Chapter 4 Peer Review Comments and EPA Response

Reviewer	Comment Number	Comment	Final Draft Response (with Scott's resolutions)
Sinha	1	A major component of EPA's Ground Water Rule (GWR) is to provide the public with increased protection against microbial pathogens in public water systems that routinely use ground water sources. To achieve this goal, it is necessary to carry out on a regular basis suitable sampling schemes to estimate viral and indicator occurrences in drinking water sources for risk characterizations. The document under review, which is Section 4 of EPA GWR EA, provides details of such an attempt based on fourteen (14) ground water well surveys.	Though no action is requested by the reviewer, EPA notes that the final GWR does not include sampling "to estimate viral and indicator occurrences" as suggested within the comment.
Sinha	2	Since ground water sources used for public consumption vary widely according to system size (number of people served), type of ownership (private, public, other), system type (treat water directly or purchase treated water) and source type (ground water, surface water), a proper risk assessment analysis ought to be carried out in a stratified fashion, providing human health risk factors in each meaningful combination category.	The final GWR EA risk model stratifies GW PWSs into 216 categories based on 3 system types (CWS, NTNCWS, TNCWS); 2 disinfection groups (with / without); 2 vulnerability groups; 2 well condition types; and 9 system sizes (i.e., 3*2*2*2*9=216). These capture differences in key risk (or risk reduction) factors such as virus concentrations; reduction in virus concentration between source and finished water; daily water consumption; days of exposure per year; and likelihood of eliminating virus occurrence from sanitary surveys or source water monitoring.
Sinha	3	Exhibit 4.1displays a national inventory of GWR system baseline in regard to system size, source type, ownership type and system type. Exhibit 4.2 provides a break-down of GWR system baseline in two categories of disinfecting and non-disinfecting systems. Exhibits 4.3–4.5 provide useful information about entry points and number of people served in each category. Two types of treatment plant flows per entry point, which can be used to compute treatment technology costs, are given in Exhibit 4.6 along with average population served per entry point for each system	EPA thanks the reviewer for the comment.

size. It is mentioned that separate calculations for publicly	
and privately owned water systems is not warranted. Exhibit	
4.7 providing treatment practices for ground water systems	
for different system sizes reveal a quite interesting fact that	
at both pre- and post-disinfection levels, chlorine is most	
widely used.	

Sinha	4	Finally, Exhibit 4.8 in this category gives the number of households served by both disinfecting and non-disinfecting systems for different system sizes. Since an important variable used in cost and benefits analysis is the number of water Total Coliform Rule (TCR) samples that would test positive for total coliform each year, Exhibit 4.11 provides national estimates of such numbers based on total coliform positive hit rates (Exhibit 4.9) and estimated number of routine total coliform samples taken from a water source per year (Exhibit 4.10). Since these numbers vary by system type and system size, estimates are provided for all combinations of system type and system	EPA thanks the reviewer for the
Sinha	5	size. Based on the limited data on system sizes provided in Exhibits 4.9 and 4.10, it is not clear how the results in Exhibit 4.11 are derived for finer divisions of system sizes! A clear explanation is warranted here.	In Section 4.2.7 "Triggered Monifinal GWR EA, EPA provides sp the calculation to estimate the nu system, per year, that are shown in of this process describes how val "Total Coliform Positive Hit Rate values from Exhibit 4.10 "Estimate Total Coliform Samples Taken P Type and Size of System", and pu illustrate how EPA estimated the
Sinha	6	Section 4.3, the core section of the report under review, provides in some detail statistical risk characterization methods based on hazard identification, exposure assessment and dose-response assessment to describe and produce an overall risk to the exposed population, both in terms of distribution of risk levels in the population and total	baseline. Though no action is requested by that the final GWR EA uses two groups (Type A representing those but mild symptoms, and Type B infectivity but potentially more so represent all viruses. These two

e comment.

