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Non-typhoidal human salmonellosis in Rio Grande do Sul, Brazil: A combined source attribution study of microbial subtyping and outbreak data

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ABSTRACT

Salmonella spp. remains the most significant foodborne pathogen in south Brazil, but its epidemiology tends to change over time. Using official and surrogate data, a microbial subtyping model attributed different *Salmonella* serovars to laying hens, pigs, broilers, and turkeys from 2005 to 2015 in Rio Grande do Sul (RS). Additional to the subtyping model, three sub-analyses of outbreak data attributed *Salmonella* spp. in humans to animal and non-animal food. Laying hens/eggs was the most important source of human salmonellosis in RS, with almost 40% (159 cases; 95% credibility interval, 43–247) attribution proportion, followed by pigs reared in Santa Catarina, a neighbor state (34.5%). The *Salmonella* serovars Enteritidis and Typhimurium were the most common serovars involved. Source-related parameters had wide credibility intervals but showed a higher risk of illness from contaminated eggs than from the other three animal-food sources. Analysis of the outbreak data corroborated the findings and indicated signs of decreasing importance for eggs and increasing importance for pork consumption.

Keywords: foodborne zoonoses, outbreaks, risk assessment, *Salmonella* source attribution, surveillance.

1. INTRODUCTION

Non-typhoidal *Salmonella* (NTS) is among the major causal agents of self-limiting gastroenteritis, a local infection that causes diarrhea (Phu Huong Lan et al., 2016) and that can lead to chronic arthritis and invasive infections sometimes. Worldwide, more than that from any other enteric pathogen, *Salmonella* infections from contaminated food alone led to the loss of 6.43 million disability-adjusted life years in 2010, a World Bank metric that adds both the years of life lost and the years lost due to disability (Kirk et al., 2015). In Brazil, NTS is considered the most common foodborne bacterial zoonosis (Finger et al., 2019), including the southern region (Tondo and Ritter, 2012).

The consumption of raw eggs in homemade mayonnaise has been related to outbreaks of the Enteritidis serovar in Brazil (Wagner et al., 2013). However, a decrease in the importance of the Enteritidis serovar has been observed more recently (Reis et al., 2018), reflecting the possible effect of preventive measures adopted in food production chains (Ministry of Agriculture, Livestock, and Supply [MAPA], 2003). Commonly, changes in NTS epidemiology take place through ecological niche occupation by other serovars (Fernandes et al., 2006; Rodríguez et al., 2017). *Salmonella* Typhimurium, related to sporadic invasive salmonellosis cases (Reis et al., 2018), is found in most animal reservoirs and appears as a candidate, as well as emerging *Salmonella* serovars such as Heidelberg, Infantis and Newport (Penha Filho et al., 2019; Reis et al., 2018), distributed in specifically few reservoirs (e.g. broilers) (Voss-Rech et al., 2014).

The associations between some *Salmonella* serovars and specific animal species suggest that some animal-derived foods act as reservoirs for particular serovars (Jackson et al., 2013; Kim,

2010; Nüesch-Inderbinen et al., 2015). Source attribution models have been used for attributing sporadic human cases or outbreaks of salmonellosis to specific sources in the last 20 years (David et al., 2013; Domingues et al., 2012; Glass et al., 2016; Guo et al., 2011; Hald et al., 2004; Miller et al., 2017; Mullner et al., 2009; Pires et al., 2010; Van Pelt et al., 1999; Wahlström et al., 2011). The occurrence of foodborne outbreaks or sporadic infections caused by *Salmonella* could be influenced by the serovars' capabilities for surviving and thriving in different hosts, and by the likelihood of several food vehicles to establish an appropriate environment for *Salmonella* growth, forming specific serovar–source combinations. Inter-serovar and food vehicle differences can be estimated from complementary surveillance data and source attribution models to improve understanding of the dynamics of human salmonellosis.

These models can be classified according to the point of attribution, specifically at exposure or consumption, reservoir, or processing. While microbial subtyping approaches attribute cases at the reservoir, analyses of data from outbreak investigations attribute it at consumption. All microbial subtyping models require a collection of temporally and spatially related isolates from humans and various sources. Therefore, an integrated foodborne disease surveillance program that provides a collection of isolates from major animal reservoirs of the foodborne pathogen would enable the construction of such models, based on the distribution of isolate subtypes across different sources and humans (Hald et al., 2004).

The subtyping of pathogens heterogeneously distributed among sources allows the application of the Bayesian classification model. All human cases with unique pathogen subtypes are assumed to originate from only one source. If subtypes isolated from only one source may

originate from other sources, from which they were not detected due to the sensitivity of surveillance systems, the importance of these unidentified sources in the salmonellosis burden will be underestimated. Most source attribution approaches require parameter estimation, e.g., estimates of serovar and source-related factors, and take into account uncertainty around estimates with credibility intervals (CrI) (Hald et al., 2004).

In Brazil and most developing countries, there is a dearth of information based on probabilistic source attribution reports, precisely where the burden of foodborne disease (FBD) is estimated to be higher (Li et al., 2019; Odeyemi, 2016; Schlundt et al., 2004). In the present study, we identify the most likely source–serovar combination of salmonellosis from 2005 to 2015 in the state of Rio Grande do Sul (RS), Brazil by modeling sporadic cases with the inclusion of the origin of the animal food sources according to its region of production across Brazil. RS is the southernmost state of Brazil and has a territorial extension of 281,730.2 km²; it is divided into 497 municipalities, and has 11.2 million inhabitants (Pessoa, 2017). In the present analysis, isolates originating from poultry, turkeys, eggs, and pigs were considered the main sources of human infection. Complementarily, trends through time, between regions in RS and the locations of preparation/consumption were obtained by modeling data of *Salmonella* outbreaks.

2. MATERIAL AND METHODS

2.1. General description of the data from humans and sources

From 2011 to 2015, sporadic cases captured by Hospital Epidemiological Surveillance Nuclei (NVEH) from Global Salm-Surv (GSS)-linked hospitals covering 8 out of 19 Regional Health Coordination (CRS) from the sub-regions of RS resulted in 247 isolates of *Salmonella* spp. From

2005 to 2015, 324 outbreaks had information at the serovar level and were from all over the state. From these, 153 were matched with serovars found in the studied sources.

There were three available origins of data for the sources: Official monitoring for poultry, turkey, and pigs; narrative review of literature of the prevalence data for pigs from Santa Catarina (SC); and outbreak data for laying hens. For both humans and sources, antigenic characterization and serovar identification were carried out in the National Reference Laboratory for Cholera and Enteric Diseases, Oswaldo Cruz Institute (FIOCRUZ, Rio de Janeiro, Brazil) with the rapid slide agglutination test using the somatic and flagellar antisera produced by the laboratory. The diagram in Figure 1 illustrates the construction of the final dataset for source attribution. This study was approved by the Research Ethics Committee at the Federal University of Rio Grande do Sul under the Certificate of Presentation for Ethical Appreciation number 85788618.1.0000.5347 on 5 July 2018.

2.2. Microbial subtyping

For the microbial subtyping approach, data were obtained from FBD outbreak surveillance from the RS State Health Surveillance Center (CEVS) from 2005 to 2015 and from the medical records of GSS-linked hospitals from 2011 to 2015. GSS was an international resource for training and capacity building in FBD surveillance developed by the World Health Organization (WHO) for improving the accurate identification and comparison of foodborne salmonellosis (WHO, 2005). In Brazil, clinical samples were obtained from a laboratory-based surveillance

network coordinated by the Bacteriology Division from the Central Public Health Laboratory, augmenting the sensitivity of *Salmonella* human surveillance in RS.

Data on the trade and transit of animal products (dos Santos et al., 2012) were obtained from the State Secretariat of Agriculture, Livestock, and Supply (Supplementary Material S1) and the distribution of different serovars in poultry meat and pigs raised in RS were obtained from federal monitoring programs from MAPA (Tables B and C, Supplementary Material S1), and from previous studies in one of the states that markets animal products to RS (da Silva et al., 2012; Kich et al., 2011; Pissetti et al., 2012).

Systematic surveillance of *Salmonella* spp. reported in animal reservoirs (e.g., broilers and turkeys) can capture a wider range of serovars in asymptomatic hosts than in clinical salmonellosis. To ensure flock health and the safety of poultry products in Brazil, MAPA has implemented the Poultry Health National Plan, which includes *Salmonella* control initiatives in the country's poultry industry. From 2004 to 2011, MAPA has implemented regular flock surveillance for detecting the *Salmonella* serovars Enteritidis, Typhimurium, and others in broilers before slaughter (Brasil, 2016).

