Salmonella Framework for Raw Poultry Products Critical Review

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Executive Summary

The Healthy People 2030 target is to reduce the *Salmonella* illness national case rate by 25 percent. Because poultry, specifically chicken and turkey, are potentially important sources of human salmonellosis illnesses, USDA FSIS has introduced a new proposed *Salmonella* Framework with the goal of reducing salmonellosis illnesses attributable to chicken and turkey by 25%. The proposed changes include a Final Product Standard that states "Raw chicken carcasses, chicken parts, comminuted chicken, and comminuted turkey are adulterated if: 1) They contain any type of *Salmonella* ≥ 10 cfu/mL or 10 cfu/g **AND** 2) Contain any detectable level of at least one of the *Salmonella* serotypes of public health significance identified for that commodity." The proposed *Salmonella* framework also includes a Statistical Process Control (SPC) program with the goal of reducing Aerobic Counts (AC) by at least one log between two sampling points in the processing plant.

The risk assessments that were conducted for this Framework demonstrate that the maximum predicted reduction in human salmonellosis illnesses following implementation of the proposed Final Product Standard is far below the HP2030 target of 25%. Even with assumptions that tend to maximize the potential decrease in human illness, this burdensome and precedent-setting program would have little noticeable impact on human salmonellosis. Some of the key deficiencies of this proposal are summarized below and are described in more detail later in this document.

In the Federal Register Notice (FRN) published August 7, 2024, four risk management questions are posed. The second risk management question asks: What is the public health impact (change in illnesses, hospitalizations, and deaths) achieved by eliminating final product contaminated with specific levels of *Salmonella* and/or specific *Salmonella* subtypes? Even though the proposed Final Product Standard includes both of these criteria (enumeration threshold and serotypes of public health significance criteria), surprisingly the risk assessments never address both of these criteria together, and therefore, all of the estimated illnesses prevented are based on an incomplete application of the proposed Final Product Standard. **It is unfathomable that USDA FSIS would propose a precedent-setting Final Product Standard on raw, not ready-to-eat (NRTE) poultry without actually evaluating the criteria of this standard.**

Another surprising deficiency in this proposed Framework is the lack of serotype-specific data for the products being considered. Specifically, serotype data were only available from the FSIS two-point chicken carcass data (rehang and post-chill); no data were collected for chicken parts, comminuted chicken or comminuted turkey, and therefore, no serotype analyses could be conducted for these products even though they are components of the proposed Final Product Standard.

I commend FSIS for undertaking this task, as there is a need to update the Performance Standards for chicken and turkey. The various models that were built have helped identify key data gaps that should be filled to more accurately estimate the efficacy of policy changes. Unfortunately, there are a significant number of data gaps in these models that limit the utility and predictive capability of the models.

Summary Points

- The proposed Final Product Standard represents perhaps the most significant change to meat inspection since HACCP. It is not analogous to STEC in ground beef or *Salmonella* in raw, not ready-to-eat not ready-to-eat (NRTE) breaded stuffed chicken. One would hope and expect that prior to implementing a regulatory change as significant as this, models would be developed to predict the potential benefit to human health and the impacts on the poultry industry. Risk assessments were developed for chicken and turkey, and the number of illnesses prevented was estimated. However, the risk assessments did not address both the enumeration-based criterion and the serotypes of public health significance criterion of the proposed Final Product Standard in the same model. It is entirely inappropriate to implement a Final Product Standard without evaluating the impact that the criteria for the standard will have on the industry and public health.
- The estimated number of illnesses prevented is inflated for chicken. The model multiplies the proportion of illnesses prevented for carcasses by all illnesses attributed to chicken and not just the number attributed to chicken carcasses. FSIS states in the chicken risk assessment that the estimated number of illnesses prevented attributed to chicken carcasses, chicken parts, and comminuted chicken are not additive, and yet the second Table 34 in the FRN sums the three products to arrive at an estimated 2,200 illnesses prevented following implementation of the enumeration-based Final Product Standard in chicken. However, if FSIS had done this calculation correctly, the numbers would be approximately 138 for carcass, 200 for parts and 1,000 for comminuted chicken, for a total of 1,338 estimated illnesses prevented. This number is much lower than the stated 2,200 as reported in Table 34.
- The risk assessments assume perfect accuracy in the diagnostic assay used to enumerate *Salmonella*. As shown in the FSIS analysis, the enumeration assay evaluated by FSIS has many false positives and false negatives, especially when the true *Salmonella* concentration is near the threshold of 10 cfu/mL(g). The impact of an imperfect diagnostic assay is an increased cost to industry (due to false positives) and a reduced benefit to public health outcomes (due to false negatives). Failing to incorporate the assay performance into the model results in fewer illnesses prevented than was predicted, perhaps by 25% or more.
- The estimated number of annual illnesses attributed to chicken and turkey in the U.S. uses an outdated under-diagnosis factor that does not account for inter-serotype (or inter-strain) differences in virulence. The *Salmonella* Framework is based heavily on the notion that there is heterogeneity in virulence among serotypes (and strains). One would expect virulent strains to cause a more severe disease, thus reducing the under-diagnosis multiplier for these virulent infections.
- The models assume that the number of illnesses attributable to specific chicken and turkey products is proportional to the number of servings of each product consumed during the year. This assumption of equal risk among different products is overly simplistic and highlights the lack of product-specific data that should have been collected (or estimated) prior to proposing a Final Product Standard.
- The virulence model for categorizing *Salmonella* serotypes, which represents a major advance in *Salmonella* bioinformatic analyses, is overly simplistic and does nothing to help reduce the impact of specific *Salmonella* serotypes derived from poultry on human health outcomes. The model treats all known and hypothetical virulence genes (factors)

equally and uses a simple cluster algorithm that labels some highly virulent serotypes as "low virulence." The model assumes that all serotypes within a cluster have equal virulence. The model assumes that a serotype found in multiple host species has equal virulence across species, implying no strain variation across species. The model actually includes more than 10,000 beef-sourced isolates in the cluster algorithm. For reasons that will be discussed below, including serocluster (and serotypes of public health significance) in the Final Product Standard criteria **reduces** the public health impact of the proposed Framework. Consequently, a better program would eliminate the serotype component of the criteria and instead focus on the enumeration-based criterion.

- The contamination distribution is used to model the initial contamination of products being regulated in this Framework. Strangely, all three chicken products (carcasses, parts and comminuted chicken) are modeled with the same distribution, even though FSIS data show that these product types have very different levels of contamination. Further, the single contamination distribution for chicken is based on a weighting of the product-specific contamination distributions, with the weighting based on the proportion of servings of each product consumed during the year (similar to the product-specific illness attribution).
- The attenuation distribution used by FSIS, which explains the growth or die-off of *Salmonella* on the product as it moves from the processing plant to the point of consumption, is the same for all chicken and turkey products. This inherently assumes that all of these products behave identically, including the likelihood that they will be mishandled and/or undercooked by the consumer.
- The dose-response function for the high virulence serotypes is based on non-poultry data for two serotypes. The final function is applied to all serotypes in the high virulence cluster, regardless of strain (and actual virulence potential) within the serotype.
- The Risk Multiplier is a biased parameter of the model, as the numerator is a surrogate for virulence of serotypes in the cluster while the denominator is a surrogate for the frequency that the serotypes are found in poultry samples in the processing plant. This confounded measure is used estimate the dose-response function of the "low virulence cluster" by reducing the dose-response function of the "high virulence cluster." This parameter, which does not actually relate to virulence at all, has no place being used to adjust a dose-response function, especially when the dose-response function for both the high and low virulence clusters could have been derived in the same way.
- The Risk Multiplier assumes that all serotypes in the cluster have an identical virulence, and further, that all strains within a serotype also have an identical virulence. The result of using the Risk Multiplier to adjust the dose-response function for the low virulence serotypes results in an inflated number of estimated illnesses that can be prevented following the implementation of the enumeration-based Final Product Standard.
- In the turkey risk assessment presented by FSIS in this Framework, data to inform multiple parameters were lacking, and consequently, chicken data were used to estimate these parameters in the turkey model. No uncertainty adjustment seems to have been included in the turkey model to account for the use of chicken data. It is inappropriate to use chicken data to estimate parameters in the turkey risk assessment, especially for a proposed regulatory change as significant as this. The agency or others should have collected the necessary data for the models prior to publishing the final *Salmonella* Framework.

Alternative Program

For reasons discussed below, a Final Product Standard is not the best way forward, at least at this time. For more than two decades, USDA-FSIS has been fixated on prevalence (presence-absence) as an indicator of risk. As I described in my comment posted to FSIS Docket No. FSIS-2022-0029 in December 2022, prevalence is not an indicator of risk, and therefore, the lack of correlation between the reduction in *Salmonella* prevalence across all poultry products and the stable incidence of human salmonellosis can be attributed to the agency targeting an inaccurate metric of risk. Rather than implementing a Final Product Standard that was never fully evaluated and, even under risk-maximizing assumptions, will fail to achieve the desired 25% reduction in human salmonellosis illnesses attributed to raw, NRTE poultry, I propose an alternative program. Specifically, FSIS should set an enumeration-based Performance Standard (such as the 10 cfu/mL or cfu/g on the products described in the Framework) of **any** *Salmonella*, not just the serotypes of public health significance identified by the models. Second, to ensure that microbiological controls in the processing plant are functioning, a proper SPC could be implemented upstream of the final product (for example, at rehang and post-chill). The combination of these two interventions will likely have a much greater impact on human salmonellosis than the program described in the Framework.

In Table 37 below, which was taken from the FRN (p. 64743), five regulatory alternatives are presented. These alternatives either maintain the status quo or make changes to the enumeration threshold for the Final Product Standard. FSIS should consider my alternative that uses SPC and an enumeration-based Performance Standard to reduce human salmonellosis attributable to poultry.

TABLE 37—REGULATORY ALTERNATIVES

Alternative [1]	Costs (medium estimate)	Benefits (medium estimate) [2]	Net (medium estimate)
1: No regulatory action (Baseline)	Continued illnesses and deaths associated with *Salmonella* from these products.	No new costs to industry	n/a.
2: The proposed rule and proposed determination.	$16.43 million compared to the baseline	$20.49 million from prevented *Salmonella* illnesses and outbreak-related recalls.	$4.06 million.
3: The proposed rule and proposed determination with a lower level for adulterated product (1 cfu/mL(g) and serotypes of public health significance).	$29.52 million compared to the baseline	$19.65 million from prevented *Salmonella* illnesses and outbreak-related recalls.	($9.88) million.
4: The proposed rule and proposed determination with a higher level for adulterated product (100 cfu/mL(g) and serotypes of public health significance).	$15.34 million compared to the baseline	$8.85 million in the form of prevented *Salmonella* illnesses and outbreak-related recalls.	($6.59 million).
5: The proposed rule and proposed determination with a lower contamination level for adulterated product of 1 cfu/mL(g) *Salmonella* regardless of serotype.	$49.96 million compared to the baseline	$34.50 million from prevented *Salmonella* illnesses and outbreak-related recalls.	($15.45 million).

