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Attribution of *Salmonella enterica* serotype Hadar infections using antimicrobial resistance data from two points in the food supply system

A. R. VIEIRA[1]*, J. GRASS[1], P. J. FEDORKA-CRAY[2], J. R. PLUMBLEE[2], H. TATE[3] AND D. J. COLE[1]

[1] *Enteric Diseases Epidemiology Branch, Centers for Disease Control and Prevention, Atlanta, GA, USA*
[2] *U.S. Department of Agriculture/Agricultural Research Service, Athens, GA, USA*
[3] *U.S. Food and Drug Administration, Silver Spring, MD, USA*

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SUMMARY

A challenge to the development of foodborne illness prevention measures is determining the sources of enteric illness. Microbial subtyping source-attribution models attribute illnesses to various sources, requiring data characterizing bacterial isolate subtypes collected from human and food sources. We evaluated the use of antimicrobial resistance data on isolates of *Salmonella enterica* serotype Hadar, collected from ill humans, food animals, and from retail meats, in two microbial subtyping attribution models. We also compared model results when either antimicrobial resistance or pulsed-field gel electrophoresis (PFGE) patterns were used to subtype isolates. Depending on the subtyping model used, 68–96% of the human infections were attributed to meat and poultry food products. All models yielded similar outcomes, with 86% [95% confidence interval (CI) 80–91] to 91% (95% CI 88–96) of the attributable infections attributed to turkey, and 6% (95% CI 2–10) to 14% (95% CI 8–20) to chicken. Few illnesses (<3%) were attributed to cattle or swine. Results were similar whether the isolates were obtained from food animals during processing or from retail meat products. Our results support the view that microbial subtyping models are a flexible and robust approach for attributing *Salmonella* Hadar.

Key words: Analysis of data, foodborne infections, surveillance.

INTRODUCTION

Each year, an estimated 1 million illnesses in the United States are caused by non-typhoidal *Salmonella* transmitted by food, resulting in more than 19 000 hospitalizations and 350 deaths [1]. Estimates of the proportion of illnesses attributable to different food sources would help public health officials set priorities for mitigating risk. Several countries use microbial subtyping source-attribution models to attribute enteric illnesses to various food sources. These models require data characterizing bacterial isolate subtypes to attribute human illnesses based on the relative distributions of different subtypes among temporally related human illnesses and food sources [2]. Models usually combine the results of *Salmonella* serotyping with additional subtyping methods, such as pulsed-field gel electrophoresis (PFGE), multilocus variable-number tandem-repeat analysis (MLVA), phage typing, or antimicrobial susceptibility testing [3–5]. In the United States, antimicrobial susceptibility and PFGE data are available for *Salmonella*.

Most *Salmonella* microbial subtyping models attribute illnesses to reservoirs (defined here as a source

* Author for correspondence: Dr A. R. Vieira, Enteric Diseases Epidemiology Branch, Centers for Disease Control and Prevention, Atlanta, GA, USA.
(Email: vht8@cdc.gov)

the pathogen depends on for survival), which are commonly food animals [6]. These models attribute illnesses to sources before cross-contamination that can occur during processing or handling at retail outlets [2]. Food safety, however, requires interventions at multiple points in the food chain. Consequently, studies are needed that can attribute illnesses along the food chain from the farm to processing plants to retail settings. In this study, we evaluated the use of antimicrobial resistance data on isolates of *Salmonella* serotype Hadar, a strongly foodborne associated serotype, collected from ill humans, food animals at the point of processing following slaughter (hereafter referred to as food animals), and retail meats, in two microbial subtyping attribution models. We compared results with those from two models using PFGE as the subtyping method. These comparisons were done with the objective of evaluating the use of data on *Salmonella* contamination on retail foods and data on antimicrobial resistance (*vs.* PFGE) in microbial subtyping models.