An exploratory study identified 16 serovars in pigs raised in RS, with only six in common with human cases (Table C, Supplementary Material S1). In SC, a neighbor state of RS and a prolific producer of pork, data on the occurrence of NTS were obtained from previous studies from the Department of Preventive Veterinary Medicine of the Federal University of Rio Grande do Sul (Table D, Supplementary Material S1). For beef cattle, the overall prevalence of *Salmonella* spp. was 1.7% in 2015 and 2016 (personal communication, Karine Bordignon, MAPA). Although beef seems to be a less important source of *Salmonella* in RS (da Silva et al. 2014), it is not

negligible, as it accounts for invasive infections caused by serovars such as Dublin or prevalent monophasic variant of Typhimurium; furthermore, traditionally, it could be consumed undercooked. Its impact on human salmonellosis cases was assessed by analyzing outbreak data, as serotyping was not performed until after the conclusion of this work.

Official monitoring data for laying hens were also not available for inclusion in the model. However, as hen eggs are recognized as important sources of human *Salmonella* infections, the serovar distribution data for reported egg-associated outbreaks were used for imputation in the model. Among all egg-related *Salmonella* outbreaks for which specimens could be subtyped and notified to CEVS from 2005 to 2015, the proportion of each *Salmonella* serovar was estimated and used as input in the model (Figure 2). These represented 77.7% of the reported subtyped *Salmonella* outbreaks.

We applied the Hald model for *Salmonella* using surveillance and monitoring data from RS from 2005 to 2015 and estimated the number of sporadic and domestic salmonellosis cases attributable to different food-producing animals (Hald et al., 2004). A sporadic case was defined as a human case that could not be associated with a recognized FBD outbreak. For outbreak-related cases, one outbreak contributed to the model with one sporadic case (e.g., index cases), whereas the remaining cases associated with an outbreak were added to the final model results after the modeling step, and attributed to the food involved in the outbreak, if known (Hald et al., 2004). Travel-related cases were not included in the model because the history of traveling before the onset of disease was not available for reported cases and they were considered domestic (de Knecht et al., 2015b).

The model assigns cases to animal reservoirs based on the distribution of *Salmonella* subtypes among animal hosts (Hald et al., 2004). The principle consists of comparing the number of human cases caused by different serovars of a pathogen with the distribution of the same serovars in different animal source foods. The model is constructed with collections of time- and space-related isolates from various food and human sources, and the amount of food available to the population is considered (De Knecht et al., 2015a; Hald et al., 2004). The expected number of human cases per source and serovar was estimated by the following equation:

$$\lambda_{ij} = M_j p_{ij} q_i a_j$$

and

$o_i \text{Poisson}(\sum_j \lambda_{ij})$, where λ_{ij} is the expected number of cases/year of serovar i of source j ; M_j is the amount of source available for consumption (in 1000 tons); p_{ij} the prevalence of serovar i in source j ; q_i is the factor related to serovar i ; a_j is source-related factor j ; and o_i is the observed number of human cases of *Salmonella* serovar i . The expected number of sporadic and domestic cases infected by *Salmonella* serovar i was estimated based on:

$$\lambda_i = \sum_j \lambda_{ij}$$

which corresponds to the observed cases affected with serovar i minus the observed cases affected by serovar i and known to be outbreak related. One case was subtracted for each outbreak so that one outbreak contributed one sporadic case (Hald et al., 2004).

As defined by Hald et al. (2004), the type-dependent factor (q_i) summarizes the characteristics of the serotype (survivability, virulence, pathogenicity), which determine its

capacity for causing infection, and the source-dependent factor (a_j) summarizes the characteristics of the source (physical properties, preparation methods, processing procedures), which determine its capacity for acting as a vehicle for *Salmonella* (Hald et al., 2004). Non-informative uniform distributions with a positive lower bound were used for the source- and serovar-dependent parameters (a_j and q_i). To ensure that the upper bounds allowed the inclusion of all possible values for the parameter, posterior distributions were evaluated visually. If distributions appeared to be disrupted, as in cases of arbitrary cut-off at the upper values, uniform distributions were expanded until they reached the required ranges (Hald et al., 2004).

The model was built using Markov chain Monte Carlo (MCMC) iterations with R software (version R 3.3.3 2017, R Development core team) and packages rjags and coda (Supplementary Material S2). Three independent Markov chains with widely dispersed initial values were performed for 120,000 iterations after a 60,000-iteration burn-in period with a thinning of 1. Autocorrelation of the Markov chain was checked as a function of delay, observing the similarity between observations as a function of the time lag between them. In case autocorrelation reached values close to 0, it was assumed to be absent. Convergence was monitored using the method developed by Gelman and Rubin (1992), and was considered to have occurred when the chains reached stability and the variance between different chains was not larger than the variance within each chain individually. String length was determined by running enough iterations to ensure that the Monte Carlo errors for each parameter were <5% of the later standard deviation.

2.3. Analysis of outbreak investigation data

Outbreak surveillance data were organized so that each reported outbreak corresponded to one observation in the final dataset amenable for analysis. Each observation contained information regarding the year of occurrence, the corresponding CRS, the population at risk, the number of sick people and fatalities associated with the outbreak, the location of the outbreak, and the food involved. Both confirmed (laboratory or epidemiologically) and suspected outbreaks were used; for incomplete fields, data were included as unknown (e.g., unknown source). Analytical epidemiological evidence corresponds to statistically significant associations between a food item and human cases in the FBD outbreak from CEVS outbreak investigations. Laboratory evidence implies that the causative agent has been detected by appropriate laboratory methods at the food source or in the production and preparation environment. Non-foodborne outbreaks were excluded from the dataset (e.g., person-to-person). The locations of outbreaks were classified as private/household or public/general depending on where the exposure took place (e.g., restaurants, hospitals) or where the food was produced or marketed (e.g., supermarket).

Here, we classified food items using an adapted commodity-based hierarchical scheme (Painter et al., 2009). The foods were divided into specific food categories of terrestrial animals, plants, and seafood. The criterion used for defining if the food was complex was to have the ingredients comprise >1 food category. Otherwise, the food was considered simple. The list of ingredients for complex foods reported was obtained by reviewing recipes online: the top three recipes from a Google search were chosen; for conflicting recipes, ingredients listed in at least two recipes were selected. Unknown source outbreaks corresponded to most of the data. In

cases without detailed information, animal-source ingredients were grouped under non-specified “meat”.

The use of outbreak data for the attribution of human FBD has been described previously (Adak et al., 2005; Greig and Ravel, 2009; Pires et al., 2010), and the applied method is based on the principles described by Pires et al. (2012). Data on reported outbreaks of *Salmonella* spp. from RS occurring in 2006–2015 were used to attribute outbreaks of human salmonellosis to specific food sources. We categorized implicated food into simple foods (i.e., constituted by ingredients belonging to one single food category) and complex foods (i.e., constituted by ingredients belonging to multiple food categories). In addition to the overall source attribution model, a set of sub-analyses were performed, estimating the importance of sources in: (1) two locations categories (general versus domestic outbreaks), (2) seven regions (Midwest, Central eastern, Capital region, Northeast, Northwest, Southeast, and South-west), and (3) three aggregated periods between 2006 and 2015.

The method used was based on the number of outbreaks caused by each food and was aimed at attributing human salmonellosis to food. The outcome was the proportion of outbreaks attributed to different food sources. To avoid potential overestimation of the proportion of disease attributed to sources that caused large outbreaks, the number of ill people reported in each outbreak was not used (Pires et al., 2010). Incriminated foods were classified based on the number of ingredients, i.e., containing a single food category or multiple, complex foods, designated by the defined criteria. If any food category was not implicated in a simple-food outbreak, the category was not considered a possible source of complex-food outbreaks. This premise may result in underestimation of the importance of some foods. However, our estimates

are data-driven, and this limitation may be overcome by including larger amounts of data or with analyses including an extended study period (Pires et al., 2010).