[1] Costs and benefits are annualized at a 7 percent discount rate over 10 years.
[2] Alternatives 2–5 have additional potential benefits from reduced risk of outbreak-related recalls and increased consumer trust.
Note: Numbers in table may not sum to totals due to rounding.

Justification for this Performance Standard approach is clearly stated in the documents released by FSIS as part of this *Salmonella* Framework proposal. For example, in the Chicken *Salmonella* Risk Assessment (SRA), FSIS states that "Public dissemination of establishment categorization has been shown to serve as a market-based incentive to encourage establishments to reduce

Salmonella contamination in failing establishments" (Chicken SRA, p. 39 and Ollinger, 2020). FSIS later states that:

> there is no mandatory enforcement action once an establishment fails a performance standard. Risk assessments of performance standards attribute improvements in public health to the actions taken by failing establishments to become passing establishments (FSIS, 2015). Those assessments assume that the motivation for improvement is via market forces that penalize failing establishments and/or reward passing establishments. Therefore, failing establishments and public health improve as an indirect effect of the performance standards (Chicken SRA, p. 126).

As seen from every prior change to the poultry Performance Standards, the industry adapts to these changes. Implementing an enumeration-based Performance Standard will push the companies to meet/exceed that Performance Standard during all production shifts and not just those on FSIS sampling days. A change in the poultry Performance Standards is the logical next step and is supported by past evidence of efficacy.

Introduction

As stated in the *Salmonella* Framework FRN, "The results of FSIS' *Salmonella* verification sampling show that the current prevalence-based performance standards approach has been effective in reducing *Salmonella* contamination in poultry. However, these measures have yet to have an observable impact on *Salmonella* illnesses." (p. 64683). This is an important admission by FSIS for several reasons. First, it implies that the poultry industry has adjusted and adapted to every change that FSIS has made to the Performance Standards over the past two decades. In other words, the voluntary Performance Standards enacted by FSIS have resulted in substantive change by the poultry industry. The observation that human illness rates have not changed is not a statement against the poultry industry but rather that FSIS was not focused on predictors of actual risk to human health. Second, the statement implies that all human salmonellosis illnesses should have declined because of the actions of the poultry industry. In reality, only those illnesses that are attributed to poultry products would have been prevented, and these estimates are based on uncertain calculations of attribution. A recent paper published in the journal Risk Analysis in 2024 found "declining trends in illness due to the poultry-associated serotypes and increasing trends in illness due to *Salmonella* serotypes not associated with poultry." (Powell, 2024). Finally, this statement by FSIS implies that the current proposed *Salmonella* Framework would have an observable impact on human salmonellosis rates. "The Healthy People 2030 target is to reduce the *Salmonella* illness national case rate of 15.3 per 100,000 population in 2016–2018 by 25 percent, or to no more than 11.5 per 100,000 population per year. Thus, to reach the 2030 target, illnesses must be reduced by 25 percent" (p. 64683). As clearly reported in the chicken and turkey SRAs and in the FRN, the best guess of the direct impact of this proposed *Salmonella* Framework would reduce human illnesses very little; the estimated percentage reduction in poultry-attributed illnesses is actually 1.99% (3,338 poultry illnesses prevented / 167,784 poultry-attributed illnesses; this number of illnesses prevented is different from Table 5 of the FRN due to an error by FSIS, as described below). This translates into a reduction in the overall *Salmonella* illness rate of 0.46% (3,338 / 723,207 total salmonellosis illnesses). Both of these decreases are far below the targeted 25% reduction.

Table 5 of the FRN shows the estimated number of illnesses prevented following implementation of an enumeration-based Final Product Standard; the estimates do not include the serotype of public health significance criterion of the Final Product Standard, as the risk assessments do not model both criteria of the proposed Final Product Standard in the same model. According to the chicken SRA, "At a level threshold of 10 cfu/mL, the number of illnesses prevented is essentially zero" (Chicken SRA, p. 104). This level (10 cfu/mL(g)) was chosen for multiple reasons, but as stated in the FRN:

> The resulting overlapping 95 percent credible intervals around the estimated number of illnesses prevented suggest that there is little meaningful difference in effectiveness between the threshold standards with respect to annual illnesses prevented. However, as discussed above, when compared with the majority of servings, chicken carcasses, chicken parts, comminuted chicken, and comminuted turkey that contain *Salmonella* at 10 cfu/mL(g) or higher present a much higher probability of illness. Thus, based on the elevated probability of illness associated with raw chicken carcasses, chicken parts, comminuted chicken, and comminuted turkey associated with *Salmonella* levels at or above 10 cfu/ mL(g), FSIS is

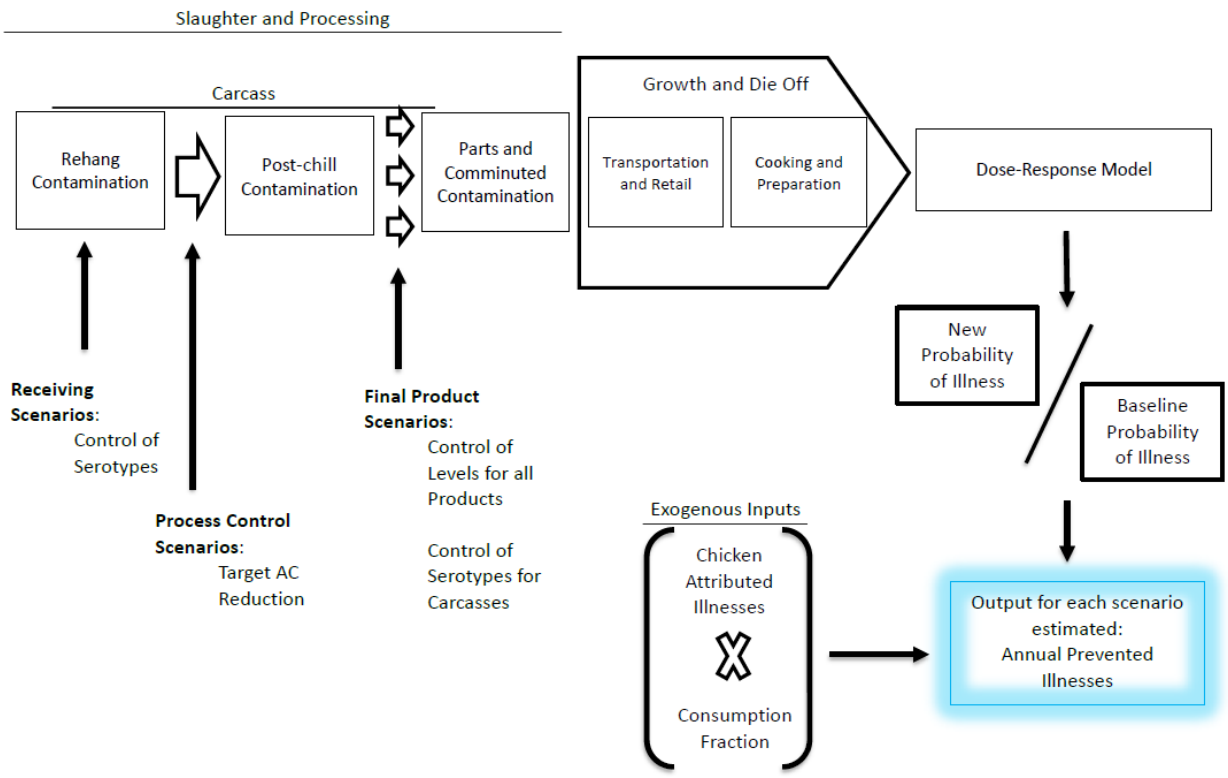
proposing 10 cfu/mL(g) as the *Salmonella* level for the proposed final product standards. (FRN, p. 64703).

TABLE 5—ANNUAL ILLNESSES PREVENTED, MOST LIKELY
[95% Credible Interval]

Threshold level	Chicken carcasses	Chicken parts	Comminuted chicken	Comminuted turkey
0.03 cfu/mL(g)	4600 (2000, 7100)	7900 (3300, 12700)	1500 (800, 2200)	2500 (700, 4900)
1 cfu/mL(g)	2400 (700, 5000)	1400 (400, 3600)	1400 (600, 2100)	2300 (600, 4800)
10 cfu/mL(g)	1000 (200, 3100)	200 (40, 700)	1000 (400, 1900)	2000 (500, 4300)
100 cfu/mL(g)	200 (0, 1500)	20 (0, 100)	600 (200, 1500)	1400 (200, 3500)

According to the FRN, "the current performance standards do not distinguish between poultry products that are heavily contaminated and that contain the most virulent type of *Salmonella* from those that contain trace amounts of a *Salmonella* with types not typically associated with foodborne illnesses in the United States" (FRN, p. 64692). Consequently, FSIS states that "the Agency has tentatively decided to phase out all current *Salmonella* performance standards for poultry" (FRN, p. 64680). While FSIS is correct that the current Performance Standards are based on prevalence and therefore do not distinguish high-load final products that might present an elevated risk to the consumer, that is not justification for saying that Performance Standards should be phased out. Instead, a new, enumeration-based Performance Standard should be created. The impact of such a program would be much greater than a Final Product Standard that is based on testing of, at most, a single lot per week. Furthermore, although the serotype and virulence profile of the *Salmonella* on a product affect the likelihood of illness to the consumer, an enumeration-based standard without the serotype criterion will have a greater impact on public health because it will target all serotypes. In other words, using an enumeration-based standard, regardless of type of *Salmonella* present, will keep all *Salmonella* at these "trace amounts" thereby imparting maximum risk reduction.

As stated in the Chicken SRA, "The amount of *Salmonella* on chicken carcasses regulated by FSIS has decreased over time. Comparison of the FSIS 2022 Exploratory Sampling post-chill data to the previous FSIS chicken carcass microbiological baseline (FSIS, 2009a) shows a 59% reduction in volume-weighted *Salmonella* prevalence (from 0.075 in 2009 down to 0.031 in 2022)." (Chicken SRA, p. 23). This lack of efficacy in reducing human illnesses should raise the key question: Is there an alternative program that would have a greater impact at reducing the human salmonellosis illness rate attributed to poultry products? The answer is "Yes." In this report, I will try to highlight some of the problems with the current proposed Framework that also support the program that I proposed above that would ultimately provide a much greater impact than this *Salmonella* Framework. I will comment on various parameters used in the SRAs. I order my comments using the schematic of the overall Framework structure (below) as a guide. I first comment attribution and estimated number of illnesses attributed to chicken and turkey. I then discuss Component 3 issues, which include the enumeration-based criterion, the serotype of public health significance criterion, the contamination distribution, the attenuation distribution, the dose-response model, and the risk multiplier. I then comment on Component 2, and specifically, the use of SPC. Finally, I comment briefly on Component 1, even though it is not currently being proposed for implementation by FSIS.



