CONCISE COMMUNICATION

Serotype Distribution of *Salmonella* Isolates from Food Animals after Slaughter Differs from That of Isolates Found in Humans

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If raw meat and poultry are the primary point of entry for *Salmonella* species into human populations, a correlation might be expected between the serotype distribution of *Salmonella* species isolated from animals at the time of slaughter and that of isolates found in humans. For 1990–1996, sufficient national data were available to permit such a comparison. A mathematical model was developed to predict serotype distributions of *Salmonella* isolates among humans on the basis of animal data. There was a significant mismatch between the serotype distributions among humans predicted by the model and those actually observed. This mismatch raises questions about the validity of the "standard" assumptions about *Salmonella* transmission on which the model was based—namely, that raw animal products are the primary source for human salmonellosis, that the risk of transmission to humans is equal for all food product categories, and that all *Salmonella* serotypes have an equal ability to cause human illness.

Salmonellosis remains a substantive cause of morbidity and mortality. Using 1996 and 1997 data from the Centers for Disease Control and Prevention (CDC) FoodNet sentinel site program, Mead et al. [1] estimated that ~1.4 million nontyphoidal *Salmonella* cases occur in the United States each year. The US Department of Agriculture (USDA) has projected that *Salmonella* infections account for medical costs and productivity losses of $600 million to $3.5 billion per year [2]. It also has been estimated that 50%–75% of human *Salmonella* infections are attributable to the presence of the organism on meat and poultry products [2].

In the 1990s, for the first time, sufficient national data became available on the serotypes of *Salmonella* species isolated from food animal carcasses to allow comparisons between *Salmonella* serotypes isolated from animals and those isolated from humans. We report here the results of such an analysis, including the development of a mathematical model to correlate serotype data from human and animal sources.

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Methods

Data on human serotypes. Data on *Salmonella* serotypes from cases of salmonellosis in humans that occurred between 1990 and 1995 were obtained from the CDC summary reports on the National *Salmonella* Surveillance System. This passive surveillance system, which was established in 1963, collects reports, from every state and the District of Columbia, of *Salmonella* isolates found in humans. The system has remained essentially unchanged except for fluctuations in reporting interests and priorities [3].

Data on raw meat and poultry contamination with Salmonella. Data were obtained from 5 studies done by the Food Safety and Inspection Service (USDA) between 1990 and 1996. The first study was the 1990–1992 *Salmonella* Species in Broilers, a National Study. The second study was the 1994–1995 Nationwide Broiler Chicken Microbiological Baseline Data Collection Program. The third study was the 1992–1993 Nationwide Beef Microbiological Baseline Data Collection Program: Steers and Heifers. The fourth study was the 1993–1994 Nationwide Beef Microbiological Baseline Data Collection Program: Cows and Bulls, and the fifth study was the 1995–1996 Nationwide Pork Microbiological Baseline Data Collection Program: Market Hogs. The principal purpose of each of these studies was to determine the national prevalence of certain indicator microorganisms and pathogens, including *Salmonella* species, on meat and poultry produced at federally inspected slaughter plants. The establishments sampled accounted for >99% of all federally inspected food products. A consistent, randomized sampling strategy was used for each food product category.

Model development. A mathematical model was developed to predict the distribution of various *Salmonella* serotypes among isolates from humans, using data on *Salmonella* serotype distribution

Table 1. Serotype distribution of *Salmonella* isolates from humans and animals, 1990–1995.

Serotype[a]	Percentage of isolates from			
	Humans (n = 33,130)	Chicken (n = 695)	Beef (n = 70)	Pork (n = 186)
Typhimurium[b]	22.6	9.9	15.7	20.4
Enteritidis	22.0	1.0	0	0
Heidelberg	6.8	17.0	0	7.0
Newport	4.6	0.7	1.4	0
Hadar	3.6	15.7	0	1.1
Thompson	1.7	6.4	2.9	0
Kentucky	0.1	17.0	8.6	0.5
Derby	0.004	0.6	1.4	28.0
Other	38.6	31.7	70.0	43.0

NOTE. Data for isolates from humans were reported through the Centers for Disease Control and Prevention, National *Salmonella* Surveillance System; data for isolates from chickens were from the US Department of Agriculture (USDA), *Salmonella* Species in Broilers, a National Study (1990–1992) and the USDA Nationwide Broiler Chicken Microbiological Baseline Data Collection Program (1994–1995); data for isolates from beef were from the USDA Nationwide Beef Microbiological Baseline Data Collection Program: Steers and Heifers (1992–1993) and the USDA Nationwide Beef Microbiological Baseline Data Collection Program: Cows and Bulls (1993–1994); and data for isolates from pork were from the USDA Nationwide Pork Microbiological Baseline Data Collection Program: Market Hogs (1995–1996).