The proportion of human salmonellosis that can be attributed to each food source was estimated based on the number of simple-food outbreaks caused by each source in the ingredients (food categories) constituting complex foods, and based on the likelihood that each of these categories was the cause of complex-food outbreaks, as described in Pires et al. (2012). Briefly, simple-food outbreak data were summarized, and the proportion of outbreaks caused by each category was used to define the distribution representing the probability that an outbreak i was caused by source j (P_j), calculated as follows:

$$P_j \text{Beta}(\text{source}S_j + 1, \text{total}S - \text{source}S_j + 1)$$

where $\text{source}S_j$ is the total number of simple-food outbreaks caused by source j , and $\text{total}S$ is the total number of simple-food outbreaks in the whole period and all regions. This probability was estimated by source using information from all CRS and over the entire period. To calculate the number of outbreaks attributed to each source, the simple-food outbreaks were assigned to the respective simple-food categories. In turn, complex-food outbreaks were divided according to each food category in the incriminated food in proportion to their likelihood of causing a simple-food outbreak, calculated by P_j . Thus, complex-food outbreaks were attributed only to categories that were incriminated in at least one simple-food outbreak.

The model was built in WinBUGS 1.4 (<http://www.mrc-bsu.cam.ac.uk/bugs/>), which defaults to the Gibbs sampler (MCMC simulation), and estimated P_j (five independent Markov chains with 60,000 iterations). For each chain, different initial values were used for P_j , widely dispersed in the target distribution (Supplementary Material S2).

3. RESULTS

3.1. General description of the data from humans and sources

From 2011 to 2015, most sporadic cases captured from GSS in RS were that of serovars Typhimurium, Enteritidis, and Infantis. Serovar Typhimurium surpassed serovar Enteritidis after 2011, and as did serovar Infantis from 2014 onwards (Reis et al., 2018). Additionally, from 2005 to 2015, 153 *Salmonella* outbreaks had information at serovar level state-wide, with the main serovars implicated being Enteritidis and Typhimurium (Figure 2). While NVEH data were used for the microbial subtyping model, outbreak data were used for both analyses, but with different discrimination power. For the analysis of outbreak data, *Salmonella* was considered at species level, resulting in a bigger sample for subset analyses.

Regarding data from putative *Salmonella* sources, 21 different serovars were obtained from 128 samples from RS poultry and turkey farms, compared to 25 human serovars obtained in NVEH (Reis et al., 2018). Five serovars were found in common in both surveillances. The main serovars found in poultry meat species and pigs are shown in Tables B and C of Supplementary Material S1, respectively.

3.2. Microbial subtyping

Serovars isolated from human cases were selected according to their occurrence in the sources of production animals, which included 15 *i* serovars. Only one subspecies of *Salmonella enterica* and three *S. enterica enterica* serovars were not detected in any of the sources and were excluded from the analysis (*S. enterica arizonae*, and the *S. enterica enterica* serovars Muenchen, Ohio, and Oranienburg). Regarding the sources, we divided broilers, turkeys, and pigs according

to the state origin (i.e., Goiás [GO], Paraná, RS, or SC), which yielded eight j sources. The remaining source accounted was laying hens/eggs, with data from outbreaks.

Considering the underrepresentation of cases (e.g., few data points in the period), we were unable to estimate the actual number of cases but could assign subtyped cases to the main sources and estimate their relative importance. The most important sources of human salmonellosis in RS between 2005 and 2015 estimated by the model were laying hens (eggs) at 39.4% (159 cases, 95% CrI 43–247), followed by 34.5% of cases attributed to pigs reared in SC (139 cases, 95% CrI 60–187). Turkeys reared in other states and broilers reared in RS were estimated as minor sources of *Salmonella*, contributing about 10% of cases each (turkeys raised in GO, PR, and SC: 42 cases, 95% CrI 15–125; chickens raised in RS: 40 cases, 95% CrI 1–139).

The most important *Salmonella* serovars isolated in the study period were Enteritidis, responsible for 43.4% of cases (175 cases, 95% CrI 150–202) and Typhimurium, contributing 39.9% of cases (161 cases, 95% CrI 137–187). Table 1 shows the estimates of the combinations of serovars and animal sources. Of all Enteritidis serovar infections, 84.6% were attributed to laying hens (148 cases, 95% CrI 48–189), while 77% of cases caused by the Typhimurium serovar originated from pigs raised in SC (124 cases, 95% CrI 45–156). Among the emerging serovars, Infantis in pigs (from SC) and Newport in turkeys (from GO, PR, and SC) were notable. The observed number (o_i) and the expected number (λ_i) of *Salmonella* serovar cases are plotted in Figure 3. The fitness of the model could be explained by the absence of serovars distributed in unknown sources.

Eggs had the highest value for source-dependent factors (a_j), followed by turkey meat, while pork had the lowest value (Table 2). However, eggs had a wide CrI range. This parameter could indicate the likelihood of infection by consuming a particular unit (for example, 1 kg) from one source compared to another source. Table 2 presents the estimated values for the serovar-dependent factors (q_i). The results suggest that the ability of the Infantis serovar to survive food processing and/or cause disease is about 40% greater than that of the Enteritidis serovar ($q = 57.7$, 95% CrI: 17.7–97.2), and about 50% greater than that of the Typhimurium serovar.

3.3. Analysis of outbreak data

Outbreak investigation data were analyzed to estimate the proportion of salmonellosis (species-level) outbreaks attributable to specific sources in the population of the different RS regions. Sub-analyses estimated the importance of sources by location (comparing general and domestic outbreaks), region (seven regions of RS), and over time (from 2006 to 2015). Due to differences in data consistency, the temporal sub-analysis used 287 outbreaks, while the other two sub-analyses used 817 outbreaks.

Figure 4 shows the proportion of foodborne outbreaks of salmonellosis from epidemiological research data, which are stratified by 3–4-year periods. We estimated that eggs were the most important source, but they showed a decline of about 13% in the period studied and a difference of about 10% with pork in the final 4 years (>36% in the first 3 years). Other food sources showed smaller variations, with a 5% increase in the proportion of chicken meat and a 5% decrease in that of vegetables.

Differences between outbreak locations were not significant (not shown). Outside the home, the proportions were higher for eggs and meat in general; in the household, the proportions for beef and pork were higher. Minor differences were found in other foods reported in outbreak investigations. In the sub-analysis between regions, a pattern was observed in the proportions of foods causing salmonellosis outbreaks, with unknown causes, eggs, and vegetables predominating, and with small differences in importance (ranking) in the proportion of the four meat source categories (Figure 5).

The probability P_j that an outbreak was caused by source j was higher for chicken and pork, followed by cattle, eggs, meat, and vegetables (Figure 6). This means that, for example, in an outbreak caused by a complex food containing pork and vegetables, the probability that pork was the cause would be 30.1% (95% CrI 19.6–42.0), and that of vegetables would be 6.4% (95% CrI 1.2–1.5).

4. DISCUSSION

Data on human NTS are scarce in South America because of the lack of an effective tracking system and efficient data recording in the countries, although the *Salmonella* underreporting rate may be lower compared to other foodborne pathogens (Wheeler et al., 1999). Most foodborne gastroenteritis cases do not require hospitalization; from those that do require hospitalization, the causative agent cannot be isolated from implicated food or clinical samples in most cases, and neither are reported to the authorities. Regional representativeness is heterogeneous, and more populated urban centers contribute to most of the reports (as seen in Figueiredo et al., 2013). Previous studies have reported on *Salmonella* foodborne outbreaks in

RS. Although different serovars were isolated from several foods, most salmonellosis outbreaks were caused by *S. Enteritidis*, especially by strain SE86 (de Oliveira et al., 2006; Oliveira et al., 2007). On the other hand, there is a possible emergence of host-adapted, versatile *S. Typhimurium* as one of the most prevalent foodborne pathogens worldwide (Hendriksen et al., 2011).

Both approaches were consistent in estimating eggs as one of the most important sources of human salmonellosis in RS. Attribution estimates using outbreak data showed similar patterns of the most important sources between the regions in RS, with emphasis on unknown sources, vegetables, eggs, and broiler meat. The impact of beef was lower compared to other sources, though its P_j value was the third higher. The absence of estimates for this source in the subtyping model can result in the misplacing of some cases when their true source is not included, particularly in the estimation of those caused by ubiquitous serovars such as *Typhimurium* (de Knecht et al., 2014a). The differences between regions may reflect data availability and quality (e.g., readiness to investigate outbreaks; due to technical constraints or geographical distance) and even different risk perceptions among outbreak investigators (i.e., reporting and investigation bias). Regional differences may be more evident when the source attribution study takes place in larger geographical areas (e.g., across countries) (Pires et al., 2012).

