ASSESSING PREVALENCE AND CHARACTERIZING *SALMONELLA* POPULATIONS WITHIN ENVIRONMENTAL RESERVOIRS

by

JARED CHRISTOPHER SMITH

(Under the Direction of Nikki W. Shariat)

ABSTRACT

Salmonella enterica is a leading cause of bacterial foodborne illness. Ubiquitous in nature, this pathogen is well characterized in food animals; however, the prevalence, diversity, and dynamics of *Salmonella* in the environment are understudied. This introduces concern of food safety threat spillover from environmental reservoirs such as wild birds and surface water. Recent outbreaks linked to wild birds and surface water demonstrate the need to identify sources of *Salmonella* in our food systems to reduce the incidence and severity of illnesses from environmental contamination. To better understand routes of *Salmonella* transmission at the environment agriculture interface, three studies were completed that aimed to assess prevalence and characterize *Salmonella* populations within environmental reservoirs. First, wild bird feces were collected from produce fields in the southeastern United States, identifying a low prevalence of *Salmonella* and a strong association of viable pathogen with fresh feces. Results of this study suggest, for the first time in this region, a limited food safety risk attributed to wild birds compared to studies on the West Coast finding a more significant risk. This led to identifying more likely routes of produce contamination, including surface water.

Second, a two-year longitudinal surveillance study of four surface creeks determined high *Salmonella* prevalence and serovar complexity, where modeling showed a positive correlation between both prevalence complexity to weather variables such as precipitation and humidity. The incidence of antimicrobial resistance and clinically relevant serovars within complex populations supports the need for consistent monitoring of surface water sources. Finally, a comparative study between two rivers in Pichincha, Ecuador found higher prevalence and complexity in the river within an urban environment compared to a river embedded in animal agriculture. This data highlights potential human health risks associated with contaminated surface water and suggests a need for continued surveillance. Overall, these studies have significantly improved our understanding of *Salmonella* ecology in various environmental reservoirs. Considerable knowledge gaps remain, such as more fully elucidating the complex influence of weather and physiochemical variables on *Salmonella* presence in surface water. Additionally, many wildlife reservoirs remain understudied, limiting our understanding of *Salmonella* ecology and transmission outside humans and food systems.

INDEX WORDS: *Salmonella enterica*, food safety, deep serotyping, environmental surveillance, wild birds, surface water, whole genome sequencing, antimicrobial resistance

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DEDICATION

To my mom and dad, Paula and Chris: Your continuous love and support are an immense part of my success. I love you both so much and hope to make you proud every day. Thank you for all that you have done for me!

To my sisters, Lauren and Kadi: I have always seen you both as role models for your commitment to achieving both personal and career goals as well as always pushing me to do the same, no matter how hard things get. I will never be able to thank you enough for the encouragement you both have given me, I love you two!

To my friends (thankfully too many to list here): I would never have reached this point without the love, support, and relief each of you have given me during one of the most challenging times of my life. I have relied on each of you and hope that, in time, I will be able to return a small piece of the joy you have brought me!

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To my lab community: we have always shared in our successes and have relied on one another to achieve greatness. I will forever be thankful for the support of Dr. Amy Siceloff, as we came to and left the lab together, supporting each other through the best and worst of times. To all of the postdoctoral, graduate, and undergraduate students who worked with me, helped with sampling, and provided critical feedback for so many projects over my time in lab; I look forward to cheering in your success that will surely follow in the years to come.

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CHAPTER 1

LITERATURE REVIEW

1.1 *Salmonella*

Salmonella was first discovered by Dr. Theobald Smith in 1885 during an investigation of pig intestines and subsequently named after his collaborator, Dr. Daniel Elmer Salmon (Salmon & Smith, 1891). This gram-negative, rod-shaped, flagellated, facultative anaerobic bacterium is part of the *Enterobacteriaceae* family. *Salmonella* is the causative agent of salmonellosis, gastroenteritis caused by *Salmonella,* which is generally self-limiting inflammation of the gastrointestinal system (Centers for Disease Control and Prevention, 2023). This can be identified by diarrhea, abdominal cramps, fever, headache, and nausea which can often be cleared naturally within 7-10 days or with the assistance of antibiotics when necessary. Salmonellosis impacts an estimated 1.35 million people in the US each year, with approximately 23,000 hospitalizations and 420 deaths annually (Centers for Disease Control and Prevention, 2023).

Salmonella **serovars**

Salmonella enterica is highly diverse, including 2,659 serovars (P. A. D. Grimont & Weill, 2007; Issenhuth-Jeanjean et al., 2014). These serovars are designated in the White-Kauffman-Le minor scheme and are phenotypically differentiated by the detection of surface antigens, including the somatic (O), flagellar (H), and capsular (Vi) antigens (Brenner et al., 2000; P. A. D. Grimont & Weill, 2007). Variability within the structure of these surface antigens is the basis for *Salmonella* serotyping. The somatic antigen is the

outermost repeating carbohydrate in the lipopolysaccharide (LPS) layer on the *Salmonella* surface. The LPS layer serves as a barrier to low pH and protects *Salmonella* from the innate immune response, where releasing the lipid A component can trigger a strong immune response (Ernst et al., 2001). Currently, there are 47 variations of the O-antigen that have been identified. All serovars with common O-antigen identity belong to the same serogroup (Grimont and Weill, 2007). *Salmonella* also possesses a flagellum containing an H antigen, of which there are 119 variations. The flagella contains one of two antigens, referred to as H1 or H2, identified by the expression of two genes encoded by the *fliC* and *fljB* genes, respectively(Andrewes, 1922; Macnab, 1996). Because flagella are often the target of the host immune response, *Salmonella* can change the translation of the flagellar antigen between the two proteins (H1/H2) in a process known as phase variation (Silverman et al., 1979). Some serovar variants cannot change phases between the H1 and H2 antigens due to mutations or deletions of either the *fliC* or *fljB* gene. These variants are designated as monophasic serovars such as serovar I 4,5,[12],i:-, a monophasic variant of serovar Typhimurium (Kauffmann, 1964). Additional serovars that lack an H2 antigen include Enteritidis, Typhi, Dublin, and Senftenberg. Notably, serovar Gallinarum does not contain the gene for either flagellar antigen, resulting in nonflagellated and nonmotile cells (P. A. D. Grimont & Weill, 2007). Finally, the Vi antigen, a linear polymer consisting of O-acetylated α-1,4-linked N-acetylgalactosaminuronic acid, is found on the surface of the *Salmonella* capsule (H. Zhang et al., 2006). This antigen is only found in serovars Typhi, Paratyphi C, and Dublin and is associated with enteric disease (Santander et al., 2008).

Salmonella enterica subspecies *enterica* can be divided into two categories that separate typhoidal from non-typhoidal salmonellae (Crump et al., 2004). Serovars Typhi, Sendai, and Paratyphi (A, B, and C), are considered typhoidal serovars and the causative agents of typhoid (enteric) fever and paratyphoid fever (Gal-Mor et al., 2014). Serovars that do not often result in enteric disease are considered non-typhoidal *Salmonella* (NTS). These serovars often induce self-limited gastrointestinal inflammation known as salmonellosis, and in some cases, invasive extra-intestinal bacteremia, that can be more life-threatening (Mandal & Brennand, 1988). At 1582 serovars, these make up the overwhelming majority of serovar diversity (Issenhuth-Jeanjean et al., 2014). Alternatively, nearly 5% of illnesses in sub-Saharan Africa and southeast Asia result in enteric fever-like illness from NTS serovars, such as Enteritidis, Typhimurium, Dublin, and Cholerasuis, where these strains are categorized as invasive NTS (iNTS) (Mandal & Brennand, 1988). The separation of typhoidal and non-typhoidal salmonellae is based on human illness; however, some serovars have unique pathogenicity within specific hosts, such as the presence of typhoid fever-like symptoms in mice colonized with serovar Typhimurium (Santos et al., 2001), serovar Gallinarum causing fowl typhoid in poultry (Pascopella et al., 1995), serovar Abortusovis causing sporadic abortions in sheep (Belloy et al., 2009), and serovar Cholerasuis causing pneumonia and septicemia in swine (Gray et al., 1996). The differences in morbidity between these serovars are an example of the phenotypic and genotypic plasticity between serovars of *Salmonella enterica* subspecies *enterica*.

While the antigenic profile is used to determine the serovar identity, other genomic differences including virulence factors (Andino & Hanning, 2015),

antimicrobial resistances (D. H. Shah et al., 2017), stress response (Gorski & Noriega, 2023), and host restriction adaptations can drastically impact the clinical relevance of a serovar (Cheng et al., 2019). *Salmonella enterica* subspecies *enterica* contains 1,586 serovars, where 80 serovars accounted for 99.9% of infections in the United States between 2018 and 2024 (CDC, 2024a; Hendriksen et al., 2011). Further, more than 75% of those cases were attributed to only 13 serovars (Figure 1.1). These differences are attributed to the requirements for a clinical case to manifest: for a serovar to colonize and cause illness in a human, it must have the virulence factors available to attach, invade, and multiply within human epithelial cells and macrophages (Ernst et al., 1990; Knodler et al., 2010; Monack et al., 1996). Additionally, serovars have different dose responses, meaning that the number of cells required to cause illness is variable between serovars (M. Kim et al., 2024; Teunis et al., 2010). For serovars attributed to non-host contamination, such as produce or water, they must be able to overcome the environmental stressors outside of the host, such as temperature (J. Shah et al., 2013; Sirsat et al., 2011), pH (Spector & Kenyon, 2012), nutrient limitation (Stenström et al., 1989), and radiation (Ormsby et al., 2024). Finally, serovars attributed to food animals must have the adaptations to survive transmission. This includes colonizing both animal and human hosts, overcoming environmental stress between hosts, and facilitating transmission from one host to another (Figure 1.2) (Siceloff et al., 2022).

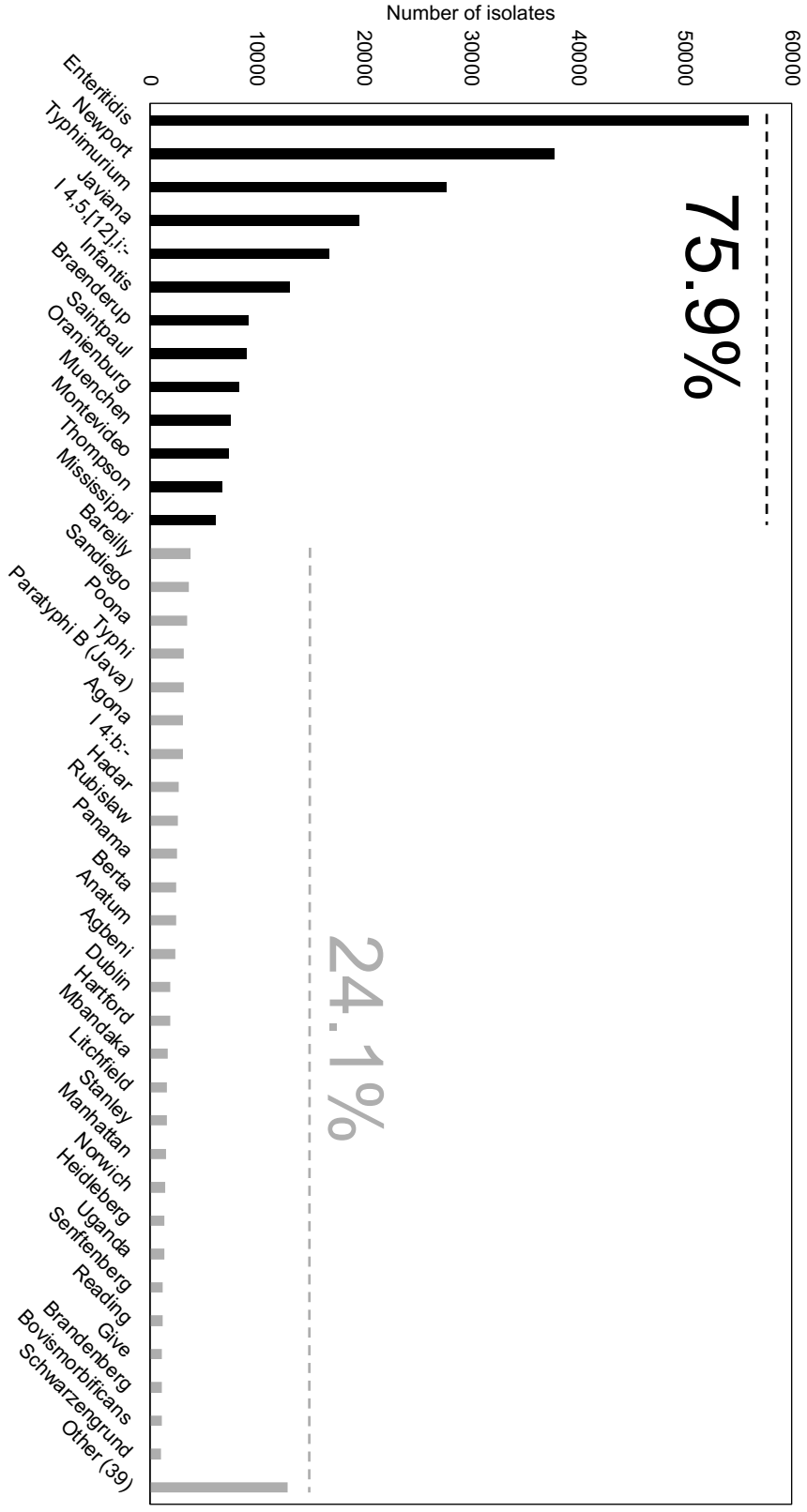
Number of isolates
0
10000
20000
30000
40000
50000
60000
Enteritidis
Newport
Typhimurium
Javiana
I 4,5,[12],i:-
Infantis
Braenderup
Saintpaul
Oranienburg
Muenchen
Montevideo
Thompson
Mississippi
Bareilly
Sandiego
Poona
Typhi
Paratyphi B (Java)
Agona
I 4:b:-
Hadar
Rubislaw
Panama
Berta
Anatum
Agbeni
Dublin
Hartford
Mbandaka
Litchfield
Stanley
Manhattan
Norwich
Heidleberg
Uganda
Senftenberg
Reading
Give
Brandenberg
Bovismorbificans
Schwarzengrund
Other (39)
75.9%
24.1%

Figure 1.1. Human *Salmonella* isolates from the United States between January 2018 and November 2024. Total isolates from January 2018 through November 2024 are shown next to their respective serovar. Serovars that account for 75.9% of total isolates are shown in black. Serovars with less than 1000 isolates during this time are grouped as "other" where this group contains data from 39 serovars. Data from CDC Beam Dashboard (CDC, 2024a).

Some serovars have adaptations that better promote environmental survival

Surviving cells will not always be introduced to humans

Transmitted cells must survive host conditions and evade immune response

If all previous steps occur, colonization can proceed

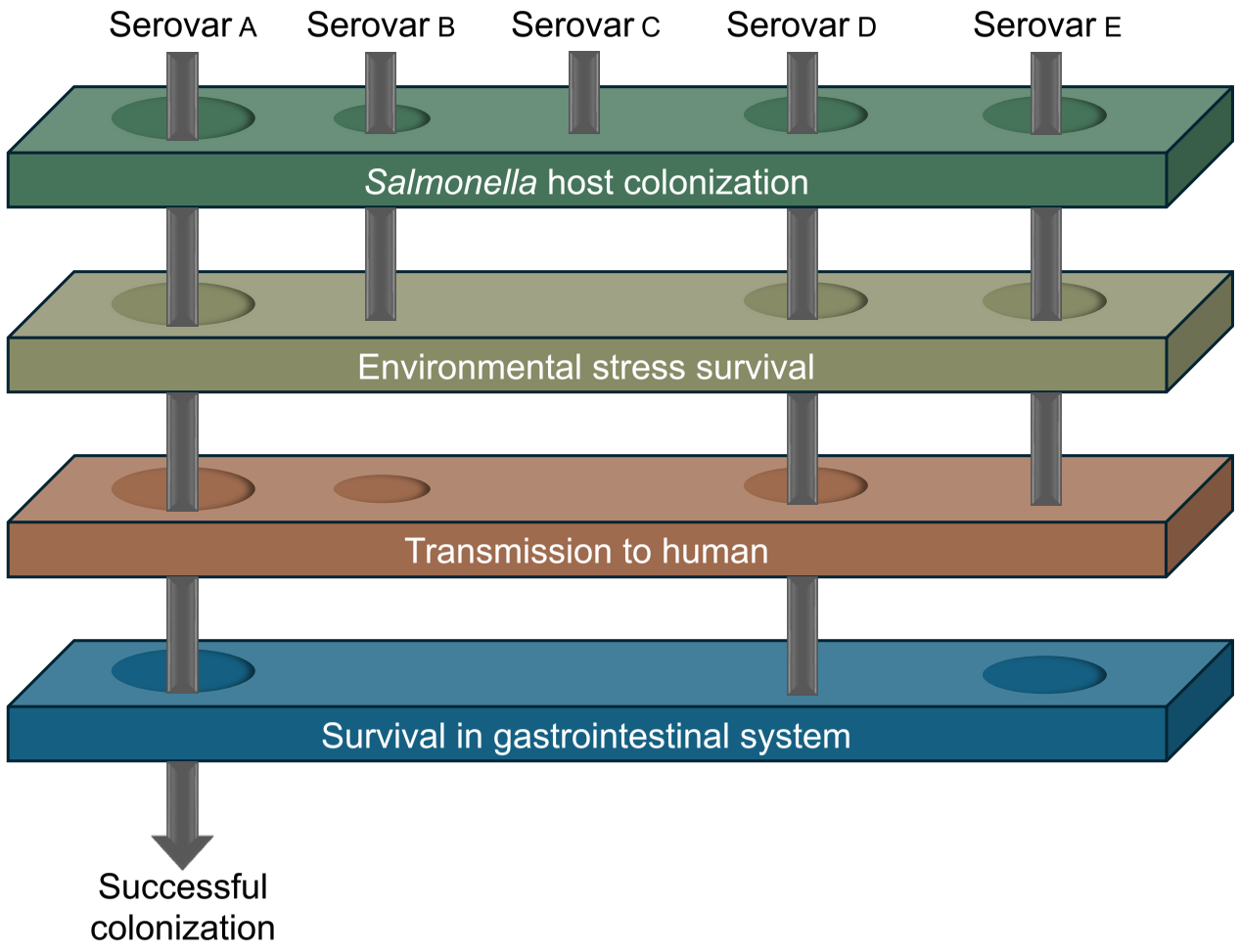

Figure 1.2. Barriers to successful human colonization by *Salmonella*. Rectangles represent obstacles that must be overcome for human colonization by *Salmonella*, where holes in a rectangle represent adaptations or opportunities for a potential serovar to overcome this obstacle. A gray arrow represents the passage of a serovar through the barriers, stopping if adaptations or opportunities are not present to move forward.

Salmonella evolution and phylogeny

The genus *Salmonella* consists of two species, *Salmonella enterica* and *Salmonella bongori* (P. A. D. Grimont & Weill, 2007; Tindall et al., 2005). These species both possess the *Salmonella* pathogenicity island 1 (SPI-1), which differentiates them from other *Enterobacteriaceae* and also promotes host invasion via a type-three secretion system (T3SS) and effector proteins (Lou et al., 2019). *S. enterica* is further differentiated by the addition of SPI-2 containing a secondary T3SS and effector proteins allowing for invasion, survival, and replication within macrophages (Figueira & Holden, 2012). The lack of SPI-2 and the evolution of non-mammalian targeted effector proteins in *Salmonella bongori* restricts hosts to cold-blooded animals, such as reptiles (Bäumler et al., 1998; Hensel, 2000). Within *Salmonella enterica*, there are currently six subspecies, *enterica* (I)*, salamae* (II), *arizonae* (IIIa), *diarizonae* (IIIb)*, houtenae* (IV), and *indica* (VI) (P. A. D. Grimont & Weill, 2007). While these six subspecies can be differentiated using biochemical tests for phenotypic and metabolic identifiers, they typically occupy separate ecological niches (Ewing & Edwards, 1986; P. A. D. Grimont & Weill, 2007). *Salmonella enterica* subspecies *enterica*, unlike other subspecies, has evolved rapidly to target colonization of warm-blooded animals and makes up more than 99% of human clinical infections (Hadjinicolaou et al., 2009). In rare cases, non-subsp. *enterica* isolates are recovered from humans; however, these are often attributed to young children with weakened immune systems or co-infections with a subsp. *enterica* serovar (Editorial team et al., 2008; Mughini-Gras et al., 2016).

Host specialization

Salmonella serovars can exhibit varying host preferences, ranging from generalist to restricted. Host restriction is the least common, where the serovar can only colonize a single species or a restricted set of organisms, and is unable to colonize most other hosts. *Salmonella* serovars Gallinarum, Typhi, and Abortusovis are examples of this designation (Uzzau et al., 2000). Poultry and aquatic birds are the sole hosts of serovars and Gallinarum and its biovar Pullorum, causing high mortality due to fowl typhoid and Pullorum disease, respectively. These serovars were common in the United States and led to high economic loss in the early 1900s before the National Poultry Improvement Plan (NPIP) put forth mitigation strategies that successfully eradicated these salmonellae from commercial flocks (Andino & Hanning, 2015; Bullis, 1977; Kabir, 2010). Serovar Typhi and the other typhoidal serovars are host-restricted to humans and higher primates (Pascopella et al., 1995). Finally, serovar Abortusovis is restricted to sheep, where it causes sporadic abortions and stillbirths in pregnant ewes (Uzzau et al., 2000).

Host-adapted serovars may specialize in a single host but are capable of colonizing one or more other hosts. Examples of this include serovars Dublin and Cholerasuis, where the primary hosts are cattle, and swine, respectively (Uzzau et al., 2000); however, illness in humans is possible following consumption of or direct contact with infected animals. Serovars Dublin and Cholerasuis are less common in humans, but illness from these serovars can be exacerbated by antimicrobial resistance (AMR) and rare systemic infections (Chiu et al., 2002). Finally, host generalists are ubiquitous in their ability to colonize a wide range of hosts, such as serovars Typhimurium and Infantis. Serovar Typhimurium is one of the leading causes of human salmonellosis in the United States and can be found in almost all warm-blooded animals (Baumler & Fang,

2013). Serovar Infantis has become more prominent over the past two decades, as shown by its prominence in poultry, cattle, swine, and turkey environments and the presence of the pESI megaplasmid containing antimicrobial and heavy metal tolerance (Gal-Mor et al., 2010; McMillan et al., 2020). Infantis now commonly identified as one of the top ten serovars identified in all regularly monitored meat and poultry products in the United States (USDA-FSIS, 2024a).

Impact of *Salmonella* on human health

Foodborne illness is observed an estimated 179 million people in the United States each year. The cases that can be directly linked to the consumption of contaminated food are estimated at 48 million (S. Hoffmann et al., 2012). Of these cases, *Salmonella* is the leading contributor to bacterial foodborne illness, hospitalizations, and deaths (S. Hoffmann et al., 2012). The economic burden of *Salmonella* illness is estimated at over $4.1 billion, accounting for the highest proportion of the $17.5 billion burden of all 15 leading foodborne pathogens (USDA-ERS, 2023). These illnesses are often caused by the consumption of contaminated food and water and are commonly identified in raw meat and uncooked produce, but can also be found in eggs, dairy, nuts, flour, and other commodities (Bintsis, 2017). To better predict the risk associated with different food groups, data is shared between federal agencies, including the Centers for Disease Control and Prevention (CDC), the United States Food and Drug Administration (FDA), and the United States Department of Agriculture (USDA) to create the Interagency Food Safety Analytics Collaboration (IFSAC). Each year, a report is released that estimates the source attribution of foodborne illness outbreaks based on data collected between 1998 and two years before the reporting year. These estimates are

developed from a model that relies more heavily on more recent outbreaks (within the previous five years) (Batz et al., 2021). Estimates are given for *Salmonella enterica*, *E. coli* O157, *Listeria monocytogenes*, and *Campylobacter jejuni* (until 2020). Annual data suggests a trend toward increasing foodborne illness attribution to meat and poultry, surpassing produce in total outbreak attribution in 2022 (Figure 1.3) (IFSAC, 2024). Other significant bacterial causes of foodborne illness include *E. coli*, *Listeria monocytogenes,* and *Campylobacter jejuni*. The sources of foodborne illness are more variable in *Salmonella* compared to other bacterial foodborne pathogens such as that of *E. coli* where vegetable row crops (62.2%) and beef (20.9%) alone account for 85.1% of estimated illnesses (IFSAC, 2024). This highlights the complex and widespread nature of *Salmonella* and its ability to cause illnesses from a variety of reservoirs.

