	0.3309	0.0419	4.6918†	3.4096†	1.2874†	0.9203	0.3649	0.0983	0.0098	0.0001	0.0000†	0.0000†	0.0000†
	NOT-PREGNANT	2821	0.328	0.0177	10.1279†	3.0955	1.3665	0.9441	0.3752	0.0681	0.0106	0.0001	0.0000	0.0000	0.0000†
Broccoli	PREGNANT	95	0.5785	0.0836	3.5508†	3.5508†	1.4480†	1.0540†	0.7997†	0.427	0.2331†	0.0884†	0.0768†	0.0436†	0.0183†
	NOT-PREGNANT	610	0.5422	0.0406	3.6304†	3.2844†	1.5446	1.1239	0.7188	0.3705	0.1766	0.0713	0.038	0.0120†	0.0019†
Bulb Vegetables	PREGNANT	596	0.1988	0.0157	2.0595†	0.9020†	0.6186	0.497	0.273	0.1293	0.0466	0.0154	0.0075	0.0014†	0.0001†
	NOT-PREGNANT	4206	0.1664	0.0056	2.7933†	0.9763	0.5396	0.4062	0.2294	0.1032	0.0313	0.0071	0.0017	0.0002	0.0000†
Cabbage	PREGNANT	79	0.2362*	0.0419	2.4063†	2.4063†	0.8008†	0.5143†	0.3068†	0.1323	0.0557†	0.0214†	0.0133†	0.0053†	0.0007†
	NOT-PREGNANT	497	0.3867	0.0396	3.9186†	3.4557†	1.2931	0.9659	0.4480	0.1848	0.0318	0.0122	0.0055	0.0027†	0.0002†
Carrot	PREGNANT	292	0.2066	0.0213	2.2291†	1.1210†	0.6540†	0.4855	0.2931	0.1277	0.0280	0.0104	0.0086†	0.0004†	0.0000†
	NOT-PREGNANT	1850	0.2493	0.0123	4.2628†	1.4977†	0.9064	0.635	0.3398	0.1279	0.0374	0.0134	0.0054	0.0001†	0.0000†
Citrus	PREGNANT	156	1.2084*	0.1988	5.4783†	4.8469†	3.2278†	3.1720†	1.7250	0.9052	0.3252	0.0000†	0.0000†	0.0000†	0.0000†

Commodity	Pregnancy ^a	N	Mean	SE	max	P99	p95	p90	p75	p50	p25	p10	p5	p1	min
	NOT-PREGNANT	877	0.5025	0.0474	13.4653†	3.2821†	2.0508	1.4286	0.7882	0.0572	0.0000	0.0000	0.0000	0.0000†	0.0000†
Corn	PREGNANT	604	0.4228*	0.0293	4.2018†	1.9473†	1.4241	1.1129	0.5854	0.2783	0.0543	0.0069	0.001	0.0001†	0.0000†
	NOT-PREGNANT	4157	0.3194	0.011	4.5603†	1.9947	1.1525	0.8296	0.432	0.1656	0.0342	0.0054	0.0018	0.0001	0.0000†
Cucumbers	PREGNANT	249	0.2944	0.0964	3.8437†	3.8437†	1.1579†	0.7107†	0.2147	0.0729	0.0402	0.0224†	0.0020†	0.0006†	0.0000†
	NOT-PREGNANT	1679	0.2187	0.0128	3.4979†	1.8698†	0.8307	0.5708	0.2627	0.096	0.0306	0.0078	0.0016	0.0000†	0.0000†
Cucurbits	PREGNANT	309	0.8714*	0.1366	14.6581†	5.2672†	3.8437†	2.9325	0.9472	0.2019	0.053	0.0276	0.0105†	0.0011†	0.0000†
	NOT-PREGNANT	1990	0.5417	0.0667	14.1597†	8.1859	2.2687	1.3743	0.4491	0.1401	0.0455	0.0123	0.0034	0.0000	0.0000†
Fruiting Vegetables	PREGNANT	601	0.778	0.0424	7.0745†	2.9737†	2.0795	1.7812	1.1326	0.6068	0.2611	0.0457	0.0061	0.0003†	0.0000†
	NOT-PREGNANT	4134	0.7454	0.0236	12.3983†	3.857	2.4173	1.6995	1.006	0.4893	0.1987	0.0328	0.0015	0.0000	0.0000†
Leafy Vegetables	PREGNANT	576	0.4900*	0.0334	4.5369†	2.8725†	1.637	1.3271	0.7105	0.2255	0.0949	0.0276	0.0011	0.0000†	0.0000†
	NOT-PREGNANT	3978	0.6133	0.0252	8.7540†	4.209	2.0113	1.5144	0.8758	0.3322	0.0844	0.0022	0.0005	0.0001	0.0000†
Legume Vegetables	PREGNANT	597	0.4019	0.0781	7.2348†	5.0871†	1.4662	0.8981	0.5025	0.0995	0.0057	0.002	0.0007	0.0002†	0.0000†
	NOT-PREGNANT	4100	0.3451	0.0178	12.6565†	3.2299	1.42	0.9059	0.3949	0.0943	0.0059	0.0015	0.0006	0.0001	0.0000†
Lettuce	PREGNANT	381	0.3715*	0.0266	3.4258†	2.1842†	1.1383†	0.8173	0.4731	0.2197	0.1133	0.0535	0.0500†	0.0317†	0.0004†
	NOT-PREGNANT	2492	0.4559	0.0192	4.8880†	3.1643	1.4736	1.0351	0.5986	0.2612	0.1094	0.0586	0.0432	0.0163	0.0002†
Onion	PREGNANT	595	0.1916	0.0157	2.0222†	0.8775†	0.5947	0.4595	0.262	0.1215	0.0434	0.0134	0.0055	0.0012†	0.0001†
	NOT-PREGNANT	4175	0.1604	0.0055	2.7597†	0.9458	0.523	0.3903	0.2239	0.0983	0.0271	0.005	0.0012	0.0001	0.0000†
Peaches	PREGNANT	317	0.1649	0.0403	2.4715†	1.3779†	1.1774†	0.6737	0.0328	0.0018	0.0000	0.0000	0.0000†	0.0000†	0.0000†
	NOT-PREGNANT	2034	0.0965	0.0108	7.3052†	1.6141	0.5932	0.2266	0.015	0.0018	0.0000	0.0000	0.0000	0.0000	0.0000†
Pear	PREGNANT	56	0.5241	0.1086	3.2842†	1.9690†	1.7184†	1.3259†	0.6036†	0.368	0.1464†	0.1166†	0.0597†	0.0020†	0.0020†
	NOT-PREGNANT	318	0.6024	0.0693	3.3764†	3.3764†	2.3513†	1.6176	0.9109	0.2354	0.1061	0.0405	0.0282†	0.0020†	0.0015†
Peas	PREGNANT	101	0.2915	0.025	1.5291†	0.8559†	0.7997†	0.5298†	0.4135†	0.2659	0.1028†	0.0506†	0.0338†	0.0145†	0.0096†
	NOT-PREGNANT	743	0.2768	0.0207	9.8985†	1.5275†	0.8697	0.6855	0.3537	0.1636	0.0703	0.0335	0.0187	0.0082†	0.0007†
Pome Fruit	PREGNANT	265	0.9136	0.1084	3.9823†	3.2779†	2.7473†	2.0214†	1.3295	0.7496	0.1310	0.0055†	0.0000†	0.0000†	0.0000†
	NOT-PREGNANT	1354	0.9177	0.0425	6.9416†	3.9486†	2.8361	2.2962	1.3911	0.6751	0.0931	0.0001	0.0000	0.0000†	0.0000†