nitoring Baseline" of the specific examples to explain number of TC+ samples per n in Exhibit 4.11. "Step 3" alues from Exhibit 4.9 ates" were multiplied by nated Number of Routine Per System, Per Year, by provides two examples to ne triggered monitoring

by the reviewer, EPA notes o representative virus ose with high infectivity 3 with low to moderate severe health effects) to o representative viral

Sinha	7	number of anticipated cases of adverse effects. Two groups of viruses, Type A representing those with high infectivity but mild symptoms and Type B with low to moderate infectivity but potentially more severe health effects, are taken as representative pathogens due to insufficient data being available on bacterial pathogen. To determine the probability, which is an important ingredient in risk assessment analyses, that a well or a sample will test positive for enterovirus or E. coli, EPA reviewed data from 23 studies and selected 14 studies whose results are subsequently used to perform the probability calculations. For each of the studies under consideration, the report provides a useful summary of study objectives,	groups are not used "due to insufficient data being available on bacterial pathogen," as the comment implies. Though no action is requested by the reviewer, EPA notes that in the final GWR EA, the number of studies reviewed by EPA is 24, from which 15 are used for the occurrence analysis.
Sinha	8	well selection procedure, data representativeness and sample results. Exhibit 4.12 shows the main results obtained from AwwaRF/AWWSCo study. Similar exhibits depicting results from other studies would have been quite useful!	The final GWR EA includes additional summary tables (4.13a - d), which summarize results for all of the GWR studies together. EPA believes that the method of selecting the studies to represent the occurrence of indicators and pathogens, as well as the level of detail of summarized data in the final EA, provides a representative and consistent basis for analysis and comparison of data for the risk assessment. EPA provided as much data as possible in consideration of the quality (detection limits, statistical representation, etc.) of available data.
Sinha	9	The first 13 full scale studies and one pilot study reported here cover a wide spectrum of states, well locations within states, and nature of wells. In addition to the above studies, results of seven new studies are also included in the probability computations. Details of these latter studies appear in Exhibit 4.13. This entire subsection of the report, subsection 4.3.2, is highly commendable.	Though no action is requested by the reviewer, EPA notes that in the final GWR EA, the number of studies reviewed by EPA is 24, from which 15 are used for the occurrence analysis.
Sinha	10	Subsection 4.5.2, is highly commendable. Since vulnerability of wells in regard to viral occurrences is of great concern, EPA categorized water source systems into two groups: more vulnerable as reflected by MCL violations under TCR and less vulnerable having no MCL violations under TCR. Exhibit 4.14 presents estimated percentage of	EPA thanks the reviewer for the comment.

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systems belonging to more vumerable type in each
combination of system size and system type category.
Another relevant information in regard to percentage of
systems disinfecting is provided in Exhibit 4.15.
Subsection 4.3.4 of Section 4.3 contains the most useful
information in terms of statistical modeling and analyses
used in the EA, providing estimates of viral pathogen hit
rates, indicator (E. coli) hit rates, viral concentrations and
co-occurrences of viruses and indicators based on the pooled
analysis of all available occurrence data. Two components
of hit rates, namely P_well and P_sample, are defined with
suitable ramifications. The basic co-occurrence model in
regard to wells is described in Exhibit 4.16 through a Venn
diagram, depicting the four scenarios: (virus, no indicator:
P_1), (indicator, no virus:P_3), (virus and indicator:P_2),
and (no virus, no indicator:P_4). While the probability
distribution of P_well is characterized by the four
probabilities:
P = 1, P = 2, P = 3, P = 4 = 1 - P = 1 - P = 2 - P = 3, P = 3 sample on the

systems belonging to more vulnerable type in each

P_1, P_2, P_3, P_4 = $1 - P_1 - P_2 - P_3$, P_sample on the other hand is defined within a well and its variation over all the wells is characterized by two beta distributions, one for virus and the other for indicator. In essence, it then results in seven parameters: P_1, P_2, P_3, alpha_virus, beta_virus, alpha_indicator, beta_indicator, whose estimation and inference is crucial for EA.