Attribution

The first step of the risk assessment approach is to estimate the number of human salmonellosis illnesses that are attributable to chicken and turkey produced and consumed in the U.S. The SRAs describe how the number of annual illnesses attributable to chicken and turkey was estimated. There were an estimated 125,115 chicken-associated and 42,669 turkey-associated *Salmonella* illnesses per year. As stated in the SRAs, this value is calculated as the product of the total number of CDC FoodNet cases per year (7,600), the share of these cases that are foodborne (66%) and of domestic origin (89%), the under-diagnosis multiplier for *Salmonella* (24.3) (Ebel, 2012c), dividing by the FoodNet catchment area (15%), and multiplying by the IFSAC attribution estimates to chicken (17.3%) or turkey (5.9%).

Given that the focus of the SRA outputs is to estimate the number of annual illnesses that can be prevented through the proposed regulatory changes, the percentage reduction in illness incidence is likely more important than the absolute number. Regardless, if the percentage of human salmonellosis cases attributable to chicken and turkey is less than the estimate used in the calculation, the actual impact on human health as a result of this proposed *Salmonella* Framework will be less than reported. These IFSAC attribution estimates are based on outbreak data and are therefore influenced by the decision by public health agencies such as the CDC to declare an outbreak or to consider an outbreak over. This will be discussed later in the report but becomes important, as many salmonellosis illnesses are considered sporadic (not linked to an outbreak) and thus do not help inform attribution estimates.

Aside from the IFSAC attribution estimates, another important parameter used in the illness estimate calculation is the under-diagnosis multiplier. This is an outdated estimate (revised by Scallan et al. 2011 and Ebel et al., 2012) and does not differentiate among serotypes or strains within serotypes; a single under-diagnosis value is used for all serotypes and strains, regardless of virulence. Given that so much of this *Salmonella* Framework is devoted to differentiating high virulence from lower virulence serotypes, it is unfortunate that more attention was not given to this parameter. One would expect virulent strains to cause a more severe disease, thus reducing the under-diagnosis multiplier for these virulent infections.

Another component to attribution estimates that is important in the *Salmonella* Framework is to attribute the illnesses to the specific chicken and turkey products being regulated in the Framework. In the chicken SRA, the estimated human salmonellosis illnesses attributed to chicken "are distributed across products by assuming the proportion of servings consumed (0.11, 0.83 and 0.06) is proportional to illnesses resulting from exposure to carcasses (whole chickens), parts and comminuted (ground) forms of chicken, respectively" (Chicken SRA p. 86). For turkey, the SRA assumed that "0.42 of all turkey-associated *Salmonella* illnesses result from exposure to comminuted (ground) turkey products, which is approximately 17,921 (Lambertini, 2021)." (Turkey SRA, p. 86). This 42% estimate by Lambertini comes from the CDC National Health and Nutrition Examination Surveys (NHANES) conducted 2013 – 2014, more than a decade ago. Regardless, attributing product-specific illnesses proportional to servings consumed assumes that all products are equally likely to cause disease, an assumption that is unlikely to be accurate. As will be seen with other parameters of the SRA models, there is considerable missing data relative to product-specific characteristics. Before proposing a Final Product Standard on these products, one would hope that these product-specific data gaps would have been filled.

Serocluster Assignment

In this *Salmonella* Framework, FSIS sought to categorize *Salmonella* serotypes by their virulence, meaning their ability to cause severe disease in people. Part of the reason for doing this was to identify serotypes of public health significance that would then become part of the Final Product Standard criteria. These virulence cluster assignments would also be used in the risk assessments to predict the number of illnesses that could be prevented through a reduction of high virulence *Salmonella* serotypes on chicken and turkey products. I commend FSIS and EpiX Analytics for using a scientific approach to categorize serotypes based on their potential virulence. However, the model that was built for this cluster assignment is flawed for various reasons, at least with respect to its application in the *Salmonella* Framework. However, as I discuss throughout this report, I do not believe that categorizing serotypes into virulence clusters is necessary, as a regulatory change that would have the biggest impact on public health would ignore serotypes and instead focus only on the enumeration-based threshold. In other words, all *Salmonella* serotypes would be targeted and not just the three identified as significant to public health for each commodity.

To separate serotypes into seroclusters, EpiX Analytics downloaded *Salmonella* genomes from sources including chicken, turkey, human and beef. The serocluster model was first published in PLoS ONE as a beef model (Fenske et al., 2023). In total, 40,038 *S. enterica* isolates of different

serotypes from these four sources were used in the model. Each genome was evaluated for the presence of known or hypothetical virulence genes, many of which have only been described in *Enterobacteriaceae* other than *Salmonella*. In total, approximately 5,000 genes were evaluated. Random forest models were created based on the presence/absence of these genes. No information was provided to the model regarding the importance of specific virulence genes in causing severe human illness. Many of the genes used in the models have not been proven to actually be *Salmonella* virulence factors (only about 150 virulence genes have evidence as being virulence factors in *Salmonella*). Therefore, the presence/absence of the virulence genes evaluated in these models may not be reflective of a strain's virulence potential. The chicken SRA specifically states "Moreover, clustering was agnostic to the biological function or role of individual virulence factors as well as point mutations or insertions/deletions of genes that can modify gene function resulting in public health risk as illustrated by the emergence of *Salmonella* Reading (Miller, 2020)" (Chicken SRA 53). I am a co-author on the Miller et al. paper and can attest to the fact that not all strains within a serotype have equal virulence, and the array of specific virulence factors (not just the total number) influences a strain's virulence.

The documents then state that "Virulence genes that were present in the majority of isolates (>95%) as well as limited gene presentation (i.e., <10 total isolates) were removed from further analysis. Hence, 193 genes available for the clustering analysis included 57 *Salmonella* VFs, 94 *E. coli* VFs, 10 *Shigella* VFs, and 32 *Yersinia* VFs. 53" (Chicken SRA, p. 53). When the clusters that were generated based on this simplistic approach are compared to an actual phylogenetic tree of *Salmonella enterica* (based on SNPs), the trees are nearly identical, showing that the cluster assignments did not accurately characterize individual serotype or individual strain virulence differences. Experimental evidence for actual invasive potential does not agree with the serocluster tree. For example, there are highly invasive serotypes falling into the "low virulence" cluster, and vice versa. As we published in the Miller et al. paper (2020), there are also major virulence differences among strains within a serotype.

Ultimately, FSIS decided to use the two-cluster model, one being labeled as "high virulence" and the other as "low virulence." Strangely, the FRN says that "The committee [NACMCF] also stated that these data show that a small number of serotypes account for most poultry-associated salmonellosis led by Enteritidis, Typhimurium, I:4,5,12:i:-, Infantis, and Heidelberg" (FRN, p. 64695). If these serotypes are poultry-associated and are associated with the highest number of illnesses, it should be an indicator that the serocluster assignment, which put Infantis and Heidelberg into the low virulence cluster, is not performing properly. FSIS states that the "clusters were validated by linking them to epidemiological data (i.e., documented outbreaks attributed to poultry sources with consideration of prevalence in animal sources from FSIS poultry sampling programs)" (Chicken SRA, p. 55). This is not validation of the model, as shown above where Infantis and Heidelberg were identified by NACMCF as being important poultry serotypes that cause human illness but that were included in the low virulence cluster. These cluster assignments were later used in the SRAs to estimate the Risk Multiplier parameter of the models, which is basically the step that FSIS says is the validation.

Furthermore, different host species can harbor different strains of the same *Salmonella* serotypes. This is important because the models developed by EpiX Analytics incorporated over 10,000 beef *Salmonella* isolates into the models. It is unknown how strongly these beef isolates altered

the cluster assignments for different *Salmonella* serotypes, as it does not appear an analysis was conducted without these isolates. According to the downloadable FSIS Bioinformatic spreadsheet, "*Salmonella* virulence does not depend on host or origin (i.e., isolation source such as chicken, turkey, beef, etc.)." This assumption is untrue, as there are considerable intra-serotype inter-strain differences in virulence, and some of this heterogeneity can be clustered by source.

Because I argue in this report that the use of *Salmonella* serotypes in the proposed regulatory changes actually reduces the public health impact of the program, it might appear that there is no need to belabor the serocluster assignment discussion. Also, as I have stated previously, the criterion of the proposed Final Product Standard involving serotypes of public health significance was not evaluated in concert with the enumeration threshold in the SRAs. However, the serocluster assignment does get used in the models in important ways, and thus it is critical to highlight the assumptions and weaknesses of the serocluster models.

For example, the arbitrary decision to divide the isolates into two clusters defined as high and low virulence then gets used to generate the Risk Multipliers that will be discussed later in this report. These Risk Multipliers are used to modify the dose-response models and to estimate the potential number of illnesses that could be prevented. Determining which serotypes belong in which cluster thus has importance to the outcomes of the SRAs, and these estimates of virulence differences among the clusters could be highly driven by a small subset of serotypes within the cluster, by a strain variant within a serotype, or by host species differences in virulence traits of isolates within a serotype (i.e., beef versus poultry isolates of the same serotype). For example, if Typhimurium is by far the dominant serotype of cluster 1 isolates, the case rate and hospitalization rate will be driven by Typhimurium and not necessarily by other serotypes within the cluster.

FSIS repeatedly states that the dose-response model developed by EpiX Analytics was "genomically validated." This is a strange (and somewhat meaningless) statement, as the dose-response model uses serotype-specific estimates from published studies. The simplistic approach to clustering isolates based on an agnostic model of virulence factor presence/absence is not a validation of a dose-response model, as described below in the dose-response section. In fact, as described above, the serocluster model is extremely crude, using only two clusters that are unable to differentiate serotypes or strains within serotypes.

The serocluster modeling approach and subsequent use of the two cluster assignments in the SRAs includes many implicit and explicit assumptions. For example, as noted by FSIS, "all serotypes in each cluster are considered to be equally virulent for the purpose of this analysis" (Taken from Table 3 of the Chicken SRA). This is a clearly flawed assumption, as FSIS has identified serotypes Infantis, Enteritidis, and Typhimurium as Key Performance Indicators (KPIs), and yet Infantis is clustered with the low virulence serotypes by the model.

In the FRN, FSIS states that:

> the scientific evidence does not support that the rising trend in Infantis illnesses is associated with chicken consumption. The emergence of Infantis in FSIS chicken sampling in 2016 did not correspond to a proportional increase in human Infantis

> illnesses, which have been on the rise in the United States since 2010. Put another way, given the volume of chicken consumed by the American public—much of which is contaminated with Infantis—if it were a high-risk poultry serotype, we would predict more Infantis illnesses. Furthermore, the 2023 chicken risk assessment, which used published genomic methods, also determined that Infantis is less virulent than many other serotypes with the exception of Kentucky. (FRN, p. 64697).