Commodity	Pregnancy ^a	N	Mean	SE	max	P99	p95	p90	p75	p50	p25	p10	p5	p1	min
Root and Tuber Vegetables	PREGNANT	612	1.0331*	0.0747	6.0423†	4.2019†	2.6676	1.9222	1.4077	0.7915	0.4405	0.1804	0.0893	0.0267†	0.0017†
	NOT-PREGNANT	4315	0.8416	0.0192	7.3177†	3.6881	2.3595	1.8614	1.1519	0.6144	0.2829	0.1249	0.0712	0.0136	0.0000†
Stalk and Stem Vegetables	PREGNANT	131	0.1541*	0.025	1.2545†	0.8364†	0.5211†	0.2939†	0.1737	0.0961	0.0458	0.0238†	0.0108†	0.0062†	0.0052†
	NOT-PREGNANT	764	0.213	0.0146	4.1523†	1.2383†	0.7425	0.5431	0.2417	0.1082	0.0391	0.0197	0.0132	0.0009†	0.0001†
Stone Fruit	PREGNANT	340	0.2749	0.0585	3.3603†	2.2333†	1.3290†	1.2172	0.2702	0.0082	0.0001	0.0000	0.0000†	0.0000†	0.0000†
	NOT-PREGNANT	2137	0.1712	0.017	7.3052†	2.1733	0.9654	0.5724	0.0811	0.0057	0.0000	0.0000	0.0000	0.0000	0.0000†
Strawberry	PREGNANT	261	0.1761	0.0353	2.5521†	2.1447†	0.8059†	0.6344†	0.1343	0.0078	0.0000	0.0000†	0.0000†	0.0000†	0.0000†
	NOT-PREGNANT	1532	0.2122	0.0245	5.8368†	2.2975†	1.085	0.7302	0.2074	0.0127	0.0000	0.0000	0.0000	0.0000†	0.0000†
Tomatoes	PREGNANT	578	0.7252	0.0409	4.9361†	2.5610†	1.926	1.6511	1.0677	0.5503	0.2484	0.0786	0.0068	0.0039†	0.0012†
	NOT-PREGNANT	3830	0.7226	0.0221	8.0503†	3.7422	2.1705	1.5201	0.9641	0.487	0.2360	0.0869	0.0339	0.0063	0.0002†
Tropical Fruits	PREGNANT	447	0.7283*	0.0817	5.2499†	3.6671†	3.2607†	2.1907	1.0625	0.4187	0.0006	0.0000	0.0000†	0.0000†	0.0000†
	NOT-PREGNANT	2700	0.4465	0.0233	10.2408†	2.7993	1.8544	1.4115	0.7564	0.0417	0.0000	0.0000	0.0000	0.0000	0.0000†
White Potatoes	PREGNANT	563	0.6993*	0.0697	5.5088†	3.2416†	2.0394†	1.5605	1.0837	0.4179	0.1491	0.0000	0.0000†	0.0000†	0.0000†
	NOT-PREGNANT	3868	0.5335	0.0194	6.8670†	3.1915	1.9811	1.4324	0.7708	0.2917	0.0078	0.0000	0.0000	0.0000	0.0000†
Total Fish	PREGNANT	153	0.7126	0.1172	5.3152†	3.7920†	2.3960†	1.5882†	0.9964	0.413	0.1826	0.0210†	0.0005†	0.0000†	0.0000†
	NOT-PREGNANT	1204	0.6456	0.0357	8.6423†	3.4532†	1.9331	1.4405	0.858	0.4265	0.1796	0.0012	0.0003	0.0000†	0.0000†
Fin Fish	PREGNANT	108	0.5895	0.1005	3.4282†	3.4282†	1.8013†	1.3664†	0.9390†	0.373	0.1677†	0.0007†	0.0002†	0.0000†	0.0000†
	NOT-PREGNANT	882	0.5877	0.0328	8.4828†	2.8633†	1.7459	1.2552	0.7956	0.4013	0.1403	0.0005	0.0001	0.0000†	0.0000†
Shell Fish	PREGNANT	75	0.5098	0.0909	2.6357†	2.6357†	1.7920†	1.4464†	0.6000†	0.4302	0.1305†	0.0113†	0.0000†	0.0000†	0.0000†
	NOT-PREGNANT	492	0.4827	0.0525	5.3172†	2.9026†	1.7853	1.0997	0.5776	0.3142	0.0988	0.0110	0.0000	0.0000†	0.0000†
Rice	PREGNANT	555	0.239	0.0383	2.4799†	1.6971†	0.9795†	0.7084	0.3377	0.0547	0.0002	0.0000	0.0000†	0.0000†	0.0000†
	NOT-PREGNANT	3690	0.2318	0.0143	5.8933†	1.9847	0.9516	0.6498	0.3018	0.0617	0.0001	0.0000	0.0000	0.0000	0.0000†
Total Grain	PREGNANT	612	2.1215*	0.0675	7.7600†	4.8763†	3.9381	3.4405	2.6515	1.9386	1.3781	1.0295	0.7923	0.5704†	0.2139†
	NOT-PREGNANT	4318	1.901	0.0353	9.7936†	5.6565	3.9351	3.3588	2.4417	1.6773	1.1244	0.7499	0.5308	0.2034	0.0003†

Commodity	Pregnancy ^a	N	Mean	SE	max	P99	p95	p90	p75	p50	p25	p10	p5	p1	min
Total Cereal	PREGNANT	612	3.0212*	0.0807	10.5650†	7.2569†	5.7144	4.7326	3.7025	2.8268	2.0819	1.5491	1.2502	0.8298†	0.5073†
	NOT-PREGNANT	4320	2.8005	0.0375	13.2925†	8.0296	5.7685	4.8472	3.5823	2.4992	1.6946	1.1404	0.8797	0.4203	0.0040†
Total Meats	PREGNANT	607	1.5989	0.053	5.9123†	4.5395†	3.3366	2.943	2.1465	1.4788	0.9328	0.5141	0.3205	0.0279†	0.0040†
	NOT-PREGNANT	4259	1.563	0.0321	12.2299†	4.9672	3.5589	3.0123	2.0343	1.3157	0.8184	0.4329	0.2532	0.0073	0.0000†
Beef	PREGNANT	540	0.707	0.0448	4.7364†	3.2504†	2.0882†	1.5265	0.9658	0.5091	0.2225	0.0459	0.0058†	0.0005†	0.0000†
	NOT-PREGNANT	3744	0.6721	0.0209	8.8360†	3.2354	2.1274	1.5758	0.905	0.4755	0.1502	0.0128	0.0017	0.0001	0.0000†
Poultry	PREGNANT	488	0.8237	0.056	4.4038†	3.4986†	2.0535†	1.7574	1.0499	0.6921	0.3300	0.1395	0.0287†	0.0000†	0.0000†
	NOT-PREGNANT	3414	0.8688	0.0259	12.2299†	3.5647	2.4044	1.8348	1.198	0.6778	0.3186	0.0964	0.0188	0.0000	0.0000†
Pork	PREGNANT	529	0.3335	0.0245	3.3536†	1.9637†	1.1193†	0.7814	0.4928	0.2015	0.0599	0.0126	0.0023†	0.0000†	0.0000†
	NOT-PREGNANT	3397	0.3549	0.0133	5.0484†	2.3334	1.1883	0.8873	0.4678	0.1772	0.0657	0.0111	0.0025	0.0001	0.0000†
Total Dairy	PREGNANT	612	5.0444*	0.2752	52.6777†	22.5236†	12.5184	10.0453	6.9105	3.8797	2.2297	0.8209	0.4249	0.0900†	0.0014†
	NOT-PREGNANT	4310	3.5385	0.1241	52.0738†	17.1615	10.5089	7.9718	4.9051	2.4097	1.0336	0.4418	0.2213	0.0305	0.0005†

^a Limited to females aged 13-49 in both PREGNANT and NON-PREGNANT categories

Notes: ‘*’ indicates mean of pregnant female is statistically significantly different from the nonpregnant female; alpha=0.05 level. Significant differences were NOT evaluated for percentiles values.