[a] Includes the 5 most common serotypes in humans and chickens, 4 of the 5 most common serotypes in market hogs, and 3 of the 5 most common serotypes in beef.

[b] Includes *Salmonella* Typhimurium var copenhagen.

among food animals and assuming (1) that raw food of animal origin is the primary source of human salmonellosis, (2) that the risk of transmission to humans is equal for each of the food product categories evaluated, and (3) that all serotypes have an equal ability to cause illness in humans. A simulation study was used to assess the significance of the observed discrepancy between "expected" and "observed" serotype distributions. In subsequent analyses, variables were manipulated to assess the impact on the model of changes in the risk associated with specific food products and/or differences in the ability of a specific serotype to cause disease. The models are presented in detail in the Appendix.

Results

Salmonella *serotypes in humans.* The numbers of cases of salmonellosis in humans that were reported between 1990 and 1995 through the National *Salmonella* Surveillance System are shown in table 1; data are included on percentage of distribution of the 5 most common *Salmonella* serotypes found in humans: Typhimurium, Enteritidis, Heidelberg, Newport, and Hadar.

Salmonella *serotypes in food animals.* In the USDA studies cited in Methods, *Salmonella* species were isolated from 22.8% of broiler chickens, 1.8% of beef carcasses, and 8.7% of market hogs sampled. In the broiler studies, the rate of contamination with *Salmonella* species decreased over time from 27.1% in 1990 to 19.6% in 1995 (χ^2 for trend, 11.5; $P < .005$). Heidelberg, Kentucky, Hadar, Typhimurium, and Thompson were the 5 most common *Salmonella* serotypes isolated from broiler chickens (table 1). Isolation of *Salmonella* Kentucky increased over the study period from 10.1% in 1990 to 23.1% in 1995 (χ^2 for trend, 8.0; $P = .005$), whereas *Salmonella* Hadar decreased from 24.0% in 1990 to 8.4% in 1995 (χ^2 for trend, 13.4; $P < .005$). In the studies of beef, *Salmonella* serotypes Typhimurium, Montevideo (12.9% of isolates), Anatum (10% of isolates), Kentucky, and Thompson were most commonly identified, and the *Salmonella* serotypes most commonly isolated from market hogs were Derby, Typhimurium, Heidelberg, Anatum (7% of isolates), and Hadar (table 1).

Application of model. We restricted our analysis to 7 major *Salmonella* serotypes (Typhimurium, Heidelberg, Newport, Hadar, Thompson, Kentucky, and Derby). All other serotypes (with the exception of *Salmonella* Enteritidis, which was excluded from the analysis because of its close association with eggs) were grouped in the category "Other." On the basis of the USDA's 1994–1996 Continuing Survey of Food Intakes by Individuals [4], we assumed an average consumption of beef as 24 g/day (SE, 1), of chicken as 21 g/day (SE, 1), and of pork as 10 g/day (SE, 0.5).

In our initial analysis, we assumed that the probability for causing illness was the same for all *Salmonella* serotypes and for each food product category. Table 2 contrasts the predicted proportion of serotypes in human infections, under these assumptions, to the proportions actually observed in the CDC database; for example, our model predicts that *Salmonella* Kentucky should constitute 14% of all isolates from humans; however, in reality, <1% of cases in humans are due to this serotype. This degree of mismatch between the expected and observed proportions is highly unlikely to have occurred by chance ($P < .0001$).

Differences in the ability of certain serotypes to cause human illness may account for this mismatch. In a subsequent analysis, we introduced the observed distribution of various serotypes

Table 2. Expected versus actual proportion of *Salmonella* disease caused by each serotype, assuming that serotypes are equally able to cause human illness and that there is equal food product risk.

Salmonella serotype	Observed proportion[a]	Expected proportion (95% CI)[b]
Typhimurium	.29	.12 (.10–.14)
Heidelberg	.09	.15 (.13–.16)
Hadar	.05	.13 (.11–.14)
Newport	.06	.007 (.002–.011)
Thompson	.02	.05 (.04–.06)
Kentucky	.001	.14 (.12–.16)
Derby	<.001	.05 (.04–.06)
Other	.49	.36 (.34–.38)

[a] Analysis excludes *Salmonella* Enteritidis isolates because of their strong association with eggs; therefore, data are the proportion of non–*Salmonella* Enteritidis isolates of each serotype and therefore are different from the proportions given in table 1.