Of the estimated 1.35 million salmonellosis cases each year, outbreaks account for only 10% of these (Scallan et al., 2011). Sporadic infections are more common, often due to the consumption of improperly cooked or cleaned food and interactions with contaminated animals, meat, or fecal material (Tack et al., 2019). To limit bias, the Foodborne Disease Active Surveillance Network (FoodNet) includes collected clinical data from ten states (Oregon, Colorado, New Mexico, Minnesota, Tennessee, Georgia, Delaware, Connecticut, and select counties in California and New York), outbreak data from the FDA, and routine surveillance of meat, poultry, and egg products by the USDA-Food Safety Inspection Service (UDSA-FSIS). FoodNet was formed in 1995 and currently includes testing for eight foodborne pathogens including *Campylobacter, Listeria, Salmonella,* STEC*, Shigella, Vibrio, Yersinia, and Cyclospora*. Between 2000 and 2022, 166,103 *Salmonella* infections have been identified on FoodNet. Serovars

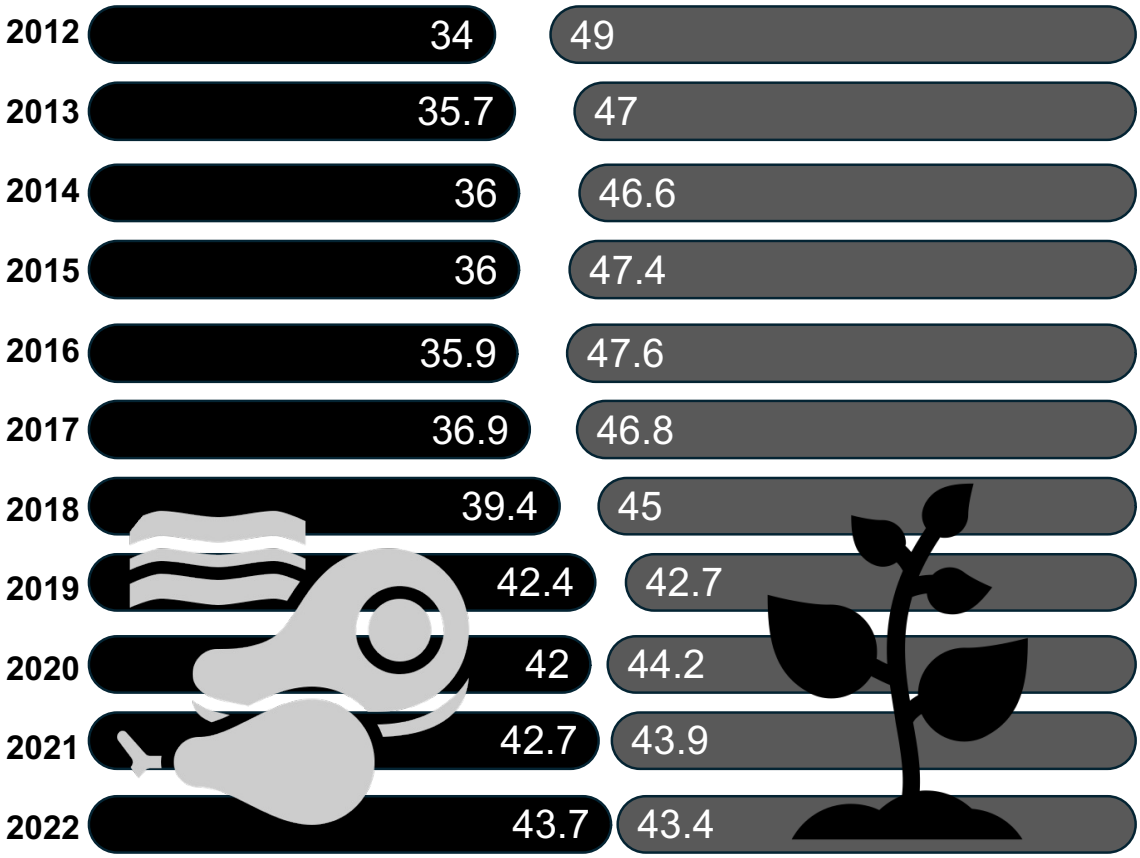

Figure 1.3. Estimated *Salmonella* foodborne illness attribution of meat and poultry vs. produce from 2012-2022. Meat and poultry (black) include chicken, pork, beef, and turkey. Produce (gray) contains fruits, seeded vegetables, vegetable row crops, sprouts, grains and beans, and "other produce" categories from each annual IFSAC report. Percentages are estimated by combining outbreak data from 1998 to each year, weighing the five most recent years more heavily than prior years. Categories not shown include dairy, eggs, seafood, game, oils-sugars, and "other meat/poultry".

Enteritidis, Typhimurium, and Newport were the top three serovars during this time, accounting for 39% of cases (Centers for Disease Control and Prevention, 2022). While Foodnet only covers approximately 16% of the US population, serovar trends align closely with data from nationwide clinical cases (Figure 1.1). During each year of this period, these three serovars were consistently the top three serovars, except for 2020, where serovar Javiana replaced Typhimurium, although the COVID-19 pandemic likely affected data from this year. Notably, these three serovars are identified most often globally, combining to account for 64.1% of all *Salmonella* cases between 2001 and 2007 (Hendriksen et al., 2011).

Additionally, antimicrobial resistance (AMR) in *Salmonella*, while generally decreasing, continues to be problematic for human clinical cases (Michael & Schwarz, 2016). AMR is a significant contributor to higher financial burden for *Salmonella* illnesses, increasing the length and cost of hospital stays and increasing morbidity of illness (Chiu et al., 2002). This is shown by a persistent multidrug-resistant strain of serovar Newport (REPJJP01) between 2016 and 2022. Beef and cattle products from Mexico associated with this outbreak were linked to high numbers of human illnesses, resulting in one-third of identified patients being hospitalized (Ford et al., 2023). To better understand the extent of AMR in the US, the National Antimicrobial Resistance Monitoring System (NARMS) was established in 1996 in partnership between the CDC, FDA, and USDA to identify incidence and patterns of antimicrobial resistance in humans, food animals, and retail meats. *Salmonella, Campylobacter, Enterococcus,* and *E. coli* isolates from humans, routine USDA surveillance and FDA retail sampling are tested using a combination of minimum inhibitory concentration (MIC) testing and whole

genome sequencing (WGS) analysis. From 1996 to 2023, 20.5% (10,808/52,471) of human clinical isolates have shown resistance to one or more antimicrobials (Figure 1.4) (CDC, 2025). The percentage of resistant isolates per year has slightly decreased over this time, likely due to improving AMR stewardship in agricultural and medical fields as well as continued efforts both nationally and internationally to combat antibiotic-resistant bacteria (CDC, 2015; FDA, 2015). While regulations such as the implementation of the veterinary feed directive and stewardship programs in hospitals have reduced the impact of AMR, alternative antibacterial approaches and improved antibiotic stewardship education are needed to reduce the burden of *Salmonella* infections.

Although *Salmonella* is an enteric pathogen, human health can be impacted through consumption of non-animal sources as well. Surprisingly, produce is also a major contributor to *Salmonella* outbreaks, accounting for the largest proportion of illnesses from outbreaks since 2012 (Figure 1.3). For these outbreaks to occur, produce must be contaminated by feces from an animal source; however, the sources of contamination are not well characterized. To better prevent these contamination events, there is a need to better understand the sources and routes of contamination on produce.

Wildlife is a largely understudied source of *Salmonella* (Langholz & Jay-Russell, 2013). As wild animals can move freely between natural habitats, agricultural land, and human-populated areas, the transmission of microbes between humans and animals is likely to occur (Dias-Alves et al., 2023). While often limited to sampling of sporadic fecal samples, *Salmonella* surveillance in wildlife has been accomplished, including racoons (Maurer et al., 2015a; Very et al., 2016), deer (Renter et al., 2006; Salas-Rosas et al., 2020; Topalcengiz et al., 2020),

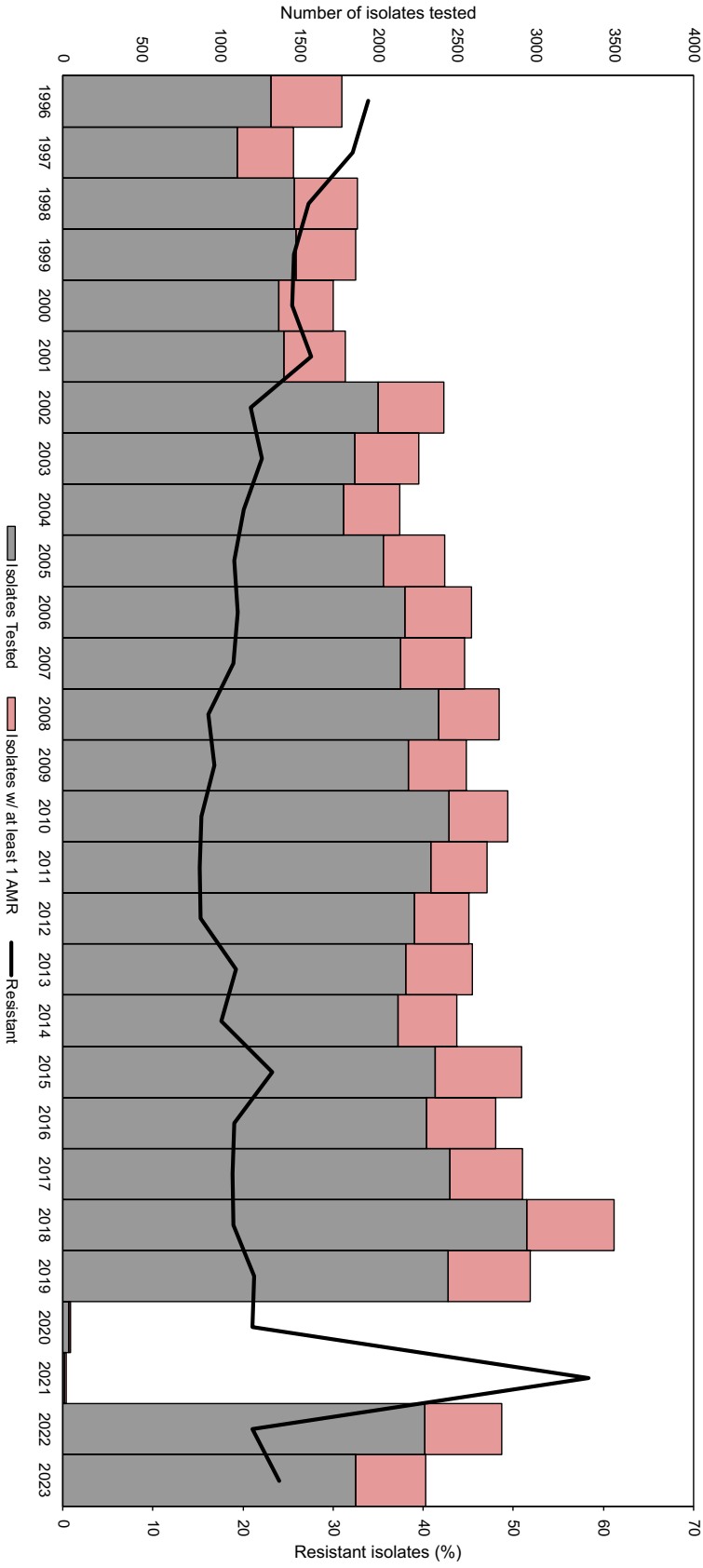
Number of isolates tested
0
500
1000
1500
2000
2500
3000
3500
4000
1996
1997
1998
1999
2000
2001
2002
2003
2004
2005
2006
2007
2008
2009
2010
2011
2012
2013
2014
2015
2016
2017
2018
2019
2020
2021
2022
2023
Isolates Tested
Isolates w/ at least 1 AMR
Resistant
0
10
20
30
40
50
60
70
Resistant isolates (%)

Figure 1.4. Instance of antimicrobial resistance in human *Salmonella* isolates (1996-2023). Human *Salmonella* isolates collected by the CDC with at least one antimicrobial resistance (red) are shown in comparison to all isolates tested (gray). The percentage of resistant isolates (black line) is shown for each year. Years 2020 and 2021 contained significantly fewer samples due to reduced sampling during COVID-19. Data adapted from CDC NORS (National Outbreak Reporting System (NORS), 2022).

reptiles (Doden et al., 2021; Pulford et al., 2019; Saelinger et al., 2006), bats (Adesiyun et al., 2009; McDougall & Power, 2021), and other animals (Millan et al., 2004; Uelze et al., 2021). These studies are necessary, as they can identify serovars not often identified in food animals. Importantly, these serovars are sometimes associated with human illness, suggesting the need for continued surveillance (Pees et al., 2023; Waltenburg et al., 2022). Due to the expertise required for extensive tracking, trapping, and sampling of these sources, longitudinal studies and large-scale sampling efforts have been limited. This knowledge gap restricts the development of mitigation strategies and risk assessment associated with wildlife.

Additionally, *Salmonella* can be found in abiotic sources such as soil and dust. This pathogen is particularly adaptable to conditions outside a host where it has been recovered as many as 332 days following application into soil (You et al., 2006). The ability of *Salmonella* to persist in the environment is exhibited in studies showing detection in dust particles, where viable isolates can be recovered after a significant reduction in moisture (Khouja et al., 2024; Pal et al., 2021). This ability to survive outside a host also allows for the transmission of *Salmonella* from a host to the environment by the spread of fecal material. Fecal samples are an effective tool for studying environmental transmission of *Salmonella,* as feces are indicative of the serovars being shed into the environment. Prevalence and quantification of *Salmonella* within various wildlife feces have been detected for 364 days, suggesting that wildlife pathogens can remain present in the environment for long periods (Topalcengiz et al., 2020). If *Salmonella* is present in the environment, the potential impact on human health can have long-term consequences (Oni et al., 2015). *Salmonella* persistence in these

abiotic sources contributes to food safety threats in produce, as transmission from soil or dust to produce can be difficult to prevent.

1.2 Produce Safety and *Salmonella*

Compared to meat and poultry, produce contaminated with *Salmonella* poses a unique problem, in that there is often no "kill" step to eliminate pathogens (Carstens et al., 2019). Cooking meat and poultry, if done properly, will prevent foodborne illness; however, most produce is eaten raw. After produce harvesting, an antimicrobial wash is often used containing an agent such as peracetic acid (PAA) or a chlorine product; however, this only affects the portion of the surface that encounters the wash (Krishnan et al., 2023). In many cases, the surface of the produce can often contain folds, holes, or other barriers to this wash, where pathogens such as *Salmonella* can "hide" from the antimicrobial agent (Barak et al., 2005). This is particularly concerning in many leafy greens including spinach and lettuce that can trap contaminants between leaves (Grivokostopoulos et al., 2022). Additionally, there is evidence to suggest that some *Salmonella* can invade produce via fruit, stems, leaves, or blossoms of fruiting vegetation, shielding the pathogen from washing (Burris et al., 2020; Hintz et al., 2010). Because of this, mitigation to prevent initial contamination is considered the most effective way to enhance produce food safety. Mitigation strategies include proper management of worker hygiene, irrigation water, soil and soil amendments, and wildlife before contacting the produce.

Between 2009 and 2023, the National Outbreak Reporting System (NORS) identified 638 *Salmonella* outbreaks from produce alone (CDC, 2024a). Significant outbreaks include the sickening of 1,127 and 1,040 people from whole onions in 2020

and 2021, respectively (CDC, 2024d). This outbreak was unique, as produce outbreaks are generally short-lived due to short shelf-life; however, onions have a long shelf-life and therefore contributed to months-long investigations during these outbreaks. A history of serovar Newport outbreaks in cucumbers and tomatoes from the eastern shore of Virginia has most recently resulted in the sickening of 275 patients and a single death in 2014. This persistent strain has been identified in at least 10 outbreaks and likely persists in the environment (Gruszynski et al., 2014a). Finally, a 2022 outbreak of serovar Typhimurium in melons led to the sickening of 87 people (FDA, 2023b). While no contamination source was identified, traceback efforts suggested a link to contaminated soil amendments (FDA, 2023a). As only a portion of these outbreak tracebacks identify the cause of produce contamination, this suggests a need for improved source attribution through rigorous surveillance testing.

Pathogen introduction to produce

The origins of foodborne pathogen contamination in fruits and vegetables are quite diverse (Figure 1.5) (Alegbeleye et al., 2018; Devarajan et al., 2023). These routes of contamination can include the application of contaminated soil or soil amendments that can transfer to growing plants or that can be splashed onto produce following rain or irrigation (Jechalke et al., 2019a; Lee et al., 2019). Biological amendments such as animal manure can have great nutritional benefits for produce plants; however, *Salmonella* can be identified at high loads and for extended periods in manure if left untreated or improperly treated (Hutchison et al., 2004; You et al., 2006). *Salmonella* has the potential to invade germinating seedlings or attach to the leaves and stems of early-stage plants (Barak & Liang, 2008; FDA, 2011). To mitigate these risks, regulations have


Used with permission – Microsoft stock images, 2025
Used with permission – Microsoft stock images, 2025
Bird droppings
Runoff
Amended soil
Splashing
Worker activity
Irrigation
Fecal contamination
Used with permission – Microsoft stock images, 2025

Figure 1.5. Potential contamination introduction routes in a produce environment. Growing fields face threats of pathogen introduction through wildlife, neighboring agriculture, irrigation water, and soil. Arrows represent examples of potential introduction (blue) and amplification (yellow) mechanisms from these sources to a growing environment.

led to more robust treatment standards for soil amendments and the time required between application and planting (FSMA Final Rule on Produce Safety, 2024).

Another route of *Salmonella* introduction to produce is following the application of contaminated irrigation water. A recent outbreak in cucumbers, which caused 551 illnesses and 155 hospitalizations, was attributed to serovars Braenderup and Africana and linked to the use of contaminated irrigation water from a canal (CDC, 2024c). Studies have shown that irrigation water can act as a reservoir for *Salmonella* and other foodborne pathogens and can be transmitted onto produce during growth (M. Cooley et al., 2007; Gorski et al., 2011). Click or tap here to enter text.Surface water is the most common source for irrigation, but also introduces the highest food safety risk, as upstream contamination can occur from wildlife, agricultural runoff, or sewage leaks (Johannessen et al., 2015). These risks can be managed using proper treatment methods including combinations of chlorination, peracetic acid, and UV radiation that are effective in reducing bacterial load in irrigation water (Krishnan et al., 2021). Municipal and well water pose a lower risk of contamination intrusion; however, application with these water sources as well as treated surface water, can collect contaminants in the soil and spread them to root vegetables (Islam et al., 2005).

Wildlife intrusion presents yet another source of produce contamination. These events can include direct contamination via defecation during foraging or indirect contamination via deposition into irrigation water or neighboring soil (Gruszynski et al., 2014a; Langholz & Jay-Russell, 2013; Topalcengiz et al., 2020). Generally, mitigating wildlife intrusion can be accomplished using barriers to physically restrict access (e.g., netting, spikes, and fences) or deterrents (e.g., decoys, sonic devices, and reflective

surfaces) (Franklin & VerCauteren, 2016). These management efforts, however, are not always feasible for large farms, as they can be quite expensive and require regular upkeep to be effective (Jung et al., 2014). Controlling wild bird intrusion is particularly challenging, as bird-specific mitigation can be costly and ineffective if not used properly. Strategies such as deterrents lose efficacy quickly and need to be performed in rotation to be successful. Additionally, physical restriction and deterrents may not be effective for all species, requiring a combination approach utilizing multiple mitigation strategies that can become overwhelming for growers (Rivadeneira et al., 2018; Varriano et al., 2025).

Food safety regulation in produce

The Food Safety Modernization Act (FSMA) was passed in 2011 and contained the most sweeping reform of US food safety laws in over 70 years (FDA, 2011). This act included the following seven primary rules to regulate food systems: The Produce Safety Rule, Preventative Controls for Human Food, Preventative Controls for Animal Food, Foreign Supplier Verification Program, Accreditation of Third-Party Auditors/Certification Bodies, Sanitary Transportation of Human and Animal Food, and Prevention of Intentional Contamination/Adulteration. The Produce Safety Rule sought to reduce the risk associated with produce contamination during growing, harvesting, and packing, where *Salmonella* is most likely to come into contact with produce (Produce Safety Rule, 2015). The rule establishes science-based minimum standards for the safe growing, harvesting, packing, and holding of produce (Produce Safety Rule, 2015). The combination of these regulations, when correctly applied, can significantly reduce the risk of *Salmonella* contamination of produce; however, contamination can still occur.

Within the Produce Safety Rule, specific requirements for agricultural water, biologically amended soils, and domestic and wild animal contamination aim to reduce the introduction of *Salmonella* and other foodborne pathogens (Produce Safety Rule, 2015). The final rule on pre-harvest agricultural water seeks to encourage growers to assess points of risk within their field and manage risks accordingly. These points include water quality, crop characteristics, environmental conditions, neighboring land use, and other relevant factors (Figure 1.6). Growers can then utilize appropriate measures such as water and soil testing to reduce contamination risk. The use of biologically amended soils is supported under the produce safety rule; however, standards are set to prevent contamination of produce. These include strict limits on detectable amounts of certain bacteria such as *Salmonella*, *Listeria*, fecal coliforms, and *E. coli*. Additionally, a minimum period of 120 days between the placement of amended soil and harvesting of produce is required to allow for bacterial die-off. If the produce has no contact with the soil, this period is shortened to 90 days. Finally, reasonable measures must be taken to identify produce contaminated from wild animal feces and avoid harvesting; however, no animal exclusion mandates are in effect. Reasonable measures can include marking contamination with a flag, where some certification agencies such as the Good Agricultural Practices (GAPs) require mandatory no-harvest zones around these contamination markers.

These regulations are based on a well-established analysis of hazards within the produce environment. Hazards are defined as any biological, chemical, or physical property that may cause a food to be unsafe for human consumption, such as contaminated water, improperly treated soil amendments, or wildlife intrusion in produce

Evaluation of on-farm risk factors:
Water use practices
Crop characteristics
Environmental conditions
Proximal land use
Water not safe or sanitary for intended use?
Yes
No
Discontinue use and take corrective measures
Neighboring land or amended soil present a hazard?
Yes
No
Implement mitigation measures within the same growing season
Other conditions likely to introduce hazards
Yes
No
Implement mitigation measures within the following year
Continue regular maintenance of water systems
Water testing to determine needed measures

Figure 1.6. Agricultural water assessment and risk-based outcomes. Growers are responsible for following this risk assessment of water sources, deciding what hazards are unique to their farm; and making the best decision for their situation. Information adapted from the Produce Safety Rule (FDA, 2015).

fields (Pathogen Reduction; Hazard Analysis and Critical Control Point (HACCP) Systems; Final Rule, 1996). Significant work has been completed to characterize these hazards, but more work is needed to translate this into risk analysis. A risk is the likelihood and consequences of hazards contributing to unwanted outcomes. For example, *Salmonella* in contaminated irrigation water is considered a hazard; however, the possibility of irrigation water contamination by agricultural runoff or ineffective treatment of contaminated water would be considered a risk. Risk assessment in a produce environment requires extensive analysis of the literature corresponding to unique farm features that affect a grower such as neighboring land use, sanitation practices, and other relevant influencing factors.