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Given the above background of model selection, a common statistical approach is to write down the likelihood function with all details, identify sufficient statistics, and apply standard statistical methods which are typically frequentist or Fisherian in nature. Of course, one can also follow the Bayesian route to solve the pertinent inference problems, especially if the frequentist approach is formidable and the latter is easier to implement. It appears that this report does not at all attempt to carry out the standard frequentist methods, not even mention about it, but rather jumps into the Bayesian solution right away. It is desirable that this report provides a valid justification for following the EPA thanks the reviewer for the comment.

EPA recognized that an uncertainty sample of parameter values would be needed to properly inform a probabilistic risk analysis. The Bayesian approach produces such a sample directly and thereby allows probabilistic treatment of uncertainty. Technically, the frequentist approach does not permit this kind of treatment because it regards the parameter values as fixed and allows probabilistic statements only about the data at hand. Frequentist statements about parameter values are couched in terms of confidence rather than probability. EPA also recognized the Bayesian approach's superior performance with complex hierarchical models, such as that used in the hitSinha

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Bayesian route, skipping the standard frequentist approach altogether. While the MCMC is a useful technical tool for the Bayesians, bootstrapping is likewise a great tool for the frequentists, and both have advantages and disadvantages. Although the construction of the likelihood function is briefly described, it would be helpful to actually provide an explicit formula for the likelihood function based on the pooled data described in subsection 4.3.2. In particular, a new Exhibit showing 1) the grand total number of wells being incorporated into the likelihood, 2) memberships of the selected wells into one of four categories, 3) values of N v, K v, N i, K i for each well, would be very helpful and is warranted especially because of the very complex nature of the pooled data from 14 viruses and E. coli occurrence studies reported in subsection 4.3.2. Obviously, 1), 2) and 3) along with the multinomial/binomial/beta distributions are the basic ingredients behind the likelihood function.

rate analysis. Although the dose-response models could have been analyzed using frequentist methods, EPA preferred to not use a mixture of methods (e.g. Bayesian for hit rates and frequentist for dose-response). The explicit formulas for the likelihood functions are now provided and discussed in detail in Section 4.3.4.1 of the Final EA.

Exhibits 4.13.b and 4.13.c in the final GWR EA show the number of wells having different combinations for virus (Nv and Kv) and E. coli (Ni and Ki), respectively. Showing the breakdown for combinations of all four (Nv, Ni, Kv, and Ki) would require a very large table. Regarding the number of wells in each category, EPA has estimates of P1, P2, P3, and P4. In each Markov Chain Monte Carlo iteration, individual wells are assigned to individual categories, based on the parameter values and the individual wells' data. EPA did monitor selected wells to observe their membership probabilities in the four groups, but monitoring all wells would have been computationally prohibitive. Wells with both virus and E. coli positives were only assigned to category P2. Wells with virus, but no E. coli positives were assigned to categories P1 and P2. Wells with E. coli, but no virus positives were assigned to categories P2 and P3. Wells with neither E. coli nor virus positives were assigned to all four categories. Among those wells with no positives of either type, those with many assays were more often assigned to category P4 than those with few assays. These are all reasonable outcomes, reflecting the limited amount of information conveyed by the data for individual wells. EPA thanks the reviewer for the comment.

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Exhibit 4.17 provides various aspects of Bayesian inference regarding the four multinomial parameters P_1, P_2, P_3, P_4 in terms of their posterior median values, and posterior 5th and 95th percentiles. Exhibits 4.18 and 4.19 do the same for the derived P well values for viruses and indicators.

Exhibits 4.20 through 4.23 provide similar aspects of the posterior distributions of P_sample based on viruses and indicators. The last two Exhibits 4.24 and 4.25 in this part display scatter plots of P_sample versus P_well separately for viruses and indicators.