Here, the trends observed in the outbreak model depict the importance of potato salad with homemade mayonnaise in *Salmonella* cases, as described in previous studies (Costalunga and Tondo, 2002; Wagner et al., 2013). The main factor contributing to the occurrence of outbreaks was the consumption of products without sanitary inspection (i.e., clandestine, informal production), mainly eggs, followed by food maintenance at room temperature for >2

hours (Costalunga and Tondo, 2002; Wagner et al., 2013). A cultural habit of the RS population is to prepare homemade mayonnaise using raw eggs mixed with oil and warm cooked potato. Often, the eggs are from small farms without sanitary inspection, increasing the probability of *Salmonella* contamination. During the preparation, *Salmonella* can multiply in the eggs due to the warm temperature and maintenance at room temperature. We attribute a significant proportion of salmonellosis to vegetable consumption, which had lower P_j weight, as a cause of simple-food outbreaks. The results suggest that between 17% and 23% of salmonellosis cases are attributed to this source, as seen in estimates from a worldwide data analysis of outbreak investigations (Greig and Ravel, 2009). Vegetables accompanied the decreased importance of eggs but could have been overestimated, possibly because potatoes are an ingredient in homemade mayonnaise.

Despite the fewer data points in the analysis of trends through time, the relative contribution of eggs showed a declining pattern, although they were the most important source in all periods, contrasting with the most significant increase of pork as a source of human salmonellosis. The compulsory use of pasteurized eggs and municipality border surveillance could be starting to control the incidence of egg-borne salmonellosis (ANVISA, 2009). This result raises the hypothesis of the diminished importance of classical sources of *Salmonella* (e.g., eggs) in FBD outbreaks. The decreasing importance of *Gallus gallus* has been observed in European countries since the implementation of new control measures in chicken farms (e.g., the introduction of live vaccines), improved hygiene, and education of consumers and food handlers (Hendriksen et al., 2011), especially after the implementation of national *Salmonella* control programs in chickens and laying hens according to European Union Regulation No. 2016/2003. In the United States, an

examination of trends in foodborne outbreaks by *S. Enteritidis* from 1979 to 2009 suggested that interventions initiated in the 1990s for curbing *S. Enteritidis* growth in eggs were essential for preventing further outbreaks (Wright et al., 2016).

Recently, pork consumption in Brazil has increased (ABPA, 2019). The analysis of outbreak data attributed an increasing proportion of cases to pork from the beginning of 2000 onwards, suggesting a change in NTS epidemiology. In RS, most pork production is related to sausage production rather than pork, which comes mainly from SC (personal communication, Carina dos Santos, Secretariat of Agriculture, Livestock and Rural Development of RS). Even with a similar prevalence of *Salmonella* in pigs from RS and SC (Tables C and D, Supplementary Material S1), the type of product and the higher amount of pork consumed from SC (Table A, Supplementary Material S1) may explain the observed results, raising the hypothesis of a potential regional chain from SC. Conversely, the incorporation of parameter M_j did not affect the attribution estimates of previous applications of the modified Hald model (Mullner et al., 2009; Mughini-Gras et al., 2014a).

There is growing evidence that pigs play a major role as sources of human salmonellosis and that the most likely route of transmission is poor handling and consumption of contaminated pigs (Mughini-Gras et al., 2014b). Just as poultry are the probable reservoirs of *S. Enteritidis*, pigs are the most likely reservoir of *S. Typhimurium* (De Knecht et al., 2015), in addition to its single-phase variant 4,[5],12:i:- and *S. Derby* (Hoelzer et al., 2011). *S. Typhimurium* seems to be more related to cases requiring hospitalization (Reis et al., 2018) and should be monitored, including its single-phase variant and antimicrobial resistance levels, as well as other emerging *Salmonella* serovars such as *Infantis*, *Heidelberg*, and *Newport* (Penha Filho et al., 2019; Reis et al., 2018).

It is assumed that the estimated values of the source and pathogen-related factors consider both the consistency of the surveillance systems and the intrinsic ability of serovars to cause disease in humans. However, it should be interpreted relatively, and not as an absolute measure. Due to the relatively few data points available for the period of analysis, the CrI were widespread, making the ranking less interpretable. The inclusion of data from different surveillance systems (e.g., CEVS and NVEH) and periods could reflect changes in the capacity for detecting different kinds of pathogenicity (e.g., generally mild, widespread foodborne outbreaks or hospitalization-related, sporadic cases), consumption/exposure habits, and the (effectiveness of) implementation of animal health programs, on *Salmonella* cases. It is possible that the fact that pork is typically consumed well-cooked influences the lower values for its source-dependent parameter. Otherwise, the higher value for eggs and turkeys agrees with a previous study covering European countries (de Knecht et al., 2015a).

Limitations

We compared the results from the combination of a microbiological and an epidemiological source attribution method to overcome their inherent limitations. The estimates account for differences in periods and populations at risk, the point of attribution, the sources investigated, and the serovar/species discrimination power. The applicability of periodic surveillance data must include the consideration that production prevalence estimates are imperfect substitutes for actual exposure and that unrepresented sources contribute to the impact of disease on the human population (Ranta et al., 2011). As time series collections increase, time-prevalence models improve and provide estimates even for periods with missing data. When insufficient surveillance or monitoring data of the herd/flock are available for a

source, slaughter samples should be surveyed and their quality as substitutes assessed (de Knecht et al., 2015b). For most sources, the data were from official monitoring programs, which do not consider informal, uninspected food production.

Most salmonellosis outbreaks originate from eggs, and consumption of animal-source food without inspection is a risk factor of foodborne outbreaks in RS (Wagner et al., 2013). In the absence of official data for laying hens, we assumed that the proportion of egg-related *Salmonella* outbreaks could partially represent exposure to this source. If the hypothesis of lower *Salmonella* prevalence in commercial, inspected laying hens holds, then the input of egg-associated *Salmonella* outbreaks could lead to overestimation of salmonellosis cases attributed to laying hens. This compromises interpretations to some degree, and they could be improved by including the data origins of more sources (e.g., with inspection).

The lower discriminatory subtyping data could result in less accurate estimates when attributing salmonellosis cases (Pires and Hald, 2010), particularly when it comes to ubiquitous types such as *S. Typhimurium* and *S. Enteritidis* (De Knecht et al., 2015a). Highly discriminatory methods are a suitable solution for (single) source attribution for a cluster of human cases, as would be the case in timely outbreak investigations. To relate groups of bacterial strains with particular reservoirs/sources and then attribute sporadic human cases to these sources, models based on both serovars and phage types allow a process to identify sufficient diversity between strains from humans and causative sources to a degree where epidemiological relatedness can be assumed (de Knecht et al., 2016).

Furthermore, there is a need to consider additional sources and transmission routes when choosing a source attribution approach, depending on the public health issues to be addressed, such as travel-related cases and direct contact contamination. Improved investigations of sporadic and outbreak-related cases, e.g., regarding travel history, which may have significant relative importance, as is the case in countries with national salmonellosis control (Hald et al., 2004; de Jong and Ekdahl 2006), and comparative exposure assessment could add value to estimates. Conversely, a systematic review of case-control studies may underestimate the importance of travel and assign cases to other exposures included in the questionnaires, such as direct contact with domestic animals and environmental sources (Pires, 2009). The inclusion of additional relevant issues could provide a comprehensive assessment of multidimensional food-related hazards, such as interactions between microbial ecology, antibiotics use, and the effectiveness of other disease control programs.

Conclusions

The combination of source attribution approaches has proven to be a valuable tool for quantifying the importance of different food sources in human salmonellosis in RS, even with the use of sparse data. Although the data were limited, which resulted in uncertainties, we believe that our study is a crucial first step towards investigating the relative contribution of different sources of salmonellosis in the state. It is also noteworthy to highlight data gaps and research needs, which will pave the way for improving source attribution efforts. Analysis of improved integrated surveillance data could yield more accurate results, cover more sources and larger areas, and thus better assist risk mitigation decisions, guide priority interventions from source to

485 consumption, and measure the effects of current control programs on the burden of *Salmonella*
486 on humans.