‘†’ indicates estimates are less statistically reliable based on $np < 8$ * ‘Design Effect’ guidance published in the *Joint Policy on Variance Estimation and Statistical Reporting Standards on NHANES III and CSFII*

Table 2: Table of Consumption Per Capita (g/kg-day)

Commodity	Pregnancy ^a	N	Mean	SE	Percent	max	P99	p95	p90	p75	p50	p25	p10	p5	p1	min
Total Vegetables	PREGNANT	612	2.7409*	0.1362	100	18.3032†	9.5662†	6.2623	4.9957	3.5249	2.3098	1.4698	0.9457	0.4169	0.2254†	0.0144†
	NOT-PREGNANT	4321	2.4304	0.0633	99.8448	17.0561†	8.7948	5.9334	4.6917	3.249	2.0329	1.1555	0.6226	0.3618	0.0454	0.0000†
Total Fruits	PREGNANT	612	1.6553*	0.13	92.4767	11.0089†	8.1086†	5.0151	4.3416	2.6476	0.9664	0.0677	--	--	--†	--†
	NOT-PREGNANT	4321	0.9821	0.0434	83.5701	16.6742†	6.1871	3.7519	2.8349	1.4133	0.3885	0.0041	--	--	--	--†
Apple	PREGNANT	612	0.3272	0.0527	38.2497	3.6472†	2.8438†	2.0214	1.25	0.2119	--	--	--	--	--†	--†
	NOT-PREGNANT	4321	0.2432	0.017	28.11	6.9416†	2.8369	1.6103	1.0502	--	--	--	--	--	--	--†
Banana	PREGNANT	612	0.3351*	0.041	63.3702	3.2114†	2.2835†	1.7832	1.2265	0.5351	--	--	--	--	--†	--†
	NOT-PREGNANT	4321	0.2018	0.0114	49.3507	6.7417†	2.2139	1.2661	0.846	0.0124	--	--	--	--	--	--†
Beans	PREGNANT	612	0.2024	0.021	46.5518	4.9616†	1.5977†	0.7963	0.7066	0.2864	--	--	--	--	--†	--†
	NOT-PREGNANT	4321	0.1759	0.0089	45.6579	4.0313†	1.6446	0.8526	0.5962	0.2132	--	--	--	--	--	--†
Berries and Small Fruits	PREGNANT	612	0.2497	0.0273	75.4711	4.6918†	2.6544†	1.2459	0.7939	0.2015	0.0338	--	--	--	--†	--†
	NOT-PREGNANT	4321	0.2155	0.0122	65.7116	10.1279†	2.6247	1.128	0.6634	0.1664	0.0090	--	--	--	--	--†
Broccoli	PREGNANT	612	0.0843	0.0152	14.5665	3.5508†	1.1927†	0.7369	0.2817	--	--	--	--	--	--†	--†
	NOT-PREGNANT	4321	0.0926	0.0088	17.0872	3.6304†	1.4667	0.6719	0.2857	--	--	--	--	--	--	--†
Bulb Vegetables	PREGNANT	612	0.1939	0.0155	97.544	2.0595†	0.9020†	0.6164	0.4936	0.2632	0.1266	0.0422	0.0104	0.0036	--†	--†
	NOT-PREGNANT	4321	0.1619	0.0054	97.3381	2.7933†	0.963	0.5334	0.4039	0.2252	0.0989	0.0270	0.0039	0.0006	--	--†
Cabbage	PREGNANT	612	0.0190*	0.0038	8.0475	2.4063†	0.4209†	0.0626	0	--	--	--	--	--	--†	--†
	NOT-PREGNANT	4321	0.0465	0.0056	12.0242	3.9186†	1.0585	0.2591	0.0173	--	--	--	--	--	--	--†
Carrot	PREGNANT	612	0.0952	0.0114	46.0861	2.2291†	1.0661†	0.4615	0.3508	0.0920	--	--	--	--	--†	--†
	NOT-PREGNANT	4321	0.1139	0.0073	45.707	4.2628†	1.1775	0.5867	0.3937	0.1050	--	--	--	--	--	--†
Citrus	PREGNANT	612	0.2868 *	0.0548	23.7326	5.4783†	3.2278†	1.7983	0.9417	--	--	--	--	--	--†	--†
	NOT-PREGNANT	4321	0.103	0.0125	20.5039	13.4653†	2.1311	0.8047	0.0724	--	--	--	--	--	--	--†
Corn	PREGNANT	612	0.4212*	0.0291	99.6278	4.2018†	1.9405†	1.4241	1.1129	0.5826	0.2783	0.0543	0.0063	0.001	--†	--†
	NOT-PREGNANT	4321	0.306	0.011	95.8244	4.5603†	1.9473	1.1319	0.8026	0.4207	0.1507	0.0265	0.0023	--	--	--†

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Commodity	Pregnancy ^a	N	Mean	SE	Percent	max	P99	p95	p90	p75	p50	p25	p10	p5	p1	min
	NOT-PREGNANT	4321	0.0453	0.004	21.2878	4.1523†	0.7754	0.2641	0.1186	--	--	--	--	--	--	--†
Stone Fruits	PREGNANT	612	0.1660*	0.0371	60.3981	3.3603†	2.2333†	1.2221	0.6737	0.0223	--	--	--	--	--†	--†
	NOT-PREGNANT	4321	0.0807	0.0088	47.1751	7.3052†	1.5078	0.5324	0.1266	0.0032	--	--	--	--	--	--†
Strawberry	PREGNANT	612	0.0866	0.0161	49.1941	2.5521†	1.7978†	0.6344	0.2448	0.0068	--	--	--	--	--†	--†
	NOT-PREGNANT	4321	0.0811	0.0102	38.2386	5.8368†	1.3393	0.5974	0.1869	--	--	--	--	--	--	--†
Tomatoes	PREGNANT	612	0.6961	0.038	95.9938	4.9361†	2.5387†	1.882	1.6297	0.9937	0.4975	0.2159	0.0232	0.0039	--†	--†
	NOT-PREGNANT	4321	0.6322	0.0206	87.4921	8.0503†	3.5407	2.0145	1.4488	0.8865	0.4136	0.1306	--	--	--	--†
Tropical Fruits	PREGNANT	612	0.5242*	0.0707	71.9747	5.2499†	3.4876†	2.3463	1.6738	0.7996	0.0018	--	--	--	--†	--†
	NOT-PREGNANT	4321	0.2651	0.015	59.3817	10.2408†	2.4093	1.5285	1.0067	0.1701	--	--	--	--	--	--†
White Potatoes	PREGNANT	612	0.6500*	0.0687	92.9504	5.5088†	3.2416†	2.0394	1.5305	1.0251	0.3950	0.0578	--	--	--†	--†
	NOT-PREGNANT	4321	0.4811	0.019	90.1768	6.8670†	3.0696	1.9173	1.3505	0.6881	0.2072	0.0002	--	--	--	--†
Total Fish	PREGNANT	612	0.1892	0.0335	26.5478	5.3152†	2.3960†	1.2774	0.5529	0.0005	--	--	--	--	--†	--†
	NOT-PREGNANT	4321	0.1855	0.014	28.7341	8.6423†	2.2373	1.0997	0.678	0.0336	--	--	--	--	--	--†
Finfish	PREGNANT	612	0.1234	0.0238	20.931	3.4282†	1.8013†	0.939	0.373	--	--	--	--	--	--†	--†
	NOT-PREGNANT	4321	0.1344	0.0113	22.8696	8.4828†	1.8532	0.9024	0.4803	--	--	--	--	--	--	--†
Shellfish	PREGNANT	612	0.0658	0.0153	12.9067	2.6357†	1.4464†	0.4763	0.0807	--	--	--	--	--	--†	--†
	NOT-PREGNANT	4321	0.0511	0.0066	10.5888	5.3172†	1.0997	0.339	--	--	--	--	--	--	--	--†
Rice	PREGNANT	612	0.2153	0.0371	90.0888	2.4799†	1.6971†	0.9795	0.66	0.2718	0.0268	--	--	--	--†	--†
	NOT-PREGNANT	4321	0.2009	0.0131	86.6753	5.8933†	1.8148	0.8548	0.6023	0.2592	0.0095	--	--	--	--	--†
Total Grain	PREGNANT	612	2.1215*	0.0675	100	7.7600†	4.8763†	3.9381	3.4405	2.6515	1.9386	1.3781	1.0295	0.7923	0.5704†	0.2139†
	NOT-PREGNANT	4321	1.898	0.0355	99.842	9.7936†	5.6565	3.9351	3.3588	2.4413	1.6768	1.1233	0.7472	0.5263	0.1959	--†
Total Cereal	PREGNANT	612	3.0212*	0.0807	100	10.5650†	7.2569†	5.7144	4.7326	3.7025	2.8268	2.0819	1.5491	1.2502	0.8298†	0.5073†
	NOT-PREGNANT	4321	2.7994	0.0375	99.9604	13.2925†	8.0296	5.7685	4.8472	3.5823	2.498	1.6930	1.1404	0.8723	0.4120	--†
Total Meats	PREGNANT	612	1.5884	0.0527	99.3392	5.9123†	4.5395†	3.3366	2.943	2.1465	1.4682	0.8934	0.5047	0.2914	0.0040†	--†
	NOT-PREGNANT	4321	1.5318	0.0306	98.0062	12.2299†	4.9672	3.5359	2.9814	2.0121	1.2954	0.7944	0.3580	0.1438	--	--†