[b] Expected proportions based on model described in the Appendix. Confidence intervals (CIs) are based on a percentile bootstrap method.

in humans from the CDC data set and solved for a serotype-specific "ability to cause human illness" variable, V_i, while maintaining product risk constant. Results are shown in table 3. Assigning *Salmonella* Typhimurium an arbitrary "ability to cause human illness" factor of 1, the observed *Salmonella* serotype distribution among humans could be accounted for if food products contaminated with *Salmonella* Kentucky were 200-fold less likely to cause human illness, when compared with products contaminated with *Salmonella* Typhimurium; food products contaminated with *Salmonella* Newport were 3.75-fold less likely to cause human illness; and so on.

To explore the impact of differences in product risk on the model, values for V_i were recalculated for scenarios in which it was assumed that one of the food product categories (i.e., chicken vs. beef vs. pork) was 10 times more likely to cause illness than were the others. As shown in table 3, even with changes of this magnitude, the model did not change substantially. Differences in virulence were still apparent when the relative product risk for each of the 3 foods was set at the most extreme value (e.g., 0).

Discussion

The idea of using serotype data to assess the relationship between *Salmonella* isolates found in humans and those of animal origin is not novel. However, the current study is one of the first to make such comparisons by taking advantage of the recent availability of data from large national studies of *Salmonella* isolates from food animal carcasses. The data both for humans and for animals have potential problems. Although methodologies were comparable, each of the studies of animals was conducted over a limited time span (in general, 2 years), and the number of positive samples and isolates serotyped is small, particularly for isolates from beef. Human data from the National *Salmonella* Surveillance System reflects passive reporting, there is considerable state-to-state variation in obtaining cultures and reporting isolates, reported isolates represent a mix of outbreak-associated and sporadic cases, and the surveillance data represent only a small fraction of the actual number of *Salmonella* infections in the United States, perhaps as few as 1% [5]. Nonetheless, the systematic collection of data and the national scope of the studies provide a reasonable starting point for making comparisons between isolates from humans and those from animals after slaughter.

For some serotypes, such as *Salmonella* Hadar, common trends across time in human and animal populations is apparent. *Salmonella* Hadar [6] was introduced into US commercial turkey flocks in the late 1970s and subsequently appeared in feed products and in chicken flocks [7]; reporting of this isolate as a major cause of human salmonellosis peaked in 1988. Given the association between *Salmonella* Hadar and poultry, the decreasing contamination of broiler chickens with *Salmonella* Hadar, from 24% in 1990 to 8% in 1995, may have been responsible for the decrease in *Salmonella* Hadar isolates from humans during the same time period. The picture for other serotypes is not as clear without an immediate, obvious correlation between the distribution of *Salmonella* serotypes among animals and that among humans.

This is, however, a very complex system. In an effort to deal with these complexities, we developed a mathematical model to relate contamination rates of raw product, consumption rates, and serotype distribution. In our initial application of this model, we assumed that all human salmonellosis (with the exception of cases caused by *Salmonella* Enteritidis) came from raw beef, chicken, or pork; that the risk of transmission to humans was equal for all food product categories; and that *Salmonella* serotypes did not differ in their ability to cause human illness. The resulting mismatch between the "expected" distribution of *Salmonella* serotypes among humans that was calculated using this model and the "real" serotype distribution pattern reported by the CDC was not unanticipated. However, the finding of such a mismatch provides a basis for questioning some of the standard assumptions used in our model.

Salmonella species clearly have multiple entry points into human populations. Traditional thinking is that the primary reservoir for the organism is the intestinal tract of food animals, with transmission associated with contamination of raw animal product by *Salmonella* species during slaughter and processing [2, 8–10]. There are raw animal products other than beef, chicken, and pork that can be contaminated with the microorganism (e.g., turkeys or raw [unpasteurized] milk). However, these products constitute a relatively small proportion of the total market; for example, in 1994, turkeys accounted for only 3.6% of poultry slaughtered in the United States (USDA, unpublished data).

Table 3. Relative ability (95% confidence interval) of each *Salmonella* serotype to cause human illness, under various assumptions of food product risk.