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488

489 **Conflict of interest statement**

490 There authors report no conflicts of interest.

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Tables

Table 1. Estimates of expected numbers of cases for major combinations of serovars and sources of livestock from different states causing human salmonellosis in Rio Grande do Sul from 2005 to 2015.

Serovar/Source (Origin State ^a)	Median	95% CrI ^b
Enteritidis/Eggs (RS)	148	(48–189)
Typhimurium/Pigs (SC)	124	(45–156)
Enteritidis/Broilers (RS)	17	(1–100)
Infantis/Pigs (SC)	16	(3–32)
Typhimurium/Turkeys (GO/PR/SC)	12	(3–85)
Newport/Turkeys (GO/PR/SC)	11	(5–18)
Typhimurium/Pigs (RS)	10	(3–12)
Typhimurium/Eggs (RS)	8	(2–40)

^a GO: Goiás; PR: Paraná; RS: Rio Grande do Sul; SC: Santa Catarina

^b Credibility interval

Table 2. Estimated values for source- and serovar-dependent factors: α_j^a and q_i , which summarize, respectively, the capacity of the source to act as a vehicle for *Salmonella*, and the likelihood of a *Salmonella* serovar to cause infection. The ranking is based on the point estimate.

	Mean	Rank	Std ^b	Median	95% CrI ^c	
a[eggs]	7.8	1	5.1	6.4	2.7	21.5
a[turkeys]	0.2	2	0.2	0.2	0.1	0.8
a[chickens]	0.0015	3	0.0024	0.0006	0.00001	0.008
a[pigs]	0.0002	4	0.0001	0.0002	0.00004	0.0005
q[Infantis]	81.3	1	15.0	84.9	44.6	99.4
q[Give]	62.6	2	24.6	65.6	12.9	98.4
q[Newport]	61.4	3	25.2	64.2	13.9	98.4
q[Enteritidis]	57.7	4	22.5	57.1	17.7	97.2
q[Typhimurium]	54.0	5	21.2	50.8	19.8	96.3
q[Corvallis]	49.5	6	26.1	47.9	6.7	96.6
q[Johannesburg]	49.4	7	26.1	47.8	6.7	96.6
q[Braenderup]	48.8	8	24.9	46.5	8.5	95.9
q[London]	21.1	9	19.1	14.9	1.7	76.5
q[Schwarzengrund]	17.5	10	17.4	11.5	0.9	67.8
q[Heidelberg]	17.4	11	18.0	11.0	0.8	69.8
q[Saintpaul]	16.2	12	13.4	12.5	1.8	52.4
q[Panama]	10.7	13	6.0	9.5	2.6	25.6
q[Agona]	3.4	14	2.7	2.7	0.4	10.5
q[Derby]	2.8	15	3.9	1.7	0.2	11.9

^a Values of $\{\alpha_j\}$ are in 10^{-5}

^b Standard deviation

^c Credibility interval

Figure legends

Figure 1. Diagram illustrating the build of the final dataset for source attribution. Dark green, blue, pink, and orange blocks represent datasets. The light green block represents surrogate data from the outbreak dataset to complement the reservoir level, even at the exposure level. The white block represents data not included in the final dataset due to a lack of subtyping.

Figure 2. The distribution of the major serovars implicated in human salmonellosis outbreak-related cases from 2005 to 2015.

Figure 3. Graph of observed and expected values of the number of cases reported according to *Salmonella* serovar.

Figure 4. Proportion of salmonellosis outbreaks attributed to foods in Rio Grande do Sul, in the triennia 2006–2008 and 2009–2011, and quadrennium 2012–2015.

Figure 5. Proportion of salmonellosis outbreaks attributed to foods in Rio Grande do Sul regions.

Figure 6. Prior prevalence (P_j) of *Salmonella* in simple foods (mean and 95% credibility interval) in Rio Grande do Sul.

738 **Supplementary Material S1. Tables (A-D)**

739 **Table A.** Values of the quantities of sources available for consumption in RS during the study period (dos Santos et
740 al., 2012).

Source/ State ^a	Amount (heads/ dozens)	Mean weight (kg)	Yield	Total (thousand t)
Pork/RS	5,729,720	100	0.72	412.5
Broilers/RS	127,724,419	5	0.83	530.0
Turkeys/RS	57,421	7	0.75	0.3
Eggs/RS	300,738	0.684	0.9	0.1
Pork/SC	29,372,467	100	0.72	2114.8
Broilers/SC	22,848,963	5	0.83	94.8
Broilers/PR	19,962,358	5	0.83	82.8
Broilers/GO	818,687	5	0.83	3.3
Turkeys/SC; PR; GO	297,881	7	0.75	1.5

741 ^a GO: Goiás; PR: Paraná; RS: Rio Grande do Sul; SC: Santa Catarina

742

Table B. Relative proportion of *Salmonella* spp. found in poultry and turkey farms in the states of Goiás, Paraná, Santa Catarina, and Rio Grande do Sul

Serovar	Broiler	Turkey
Enteritidis	30.8%	0.6%
Schwarzengrund/Bredeney	8.1%	0.6%
Mbandaka	6.0%	2.5%
Heidelberg	4.5%	0.6%
Infantis	4.1%	2.5%
Agona	3.6%	35.7%
Muenchen	3.6%	0.0%
Saintpaul	3.3%	5.1%
Typhimurium	2.9%	2.5%
Oranienburg	2.6%	5.7%
Newport	2.4%	5.1%
Senftenberg	2.1%	9.6%
Typhimurium/4,[5],12:i:-	2.1%	4.5%
Minnesota	2.1%	0.6%
Lille	1.9%	0.6%
Brackenridge	1.7%	0.0%
Poona	1.7%	0.0%
Worthington	1.4%	1.3%
Derby	1.4%	0.0%
Anatum	1.2%	1.9%
Javiana	1.0%	1.9%
Hadar	0.0%	12.1%

747 **Table C.** Exploratory study by MAPA on the relative proportion of *Salmonella* spp. found in pig farms in Rio Grande
 748 do Sul.

Serovar	Proportion
Anatum	2.7%
Derby	10.3%
Give	0.7%
Infantis	1.4%
Lexington	0.7%
Ohio	1.4%
Oslo	2.1%
Panama	4.8%
Paratyphi B	0.7%
Rissen	0.7%
Typhimurium	22.6%
4,[5],12:i:-	14.4%
Enteritidis	0.7%
Manhattan	0.7%
Rubislaw	1.4%
Javiana	0.7%
Not identified	34.2%

749

750

Table D. Summary of studies used to estimate the proportion of *Salmonella* serovars in pigs reared in Santa Catarina.

Serovars	Sample (carcasses)	Proportion	Reference
Derby	109	11.9%	da Silva et al. (2012)
Derby	98	2.0%	Kich et al. (2011)
Derby	252	7.1%	Pissetti et al. (2012)
Infantis	252	2.4%	Pissetti et al. (2012)
London	252	0.8%	Pissetti et al. (2012)
Mbandaka	252	0.4%	Pissetti et al. (2012)
O:4,5 (B)	98	6.1%	Kich et al. (2011)
O:4,5 (B)	252	0.4%	Pissetti et al. (2012)
O:4,5:-:1.2 (Stanley)	98	1.0%	Kich et al. (2011)
O:6,7:r:- (Infantis)	252	0.4%	Pissetti et al. (2012)
Ohio	252	0.4%	Pissetti et al. (2012)
Panama	109	1.8%	da Silva et al. (2012)
Panama	98	4.1%	Kich et al. (2011)
Panama	252	0.4%	Pissetti et al. (2012)
Typhimurium	109	16.5%	da Silva et al. (2012)
Typhimurium	98	11.2%	Kich et al. (2011)
Typhimurium	252	17.5%	Pissetti et al. (2012)

755 **Supplementary Material S2.** Codes for the microbial subtyping model (R) and the outbreak data model (WinBUGS)

756

757 Code A. *Salmonella* microbial subtyping model

758

759 setwd("~/R")

760 bd.obs= read.csv2('casos_Ser15_spo.csv')

761 bd.prev= read.csv2('prev_15_9.csv')

762 bd.qtdes= read.csv2('quantidades9kg.csv')

763 library(rjags)