Commodity	Pregnancy ^a	N	Mean	SE	Percent	max	P99	p95	p90	p75	p50	p25	p10	p5	p1	min
Beef	PREGNANT	612	0.6359	0.0455	89.9432	4.7364†	3.2504†	1.7828	1.4426	0.9422	0.4735	0.1266	--	--	--†	--†
	NOT-PREGNANT	4321	0.5758	0.0192	85.676	8.8360†	3.13	1.9776	1.4571	0.8329	0.3664	0.0320	--	--	--	--†
Poultry	PREGNANT	612	0.6661	0.0529	80.8758	4.4038†	3.4986†	1.987	1.5126	0.9774	0.5249	0.0680	--	--	--†	--†
	NOT-PREGNANT	4321	0.6677	0.022	76.8475	12.2299†	3.4113	2.1714	1.635	0.9934	0.4598	0.0027	--	--	--	--†
Pork	PREGNANT	612	0.2851	0.0211	85.4822	3.3536†	1.6992†	1.0094	0.7513	0.3804	0.1709	0.0220	--	--	--†	--†
	NOT-PREGNANT	4321	0.2764	0.0108	77.8779	5.0484†	2.2973	1.0677	0.7863	0.3506	0.107	0.0013	--	--	--	--†
Total Dairy	PREGNANT	612	5.0444*	0.2752	100	52.6777†	22.5236†	12.5184	10.0453	6.9105	3.8797	2.2297	0.8209	0.4249	0.0900†	0.0014†
	NOT-PREGNANT	4321	3.5258	0.1246	99.6417	52.0738†	17.1615	10.4803	7.9399	4.9026	2.407	1.0216	0.4305	0.2122	0.0187	--†

^a Limited to females aged 13-49 in both PREGNANT and NONPREGNANT categories

Notes:

‘*’ indicates mean of pregnant female is statistically significantly different from the nonpregnant female; alpha=0.05 level. Significant differences were NOT evaluated for percentiles values.

‘†’ indicates estimates are less statistically reliable based on $np < 8$ * ‘Design Effect’ guidance published in the *Joint Policy on Variance Estimation and Statistical Reporting Standards on NHANES III and CSFII*

“--” indicates either no reported per capita consumption at this percentile, or per capita consumption is <0.0001 g/kg bw

Appendix 4: SAS codes

```
libname NCEA "F:\NCEA Pregnant women";

PROC IMPORT OUT= Work.FCID_description
            DATATABLE= "FCID_Code_Description"
            DBMS=ACCESS REPLACE;

Database = "G:\WWEIA-FCID\Final Databases\WWEIA FCID 2003-08 (1-17-13).mdb";
SCANMEMO=YES;
USEDATE=NO;
SCANTIME=YES;
RUN;

proc contents data=WORK.FCID_description ;
run;

PROC IMPORT OUT= WORK.demo_0308
            DATATABLE= "WWEIA_Demo_0308"
            DBMS=ACCESS REPLACE;
DATABASE= "G:\WWEIA-FCID\Final Databases\WWEIA FCID 2003-08 (1-17-13).mdb" ;
SCANMEMO=YES;
USEDATE=NO;
SCANTIME=YES;
RUN;

proc contents data=WORK.demo_0308 ;
run;

PROC IMPORT OUT= NCEA.commodity_Intake_0308
            DATATABLE= "Commodity_Intake_0308"
            DBMS=ACCESS REPLACE;
DATABASE= "G:\WWEIA-FCID\Final Databases\WWEIA FCID 2003-08 (1-17-13).mdb" ;
SCANMEMO=YES;
USEDATE=NO;
SCANTIME=YES;
RUN;

proc sort data=WORK.demo_0308;
by seqn;
run;
```

```

libname rp1 xport "F:\NCEA Pregnant women\RHQ_c.xpt";
libname dm1 xport "F:\NCEA Pregnant women\demo_c.xpt";
libname rp2 xport "F:\NCEA Pregnant women\RHQ_d.xpt";
libname dm2 xport "F:\NCEA Pregnant women\demo_d.xpt";
libname rp3 xport "F:\NCEA Pregnant women\RHQ_e.xpt";
libname dm3 xport "F:\NCEA Pregnant women\demo_e.xpt";

options mprint;

data NCEA.data_34;
merge
dm1.demo_c(keep=seqn riagendr ridageyr ridreth1 RIDEXPRG sdmvpsu sdmvstra)
rp1.RHQ_c(keep=seqn RHD143 RHQ200 );
by seqn;
run;

data NCEA.data_56;
merge
dm2.demo_d(keep=seqn riagendr ridageyr ridreth1 RIDEXPRG sdmvpsu sdmvstra)
rp2.RHQ_d(keep=seqn RHD143 RHQ200 );
by seqn;
run;

data NCEA.data_78;
merge
dm3.demo_e(keep=seqn riagendr ridageyr ridreth1 RIDEXPRG sdmvpsu sdmvstra)
rp3.RHQ_e(keep=seqn RHD143 RHQ200);
by seqn;
run;

data demographic1;
set NCEA.data_34 NCEA.data_56 NCEA.data_78;
varunit=sdmvpsu ;
varstrat=sdmvstra;
age=ridageyr;
sex=riagendr;
if RIDEXPRG=1 then preg_f=1;
else if RIDEXPRG=2 then preg_f=0;
run;

PROC SQL;
CREATE TABLE demographic2 AS
SELECT a.*, b.preg_f, b.RHQ200, b.RHD143, b.RIDEXPRG, b.age, b.sex
FROM demo_0308 as a inner join demographic1 as b
ON a.seqn=b.seqn
;

```

```
QUIT;
```

```
proc contents data= demographic2;  
run;
```

```
Proc sql;  
CREATE TABLE FCID_commodity2 AS  
SELECT *  
FROM NCEA.commodity_Intake_0308 as a inner join FCID_description as b  
ON a.FCID_CODE=b.FCID_code  
;  
QUIT;
```

```
proc contents data=FCID_commodity2 ;  
run;
```

```
data NCEA.FCID_commodity2;  
set FCID_commodity2;  
run;
```

```
data NCEA.demographic2;  
set demographic2;  
run;
```

```
+++++ SAS MACRO +++++;
```

```
%MACRO consumption();
```

```
Proc sql;  
CREATE TABLE FCID_group AS  
SELECT unique FCID_desc  
FROM FCID_commodity3  
;  
QUIT;
```

```
ods rtf file="&PATH\FCID_group.rtf";  
title "FCID Group ";  
proc print data= FCID_group;  
run;  
ods rtf close;
```

```

proc sort data=FCID_commodity3;
by seqn daycode;
run;

Proc univariate data =FCID_commodity3 noprint;
var intake_bw;
by seqn daycode;
output out = total_food_day sum = total;
run;

Proc transpose data = total_food_day out = average_total;
var total;
by seqn;
ID daycode;
run;

*proc print data=average_total;
*run;

Data average_total (keep = seqn average);
set average_total;
if _1 = . then _1 = 0;
if _2 = . then _2 = 0;
average = (_1+_2)/2;
run;

*proc print data=average_total;
*run;

proc sort data=demographic2;
by seqn;
run;

*====> Merge individual with the demographic file <====*;

Data total_food;
merge work.demographic2 average_total;
by seqn;
run;

Data total_food;
set total_food;
if average = . then do eater = "none_eater"; con_amt = 0; output;end;
else do eater = "eater"; con_amt = average; output;end;
run;