MATERIALS AND METHODS

Data sources

The National Antimicrobial Resistance Monitoring System (NARMS) conducts antimicrobial susceptibility testing on *Salmonella enterica* isolates collected from humans, retail meats, and food animals. NARMS is a collaboration between the Centers for Disease Control and Prevention (CDC), the U.S. Food and Drug Administration (FDA), and the U.S. Department of Agriculture (USDA) [7]. CDC began the human surveillance component of NARMS in 1996, FDA launched the retail meat surveillance component of NARMS in 2002, and USDA started the food animal surveillance component of NARMS in 1997.

We used antimicrobial resistance patterns among serotype Hadar isolates from humans, food animals, and retail food submitted to NARMS. Isolates from ill people were obtained from specimens submitted to clinical laboratories and forwarded to state public health laboratories. Initially, participating states submitted every tenth non-typhoidal *Salmonella* isolated from humans to CDC's NARMS laboratory. When NARMS went nationwide in 2003, sampling decreased to every twentieth isolate. NARMS retail meat monitoring was conducted by the FDA's Center for Veterinary Medicine as previously described [8]. NARMS monitoring of food animals was conducted by USDA's Bacterial Epidemiology and Antimicrobial Resistance Research Unit of the Agricultural Research Service as previously described [9]. Our analysis included susceptibility-tested isolates of *Salmonella enterica* serotype Hadar from humans with a specimen collection date from 1996 to 2012, susceptibility-tested food animal isolates (chicken, swine, turkey, cattle) with a sample collection date from 1997 to 2012, and susceptibility-tested retail meat isolates (chicken parts, pork chops, ground turkey, ground beef) with a sample collection date from 2002 to 2012.

Broth microdilution (Sensititre, Trek Diagnostics Systems, Thermo Fisher Scientific Inc., USA) was used to determine the minimum inhibitory concentration levels for antimicrobial agents tested during all years of this analysis, including amoxicillin/clavulanic acid, ampicillin, cefoxitin, ceftiofur, ceftriaxone, chloramphenicol, ciprofloxacin, gentamicin, nalidixic acid, streptomycin, kanamycin, sulfisoxazole, tetracycline, and trimethoprim/sulfamethoxazole. We only included result for susceptibility testing with antimicrobial drugs used in every year of the study period. Resistance was defined by the Clinical and Laboratory Standards Institute (CLSI) interpretive standards, when available [10]. For streptomycin, where no CLSI interpretive criteria for human isolates exist, the resistance breakpoint was 64 μg/ml (FDA, 2009). Testing was performed according to the manufacturer's instructions using the following quality-control strains: *Escherichia coli* ATCC 25 922, *Staphylococcus aureus* ATCC 29 213, *Enterococcus faecalis* ATCC 29 212, and *Pseudomonas aeruginosa* ATCC 27 853.

We analysed PFGE patterns based on single-enzyme *Xba*I fragments for all serotype Hadar isolates (from humans, food animals, and retail meats) collected from 1999 to 2012 by PulseNet, the national molecular subtyping network for enteric bacteria, which is coordinated by CDC. Public health laboratories follow the same PFGE subtyping protocols and standards, making it possible to compare the relatedness of bacteria based on DNA banding patterns resolved using electrophoresis (http://www.cdc.gov/pulsenet/PDF/ecoli-shigella-salmonella-pfge-protocol-508c.pdf). The PulseNet database contains PFGE data on human isolates collected through NARMS, food animal isolates tested by USDA VetNet (a network created in 2003 to determine PFGE patterns of animal isolates collected from USDA Food Safety and Inspection Service (FSIS) inspected slaughter and processing facilities), and from retail meat isolates tested by FDA [11, 12].

Data analysis

A resistance profile was determined for each isolate tested in NARMS. We considered each discrete antimicrobial resistance profile or PFGE *Xba*I pattern to represent a unique subtype of serotype Hadar. For data collected from food animals, we estimated subtype prevalence in each source by dividing the number of samples from an animal source in which cultures yielded that subtype by the total samples from that source that were cultured for *Salmonella* (USDA FSIS verification testing). Because the number of samples from retail meat cultured for *Salmonella* was similar for each food source, we assumed the number of samples for which cultures yielded each serotype Hadar subtype in each meat source was an estimate of subtype prevalence (i.e. no denominator was used to estimate subtype prevalence in each meat source).