764 Loading required packages: coda

765 Linked to JAGS 4.2.0

766 Loaded modules: basemod,bugs

767 writeLines(
 768 + "model{
 769 + for(i in 1:15){
 770 + for(j in 1:nsource){
 771 + lambdaij[i,j] <- prevs[i, j]*qtdes[j]*a[j]*q[i]
 772 + }
 773 + casos[i] ~ dpois(lambdai[i])
 774 + lambdai[i] <- sum(lambdaij[i, 1:nsource])
 775 + }
 776 +
 777 + for(j in 1:nsource){
 778 + lambdaj[j]<-sum(lambdaij[1:15, j])
 779 + }
 780 + a[1]~dunif(0,100)
 781 + a[2]~dunif(0,100)
 782 + a[3]~dunif(0,100)
 783 + a[4]~dunif(0,100)
 784 + a[5]<-a[1]
 785 + a[6]<-a[2]
 786 + a[7]<-a[2]
 787 + a[8]<-a[2]
 788 + a[9]<-a[3]
 789 +
 790 + q[1]~dunif(0,100)
 791 + q[2]~dunif(0,100)
 792 + q[3]~dunif(0,100)
 793 + q[4]~dunif(0,100)
 794 + q[5]~dunif(0,100)
 795 + q[6]~dunif(0,100)
 796 + q[7]~dunif(0,100)
 797 + q[8]~dunif(0,100)
 798 + q[9]~dunif(0,100)
 799 + q[10]~dunif(0,100)
 800 + q[11]~dunif(0,100)
 801 + q[12]~dunif(0,100)
 802 + q[13]~dunif(0,100)

```

803 + q[14]~dunif(0,100)
804 + q[15]~dunif(0,100)
805 +
806 + }
807 + ", "modelo_a_related9_q_uniform15")
808 # Defining data as a list, as in BUGS
809 nsero = 15 #n serovar (i index)
810 nsource = 9 #n animal source (j index)
811 dadosjags = list(nsero=15,
812 + nsource = 9,
813 + casos = array(bd.obs$ncasos),
814 + prevs = array(bd.prev$prev, c(nsero, nsource)),
815 + qtde = array(bd.qtde$qtde)
816 + )
817 # Defining data as a list, as in BUGS
818 > inits = list(list(a=c(1,33,66,99,NA,NA,NA,NA,NA),q=c(1,8,15,22,29,19,89,36,43,50,57,64,71,78,85)),
819 + list(a=c(33,66,99,1,NA,NA,NA,NA,NA),q=c(8,15,22,29,36,89,19,43,50,57,64,71,78,85,1)),
820 + list(a=c(66,99,1,33,NA,NA,NA,NA,NA),q=c(15,22,29,36,43,6,88,50,57,64,71,78,85,1,8)))
821 # Defining burn-in, number of simulations, thinning and parameters for the required results
822 burn = 60000
823 nsim = 120000
824 nthin = 1
825 parms = c("a","q","lambdai", "lambdaj", "lambdaij")
826 # Initializing the model with a chain if 1000 iterations for the adaptation
827 > m <- jags.model("modelo_a_related9_q_uniform15.txt", dadosjags, inits, n.chains=3,n.adapt=1000)
828 Compiling model graph
829 Resolving undeclared variables
830 Allocating nodes
831 Graph information:
832 Observed stochastic nodes: 15
833 Unobserved stochastic nodes: 19
834 Total graph size: 400
835 Initializing model
836 |+++++++++++++++++++++++++++++++++++++| 100%
837 # Updating the burn-in
838 update(m,burn)
839 |*****| 100%
840 # Getting the final sample
841 mcmc <- coda.samples(m, parms, n.iter=nsim,thin=nthin)
842 |*****| 100%
843
844

```

```

845 Code B. Salmonella source attribution model using outbreak data
846
847 # model for Salmonella enterica and 12 sources (including water): periods' sub-analysis
848 # data set: first all simple foods (o in s1: S)
849 # complex foods (o in c1:C)
850
851
852 # variables:
853 # out[i] = outbreak per pathogen
854 # s = outbreak observation in simple foods
855 # i = outbreak observation in complex foods
856 # j = source
857 # PriorS = prior knowledge we gain from simple food outbreaks for each source
858 # F = presence or absence of source j in complex food f, matrix
859 #sources:
860 #1Eggs2Dairy3GoatMilk4Meat5Poultry6Chicken7Ducks8Turkey9Beef10Pork11Lamb12Mutton13Game14F
861 ruitsNuts15Vegetables16GrainsBeans17OilsSugar18Seafood19Water
862
863 #1Beef2Pork3Poultry4Meat5Dairy6Eggs7Seafood8FruitsNuts9GrainsBeans10Vegetables11OilSugar12
864 Water
865
866 model
867 {
868
869 # Proportion of unknowns
870
871
872 propukAA <- unknownAA * 100/ totaloutbreaksAA
873 propukBB <- unknownBB * 100/ totaloutbreaksBB
874 propukCC <- unknownCC * 100/ totaloutbreaksCC
875
876
877
878 # Variable to estimate attribution of complex foods, separated loop (out of order)

```



```

879
880
881  #1st triennium
882  for (i in AAc1:AAc2 ) {
883    b[i] <- sum(a[1:12,i])
884
885  }
886
887  #2nd triennium
888  for (i in BBc1:BBc2 ) {
889    b[i] <- sum(a[1:12,i])
890
891  }
892
893  #3rd triennium
894  for (i in CCc1:CCc2){
895    b[i] <- sum(a[1:12,i])
896
897  }
898
899  for (j in 1:12) {
900
901    # PRIOR
902
903
904    sourceS[j] <- sum(S[j,1:totalS])
905
906
907    z[j] <- sourceS[j] +1
908    y[j] <- totalS - sourceS[j] +1
909    PriorS[j] ~ dbeta(z[j], y[j])
910

```

```

911
912
913  # BY YEAR
914
915
916  #1st triennium
917
918  # SIMPLE FOODS
919  sourceSAA[j] <- sum(F[j, AAs1:AA2])
920
921  #COMPLEX FOODS
922  for (i in AAc1:AAc2) {
923
924    a[j,i] <- PriorS[j]*F[j,i]
925    c[j,i] <- a[j,i]/b[i]
926    d[j,i] <- c[j,i]*PriorSU[j]
927
928  }
929
930  #2nd triennium
931
932  # SIMPLE FOODS
933  sourceSBB[j] <- sum(F[j, BBs1:BBs2])
934
935  #COMPLEX FOODS
936  for (i in BBc1:BBc2) {
937    a[j,i] <- PriorS[j]*F[j,i]
938    c[j,i] <- a[j,i]/b[i]
939    d[j,i] <- c[j,i]*PriorSU[j]
940
941  }
942

```

```

943  #3rd triennium
944
945  # SIMPLE FOODS
946  sourceSCC[j] <- sum(F[j, CCs1:CCs2])
947
948  #COMPLEX FOODS
949  for (i in CCc1:CCc2) {
950    a[j,i] <- PriorS[j]*F[j,i]
951    c[j,i] <- a[j,i]/b[i]
952    d[j,i] <- c[j,i]*PriorSU[j]
953
954  }
955
956
957  sourceCAA[j] <- sum(c[j,AAc1:AAc2])
958  sourceCBB[j] <- sum(c[j,BBc1:BBc2])
959  sourceCCC[j] <- sum(c[j,CCc1:CCc2])
960
961
962  TotalAA[j] <- sourceSAA[j] + sourceCAA[j]
963  TotalBB[j] <- sourceSBB[j] + sourceCBB[j]
964  TotalCC[j] <- sourceSCC[j] + sourceCCC[j]
965
966
967  AttribAA[j] <- (TotalAA[j] * 100 / totaloutbreaksAA)
968  AttribBB[j] <- (TotalBB[j] * 100 / totaloutbreaksBB)
969  AttribCC[j] <- (TotalCC[j] * 100 / totaloutbreaksCC)
970
971      }
972  PriorSU[1]<-1
973  PriorSU[2]<-1
974  PriorSU[3]<-1

```

```

975   PriorSU[4]<-1
976   PriorSU[5]<-1
977   PriorSU[6]<-1
978   PriorSU[7]<-0
979   PriorSU[8]<-0
980   PriorSU[9]<-1
981   PriorSU[10]<-1
982   PriorSU[11]<-0
983   PriorSU[12]<-1
984           }
985
986
987   # DATA
988
989
990   list(
991
992     AAs1  = 1 ,
993     AAs2  = 12 ,
994     AAc1  = 13 ,
995     AAc2  = 151 ,
996
997
998     BBs1  = 184 ,
999     BBs2  = 189 ,
1000    BBc1  = 190 ,
1001    BBc2  = 244 ,
1002
1003
1004    CCs1  = 259 ,
1005    CCs2  = 263 ,
1006    CCc1  = 264 ,