This is a highly flawed assessment and is a clear indication that the serocluster model failed. As will be discussed later in the dose-response section, *S.* Infantis was modeled as having one of the lowest infectious doses. On the next page of the FRN, FSIS states "The median *Salmonella* dose predicted to result in 50 percent of exposed individuals becoming ill (IllD50) was 3,360 cfu (95 percent range: 18–3.2×10^{9}), 1,500 cfu (38–8.8×10^{7}), and 1 cfu (0.69–1.0×10^{6}) for Enteritidis, Typhimurium and Infantis, respectively" (FRN, p. 64698). From this statement, it should be apparent that there are strains of *S.* Infantis that have a very low infectious dose, demonstrating the high virulence of some of the *S.* Infantis strains. FSIS states "Given the notable concern of the *Salmonella* Infantis REPJFX01 strain raised by the CDC and other public health experts, FSIS is requesting comment on the possible inclusion of Infantis as a serotype of public health significance" (FRN, p. 64697). Given that the REPJFX01 strain is just one of many within the Infantis serotype, it would seem inappropriate to label an entire serotype as significant to public health. The serocluster model fails to incorporate intra-serotype strain differences, which are key to understanding virulence differences among isolates.

Enumeration-Based Standard

The schematic depiction of an enumeration-based standard is shown in the figure below, which is taken from Figure 22 of the Turkey SRA. Before the enumeration standard is applied, lots of product are either above or below this threshold. However, the true status of each lot is unknown. The fraction of lots that pass (ω) the standard can still get people sick, but the load of *Salmonella* in these lots will generally be lower than the lots that fail (1-ω). After the standard is applied, there will still be lots that are truly above or below the enumeration threshold (ω), but only those lots sampled and tested by FSIS and that exceed the threshold would be diverted (α). As can be seen in the formulas below, the serocluster assignment is used in the estimation of the number of illnesses; the dose-response section below shows how illness associated with the high virulence cluster (C1) is more likely at the same dose than the lower virulence cluster (C2).

As can be seen in this schematic, there is no parameter for diagnostic test accuracy. In other words, the model does not seem to account for mistakes in determining whether a sample is above or below the 10 cfu/mL(g) threshold. As stated in both SRAs:

> All public health outcome predictions presented in this chapter are based on a determination of pass/fail status of each lot using a test with high accuracy, and the testing method used for risk management option implementation should be considered when evaluating the results below, as discussed in the NACMCF 2023 response (NACMCF, 2023)." (Chicken SRA, p. 89 and Turkey SRA, p. 77).

In other words, the model assumes a test of perfect accuracy.

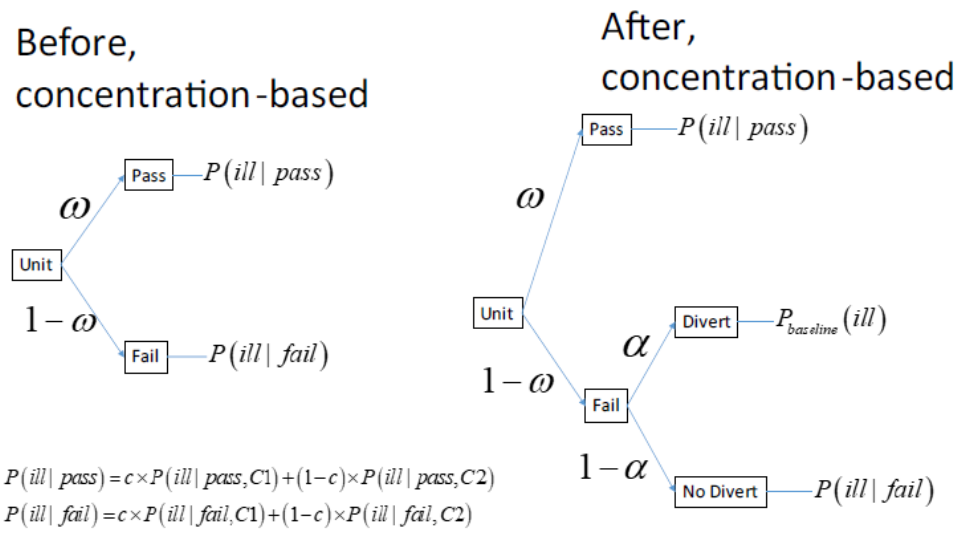


Figure 22: Schematic depiction of the possible pathways which product moves before and after implementation of a concentration-based diversion strategy.

The 2x2 table below shows how we can look at the true status of a sample (above or below the 10 cfu threshold) and whether the laboratory test gives a result above or below the threshold. The squares in the 2x2 table show whether the result would be considered True Negative, False Positive, True Positive or False Negative. The sensitivity of the assay can be thought of as the True Positive rate, calculated as TP/(TP+FN). The specificity can be thought of as the True Negative rate, calculated as TN/(TN+FP). Using this information, we can assess whether the assumption of a test with high accuracy is upheld.

		True Status	
		< 10 cfu / g	**≥ 10 cfu / g**
Test Status	**< 10 cfu / g**	True Negative	False Negative
	≥ 10 cfu / g	False Positive	True Positive
		Specificity	**Sensitivity**

FSIS reports using the bioMérieux's GENE-UP® QUANT *Salmonella* test (Quant) to test samples for *Salmonella* load and comparing these results to the MPN method. When assessing the performance of a quantitative assay, it is important to know the Limit of Quantification (LOQ), which can be defined as the lowest amount of analyte in a sample which can be quantitatively determined with suitable precision and accuracy. According to the FRN, "the lower LOQ for the *Salmonella* tests utilized by FSIS was 10 cfu/mL" (FRN, p. 64712). According to FSIS, "The limit of detection (LOD) of the quantitative polymerase chain reaction (qPCR) enumeration technology used by FSIS at present is 10 cfu/g or /mL (FSIS, 2022b)" (Chicken SRA, p. 25). If the LOD is 10 cfu/mL, then the LOQ should be higher, as it is typically impossible to have quantitative accuracy at the same level as the LOD.

The table below is taken from Table 2 of the Exploratory *Salmonella* Sampling Report document. It shows the results of the testing conducted by FSIS with samples that were spiked to known concentrations of a *Salmonella* Typhimurium isolate. The row showing results for Actual *Salmonella* level of 5 cfu/mL represents samples that are truly below the 10 cfu threshold. As can be seen, the Quant assay determined that 17 of 60 samples had *Salmonella* levels above 10 cfu/mL. This gives a Specificity of 72%. Stated another way, 28% of the time, product would be falsely identified as failing the Final Product Standard. This result represents a cost for the poultry company because of diversion of product falsely identified as failing the standard. The row showing results for Actual *Salmonella* level of 50 cfu/mL represents samples that are truly above the 10 cfu threshold. As can be seen, the Quant assay determined that 15 of 60 samples had *Salmonella* levels below 10 cfu/mL. This gives a Sensitivity of 75%. Stated another way, 25% of the time, product would be falsely identified as meeting the standard and consequently the product would be shipped into commerce. This represents a failure of the proposed system and reduces the predicted impact that the proposed Final Product Standard will have on reducing human salmonellosis illnesses.

QUANT Method				MPN Method			
Actual Salmonella level (CFU/mL)	Counts Below 10 CFU/mL	Counts Above 10 CFU/mL	Method Accuracy	Actual Salmonella level (CFU/mL)	Counts Below 10 CFU/mL	Counts Above 10 CFU/mL	Method Accuracy
5	43/60	17/60	72%	5	60/60	0/60	100%
10	40/60	20/60	33%	10	35/60	25/60	42%
50	15/60	45/60	75%	50	15/60	45/60	75%

It should be clear from this analysis that the assay being used currently by FSIS is not highly accurate at the concentration being proposed for the enumeration-based Final Product Standard. This is highly problematic for a program designed around an adulterant standard. However, if the discussion were to shift to an enumeration-based Performance Standard, as I outlined at the start of this report, there would be much less concern whether an individual lot falsely tests positive or negative, as the company would be using the assay on all production lots and would better understand the importance of a single result in the context of the overall trends in the processing plant.

Furthermore, the FRN states that "*Salmonella* screening results and quantification results would routinely be available 2 days after a sample is taken. For samples above the quantification threshold, an additional 3 days may be necessary for a confirmed positive or negative result." (FRN, p. 64707). This is likely a best-case scenario and would be a major challenge for the production of some of the products included in this regulatory change. However, we should not be hindered by current technology as we develop a plan. If 10 cfu/mL(g) is determined to be a relevant public health threshold, then a program can still be designed around this level. Using an imperfect test with a delayed turnaround would be problematic in an enumeration-based Final Product Standard system but might work suitably in an enumeration-based Performance Standard system. The Final Product Standard requires test-and-hold with severe consequences for samples exceeding the threshold. I can envision a situation in which companies choose to destroy all product tested in the FSIS lot rather than wait for the test results. This does not help public health because in this situation, no other production lots might get tested for *Salmonella* concentration. An enumeration-based Performance Standard does not suffer from the possible disincentive to companies of testing product not included in the FSIS sampled lot.

Contamination Distribution

The SRAs use a lognormal distribution to model the initial contamination of the products being regulated in the proposed Final Product Standard. The Chicken SRA describes different contamination distributions for carcasses, parts and comminuted product (see Chicken SRA, pp. 73-75). The figure below, which is taken from Figure 21 of the Turkey SRA, shows the differences in contamination across the three chicken products as well as comminuted turkey. Regardless of the fact that the three chicken products have different contamination distributions, the Chicken SRA uses a single contamination distribution for all three products. As stated in the SRA, "a lognormal distribution (Log10Normal(-3.037117, 1.279985)) was used that reflected the initial contamination of a mixture of the three raw chicken products – carcasses, parts and comminuted – according to their relative frequencies of consumption (see subsection Chicken Consumption)." (Chicken SRA, p. 90). In other words, the three different contamination distributions for the three different chicken products were combined into a single distribution, with the weighting of each individual distribution based on the frequency of consumption of that product (11%, 83% and 6% for whole carcass, parts and comminuted, respectively). The estimated number of illnesses attributed to chicken was partitioned using the same breakdown. Overall, the chicken model has failed to assess risk associated with each individual product, and the use of a single contamination distribution for all three chicken product types is another example.

The contamination distribution for the turkey SRA is also confusing. In the turkey SRA, FSIS states that "A simplifying assumption was adopted due to the lack of complete data in terms of *Salmonella* contamination across all raw turkey products. The role and contribution from turkey carcasses and turkey parts is highly uncertain. On the other hand, robust data on the *Salmonella* contamination of raw chicken products provides a reliable starting point." (Turkey SRA, p. 80). The document continues by saying "Therefore, a lognormal distribution (Log10Normal(-3.037117, 1.279985)) was used to reflect the initial contamination of *Salmonella* in a mixture of raw poultry products… Hence, under the current approach, the method will likely overestimate

the risk associated to comminuted turkey final products" (Turkey SRA, p. 81). However, in the remainder of the document, it appears that a different lognormal distribution (Log10Normal(-4.857, 2.333) was used in the risk calculations. This other contamination distribution better reflects the comminuted turkey data shown in the figure below.