There are two aspects of characterizing virus occurrences in source water used by public ground water wells, namely, hit rates dealing with presence/absence of virus in water and virus concentrations analysis in virus-positive well water. To analyze the first aspect, Exhibit 4.26 represents the cumulative probability of having an indicator positive as a function of assay number based on data from all wells surveyed. This is useful information because even if a virus is present, it may take several assays before it can be detected.

Exhibit 4.27 depicts similar findings based only on virus positive wells.

The second aspect pathogen concentration analysis is carried out based on data from three key studies, one study providing information on more vulnerable wells and two studies providing information on less vulnerable wells. The findings from these studies are summarized in Exhibits 4.28, 4.29 and 4.30.

Sinha15Due to lack of sufficient data, it is assumed in the above
occurrence analysis that the same hit rates and viral
concentrations pertain to both sensitive and non-sensitive
wells. Obviously, wells in sensitive aquifers are more likely
to be virus positive and have higher virus concentrations
than wells in non-sensitive aquifers.Sinha16Exhibits 4.31ab describe how allocation of benefits changes,
assuming no difference and a difference of twice the rate for
sensitive as against non-sensitive wells.
The last Exhibit 4.33 provides a summary of uncertainties

affecting GWR baseline estimates due to limitations of available data.

EPA thanks the reviewer for the comment.

EPA thanks the reviewer for the comment.

		To sum up, on the whole, the report is excellent in terms of meeting the objectives and presenting the results with clarity and enough details. However, some points raised above and below under specific comments need to be addressed and included in the report.	
Sinha	17	The multinomial model used to describe the occurrences of four cell frequencies (well categories) is quite standard in this kind of context, and is a very reasonable assumption.	EPA thanks the reviewer
Sinha	18	Statistical modeling of a fraction by a beta distribution is again standard. Due to lack of sufficient data, it is assumed in the report that the same parameters apply for wells in the two categories P_1 and P_2 for detecting virus. This may obviously introduce some bias in the subsequent computations of risk factors. It would be desirable to estimate the beta parameters separately for the two categories even if there is scanty data in each set just to verify if the computed estimates of the beta parameters are close so that the assumption of same parameters becomes tenable.	Section 4.3.4.1 discusses estimating different Psar Also, refer to the respons Another reviewer also m regard. Following throug found that even a small e model could not be supp an analysis with only one more complex than our r complex than the model and found that the data w respect to this new paran the data limitation that p Psample distributions for tables (4.13b and 4.13c) amount of data available distributions of wells by positives. Exhibit 4.13c number of E. coli assays tables clearly show that t
Sinha	19	Computation of concentration level for a well on days when virus is present is based on a random selection from the set of positive virus measured values (bootstrapping!) obtained from the entire survey data. Naturally, this may induce some bias in subsequent risk computations, which can be avoided to some extent by limiting the universe to wells belonging to similar categories.	positive assays are very s EPA believes that the po virus concentrations sele addressed by the more very that uses concentration v categories. An alternative data for only noncommu virus concentration value

er for the comment.

es the data limitation that precludes ample distributions for P1 and P2. onse to Stedinger comment 5. made some suggestions in this ough on these suggestions, EPA embellishment of the current ported by the data. EPA attempted ne additional parameter (slightly reported model and slightly less el suggested by the second reviewer) were nearly noninformative with ameter. Section 4.3.4.1 discusses precludes estimating different or P1 and P2. EPA has added two c) to better illustrate the limited le. Exhibit 4.13b shows y numbers of virus assays and virus Sc shows distributions of wells by ys and E. coli positives. These two the numbers of wells with multiple small. otential for introducing bias in the

lected for individual wells is versus less vulnerable stratification values observed in wells in those ive analysis using the Pennsylvania unity wells was also done so that ues for noncommunity wells are