Salmonella serotype	Assuming equivalent product risk	Assuming 10-fold higher risk from beef	Assuming 10-fold higher risk from chicken	Assuming 10-fold higher risk from pork
Typhimurium	1.00	1.00	1.00	1.00
Heidelberg	0.25 (0.20–0.30)	0.46 (0.32–0.60)	0.18 (0.15–0.23)	0.49 (0.36–0.65)
Hadar	0.15 (0.12–0.19)	0.28 0.20–0.38)	0.11 (0.08–0.14)	0.44 (0.33–0.58)
Newport	3.75 (2.07–9.67)	2.95 (1.30–15.96)	2.98 (1.63–8.52)	10.18 (6.15–33.48)
Thompson	0.17 (0.13–0.22)	0.23 (0.15–0.36)	0.12 (0.09–0.17)	0.59 (0.39–0.76)
Kentucky	0.005 (0.004–0.006)	0.006 (0.004–0.009)	0.003 (0.003–0.004)	0.0014 (0.0011–0.0019)
Derby	0.0004 (0.0001–0.0006)	0.0007 (0.0002–0.0011)	0.002 (0.0005–0.0029)	0.0002 (0.000045–0.00022)
Other	0.56 (0.47–0.65)	0.46 (0.35–0.59)	0.54 (0.44–0.66)	0.71 (0.57–0.87)

Products that have been cooked or further processed before sale to consumers generally have been considered to be less of a risk to humans than raw products, an assumption which may not be completely correct. Similarly, there may be a need to further explore the relative contribution, to the total *Salmonella* burden, of other possible reservoirs, including raw produce, pets, and colonized human food handlers.

In our initial model, we assumed that risk was equal for all animal product classes (i.e., beef, chicken, and pork). There also may be problems with this assumption. Consumers have been told repeatedly about risks associated with raw chicken and may take greater precautions in the kitchen when handling chicken than when handling beef or pork, to avoid cross-contamination. There definitely are differences in cooking practices, with chicken traditionally cooked well done, compared with beef (including ground beef), which may get minimal cooking. Surveys in the early 1990s indicated that 23%–25% of US consumers preferred their hamburgers cooked rare or medium rare [11]. However, product risk did not appear to have as strong an impact on our model as did potential differences in the ability of certain serotypes to cause human illness. The model was not greatly affected by 10-fold increases in the risk of specific product classes, and, even when product risk was maximized, it was still necessary to include serotype-specific differences in the ability to cause illness, to match the observed distribution of *Salmonella* serotypes among humans.

The concept that all *Salmonella* serotypes are equally able to cause human disease follows, in part, from early volunteer studies with several *Salmonella* serotypes, including Typhimurium, Anatum, Meleagridis, Newport, Derby, Bareilly, Pullorum, Sofia, and Bovis-morbificans [12]. However, these studies were limited in size and scope and did not include many of the serotypes that are now commonly isolated from animals or humans. Studies of animals have clearly shown that certain *Salmonella* serotypes are more virulent than others [13]. It also is recognized that certain *Salmonella* serotypes are more "human adapted" and more likely to cause invasive disease and bacteremia [13–15]. In this setting, it is reasonable to hypothesize that serotypes also differ in their overall ability to infect the human intestinal tract and cause illness, which is related to factors such as differences in virulence and infectious dose. The "ability to cause human illness" variable in our model reflects this concept. The actual values provided by the model must be interpreted carefully in view of the uncertainties about our underlying assumptions and the quality of the data sets. Nonetheless, our results raise serious questions about the appropriateness of assigning equal public health significance to colonization of a food product with *Salmonella* Kentucky versus *Salmonella* Typhimurium (or *Salmonella* Newport).

The differences in "ability to cause human illness" noted by the model also may reflect segregation of isolate populations among humans and animals—that is, certain serotypes may be transmitted preferentially within human populations, whereas other serotypes may be limited primarily to animal populations. If true, this hypothesis raises further questions about our assumption that animals are the predominant source for the *Salmonella* strains that cause human illness. In reality, we probably are dealing with a complex natural system in which certain serotypes are preferentially transmitted by specific raw food products, whereas others are derived from alternative sources. These complexities need to be considered when regulatory approaches to *Salmonella* species control are being developed. In addition, our studies underscore the need for further data collection, for biologic studies of serotype-specific differences in disease causation, and for more-sophisticated modeling that will allow us to understand the ebb and flow of these pathogens through human and animal populations.