1.3 *Salmonella* in wild birds

While *Salmonella* is best characterized in humans and food animals, wild animals such as wild birds have been understudied (Franklin & VerCauteren, 2016; Hernandez et al., 2012; Navarro-Gonzalez et al., 2020; O. M. Smith, Edworthy, et al., 2020). The term "wild birds" refers to all non-domesticated avian species, excluding domesticated poultry and pet birds. Wild birds can be classified taxonomically, and can also be grouped by their social behaviors, such as songbirds (passerines), waterbirds (ducks, cranes, etc.), and larids (gulls, terns, etc.) (Prum et al., 2015). By grouping species into these categories, social behavior including foraging and nesting can provide insight into their risk to human health concerning *Salmonella*. While many birds are asymptomatic carriers of *Salmonella,* songbirds are the most studied group due to their susceptibility to increased morbidity caused by salmonellosis (Alley et al., 2002; Hall & Saito, 2008; Hernandez et al., 2012; Tizard, 2004). Common songbirds include finches, sparrows, and

woodpeckers, where these birds generally feed on insects, fruits, and seeds. As social species, they will also frequent bird feeders and common gathering spaces. This social nature has contributed to the high transmissibility of salmonellosis within their populations and also to incidences of transmission to humans (M. H. Murray et al., 2021; Patel et al., 2023).

A single bird can be colonized via the consumption of contaminated food or water, then proceed to transmit the bacteria to other individuals in a flock. Waterbirds are particularly high risk for *Salmonella* as a significant portion of their activity is in or near surface water, where *Salmonella* can be readily found (M. H. Murray et al., 2021). Surface water is defined as any accessible water source above ground, including freshwater sources such as lakes, rivers, and aquifers, but also non-freshwater sources like brackish water and oceans. Ponds and lakes where waterbirds are commonly found tend to be collection points for human and animal waste runoff (Gorski et al., 2022; Murphy et al., 2022; Rodrigues et al., 2019; Weller et al., 2020); therefore, it is unsurprising that waterbirds have been shown to have increased prevalence of *Salmonella* (Hernandez et al., 2016; Krawiec et al., 2015; Tardone et al., 2020). Larid species are commonly found along shorelines, where a natural diet consists of a variety of crustaceans, insects, and fish, but will also include scavenging on animal carcasses (Andersson, 1970). This scavenging behavior has led to significant overlap in habitat with humans, where species such as seagulls will often forage from waste facilities (Olsen & Larsson, 2004; Ramos et al., 2010) leading to increased *Salmonella* prevalence (Antilles et al., 2021).

Other groupings of birds such as birds of prey (falcons, owls, etc.) and Pelecaniformes (pelicans, ibises, etc.) have been studied as well, where predatory species are estimated to have high foodborne pathogen prevalence due to the consumption of contaminated rodents or birds (Tizard, 2004). As urban populations grow, more species are driven from native environments, where interactions with humans have led to increased *Salmonella* prevalence and food safety risks, such as in the case of the white ibis (Hernandez et al., 2016). Alternatively, migratory species such as sandhill cranes have a range of approximately 5,000 miles, where dissemination of clinically relevant bacteria is of higher concern during this migration period (Callaway et al., 2014; Elsohaby et al., 2021). *Salmonella* is most often explored in its ability to spread directly from birds to humans such as in the 2021 outbreak or to cause explosive disease within wild bird populations, as described in the mass mortality of songbirds in the US in 2009 (Hernandez et al., 2012; Patel et al., 2023).

Salmonellosis and adaptation of *Salmonella* in avian hosts

Salmonella does not commonly cause illness in birds but rather is carried as a commensal organism in the intestinal tract (Janecko et al., 2015; Silva et al., 2018). There are, however, significant exceptions. (Cohen et al., 2021; Fukui et al., 2014; Hernandez et al., 2012; Mather et al., 2016; Patel et al., 2023; Refsum et al., 2003). In poultry, *Salmonella* serovars Gallinarum and its biovar Pullorum are associated with fowl typhoid and Pullorum disease, respectively, where both are largely fatal (Bullis, 1977). Genomic alterations including those responsible for the expression of SPI-1 and metabolism have reduced the ability of these pathogens to colonize non-poultry hosts (Langridge et al., 2015) Due to the host-specific nature of these serovars and their ability to cause high

levels of mortality, testing and culling programs have successfully eliminated them from the commercial poultry industry in the United States (Bullis, 1977; Kabir, 2010).

Sporadic cases of *Salmonella* epidemics in wild birds do occur, where serovar Typhimurium is generally the causative agent (Cohen et al., 2021). While no system is in place to regularly monitor *Salmonella* presence and diversity in wild birds, research groups and diagnostic laboratories have identified *Salmonella* in deceased birds (Hall & Saito, 2008; Hernandez et al., 2012). Avian *Salmonellosis* is often expressed through neurological dysfunction, ruffled feathers, and esophageal lesions that make swallowing difficult and lead to weight loss (Friend et al., 2015). Outbreaks are often widespread and occur in winter months due to increased reliance on feeders and heightened transmission during migration (Refsum et al., 2003). A recent outbreak was particularly interesting as it involved avian mortality and human illness (Patel et al., 2023). At least 30 people contracted salmonellosis due to contact with dead birds or contaminated feeders, supporting the need for improved sanitation practices in supplemental bird feeding. By focusing studies on avian salmonellosis, the knowledge of *Salmonella* in wild birds is biased toward only the most invasive strains in birds. This may not be an accurate reflection of strains present across wild bird species including those that could have a more significant impact on human health. For effective risk assessment within wild birds, more information about the prevalence and nature of *Salmonella* in this host is needed.

Avian host adaptations are well documented in serovar Typhimurium, where the definitive phage types (DTs) 40, 56v, and 160 have become widespread within migratory birds and have led to outbreaks of explosive mortality in waterfowl, songbirds, and other species (Alley et al., 2002; Cohen et al., 2021; Fu et al., 2022; Fukui et al., 2014;

Hernandez et al., 2012; Patel et al., 2023; Refsum et al., 2003). These phage types often have distinct genomic lineages that include a loss of the pSLT virulence plasmid (Mather et al., 2016). A second example of this is a newly identified serovar, Tirat-Zvi, which excels in the colonization of passerine species while also showing reduced virulence in mice (Cohen et al., 2024). Additionally, these host-adapted strains are also found to have an accumulation of pseudogenes, or alterations that cause translated proteins to no longer function normally, which often result in the loss of function for virulence factors (Cohen et al., 2021). The loss of these genes that facilitate general colonization and the retention of genes specific to the colonization of avian species has led to significant differences in virulence between wild birds and other hosts (Cohen et al., 2021). This process of transitioning from a host-generalist to a host-specialist by the formation of pseudogenes and gene inactivation is known as genome decay (Baumler & Fang, 2013; Langridge et al., 2015). While specific genes allowing for the increased virulence of these strains have not been identified, the reduction of fitness within non-target hosts is hypothesized to increase fitness within the specific host.

Wild birds provide a unique *Salmonella* risk as they can cover large distances and do not face the same barriers to movement as many other wildlife (Elsohaby et al., 2021; Rivadeneira et al., 2018). This likely facilitates the accumulation and spread of *Salmonella* in birds from a variety of sources and geographic locations, making them a particularly interesting subject of study. To further investigate avian *Salmonella* isolates, Enterobase is a useful database containing genomes from a variety of human, animal, and environmental sources from around the world (Zhou et al., 2020). This database currently contains 2,179 isolates of *Salmonella* that have been associated with wild avian sources.

Of these, 1079 are serovar Typhimurium. This is unsurprising as this generalist serovar is frequently identified in many different hosts, including wild birds (Alley et al., 2002; Cohen et al., 2021; Fukui et al., 2014). Additionally, 154 isolates of serovar Enteritidis and 134 isolates of serovar Infantis are present that are linked to wild birds. Notably, these three serovars that are frequently associated with avian hosts also account for 33% of human clinical *Salmonella* isolates in the United States, suggesting a potential association between these hosts (Figure 1.1).

Salmonella prevalence in wild birds

In the United States, regional testing as part of smaller research studies has shown that *Salmonella* prevalence in birds ranges from 0.5% to 2.5% on the West Coast and 1.9% in the Southwest (Gorski et al., 2011; Jay-Russell & Justice-Allen, 2014; Navarro-Gonzalez et al., 2020; Rivadeneira et al., 2016; O. M. Smith, Edworthy, et al., 2020; O. M. Smith, Olimpi, Navarro-Gonzalez, et al., 2022). Studies outside of the United States, including those in Europe, South America, the Middle East, and Eastern Asia regularly find similar levels of *Salmonella* prevalence in wild birds (Cardoso et al., 2021; Cohen et al., 2021; Lawson et al., 2011; Palmgren et al., 2006). As urbanization and industrialization reduce the natural habitat for wild birds, it is unsurprising that interactions between humans in urban populations or animal agriculture increase. Many of these studies have identified human-associated serovars carried in wild birds, including those more commonly identified in human infections (Hernandez et al., 2016; O. M. Smith, Edworthy, et al., 2020). Wild birds interacting with human populations can become colonized with *Salmonella* via foraging in garbage and refuse (i.e. pigeons, crows, ibises) (Hernandez et al., 2016; Ramos et al., 2010), while those interacting with

animal agriculture (i.e. starlings, blackbirds, and sparrows) can be recipients of transmission from feed, feces, or irrigation water in these settings (Kirk et al., 2002; O. M. Smith, Olimpi, & Karp, 2022).

Public health risks attributed to wild birds

Wild birds can transmit *Salmonella* to humans directly and indirectly. Recently, a 2021 outbreak in humans was attributed to wild birds, where human interactions with bird feeders and deceased birds lead to illness (Patel et al., 2023). In 2008, the CDC reported an outbreak of *Salmonella* Typhimurium linked to peanuts (Wittenberger & Dohlman, 2010). Source tracking later identified that this strain was found in the peanut fields and also in wild birds that were sampled around the fields (Hernandez et al., 2012). Although wild birds were not directly implicated in this outbreak, closely related strains found in birds support the hypothesis that feces from the wild birds contaminated the peanut products. This form of contamination has also been linked to other pathogens, including the 2004 outbreak of *Campylobacter jejuni* that was caused by feces from sandhill cranes on pea plants in Alaska; however, this case involved many unique factors such as the consumption of raw, uncleaned product at a local level and the farm location being near a large wildlife refuge (Gardner et al., 2011). Previous work has investigated factors that affect the survival and transmissibility of *Salmonella* in feces at these foraging sites, finding that recovery can occur for weeks after inoculation (Oni et al., 2015; Topalcengiz et al., 2020). Within animal agriculture environments, wild birds can also indirectly impact human health. Some species such as European starlings, brown-

headed cowbirds, and Brewer's blackbirds are categorized as pest species within concentrated animal feeding operations (CAFOs) where they will forage on the animal feed and fecal material from livestock (Palmer, 1976). Here, *Salmonella* and other foodborne pathogens can be transmitted from birds to livestock, contributing to the pathogen burden in these food production operations (Carlson et al., 2011, 2020).

In the US, wild birds have been identified as a risk for *Salmonella* contamination in regions dominated by monoculture crop production and animal agriculture (O. M. Smith, Edworthy, et al., 2020). Studies around produce fields have identified a range of prevalence, from 0.5% in west coast studies (Gorski et al., 2011; Navarro-Gonzalez et al., 2020; O. M. Smith, Edworthy, et al., 2020) and 1.9% in a southwest study (Rivadeneira et al., 2016). FSMA and the Produce Safety Rule, while urging growers to manage wildlife contamination, do not set guidelines on how to prevent intrusions, which adds to the challenges growers face in trying to control wildlife. The efficacy of wild bird mitigation strategies such as decoys, lasers, air cannons, and falconry have been debated, leading to conflicting measures to reduce bird intrusion (Rivadeneira et al., 2018; Varriano et al., 2025). Conflict is also demonstrated in the debate over the protection or removal of riparian zones (Karp et al., 2015; Strawn et al., 2013). These strips of non-crop vegetation along the perimeter of produce fields are thought to encourage the foraging of wild birds and insects; however, others argue that any encouragement of wildlife near fields increases risk. Considering the relatively low prevalence of *Salmonella* within wild birds from previous studies, the financial burden of mitigation and the potential impact of ecological disruption have recently been questioned (Olimpi et al., 2022, 2024; O. M. Smith, Snyder, et al., 2020) calling for co-management practices

instead. This lack of confidence in risk assessment and mitigation strategies produces a food safety question; what role do wild birds have in pathogen transmission to fresh produce? To answer this question, it is first necessary to identify what is known about the potential for avian *Salmonella* transmission in produce fields.

1.4 Surface water

For produce growers using surface water, a combination of microbial testing and mitigation strategies ensures that only water within a safe range is used (FDA, 2011). Currently, there are no tests available that can directly and efficiently test for *Salmonella* presence. Most tests target a combination of total coliforms, fecal coliforms, and generic *E. coli* within water. These organisms are associated with fecal contamination and are used because they can serve as indicators for enteric pathogens such as *Salmonella.* However, previous work has shown an inconsistent relationship between these indicator species and *Salmonella* (Gu et al., 2021; McEgan et al., 2013; Weller et al., 2020). Although these regulations are in place to prevent the use of unsafe water, *Salmonella* outbreaks in produce continue to implicate contamination in irrigation sources (CDC, 2024c; FDA, 2021). This highlights a critical need to understand *Salmonella* in surface water, which has been well-studied (Gorski et al., 2022; Gu et al., 2019; Hintz et al., 2010; Li et al., 2015; Micallef et al., 2012).

Salmonella **introduction and survival in surface water**

Salmonella is an enteric pathogen, suggesting that aquatic environments would be inhospitable; however, multiple domestic and international studies have shown high *Salmonella* prevalence in water (Chen et al., 2024; Deaven et al., 2021; Haley et al., 2009; H. Liu et al., 2018; Murphy et al., 2022; Toro et al., 2022). *Salmonella* can be

introduced to surface water via direct or indirect contamination by humans and animals (Casanova et al., 2020; Li et al., 2015; Topalcengiz et al., 2019). When wildlife or livestock have direct contact with water, defecation is likely to occur. Human sewage is also often directed to rivers, providing an influx of new pathogens while also providing fresh nutrients that facilitate pathogen proliferation (Olds et al., 2018). The treatment of this sewage greatly reduces its microbial impact; however, studies have identified *Salmonella* following this treatment as it returns to surface water (Kinde et al., 1997; Odjadjare & Olaniran, 2015). Wastewater conveyance, or the carriage of sewage to the treatment plants which often occurs alongside waterways, can also experience leakes that introduce pathogens (Roehrdanz et al., 2017). Alternatively, indirect contamination often takes the form of agricultural runoff (Gorski et al., 2011). For example, precipitation events can push fecal material from animal agriculture fields downhill to the lowest areas where it will eventually find a surface water source. Surface water can be naturally formed such as creeks or ponds, or can be manmade for fecal runoff of cattle feed yards or dairies, such as lagoons. Presumably, runoff from non-agricultural land can also wash fecal material deposited by wildlife into surface water. Where improperly treated biologically amended soils are used in plant agriculture, they, too, can be washed away following precipitation and irrigation (You et al., 2006). Barriers and treatments can be used to limit this runoff; however, such mitigation is often costly, which reduces the sustainability of these approaches.

In the water, *Salmonella* encounters a variety of stressors that it must overcome to survive. These include osmotic pressure, oxidative stress, pH, temperature, and radiation as well as a significantly diluted supply of essential nutrients (Winfield & Groisman,

2003). Some studies have suggested that *Salmonella* merely persists in the water in a metabolically inactive state or survives in a suboptimal state until re-entering a host (Roszak et al., 1984; Santo Domingo et al., 2000). Alternatively, *Salmonella* is capable of proliferating, either planktonically, or within biofilms or free-living protozoa (Brandl et al., 2005; Byappanahalli et al., 2009; Gaertner et al., 2011; Sha et al., 2013). Previous work has shown that viable *Salmonella* can be found in freshwater after 300 days without any added nutrients (Topalcengiz et al., 2019) and was detected five years after being inoculated into sterile water (Liao & Shollenberger, 2003). Others have observed a significant decrease in viability within days of inoculation into water (Oguadinma et al., 2022). This suggests that *Salmonella* could enter a viable but nonculturable (VBNC) state that allows it to persist for long periods.

Methods for *Salmonella* sampling from surface water

The two overarching methods for *Salmonella* sampling in water are Moore swabs and membrane filtration. Moore swabs are a piece of cheesecloth that is tied in the middle and placed in a sampling environment for 24-48 hours where free-floating *Salmonella* can become caught in the fabric (Moore, 1948; Sikorski & Levine, 2020). The swab is then collected and placed into culture media. As a more efficient method, modified Moore Swabs are collected on-site for 2-30 minutes. Here, a larger piece of cheesecloth is rolled tightly and placed inside a PVC cassette where between 0.1L to 10L of water is pumped through the cheesecloth filter (Bisha et al., 2011). This actively filters any *Salmonella* floating by the filter rather than relying on passive capture. The cheesecloth filter can then be removed and cultured, similar to a conventional Moore swab. Alternatively, membrane filtration traditionally begins by collecting a set volume of

water, generally between 100mL to 1L (Entis, 1990; Lindquist et al., 2007; H. Liu et al., 2018; McEgan et al., 2013). This sample is then filtered using vacuum filtration or adding in perlite "pool" filter into the sample (Meinersmann et al., 2008). Work completed by the CDC has also led to the development of a highly sensitive membrane filtration method known as dead-end ultrafiltration (DEUF) where up to 100L of water are pumped on-site through a small filter on-site before backflushing the filter into 100mL to 500mL of solution that can then be cultured (C. M. Smith & Hill, 2009).

These protocols have advantages and disadvantages that should be considered when choosing research methods. Moore swabs allow the researcher to examine the population of *Salmonella* in the water over time (Bisha et al., 2011; Cho et al., 2020). Additionally, modified Moore swabs can be utilized to analyze varying volumes of water, giving researchers control of the sample size. Prior work compared *Salmonella* recovery in 0.1L, 1L, and 10L using this method and found that 10L samples had 43.5 and 25.5 times better recovery than 1L and 0.1L, respectively (Sharma et al., 2020). Membrane filtration has the benefit of higher sensitivity compared to Moore swabs (Kraft et al., 2023), as samples from this collection method can be quantified due to the concentration of bacteria in a small filter. Additionally, the resulting backflush can be split into multiple growth conditions, allowing for the detection of various organisms from a single sample (Amin et al., 2020; P. Liu et al., 2012; McEgan et al., 2013). Alternatively, Moore swabs are advantageous due to their comparatively low cost and ease of use (P. Liu et al., 2021); however, this method requires researchers to return to the sampling site the following day, making this method less time-efficient than others (McEgan et al., 2013). These

advantages and disadvantages demonstrate the need to take careful consideration of collection methods when sampling water sources.

Salmonella prevalence in water

Salmonella prevalence in water has been well characterized in the United States, with studies taking place in major agricultural regions, including the Southeast, West Coast, Mid-Atlantic, and Northeast (Figure 1.7). The largest proportion of these studies have been completed in the Southeast, where prevalence ranges from 4.8% to 100% (Bell et al., 2015; Gu et al., 2019; Haley et al., 2009; Havelaar et al., 2017; Li et al., 2014; Luo et al., 2015; McEgan et al., 2013, 2014; Murphy et al., 2022, 2024; Rajabi et al., 2011; Vereen et al., 2013). Sampling in the Mid-Atlantic has resulted in a prevalence ranging from 4.2% to 65% (Acheamfour et al., 2024; Callahan et al., 2019; S. Kim et al., 2023; Kraft et al., 2023; Micallef et al., 2012; Sharma et al., 2020). A smaller number of studies in the Northeast, including those in New York and Pennsylvania, have found *Salmonella* prevalence ranging between 4.6 and 49% (Deaven et al., 2021; Strawn et al., 2013; Weller et al., 2020). West Coast studies have been concentrated in agricultural regions of California, where prevalence ranges from 6% to 65% (Benjamin et al., 2013; M. B. Cooley et al., 2014; Gorski et al., 2011, 2022; Partyka et al., 2018). Additionally, international sampling has included a wide range of global regions, where similarly variable *Salmonella* prevalence ranged from

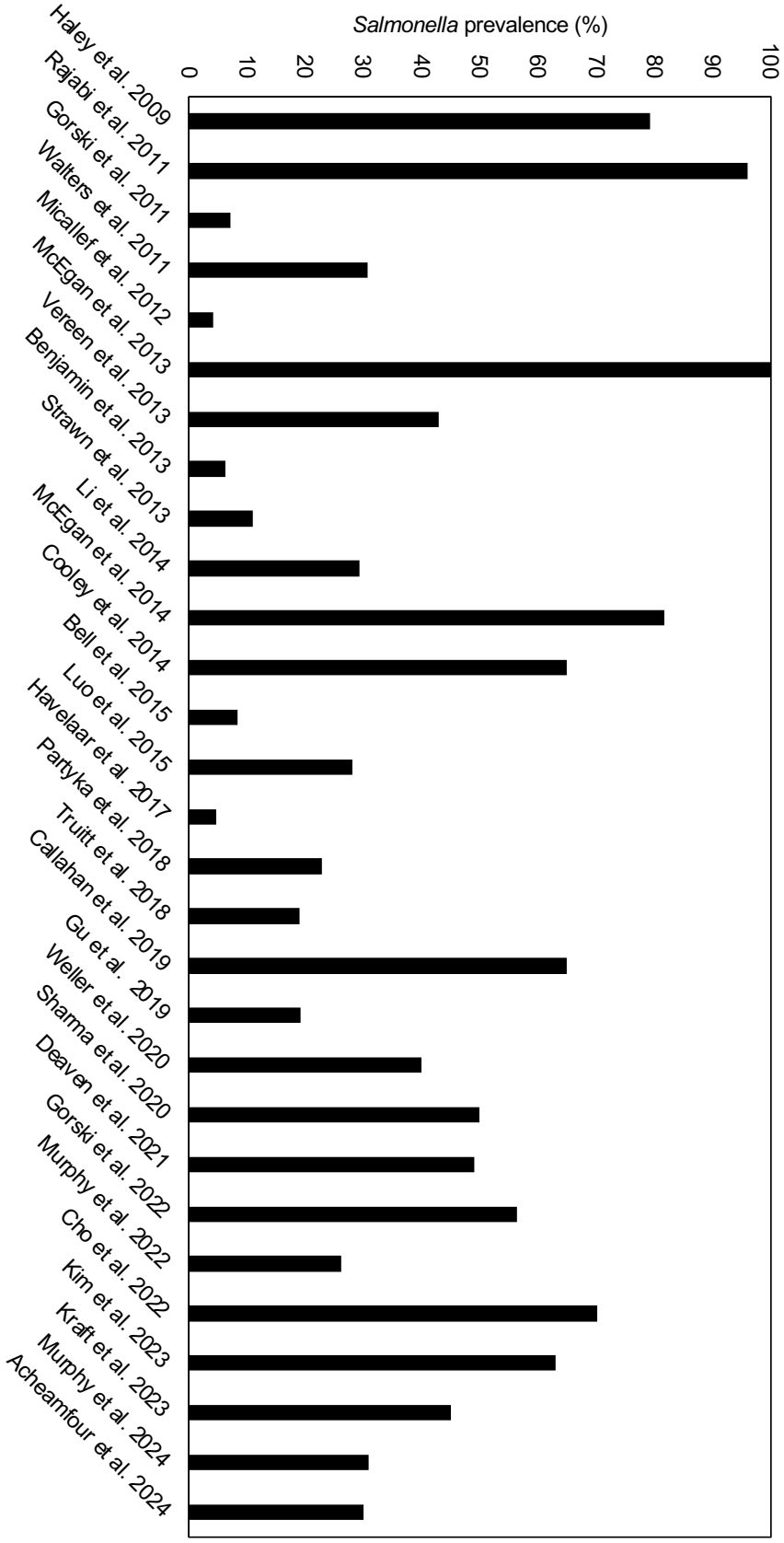
Salmonella prevalence (%)
0
10
20
30
40
50
60
70
80
90
100
Haley et al. 2009
Rajabi et al. 2011
Gorski et al. 2011
Walters et al. 2011
Micallef et al. 2012
McEgan et al. 2013
Vereen et al. 2013
Benjamin et al. 2013
Strawn et al. 2013
Li et al. 2014
McEgan et al. 2014
Cooley et al. 2014
Bell et al. 2015
Luo et al. 2015
Havelaar et al. 2017
Partyka et al. 2018
Truitt et al. 2018
Callahan et al. 2019
Gu et al. 2019
Weller et al. 2020
Shama et al. 2020
Deaven et al. 2021
Gorski et al. 2022
Murphy et al. 2022
Cho et al. 2022
Kim et al. 2023
Kraft et al. 2023
Murphy et al. 2024
Acheamfour et al. 2024

Figure 1.7. *Salmonella* prevalence in surface water studies in the United States. Chronological display of various studies within the United States that tested for *Salmonella* prevalence in surface water. Prevalence is measured in percentage and labeled with the first author and the year of study.