We developed and compared results from four source-attribution subtyping models:

AR-animal model. Antimicrobial resistance data from food animals were used to subtype isolates and attribute human illnesses.

AR-retail model. Antimicrobial resistance data from retail meats were used to subtype isolates and attribute human illnesses.

PFGE-animal model. PFGE *Xba*I data from food animals were used to subtype isolates and attribute human illnesses.

PFGE-retail model. PFGE *Xba*I data from retail meats were used to subtype isolates and attribute human illnesses.

We used a direct estimation method first proposed in The Netherlands to find the proportion of human infections attributable to each source [13]. We calculated the number of human serotype Hadar infections attributable to each source using the estimated prevalence of isolate subtypes in each source:

$$\lambda_{ji} = \left(P_{ij}/\sum\nolimits_j P_{ij}\right)^* o_i,$$

where λ_{ij} is the expected number of infections of subtype i attributed to source j; P_{ij} is the proportion of subtype i isolates from source j; and o_i is the observed number of infections per subtype.

For each subtype, the number of human infections attributed to each source was proportional to how often that subtype was isolated from that food or animal source, compared to the other sources. For example, if 20 of 100 isolates of a single subtype came from source A and 80 from source B, then four times more infections were attributed to source B. The expected number of infections attributed to each source is the sum of infections caused by all subtypes attributed to that source ($\sum i\lambda_{ij}$). Infections with a subtype not detected in any of the sources were not attributed to a source. We calculated uncertainty around model estimates using bootstrap resampling of the source isolates and infections. For each model, we obtained a set of 1000 replicates from the original data to generate a bootstrap distribution of food source-attribution fractions associated with each source. Then we used the two values containing the central 95% of the bootstrap distribution as estimates of the 95% confidence interval (CI), also referred to as the percentile method. Models were set up using SAS Enterprise Guide 4.3 (SAS Institute Inc., USA).

Using the AR-animal model, we also estimated source attribution for four periods: 1996–2000, 2001–2004, 2005–2008, and 2009–2012.

RESULTS

We obtained antimicrobial resistance patterns of 345 human isolates and PFGE patterns of 2303 human isolates of *Salmonella* serotype Hadar isolated from 1996 to 2012. We obtained antimicrobial resistance patterns of 1406 food animal isolates and PFGE patterns of 443 food animal isolates of serotype Hadar isolated from 1997 to 2012. We obtained antimicrobial resistance patterns of 305 retail meat isolates and PFGE patterns of 260 retail meat isolates of serotype Hadar isolated from 1997 to 2011. Table 1 shows data on the number of different food animal and retail food samples analysed and the proportion that yielded serotype Hadar. A higher proportion of samples from turkeys and ground turkey than other sources yielded serotype Hadar.

We identified 29 antimicrobial resistance patterns, varying in resistance from none to seven antimicrobials, among the 345 human isolates (28 pansusceptible isolates). Twenty of these patterns (comprising 96% of 345 isolates) were also present in food animal isolates, and nine (84% of 345 isolates) were also present in retail meat isolates.

In contrast, we found 76 antimicrobial resistance patterns, varying in resistance from none to 12 antimicrobials, among the 1406 food animal isolates (80 pansusceptible isolates). Twenty-four of these patterns were also present in more than one type of food animal; 91% of food animal isolates had one of these

Table 1. *Summary of testing of food animal and retail meat isolates of* Salmonella *serotype Hadar, 1997–2012*