```

```

                                Data total_food1;
                                set total_food;

                                * preg_f variable is a recoding of ridexprg variable ;
                                *if RIDEXPRG=1 then preg_f=1;
                                *else if RIDEXPRG=2 then preg_f=0;

                                if preg_f=1 and sex=2 and age > 12 and age < 50 then Preg =
                                "P";
                                else if preg_f=0 and sex=2 and age > 12 and age < 50 then
                                Preg = "N" ;

                                preg_female_eater = Preg ;
                                if average = . then preg_female_eater =
                                "non_eater";

                                run;

**** pregnant and non-pregnant female consumption per capita *****;

proc format;
value $ prgy
"P" = " Pregnant 13-49 "
"N" = " Not pregnant 13-49 " ;

run
;

                                proc contents data=total_food ;

                                run;

Proc surveymeans data = total_food1;

                                cluster sdmvpsu;
                                strata sdmvstra / NOCOLLAPSE ;
                                weight WT6_2DAY;
                                Domain preg;
                                var con_amt;
                                *format preg $prgy.;
                                ods output domain = pregnant_female (drop =
                                domainlabel) ;

                                run;

proc print data=pregnant_female ;

                                run;

proc contents data= pregnant_female ;

                                run;

```

```

proc sort data=total_food1;
by preg;
run;

Proc univariate data = total_food1      ;

                                weight WT6_2DAY;
                                var con_amt;

                                output out = pregnant_female1      min=min
p1=p1 p5=p5 p10=p10 p25=p25 p50=p50 p75=p75 p90=p90 p95=p95 p99=p99 max=max ;
                                by preg;

                                run;

data pregnant_female2      ;
set pregnant_female1 ;
if _N_ = 1 then delete;

run;

data pregnant_female;
set pregnant_female;
if preg ="P" then order =1;
else order=2;
run;

data pregnant_female2;
set pregnant_female2;
if preg ="P" then order =1;
else order =2;
run;

proc sort data =pregnant_female2;
by order;
run;

proc sort data =pregnant_female;
by order;
run;

proc print data=pregnant_female;
run;

*=== Proportion of eater ===*;

Proc sort data = total_food1;
by preg;
run;

Proc freq noprint data = total_food1;
by preg;
weight WT6_2DAY;

```

```

table preg_female_eater /out =
proportion_pregnancy (drop=count) ;
run;

proc print data=proportion_pregnancy;
run;

Data proportion_pregnancy (drop = preg );
    set proportion_pregnancy;
    if _N_ = 1 then delete;

run;

Data proportion_pregnancy;
    set proportion_pregnancy;

    if Preg_female_eater = "P" or
Preg_female_eater = "N" ;

run;

data proportion_pregnancy ;
set proportion_pregnancy ;
if preg_female_eater ="P" then order=2;
else order=1;

run;

proc sort data=proportion_pregnancy ;
by order;
run;

proc print data=proportion_pregnancy ;
run;
data proportion_pregnancy ;
set proportion_pregnancy ;
keep order percent;
run;

proc print data=proportion_pregnancy ;
run;

**** combine proportion, percentile and mean *****;

data pregnant_female_summary;
merge pregnant_female proportion_pregnancy pregnant_female2 ;
by order;
run;

proc print data=pregnant_female;
run;

proc print data=pregnant_female2;
run;

```

```

data pregnant_female_summary;
set pregnant_female_summary;

drop varname order ;
*label percent='proportion_eater';

run;

proc contents data= pregnant_female_summary;
run;

proc print data=pregnant_female_summary;
run;

ods csv file="&PATH\&NAME percapita .csv";
proc print data=pregnant_female_summary;
format LowerCLMean Mean Percent StdErr UpperCLMEAN max min p1 p5 p10 p25 p50 p75
p90 p95 p99 14.4 ;
format preg $prgy.;
run;

ods csv close;

proc print data=pregnant_female_summary ;
run;

ods rtf file="&PATH\&NAME regression_per_capita.rtf";
ods graphics on;
title " per capita ";
Proc surveyreg data = total_food1 ;
class preg;
                                cluster sdmvpsu;
                                strata sdmvstra/NOCOLLAPSE;
    model con_amt=preg /vadjust=none;
    lsmeans preg /diff cl plots=( meanplot(cl));
                                weight WT6_2DAY;
    format preg $prgy.;
                                run;

ods graphics off;
ods rtf close;

***** pregnant and non-pregnant female consumption eater only *****;

```

```

data total_food2;
set total_food1;
if average ne .;
run;

Proc surveymeans data = total_food2 ;
                                cluster sdmvpsu;
                                strata sdmvstra ;
                                weight WT6_2DAY;
                                Domain preg;

                                var con_amt;

                                ods output domain = pregnant_female_eaters
(drop = domainlabel) ;

run;

data pregnant_female_eaters;
set pregnant_female_eaters;
if preg="P" then order =1;
else order=2;
run;

proc print data=pregnant_female_eaters ;
run;

proc sort data=total_food2;
by preg;
run;

Proc univariate data = total_food2 ;

                                weight WT6_2DAY;
                                var con_amt;

                                output out = pregnant_female_p      min=min
p1=p1 p5=p5 p10=p10  p25=p25 p50=p50  p75=p75  p90=p90  p95=p95  p99=p99  max=max ;
                                by preg;

run;

data pregnant_female_p ;
set pregnant_female_p ;
if _N_ = 1 then delete;

run;
data pregnant_female_p;
set pregnant_female_p;
if preg= "P" then order =1;
else order=2;
run;

proc print data=pregnant_female_p ;
run;

```

```

proc sort data=pregnant_female_p;
by order;
run;
proc sort data=pregnant_female_eaters;
by order;
run;
title " female eaters only " ;
proc print data=pregnant_female_eaters;
run;

data prg_female_eaters;
merge pregnant_female_eaters pregnant_female_p ;
by order;
run;

data prg_female_eaters1;
set prg_female_eaters;
drop varname order ;
run;
title " female eaters only ";
proc print data= prg_female_eaters1;
run;

ODS CSV FILE= "&PATH\&NAME eaters.CSV";
proc print data=prg_female_eaters1 ;
format LowerCLMean Mean StdErr UpperCLMEAN max min p1 p5 p10 p25 p50 p75 p90 p95
p99 14.4 ;
format preg $prgy.;
run;

ods csv close;

ods rtf file= "&PATH\&NAME regression_female_eaters.rtf" ;

ods graphics on;
title " Eaters Only ";
Proc surveyreg data = total_food2 ;
class preg;
                                cluster sdmvpsu;
                                strata sdmvstra / NOCOLLAPSE;
model con_amt=preg /vadjust=none;
lsmeans preg/diff cl plots=( meanplot(cl));
                                weight WT6_2DAY;

format preg $prgy.;

run;