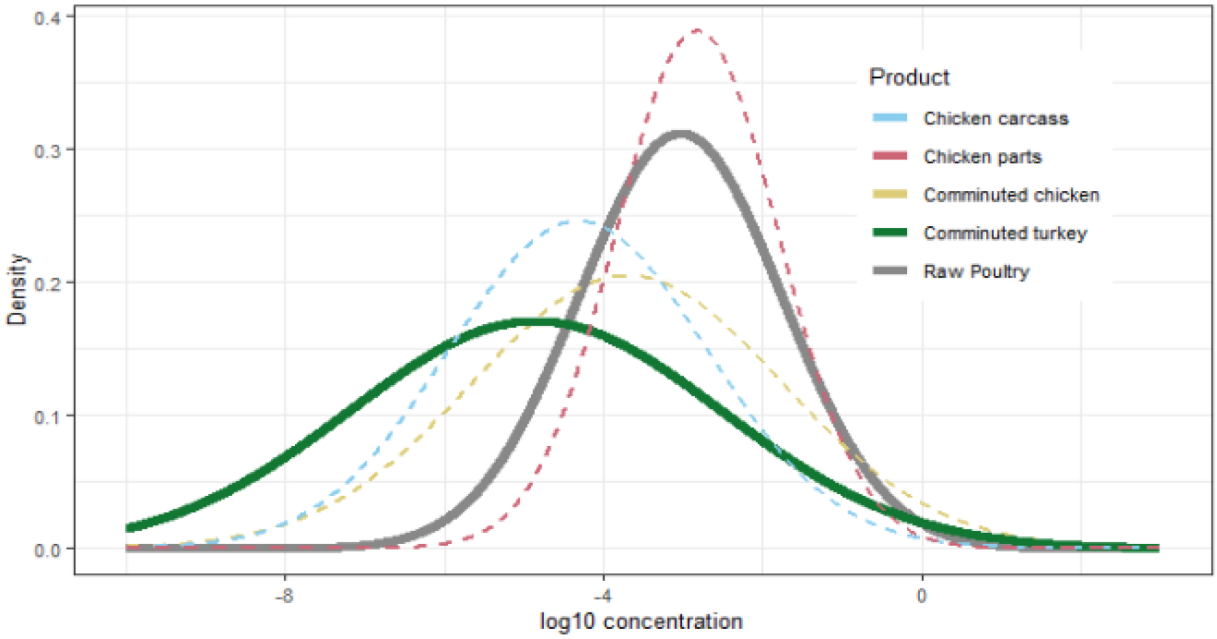


Figure 21: Concentration distribution comparison between comminuted turkey and comminuted chicken and the overarching *Salmonella* contamination distribution in raw poultry products.

Attenuation Distribution

The SRAs include a parameter that adjusts the dose of *Salmonella* on the final product during its transit from the processing plant to the point of consumption. As stated in the SRAs:

> We summarize the effects of the myriad of pathways contaminated product may follow from the end of processing through commerce and preparation using an attenuation distribution. This attenuation distribution captures the variability associated with mixing, partitioning, growth, cooking and serving size processes between production and consumption. A lognormal attenuation distribution (μ = −5.00 log10, σ = 1.91 log10) was calibrated previously for chicken. (Turkey SRA, p. 36).

The SRA then states "Lacking alternative estimates, this default attenuation distribution is used across analyses of chicken products (carcasses, parts, comminuted chicken) as well as comminuted turkey (which is generally handled/consumed in a similar manner as comminuted chicken products)" (Turkey SRA, p. 36).

While the intent of the attenuation distribution is to allow the contamination level to be amended given the potential growth or death of *Salmonella* on the poultry product from the processing plant to point of consumption, the fact that a single distribution was used for all products in both

chicken and turkey seems pointless. Perhaps this is why the SRAs state "both the attenuation and dose-response functions have limited influence on the full model's estimates; i.e., the full model's results are not highly influenced by either attenuation or dose-response. Nevertheless, application of attenuation and dose-response are necessary for improved accuracy in estimates as the threshold increases." (Chicken SRA, p. 113). It is concerning that the SRA models were intended to model illnesses prevented after the regulatory changes, but this important attenuation parameter was not adjusted by product type. It is hard to imagine that *Salmonella* attenuation is identical among chicken carcasses, chicken parts and comminuted chicken, given that this attenuation is meant to include all variability associated with "mixing, partitioning, growth, cooking and serving size processes between production and consumption." Even more concerning is the complete lack of data for comminuted turkey, thus requiring the use of a chicken parameter derived in a 2015 publication by Ebel and Williams. Given the importance of the proposal to establish a Final Product Standard whose efficacy is based on the ability to reduce human illnesses, this important parameter should have been based on better data that are species and product specific.

Risk Multiplier

The Risk Multiplier (RM) is a very important parameter of the SRAs but makes assumptions that are problematic. It is meant to show the increased risk associated with the high virulence serocluster when compared to the low virulence seroculster, but in reality it is a confounded parameter that combines the importance of the serocluster to human illnesses weighted by the frequency that serotypes within the cluster are found in poultry samples. According to FSIS, "the clusters were validated by linking them to epidemiological data (i.e., documented outbreaks attributed to poultry sources with consideration of prevalence in animal sources from FSIS poultry sampling programs). In this sense, the relative risk estimate is skewed towards strains to which a poultry consumer is likely to be exposed." (Chicken SRA, p. 55). The RM is used to modify the dose-response curve of the high virulence cluster to create a curve for the low virulence cluster, which subsequently affects the predicted number of illnesses prevented through the implementation of the proposed regulatory changes.

To understand the problems with this parameter, it is necessary to explore its derivation. As shown in the figure below (Figure 3 from the Turkey SRA), the RM derivation relies on the serocluster model previously discussed. The numerator of the RM for a given serocluster is the weighted proportion of the time that serotypes within each cluster are associated with salmonellosis outbreaks attributed to chicken or turkey. In other words, the numerator is meant to reflect a measure of serocluster severity. However, only illnesses that have been declared by a public health agency such as the CDC as being part of an outbreak are included in the numerator. Serotypes that cause disease sporadically would not be accounted for in this calculation. Also, the size and duration of outbreaks can be subjectively affected by the public health agencies, which would affect the RM estimation. As an example of this, I will briefly explore the yearlong *Salmonella* Infantis outbreak from 2018-2019 (https://archive.cdc.gov/#/details?url=https://www.cdc.gov/salmonella/infantis-10-18/index.html). In February 2019, the outbreak was declared over by the CDC. This was not because *Salmonella* Infantis had been eliminated from chicken or because there were no new

illnesses caused by this serotype. On the contrary, the outbreak was declared over because the incidence rate had reached a stable level. All of the cases of *S.* Infantis attributed to chicken that occurred after the outbreak was declared over would not be included in the RM estimation.

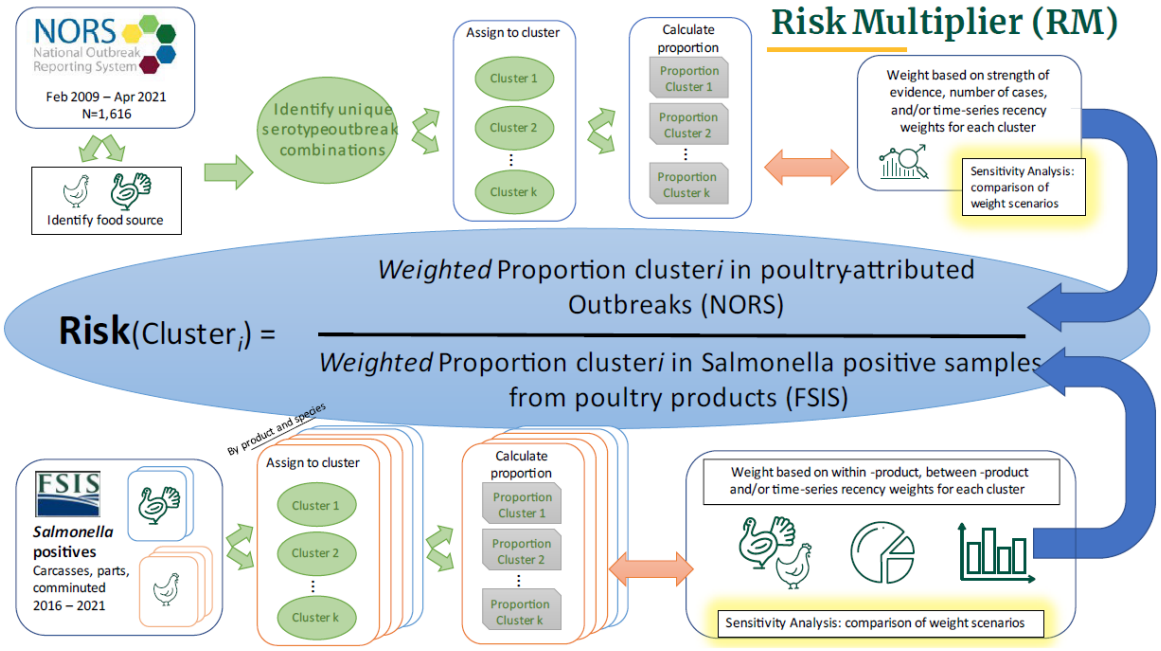


Figure 3: FSIS diagram of risk multiplier estimation for each serocluster.

The denominator of the RM is frequency-weighted, representing the proportion of poultry samples that have one of the serotypes in the respective cluster. These are samples collected by FSIS and are therefore isolates collected from within the processing plant. It is unknown how many of these isolates ended up in commerce and subsequently have the ability to expose the consumer. It is also impossible to know how the culture media used by FSIS affects the serotype detected in the sample (Singer et al., 2009). Regardless, it is unclear why a measure of a serotype's frequency in poultry samples would be used to adjust a dose-response relationship. This is why I consider the RM parameter to be a confounded metric, combining both outbreak potential (a surrogate of virulence) and FSIS-sample prevalence (a surrogate of frequency that has nothing to do with virulence). This type of confounded metric should not be used to adjust a dose-response function.

The end result is a ratio that does not reflect what the modelers seem to think it does. The RM ratio is calculated as 5.66 (2.15/0.38), representing the high virulence (C1) cluster risk over the low virulence (C2) cluster risk. This is shown in the table below, which is taken from Table 13 in the Chicken SRA. FSIS states "the probability of illness per serving from C1 exposures is 5.66 times larger than the probability of illness per serving from C2 exposures. Therefore, the parameters for the two dose-response function must be selected to maintain this relative probability of illness" (Chicken SRA, p. 91). This constraint on the dose-response functions will be discussed more below, but in essence this assumes that the outcome of the dose-response of C2 (probability of illness given a certain dose) is homogenously 5.66 times lower than C1.

Table 13: Risk multiplier estimation including the 95% confidence interval for k=2 seroclusters derived by EpiX Analytics.