	Food animals after slaughter					Retail meat				
		Serotype Hadar					Serotype Hadar			
		Antimicrobial resistance		PFGE			Antimicrobial resistance		PFGE	
Animal source (food animal/retail meat)	Samples analysed (*n*)	Isolates tested (*n*)	Patterns identified (*n*)	Isolates tested (*n*)	Patterns identified (*n*)	Samples analysed (*n*)	Isolates tested (*n*)	Patterns identified (*n*)	Isolates tested (*n*)	Patterns identified (*n*)
Turkeys/ground turkey	24 305	880	64	368	20	12 675	264	31	228	39
Chicken/chicken breasts	124 923	446	32	23	3	12 713	32	6	31	7
Swine/pork chops	74 626	52	6	45	6	12 716	9	3	1	1
Cattle/ground beef	355 316	28	9	7	4	12 741	0	0	0	0
Total	579 170	1406	—	443	—	50 845	305	—	260	—

PFGE, Pulsed-field gel electrophoresis.

24 patterns. We observed 42 resistance patterns (8% of food animal isolates) only among turkey isolates. Nine patterns (1% of food animal isolates) were unique to chicken, one pattern was observed in a single isolate from swine, and no pattern was unique to cattle.

We found 33 resistance patterns, varying in resistance from none to nine antimicrobials, among the 345 retail meat isolates (five pansusceptible isolates). Six patterns (75% of retail meat isolates) were present in more than one retail meat source. Twenty-five patterns (24% of retail meat isolates) were present only among turkey isolates, and two patterns (1% of retail meat isolates) were present only in chicken isolates.

We identified 248 PFGE *Xba*I patterns among human isolates. Nineteen of these patterns (70% of isolates) were present in food animal isolates, and 31 (68% of isolates) were present in retail meat sources. In contrast, we found only 23 PFGE *Xba*I patterns among food animal isolates. Turkey isolates had 20 PFGE *Xba*I patterns (368 isolates), whereas chicken isolates had six PFGE *XbaI* patterns (45 isolates), swine isolates had three patterns (23 isolates), and cattle isolates had four patterns (seven isolates). We observed 12 PFGE patterns only among turkey isolates, and two PFGE patterns were unique to chicken isolates. No PFGE patterns were unique to swine or cattle.

We found 43 PFGE *Xba*I patterns among retail meat isolates. Ground turkey isolates had 39 PFGE *Xba*I patterns (228 isolates), whereas chicken isolates had seven *Xba*I patterns (31 isolates). We observed 35 PFGE patterns only among ground turkey isolates and four PFGE patterns unique to chicken isolates. There were no PFGE *Xba*I data available from ground beef and pork chop isolates obtained at retail.

Among the models using antimicrobial susceptibility testing data to subtype isolates, the model that used data from food animals attributed more illnesses to sources (96%, 95% CI 91–98) than the model that used retail meat data (84%, 95% CI 80–88). Both models using antimicrobial susceptibility testing data attributed more human infections to a source than those using PFGE data, which attributed only 70% (95% CI 66–74) of infections to a source using food animal data and 68% (95% CI 61–73) to a source using retail meat data.

All models yielded similar estimates, with most human infections attributed to turkey. For the models using antimicrobial susceptibility testing data, among attributed infections, 86% (95% CI 82–89) were attributed to turkey using animal data and 86% (95% CI