```

```
ods graphics off;
```

```
ods rtf close;
```

```
%MEND;
```

```
**** end of SAS MACRO ;
```

```
libname NCEA "F:\NCEA Pregnant women";
```

```
data demographic2 ;  
set NCEA.demographic2;  
run;
```

```
data FCID_commodity2 ;  
set NCEA.FCID_commodity2;  
com_code=FCID_code;  
run;
```

```
%LET NAME = Apple; * replace the value of the name variable to reflect the name of  
the commodity group;
```

```
%LET PATH = F:\NCEA Pregnant women; * replace the path name where you want to store  
the file;
```

```
*====> Apple <====*;
```

```
data FCID_commodity3;  
set FCID_commodity2;  
* replace the commodity codes below if consumption is required for different commodity  
groups*;  
if com_code in ( 1100009000 , 1100009001 , 1100007000 , 1100008000 , 1100008001  
                , 1100011000 , 1100011001)  
run;
```

```
%consumption();
```

```
*====> Total Vegetables <====*;
```

```
data FCID_commodity3;  
set FCID_commodity2;
```

```
if com_code in ( 1800002000 , 401005000 , 103015000 , 103015001 ,  
                9500016000
```

```

,      103017000 ,      401018000 ,      9500019000 ,      902021000 ,      9500022000
,      1901029000 ,      1901029001 ,      1901028000 ,      1901028001 ,      602033000
,      603036000 ,      603038000 ,      602037000 ,      603039000 ,      603040000
,      603041000 ,      603042000 ,      601043000 ,      601043001 ,      101050000
,      101050001 ,      200051000 ,      9500054000 ,      501061000 ,      502063000
,      501062000 ,      501061001 ,      501064000 ,      501069000 ,      502070000
,      501072000 ,      501071000 ,      9500073000 ,      901075000 ,      402076000
,      101078000 ,      101078001 ,      901075000 ,      103082000
,      103082001 ,      501083000 ,      101084000 ,      402085000 ,      402085001
,      402087000 ,      902088000 ,      603099000 ,      603098000 ,      603098001
,      101100000 ,      200101000 ,      902102000 ,      302103000 ,      401104000
,      1902105000 ,      1902105001 ,      1901118000 ,      1901118001 ,      1902119000
,      1902119001 ,      401138000 ,      103139000 ,      200140000 ,      1901144000
,      1902143000 ,      402152000 ,      301165000 ,      301165001
,      103166000 ,      103167000 ,      103166001 ,      101168000 ,      9500177000
,      603182000 ,      603182001 ,      1901184000 ,      1901184001 ,      502194000
,      501196000 ,      302198000 ,      1901202000 ,      401204000 ,      401205000
,      1901220000 ,      1901220001 ,      802234000 ,      301237000 ,      301238000
,      301238001 ,      301237001 ,      302239000 ,      9500243000 ,      1901249000
,      1901249001 ,      401248000 ,      101250000 ,      101251000 ,      101251001
,      603256000 ,      603256001 ,      601257000 ,      603258000 ,      602259000
,      602255000 ,      602255001 ,      802270000
,      802271000 ,      802271001 ,      802270001 ,      1902274000 ,      1902274001
,      802272000 ,      802273000 ,      802272001 ,      9500275000 ,      103296000
,      103297000 ,      103297001 ,      103298000 ,      103298001 ,      103300000
,      103300001 ,      103299000 ,      103299001 ,      902308000 ,      401313000
,      101316000 ,      200317000 ,      101314000 ,      200315000 ,      502318000
,      402322000 ,      101327000 ,      101331000 ,      200332000 ,      1901334000
,      9500335000 ,      9500335001 ,      301338000 ,      603348000 ,      603348001
,      600347000 ,      1902354000 ,      1902354001 ,      902356000 ,      902356001
,      902357000 ,      902357001 ,      103366000 ,      103366001 ,      402367000
,      103371000 ,      801374000 ,      801375000 ,      801378000 ,      801378001
,      801376000 ,      801376001 ,      801377000 ,      801377001 ,      801380000
,      801375001 ,      103387000 ,      502389000 ,      101388000 ,      9500397000
,      9500398000 ,      901399000 ,      103407000
,      103406000) ;

```

```
run;
```

```
%consumption();
```

```
*====> Total Fruit <====*;
```

```
data FCID_commodity3;
```

```
set FCID_commodity2;
```

```

if com_code in (      9500001000 ,      1100009000 ,      1100009001 ,      1100007000 ,
1100008000 ,      1100008001 ,      1100011000 ,      1100011001 ,
1202012000 ,      1202013000 ,      1202012001 ,      9500020000 ,
9500023000 ,      9500024000 ,      9500024001 ,      9500023001 ,
1301055000 ,      1302057000 ,      1302057001 ,      1301058000 ,
9500060000 ,      9500074000 ,      9500089000 ,      1201090000 ,
1201090001 ,      1001106000 ,      1001107000 ,      9500112000 ,
9500111000 ,      9500111001 ,      9500113000 ,      1100129000 ,
1307130000 ,      1307131000 ,      1307130001 ,      1302136000 ,
1302137000 ,      9500141000 ,      802148000 ,
1302149000 ,      9500151000 ,      9500153000 ,      9500154000 ,
1302174000 ,      1304175000 ,      9500178000 ,      1003180000 ,
9500183000 ,      9500183001 ,      1302191000 ,      9500193000 ,
1304195000 ,      1002197000 ,      1002199000 ,      1002201000 ,
1002206000 ,      1301208000 ,      9500209000 ,      1100210000 ,
9500211000 ,      9500212000 ,      9500214000 ,      9500215000 ,

```

```

    9500216000 ,    9500215001 ,    1303227000 ,    1202230000 ,
    1001240000 ,    1001242000 ,    9500245000 ,    9500246000 ,
    9500245001 ,    9500252000 ,    9500252001 ,    ,    9500254000 ,
    1202260000 ,    1202261000 ,    1202261001 ,    1202260001 ,
    1100266000 ,    1100267000 ,    1100266001 ,    9500277000 ,
    9500279000 ,    9500280000 ,    9500279001 ,    9500283000 ,
    9500284000 ,    1203285000 ,    1203287000 ,    1203287001 ,
    1203286000 ,    1203286001 ,    1203285001 ,    9500289000 ,
    1003307000 ,    1100310000 ,    1301320000 ,    1301320001 ,
    9500333000 ,    9500346000 ,    9500351000 ,    ,    9500358000 ,
    1307359000 ,    1307359001 ,    9500361000 ,    9500368000 ,
    1001369000 ) ;
run;

%consumption();

*====> Banana <====*;
data FCID_commodity3;
set FCID_commodity2;

if com_code in ( 9500023000 , 9500024000 , 9500024001 , 9500023001);
run;

%consumption();

*====> Beans <====*;
data FCID_commodity3;
set FCID_commodity2;

if com_code in ( 603035000 ,    603030000 ,    603032000 ,    602031000 ,    603034000
    ,    602033000 ,    603036000 ,    603038000 ,    602037000 ,    603039000
    ,    603040000 ,    603041000 ,    603042000 ,    601043000 ,    601043001);
run;
%consumption();

*====> Berries and small fruit <====*;
data FCID_commodity3;
set FCID_commodity2;

if com_code in ( 1301055000 ,    1301058000 ,    1301208000 ,    1301320000 ,
1301320001
    ,    1302057000 ,    1302057001 ,    1302136000 ,    1302137000 ,
1302149000
    ,    1302174000 ,    1302191000 ,    1307130000 ,    1307130001 ,
1307131000
    ,    1304175000 ,    9500177000 ,    9500178000 ,    1304195000 ,
1303227000
    ,    1307359000 ,    1307359001);

run;
%consumption();

```

```

*====> Broccoli <====*;

data FCID_commodity3;
set FCID_commodity2;

    if com_code in ( 501061000 ,    501061001);
run;
%consumption();

*====> Bulb Vegetables <====*;

data FCID_commodity3;
set FCID_commodity2;

if com_code in ( 301165000 ,    301165001 ,    302198000 ,    301237000 ,
    301237001 ,    301238000 ,    301238001 ,    302239000 ,
    301338000 ,    302103000 ,    302338500);
run;

%consumption();

*====> Cabbage <====*;
data FCID_commodity3;
set FCID_commodity2;

if com_code in ( 501069000 ,    501072000 ,    501071000);
run;

%consumption();

*====> Carrots <====*;

data FCID_commodity3;
set FCID_commodity2;

    if com_code = 101078000;
run;

%consumption();

*====> Citrus <====*;
data FCID_commodity3;
set FCID_commodity2;

if com_code in ( 1001106000 ,    1001107000 ,    1003180000 ,    1002197000
    ,    1002199000 ,    1002201000 ,    1002206000 ,    1001240000 ,
    1001242000 ,    1003307000 ,    1001369000);

run;

%consumption();

*====> Corn <====*;

data FCID_commodity3;
set FCID_commodity2;

```

```

if com_code in (1500122000 , 1500120000 , 1500120001 , 1500121000 ,
1500121001
, 1500123000 , 1500123001 , 1500126000 , 1500127000 ,
1500127001);

run;

%consumption();

*====> Cucumber <====*;
data FCID_commodity3;
set FCID_commodity2;

if com_code = 902135000;
run;
%consumption();

*====> Cucurbits <====*;

data FCID_commodity3;
set FCID_commodity2;

if com_code in ( 901075000 , 901187000 , 901399000 , 902021000
, 902088000 , 902102000 , 902135000 , 902308000 , 902309000
, 902356000 , 902356001 , 902357000 , 902357001);
run;

%consumption();

*====> Fruiting Vegetable <====*;

data FCID_commodity3;
set FCID_commodity2;

if com_code in (802148000 , 802234000 , 802270000 , 802270001 , 802271000
, 802271001 , 802272000 , 802272001 , 802273000 , 801374000
, 801375000 , 801375001 , 801376000 , 801376001 , 801377000
, 801377001 , 801378000 , 801378001);

run;

%consumption();

*====> Lettuce <====*;
data FCID_commodity3;
set FCID_commodity2;

if com_code in ( 401204000 , 401205000);
run;

%consumption();

*====> Leafy Vegetables <====*;