	Cluster 1	Cluster 2	Not Assigned
Proportion in outbreaks (numerator)	0.71 [0.58; 0.83]	0.25 [0.14; 0.38]	0.039 [0.012; 0.081]
Proportion in poultry (denominator)	0.33 [0.31, 0.35]	0.66 [0.64; 0.68]	0.010 [0.006; 0.017]
Risk multiplier	**2.1 [1.7; 2.5]**	**0.38 [0.21; 0.58]**	**3.9 [1.1; 9.1]**

There was supposedly a sensitivity analysis conducted on the RM parameter, but this sensitivity analysis only focused on the weighting scheme used for the numerator and denominator. The sensitivity analysis did not address the importance of having all strains of a serotype in the same cluster nor the assumption that all serotypes in the cluster represent an equal risk.

Finally, data were clearly lacking from turkey. FSIS states in the chicken SRA that "including turkey data does not change the results of this chicken *Salmonella* serocluster risk analysis. On the other hand, turkey data is not sufficient to determine risk, and chicken data is necessary for a turkey analysis" (Chicken SRA, p. 61). This is due to limited serotype information from turkey samples. To complete the turkey SRA, chicken data were often used in parameter estimation. This is a clear indication that data gaps exist and that additional data need to be collected, especially prior to implementing a regulatory change as significant as a Final Product standard in raw, NRTE poultry products.

Dose-Response

Dose-response models are needed in these quantitative SRAs because the likelihood of becoming ill following exposure to *Salmonella* depends on the dose as well as the virulence of the *Salmonella* strain. Many dose-response functions have been derived for different *Salmonella* serotypes based on data collected during outbreaks. The functions that are derived around the collected data do not necessarily predict the likelihood of infection (and illness) at the lower ends of these dose-response curves. According to FSIS, "The median *Salmonella* dose predicted to result in 50 percent of exposed individuals becoming ill (IllD50) was 3,360 cfu (95 percent range: 18–3.2×109), 1,500 cfu (38–8.8×107), and 1 cfu (0.69–1.0×106) for Enteritidis, Typhimurium and Infantis, respectively" (FRN, p. 64697-8). As stated previously, even though Infantis is predicted to have a very low infectious dose (implying high virulence), the flawed application of the serocluster model includes Infantis with the low virulence cluster.

There is a strange commentary on virulence and dose in the FRN. FSIS states:

> The 2023 risk profile identified 32 *Salmonella* serotypes of concern linked to foodborne *Salmonella* outbreaks from chicken and turkey products. These identified serotypes of concern informed all subsequent risk management questions, including whether exposure to a small number of these serotypes result in foodborne illness. Because the *Salmonella* serotypes of public health significance identified in the final product standards are among the 32 *Salmonella* serotypes of concern identified in the risk profile and risk assessments, it is

> reasonable to conclude that the serotypes of public health significance in the final product standards all cause illness at a relatively low dose." (FRN, p. 64698).

This is a strange justification for dose-response conclusions, Also, key "low virulence" serotypes that have strains with higher virulence were included in cluster 2.

To generate the dose-response function for cluster 1 (the high virulence cluster), EpiX Analytics used "data from the literature on Enteritidis and Typhimurium (two primary serotypes in cluster 1) and scaled a second dose-response model for the lower virulence cluster 2 based on the risk multiplier ratios" (Chicken SRA, p. 64). In other words, dose-response data were used for two of the serotypes in the high virulence cluster. The outbreaks from which the dose-response data were derived were rarely associated with poultry products. Once the dose-response function was derived, this function was used for all serotypes and strains within the cluster, thereby making the implicit assumption that every strain of every serotype in the cluster would behave about the same (an assumption that is not supported by the data).

To generate the dose-response function for cluster 2 (the low virulence cluster), one might expect EpiX Analytics to use data from outbreaks caused by serotypes in cluster 2 in a manner similar to cluster 1. This is not what was done, however. For cluster 2, the dose-response function was derived by assuming that the Risk Multiplier expresses the difference in virulence between clusters 1 and 2 and therefore can also be used to modify the dose-response function. In other words, because the Risk Multiplier was estimated to be 5.66, the high virulence cluster dose-response was reduced 5.66-fold. FSIS states that an RM value of 5.66 indicates that "the probability of illness per serving from C1 exposures is 5.66 times larger than the probability of illness per serving from C2 exposures. Therefore, the parameters for the two dose-response function must be selected to maintain this relative probability of illness." (Chicken SRA, p. 91).

As can be seen in the equation below, EpiX Analytics assumed that the ratio of the two dose-response functions (left side of equation) should be equal to the Risk Multiplier (right side of equation). Consequently, the low virulence cluster dose-response function has no basis in data collected from the dose-response functions of serotypes contained within the cluster but instead on the flawed Risk Multiplier parameter. The RM parameter uses a frequency-based denominator (frequency of *Salmonella* in FSIS-sampled product) which should not be used to modify a dose-response function.

$$\frac{\int R_1(d)h(d)\partial d}{\int R_2(d)h(d)\partial d} = \frac{RR_1}{RR_2}$$

After performing sensitivity and uncertainty analyses on parameters such as the dose-response functions, FSIS states that:

> Such findings support the general idea that both the attenuation and dose-response functions have limited influence on the full model's estimates; i.e., the full model's results are not highly influenced by either attenuation or dose-response. Nevertheless, application of attenuation and dose-response are necessary for improved accuracy in estimates as the threshold increases. (Chicken SRA, p. 113).

Perhaps the reason for the limited influence of the dose-response functions on the model outputs is that a single function was derived for every strain of every serotype in the high virulence cluster, and then this same function was reduced 5.66-fold to account for all strains of all serotypes in the low virulence cluster. This lack of precision and heterogeneity around the dose-response function would clearly lead to the parameter being considered unimportant in the model.

FSIS justifies making *Salmonella* an adulterant by comparing this current approach to that taken with Shiga toxin-producing *E. coli* (STEC). This is an entirely fallacious comparison for multiple reasons. First, the infectious dose of the two organisms is very different. STEC have a much lower estimated infectious dose than *Salmonella*. Second, the severity of the illness caused by the two is drastically different. STEC infections result in hospitalization and often death, with many patients developing hemolytic uremic syndrome (HUS). Most salmonellosis cases are self-limiting, and very few need additional therapy beyond supportive care. Third, *Salmonella* is ubiquitous in the poultry industry, which means that most flocks will be positive for *Salmonella*. *Salmonella* might not be present in every bird within the flock, but in poultry production, the flock is the unit. STEC infect the individual animal (cow), and carriers can be identified and culled without sacrificing the entire herd. For *Salmonella*-positive flocks, the entire flock would have to be eliminated or diverted, as it is not feasible to test each individual animal. On the farm, STEC was always a much rarer bacterium than *Salmonella*, and *Salmonella* is very well-adapted to the bird. Fourth, STEC do not cause disease in cattle, whereas *Salmonella* can sometimes be an avian pathogen. Finally, cattle and poultry slaughtering processes are very different. These are just some of the differences between *Salmonella* and STEC; STEC should not be used as an analogous organism to justify this proposed Framework.

Overestimation of Chicken-Attributed Illnesses Prevented

The expected number of illnesses prevented is shown in various tables in the documents, including in the FRN. For example, Table 5 in the FRN says that 1,000, 200 and 1,000 illnesses would be prevented due to consumption of chicken carcasses, chicken parts, and comminuted chicken, respectively. The second Table 34 in the FRN gives the same numbers (shown below), but also adds a total for all chicken products: 2,200. I am focusing on the High column, as these results reflect the baseline enumeration-based Final Product Standard model. It is strange that this table sums all chicken-attributed illnesses prevented into one number. Specifically, FSIS states that "The 2023 chicken risk assessment assessed the effect of a carcass final product standard on all chicken associated illnesses, including those from parts and comminuted product consumption, but could not assess the effect of carcasses and secondary products standards sequentially. As such, the 2023 chicken risk assessment estimates for chicken products are not additive." (FRN, p. 64738). In other words, we cannot sum the chicken product numbers in Table 5 (or Table 34) to estimate the total number of illnesses that might be prevented. To show how the number of prevented illnesses attributed to chicken is inflated, I will work through the derivation of the numbers in Tables 5 and 34 of the FRN.

TABLE 34—ESTIMATED NUMBER OF ILLNESSES PREVENTED BY PRODUCT

Product	Prevented illnesses		
	Low	Medium	High
Chicken products:	240	1,000	2,200
Chicken carcasses	240	1,000	1,000
Chicken parts	.....	.....	200
Comminuted chicken	.....	.....	1,000
Comminuted turkey	525	2,100	2,100
Total	765	3,100	4,300

Within the Chicken SRA, FSIS states that "A chicken carcass performance standard that diverts test-positive lots based on a threshold level of 0.033 cfu/mL (i.e., 1 cfu of *Salmonella* per 300 mL poultry rinsate) is the most effective risk management option to reduce foodborne *Salmonella* from chicken carcasses, with 4,700 illnesses prevented annually, which equates to 3.8 percent of the approximately 125,000 overall chicken illnesses that occur each year. The public health impact (in terms of illnesses prevented) of the chicken carcass final product standards encompasses the illnesses estimates for all secondary chicken products, as the majority of those secondary products are fabricated from carcasses." (Chicken SRA, p. 30). As can be seen in this calculation, the estimated number of illnesses prevented for chicken carcasses includes all illnesses prevented for parts and comminuted chicken as well. This can be seen using some of the tables included in the Chicken SRA document.

First, Table 11 of the Chicken SRA shows parameter estimates for the model. Below, I have extracted the row from Table 11 that shows the number of illnesses attributed to each of the chicken products (p. 51). The number of illnesses listed for carcasses is actually the total number of illnesses attributed to chicken consumption. Again, it appears that the reason for including all illnesses under "carcasses" is because of the statement that "the chicken carcass final product standards encompasses the illnesses estimates for all secondary chicken products, as the majority of those secondary products are fabricated from carcasses." (Chicken SRA, p. 30 and p. 156).

number of illnesses before policy	λ_{ill}	carcasses=125,115 parts=103,845 comminuted=7507	illnesses/year

Table 38 of the Chicken SRA (shown below) reports the parameter distributions for the percent reduction in illnesses for the various chicken products. If we look at the Pert distribution for the 0.03 cfu/mL and for chicken carcasses, the distribution is a Pert(0.0095, 0.0369, 0.0444). The three parameters of the Pert distribution are the Minimum, Mode and Maximum values. The median of the Pert can be derived with the formula (Min + (6*Mode) + Maximum) / 8, which gives a median percent reduction of 0.0344. To arrive at the number of illnesses prevented, FSIS multiplied 3.8% (rather than the mode, 3.69% or median, 3.44% from the Pert distribution) by the total number of chicken-attributed illnesses (125,115) rather than the total number of chicken carcasses-attributed illnesses (13,763, see Table 28 of Chicken SRA). Multiplying 125,000 * 0.038 = 4,750, which is basically the number that FSIS cites in the paragraph included above.