7.1% to 78.4% (Afema et al., 2016; Arvanitidou et al., 2005; Chen et al., 2024; Díaz-Torres et al., 2020; Ho et al., 2018; Huang et al., 2014; Mahagamage et al., 2020; Polo et al., 1998; Ruiz et al., 1987; Santiago et al., 2018; Setti et al., 2009; Song et al., 2018; Thomas et al., 2013; Toro et al., 2022). These studies show that *Salmonella* can be regularly identified in surface water; however, the prevalence can vary significantly.

In addition to regional differences, the type of water in these studies may influence *Salmonella* prevalence. Generally, water collected from moving sources such as rivers, creeks, and streams yielded *Salmonella* in more than 50% of samples for many studies (Cho et al., 2022; Haley et al., 2009; McEgan et al., 2013, 2014; Rajabi et al., 2011). Alternatively, studies that used stagnant water such as ponds or agricultural water reservoirs often contained *Salmonella* in less than 25% of samples (Gu et al., 2019; Havelaar et al., 2017; Micallef et al., 2012; Partyka et al., 2018; Strawn et al., 2013; Truitt et al., 2018). This suggests *Salmonella* may benefit from the circulation of nutrients or mixing of microbial communities in larger creeks and rivers.

Environmental factors influencing *Salmonella* potential in water

The presence of *Salmonella* in water can be influenced by meteorological factors, such as temperature, wind, and solar radiation, and also by physiochemical factors such as pH, turbidity, dissolved oxygen, and total dissolved solids (Weller et al., 2020). Prior work has identified positive correlations between *Salmonella* prevalence and higher temperatures, including both air and water temperatures (Deaven et al., 2021; Gorski et al., 2011; Haley et al., 2009; H. Liu et al., 2018; Murphy et al., 2022; Thomas et al., 2013; Weller et al., 2020). Additionally, the incidence of rainfall before sampling was often associated with increased *Salmonella* recovery (Deaven et al., 2021; Setti et al.,

2009; Strawn et al., 2013; Thomas et al., 2013; Weller et al., 2020). The association of *Salmonella* prevalence with the presence of indicator species such as *E. coli* was mixed, with some finding positive associations (Weller et al., 2020) and others finding negative correlations (McEgan et al., 2013). Alternatively, many studies found a lack of correlation between these same variables, such as rain (Goyal et al., 1977; McEgan et al., 2013; Santiago-Rodriguez et al., 2012), temperature (Strawn et al., 2013), and indicator species (McEgan et al., 2013; Murphy et al., 2022). Rarely, negative correlations were identified; however, these were not consistently found across studies, for example with conductivity (McEgan et al., 2013). Inconsistencies between these variables highlight the complexities associated with understanding which factors most greatly influence *Salmonella* in water. A better understanding of how these factors affect *Salmonella* introduction and viability in surface water is necessary for building risk assessment models. If these predictive tools could be reliably implemented, growers would be able to monitor water and weather conditions and make more informed decisions about safe water usage.

Global surface water research and improvement efforts

Salmonella cases worldwide are estimated as high as 176 million illnesses annually, where enteric fever from typhoidal serovars contributes to 26 million cases (CDC, 2024b). Notably, infections from typhoidal-serovars almost exclusively occur in Latin America, Sub-Saharan Africa, and Southeast Asia (IHME, 2021b, 2021a). Alternatively, the United States accounted for less than 500 culture-confirmed cases between 2016-2018, a majority of which were attributed to international travel (CDC,

2024b). Typhoidal infections often occur via contact with contaminated water, making surface water studies in these regions particularly important.

Antibiotics are regularly used for human and animal health around the world; however, their misuse has contributed to an increasing concern of AMR, becoming a threat to human health in the United States and internationally. AMR directly caused 1.27 million deaths in 2019 and contributed to 4.95 million deaths, making it a top concern of the World Health Organization (C. J. L. Murray et al., 2022; WHO, 2023). Antimicrobial-resistant *Salmonella* are regularly identified in surface water, with prevalence ranging from 2% to 100% (Berge et al., 2006; Casanova et al., 2020; Chen et al., 2024; Cho et al., 2022; Dolejská et al., 2009; Gorski et al., 2011; Jokinen et al., 2015; Li et al., 2014; Luo et al., 2015; McEgan et al., 2014; Meinersmann et al., 2008; Micallef et al., 2012; Nguyen et al., 2021; Patchanee et al., 2010; Somda et al., 2021; Suhartono et al., 2021). Varying levels of antibiotic stewardship exist across different geographic regions (Maron et al., 2013). Without proper use and regulation, antimicrobial residues can influence bacterial populations and the presence of AMR genes in the environment (Beattie et al., 2018; Carroll et al., 2015; Dolejská et al., 2009; Laborda et al., 2022; Ramey & Ahlstrom, 2020). Surface waters have been known to act as a reservoir for antimicrobials, which can facilitate transmission to the environment and genetic transfer between microbes (Chen et al., 2024; Wilkinson et al., 2022).

Due to the increasing threat of enteric fever and antimicrobial resistance, global studies focusing on *Salmonella* in surface water are needed to better understand transmission and introduction sources. To address this, a team of researchers from Latin America, in a coordinated effort with the Joint Institute for Food Safety and Applied

Nutrition (JIFSAN), have been collecting water samples beginning in 2019. The overall prevalence in these three studies was 45.4%, where teams in Brazil, Chile, and Mexico found individual prevalence of 68.2%, 33.1%, and 62.9%, respectively (Chen et al., 2024). In these studies, antimicrobial resistance was prominent, where 33.8% of all isolates were resistant to at least one antimicrobial. (Chen et al., 2024). In Chile, *Salmonella* prevalence was identified at 28.1% between two major agricultural regions where crop presence was the most significant factor in predicting pathogen presence (Toro et al., 2022). Similar to studies in the United States (Deaven et al., 2021; Gorski et al., 2011; Haley et al., 2009; Murphy et al., 2022), this study found that seasonality was a driving factor for prevalence. While much of the research from this joint effort is forthcoming, the data made available from these studies thus far shows the need to continue surveillance of agricultural water internationally as a means of reflecting the food safety risks associated with contaminated water. This research and others around the world continue to improve our understanding of *Salmonella* distribution, diversity, and influencing factors. While not specific to *Salmonella*, the assessment of implemented measures to improve water quality found that conditions remained consistent, rather than degrading, following large urbanization (Johannessen et al., 2015). This demonstrates the need for improved management strategies as well as the benefit of targeted water quality regulations. Additionally, work completed in Mexico supported previous findings of high *Salmonella* levels in summer months (Estrada-Acosta et al., 2018; González-López et al., 2022). This work also identified a strain of serovar Oranienburg over multiple months, suggesting potential adaptations for persistence. Dynamics involved in the persistence

and transmission of *Salmonella* have not been well characterized prior to this work, highlighting the importance of elucidating these pathways.

Global urbanization, waste management practices, and improper use of waterways contribute to contamination of surface water across the (Tickner et al., 2017). This use of improperly managed water contributes to the estimated 93.8 million global cases of gastroenteritis and 59,100 deaths annually caused by *Salmonella* species (Majowicz et al., 2010; Roth et al., 2018). In their 2023 sustainable development goals report, the United Nations (UN) stated that 2.2 billion people around the world lacked access to safely managed drinking water. Because of this, the UN has listed access to clean water and sanitation as one of their 17 major goals to meet before 2030, indicating the importance of this natural resource to people around the world (Burden of Disease Attributable to Unsafe Drinking-Water, Sanitation and Hygiene, n.d.). This global effort to improve availability and access to safe water will rely on international research efforts to provide data used to meet these goals.

1.5 Methods of *Salmonella* isolation

Traditional *Salmonella* isolation includes four steps: 1) pre-enrichment, 2) selective enrichment, 3) indicator plating, and 4) confirmation (Figure 1.8). Depending on the sample type being tested, the procedure may vary between protocols following the Bacterial Analytical Manual (BAM) method (FDA, 2024a) or Microbiology Laboratory Guidebook (MLG) (USDA-FSIS, 2024b). In each protocol, these steps seek to promote *Salmonella* growth by inhibiting competing microbes while developing an environment suitable for *Salmonella* growth. The BAM is followed by the FDA for produce and environmental sampling, while MLG is followed by the USDA-FSIS for the culture of

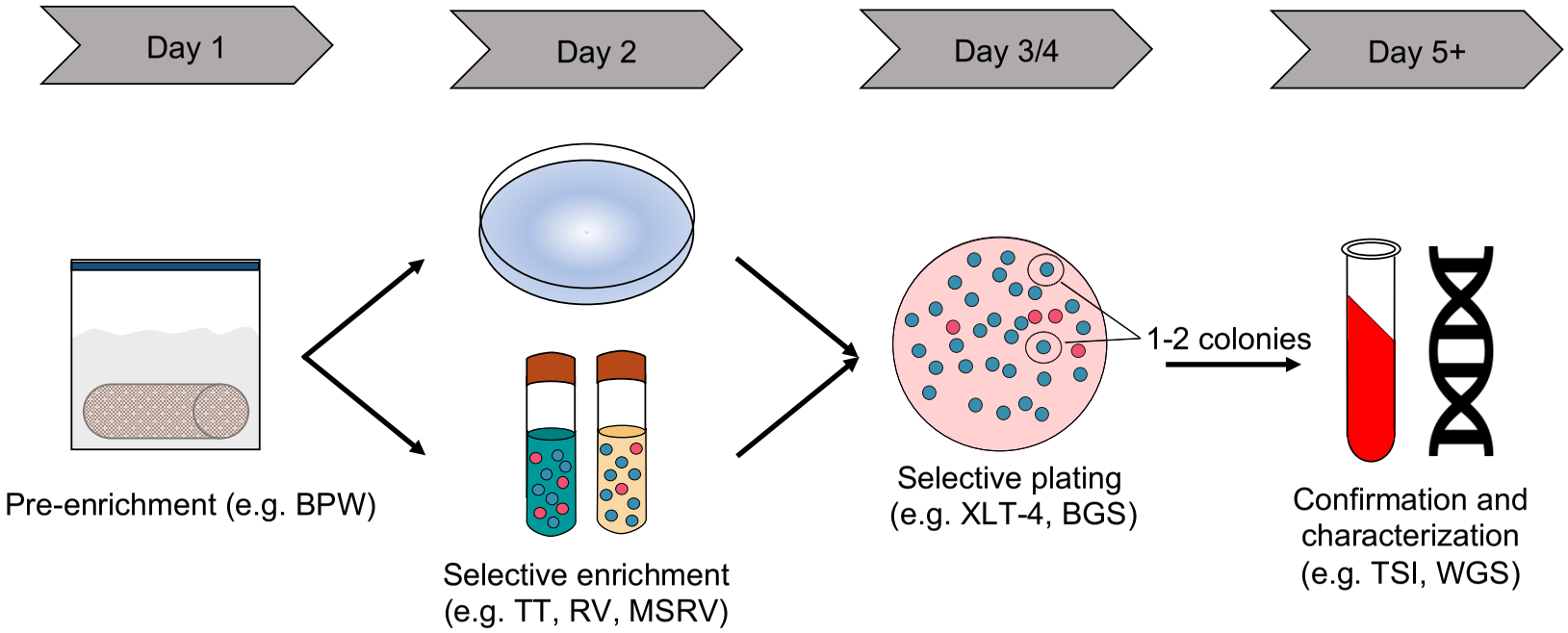
Day 1
Day 2
Day 3/4
Day 5+
Pre-enrichment (e.g. BPW)
Selective enrichment
(e.g. TT, RV, MSRV)
Selective plating
(e.g. XLT-4, BGS)
1-2 colonies
Confirmation and
characterization
(e.g. TSI, WGS)

Figure 1.8. *Salmonella* isolation flow diagram. The general flow of *Salmonella* isolation begins with a pre-enrichment, followed by selective enrichment and selective plating. Confirmation of presumptive positive isolates concludes isolation.

meat, poultry, and pasteurized egg products. Chemical and physical differences in sample composition, such as pH, water activity, and fat content can drastically affect the recovery of *Salmonella* due to potential interactions with media or other microbial growth (Gibson et al., 1988). Additionally, recovery can be influenced by growth media, selecting for serovars better adapted to the conditions in one media but selecting against serovars without those adaptations (Gorski, 2012), Therefore, ensuring that the right protocol for a given matrix is key to effective isolation of *Salmonella*.

A non-selective pre-enrichment or "recovery" medium is often used as the first step of *Salmonella* isolation, as cells recovered from environmental sampling are often damaged and unable to compete (Budu-Amoako et al., 1992). Buffered peptone water (BPW) is often used for this step, as it contains peptones as a nutrient source and sodium chloride for electrolytes, but no selective agents. In samples from produce or food animal processing where antimicrobials are used, neutralizing agents are added to the recovery medium to prevent the continuation of antimicrobial properties from further injuring *Salmonella*. These neutralizing media including Dey-Engley (DE) broth used during FDA for environmental sampling during outbreak investigations and neutralizing BPW (nBPW) used by the USDA-FSIS, to prevent the continuation of antimicrobial properties from further injuring *Salmonella* (FDA, 2024b; Gamble et al., 2017). Samples are incubated between 37°C and 42°C for up to 24 hours to allow for the recovery of sub-lethally injured cells. As this medium is non-selective, background and competing microbes will also grow and potentially outcompete *Salmonella*, therefore an additional step is necessary to isolate *Salmonella* from the non-target bacteria (Busse, 1995; Rasamsetti et al., 2021).

Following pre-enrichment, samples are then transferred into one or more selective enrichment media. The two most common media used for *Salmonella* isolation are tetrathionate (TT) and Rappaport-Vassiliadis (RV) broths. TT broth takes advantage of a metabolic pathway present in many *Salmonella* serovars but missing in other species (Winter et al., 2010). This pathway allows *Salmonella* to metabolize tetrathionate as an energy source while providing no additional energy source for other species, preventing the growth of competing bacteria (D'aoust, 1981; Knox, 1945). Additionally, bile salts are used to inhibit gram-positive bacterial growth along with the presence of calcium chloride to balance pH, as sulfuric acid is produced by metabolic activity (W. Moats, 1981; Winter et al., 2010). An alternative TT formulation, TT Hajna, includes further selective ingredients including brilliant green and sodium desoxycholate, both of which further inhibit the growth of gram-positive bacteria (Hajna & Damon, 1956). RV broth contains a combination of low pH, malachite green, and magnesium chloride. *Salmonella* survives well at lower pH, while malachite green inhibits the growth of coliforms that are often identified in environments with *Salmonella* (Rappaport et al., 1956; Vassiliadis, 1983; Vassiliadis et al., 1981). Magnesium chloride acts to increase the osmotic pressure, creating a hypertonic solution that gram-negative organisms can tolerate, but gram-positive organisms cannot (Peterz et al., 1989). While both media are acceptable for recovery of *Salmonella,* TT medium has improved recovery when low microbial loads are present (Hammack et al., 1999). RV medium can often be altered depending on sample type, including the addition of novobiocin to further reduce gram-positives, or the addition of agar to form a semi-solid medium that retains selectivity while also allowing for the separation of motile and non-motile organisms (Aspinall et al., 1992; KOMATSU

& RESTAINO, 1981). After inoculation into selective enrichment media, cultures are incubated for an additional 24 hours.

Following enrichment, samples are then streaked onto an indicator plate that continues to selectively enrich for *Salmonella* while utilizing a visual change in the color of the colony or plate to identify presumptive-positive *Salmonella* colonies. Xylose Lysine Tergitol-4 (XLD-4), Brilliant Green Sulfate (BGS), and Xylose Lysine Deoxycholate (XLD) agar are commonly used. Both XLD and XLT-4 agar utilize a trait in most *Salmonella* serovars, the ability to produce hydrogen sulfide (H_2S) during fermentation, to turn H_2S (+) colonies black while leaving H_2S (-) colonies white (Taylor, 1965). XLT-4 utilizes tergitol, a surfactant that is bacteriostatic against gram-positive bacteria and *Proteus* species (Miller et al., 1991). XLD does not utilize tergitol; however, novobiocin is often added at concentrations ranging from 5ug/mL to 80ug/mL to reduce gram-positive bacteria (Restaino et al., 1982). BGS agar utilizes brilliant green to reduce gram-positive bacteria and the inability of *Salmonella* to ferment lactose by introducing phenol red to turn fermenting bacteria yellow, leaving non-fermenting bacteria pinkish red (W. A. Moats & Kinner, 1974). Finally, confirmation of *Salmonella* is needed after indicator plating to mitigate false positives from visually similar colonies such as surviving *Proteus* species on XLD or *Citrobacter* species on XLT-4. These confirmation assays can include biochemical tests with triple sugar iron slants (Krumwiede & Kohn, 1917), physical reactions with serum agglutination assays (Olopoenia & King, 2000), or molecular confirmation using polymerase chain reaction (PCR) for the *invA* gene (Rahn et al., 1992).

Variations of the culture methods described above include delayed secondary enrichment (DSE) and consecutive selective enrichment. DSE is the process of inoculating pre-enrichment culture into TT broth, the medium is left at room temperature for seven days, where the culture is then transferred to fresh TT and incubated for 24 hours (Waltman et al., 1991). Consecutive selective enrichment consists of pre-enrichment culture inoculation into TT broth and, after 24 hours of incubation, passaging this culture into RV broth for an additional 24 hours before plating (Rigby & Pettit, 1980). Additionally, alternative selective media have been used for the growth of *Salmonella*, including selenite broth and its variable formulations which utilize sodium selenite to inhibit the growth of gram positives and some gram negatives, while permitting *Salmonella* growth (Zimboro et al., 2009). A semisolid agar formulation of RV medium is also commonly used to maintain the same selective pressure as traditional RV while further selecting for motility from the inoculation site (Aspinall et al., 1992).

1.6 Characterizing *Salmonella* isolates

Following *Salmonella* confirmation, the next step in characterization includes serotyping to identify the serovar, which is then followed by subtyping to provide more granular strain information.

Serotyping

Traditional serotyping is performed via serum agglutination (Wattiau et al., 2011). This process utilizes the antigenic structure of *Salmonella* described in the White-Kauffman-Le minor scheme (P. A. D. Grimont & Weill, 2007). A complete serotyping analysis requires the possession of antisera for 46 somatic, 119 flagellar, and the capsular antigen (B. Liu et al., 2014; McQuiston et al., 2004). For each isolate, a separate test is

conducted for the somatic antigen along with testing for both H1 and H2 flagellar antigens, which can exist due to the ability of *Salmonella* to phase switch between the expression the *fliC* and *fljB* genes, and finally testing for the presence or absence of the capsular Vi antigen (P. A. D. Grimont & Weill, 2007; Silverman et al., 1979). Due to the cost associated with obtaining all necessary antisera and the difficulty in applying these tests for multiple isolates, this analysis is often conducted by specialty diagnostic laboratories. Alternatively, the identification of the O-antigen alone, referred to as serogrouping, can be used to gather general information from isolates and can be a useful research tool for screening isolates.

Alternative serotyping methods include molecular approaches: one of the first methods used was the amplification of abequose (*rfbJ*) and paratose synthase genes (*rfbS*) of common *Salmonella* O-groups (A, B, C2, and D). The presence of these group-specific sequences can be used to identify the serotype of an isolate (Luk et al., 1993). Additional work found group-specific sequences within the *rfb* gene cluster for groups E (Wang et al., 1992), H (Fitzgerald et al., 2003), O:35 (Wang & Reeves, 2000), and O:54 (Keenleyside et al., 1994). Due to the variability of the *rfb* gene cluster, a multiplex PCR was created to differentiate the presence of common serogroups (B, C1, C2, D, E, and O:13) using a Luminex platform (Dunbar et al., 2003; Fitzgerald et al., 2007). Further inclusion of group-specific and flagellar genes led to the development of a *Salmonella* multiplex assay for rapid typing (SMART) analysis to serogroup the 50 most common serovars in the United States (Leader et al., 2009). These methods utilize variability within surface antigen genes to separate serotypes; however, this can be accomplished

through other methods as well and many subtyping approaches described below can also accurately indicate serovar identity.

Non-antigenic molecular serotyping methods include insertion sequences, ribosomal variation, and other genomic targets. Insertion sequences (IS), primarily IS200, were identified as a prominent trait within a majority of human clinical isolates, where mutational variation within this sequence has been used for serotype identification and the additional distinction between some strains (Gibert et al., 1990; Lam & Roth, 1983; RUBINO et al., 1998). Additionally, utilizing variation within ribosomal DNA, by restriction enzyme mapping of 23S and 16S genes (Altwegg et al., 1989; Esteban et al., 1993) and the intergenic space, known as intergenic sequence ribotyping (ISR) (Lagatolla et al., 1996) can be used to differentiate serovars. Another method for serogrouping is phage-typing, which utilizes the highly specific viral receptor proteins to identify the O-group of an isolate (Anderson et al., 1977; Anderson & Williams, 1956; Demczuk et al., 2003). These methods, while efficiently serotyping isolates, cannot obtain significant strain information that is useful for outbreak tracebacks.

Subtyping

To better differentiate strain information, pulsed-field gel electrophoresis (PFGE) was developed to give isolates a genetic "fingerprint" that can be compared to other isolates and identify potential outbreaks. This method utilizes restriction enzymes (typically XbaI, BlnI, SpeI, SfiI, PacI, and NotI for *Salmonella*) to cut the chromosome at various locations, resulting in a series of DNA bands of different lengths (Schwartz & Ft Cantor, 1964). An oscillating electric current is then used to separate these large bands on an agarose gel; the resulting gel image can then be compared to a PFGE database (e.g.,

CDC's PulseNet) to determine the genetic proximity of related isolates (Ricke, 2014). This method can discern strains within a serovar, but as this subtyping method only shows lengths of DNA and not sequence, it is not always sufficient for outbreak tracking, especially in the case of highly clonal serovars (Allard et al., 2013).

To satisfy the need to track outbreaks using DNA sequence information, multilocus sequence typing (MLST) was developed to elucidate the sequence of seven highly conserved housekeeping genes for serovar Typhi: *aroC*, *dnaN*, *hemD*, *hisD*, *purE*, *sucA*, and *thrA* (Kidgell et al., 2002). In this process, all seven genes are individually amplified by PCR and sequenced. Each gene is given an allelic identifier and the combination of the seven alleles is represented by a sequence type (ST). The strain is then given an overall sequence type (MLST) and compared to other strains, where a match in six out of seven genes and a change in the seventh gene would result in a different sequence type. This subtyping procedure is effective in characterizing non-typhoidal *Salmonella* strains; however, the depth of subtyping is limited to the sequence of these seven genes, therefore more closely related strains or highly clonal serovars, such as Enteritidis, are not able to be separated using this method (Achtman et al., 2012; Maiden, 2006).