```

```

data FCID_commodity3;
set FCID_commodity2;

if com_code in ( 200051000 , 200101000 , 200140000 , 200315000 , 200317000
, 200332000 , 401005000 , 401018000 , 401104000 , 401133000
, 401134000 , 401138000 , 401150000 , 401204000 , 401205000
, 401248000 , 401313000 , 401355000 , 401355001 , 402076000
, 402085000 , 402085001 , 402087000 , 402152000
, 402322000 , 402367000 , 501061000 , 501061001 , 501062000
, 501064000 , 501069000 , 501071000 , 501072000 , 501083000
, 501196000 , 502063000 , 502070000 , 502117000 , 502194000
, 502229000 , 502318000 , 502389000 , 9500054000 , 9500335000
, 9500335001 , 9500398000);

run;

```

```
%consumption();
```

```
*====> Legume Vegetables <====*;
```

```

data FCID_commodity3;
set FCID_commodity2;

```

```

if com_code in ( 600347000 , 603348000 , 603348001 , 600349000 , 600349001
, 601043000 , 601043001 , 601257000 , 602031000 , 602033000
, 602037000 , 602255000 , 602255001 , 602259000 , 603030000
, 603032000 , 603034000 , 603035000 , 603036000 , 603038000
, 603039000 , 603040000 , 603041000 , 603042000 , 603098000
, 603098001 , 603099000 , 603182000 , 603182001 , 603203000
, 603256000 , 603256001 , 603258000);

run;

```

```
%consumption();
```

```
*====> Onion <====*;
```

```

data FCID_commodity3;
set FCID_commodity2;

```

```

if com_code in ( 301237000 , 301238000 , 301238001 , 301237001 ,
302239000);

run;

```

```
%consumption();
```

```
*====> Pea <====*;
```

```

data FCID_commodity3;
set FCID_commodity2;

```

```

if com_code in ( 603256000 , 603256001 , 601257000 , 603258000 , 602259000
, 602255000 , 602255001);

run;
%consumption();

```

```
*====> Peach <====*;
```

```

data FCID_commodity3;

```

```

set FCID_commodity2;

if com_code in ( 1202260000 , 1202261000 , 1202261001 , 1202260001);
run;
%consumption();

*====> Pear <====*;
data FCID_commodity3;
set FCID_commodity2;

if com_code in ( 1100266000 , 1100267000 , 1100266001);
run;

%consumption();

**** Pome Fruit ****;
data FCID_commodity3;
set FCID_commodity2;

if com_code in ( 1100007000 , 1100008000 , 1100008001 ,
1100009000 , 1100009001
, 1100011000 , 1100011001 , 1100129000 , 1100210000
, 1100266000
, 1100266001 , 1100267000 , 1100310000);
run;

%consumption();

*====> Root and Tuber Vegetables <====*;

data FCID_commodity3;
set FCID_commodity2;

if com_code in ( 103015000 , 103015001 , 103017000 , 101050000 , 101050001
, 200051000 , 101052000 , 101052001 , 101067000 , 101078000
, 101078001 , 103082000 , 103082001 , 101084000 , 101100000
, 103139000 , 103166000 , 103167000 , 103166001 , 101168000
, 101190000 , 101250000 , 101251000 , 101251001 , 103296000
, 103297000 , 103297001 , 103298000 , 103298001 , 103300000
, 103300001 , 103299000 , 103299001 , 101316000 , 101314000
, 101327000 , 103366000 , 103366001 , 103371000 , 103387000
, 101388000 , 9500397000 , 103407000 , 103406000) ;
run;
%consumption();

*====> Strawberries <====*;

data FCID_commodity3;
set FCID_commodity2;

if com_code in ( 1307359000 , 1307359001);
run;

```

```

%consumption();

*====> Stalk and Stem Vegetables <====*;
data FCID_commodity3;
set FCID_commodity2;

if com_code in ( 9500016000 , 9500019000 , 9500022000 , 2100228000 ,
9500243000);
run;

%consumption();

*====> Stone Fruit <====*;

data FCID_commodity3;
set FCID_commodity2;

if com_code in ( 1202012000 , 1202012001 , 1202013000 , 1201090000 ,
1201090001
, 1202230000 , 1202260000 , 1202260001 , 1202261000 , 1202261001
, 1203285000 , 1203285001 , 1203286000 , 1203286001 , 1203287000
, 1203287001);
run;
%consumption();

*====> Tropical Fruit <====*;

data FCID_commodity3;
set FCID_commodity2;

if com_code in ( 9500001000 , 9500022000 , 9500023000 , 9500023001 ,
9500024000 , 9500024001 , 9500060000 , 9500074000 , 9500089000 ,
9500111000 , 9500111001 , 9500112000 , 9500113000 , 9500141000 ,
9500151000 , 9500153000 , 9500154000 , 9500183000 , 9500183001 ,
9500193000 , 9500209000 , 9500211000 , 9500212000 , 9500214000 ,
9500215000 , 9500215001 , 9500216000 , 9500245000 , 9500245001 ,
9500246000 , 9500252000 , 9500252001 , 9500254000 , 9500279000 ,
9500279001 , 9500280000 , 9500283000 , 9500284000 , 9500289000 ,
9500333000 , 9500346000 , 9500351000 , 9500358000 , 9500361000 ,
9500368000);
run;

%consumption();

*====> Tomatoes <====*;

data FCID_commodity3;
set FCID_commodity2;

if com_code in ( 801375000 , 801378000 , 801378001 , 801376000 , 801376001
, 801377000 , 801377001 , 801375001);
run;

%consumption();

```

```

*====> White Potatoes <====*;

data FCID_commodity3;
set FCID_commodity2;

if com_code in ( 103296000 , 103297000 , 103297001 , 103298000 , 103298001
, 103300000 , 103300001 , 103299000 , 103299001);

run;

%consumption();

*====> Total Fish <====*;

data FCID_commodity3;
set FCID_commodity2;

if com_code in ( 8000157000 , 8000158000 , 8000159000 , 8000160000 ,
8000161000 , 8000162000);
run;

%consumption();

*====> Total ShellFish <====*;

data FCID_commodity3;
set FCID_commodity2;

if com_code in ( 8000161000 , 8000162000);
run;

%consumption();

*====> Total Finfish <====*;

data FCID_commodity3;
set FCID_commodity2;

if com_code in ( 8000157000 , 8000158000 , 8000159000 , 8000160000);
run;

%consumption();

***** Rice *****;

data FCID_commodity3;
set FCID_commodity2;

```

```

if com_code in ( 1500326000 ,      1500326001 ,      1500324000 ,      1500324001 ,
1500325000 ,      1500325001 ,      1500323000 ,      1500323001);
run;

%consumption();

*====> Total Grain <====*;
data FCID_commodity3;
set FCID_commodity2;

if com_code in ( 950006000 ,      1500025000 ,      1500025001 ,      1500026000 ,
1500026001
,      1500027000 ,      1500065000 ,      1500066000 ,      1500120000 ,      1500120001
,      1500121000 ,      1500121001 ,      1500122000 ,      1500123000 ,      1500123001
,      1500126000 ,      1500127000 ,      1500127001 ,      1500226000 ,      1500231000
,      1500232000 ,      1500232001 ,      1500233000 ,      1500233001 ,      9500306000
,      9500311000 ,      1500323000 ,      1500323001 ,      1500324000 ,      1500324001
,      1500325000 ,      1500325001 ,      1500326000 ,      1500326001 ,      1500328000
,      1500329000 ,      1500344000 ,      1500381000 ,      1500381001 ,      1500401000
,      1500401001 ,      1500402000 ,      1500402001 ,      1500403000 ,      1500404000
,      1500405000);
run;
%consumption();

*====> Total Cereal Grains <====*;
data FCID_commodity3;
set FCID_commodity2;

if com_code in ( 1500025000 ,      1500025001 ,      1500026000 ,      1500026001 ,
1500027000
,      1500065000 ,      1500066000 ,      1500120000 ,      1500120001 ,      1500121000
,      1500121001 ,      1500122000 ,      1500123000 ,      1500123001 ,      1500124000
,      1500124001 ,      1500126000 ,      1500127000 ,      1500127001 ,      1500226000
,      1500231000 ,      1500232000 ,      1500232001 ,      1500233000 ,      1500233001
,      1500233000 ,      1500233001 ,      1500324000 ,      1500324001 ,      1500325000
,      1500325001 ,      1500326000 ,      1500326001 ,      1500328000 ,      1500329000
,      1500344000 ,      1500345000 ,      1500381000 ,      1500381001 ,      1500401000
,      1500401001 ,      1500402000 ,      1500402001 ,      1500403000 ,      1500404000
,      1500405000 ,      950006000 ,      9500306000 ,      9500311000);
run;

%consumption();

*====> Total Beef <====*;

data FCID_commodity3;
set FCID_commodity2;

if com_code in ( 3100044000 ,      3100044001 ,      3100045000 ,      3100046000 ,
3100046001 ,      3100047000 ,      3100047001 ,      3100048000 ,      3100049000 ,
3100049001);
run;