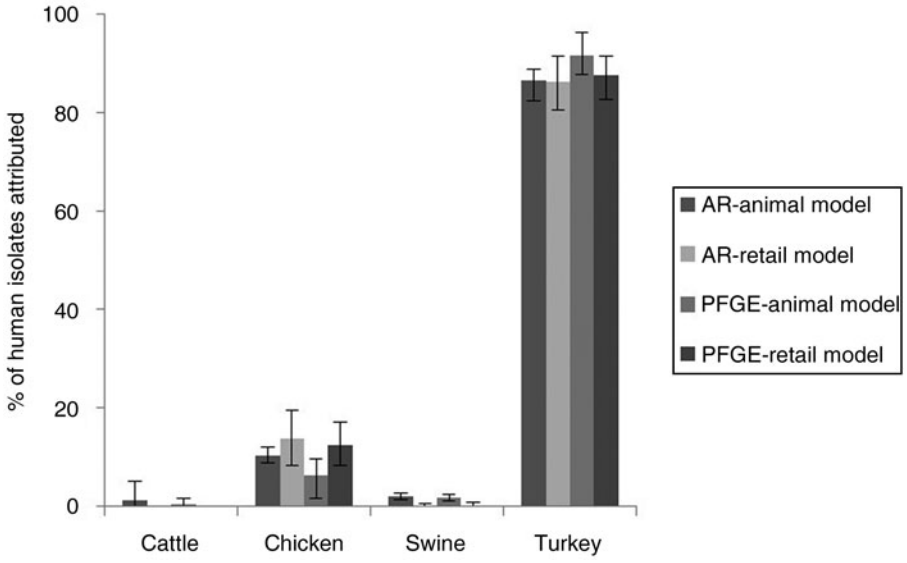

Fig. 1. Proportion of *Salmonella enterica* serotype Hadar human isolates[a] attributable to each source using four subtyping models[b], 1997–2012. [a] Proportions based on attributed isolates only. [b] Source-attribution subtyping models used: antimicrobial resistance (AR) data from animal isolates to attribute human illnesses to food animal sources (AR-animal model); antimicrobial resistance data from animal isolates to attribute human illnesses to retail meat isolates (AR-retail model); pulsed-field gel electrophoresis (PFGE) data from animal isolates to attribute human illnesses to food animal sources (PFGE-animal model); PFGE data from retail meat isolates to attribute human illnesses to sources at retail (PFGE-retail model).

80–91) using retail turkey meat. Chicken was the next most prevalent source; 10% (95% CI 9–12) of infections were attributed to chicken using data from chicken and 14% (95% CI 8–20) using data from retail chicken. For the models using PFGE data, among attributed infections, 91% (95% CI 88–96) were attributed to turkey using food animal data and 87% (95% CI 82–91) to turkey using meat data (Fig. 1). Few illnesses were attributed to beef or pork in any of the models. Because more isolates were available from food animals than retail food sources, and more of these had antimicrobial susceptibility than PFGE results, attribution estimates from the model using these data had the smallest confidence intervals.

Most infections were attributed to turkey during each of the four periods. This proportion was lowest during 2009–2012, when the proportion not attributed to any source was also highest (Fig. 2).

DISCUSSION

Our results demonstrate that antimicrobial resistance data can be used to characterize pathogen subtypes in a microbial subtyping source-attribution model, providing similar results to models using PFGE data. However, models using antimicrobial resistance data to subtype serotype Hadar attributed more human infections to sources than models based on PFGE data. This resulted from the higher number of isolates with antimicrobial resistance data available and the many resistance subtypes detected among isolates.

Use of our model to attribute human infections to sources assumes each antimicrobial resistance pattern is stable over time in different food sources. Likewise, we assumed antimicrobial resistance had not developed during treatment of people from whom samples were collected. Using this model to assess change over time also requires the assumption that variability in resistance to antimicrobials is driven mostly by changes in resistance among isolates from food animals, not by changes among human isolates. The attribution estimates were fairly similar over time, suggesting that among the sources of serotype Hadar included in our model, the relative role of each has not changed over the past several years. However, FSIS increased its targeted sampling of food animals in 2006 because of a new risk-based criteria [14]. If this change in sampling resulted in higher frequencies of *Salmonella* isolation from food sources, or higher probabilities of selection of resistant isolates from food sources, the model estimating changes over time could have overestimated