Sinha	19	The assumption of same hit rates and same viral concentrations for sensitive and non-sensitive wells, which is made due to lack of sufficient data, can lead to potential bias in the analysis. It may be worthwhile to carry out the risk analysis with one beta distribution of P_sample using the parameters (alpha_ns, beta_ns) for non-sensitive wells and another distribution of P_sample using the parameters (alpha_s, beta_s) for sensitive wells, taking alpha_s = c (alpha_ns) and beta_s = d(beta_ns) for known c > 1 and d > 1 for both virus and indicator. Several pairs of values of (c, d) can be tried to hopefully to come up with a robust choice!	The reviewer believes that it is worthwhile to include two beta distributions (one beta distribution of P_sample using the parameters (alpha_ns, beta_ns) for non-sensitive wells, and another distribution of P_sample using the parameters (alpha_s, beta_s) for sensitive wells). The data needed to do such an analysis are not available (noted in Chapter 5 of EA, p. 5-94, lines 45 - 46); although this data limitation was not made explicit in Chapter 4. Most of the surveys do not reliably classify wells as either sensitive or non- sensitive (noted in Chapter 5, p. 5-94, line 52). Such identification would be required if the data are to be used as the reviewer suggests. Therefore, estimating separate hit rate distributions for sensitive and non-sensitive wells is not feasible.
Stedinger	1	I see no justification for the exponents on this page to be other than unity.	EPA used exponential values with 5 or more significant digits in the final EA, as well as the Model Systems Report, to be consistent with the treatment of data in other rules (e.g., Stage 2 Disinfection Byproduct Rule, and Long Term 2 Surface Water Treatment Rule) that have used the same formulas.
Stedinger	2	If the virus come in clumps, then many our dose-response relationship also relates to clumps, and we are correct.	EPA thanks the reviewer for the comment.
Stedinger	3	Exhibit 4.14 needs more information so I can evaluate precision. How many wells for each combination of system size and category? Does the data justify some many digits? Can we just as well have the same concentration for all wells servicing less than a million? Why not?	The details of the underlying data are provided in Exhibit B.18 in Appendix B.
Stedinger	4	P(sample) includes a detection rate. Often the recovery rate for these pathogens is not unity, and such failure to detect is included in P(sample). This should be mentioned in the paragraph starting "There are a number"	EPA has addressed recovery and related issues at the beginning of Section 4.3.2 under the heading "General Considerations for Interpreting Viral Occurrence Data" (including Uncertainty).
Stedinger	5	I am troubled that Pv(sample) and Pi(sample) are assumed to be independent of one another. I suspect, as E[Pv(well)]	EPA has found that even a small embellishment of the current model could not be supported by the data. EPA

and E[Pi(well)] showed strong correlation across wells, one should also see strong dependence among Pv(sample) and Pi(sample). AND this should be important to the economic analysis because it says we are likely to see indicate organisms at those sites where we more often see viruses showing up.

Stedinger	6	Mathematically it may be difficult to adopt a bivariate beta distribution, called a Diriclet distribution, with the desired joint distribution. But if one used marginal probit distributions, then one could add on parameter corresponding to the cross-correlation between the standard normal variates, and one would have the needed joint
		distribution of the Pv(sample) and Pi(sample) without much modeling complexity.
Cto dia ana	7	
Stedinger	7	Sorry to have anything but positive things to say, but this potentially important cross-correlation is not mentioned in
		the report, seems potentially important to the analysis, and could be included without great difficulty.
Stedinger	8	Page 4-50 non-informative priors: I think a uniform is a
21128	-	GOOD prior to adopt. But it is not the non-informative
		prior. The strictly non-informative lets and go to zero.
		Zellner and some others use $\alpha = \beta = \frac{1}{2}$. The analysis is fine: the justification is wrong.

. .

Stedinger 9 Correct notation: $dbeta(\pi \mid \alpha, \beta) = ...$ use a vertical line.

attempted an analysis with only one additional parameter (slightly more complex than our reported model and slightly less complex than the model suggested above) and found that the data were nearly noninformative with respect to this new parameter. EPA has added two tables (4.13b and 4.13c) to better illustrate the limited amount of data available. Exhibit 4.13b shows distributions of wells by numbers of virus assays and virus positives. Exhibit 4.13c shows distributions of wells by number of E. coli assays and E. coli positives. These two tables clearly show that the numbers of wells with multiple positive assays are very small.