Analogous to MLST, Clustered Regularly Interspaced Short palindromic repeats (CRISPR) typing is an alternate subtyping method. The CRISPR system in *Salmonella*, like many other prokaryotes, evolved to defend against conjugative plasmids and viral predation via acquired immunity (Grissa et al., 2007; Touchon & Rocha, 2010). In *Salmonella enterica* subspecies *enterica*, the presence of two CRISPR loci, separated by approximately 20kb, consist of highly conserved direct repeats 29 nucleotides long and

highly variable spacers 32 nucleotides long (Fabre et al., 2012; F. Liu, Barrangou, et al., 2011; F. Liu, Kariyawasam, et al., 2011). Following the survival from a conjugative plasmid or viral infection, a 32-nucleotide sequence from the foreign DNA is added at the leading end of the CRISPR loci. These loci, along with CRISPR-associated (*cas*) genes can then be expressed, where this sequence can be used to identify the presence of a repeated invasion by the same foreign DNA (Barrangou et al., 2007; Garneau et al., 2010; Pourcel et al., 2005). Interestingly, this system is no longer functional as a defense mechanism in *Salmonella*; however, the presence of the CRISPR loci remains consistent (Shariat et al., 2015).

CRISPR-typing utilizes the hypervariability of the spacer sequences between isolates belonging to the same serovar as a subtyping approach in *Salmonella* (F. Liu, Barrangou, et al., 2011). This process involves the independent amplification of both CRISPR loci and the comparison of their sequences, which, depending on the application, can be compared to a database of analyzed isolates (Fabre et al., 2012; Shariat & Dudley, 2014). This method was developed originally in *Mycobacterium tuberculosis* (Kamerbeek et al., 1997) and was called spoligotyping, and has since been adapted to Group A *Streptococcus* (Hoe et al., 1999), *Campylobacter* (Schouls et al., 2003), and *E. coli* (Delannoy et al., 2012). Notably, the CRISPR system remains active in *M. tuberculosis* and Group A *Streptococcus*, and is no longer active in *Campylobacter* and *E. coli*; however, these systems are commonly identified across tested isolates (Wei et al., 2019, Nozawa et al., 2011, Yeh et al., 2024, Touchon et al., 2011). Similarly to the hypervariability of CRISPR regions, virulence genes experience significant selective pressure and therefore evolve at a higher rate (Endo et al., 1996). Combining sequence

analysis of *Salmonella* virulence genes *fimH* and *sseL* with CRISPR loci improved the subtyping capability of nine clinically relevant serovars in a process called CRISPR-multi-virulence-locus sequence typing (CRISPR-MVLST) (F. Liu, Kariyawasam, et al., 2011). This method has since been adapted to many other *Salmonella* serovars (Almeida et al., 2017; Shariat, DiMarzio, et al., 2013; Shariat, Kirchner, et al., 2013; Shariat, Sandt, et al., 2013; Vilela et al., 2024; Vosik et al., 2018). Similar to MLST, this method is restricted in its subtyping discrimination, as analysis based on a small number of variable genomic regions does not account for the full genomic diversity between strains. Interestingly, the conservation of these molecular targets within a serovar also allows for CRISPR-typing and MLST to be used for serotyping. For example, CRISPR spacer content is highly associated with serovar identity (Deng et al., 2015; Fabre et al., 2012), as is MLST (Achtman et al., 2012).

Advances in sequencing technology have allowed scientists to utilize bacterial genomes for strain discrimination rather than individual components, known as whole genome sequencing (WGS). This technology is used to characterize individual genomes that can be compared to one another or to entire databases to assess genomic differences, such as gene presence or absence, insertion of genetic elements including plasmids and transposons, or single nucleotide polymorphisms (SNPs). These differences can then be used to characterize the phylogenetic relationship of strains, where closely related strains can be identified during outbreak investigations. This highly discriminatory subtyping tool is the foundation for GenomeTrakr, an open-source database of whole genome sequences used to detect closely related strains and identify sources of transmission (Allard et al., 2016; Timme et al., 2019). In these cases, WGS can identify SNPs and

more targeted genetic characteristics to trace outbreaks (M. Hoffmann et al., 2016). SNP-based characterization using WGS can include as few as the seven genes used in traditional MLST, or as much as the whole genome (wgMLST), with varying levels of depth, including ribosomal (rMLST) and core genome (cgMLST) (Mohammed & Thapa, 2020). The utility of WGS for outbreak tracebacks and higher-resolution characterization has been demonstrated by its ability to outperform PFGE, MLST, and other tools in recent outbreaks (Allard et al., 2012, 2013; den Bakker et al., 2014; Deng et al., 2015; M. Hoffmann et al., 2016; Lienau et al., 2011). Additionally, WGS allows for more robust serovar characterization by mapping serovar-specific genes beyond those responsible for surface antigens, such as the *Salmonella in silico* Typing Resource (SISTR) and SeqSero 2.0 (Yachison et al., 2017; Yoshida et al., 2016; S. Zhang et al., 2019a). SISTR identifies serovars via O-antigen flipase (*wzx*) and polymerase (*wzy*) genes and the *fliC* and *fljB* genes for the H1 and H2 determination, while SeqSero 2.0 targets the *rfb* gene cluster for the O-antigen and the *fliC* and *fljB* genes for H-antigen determination. For these reasons, WGS has become firmly established as the gold standard for *Salmonella* serotyping and subtyping. Having first been developed by the Center for Food Safety and Applied Nutrition at the US-FDA, WGS was adopted in 2014 by the CDC and USDA-FSIS for their *Salmonella* analyses.

1.7 Limitations of *Salmonella* isolation and characterization

All methods previously described above rely on the isolation of an individual isolate following *Salmonella* enrichment. Traditional isolation techniques rely on the picking of one to five colonies for characterization (FDA, 2024a; USDA-FSIS, 2024b); however, prior observations have suggested that six colonies would need to be picked to

reliably identify two serovars at equal proportions in a population (Cason J. et al., 2011). If a serovar composes only 10% of a population, 32 colonies would need to be picked. This depth of colony picking presents a serious limitation in *Salmonella* surveillance and source tracking analysis. Considering the upper limit of five picked colonies from a positive sample, this will often result in picking only the most abundant serovar or serovars in a mixed population. Consequently, serovars that account for a smaller proportion of the population are less likely to be detected, where these less abundant serovars have the potential to contribute to human illness (Berghaus et al., 2013; Cason J. et al., 2011).

To overcome this problem, a tool was developed that would be able to detect the presence of multiple serovars in a sample. CRISPR-SeroSeq is a deep serotyping method that utilizes the serovar-specific CRISPR spacers to differentiate serovars and provide a relative abundance of each serovar in a single sample (Thompson et al., 2018). In contrast to CRISPR-Typing, where each entire CRISPR locus is amplified by PCR, the primers used in CRISPR-SeroSeq target the highly conserved direct repeat sequences. This generates short sequences that collectively present the spacer content of *Salmonella* present in a given sample. By comparing the short sequence reads to a database of known spacer sequences, the serovars can be determined. Further, the total number of sequence reads belonging to each serovar within a sample can be used to determine the relative frequency of each serovar. Further, polyphyletic deliniations can be identified where a serovar can contain two or more distinct CRISPR profiles, indicating a potential evolutionary recombination event (Cherchame et al., 2022; Worley et al., 2018). This process has been used to identify multiserovar populations within animal agriculture and

environmental samples, (Deaven et al., 2021; Siceloff et al., 2021; Thompson et al., 2018). Results from Thompson et al. 2018 showed that this technology can confidently identify serovars constituting as little as 0.01% of the population, though this is dependent on the depth of sequencing. In the first application of this technology, 91% of positive samples were found to contain multiple serovars, and up to four serovars were identified in a single sample. Siceloff et al. 2021 provided a parallel comparison of traditional isolation and CRISPR-SeroSeq deep serotyping in cattle fecal samples, where deep serotyping identified tetracycline-resistant serovar Reading in multiple isolates that were not identified by picking colonies. This provides context to the importance of identifying and characterizing all serovars in a population, as a tetracycline treatment given to these cattle would have allowed serovar Reading to survive. Relevant to the work here, Deaven et al. 2021 identified highly complex *Salmonella* populations in surface water, including up to ten serovars in a single sample. In this study, 80% of positive samples contained multiple serovars, most of which (78%) contained one of the top ten serovars associated with human illness. Importantly, serovars Typhimurium and Enteritidis were masked by other serovars in 78% and 71% of samples when present, respectively. This suggests a decreased likelihood of detecting these important serovars using traditional isolation methods. Additionally, this work began to characterize potential influences of *Salmonella* population complexity, such as precipitation and subsequent river discharge. The width of the Susquehanna River introduced a challenge in this study, limiting the ability to assess the full complexity of this water source. To overcome this challenge, more regular sampling of smaller creeks would better represent downstream and temporal changes. Collectively, this data suggests that *Salmonella*

populations in animals and the environment are highly complex and that these mixed populations need to be fully characterized to properly evaluate food safety risks. This method has transformed what we know about *Salmonella* ecology and serovar dynamics, and these findings suggest a need to re-evaluate and more deeply characterize the diversity and dynamics of *Salmonella* serovars in different environments and hosts.

1.8 Objectives for this dissertation

Salmonella enterica is a leading cause of bacterial foodborne illness in the United States. In addition to its high prevalence in food animals, *Salmonella* is commonly found in environmental sources such as wildlife and water; however, prevalence within these sources can be variable. Because of this, potential sources of contamination exist within a variety of scenarios (CDC/FDA/USDA, 2023). *Salmonella* is comprised of over 2,600 serovars, characterized by somatic (O) and flagellar (H) antigens (P. A. D. Grimont & Weill, 2007). In addition to antigenic differentiation, phenotypic and genotypic differences including host restriction (Pascopella et al., 1995), virulence factors (Cheng et al., 2019), and antimicrobial resistance (D. H. Shah et al., 2017) exemplify the genomic placidity of *Salmonella*. Only 80 serovars account for 99% of human clinical cases in the US, and 13 of these contribute to over 75% of cases (CDC, 2024a). Many environmental sources of *Salmonella* are understudied such as wildlife and water, resulting in the inability to mitigate risks associated with these sources. It is critical to identify potential routes of contamination and prevent their entry into our food systems to reduce the number of illnesses attributed to *Salmonella*.

One challenge in this field of research is the lack of studies on the environmental prevalence of *Salmonella*. While animal agriculture and processing account for the bulk

of the literature about prevalence and contamination, environmental sources including wildlife and water require further investigation. The prevalence and diversity of serovars found within these understudied sources can provide important information about the routes of contamination that are not being protected. Previous work from our lab has demonstrated that *Salmonella* often exists in multiserovar populations, where traditional isolation techniques often only identify the dominant serovar in a population (Thompson et al., 2018). This creates a unique challenge in risk assessment, as less abundant serovars may contribute to negative health outcomes if left unidentified and untreated (Siceloff et al., 2021).

To address these obstacles and develop a further understanding of *Salmonella* prevalence and population dynamics in the environment, my doctoral research aims to evaluate the prevalence and diversity of *Salmonella* populations within previously understudied environmental reservoirs. This was completed by first assessing the risk associated with wild bird transmission of *Salmonella* in produce fields in the southeastern United States. In the first survey of *Salmonella* within wild birds in this region, I found a notable decrease in prevalence compared to studies on the West Coast, where pathogen viability was strongly associated with the freshness of feces. This work demonstrates a regional difference in risk assessment that growers can utilize for individual management strategies. I then characterized the prevalence and population of *Salmonella* in four distinct creeks over two years in the southeastern United States. This study found high *Salmonella* recovery and complexity associated with increased precipitation and humidity, while proximal land use was found to have a less significant influence. Additionally, many serovars not commonly found in food animal surveillance testing

were identified in water samples, suggesting a potential reservoir for clinically relevant *Salmonella*. Finally, I explored the population and prevalence of *Salmonella* in the rivers of both agricultural and urban rivers in Pinchinca, Ecuador to identify similarities and differences between these water sources. There I found significantly increased complexity within sites of the urban river compared to the agricultural river, including many clinically relevant serovars. Additionally, signs of persistence and downstream transmission were identified, suggesting a need to monitor and manage these important sources for safe, clean water. In conclusion, my work combines longitudinal studies with genomic and population sequencing to assess the prevalence, diversity, and risk associated with different environmental sources of *Salmonella.*

To establish a more robust understanding of the transmission of *Salmonella* between humans, food systems, and the environment to develop more effective food safety mitigation strategies, I employed two specific aims:

1. Assess the prevalence and complexity of *Salmonella* populations in wild birds and surface water sources.
2. Evaluate risk factors that influence *Salmonella* viability within wild bird and surface water environments.

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CHAPTER 2

PREVALENCE AND MOLECULAR CHARACTERIZATION OF *SALMONELLA* ISOLATED FROM WILD BIRDS IN FRESH PRODUCE ENVIRONMENTS[1]

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2.1 Abstract

Wild birds pose a difficult food safety risk to manage because they can avoid traditional wildlife mitigation strategies, such as fences. Birds often use agricultural fields and structures as foraging and nesting areas, which can lead to defecation on crops and subsequent transfer of foodborne pathogens. To assess the food safety risk associated with these events, wild bird feces were collected from produce fields across the southeastern United States during the 2021 and 2022 growing seasons. In total 773 fecal samples were collected from 45 farms across Florida, Georgia, South Carolina, and Tennessee, and 2.1% ($n = 16$) of samples were *Salmonella*-positive. Importantly, 75% of *Salmonella* were isolated from moist feces, showing reduced *Salmonella* viability when feces dry out. 16S microbiome analysis showed that presence of culturable *Salmonella* in moist feces correlated to a higher proportion of the Enterobacteriaceae family. From the *Salmonella*-positive samples, 62.5% (10/16) contained multi-serovar *Salmonella* populations. Overall, 13 serovars were detected, including six most commonly attributed to human illness (Enteriditis, Newport, Typhimurium, Infantis, Saintpaul, and Muenchen). PCR screening identified an additional 59 *Salmonella*-positive fecal samples, which were distributed across moist ($n = 44$) and dried feces ($n = 15$). On-farm point counts and molecular identification from fecal samples identified 57 bird species, including for 10 *Salmonella*-positive fecal samples. Overall, there was a low prevalence of *Salmonella* in fecal samples, especially in dried feces, and we found no evidence of *Salmonella* transmission to proximal foliage or produce. Fecal samples collected in farms close together shared highly related isolates by whole genome sequencing and also had highly similar *Salmonella* populations with

comparable relative frequencies of the same serovars, suggesting the birds acquired *Salmonella* from a common source.

2.2 Introduction

Salmonella enterica is a leading contributor of bacterial foodborne illness in the United States (Scallan et al., 2011; Tack et al., 2019). While *Salmonella* is an enteric pathogen, it can be found in non-host environments, such as surface water and soil, as well as on produce (Critzer and Doyle, 2010; Gorski et al., 2011; Strawn et al., 2013; Reddy et al., 2016; Bardsley et al., 2021; Deaven et al., 2021), where it can survive and cause outbreaks (CDC, 2023). Consumption of contaminated produce causes an estimated 44.2% of salmonellosis cases in the United States [The Interagency Food Safety Analytics Collaboration (IFSAC), 2022]. In produce, contamination can occur through water, soil, equipment, personnel, and wildlife introduction events (Alegbeleye et al., 2018; Rodrigues et al., 2019; Devarajan et al., 2021, 2023). Because produce is often eaten raw and post-harvest kill steps are limited, there is a significant need to understand and mitigate potential sources of contamination. The Standards for the Growing, Harvesting, Packing, and Holding of Produce for Human Consumption (Produce Safety Rule, 74354, 2015) went into effect in 2016 as a part of the Food Safety Modernization Act. This rule set the first federally mandated standards for the safe production of fruits, vegetables, and nuts, and includes requirements for microbial quality of production and postharvest water, soil amendments, cleaning and sanitation practices, worker training and hygiene, and wildlife mitigation in order to reduce the likelihood of foodborne pathogen-contamination to produce. While many of these standards have clear guidelines, wildlife mitigation is often limited to physical barriers to prevent foraging from deer,

raccoons, and other land animals (Hamilton et al., 2015). These precautions do little to prevent the intrusion of birds, which can easily fly into fields to forage for plants, insects, or small rodents. Birds are a further challenge as they can become accustomed to deterrents and often fly long distances while migrating (Rivadeneira et al., 2018; Elsohaby et al., 2021).

Wild birds are known to carry foodborne pathogens, including *Salmonella enterica* subspecies *enterica* (Tizard, 2004). Studies performed in the western and southwestern United States found *Salmonella* prevalence in wild birds at 0.5–6.5% (Gorski et al., 2011; Rivadeneira et al., 2016; Navarro-Gonzalez et al., 2020). Additionally, flocks of wild bird can spread disease among individuals when congregating at common food and water sources (Hernandez et al., 2016). Outside of explosive mortality events caused by *Salmonella* serovar Typhimurium (Hernandez et al., 2012), *Salmonella* does not typically elicit symptoms in wild birds, so healthy carriers can transmit this pathogen without suffering from salmonellosis (Prosser et al., 2011). Transmission of pathogens from birds to produce can occur through defecation when birds are flying over fields or foraging for food. An outbreak of *Salmonella* serovar Typhimurium in 2009 found matching strains in birds, peanut crops, and human clinical cases (Hernandez et al., 2012). While birds can benefit farms by providing services like natural pest control (Karp et al., 2013), their habituation in production environments could play a role in the transmission of foodborne pathogens via fresh produce.

Salmonella enterica is a diverse species, consisting of over 2,600 distinct serovars that are categorized by their unique O (somatic) and H (flagellar) antigens (Grimont and Weill, 2007; Issenhuth-Jeanjean et al., 2014). Genomic diversity between these serovars

has led to differences in host specificity, pathogenesis, and antibiotic resistance profiles (Uzzau et al., 2000; Cheng et al., 2019). While some serovars are most typically found in a small number of reservoirs (e.g., serovar Enteritidis is most closely linked to poultry), others, such as serovar Typhimurium, are ubiquitous and found in a variety of different hosts. Further, *Salmonella* is often detected in food animal production systems and the environment as mixed populations of multiple serovars (Deaven et al., 2021; Siceloff et al., 2021, 2022; Obe et al., 2023). In some instances, low frequency serovars in these populations may have greater potential impacts on public health when they have clinically relevant antimicrobial resistance profiles (Siceloff et al., 2022) or are more often associated with human illness (Deaven et al., 2021). Traditional isolation techniques that rely on picking a small number of colonies from selective agar are unable to resolve complex multi-serovar *Salmonella* populations (Cason et al., 2011). This hurdle is overcome by deep serotyping approaches such as CRISPR-SeroSeq, which can resolve the relative frequencies of multiple serovars in a single sample (Thompson et al., 2018).

In this study, we investigated the role of wild birds in the transmission of *Salmonella* to produce foliage in the southeastern United States. This study region includes more than 12 million acres of cropland (CroplandCROS, 2022) where produce such as tomatoes, peppers, eggplant, and other fruit, vegetable, and nut crops are significant economic contributors. Wild bird feces were collected from produce fields over a two-year period and cultured for *Salmonella*. Deep serotyping and whole genome sequencing were performed to assess *Salmonella* populations and to estimate source

attribution. Additionally, wild bird species were identified with both physical and molecular techniques to associate pathogen transmission risk.

2.3 Materials and Methods

2.3.1 Site selection and overview of study design

To study the impact of wild bird activity upon produce contamination, 45 different farms across the southeastern United States (Tennessee, Georgia, Florida, and South Carolina) were visited between 1–6 times (average 2.4 visits/farm). Produce grown on these farms included peppers (bell, banana, and jalapeño), eggplant, cucumbers, tomatoes, squash, grapes, pole beans, and okra. These above ground produce were chosen because they pose a greater risk for human illness should they be contaminated, as many are often eaten raw. Additionally, selecting produce growing above ground reduced the incidence of identifying contamination from on-ground sources, such as soil, or rodents or other small wildlife that primarily forage on the soil surface. Farms in this study were diverse and included organic and conventional farms, commercial and family-run operations, mono- and polyculture farms, and some had livestock on and around the farm. To best measure the effect of seasonality on the prevalence of *Salmonella*, repeated sample collections were completed at farms, up to three times per sampling season (May–October), where possible. During each sampling visit, crops around the perimeter and the interior of the fields were inspected to identify wild bird fecal samples. When fecal samples were identified, the leaf containing the feces was removed and homogenized for culturing *Salmonella*. To evaluate the necessity of exclusion zones encouraged by groups such as the Leafy Green Marketing Agreement (LGMA), surface swabs of a piece of produce beneath the fecal sample and from the leaf of a neighboring plant downwind

were also collected. *Salmonella* was first identified by culture. Samples found positive for *Salmonella* culture were then further analyzed with additional molecular tools (e.g., PCR, WGS, and CRISPR-SeroSeq). The culture-negative samples were then analyzed by a *Salmonella* PCR.

2.3.2 Sample collection

Fecal samples were collected between sunrise and 11 am to capture on-field bird activity while also limiting UV exposure and reducing the opportunity for desiccation. Upon arriving at a farm, sampling was conducted around the perimeter of each field, followed by a step-wise sampling through the interior of the field. On smaller farms (or small (<1 acre) fields on a large farm), all individual rows were surveyed. When a fecal sample was identified, it was visually scored for moisture as either 1 (moist) or 0 (dry) as an indicator of freshness. Then, the leaf containing the fecal sample was removed, inserted into 2 mL buffered peptone water (BPW, Hardy Diagnostics, Ohio, USA) recovery media, and placed on ice until culturing (within 24 h). Because the fecal samples were small and because in some cases removing them from the leaf would lose some of the fecal material, the entire leaf was removed from the plant and then the portion containing just the feces and the leaf material directly under the feces were isolated and collected. To test for transmission of *Salmonella* from the fecal sample, the surface of a piece of produce under the leaf was swabbed, along with a leaf of a neighboring plant downwind from the fecal sample. These swabs were collected by soaking a sterile cotton ball in 3 mL of BPW and using sterile forceps to drag it across the top and bottom of the neighboring leaf and across the entire surface of the produce. Swabs were placed in a

cooler with ice packs and stored at 4°C for no more than 24 h or until culturing could begin in the laboratory.

2.3.3 *Salmonella* culturing

Fecal samples were homogenized by hand into the 2 mL of recovery media. For *Salmonella* isolation, 750uL of the homogenate was transferred into a culture tube containing 9.25 mL BPW and incubated at 42°C for 24 h. Then, this was sub-inoculated into 9 mL Tetrathionate (TT, Neogen Diagnostics, Michigan, USA) and 9.9 mL Rappaport-Vassiliadis (RV, Hardy Diagnostics, Ohio, USA) selective enrichment broths in parallel and incubated for 24 h at 37°C before being streaked onto Xylose Lysine Tergitol-4 agar plates (XLT-4, Hardy Diagnostics, Ohio, USA). The plates were incubated at 37°C for 24 h and inspected for black colonies as an indicator of presumptive *Salmonella* colonies. If no H2S-positive colonies were present, the plates were re-incubated for another 24 h. Up to 2 colonies from each sample were selected and were re-streaked onto XLT-4 for isolation if needed. *Salmonella* isolates were grown in Luria Broth (LB, Hardy Diagnostics, Ohio, USA) where aliquots were used to make frozen glycerol stocks and for DNA isolation. If we observed presumptive *Salmonella* colonies, we then returned to the swabs from produce and neighboring foliage and cultured these using the same protocol.