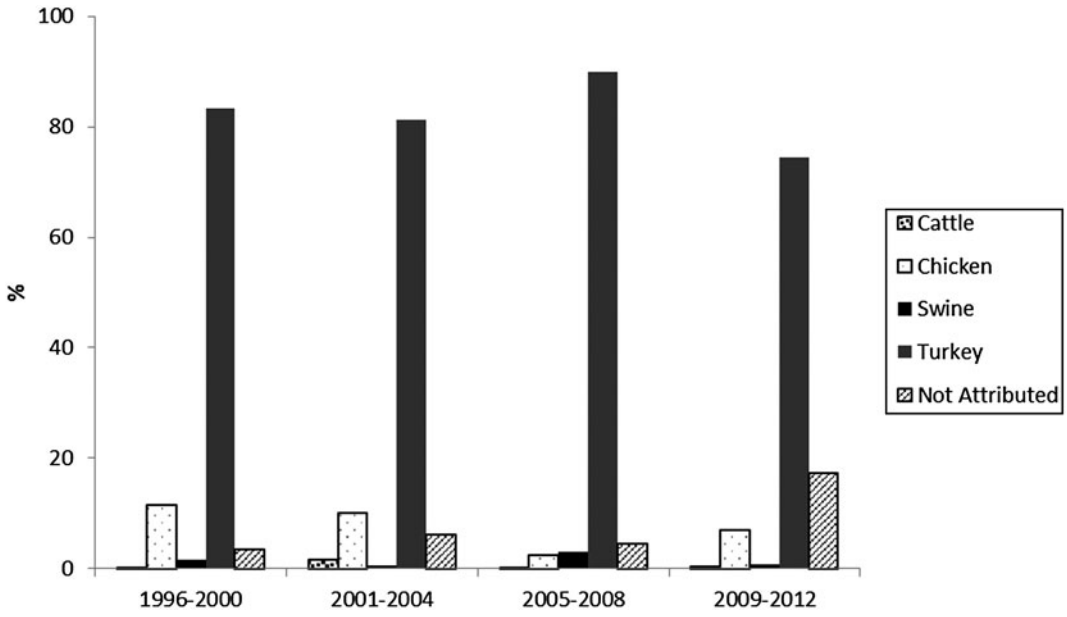

Fig. 2. Proportion of human *Salmonella enterica* serotype Hadar isolates attributable to food animal sources, using antimicrobial resistance data, 1996–2012.

the increase in the percentage of human infections attributable to turkey in 2005–2008.

Microbial subtyping attribution models are based on the assumption that pathogen subtypes are heterogeneously distributed among food sources [2]. Therefore, microbial subtyping models using one level of subtyping, such as *Salmonella* serotype [15, 16] to distinguish between pathogen strains may not be as reliable as those using more discriminatory subtyping methods that increase the reservoir specificity of isolates. Consequently, countries with well-integrated surveillance systems have proposed the use of additional levels of subtyping to improve the discriminatory ability of attribution models, such as subtypes defined by both serotype and PFGE pattern, or subtypes defined by serotype, phage type, and antimicrobial resistance patterns [3]. Hald *et al.* [5] first demonstrated the utility of antimicrobial resistance data on a microbial subtyping model. In that study, better distinction between isolates from different sources was achieved through the inclusion of phage typing on fully susceptible isolates. In our study, inclusion of antimicrobial resistance data resulted in greater discrimination of isolates obtained from food animal isolates than PFGE patterns. Inclusion of multiple *Salmonella* serotypes and antimicrobial resistance data may provide better discrimination of sources by increasing the heterogeneity of isolates among sources in the model. However, models using antimicrobial resistance data rely on the assumption that the distributions of antimicrobial resistance differ among specific animal sources, which may be valid for some serotypes but not others.

Defining subtypes for more than one serotype using antimicrobial resistance data may result in decreased discriminatory power if all serotypes in the model are not equally likely to exhibit specific antimicrobial resistance patterns when isolated from the same source. Another limitation of using antimicrobial resistance data for subtyping is the assumption that resistance phenotypes that are used to define a pattern are independent. Some genes, like bla_{CMY}, can cause resistance to multiple antimicrobial drugs and may affect the results of the attribution model.