See above response to Stedinger, Comment 5.

See above response to Stedinger, Comment 5.

Technically, each can be called non-informative. The Jefferies prior (alpha = beta = $\frac{1}{2}$) places greater mass in the intervals 0-0.1 and 0.9-1, than in other equally sized intervals, so can be regarded as informative. However, providing this level of detail would be too technical for the intended audience, and has negligible influence on the results.

Please note that either notation is correct. Therefore, EPA has chosen the one currently used in the EA and has made no further change. EPA's notation is that which is recognized by MathCAD, the software used for processing the WinBUGS output. Therefore, EPA has chosen the notation currently used in the EA and has made no further

change.

Stedinger	10	Page 4-51: I am away from the office and do not have Gelman et al. with me. It makes sense to parameterize the beta in terms of its mean, lets call it mu. and another parameter. Then please use that notation later, as in exhibit 4.19 which plots the the two means against each other, but lacks a notation to explain what is happening. Same trouble exhibit 4.22. The later discussion is confusing because of a lack of notation for the mean of the beta distributions, and here is where that notation should be introduced.	EPA agrees with the reviewer comments. The Agency has reviewed and updated the final GWR EA to ensure that text and exhibit titles refer to Psample means as appropriate.
Stedinger	11	But then what do you call the second parameter. Why not just the "sample size" = $\alpha + \beta$?	Although EPA agrees with the reviewer, EPA decided to remain consistent with the Gelman paper. No change was made to the final GWR EA.
Stedinger	12		n/a
Stedinger	13		n/a
Stedinger	14	I did not see what priors you used on the mean (uniform on [0,1] would be reasonable) and the sample size. This should have required some thought, and I only see here the phase "disperse uniform priors." What does that mean? And how would it apply to the inverse of the sample size?	Priors on means range from 0.01 to 0.4. When the range was broadened, MCMC algorithm sometimes selected extreme values for which likelihood could not be computed (i.e., underflow). Restricting to this range, the MCMC sample stayed well away from the limits, so the prior does not appear to have influenced the posterior; this kept the algorithm from crashing.
Stedinger	15	Page 4-52 – top - Were the 10,000 samples all different or did they include repeats. IF the proposal ldistribution is rejected, then the same parameters are repeated. For low acceptance rates such as 10-20%, 10,000 samples may represent only 1,000-2,000 different values!	There were no repeats within the 10,000 samples. The set of 10,000 was narrowed from a larger set of 500,000 samples. Only every 50th set of values was saved. Later, this set was narrowed again by factor-of-ten; sampling every 500th set effectively removed autocorrelation.
Stedinger	16	Page 4-55: The discussion gets very confusing here because you fail to use the notation that was introduced on page 4- 51. Define the parameters of the beta distribution to be the mean and a sample size (or the median of a tobit distribution) and then talk about the mean here. The failure to have a notation to describe what is being discussed makes for a very unclear discussion. And tell me something about	The final GWR EA includes two new Exhibits (4.20 and 4.21) with explanations of the shapes of the Psample distributions, which addresses the variance issue. EPA has also provided additional clarifying discussion of the precision parameters.