2.3.4 DNA isolation and *Salmonella* PCR screen

The total genomic DNA was isolated from 500uL of the fecal/recovery media homogenate using the Genome Wizard kit (Promega, Wisconsin, USA), with the additional step of grinding the fecal pellet with a sterile mortar and pestle to disrupt the fecal particles before beginning the extraction. Prior to any PCR (i.e., for *Salmonella* or

for COI), DNA from this fecal/recovery media homogenate was screened with an internal amplification control (IAC) PCR to identify the presence PCR inhibitors (Rosenstraus et al., 1998). The primers for IAC PCR were IAC_F (5'-AGTTGCAGTGTAACCGTCATGT-3′) and IAC_R (5′-TCGACGAGACTCTGCTGTTAAG-3′) and the IAC template control sequence was IAC (5'-AGTTGCAGTGTAACCGTCATGTACCAGTAATCTGCGTCGCACGTGTGCACCTA GTCTA ATCACTTATGACTCAGATAACTTAACAGCAGAGTCTCGTCGA-3′). For each reaction, the following components were mixed: 39.5uL sterile water, 5uL 10x Taq Buffer, 0.5uL 10uM forward primer, 0.5uL 10uM reverse primer, 0.3uL 100 mM dNTPs, and 1 U Taq polymerase, before 2uL of bird fecal DNA was added as template. Cycle conditions were as follows: 95°C for three minutes followed by 40 cycles of 95°C for 30 s, 56°C for 30 s, and 72°C for 30 s. This was followed by a final elongation of 72°C for two minutes and resting at 4°C. PCR products were visualized by gel electrophoresis. Where there was no amplification, suggesting the presence of PCR inhibitors, a 1:10 dilution of the bird fecal sample DNA was made, and the PCR repeated. In this study, nearly 10% (n = 75) of samples contained PCR inhibitors as shown by the IAC PCR. This inhibition was resolved when the template was diluted 10-fold in molecular grade water, and this dilution was used for all subsequent PCRs.

For the *Salmonella* screening, an invA PCR was used (Rahn et al., 1992). In this PCR, primers – InvA_F1 (5'-AACGTGTTTCCGTGCGTAAT-3′) and InvA_R1 (5′-TCCATCAAATTAGCGGAGGC-3′) were mixed with 38.5uL sterile water, 5uL 10x Taq Buffer, 2uL of 6.25ug/mL BSA, 1uL 10uM forward primer, 1uL 10uM reverse

primer, 0.25uL 100 mM dNTPs, and 1 U Taq polymerase before 2uL of bird fecal sample DNA was added as template. Cycling conditions began with an initial melting temperature of 95°C for three minutes followed by 40 cycles of the following: 95°C for 30 s, 56°C for 30 s, and 72°C for 30 s. A final elongation temperature of 72°C for two minutes was completed before resting at 4°C.

2.3.5 *Salmonella* weather analysis

For each site, weather data from the day prior to collection, including total precipitation, average wind, average humidity, and high temperature values were determined using the closest USGS weather stations. To identify relationships between weather variables and moist feces, we conducted a series of binomial generalized linear mixed models (GLMM) using the glmmTMB package V1.1.7 (Brooks et al., 2023) within R V4.1.1. All continuous variables were standardized prior to analysis. Visits nested within farm and year were used as random effects. We ran models using individual variables as a fixed effect in each model and considered different additive configurations of other weather variables. We assessed multicollinearity using the performance package V0.10.3 (Lüdecke et al., 2021) and homogeneity of variance using the DHARMa package V0.4.6 (Hartig and Lohse, 2022); models meeting these assumptions (i.e., VIF < 5 and equally distributed residuals, respectively) were retained for comparison. Models were compared using the Akaike Information Criterion adjusted for small sample sizes (AICc) using the R package AICcmodavg V2.3–2. We considered "top models" as those with $\Delta AICc \leq 2$ (Burnham and Anderson, 2002). The same weather stations were used to calculate the monthly average weather values (Supplementary Figure S2.1).

2.3.6 Whole genome sequencing

Total genomic DNA from *Salmonella* isolates was extracted using a Promega Genome Wizard DNA extraction kit (Promega, Wisconsin, USA) and sequenced on an Illumina MiSeq 500 cycle v2 chemistry kit (Illumina, California, USA). The sequence reads were assembled using SPAdes de-novo assembly (Version 3.15.5) (Bankevich et al., 2012) and the serovar determined using SeqSero 2.0 (Zhang et al., 2019). Sequences were uploaded to Enterobase (Zhou et al., 2020) where sequence types (ST) could be predicted and used to identify related isolates. Phylogenetic relatedness was visualized through GrapeTree and allelic differences were used to identify the closest related source type. The assembled genomes were uploaded to NCBI (Accession numbers SAMN33186945, SAMN33186956, SAMN33186963, SAMN33186964, SAMN33186971, SAMN33186984, SAMN33187804, SAMN33187835, SAMN33187836, SAMN33187842, SAMN33187843, SAMN33187878, SAMN33187961, SAMN33187962, SAMN33187972, SAMN33225914, SAMN37196586, SAMN37196587, and SAMN37196588).

2.3.7 *Salmonella* population analysis

To identify the populations of *Salmonella* within wild bird feces, TT and RV enrichments from *Salmonella* culture positive samples were processed individually by centrifuging 1 mL of each selective enrichment at 14,000 rpm for three minutes. Total genomic DNA was isolated from the resulting pellet using a Promega Genome Wizard Kit and resuspended in 200uL of molecular-grade water. A total of 2 µL of this template was used in the PCR for CRISPR-SeroSeq with primers targeting the conserved direct repeat sequences within *Salmonella* CRISPR arrays (Thompson et al., 2018; Siceloff et

al., 2022). Primers also included index sequences which facilitated multiplexed, high throughput sequencing. PCR products were purified using the Ampure system (Beckman Coulter, Indianapolis, IN) and pooled in approximate equimolar ratios. Pooled libraries were sequenced using the Illumina NextSeq 550 platform (Illumina, California, USA) mid output 150 cycle v2.5 kit with single-end reads. A water negative-control and a positive control containing *Salmonella* serovar Enteritidis genomic DNA with a known CRISPR profile were included in the library. Sequence reads were scanned and matched in a local BLAST search to a lab-curated database of over 150 serovars (Siceloff et al., 2022).

Serovars were called only if they contained multiple CRISPR spacers that were unique to that serovar. Where there were sufficient *Salmonella* sequence reads (>1,000 reads) for both the TT and RV enrichments the relative frequency of each serovar was normalized across both enrichments to provide a single serovar profile.

2.3.8 Microbiome analysis

All 16S rRNA Illumina-tag PCR reactions were performed on DNA extracts per the Earth Microbiome Project protocol (Walters et al., 2016). Negative controls (molecular grade water) were processed in parallel with the samples for PCR amplification. PCR products were pooled in batches of ~200 samples each and gel purified on a 2% agarose gel using the QIAquick Gel Purification Kit (Qiagen, Frederick, Maryland, USA). Before sequencing, purified pools were quality checked using an Agilent 2100 BioAnalyzer and Agilent DNA High Sensitivity DNA kit (Agilent Technologies, Santa Clara, California, USA). The purified pools were stored at −20°C, then sequenced using an Illumina MiSeq 500 cycle v2 chemistry kit (Illumina, California,

USA). Raw data were processed, analyzed, and quality checked with QIIME2 (Bolyen et al., 2019) before forward and reverse reads were merged and chimeras removed with DADA2 (Callahan et al., 2016). DADA2 was also used to assign sequences to amplicon sequence variants (ASVs) using a pre-trained Silva 132 Database (Quast et al., 2012). MAFFT (Katoh and Standley, 2013) and FastTree (Price et al., 2010) were used to create a rooted phylogenetic tree using representative ASVs. Additionally, a biomarker analysis was completed to identify taxonomic groups that were differentially abundant within groupings of samples (*Salmonella* Culture, *Salmonella* PCR, and No *Salmonella*) using LEfSe (Segata et al., 2011) by normalizing the ASVs with the counts per million method and a differential abundance value of p of <0.05 and a log (LDA) score of at least 1.0.

2.3.9 Bird species identification

Wild birds were identified in two ways: physical identification (i.e., point counts) of birds present around and in fields, and molecular identification from feces. Point counts were conducted at all field locations on sample days between 6 and 10 am. One point count was done for every 10 hectares (ha) of sampled field when field conditions and harvesting schedules allowed. Points on the same farm were at least 200 m apart. Points were positioned approximately 90 m away from the edge of fields to overlap with bacterial sampling areas while still capturing birds moving in and out of produce. All birds seen and heard within a 100-m radius during a 10-min period were recorded, along with distance, detection method, and habitat. During the 10 min, birds were counted in sub-periods of three, three, and four minutes. Only new species were counted after the first sub-period to avoid counting the same individual multiple times. Birds flying overhead were excluded unless they were a species that forages aerially (e.g., swallows),

in which case a note was made that they were “aerial foraging.” The same observer conducted all counts for both years of sampling. Birds were categorized as in-field if they were observed interacting with produce (e.g., in tunnels, perching on produce stakes, or on produce plants) and other birds were categorized as off-field.

Molecular identification of wild bird species from fecal samples was completed using 2uL of DNA isolated from fecal samples as part of a PCR to amplify the Cytochrome C Oxidase Subunit I (COI). The sequence variability of the COI gene between bird species enables species identification. Many COI PCR assays were attempted, following published protocols (Hebert et al., 2003; Ivanova et al., 2007; Kerr et al., 2007; Joo and Park, 2012), but either did not yield amplicons or failed to produce quality sequences. This PCR used the following primers: COI_F1 (5’-CGCYTWAACAYTCYGCCATCTTACC-3′) and COI_R1 (5′-ATTCCTATGTAGCCGAATGGTTCTTT-3′) (Patel et al., 2010). For each reaction, the following were mixed into a 50uL reaction: 38.5uL sterile water, 5uL 10x Taq Buffer, 2uL 25 mM MgCl2, 1uL 10uM forward primer, 1uL 10uM reverse primer, 0.3uL 100 mM dNTPs, and 1 U Taq polymerase along with adding 2uL of DNA template. The mix was run on the following PCR program: Initial melting of 95°C for four minutes was followed by five cycles of 95°C for 30 s, 59°C for 30 s, and 68°C for 45 s. This was followed by 40 cycles of 95°C for 30 s, 56°C for 30 s, and 68°C for 45 s and a final two-minute elongation step. Appropriately sized amplicons were sequenced in the forward and reverse direction by Eton Bioscience Inc. (Research Triangle Park, NC). SeqMan (Lasergene, DNA Star) was used to assemble the forward and reverse reads into a single

sequence, which was then compared to two databases: NCBI BLAST, and the Barcode of Life Database (Meiklejohn et al., 2019) with a 97% nucleotide identity threshold.

2.4 Results

During 2021 and 2022, 109 farm visits were performed across the southeastern United States, including Tennessee (n = 4 farms), North Georgia (n = 8), South Georgia (n = 20), South Carolina (n = 10), and North Florida (n = 3; Table 2.1). Farms ranged in size from 1.6–233 acres and included 13 small or independently owned farms (1.6–33.3 acres), as well as 32 large commercial farms (6.95–233 acres). Over the two seasons, 773 fecal samples were collected: 227 samples in 2021 and 546 in 2022. In total, 43.6% (337/773) of fecal samples were scored as moist, including 152 in 2021 and 185 in 2022, while 56.4% (436/773) were scored as dry, including 75 in 2021 and 361 in 2022 (Figure 2.1A).

By culture, *Salmonella* was isolated in 16 samples (16/773 total samples; 2.1%); 15 were identified in the first year of collection (15/227, 6.6%) and one was identified in the second year (1/546, 0.2%). Three quarters (12/16) of *Salmonella* samples were recovered from moist fecal samples (Figure 2.1B). *Salmonella*-positive samples were found in South Georgia (n = 10), Florida (n = 4), and North Georgia (n = 2). There was no recoverable incidence of transmission from fecal samples to produce below leaves with feces, nor to neighboring plants downwind. We screened all samples not confirmed positive by culture using a PCR targeting the *Salmonella* invA gene and detected *Salmonella* in 59 additional fecal samples, bringing the total *Salmonella*-positive samples to 75 (9.7%) (Table 2.2). Similar to culture-positive fecal samples, *Salmonella* was more commonly detected in the first year of collection, with 16.5% (35/212) of culture-

Table 2.1. Sampling distribution across the Southeast.

State	Number of farms (Number of visits)	Number of fecal samples collected
TN	4 (16)	218
SC	10 (29)	225
GA-N	8 (31)	235
GA-S	21 (27)	76
FL	3 (6)	19
Total	**45 (109)**	**773**

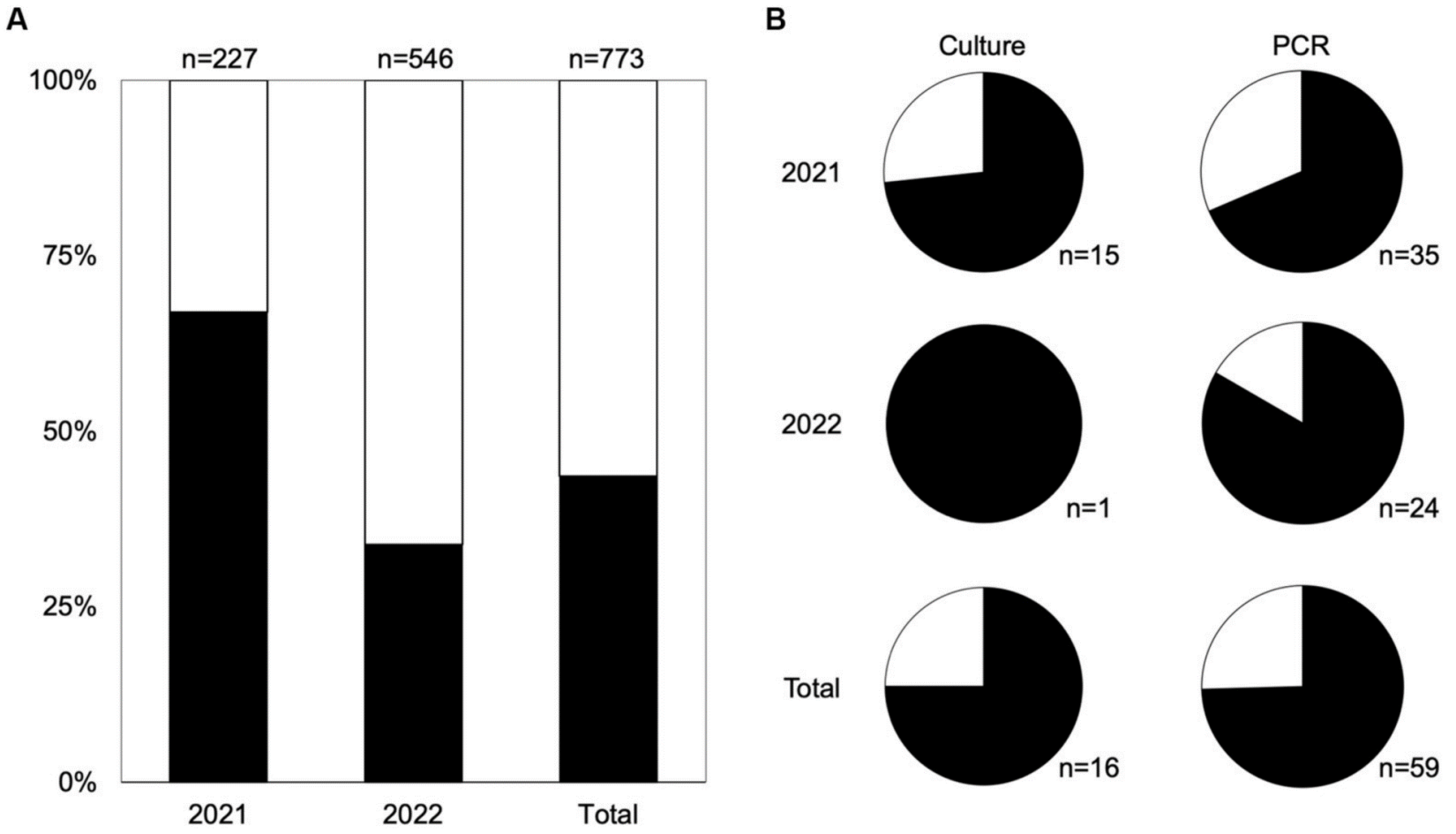
A
n=227
n=546
n=773
100%
75%
50%
25%
0%
2021
2022
Total
B
Culture
PCR
2021
n=15
n=35
2022
n=1
n=24
Total
n=16
n=59

Figure 2.1. **Moist feces support survival of *Salmonella* better than dry feces**. **(A)** The distribution of moist (black) and dry (white) feces per year and in total. **(B)** Proportion of *Salmonella*-positive samples in both culture positive (left) and PCR-positive samples (right) and the number of positive samples is indicated below each pie chart. Moist feces are shown in black and dry feces shown in white.

Table 2.2. ***Salmonella*** **prevalence increases with inclusion of molecular detection**.

	Fecal Samples	Viable *Salmonella*	Prevalence (%)	Additional PCR Positive	Prevalence (%)	Total	Prevalence (%)
Year 1	227	15	6.6	35	15.4	50	22
Year 2	546	1	0.2	24	4.4	25	4.6
Total	**773**	**16**	**2.1**	**59**	**7.6**	**75**	**9.7**

negative samples from 2021 being PCR-positive, while 4.4% (24/545) of culture-negative samples from 2022 were PCR-positive. The proportion of PCR positive samples in moist and dry feces matched the culture data, with three quarters (74.6%, 44/59) of the PCR-positive fecal samples being moist, compared to a quarter from dry feces (25.4%, 15/59) (Figure 2.1B). Overall, *Salmonella* was significantly more likely to be detected in moist samples than dried samples [$\chi 2(1, n = 773) = 6.55, p < 0.05$].

Given the positive association between *Salmonella* presence (by culture and by PCR) and moist feces, we used a binomial generalized linear mixed model (GLMM) to explore weather factors that could influence fecal moisture. We identified four top models (i.e., ΔAICc <2) (Supplementary Table S1). Precipitation the day before sample collection was included in three of the top models and was positively associated with moist feces. During most sampling months in 2022, monthly cumulative precipitation was lower than in 2021 (Supplementary Figure S2.1), which may explain the reduced *Salmonella* detection in 2022. Humidity was also included in three models and had negative correlations with moist feces. Although temperature did not appear in our models, we expect that high temperatures would contribute to drying the feces. During May–July, the average temperatures were hotter in 2022 than in 2021 in all sampled regions, which, in combination with reduced precipitation may also contribute to the reduced *Salmonella* detection in feces in the second year of sampling.

We assessed total microbial diversity in each fecal sample (n = 773) by 16S rRNA sequencing, with 720 samples passing quality control. Weather variables (precipitation, temperature, humidity, and wind) did not have a strong positive or negative (±0.30) impact to alpha diversity (data not shown). *Salmonella* was not found to affect species

richness when comparing culture-only positives or PCR positives to the *Salmonella* negative group (data not shown). Because the number of *Salmonella* culture positive samples was low, we presented the microbiome data stratified into six different groups, based on *Salmonella* status and fecal moisture. The group containing *Salmonella* culture positives from dry feces was removed from the groups, as the low number of samples (n = 4) reduced significant findings. Within moist feces, the Enterobacteriaceae family was significantly enriched in samples containing culturable *Salmonella* compared to samples containing only molecularly detectable *Salmonella* or no detectable *Salmonella* (Figure 2.2). This included a significant increase in the Escherichia-Shigella genera (these cannot be separated using 16S) in the *Salmonella*-culture group, rather than *Salmonella* (data not shown) (Wilcoxon rank sum test adjusted p value <0.05).

Whole genome sequencing was completed on 19 isolates (JSBird1-JSBird19) (Supplementary Table S2), and eight serovars were subsequently identified: Hadar (5 isolates), Give (4), Newport (4), Saintpaul (2), Kentucky (1), Mississippi (1), Muenchen (1), and Typhimurium (1) (Table 2.3). Using Enterobase, we next searched for related isolates. Four serovar Hadar isolates were closely related to each other (JSBird3-JSBird5 and JSBird10) and to isolates collected from ground turkey meat (within the same HeirC2 cgMLST cluster) (Supplementary Figure S2A,B). The fifth serovar Hadar isolate (JSBird11) was more closely related to an isolate from chicken meat (within the same HeirC5 cgMLST cluster) than to the other serovar Hadar isolates we isolated (Supplementary Figure S2.2C). Serovar Typhimurium and Kentucky were both isolated from the same fecal sample (F26) and both isolates were most closely related to isolates from chicken (each was within the same HeirC5 cluster of a chicken isolate). Serovar

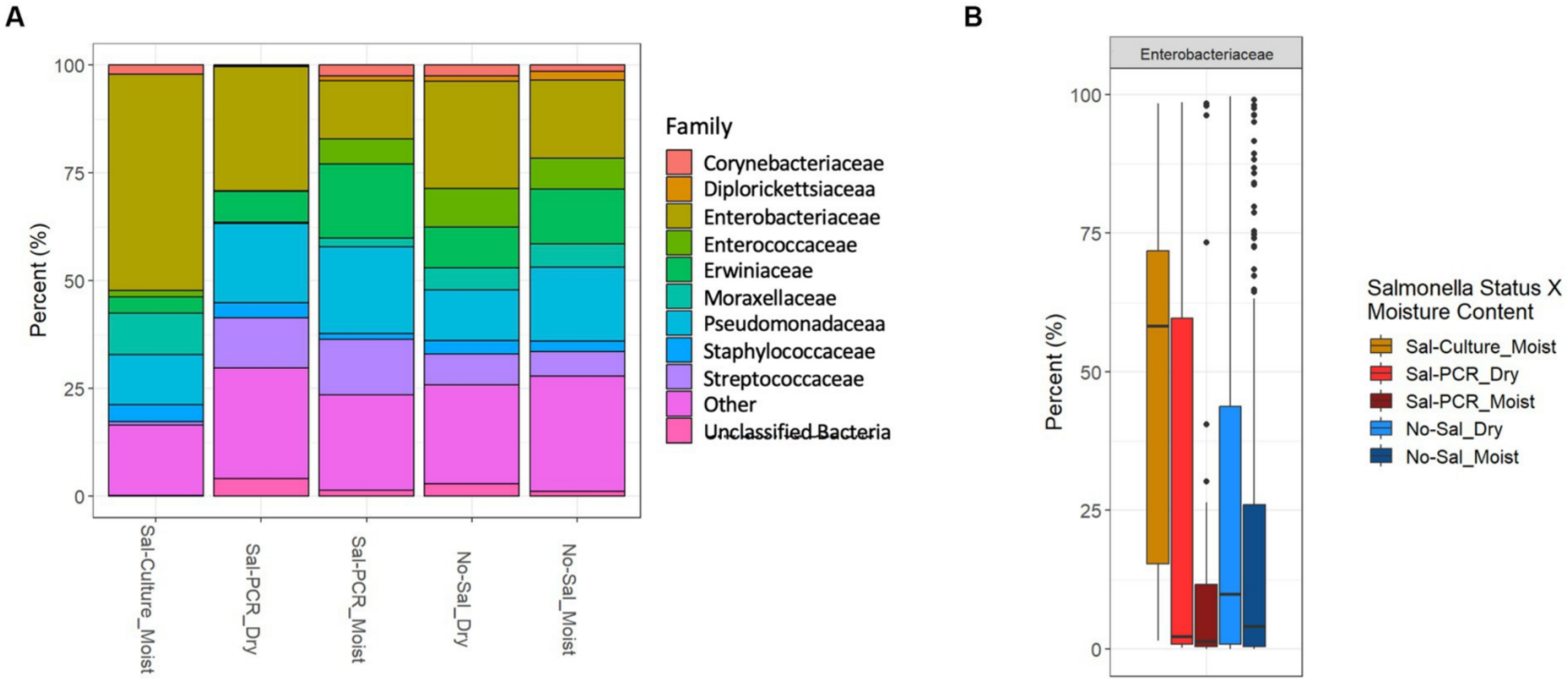
A
100
75
50
25
0
Percent (%)
Sal-Culture_Moist
Sal-PCR_Dry
Sal-PCR_Moist
No-Sal_Dry
No-Sal_Moist
Family
Corynebacteriaceae
Diplorickettsiaceaa
Enterobacteriaceae
Enterococcaceae
Erwiniaceae
Moraxellaceae
Pseudomonadaceaa
Staphylococcaceae
Streptococcaceae
Other
Unclassified Bacteria
B
Enterobacteriaceae
100
75
50
25
0
Percent (%)
Salmonella Status X
Moisture Content
Sal-Culture_Moist
Sal-PCR_Dry
Sal-PCR_Moist
No-Sal_Dry
No-Sal_Moist

Figure 2.2. 16S sequencing of bird feces shows microbial community differences in *Salmonella* culture positive samples. **(A)** 100% bar graph of mean abundances of the 10 most prominent families identified across the entire dataset are displayed when summarized by *Salmonella* group [*Salmonella* culture positive from moist feces (Sal-Culture_Moist), *Salmonella* PCR positive from dry feces (Sal-PCR_Dry), *Salmonella* PCR positive from moist feces (Sal-PCR_Moist), no *Salmonella* from dry feces (No-Sal_Dry), and no *Salmonella* from moist feces (No-Sal_Moist)]. All taxa outside the top 10 taxa are classified as "Other." **(B)** Differential relative abundance boxplots of prominent Enterobacteriaceae are displayed with significantly (Wilcoxon Rank Sum test, adjusted value of $p < 0.05$) different pairwise relationships displayed.