```

1007	CCc2 = 282 ,
1008	
1009	
1010	
1011	unknownAA= 32 ,
1012	unknownBB= 14 ,
1013	unknownCC = 5 ,
1014	
1015	totaloutbreaksAA = 183 ,
1016	totaloutbreaksBB = 75 ,
1017	totaloutbreaksCC = 29 ,
1018	
1019	totalS = 61 ,
1020	
1021	S=structure(.Data=
1022	c(0,0,0,1,0,0,0,0,0,0,1,0,0,0,0,0,0,0,0,0,0,1,0,0,0,0,1,1,0,0,1,0,
1023	0,0,0,0,0,0,
1024	0,0,0,0,0,1,1,0,1,0,0,0,0,0,0,1,1,1,0,0,0,0,0,0,0,0,0,0,0,0,1,0,0,0,0,0,0,0,1,0,1,1,0,1,0,1,1,1,0,
1025	0,1,1,1,0,
1026	0,1,1,0,1,0,0,1,0,1,0,0,1,0,0,0,0,1,0,1,0,0,1,0,1,0,1,0,0,0,0,0,1,1,0,0,1,0,1,1,0,1,0,0,0,0,
1027	1,0,0,0,1,
1028	0,0,0,0,0,0,0,0,0,0,0,0,0,0,1,0,0,0,0,1,0,0,0,0,0,0,0,0,0,1,0,
1029	0,0,0,0,0,
1030	0,
1031	0,0,0,0,0,
1032	0,0,0,0,0,0,0,0,0,0,0,0,0,0,1,0,0,0,0,0,0,0,0,0,0,1,0,0,0,0,0,0,0,0,0,0,1,0,0,0,0,0,0,0,0,0,0,0,0,0,1,
1033	0,0,0,0,0,
1034	0,
1035	0,0,0,0,0,
1036	0,
1037	0,0,0,0,0,
1038	1,0,
1039	0,0,0,0,0,

[illegible]

[illegible]


```

1153 # model for SALMONELLA and 12 sources (including water): regions' sub-analysis
1154 # data set: first all simple foods (o in s1: S)
1155 # complex foods (o in c1:C)
1156
1157
1158 # variables:
1159 # out[i] = outbreak per pathogen
1160 # s = outbreak observation in simple foods
1161 # i = outbreak observation in complex foods
1162 # j = source
1163 # PriorS = prior knowledge we gain from simple food outbreaks for each source
1164 # F = presence or absence of source j in complex food f, matrix
1165 #sources:
1166 #1Eggs2Dairy3GoatMilk4Meat5Poultry6Chicken7Ducks8Turkey9Beef10Pork11Lamb12Mutton13Game14F
1167 ruitsNuts15Vegetables16GrainsBeans17OilsSugar18Seafood19Water
1168
1169 #1Beef2Pork3Poultry4Meat5Dairy6Eggs7Seafood8FruitsNuts9GrainsBeans10Vegetables11OilSugar12
1170 Water
1171
1172
1173 model
1174 {
1175
1176 # Proportion of unknowns
1177
1178
1179
1180
1181 propukCOC <- unknownCOC * 100/ totaloutbreaksCOC
1182 propukCOR <- unknownCOR * 100/ totaloutbreaksCOR
1183 propukMPA <- unknownMPA * 100/ totaloutbreaksMPA
1184 propukNE <- unknownNE * 100/ totaloutbreaksNE
1185 propukNO <- unknownNO * 100/ totaloutbreaksNO

```

```

1186   propukSE <- unknownSE * 100/ totaloutbreaksSE
1187   propukSO <- unknownSO * 100/ totaloutbreaksSO
1188
1189
1190   # Variable to estimate attribution of complex foods, separated loop (out of order)
1191
1192   #COC
1193   for (i in COCc1:COCC2 ) {
1194     b[i] <- sum(a[1:12,i])
1195
1196   }
1197
1198   #COR
1199   for (i in CORc1:CORc2) {
1200     b[i] <- sum(a[1:12,i])
1201
1202   }
1203
1204   #MPA
1205   for (i in MPAc1:MPAc2) {
1206     b[i] <- sum(a[1:12,i])
1207
1208   }
1209
1210   #NE
1211   for (i in NEc1:NEc2 ) {
1212     b[i] <- sum(a[1:12,i])
1213
1214   }
1215
1216   #NO
1217   for (i in NOc1:NOc2 ) {

```

```

1218   b[i] <- sum(a[1:12,i])
1219
1220   }
1221
1222   #SE
1223   for (i in SEc1:SEc2) {
1224     b[i] <- sum(a[1:12,i])
1225
1226   }
1227
1228   #SO
1229   for (i in SOc1:SOc2 ) {
1230     b[i] <- sum(a[1:12,i])
1231
1232   }
1233
1234
1235
1236
1237   for (j in 1:12) {
1238
1239     # PRIOR
1240
1241
1242     sourceS[j] <- sum(S[j,1:totalS])
1243
1244
1245     z[j] <- sourceS[j] +1
1246     y[j] <- totalS - sourceS[j] +1
1247     PriorS[j] ~dbeta(z[j], y[j])
1248
1249

```

```

1250  # BY REGION
1251  #COC
1252  # SIMPLE FOODS
1253    sourceSCOC[j] <- sum(F[j,COCs1:COCs2])
1254  # COMPLEX FOODS
1255    for (i in COCc1:COCc2) {
1256      a[j,i] <- PriorS[j] * F[j,i]
1257      c[j,i] <- a[j,i] / b[i] }
1258  #COR
1259  # SIMPLE FOODS
1260    sourceSCOR[j] <- sum(F[j,CORs1:CORs2])
1261  # COMPLEX FOODS
1262    for (i in CORc1:CORc2) {
1263      a[j,i] <- PriorS[j] * F[j,i]
1264      c[j,i] <- a[j,i] / b[i] }
1265  #MPA
1266  # SIMPLE FOODS
1267    sourceSMPA[j] <- sum(F[j,MPAs1:MPAs2])
1268  # COMPLEX FOODS
1269    for (i in MPAc1:MPAc2) {
1270      a[j,i] <- PriorS[j] * F[j,i]
1271      c[j,i] <- a[j,i] / b[i] }
1272  #NE
1273  # SIMPLE FOODS
1274    sourceSNE[j] <- sum(F[j,NEs1:NEs2])
1275  # COMPLEX FOODS
1276    for (i in NEc1:NEc2 ) {
1277      a[j,i] <- PriorS[j] * F[j,i]
1278      c[j,i] <- a[j,i] / b[i] }
1279  #NO
1280  # SIMPLE FOODS
1281    sourceSNO[j] <- sum(F[j,NOs1:NOs2])

```

```

1282  # COMPLEX FOODS
1283  for (i in NOc1:NOc2 ) {
1284    a[j,i] <- PriorS[j] * F[j,i]
1285    c[j,i] <- a[j,i] / b[i] }
1286  #SE
1287  # SIMPLE FOODS
1288    sourceSSE[j] <- sum(F[j,SEs1:SEs2])
1289  # COMPLEX FOODS
1290  for (i in SEc1:SEc2 ) {
1291    a[j,i] <- PriorS[j] * F[j,i]
1292    c[j,i] <- a[j,i] / b[i] }
1293  #SO
1294  # SIMPLE FOODS
1295    sourceSSO[j] <- sum(F[j,SOs1:SOs2])
1296  # COMPLEX FOODS
1297  for (i in SOc1:SOc2 ) {
1298    a[j,i] <- PriorS[j] * F[j,i]
1299    c[j,i] <- a[j,i] / b[i] }
1300
1301  sourceCCOC[j] <- sum(c[j,COCc1:COCc2 ])
1302  sourceCCOR[j] <- sum(c[j,CORc1:CORc2])
1303  sourceCMPA[j] <- sum(c[j,MPAc1:MPAc2 ])
1304  sourceCNE[j] <- sum(c[j,NEc1:NEc2])
1305  sourceCNO[j] <- sum(c[j,NOc1:NOc2])
1306  sourceCSE[j] <- sum(c[j,SEc1:SEc2])
1307  sourceCSO[j] <- sum(c[j,SOc1:SOc2])
1308
1309  TotalCOC[j] <- sourceSCOC[j] + sourceCCOC[j]
1310  TotalCOR[j] <- sourceSCOR[j] + sourceCCOR[j]
1311  TotalMPA[j] <- sourceSMPA[j] + sourceCMPA[j]
1312  TotalNE[j] <- sourceSNE[j] + sourceCNE[j]
1313  TotalNO[j] <- sourceSNO[j] + sourceCNO[j]