Stedinger	17	the variance parameter, not just the mean of the betas (exhibit 4.22). I am not sure that exhibit 4-56 makes sense because the percentile are defined in terms of the mean, but the distribution showed reflect the value of two parameters. So I am not sure at all what is happening with the precision parameter of the beta distribution. Exhibit 4.22 and 4.23 are nice, particularly together. But they tell me nothing about the precision parameters. How about a scatter plot of the means and the precision parameter? I would like to know something about the precision parameter.	The shape of the distribution curves provides an indication of the range of parameters. "Bathtub-shaped" curves are cases in which sample size is small (less than 1). Curves with clear non-zero modes are cases in which sample size is larger (greater than 2). See also Exhibits 4.20 and 4.21 and their accompanying text for further discussion of distribution shapes.
Stedinger	18	Exhibit 4.23 – what is correlation between the two parameters?	See above response to Stedinger, Comment 5. The correlation coefficient is 0.27 (Pearson's r, based on MCMC sample of size 10,000).
Stedinger	19	Page 4-58 First sentence: It is the MEAN of P(sample) versus P(well)!	EPA has edited the text to indicate that these are Psample means.
Stedinger	20	Page 4-59 These are the MEAN of P(sample) versus P(well)! The figure titles and the axis labels are wrong. Use the notation from page 4-51.	EPA has corrected this typographical error.
Stedinger	21	Page 4-60 Something went wrong here. The equation on this page makes no sense, and the description of the probability analysis makes little sense. I cannot figure out what F(n,i) should be, or how it was computed. The probability the first success occurs on the rth trial is simple a geometric distribution given the probability of a success. But I cannot figure out what was done here.	EPA has provided an expanded discussion of this equation to clarify the parameter definitions, and to explain complexities introduced when integrating over the beta- distributed Psample.
Stedinger	22	The equation with the four gamma functions simplifies easily, but I cannot figure out what it might correspond to. I do not know what exhibit 4.26 is about. And the equation on page 4-61 seems to be important, but, again, I cannot figure out what it is doing. I would think for each well one generates a single Pi(sample). Then the number of trials until one gets a positive is a geometric distribution, whose CDF goes to one (not like Exhibit 4.26 which does not go to	Exhibit 4.26 considers the total population of wells ("All Wells"), including those with no indicator occurrence, and shows the cumulative probability of having an indicator on or before the indicator assay number. The equation converts the fraction of wells with indicator occurrence to fraction of all wells. EPA has provided further clarifying discussion in the final GWR EA, including the integrals behind the gamma functions.

Stedinger	23	one). One could then average these geometric distritubtions across wells. That is what exhibit 4.26 should be. Or does it also include the probability at well would ever generate a positive? *** Page 4-63 To assume the distributions of viruses that you have not seen is the same as those you have seen seems unreasonable. We need some real data. Some data or justification seems to be required. How does the occurance of the viruses in the human population (and thus the source) differ? Does the distribution matter to the economic analysis?	EPA thanks the reviewer for the comment; however, there is no relevant information on the distribution of these viruses in humans. Because of these data limitations, it is necessary to assume that what has been observed is representative of other wells.
Stedinger	24	*** Exhibit 4.30 The values in this exhibit are just very different than those in Exhibit 4.29. EPA should not mix these non-community wells with those in 4.29.	EPA thanks the reviewer for the comment. However, EPA has not made any changes to the final GWR EA based on this comment, because the alternate analysis treats noncommunity wells separately with respect to virus concentration estimates.
Stedinger	25	 Page 4-66 I think it is just fine for EPA to use the empirical distribution of the observed values, after the Pennsylvania non-community values are separated from the Awwarf/AWWASCo study. But the analysis should not some concerns. *** One can no longer address uncertainty in the distribution parameters when one uses an empirical distribution, instead of a parametric distribution that has parameters. ***The values in Exhibits 4.28 – 4.30 ARE NOT CONCENTRATIONS. THEY ARE ESTIMATES OF CONCENTRATIONS. Those estimates have large variation (error), and thus collectively these estimates have greater variance that the real concerntations do. As an example of this kind of problem see: Reis, D. S., Jr., J. R. Stedinger, and E. S. Martins, Bayesian generalized least squares regression with application to log Pearson type 3 regional skew estimation, Water Resour. Res., 41, W10419, doi:10.1029/2004WR003445, 2005. 	EPA has added a clarifying discussion in the final GWR EA regarding trade-offs between the uncertainty of using "bootstrap," and the uncertainty of using a distribution where data are a poor fit. In addition, EPA has ensured that the final GWR EA clearly indicates that these are concentration estimates, and not actual concentrations, as the reviewer has pointed out.