Appendix

Mathematical Model

Let P_i stand for the probability that a person in the population acquires disease caused by *Salmonella* of serotype i from a random meal, where i ranges from 1 to k different serotypes. Then, the proportion of cases of human salmonellosis caused by a particular serotype, i_0, is

$$\frac{P_{i_0}}{\sum_{i=1}^{k} P_i} . \qquad \text{(A1)}$$

Considering only those serotypes transmitted exclusively by beef, chicken, or pork, $P_i = P_{i_b} + P_{i_c} + P_{i_p}$, where P_{i_b}, P_{i_c}, and P_{i_p} refer to the probability of acquiring salmonella of type i from beef, chicken, or pork, respectively. P_{i_b} can be calculated as the product of the following terms: b, the probability that a meal will include beef; b_S, the probability that the beef is contaminated with *Salmonella* species; $b_{i|S}$, the probability that *Salmonella* species in beef will be serotype i; and $b_{\text{inf}|i}$, the probability that consumption of the beef with *Salmonella* serotype i will lead to human infection.

We modeled $b_{\text{inf}|i}$ as the product of terms related to the ability of the serotype to cause human illness and to the effect of beef (relative to chicken or pork) on the probability of infection ("product risk"). These parameters may be denoted as V_i and b_r, respectively.

P_{i_c} and P_{i_p} can be calculated as the products of similarly defined terms. Substituting these new parameters into expression (A1), the proportion of cases of human salmonellosis caused by serotype i_0 can be expressed as

$$\frac{bb_S b_{i_0|S} V_{i_0} b_r + cc_S c_{i_0|S} V_{i_0} c_r + pp_S p_{i_0|S} V_{i_0} p_r}{\sum_{i=1}^{k} (bb_S b_{i|S} V_i b_r + cc_S c_{i|S} V_i c_r + pp_S p_{i|S} V_i p_r)} . \qquad \text{(A2)}$$

Under the assumptions that each *Salmonella* serotype has an equal ability to cause illness and that the risk of infection is equal for all 3 product classes (beef, chicken, and pork), the ability to cause illness and the product risk parameters cancel from expression (A2). Thus, expression (A2) reduces to the following:

$$\frac{bb_{\mathrm{S}}b_{i_0|\mathrm{S}} + cc_{\mathrm{S}}c_{i_0|\mathrm{S}} + pp_{\mathrm{S}}p_{i_0|\mathrm{S}}}{\sum_{i=1}^{k}(bb_{\mathrm{S}}b_{i|\mathrm{S}} + cc_{\mathrm{S}}c_{i|\mathrm{S}} + pp_{\mathrm{S}}p_{i|\mathrm{S}})}. \quad \text{(A3)}$$

Data are available to estimate all the parameters in expression (A3). Therefore, using these estimates, it is possible to calculate the expected proportion of cases of *Salmonella* infection in humans that is due to each serotype. We compared the expected proportions under this model to the observed proportions in our data and computed a measure of discrepancy (defined as the sum of the absolute differences between the expected and observed proportions for each serotype). To determine whether the observed discrepancy could have been due to the sampling variation of our parameter estimates, we simulated 10,000 sets of parameter estimates from their estimated distributions and 10,000 corresponding values of the discrepancy calculated under the assumptions of equal virulence and product risk. We then compared the discrepancy observed in our data with the distribution of discrepancies calculated from these simulated estimates.

Data are available for all the parameters in expression (A2), with the exception of the paramaters for serotype-specific ability to cause illness (V_i) and for product risk (b_r, c_r, and p_r). To determine the relative ability of the different serotypes to cause human illness, we substituted the observed proportion of cases of *Salmonella* infection caused by each serotype and all the available parameter estimates into expression (A3) for each of the k serotypes. Then, we solved these equations for V_i under various assumptions about the values for the product risk variables. There are no unique solutions for V_i: if the equations are solved by the set V_i^*, then the equations also will be solved by V_i^* multiplied by an arbitrary constant. Thus, there is no information in these equations about absolute values for V_i; however, there is information in these equations about relative values for V_i. To find the relative ability of each serotype to cause illness, we set the value of V_i to 1.0 for one of the strains and then solved for the remaining $k-1$ values of V_i. This resulted in a linear system of $k-1$ equations and $k-1$ unknowns, so that the solution was available in closed form.

The resulting estimates of relative ability to cause illness can be thought of as maximum likelihood estimates, since they result from substituting maximum likelihood estimates (sample proportions) for each parameter in the model. To construct confidence intervals, we used the percentile bootstrap method, simulating parameter estimates as described above.

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