Several microbial subtyping attribution models use surveillance data from animal isolates obtained at the farm and attribute human infections to the animal reservoir [17]. However, Guo *et al.* [15] attributed human infections to the point of food animal processing and assumed that all human infections are attributable to processed animal products or food products contaminated by animal products after processing. In our study, we attributed infections to source isolates sampled from different points of the food chain and compared the results. It is possible that differences in attribution results when using data from food animals *vs.* retail meats reflect changes in contamination that may occur between these points in the food chain. However, the differences in attribution results may simply reflect different data sources. Moreover, the food

products sampled at each point were not the same. The retail foods sampled were a subset of the foods derived from the food animals. Nonetheless, both models attributing human infections to retail meats and to food animals yielded similar results, suggesting heterogeneity in antimicrobial resistance patterns among sources is stable between these two points in the food system. The model that attributed infections using data from food animals attributed slightly fewer illnesses to chicken and slightly more to turkey than the model that used retail food data. Although not significantly different, this attribution may reflect relatively lower contamination of chicken parts relative to other chicken products sampled during slaughter and processing, or it may reflect factors after processing such as handling practices, refrigeration, or cross-contamination of foods at retail. Comparison of the results of models that use animal *vs.* food isolate data may suggest the original reservoir sources or points of contamination for pathogens introduced via other pathways. Using data at a level below the reservoir would allow targeting pathogens that are prone to disseminate through other pathways in addition to food.

NARMS collects and tests isolates from human infections, food animals after slaughter, and retail meats, providing the opportunity to evaluate estimates of the percentage of human infections attributable to specific food sources at different points in the food chain. Microbial subtyping methods used in source-attribution models need to have similar data collection and isolate analysis methods used on all samples from humans and food sources so that subtypes are characterized uniformly across all possible sources. We had more data on antimicrobial resistance from *Salmonella* isolates obtained from humans, food animals, and retail meats readily available in NARMS than PFGE data in PulseNet. The high level of standardization of PulseNet and NARMS surveillance makes both ideal for source-attribution models. PulseNet participating laboratories must meet annual proficiency testing requirements for analysis and interpretation. NARMS, which has collected a similar number of isolates from different food types, requires participating laboratories to use the same antimicrobials and interpretive criteria for isolates collected from each sample source and perform quality assurance exercises to ensure similar results for a given panel of isolates.

The direct estimation approach to source attribution used in our analysis assumes that all food sources contaminated with serotype Hadar are equally likely to cause human illness. More sophisticated subtyping attribution models have been proposed [3, 4]. However, since the objective of our study was to compare the use of different subtyping methods (antimicrobial resistance and PFGE) and data sources, we chose a model approach of easier application and interpretation. By applying the same approach on the four proposed models, we do not expect that the choice of modelling approach impacts our model comparisons. Likewise, our approach does not address differences in exposure to individual sources, and microbial subtyping attribution models that include estimates of consumption for each food source may yield more reliable attribution estimates [17]. Also, reported international travel and outbreak association could not be assessed for the human infections included in the analysis and this may cause bias in the attribution estimates. In addition, we assumed that human infections included in the analysis are attributable to one of the sources in the model. Previous studies suggest that the sources we included are the major food sources of serotype Hadar, but pigs were estimated as the most important source of this serotype in Europe [18–20], highlighting the importance of considering potential spatial variations in the sources of *Salmonella* serotypes. Other possible sources and transmission pathways should be investigated if surveillance data are available. We were unable to use this approach to study serotypes that do not have systematically collected surveillance data for major reservoirs of human infection (e.g. *Salmonella* serotype Enteritidis is associated with shell eggs, but we lacked microbiological data on *Salmonella* in eggs). Likewise, attribution of human infections to non-foodborne sources of *Salmonella* cannot be done using this model because of the lack of surveillance data.

Our source-attribution models using data from food animals and from retail meats yielded similar attribution estimates, suggesting that subtyping data collected from points in the food production chain other than the reservoir may be used in source-attribution studies assessing *Salmonella* Hadar food sources. Although we developed four models to demonstrate the method, models that combine data from several subtyping methods and several sources might increase the proportion of illnesses that can be attributed to sources.

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DECLARATION OF INTEREST

None.

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