Table 38: Descriptions of the uncertainty distributions for the parameters used to estimate annual illnesses prevented are shown.

Concentration threshold*	Variable	Chicken carcasses	Chicken parts	Comminuted chicken
	λ_{ill}, mean (95% CI)	125,000 (73,000 – 193,000)	104,000 (60,000 – 160,000)	8,000 (4,000 – 12,000)
0.03 cfu/mL	percent reduction	Pert(min=0.0095, mode=0.0369, max=0.0444)	Pert(min=0.0192, mode=0.0757, max=0.1005)	Pert(min=0.096, mode=0.1964, max=0.2033)
1 cfu/mL	percent reduction	Pert(0.0016, 0.0194, 0.0392)	Pert(0.0011, 0.0138, 0.0383)	Pert(0.04, 0.1852, 0.2252)
10 cfu/mL	percent reduction	Pert(0.0003, 0.0083, 0.03)	Pert(0, 0.0021, 0.0083)	Pert(0.0148, 0.1389, 0.2267)
100 cfu/mL	percent reduction	Pert(0.0001, 0.0017, 0.0186)	Pert(0, 0.0002, 0.0008)	Pert(0.0042, 0.0817, 0.2189)

To see how the number of illnesses prevented might be more accurately estimated, we can again use Table 38 (shown above) from the Chicken SRA. At the 10cfu/mL(g) concentration threshold, the chicken carcass distribution is modeled as a Pert(0.0003, 0.0083, 0.03). The median of this Pert distribution equals approximately 1.0% percent reduction in illness. Using this median estimate and the total number of chicken carcass-attributed illnesses (13,763) instead of the total number of all chicken-attributed illnesses gives an estimated 138 illnesses prevented from carcass-attributed illnesses, thus giving a total of 138+200+1,000=1,338 illnesses prevented, not 2,200. A total of 1,338 illnesses prevented represents approximately 1.07% of estimated chicken-attributed illnesses prevented with this Final Product Standard policy (1,338/125,115). This number of illnesses prevented does not include the serocluster criterion of the proposed Final Product Standard, which would further reduce the number of illnesses prevented.

In summary, the total estimated number of illnesses prevented from chicken consumption is likely between 1,000 and 1,500 under this proposed enumeration-based Final Product Standard. The number 2,200 shown in Table 34 of the FRN is double counting the carcass-associated illness estimates.

Component 2: Enhanced process control

I fully support the addition of a proper statistical process control (SPC) program, as I've discussed earlier under my Alternative Program proposal. The purpose of this upstream SPC is to ensure that the microbial interventions of the processing plant are functioning properly. As stated throughout the documents included with the *Salmonella* Framework proposal, published studies and "unpublished data provided by the poultry industry and university researchers suggests that indicator bacteria have very limited predictive value for the prevalence of *Salmonella.*" (FRN, p. 64711). Because these results are not predictive of *Salmonella* outcomes, companies should have flexibility in the design of the SPC program, as long as biologically relevant reductions in indicator bacteria (and therefore efficacious microbial interventions) can be documented.

Collecting samples at rehang and post-chill, as proposed by FSIS, seems like logical locations in the plant to conduct the SPC program. In a previous paper on which I am co-author (Berghaus et al., 2013), rehang was predictive of post-chill and would be a good in-plant location for sampling to document reductions in indicators such as Aerobic Count (AC). Again, there should be flexibility here regarding which bacterial indicators to monitor and what reduction in bacterial load between sampling sites represents a successful program.

The FSIS documents are confusing because, at times, the proposed SPC program is described as two-sided. For example, the FRN states that "If the process exceeds an upper or a lower specification limit, the product does not meet the specification even if it is operating without assignable causes and is in control" (FRN, p. 64710). Why would a reduction below the minimum be considered out of control? One would think that reductions that exceed lower specification limits would be a good thing. The SPC program should be one-sided, in which results that exceed the upper specification limit are considered out of compliance.

I am concerned about FSIS' opinion regarding the use of EB or *Salmonella* in the SPC. Specifically, FSIS sates that "microbial monitoring of EB or *Salmonella* is unlikely to yield the reliable quantified results necessary for an individual establishment to support SPC monitoring" (FRN, p. 64712). This conclusion is based on some samples having EB or *Salmonella* loads below the LOQ. However, why is left-censored data a bad thing? Microbial levels below the LOQ should represent a desirable outcome, and if the post-chill samples have results below the LOQ, this should indicate a successful intervention. Regardless, FSIS states that "the current data shows that AC is more likely to yield reliably detectable quantified microbial results compared to either EB or *Salmonella* for most establishments" (FRN, p. 713), and while this is not a good reason to use AC as the indicator organism for an SPC program, AC might be an easier microbial target to monitor for many of the poultry processing plants. The combination of an actual SPC for AC upstream with an enumeration-based Performance Standard should provide a large impact on salmonellosis illnesses, but as I have stated previously, this was not modeled in the current *Salmonella* framework.

The proposed level of reduction in AC, as described in the *Salmonella* Framework, is also confusing. According to FSIS, the:

> recent chicken risk assessment concluded that a hypothetical AC reduction standard could achieve a 25 percent reduction in *Salmonella* illnesses attributed to chicken only if microbiological criteria based on 2.5–3.0 log reduction or no AC tests exceed 10 cfu/mL at the post-chill location. The risk assessment concluded that AC is only moderately correlated with the occurrence of *Salmonella* and thus an AC based standard would perform less well than a *Salmonella* standard. (FRN, p. 64713).

The FRN goes on to state that the "2023 turkey risk assessment reported that the correlation between AC or EB and *Salmonella* prevalence is weak, and it was not possible to fully assess the public health impact of monitoring and enforcing process control from rehang to post-chill" (FRN, p. 64713). FSIS then states that "Based on these findings, the Agency would consider an establishment's target change criteria to meet the requirements in 9 CFR 381.65(g) when its MMP sets an expected reduction of at least 1.0 log in detected microbial levels between sampling locations" (FRN, p.64714).

It is extremely confusing to read through the SRAs and the FRN in which data are presented that show that the industry already reduces AC by 2-3 logs, and that this reduction might be correlated with reduced *Salmonella* prevalence post-chill, at least in chicken. However, these same documents then propose a threshold for AC reduction equal to 1 log. How does a 1 log reduction help? Is this small reduction correlated with reductions in *Salmonella*? Is a 1 log

reduction indicative of a process that is in control? Was any modeling conducted to evaluate a 1 log reduction?

Another confusing aspect of the SPC proposal by FSIS is the lack of consideration of incoming load in the SPC thresholds. If the goal is to reduce by 1 log (or better yet, 2 to 3 logs, as I have mentioned above in my Alternative Program section), companies would be rewarded for having high *Salmonella* loads at rehang because it would be easier to reduce this high level of contamination. For a company whose *Salmonella* loads are already low at rehang, it would be harder to reduce those low loads further. The SPC program needs to include a biologically relevant reduction target between sampling points as well as consideration for the incoming load of the indicator organism at rehang.

The Chicken SRA states that:

> As a result of these weak relationships between AC and *Salmonella* prevalence, it follows that the correlation between AC and *Salmonella* serotypes or levels is also weak. Therefore, it was not possible to assess the risk management question regarding the public health impact (illnesses, hospitalizations, and deaths) of monitoring/enforcing process control from rehang to post-chill in the same manner as it was estimated for final product standards. (Chicken SRA, p. 34).

Thus, it appears that we have no idea how useful an SPC program will be in reducing *Salmonella* illnesses. Regardless, I still believe that a proper SPC program linked to an enumeration-based Performance Standard will have a considerable impact on poultry-attributed *Salmonella* illnesses.

Component 1: Incoming flock testing

Although FSIS states that it "considered the available scientific research as well as input from the NACMCF and concluded that, at this time, the research does not support the use of a threshold for test results at the receiving step to reduce or eliminate *Salmonella* from raw poultry products" (FRN, p. 64680), I will briefly address Component 1 of the Framework because it relates to a specific question that FSIS has posed. Namely, the FRN states that "If FSIS finalizes the proposed final product standards, the Agency intends to re-evaluate the serotypes of public health concern every 3–5 years at a minimum and whenever new information on *Salmonella* serotypes associated with human illness become available" (FRN, p. 64679). This has relevance in the context of Component 1 and live production *Salmonella* interventions.

Chicken and turkey companies spend a considerable amount of time and money controlling *Salmonella* in live production. Their obvious goal is to reduce *Salmonella* loads as much as possible before the birds go to the processing plant so that the in-plant interventions can be as efficacious as possible. The lower the incoming load, the more effective these interventions are likely to be. Regulating companies on incoming load or serotypes is misguided, as different companies will pursue different approaches to ensure that their final product is as low risk as possible. Consequently, FSIS should stay focused on ensuring that plants have adequate process control (through the SPC Component 2) and are keeping *Salmonella* loads below an enumeration-based threshold (variation of Component 3).

Poultry companies already focus on *Salmonella* preharvest control in various phases of production, including the commercial birds, the hatchery, and the breeders. It is very difficult to control *Salmonella* completely, as *Salmonella* is a commensal organism in chickens and turkeys and is well-adapted to the avian host. Controlling *Salmonella* has always been a challenge, and preharvest interventions are expensive and imperfect. I previously published a paper showing that companies were practicing preharvest intervention but with limited success due to imperfect solutions (Hwang & Singer, 2020). No preharvest interventions have proven to prevent *Salmonella* presence on incoming chicken flocks. If there were such interventions, then the entire poultry industry would have incorporated them rather than spend an inordinate amount of time and money with postharvest measures.

Controlling specific serotypes of *Salmonella* is extremely problematic, as there are very few serotype-specific mitigation strategies. FSIS states that "The committee also noted that *Salmonella* vaccination is one breeder-level pre-harvest intervention that contributes to an overall reduction and/or elimination of specific *Salmonella* serotypes. The committee stated that the most effective vaccination strategy is to focus on vaccination of breeder flocks and reduce vertical transmission of *Salmonella.*" (FRN, p. 64719). Vaccination is a key intervention approach, and companies actively use *Salmonella* vaccines, including autogenous vaccines targeting key serovars. Vaccination is already being used in the breeder flocks, as many of the serotypes of public health significance can be transmitted vertically, from the breeder hens to their progeny. As stated in the FRN, "As part of its response, the committee noted that vaccination programs have been incorporated on U.S. farms. The committee described such vaccination programs as an effective management practice for controlling *Salmonella* at pre-harvest and noted that vaccines are likely the only serotype-specific intervention strategies." (FRN, p. 64720). What is being proposed is not novel and has been a strategy for the poultry industry for many years.