Table 2.3. Bird *Salmonella* isolates are related to isolates from a variety of sources.

Sample ID	Serovar	Farm Collected	Most closely related source type (genomic distance)	Isolate Reference
JSBird1	Typhimurium	F5	Chicken (3)	SRR10883419
JSBird2	Kentucky	F5	Chicken (3)	SRR21413100
JSBird3	Hadar	F1	Turkey (2)	SRR3664900
JSBird4	Hadar	F2	Turkey (2)	SRR3664900
JSBird5	Hadar	F2	Turkey (2)	SRR3664900
JSBird6	Give	F8	River Water (24)	SRR2050944
JSBird7	Give	F18	River Water (24)	SRR2050944
JSBird8	Give	F18	River Water (23)	SRR2050944
JSBird9	Give	F18	River Water (23)	SRR2050944
JSBird10	Hadar	F9	Turkey (2)	SRR3664900
JSBird11	Hadar	F26	Chicken (5)	SRR1122614
JSBird12	Muenchen	F7	No Similarity	N/A
JSBird13	Newport	F7	Human (8)	SRR1646204
JSBird14	Saintpaul	F7	Human (29)	SRR6231044
JSBird15	Newport	F7	Human (7)	SRR1646204
JSBird16	Mississippi	F23	Human (46)	SRR9640338
JSBird17	Newport	F7	Human (5)	SRR16925338
JSBird18	Newport	F7	Human (7)	SRR1646204
JSBird19	Saintpaul	F7	Human (30)	SRR6231044

Newport was identified four times, including two different Newport isolates from the same fecal sample (F7-5). Interestingly, while the closest whole genome match to these isolates was a single human isolate, they were also closely related to a number of serovar Newport isolates collected from surface waters in Georgia in 2011 (Supplementary Figure S2.3). For isolates belonging to serovars Give, Mississippi, Saintpaul, and Muenchen there were no other isolates in Enterobase that aligned closely, which limits assessment of potential sources for these isolates.

Deep serotyping by CRISPR-SeroSeq was performed on 14 samples. Two libraries failed to produce enough sequence reads, despite two attempts, and these both came from dry fecal samples. In total, 13 different serovars were identified (Figure 2.3). In these samples, 71% (10/14) had *Salmonella* populations consisting of multiple serovars, with an average of 2.6 serovars per sample (range, 1–7 serovars per sample). Serovars included Saintpaul (n = 6), Hadar (n = 5), Newport (n = 4), Kentucky (n = 4), Enteritidis (n = 4), Braenderup (n = 4), Give (n = 3), Rubislaw (n = 2), Heidelberg (n = 1), Infantis (n = 1), Muenchen (n = 1), Typhimurium (n = 1), and Mississippi (n = 1). Importantly, serovars Enteritidis, Infantis, and Braenderup, which were in the top 10 serovars found to cause human illness between 2019–2021 (Centers for Disease Control and Prevention, 2022), were always outnumbered by other serovars when they were present (outnumbered serovars have thinner connecting lines in Figure 2.3), and unsurprisingly, we did not isolate these by culture. In congruence with our whole genome sequence analyses, samples collected from the same sites on the same days often contained similar *Salmonella* populations. For example, two of the three fecal samples collected from farm 18 (F18-2,3) had nearly identical *Salmonella* profiles (serovars

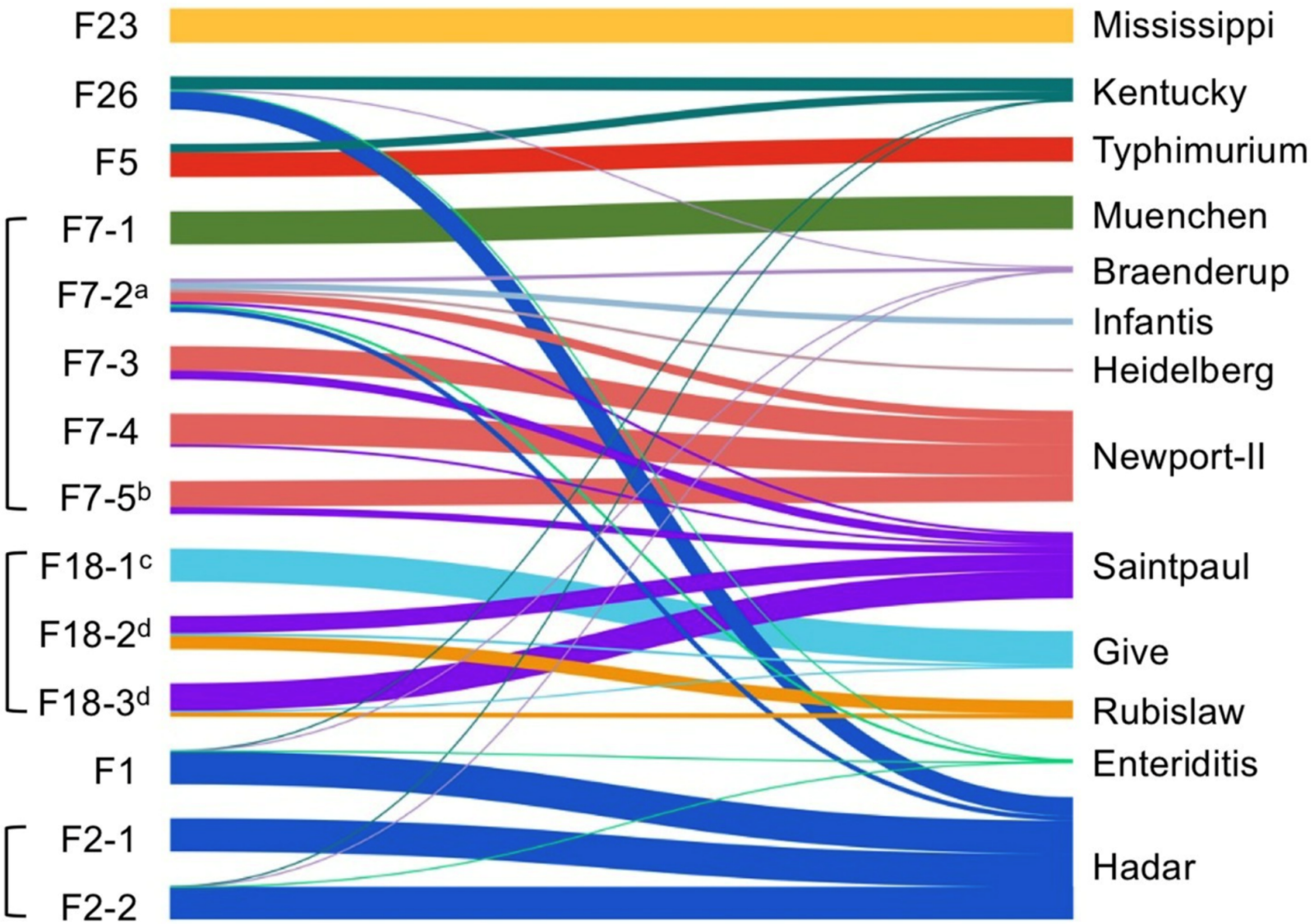
F23
F26
F5
F7-1
F7-2a
F7-3
F7-4
F7-5b
F18-1c
F18-2d
F18-3d
F1
F2-1
F2-2
Mississippi
Kentucky
Typhimurium
Muenchen
Braenderup
Infantis
Heidelberg
Newport-II
Saintpaul
Give
Rubislaw
Enteriditis
Hadar

Figure 2.3. Multiserovar *Salmonella* populations exist in wild bird feces. A Sankey plot showing the sample (left nodes, indicated by the farm where the sample was collected) and the *Salmonella* serovar population within each sample. The colored bars represent different serovars (right nodes) and the thickness of the bars represent the relative abundance of each serovar within a population. Brackets around samples indicate that samples were collected from the same farm on the same day. For samples with a superscript alphabet, we were able to determine the bird species: [a]chipping sparrow, [b]house sparrow, [c]cattle egret, and [d]fish crow.

Saintpaul, Rubislaw, and Give) with respect to the serovars that were present and their relative frequency within each sample.

Point counts were performed at each farm visit and identified 1,123 individuals. This included 51 species, with the most prevalent being the northern cardinal (*Cardinalis cardinalis*) (n = 48 visits where species was observed), the northern mockingbird (*Mimus polyglottos*) (n = 46), and the barn swallow (*Hirundo rustica*) (n = 45) (Table 2.4). A total of 31 species were observed in-field, with the most common being the song sparrow (*Melospiza melodia*) (n = 19), eastern phoebe (*Sayornis phoebe*) (n = 14), northern cardinal (n = 13), chipping sparrow (*Spizella passerine*) (n = 13), and mourning dove (*Zenaida macroura*) (n = 11). Of these, *Salmonella* was detected in four species, including three times from chipping sparrows. Off-field species included the barn swallow (n = 39), northern mockingbird (n = 38), and the Carolina wren (*Thryothorus ludovicianus*) (n = 38). Notably, some species were not often identified, but when present, were found in large numbers. For example, the rock pigeon (*Columba livia*) was only observed during four visits, but 147 individuals were recorded (Supplementary Figure S2.4). Rock pigeons were not in the top ten most frequently observed bird species across this study; however, they were the first and second highest in terms of total individuals off-field and in-field, respectively. Similarly, the common grackle (*Quiscalus quiscula*) was also observed off-field during four visits, but 83 individuals were recorded. Molecular species identification was done via PCR and sequencing of the COI gene was completed on 161 (20.8%) samples. This identified 24 species with the most common being the eastern bluebird (*Sialia sialis*) (n = 36) and the northern mockingbird (n = 19) (Table 2.4). The individuals that were culture-positive for *Salmonella* were a chipping

sparrow, an eastern bluebird, a cattle egret (Bubulcus ibis), a house sparrow (Passer domesticus), and two fish crows (Corvus ossifragus). Because we only identified the bird species in 13 *Salmonella*-positive fecal samples, conclusions based on the *Salmonella* status of specific bird species are limited.

2.5 Discussion

This study investigated the impact of wild birds on food safety by surveying *Salmonella* in wild bird feces deposited on foliage on produce farms over a two-year period in the Southeast. Our study demonstrated that the overall prevalence of culturable *Salmonella* in the Southeast was 2.1%, but this differed greatly between 2021 (6.6%) and 2022 (0.2%). Studies have been completed in other regions include the west coast where *Salmonella* prevalence ranged from 0.5% in cultured fecal samples (Gorski et al., 2011; Franklin and VerCauteren, 2016; Navarro-Gonzalez et al., 2020; Smith et al., 2020) to 2.5% in cultured bird gastrointestinal tracts (Kirk et al., 2002), and the Southwest where one study found a 1.9% prevalence in bird feces (Rivadeneira et al., 2016). Other studies outside of the US have included Europe (Palmgren et al., 2006; Lawson et al., 2011), South America (Cardoso et al., 2021), and the Middle East (Cohen et al., 2021). The overall prevalence identified in the current study aligns with this body of literature. Unlike most studies that sampled fresh feces (i.e., collected directly from a bird), this study offered us the opportunity to evaluate whether *Salmonella* is likely to be recovered from defecated material on foliage. *Salmonella* was isolated by culture and also detected by PCR three times more frequently in moist feces (presumably deposited within a few hours of collection) compared to dry feces. This suggests that *Salmonella* survival in feces is dynamic and the population reduces as the feces dry. While prior work has shown

Table 2.4. Bird species identified by bird counts and by molecular analysis.

Species	In field		Off field		Total species observations	Molecular observations (COI)	*Salmonella* culture	*Salmonella* PCR	Total *Salmonella* positive
	Number of species observations	Total number of individuals	Number of species observations	Total number of individuals					
Song sparrow (*Melospiza melodia*)	19	33	6	8	25	0	0	0	0
Eastern phoebe (Sayornis phoebe)	14	16	21	23	35	5	0	1	1
Northern cardinal (*Cardinalis cardinalis*)	13	25	35	41	48	14	0	1	1
Chipping sparrow (*Spizella passerina*)	13	16	22	32	35	9	1	2	3
Mourning dove (*Zenaida macroura*)	11	67	26	44	37	9	0	1	1
Northern mockingbird (*Mimus polyglottos*)	8	13	38	47	46	19	0	0	0
Eastern bluebird (*Sialia sialis*)	7	11	15	25	22	36	1	0	1
Barn swallow (*Hirundo rustica*)	6	18	39	59	45	0	0	0	0
Killdeer (*Charadrius vociferus*)	6	6	2	4	8	0	0	0	0
Red-winged blackbird (*Agelaius phoeniceus*)	5	5	4	7	9	7	0	0	0
House finch (*Haemorhous mexicanus*)	4	8	19	56	23	11	0	1	1
Eastern meadowlark (*Sturnella magna*)	4	4	2	3	6	0	0	0	0
Indigo bunting (*Passerina cyanea*)	4	4	0	0	4	10	0	1	1
American Crow *(Corvus brachyrhynchos)*	3	8	16	29	19	12	0	0	0
European collared dove (*Streptopelia decaocto*)	3	4	2	2	5	0	0	0	0
American goldfinch (*Spinus tristis*)	3	3	4	4	7	0	0	0	0
Rock pigeon (*Columba livia*)	2	45	2	102	4	0	0	0	0
Cattle egret (*Bubulcus ibis*)	2	13	2	5	4	1	1	0	1
Carolina wren (*Thryothorus ludovicianus*)	2	3	38	46	40	0	0	0	0
Field sparrow (*Spizella pusilla*)	2	3	6	6	8	1	0	0	0
Eastern kingbird (*Tyrannus tyrannus*)	2	3	1	1	3	2	0	0	0
Blue jay (*Cyanocitta cristata*)	2	2	20	25	22	0	0	0	0
Tufted titmouse (*Baeolophus bicolor*)	2	2	9	9	11	0	0	0	0
Ruby-throated hummingbird (*Archilochus colubris*)	2	2	0	0	2	0	0	0	0
Eurasian collared dove (*Streptopelia decaocto*)	1	6	0	1	1	0	0	0	0
European starling (*Sturnus vulgaris*)	1	3	4	16	5	3	0	0	0
Common ground dove (*Columbina passerina*)	1	2	1	2	2	0	0	0	0
White-eyed vireo (*Vireo griseus*)	1	1	10	10	11	0	0	0	0
Blue grosbeak (*Passerina caerulea*)	1	1	7	7	8	7	0	0	0
Brown thrasher (*Toxostoma rufum*)	1	1	5	5	6	0	0	0	0
Downy woodpecker (*Picoides pubescens*)	1	1	5	5	6	0	0	0	0
Eastern towhee (*Pipilo erythrophthalmus*)	0	0	11	13	11	0	0	0	0
Red-bellied woodpecker (*Melanerpes carolinus*)	0	0	7	9	7	0	0	0	0
Carolina chickadee (*Poecile carolinensis*)	0	0	5	12	5	0	0	0	0
Chimney swift (*Chaetura pelagica*)	0	0	5	11	5	1	0	0	0
Common grackle (*Quiscalus quiscula*)	0	0	4	83	4	0	0	0	0
Red-shouldered hawk (*Buteo lineatus*)	0	0	4	6	4	0	0	0	0
Pine warbler (*Setophaga pinus*)	0	0	3	3	3	0	0	0	0
Black vulture (*Coragyps atratus*)	0	0	2	9	2	0	0	0	0
American robin (*Turdus migratorius*)	0	0	2	6	2	0	0	0	0
American kestrel (*Falco sparverius*)	0	0	2	2	2	0	0	0	0
Brown-headed nuthatch (*Sitta pusilla*)	0	0	2	2	2	0	0	0	0
Painted bunting (*Passerina ciris*)	0	0	2	2	2	1	0	0	0
Yellow-throated vireo (*Vireo flavifrons*)	0	0	2	2	2	0	0	0	0
Northern rough-winged swallow (*Stelgidopteryx serripennis*	0	0	1	4	1	0	0	0	0
House sparrow (*Passer domesticus*)	0	0	1	1	1	5	1	0	1
Northern parula (*Setophaga americana*)	0	0	1	1	1	0	0	0	0
Pileated woodpecker (*Dryocopus pileatus*)	0	0	1	1	1	0	0	0	0
Red-tailed hawk (*Buteo jamaicensis*)	0	0	1	1	1	0	0	0	0
Tree swallow (*Tachycineta bicolor*)	0	0	1	1	1	0	0	0	0
White-breasted nuthatch (*Sitta carolinensis*)	0	0	1	1	1	0	0	0	0
Blue-gray gnatcatcher (*Polioptila caerulea*)	0	0	0	0	0	1	0	0	0
Brown-headed cowbird (*Molothrus ater*)	0	0	0	0	0	2	0	0	0
Eastern wood pewee (*Contopus virens*)	0	0	0	0	0	1	0	0	0
Fish crow (*Corvus ossifragus*)	0	0	0	0	0	2	2	0	2
Great crested flycatcher (*Myiarchus crinitus*)	0	0	0	0	0	1	0	0	0
Summer tanager (*Piranga rubra*)	0	0	0	0	0	1	0	0	0
TOTAL	146	329	419	794	565	161	6	7	13

Salmonella can survive in feces up to 291 days (Topalcengiz et al., 2020) and can have improved survival in low moisture environments (Oni et al., 2015), these studies were performed in controlled laboratory experiments and do not necessarily reflect conditions in a produce field. The fecal samples we collected had a much larger surface area to volume ratio and, therefore, are likely to dry out faster than homogenized laboratory samples.

Our statistical models suggest that precipitation the day before sampling positively influences the moisture of wild bird fecal samples, which is expected. Comparison of precipitation during sampling months in both years supports this relationship, with lower precipitation in 2022 than in 2021 likely accounting for decreased moisture and therefore a reduced *Salmonella* recovery. One model included a negative correlation between increased wind and moist feces, which is also expected as increased wind would dry the feces more rapidly. Alternatively, humidity showed a negative influence on fecal moisture in three different models. This seems counterintuitive; however one study of *Salmonella* survival in a controlled environment also saw a negative association between humidity and pathogen recovery from turkey feces (Oni et al., 2015).

Most studies of *Salmonella* in wild birds have involved capturing birds and collecting fresh feces or swabbing the cloaca (Gorski et al., 2011; Hernandez et al., 2016; Navarro-Gonzalez et al., 2020; Murray et al., 2021) while others have applied molecular techniques (i.e., PCR) to identify *Salmonella* in bird feces (Rivadeneira et al., 2016; Smith et al., 2020; Zhao et al., 2020; Olimpi et al., 2022). PCR is a very sensitive method for pathogen detection, and we detected nearly five times as many *Salmonella*-positive

fecal samples when we used PCR compared to culture. *Salmonella* has been shown to be detectable by PCR up to 10 days after inoculation; however, significant reduction occurs after four days (Lopez-Velasco et al., 2015). There are three possibilities that could explain the discrepancy between the culture results and the PCR results: (i) PCR can detect dead *Salmonella*; (ii) PCR can detect viable but non-culturable (VBNC) *Salmonella*; and (iii) because PCR is more sensitive than culture, it is possible that where the amount of *Salmonella* in the feces was very low, we were not able to recover it from culture but could detect it by PCR. We note that the background microflora was not particularly high in the selective enrichment broths nor on the XLT-4 plates, so we do not suspect that this contributed to not being able to detect *Salmonella* via our culture methods. We did attempt to serotype the PCR-detected *Salmonella* using the ISR method (Guard et al., 2022) to determine whether there were any serovar associations with PCR versus culture, but we were unsuccessful. The *Salmonella* detected by culture may have been present in higher loads, which allowed us to isolate it more easily, although we did not quantify *Salmonella*. Whether the PCR-only positive samples represent VBNC cells and pose a food safety risk should be a focus of future studies, especially as PCR-based diagnostic assays are more commonly being used to screen food products.

In addition to completing a surveillance study, this work also assessed the need for and efficacy of no-harvest buffer zones around feces in a production environment (Hamilton et al., 2015). While produce directly contacting feces cannot be harvested, the Produce Safety Rule does not require the establishment of no-harvest buffer zones, nor does it recommend suggested distances surrounding contaminated produce to exclude from harvest. Depending on the recommended buffer zone radius and the impacted

commodity type, buffer recommendations could have a substantial economic impact on growers and could be excluding produce that is safe for consumption. *Salmonella* was not isolated from additional plant samples below foliage with fecal contamination nor from neighboring plants downwind. However, depending on the weather or other climate factors, the rate at which feces dry on the plant surface may vary; this may be important to consider since our data shows that culturable *Salmonella* is primarily present in moist feces. The low incidence of *Salmonella* in bird feces and the lack of evidence supporting spread to adjacent plants in this study may be useful data for growers as they establish procedures for managing bird feces before and during harvest.

Alongside determining *Salmonella* prevalence in bird fecal samples, a deeper analysis was conducted into individual *Salmonella* isolates and serovar populations. Previous work has shown a high level of diversity within bird feces, including identifying as many as three serovars of *Salmonella* from a single sample (Antilles et al., 2021). Our culture-based analysis supported this high diversity by identifying eight serovars among 19 isolates. High-resolution analysis by deep serotyping revealed even great serovar diversity, by detecting 13 serovars across 14 samples. Further, we showed that 62.5% of culture positive samples contain multiple serovars, which included one fecal sample that contained seven different serovars (F7-2). Six serovars identified here (serovars Enteriditis, Newport, Typhimurium, Infantis, Saintpaul, and Muenchen) were determined by the Centers for Disease Control (CDC) to be among the top 10 serovars associated with human illness between 2019–2021 (Centers for Disease Control and Prevention, 2022). Additionally, serovars Hadar, Heidelberg, and Braenderup have all been linked to human outbreaks in produce or animal products in the past ten years (CDC, 2023) and

were also found in our samples. Importantly, serovars Enteritidis and Braenderup were each found in four different fecal samples and in each of these, they were significantly outnumbered by other serovars that are not known to be associated with human foodborne illness. For example, in one sample (F2-2), serovar Hadar constituted 95.9% of the total *Salmonella,* and serovar Enteritidis was only 0.1%. As our results demonstrated, using traditional culture-based *Salmonella* isolation, serovars Enteritidis and Braenderup were never detected, indicating that these important serovars were overlooked. From the five serovar Hadar isolates identified, four were closely related (within the same hierCC 2 cluster on Enterobase), to isolates from commercial turkeys (Supplementary Figure S2.1B). It should be noted that there is no commercial turkey production within at least 200 miles of the location of these farms and the turkey isolates were from 2012–2016. The fifth serovar Hadar isolate was related (within the same hierCC 5 cluster) to a chicken isolate, though that isolate was collected in 2015 from Oregon. While chicken production in the southeast is well established, further research is needed to determine whether and how wild birds acquire *Salmonella* from commercial poultry operations (e.g., from foraging on poultry farms, or from encountering contaminated poultry manure on produce farms). Interestingly, the four serovar Newport isolates most closely matched to human isolates; however, they were also closely related to isolates collected from fresh water sources in Georgia (Supplementary Figure S2.2). Two different Newport isolates came from the same fecal sample (F7-5), where one was identified from each selective enrichment broth, indicating there is also strain diversity within single fecal samples. Deep serotyping showed that samples collected from the same farm often had similar *Salmonella* serovar populations in addition to closely related isolates, suggesting that

similar sources of *Salmonella* may occur in the environment that contribute to contamination in wild birds, or that a single bird was defecating multiple times in the same field. Alternatively, for birds that flock together (e.g., crows), this similarity may reflect transmission within a flock, for example at common feeding or watering locations. Overall, our findings indicate that wild birds have the potential to obtain and transmit *Salmonella* from a wide range of sources over large geographic areas.