```

```

1314 TotalSE[j] <- sourceSSE[j] + sourceCSE[j]
1315 TotalSO[j] <- sourceSSO[j] + sourceCSO[j]
1316
1317 AttribCOC[j] <- (TotalCOC[j] * 100) / totaloutbreaksCOC
1318 AttribCOR[j] <- (TotalCOR[j] * 100) / totaloutbreaksCOR
1319 AttribMPA[j] <- (TotalMPA[j] * 100) / totaloutbreaksMPA
1320 AttribNE[j] <- (TotalNE[j] * 100) / totaloutbreaksNE
1321 AttribNO[j] <- (TotalNO[j] * 100) / totaloutbreaksNO
1322 AttribSE[j] <- (TotalSE[j] * 100) / totaloutbreaksSE
1323 AttribSO[j] <- (TotalSO[j] * 100) / totaloutbreaksSO
1324     }
1325     }
1326
1327
1328 # DATA
1329 list(
1330   COCs1 = 1 ,
1331   COCs2 = 2 ,
1332   COCc1 = 3 ,
1333   COCc2 = 29 ,
1334
1335   CORs1 = 35 ,
1336   CORs2 = 38 ,
1337   CORc1 = 39 ,
1338   CORc2 = 90 ,
1339
1340   MPAs1 = 104 ,
1341   MPAs2 = 120 ,
1342   MPAc1 = 121 ,
1343   MPAc2 = 225 ,
1344
1345   NEs1 = 271 ,
1346   NEs2 = 281 ,
1347   NEc1 = 282 ,

```

1348 NEc2 = 400 ,
 1349
 1350 NOs1 = 446 ,
 1351 NOs2 = 470 ,
 1352 NOc1 = 471 ,
 1353 NOc2 = 714 ,
 1354
 1355 SEs1 = 790 ,
 1356 SEs2 = 790 ,
 1357 SEc1 = 791 ,
 1358 SEc2 = 803 ,
 1359
 1360 SOs1 = 810 ,
 1361 SOs2 = 810 ,
 1362 SOc1 = 811 ,
 1363 SOc2 = 815 ,
 1364
 1365
 1366
 1367
 1368 unknownCOC = 5 ,
 1369 unknownCOR = 13 ,
 1370 unknownMPA = 45 ,
 1371 unknownNE = 45 ,
 1372 unknownNO = 75 ,
 1373 unknownSE = 6 ,
 1374 unknownSO = 2 ,
 1375 ,
 1376 totaloutbreaksCOC = 34 ,
 1377 totaloutbreaksCOR = 69 ,
 1378 totaloutbreaksMPA = 167 ,
 1379 totaloutbreaksNE = 175 ,
 1380 totaloutbreaksNO = 344 ,
 1381 totaloutbreaksSE = 20 ,
 1382 totaloutbreaksSO = 8 ,
 1383

[illegible]

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1612 **# INITIAL VALUES**

1613

1614 `list(PriorS= c(0.1,0.1,0.1,0.1,0.1,0.1,0.1,0.1,0.1,0.1,0.1,0.1,0.1,0.1,0.1,0.1,0.1,0.1))`

1615

1616 `list(PriorS`

1617 `=c(0.01,0.01,0.01,0.01,0.01,0.01,0.01,0.01,0.01,0.01,0.01,0.01,0.01,0.01,0.01,0.01,0.01,0.01))`

1618

1619 `list(PriorS =c(0.2,0.2,0.2,0.2,0.2,0.2,0.2,0.2,0.2,0.2,0.2,0.2,0.2,0.2,0.2,0.2,0.2,0.2))`

1620

1621 `list(PriorS`

1622 `=c(0.05,0.05,0.05,0.05,0.05,0.05,0.05,0.05,0.05,0.05,0.05,0.05,0.05,0.05,0.05,0.05,0.05,0.05))`

1623

1624 `list(PriorS =c(0.3,0.3,0.3,0.3,0.3,0.3,0.3,0.3,0.3,0.3,0.3,0.3,0.3,0.3,0.3,0.3,0.3,0.3))`

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```

1659 # model for SALMONELLA and 12 sources (including water): locations' sub-analysis
1660 # data set: first all simple foods (o in s1: S)
1661 # complex foods (o in c1:C)
1662
1663
1664 # variables:
1665 # out[i] = outbreak per pathogen
1666 # s = outbreak observation in simple foods
1667 # i = outbreak observation in complex foods
1668 # j = source
1669 # PriorS = prior knowledge we gain from simple food outbreaks for each source
1670 # F = presence or absence of source j in complex food f, matrix
1671 #sources:
1672 #1Eggs2Dairy3GoatMilk4Meat5Poultry6Chicken7Ducks8Turkey9Beef10Pork11Lamb12Mutton13Game14F
1673 ruitsNuts15Vegetables16GrainsBeans17OilsSugar18Seafood19Water
1674
1675 #1Beef2Pork3Poultry4Meat5Dairy6Eggs7Seafood8FruitsNuts9GrainsBeans10Vegetables11OilSugar12
1676 Water
1677
1678
1679 model
1680 {
1681
1682 # Proportion of unknowns
1683
1684
1685
1686
1687 propukG <- unknownG * 100/ totaloutbreaksG
1688 propukH <- unknownH * 100/ totaloutbreaksH
1689
1690
1691 # Variable to estimate attribution of complex foods, separated loop (out of order)

```

```

1692
1693  #G (General)
1694  for (i in Gc1:Gc2 ) {
1695    b[i] <- sum(a[1:12,i])
1696
1697  }
1698
1699  #H (Household)
1700  for (i in Hc1:Hc2) {
1701    b[i] <- sum(a[1:12,i])
1702
1703  }
1704
1705
1706
1707  for (j in 1:12) {
1708
1709    # PRIOR
1710
1711
1712    sourceS[j] <- sum(S[j,1:totalS])
1713
1714
1715    z[j] <- sourceS[j] +1
1716    y[j] <- totalS - sourceS[j] +1
1717    PriorS[j] ~dbeta(z[j], y[j])
1718
1719
1720
1721
1722
1723

```

```

1724  # BY LOCATION
1725
1726
1727  #G
1728
1729  # SIMPLE FOODS
1730    sourceSG[j] <- sum(F[j,Gs1:Gs2])
1731
1732  # COMPLEX FOODS
1733    for (i in Gc1:Gc2) {
1734
1735      a[j,i] <- PriorS[j] * F[j,i]
1736      c[j,i] <- a[j,i] / b[i]
1737
1738    }
1739
1740
1741  #H
1742
1743  # SIMPLE FOODS
1744    sourceSH[j] <- sum(F[j,Hs1:Hs2])
1745
1746  # COMPLEX FOODS
1747    for (i in Hc1:Hc2) {
1748
1749      a[j,i] <- PriorS[j] * F[j,i]
1750      c[j,i] <- a[j,i] / b[i]
1751
1752    }
1753
1754

```



```

1755
1756
1757
1758   sourceCG[j] <- sum(c[j,Gc1:Gc2 ])
1759   sourceCH[j] <- sum(c[j,Hc1:Hc2])
1760
1761
1762   TotalG[j] <- sourceSG[j] + sourceCG[j]
1763   TotalH[j] <- sourceSH[j] + sourceCH[j]
1764
1765
1766   AttribG[j] <- (TotalG[j] * 100) / totaloutbreaksG
1767   AttribH[j] <- (TotalH[j] * 100) / totaloutbreaksH
1768
1769       }
1770   }
1771
1772
1773   # DATA
1774
1775
1776   list(
1777
1778
1779     Gs1 = 1 ,
1780     Gs2 = 20 ,
1781     Gc1 = 21 ,
1782     Gc2 = 243 ,
1783
1784     Hs1 = 313 ,
1785     Hs2 = 354 ,
1786     Hc1 = 355 ,

```

[illegible]

[illegible]