As shown in a previous paper, it can take a considerable amount of time (at least 18 months) for the effects of vaccination of breeders to be seen in commercial birds, and this is assuming high efficacy of vaccine (Liljebjelke et al., 2005). This fact has been known for many years. The FSIS documents seem to assume that vaccination is a key method for eliminating a serotype of concern. *Salmonella* Heidelberg is given as an example. However, this is a simplistic view of the reasons for the decline of *S.* Heidelberg. *S.* Infantis appeared in U.S. poultry, as well as poultry around the world, without warning. Broiler companies have focused on this serotype for years. There seems to be an expectation by the public health agencies that this serotype should have been eliminated already and yet it remains a main serotype seen in broiler production and on final product. There is no perfect approach to *Salmonella* elimination from poultry, and to assume that the presence of *Salmonella* in incoming birds demonstrates a lack of focus on preharvest measures is inaccurate.

Given that it can take 18 months or more to see an effect of the vaccine in breeders on *Salmonella* in commercial birds, changing the serotypes of public health significance on a timeframe of less than 3 years would be problematic for vaccine design and administration within the entire production system. A timeframe of 5 years or more for changing the serotypes of public health significance ensures that the production companies are not constantly changing their focus without sufficient time to see the effects of the vaccination program. In other words,

if a public health agency notices a new serotype causing human illnesses, and if this serotype is associated with poultry, it will take two years or more to implement a vaccine intervention and see the downstream effect. Companies need to be notified as early as possible about a possible new serotype of concern so that they can begin targeting that emerging serotype in live production. Changing the serotypes on which the companies are being regulated (which is different than notifying the companies of a new signal of concern) assumes that the intervention can quickly reduce the incidence of this serotype. Just because "new information on *Salmonella* serotypes associated with human illness" might become available, it still takes time (years) to change the intervention program (vaccine) and see the downstream effect.

Other Comments

- Throughout the proposed *Salmonella* Framework, there are many examples of data gaps that should have been filled prior to issuing this proposed drastic change to poultry inspection. For example, many of the parameters used in the turkey SRA are missing turkey-specific data; chicken data are used as a surrogate for the turkey data. Data are often lacking regarding the chicken products being regulated under this proposed Framework. It seems entirely inappropriate to propose a regulatory change of this magnitude without data specific to the products being regulated.
- The product "chicken parts" is included in this proposed Framework, but no real definition is given to this product. It does not seem reasonable to assume that chicken wings, chicken thighs, chicken breasts, and other chicken parts behave the same with respect to the model parameters. This is not a homogenous category.
- FSIS states that "Since consumers are unable to distinguish between products in the marketplace that have higher probabilities of resulting in *Salmonella* illness and those with lower probabilities, both types of products are sold at the same price point. Under such market conditions, establishments are disincentivized from investing in food safety measures and controlling for *Salmonella.* This results in an increased risk of *Salmonella* illnesses, and, in consequence, an increased risk of outbreaks and outbreak-related recalls for establishments." (FRN, p. 64741). This is an incredibly inflammatory opinion. Does FSIS have data to back up this statement?
- Throughout the *Salmonella* Framework documents, FSIS clearly shows a major disparity between large and small volume establishments. For example, FSIS states in the Chicken SRA "For the poultry industry, *Salmonella* and *Campylobacter* occurrence is more frequent on products produced by lower-volume establishments. The opposite phenomenon is observed in the pork and beef industries, where a small number of large establishments account for the majority of the contaminated product reaching consumers (Williams, 2022)." (Chicken SRA 68). There is the additional statement that "*Salmonella* carcass post-chill contamination is now predominantly in low-volume production establishments (those establishments slaughtering less than 10 million carcasses per year)." (Chicken SRA, p. 24). Figure 2 from the Chicken SRA (p. 24), shown below, demonstrates that the small volume establishments have a larger problem with *Salmonella*. Why is implementation of the proposed Framework delayed for these establishments? The estimated number of illnesses prevented does not account for this delayed implementation, and thus the impact of the proposed program may not be realized for years.

- The *Salmonella* Framework documents repeatedly state that the risk assessments and other models were peer-reviewed. However, the actual peer-review process used does not appear to be the same as that used by peer-reviewed journals. For example, how were the reviewers selected? Reviewers were given a very short window of time to review these massive documents/models. How did the agency respond to the reviewers' concerns? Did the reviewers get to review the revisions made by FSIS, as would be done in a peer-review process of a journal article? Did the reviewers have the option to "reject" or "not accept" the risk assessment, or parts of the risk assessment, as would be done in a peer-reviewed journal article? In summary, I do not consider these documents to be "peer-reviewed" in the sense with which many scientists are familiar.

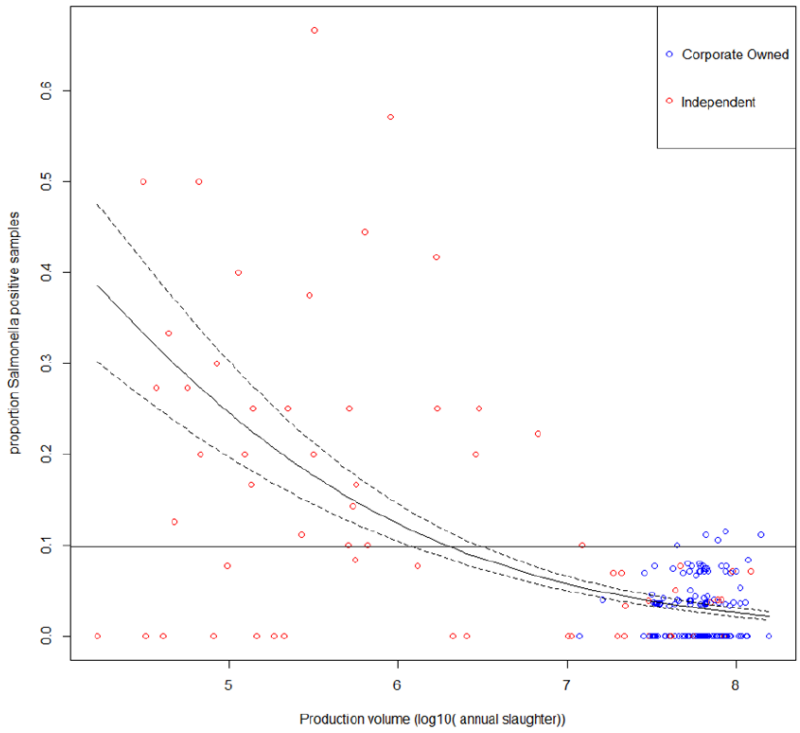


Figure 2: Relationship between establishment production volume, ownership, and presence of *Salmonella* on chicken carcasses.

Salmonella **Framework – Comments Requested by FSIS**

- The proposed timeline for re-evaluating serotypes of public health concern (every 3-5 years).
  Response: As I have written in my Component 1 section, it is concerning that the regulated serotypes of public health significance could be changed at a frequency that is impossible to meet by the industry. It can take 18 months or longer to see downstream effects of vaccine use in breeders. The industry would benefit from being informed of a new serotype signal detected by the public health agencies. The companies could then begin a vaccination program well in advance of a regulatory change in serotypes of public health significance.

- Phasing out performance standards and no longer using *Salmonella* sampling results to categorize establishments and no longer publishing categories on the FSIS website.
  Response: The performance standards for chicken and turkey have been effective at reducing *Salmonella* on tested products, as clearly stated by FSIS in the *Salmonella* Framework documents. Part of the industry's ability and willingness to adapt to the performance standards voluntarily is due to the publishing of establishment results. As stated in my overall assessment of the Framework, a new Performance Standard based on enumeration-based thresholds is a logical and likely effective advancement to poultry inspection. Publishing the establishments' categorization based on the enumeration-based Performance Standards will likely help ensure that the industry adapts to this new standard.

- The full risk model and the uncertainty and sensitivity analyses, whether they are fit for the purpose of determining the serotypes of public health significance, and what model adjustments or other approaches FSIS should consider in the determination to adapt to evolving data, technology, and analytical methods
  Response: The full risk model, while representing a massive effort by many scientists, is not fit nor necessary for determining the serotypes of public health significance.

- Whether EpiX Analytics serotype clustering and dose-response adjustment (i.e., risk multiplier) used the best available data and genetic factors relevant to *Salmonella* risk and contamination in the US population
  Response: The serotype clustering and dose-response adjustment did not use the best available data. The serotype clustering analysis was more of a phylogenetic tree rather than a clustering based on virulence. The model treated all genes (including putative genes that have no known function within *Salmonella*) to relate isolates. No weighting was performed based on importance of a gene or set of genes. The model does not capture strain variation. Also, the original model was based on beef isolates (Fenske et al., 2023). The issue is that any strain variation within a serotype affects the cluster assignments and subsequent RM estimates. Increasing the number of isolates with non-poultry sources will add these non-poultry genetic arrangements to the algorithm, thereby not reflecting the risk that isolates from poultry possess.

- Potential improvements to the serotype clustering robustness analysis and the risk multiplier sensitivity analysis

Response: The sensitivity analysis on the risk multiplier was useless. It did not address the major concerns I detailed above, including the fact that this RM should never have been used to adjust a dose-response function. What would have happened if a single RM was developed for each serotype, with one serving as a baseline? Where would Infantis fall? Regardless, before the RM can be useful, we must understand how strain differences within serotype affect the RM.

- Possible inclusion of Infantis as a serotype of public health significance
  Response: The clustering algorithm developed by EpiX Analytics and used by FSIS to assign seroclusters and to derive the Risk Multiplier is flawed. Not all strains within a serotype are homogenous, including with respect to virulence. Further, not all serotypes included in a serocluster are homogenous. There are clearly strains of Infantis that are more virulent than others in the serotype. Similar to a paper we published on *Salmonella* Reading, it is necessary to identify the traits that differentiate strains of Infantis and then figure out how to develop a clustering algorithm that can actually account for these intra-serotype inter-strain differences. At this point in time, the use of the serocluster algorithm is unnecessary and highly flawed and leads to inaccurate estimates of illnesses prevented.

- Available technologies and methods for quantification and serotyping
  Response: The current technologies and methods for quantification and serotyping are not currently ready to be used for the Final Product Standard proposed in this Framework. First, the LOQ is too high. As stated in the documents, the LOD of the assay is around 10 cfu/mL(g), which also happens to be the threshold of the Final Product Standard. There is no way that the LOQ can be the same as the LOD. Having an error-prone test for use with a Final Product Standard is inappropriate and problematic. However, this same test might function fine when used in an enumeration-based Performance Standard system. For serotyping, the results take too long. Product cannot be held that long. Regardless, the maximum public health benefit of this program is by using an enumeration-based standard without a serotype criterion, and therefore, the serotyping methods are irrelevant at this time.

- Whether the Agency should phase out the current performance standards as the Agency implements the final product standards or if the Agency should retain the current performance standards and later determine if these standards are still needed when evaluating the effectiveness of the proposed final product standards
  Response: The Agency should phase out the current performance standards and implement an enumeration-based Performance Standard. There is no need to implement a Final Product standard. I have explained this in detail in my report.

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