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Stedinger	26	Page 4-67 Concentration data is NOT available. Estimates of concentration data is available.	EPA has ensured that the final GWR EA clearly indicates that these are concentration estimates, and not actual concentrations, as the reviewer has pointed out.
Stedinger	27	Page 4-68 To animals not have viruses to which humans are susceptible? LINE (32) provies insufficient data TO EXPLORE ALL OF THESE RELATIONSHIPS.	EPA thanks the reviewer for the comment. As discussed in Chapter 5, EPA has focused its risk and benefits analysis on human pathogenic viruses and used two virus types (Type A and Type B) to represent the range of pathogens to which humans are exposed from ground water.
Stedinger	28	Pagge 4-69 Why bother for a factor of 2. It seems that a much LARGER difference is to be expected.	EPA thanks the reviewer for the comment.
Stedinger	29	 Page 4-73 (1):I agree with EPA that I am concerned about the failure to better link the occurrence of indicator organism and viruses. I suggested that this be done in the distribution of P(sample) in my discussion above. (2):I agree with EPA that a better model of the importance of sensitive and nonsensitive wells should be established. The conventional wisdom and I had heard it was that if a well was protects why hundreds of feet of fine soil, then no contamination of the well should occur. That is why my comment that a factor of two in the analysis was almost nothing. It is likely to be a difference between no contamination being possible, and contamination being likely. This does not mean that a well owner will always realize a well is sensitive. 	As noted above in response to reviewer comments on Chapter 4, most of the surveys do not reliably classify wells as either sensitive or non-sensitive. Therefore, data are insufficient to stratify the occurrence analysis by sensitive and non-sensitive wells.
Stedinger	30	2.1: This seemed reasonable. None should be empty. The data is allowed to determine the appropriate probabilities. Unfortunately the indicator organisms are not perfect in groundwater.	EPA thanks the reviewer for the comment.
Stedinger	31	2.2:We do not have enough data, and given that limitation this assumption is reasonable. However, it might be possible to fit a separater beta distribution for each of the two cases, and this would be a reasonable option to explore. The problem is that there may not be enough cases of virus without indicator organisms. And in this case, the data that drive the fit of the beta distribution are in fact mostly the	See above response to Stedinger, Comment 5.

case of interest.

Stedinger 32 2.3: This seems fine to me. The distribution of the observed values has as a mean the mean of the measurements, and this is what is critical for health assessment. The actual distribution of the values does not seem to be critical. It is the mean number of virsuses that determines how many illnesses occur (right? I do not have the health chapter.).

But the analysis should recognize these are not concentrations, but estimates of concentrations. Thus the measurement process may underestimate concentrations, and certainly is noisy.

Stedinger 33 2.4: I agree with EPA that a better model of the importance of sensitive and non-sensitive wells should be established. The conventional wisdom and I had heard it was that if a well was protects why hundreds of feet of fine soil, then no contamination of the well should occur. That is why my comment that a factor of two in the analysis was almost nothing. It is likely to be a difference between no contamination being possible, and contamination being likely. This does not mean that a well owner will always realize a well is sensitive.

I agree with EPA that I am concerned about the failure to better link the occurrence of indicator organism and viruses. I suggested that this be done in the distribution of P(sample) in my discussion above. This error will distort the value of monitoring efforts because monitoring will be more effective at identifying site with high viral loads. I would encourage EPA to address this issue. EPA thanks the reviewer for the comment and has ensured that the final GWR EA clearly indicates that these are concentration estimates, and not actual concentrations, as the reviewer has pointed out.

As noted above in response to reviewer comments on Chapter 4, most of the surveys do not reliably classify wells as either sensitive or non-sensitive. Therefore, data are insufficient to stratify the occurrence analysis by sensitive and non-sensitive wells.