```

```

%consumption();

*====> Total Pork <====*;
data FCID_commodity3;
set FCID_commodity2;

if com_code in ( 3400290000 , 3400290001 , 3400291000 , 3400292000 ,
3400292001 , 3400293000 , 3400293001 , 3400294000 , 3400295000);
run;

%consumption();

*====> Total Poultry <====*;
data FCID_commodity3;
set FCID_commodity2;

if com_code in ( 4000093000 , 4000093001 , 4000094000 , 4000095000 ,
4000095001
, 4000096000 , 4000096001 , 4000097000 , 4000097001 , 5000382000
, 5000382001 , 5000383000 , 5000383001 , 5000384000 , 5000384001
, 5000385000 , 5000385001 , 5000386000 , 5000386001 , 6000301000
, 6000302000 , 6000303000 , 6000304000 , 6000305000);
run;

%consumption();

*====> Total Meats <====*;
data FCID_commodity3;
set FCID_commodity2;

if com_code in ( 3100044000 , 3100044001 , 3100045000 , 3100046000 ,
3100046001
, 3100047000 , 3100047001 , 3100048000 , 3100049000 , 3100049001
, 3200169000 , 3200170000 , 3200171000 , 3200172000 , 3200173000
, 3300189000 , 3400290000 , 3400290001 , 3400291000 , 3400292000
, 3400292001 , 3400293000 , 3400293001 , 3400294000 , 3400295000
, 3500339000 , 3500339001 , 3500340000 , 3500341000 , 3500341001
, 3500342000 , 3500343000 , 3800221000 , 3900312000 , 4000093000
, 4000093001 , 4000094000 , 4000095000 , 4000095001 , 4000096000
, 4000096001 , 4000097000 , 4000097001 , 5000382000 , 5000382001
, 5000383000 , 5000383001 , 5000384000 , 5000384001 , 5000385000
, 5000385001 , 5000386000 , 5000386001 , 6000301000 , 6000302000
, 6000303000 , 6000304000 , 6000305000);
run;

%consumption();

*====> Total Dairy <====*;

data FCID_commodity3;
set FCID_commodity2;

```

```

if com_code in ( 3600222000 , 3600222001 , 3600223000 , 3600223001 ,
3600224000 , 3600224001 , 3600225001);
run;

%consumption();
*Minimum sample size calculation for upper and lower percentiles for pregnant
and non-pregnant female* ;
* This sample size calculation does not depend on a particular commodity or
commodity groups*;

libname NCEA "F:\NCEA Pregnant women";

data demographic2 ;
set NCEA.demographic2;
run;

data FCID_commodity2 ;
set NCEA.FCID_commodity2;
com_code=FCID_code;
run;

data FCID_commodity3;
set FCID_commodity2;
run;

proc sort data=FCID_commodity3;
by seqn daycode;
run;

Proc univariate data =FCID_commodity3 noprint;
var intake_bw;
by seqn daycode;
output out = total_food_day sum = total;
run;

Proc transpose data = total_food_day out = average_total;
var total;
by seqn;
ID daycode;
run;

Data average_total (keep = seqn average);

```

```

    set average_total;
    if _1 = . then _1 = 0;
    if _2 = . then _2 = 0;
    average = (_1+_2)/2;
run;

```

```

proc sort data=demographic2;
by seqn;
run;

```

```

*====> Merge individual with the demographic file <====*;

```

```

Data total_food;
    merge work.demographic2 average_total;
    by seqn;
run;

```

```

*==== Pregnant Female Varaince Inflation Factor and sample size calculation
====*;

```

```

                                Data total_food1;
                                set total_food;

                                if preg_f=1 and sex=2 and age > 12 and age < 50
then Preg = "P";
                                else if preg_f=0 and sex=2 and age > 12 and age < 50
then Preg = "N" ;

                                run;

```

```

proc format;
value $ prgy
"P" = " Pregnant 13-49  "
"N" = " Not pregnant 13-49  " ;

run
;

```

```

data total_food1;

```

```

set total_food1;
weight1=WT6_2DAY**2;
weight2=WT6_2Day;
run;

data total_food_prg;
set total_food1;
if preg= "P";
run;

data total_food_nprg;
set total_food1;
if preg = "N";
run;

Proc surveymeans data = total_food_prg ;
                    cluster sdmvpsu;
                    strata sdmvstra;
                    var weight1 weight2;

                    run;

data VIF2;
input numerator denominator;
datalines;
138257603 6253.756527
;
run;

data VIF2;
set VIF2;
denominator=denominator**2;
run;

data VIF2;
set VIF2;
VIF=numerator/denominator;
n_01=8*(VIF/0.01); * sample size required for 1st percentile *;
n_05=8*(VIF/0.05); * sample size required for 5th percentile *;
n_10=8*(VIF/0.10); * sample size required for 10th percentile *;
n_25=8*(VIF/0.25); * sample size required for 25th percentile *;
n_75=8*(VIF/0.25); * sample size required for 75th percentile *;
n_90=8*(VIF/0.10); * sample size required for 90th percentile *;
n_95=8*(VIF/0.05); * sample size required for 95th percentile *;
n_99=8*(VIF/0.01); * sample size required for 99th percentile *;
n_99_99= 8*(VIF/0.0001); * sample size required for 99.99th percentile *;
run;
ods rtf file="F:\NCEA Pregnant women\Modification\ sample size pregnant
women.rtf";

title 'pregnant women sample size ';

```

```

proc print data=VIF2;
run;
ods rtf close ;

*****;

***** Not-pregnant female VIF and sample size calculation *****;

Proc surveymeans data = total_food_nprg ;
                                cluster sdmvpsu;
                                strata sdmvstra;
                                var weight1 weight2;
                                *ods output statistics=VIF2 (keep=mean
varname) ;
                                run;

data VIF3;
input numerator denominator;
datalines;
527057037 14616
;
run;

data VIF4;
set VIF3;
denominator=denominator**2;
run;

proc print data=VIF4;
run;

data VIF4;
set VIF4;
VIF=numerator/denominator;
n_01=8*(VIF/0.01); * sample size required for 1st percentile *;
n_05=8*(VIF/0.05); * sample size required for 5th percentile *;
n_10=8*(VIF/0.10); * sample size required for 10th percentile *;
n_25=8*(VIF/0.25); * sample size required for 25th percentile *;
n_75=8*(VIF/0.25); * sample size required for 75th percentile *;
n_90=8*(VIF/0.10); * sample size required for 90th percentile *;
n_95=8*(VIF/0.05); * sample size required for 95th percentile *;
n_99=8*(VIF/0.01); * sample size required for 99th percentile *;
n_99_99= 8*(VIF/0.0001); * sample size required for 99.99th percentile *;
run;

ods rtf file="F:\NCEA Pregnant women\Modification\ sample size not-pregnant
women.rtf";
title ' sample size non-pregnant';
proc print data= VIF4;

```

run;

Appendix 5 :

References:

Appendix B: ANALYTIC AND REPORTING GUIDELINES:

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