Bird species were identified in this study using both physical and molecular methods. Other studies have used a more direct collection approach where birds are caught using nets or traps followed by the collection of feces or swabbing the cloaca (Gruszynski et al., 2014; Fuentes-Castillo et al., 2019; Navarro-Gonzalez et al., 2020). In these instances, bird species can be identified quite easily, and the sample is fresher. Alternatively, the collection method used in this study resulted in lower molecular characterization of bird species (20.8% identified). However, it was non-invasive and provided an opportunity to investigate bird species actively defecating on the field, not just those primarily foraging in adjacent habitats. Data collected in this study identified 51 species of bird from point counts and 24 species from COI, for a total of 57 species. Molecular detection from bird feces allowed for the identification of six additional species, including the fish crow, which was identified in two *Salmonella* culture positive samples but not identified during point counts. This demonstrates the importance of the two complementary methods for bird identification.

We categorized birds from our point counts as in-field or off-field. The off-field category included species that are often associated with agricultural structures (e.g., barns, packing houses, fences) or other structures (e.g., powerlines adjacent to the farm),

such as the barn swallow (n = 39 species observations off-field), house finch (*Haemorhous mexicanus*) (n = 19), European starling (*Sturnus vulgaris*) (n = 4), and rock pigeon (n = 2). Of these, only a single house finch fecal sample tested positive for *Salmonella*. Although rock pigeons were observed twice off-field, the total number of individuals was 102, suggesting that flock size may also be relevant with respect to understanding the risk posed by different species. The off-field category also included birds found away from the farm premises (e.g., in tree line, neighboring pasture) and included the Carolina wren (n = 26), woodpecker (n = 12), white-eyed vireo (*Vireo griseus*) (n = 8), and eastern towhee (*Pipilo erythrophthalmus*) (n = 3). This latter group poses the lowest risk of pathogen transmission because they are infrequently observed interacting with produce. Although we were not able to identify the bird species for the majority of *Salmonella*-positive fecal samples, of the ones we were able to identify, none belonged to this category. Conversely, birds on agricultural structures and in-field pose a higher food safety risk because of their interactions with farm livestock and produce, so deterrents targeting these species would be more effective. Three-quarters (10/13) of *Salmonella*-positive fecal samples were from birds that were also observed as in-field during point counts. For the three that were not observed, one was a cattle egret and the other two were fish crows. Interestingly, fish crows are associated with water and both had *Salmonella* serovars Give and Rubislaw (F18-2, -3), which are associated with surface water (Haley et al., 2009; Gorski et al., 2011; McEgan et al., 2014; Maurer et al., 2015; Callahan et al., 2019; Deaven et al., 2021).

Mitigating risks associated with wild birds in produce fields remains a complicated issue that will require a One Health approach to fully understand how the

interaction of animals (including wildlife, such as birds, and food animals), the environment, and human activity contribute to *Salmonella* ecology. In this work, we found a low prevalence of *Salmonella*, however; serovars associated with human illness were often identified when *Salmonella* was present. Moreover, the prevalence increased from 2% to over 9% when molecular detection was included, suggesting that different methods of detection can influence the establishment of risk due to this environmental source of *Salmonella*. The complexity of this problem is highlighted by our whole genome analysis showing that *Salmonella* isolates recovered in this study were related within 10 pairwise allelic differences (PADs) to isolates from a range of sources including humans, animal agriculture, and the environment, as well as some without any links to these sources. The freshness of the wild bid feces was shown to impact viability of *Salmonella*; however, more work will need to be completed to show how risk of feces changes with time and if certain serovars are better adapted to this environment. While factors affecting the prevalence of *Salmonella* within wild birds and *Salmonella* survival within feces are not fully understood, the findings presented here contribute to our understanding of these complex food safety systems.

2.6 Data availability statement

The datasets presented in this study can be found in online repositories. The names of the repository/repositories and accession number(s) can be found in the article.

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2.9 Conflict of interest

The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

2.10 Author Contributions

JCS: Data curation, Formal analysis, Investigation, Writing – original draft, Writing – review & editing, Visualization. SV: Data curation, Formal analysis, Investigation, Writing – review & editing. KR: Resources, Writing – review & editing. ZS: Resources, Writing – review & editing. JLD: Resources, Writing – review & editing. JS: Resources, Writing – review & editing. LLD: Conceptualization, Funding acquisition, Investigation, Methodology, Project administration, Resources, Supervision, Writing – review & editing. WES: Conceptualization, Funding acquisition, Investigation, Methodology, Project administration, Supervision, Writing – review & editing. NWS: Conceptualization, Data curation, Formal analysis, Funding acquisition, Investigation, Methodology, Project administration, Supervision, Writing – original draft, Writing – review & editing.

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CHAPTER 3

A NEED FOR INCREASED ENVIRONMENTAL SURVEILLANCE TO CLOSE THE ONE HEALTH LOOP[1]

[1]Jared C. Smith, Amy T. Siceloff, Sherwin M. Shirazi, Rebecca L. Bell, Nikki W. Shariat. 2025. To be submitted to a peer-reviewed journal. JCS, ATS, and NWS conceived and planned the study.

3.1 Abstract

Salmonella enterica is a leading cause of bacterial foodborne illness, often transmitted through contaminated food and water. Considerable reductions of *Salmonella* contamination in meat and poultry products do not wholly contribute to decreased foodborne illness, highlighting the need to define alternative reservoirs and transmission pathways. In this study, we collected samples from four distinct creeks over 24 months to characterize *Salmonella* serovar diversity and utilized phylogenetic approaches, along with proximal land use analyses, to identify relationships between environmental reservoirs and hosts. Across 19 sites, including animal agriculture, suburban, and forested areas, 10L water samples were collected using modified Moore swabs (n = 456), and cultured for *Salmonella*, followed by whole genome sequencing of isolates and deep serotyping of multiserovar populations. Overall prevalence was 69% (314/456), and generalized linear mixed models showed that seasonal weather patterns, including precipitation and humidity, significantly influenced recovery and complexity in comparison to surrounding land use. Antimicrobial resistance was detected in 11% (33/314) of isolates, with 21% (7/33) classified as multidrug resistant. CRISPR-SeroSeq identified 37 serovars, and multiserovar populations were detected in 89% (229/258) of positive samples with sequencing data, averaging 3.7 serovars per sample (range: 1-13). Comparison with national food animal production monitoring showed limited serovar overlap, with serovar Rubislaw dominating water samples but absent in agricultural datasets. Collectively, these results demonstrate extensive serovar diversity within *Salmonella* populations in freshwater systems, including clinically relevant serovars, and

emphasize the need to develop a robust surveillance platform for source attribution and, ultimately, prevention of future outbreaks.

3.2 Significance statement

Contaminated surface water significantly contributes to global *Salmonella* illnesses, marking a critical need to assess serovars present and determine environmental variables affecting the population dynamics in this reservoir. We found that complex multiserovar populations, often including pathogenic serovars, occur in surface water regardless of proximal land use. Notably, many aquatic serovars are not detected in animal agriculture monitoring. However, limited serotyping data is available for alternative reservoirs of foodborne illness, namely wildlife, which hinders source attribution. Phylogenetic analysis revealed aquatic antimicrobial-resistant Infantis and Typhimurium isolates were more closely related to clinical than animal-source isolates. This study highlights a significant gap in understanding environmental *Salmonella* transmission and underscores the importance of a One Health surveillance approach to protect public health.

3.3 Introduction

The WHO reports that 1.4 million deaths worldwide could be prevented by improving water safety measures and reducing the load of contaminating bacteria (WHO, 2023). Notably, *Salmonella enterica* subsp. *enterica* is one of four key global causes of diarrheal diseases, and serovar Typhi alone contributes an estimated 9 million illnesses and 110,000 deaths through the consumption of water contaminated with fecal material (https://www.who.int/news-room/fact-sheets/detail/typhoid). While typhoidal *Salmonella* illnesses are uncommon in the United States (CDC, 2024), there are an estimated 1.35

million non-typhoidal illnesses each year, resulting in 26,500 hospitalizations, 420 deaths (CDC, *Salmonella*, 2024), and a total cost of $4.1 billion (U.S. Department of Agriculture (USDA), Economic Research Service (ERS). Cost Estimates of Foodborne Illnesses). Non-typhoidal *Salmonella enterica* subsp. *enterica,* hereafter *Salmonella,* is also a contributing microorganism to the global threat of antibiotic resistance, as several resistant serovars have been affecting the food chain (WHO). However, 60-80% of salmonellosis cases are not connected to a known outbreak, which underscores a critical lack of knowledge needed to prevent future illnesses (WHO). Final products from the four main food animals in the United States (beef, chicken, pork, turkey) are routinely tested by the United States Department of Agriculture – Food Safety and Inspection Service (USDA-FSIS). In response to an ever-increasing number of related foodborne outbreaks, produce is also screened for pathogens in the U.S. Notably, an estimated 43.9% of *Salmonella* illnesses in the United States can be attributed to the consumption of contaminated produce, while 42.7% can be attributed to meat and poultry (IFSAC, 2024). These surveillance programs are necessary to protect consumers but also widen the knowledge gap of *Salmonella* transmission as wildlife and other environmental contributors to contamination in food production are not monitored. For example, contaminated water is believed to be a significant contributor to disseminating foodborne pathogens in produce (Bell et al., 2021). Most recently in cucumbers, serovars Braenderup and Africana infected 551 individuals, where both serovars were found in the agricultural water at the farms where the outbreaks originated (FDA, 2024). It is likely that contaminated irrigation water contributes to many produce-related outbreaks but these traceback investigations do not often result in exact sources due to the consumption

or spoilage of products, along with the clearing of fields following harvesting. Additionally, produce contamination may occur both in the field and in packinghouses, further increasing the difficulty of attributing sources of *Salmonella*. Because meat, poultry, and egg surveillance, along with clinical isolates, comprise the majority of publicly available data, any large-scale genomic attribution studies are skewed towards animal agriculture. Subsequently, the risk is not properly assessed for serovars arising from alternative sources, such as wildlife or contaminated water.

There are over 2,600 *Salmonella* serovars, characterized by their combination of somatic (O) and flagellar (H) antigens (Grimont and Weill, 2007; Issenhuth-Jeanjean et al., 2014). Significant phenotypic and genotypic plasticity can be found across serovars, including host restriction (Uzzau et al., 2000), antimicrobial resistance (Shah et al., 2016), virulence factors (Cheng et al., 2019), and stress response (Gorski and Noriega, 2023). Most serovars are host generalists or host-adapted, which can lead to patterns of serovar isolation from the same sources repeatedly. While some wildlife reservoirs are often found with host-adapted serovars or strains, such as serovar Cholerasuis in wild boars and some subtypes of serovar Typhimurium circulating in wild birds (Uelze et al. 2021; Patel et al. 2023; Hernandez et al. 2016; Chiu et al., 2002; Cohen et al., 2021), serovar association from sources such as deer (Renter et al., 2006; Salas-Rosas et al. 2020; Topalcengiz et al. 2020), reptiles (Doden et al. 2021), raccoons (Very et al. 2016; Maurer et al. 2015), and other wildlife (Gorski et al. 2011; Millan et al. 2004; Maurer et al., 2015) are largely understudied. Additionally, wild animals are recognized as carriers of antimicrobial resistance (AMR), so identifying points of introduction and transmission

within the environment is critical to understanding the public health risk posed by wildlife activity. (Carroll et al., 2015; Ramey et al., 2020; Laborda et al., 2022).

In addition to its presence in a variety of animal hosts and surface water, *Salmonella* is often found in dust and soil as well (Whyte et al., 2003; Pal et al., 2021; Jacobsen and Bech, 2012; Jechalk et al., 2019; Bell et al., 2015; Deaven et al., 2021; Gorski et al., 2022; Haley et al., 2009). As an enteric pathogen, *Salmonella* likely enters these abiotic environments via fecal contamination from humans, wildlife, and animal agriculture (Liu et al., 2018; Santo Domingo et al. 2000; Abulreesh, 2012) and may be maintained following continual re-introduction or via persistence (Byappanahall et al., 2009; Sha et al., 2013; Gaertner et al., 2011; Brandl et al., 2005; Bell et al., 2015; Li et al., 2015). As *Salmonella* often exists as mixed serovar populations (Thompson et al., 2018; Obe et al., 2023; Siceloff et al., 2022), surface water may serve as a conduit for highly complex populations following contamination from multiple hosts (Deaven et al., 2021; Gorski et al., 2011; Maurer et al., 2015b; Micallef et al., 2012). *Salmonella* has been shown to survive at least 300 days in a freshwater source (Topalcengiz et al., 2019), indicating that, while growth may be limited, survival for extended periods is possible. Previous research (Haley et al., 2009; McEgan et al. 2013; Murphy et al., 2022; Weller et al., 2020; Truitt et al., 2018; Deaven et al., 2021) has indicated that meteorological variables, including temperature and precipitation, can influence *Salmonella* recovery from surface water.

Conventional *Salmonella* isolation by culture-based methods can be quite sensitive; however, previous work has demonstrated that the use of selective enrichment media, is necessary for *Salmonella* recovery but leads to skewed population proportions

(Gorski et al., 2024). While media bias can result in discordance between true and observed proportions, traditional culturing has the advantage of recovering isolates that can then be subtyped. Molecular approaches such as rapid detection through qPCR can identify positive samples quickly; however, no strain analysis for outbreak traceback is possible using this method. As an alternative approach, deep serotyping using CRISPR-SeroSeq (serotyping based on the sequences of native *Salmonella* CRISPRs) is an amplicon-based next-generation sequencing approach that profiles the relative frequency of multiple *Salmonella* serovars in a single sample (Thompson et al. 2018). Applying this method to *Salmonella*-positive surface water samples has shown that rivers harbor complex *Salmonella* populations (Deaven et al., 2021). At the time, this was attributed to the large size of the watershed (Susquehanna R. watershed, Pennsylvania), and that the land use in that region was diverse. The current study was designed to apply this methodology to gain an improved representation of the *Salmonella* population within a watershed, such that smaller creeks were sampled, and to compare serovar differences between land that was selected for one primary use (i.e., human activity, animal agriculture, or national forest).

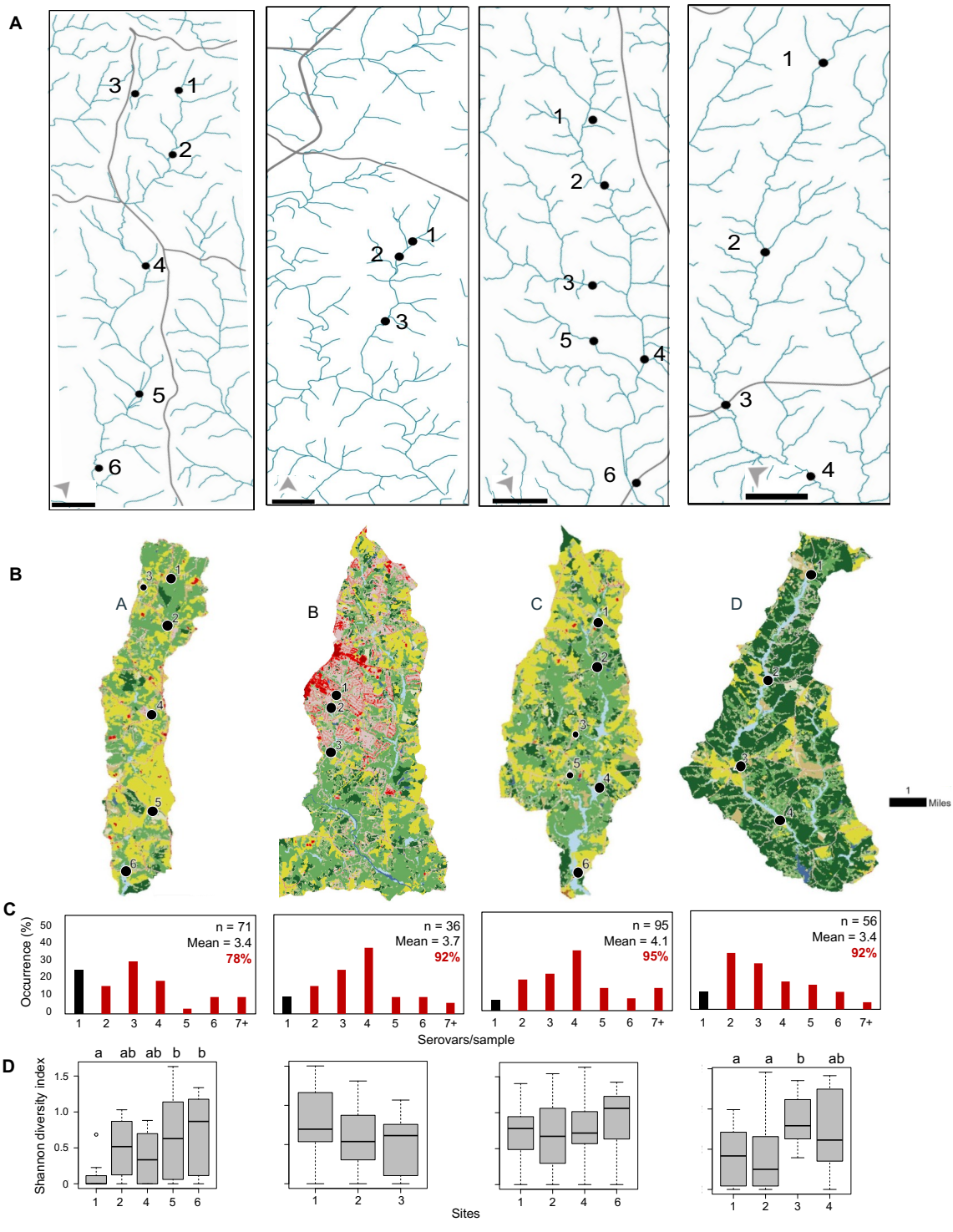
In this study, we sought to reveal the prevalence, population complexity, and antimicrobial resistance of non-typhoidal *Salmonella* within diverse freshwater aquatic environments representing four distinct creeks in the southeastern United States. Additionally, we investigated the influence of proximal land-attribution and weather on *Salmonella* in these systems. We utilized a total of 19 collection sites across the four creeks, where 10L water samples were collected from each site monthly over two years (November 2021 – October 2023; n = 456). From each sample, *Salmonella* was isolated

by traditional culturing techniques followed by a deep sequencing method of serotyping to characterize serovar populations. Additionally, antimicrobial resistance testing was performed. Generalized linear mixed models (GLMMs) were developed based on *Salmonella* prevalence and population complexity, weather patterns, and land-attribution data to determine significant contributors to *Salmonella* incidence and complexity as a risk assessment tool.

3.4 Results

Over 24 months (November 2021-October 2023), water samples were collected at 19 sites across four creeks using modified Moore swabs (Figure S3.1A). Each creek was selected for a different prominent land use, including animal agriculture (Creek A and C), a suburban community (B), and a national forest (D) (Figure 3.1A). In total, 69% (314/456) of samples were *Salmonella*-positive (Figure S3.1B). Prevalence differed across the creeks ($p < 0.05$, Chi-squared Test) and was highest in Creek C (78%; 112/144), and lowest in Creek A (63%; 91/114); both systems were adjacent to animal agriculture. Prevalence also differed by season ($p < 0.001$, Chi-squared Test), being highest in Spring (93%; 106/114) and lowest in Summer (47%; 54/114). Differences in *Salmonella* recovery were observed across culturing methods and fluctuated based on the season as well (Figure S3.1B).

A generalized linear mixed model (GLMM) was used to collectively analyze weather and land attribution data as well as the non-linear and interaction effects between and among variables to determine the likelihood of *Salmonella* detection and population complexity (Tables S3.1, S3.2). The models used fall as the baseline season (intercept), with the other seasons reported in relation to fall, and resulted in significance values and


A
B
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Miles
n = 71
Mean = 3.4
78%
n = 36
Mean = 3.7
92%
n = 95
Mean = 4.1
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n = 56
Mean = 3.4
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Serovars/sample
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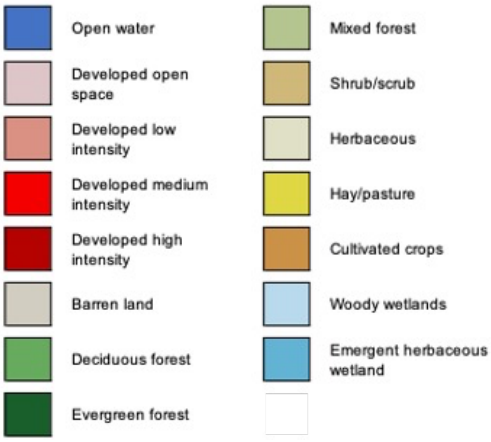


Figure 3.1. Sampling sites, multiserovar prevalence, and diversity of creeks. A) Each creek is shown with its corresponding land attribution from the national land cover database. Individual sites are shown as black circles with numbers representing each site. "Branching" sites off the main creek stem are shown as smaller black circles. B) Results of deep serotyping reveal the occurrence of multiserovar populations (red bar) compared to single-serovar populations (black bar). The number of positive samples, average serovars/sample, and percent of multiserovar populations for each creek are included. C) Shannon diversity indices of sites along the main stem of the creek, where lowercase letters show significant differences between sites using a Kruskal-Wallis and Dunn post-hoc test.

odds ratios (OR) to describe interactions between variables tested and *Salmonella* recovery. The likelihood of *Salmonella* detection was similar in winter and fall ($p > 0.05$), then higher in spring ($p < 0.05$, OR = 2.09, CI 1.26-3.48) and summer ($p < 0.05$, OR = 0.06, CI 0.04-0.01). Wind speed ($p < 0.05$, OR = 1.21, CI 1.1-1.32) and precipitation ($p < 0.05$, OR = 1.21, CI 1.13-1.29) corresponded with increased likelihood of detection, along with the presence of hay pasture ($p < 0.05$, OR = 1.03, CI 1.02-1.05) and mixed forest ($p < 0.05$, OR = 1.12, CI 1.08-1.17) as surrounding land. Similar results were obtained when modeling for serovar complexity, except summer ($p > 0.05$, OR = 0.60, CI 0.3-1.21) was not a significant predictor while winter was ($p < 0.05$, OR = 0.48, CI 0.26-0.87). Additionally, humidity ($p < 0.05$, OR = 1.06, CI 1.03-1.08) was found to increase the likelihood of complex *Salmonella* populations. Spring, as defined by sampling month, was the greatest predictor of *Salmonella* prevalence and complexity. The relationship between weather variables was explored as well using the Pearson correlation coefficient and revealed a strong association between maximum temperature and minimum temperature ($r = 0.89$), along with radiation ($r = 0.75$) (Figure S3.2).

From the antimicrobial susceptibility testing, 11% (33/314) of *Salmonella* isolates displayed AMR phenotypes, and 21% (7/33) of these were classified as multidrug resistant (MDR) (Figure S3.3). The most common resistance was to streptomycin (29/33, 88%), and one serovar Saintpaul isolate was resistant to seven antibiotics. These AMR isolates represented 15 serovars, as determined by whole genome sequencing, with serovar Anatum as the most predominant (6/33 isolates), comprising the majority of the MDR isolates (5/7). Out of the 15 serovars, seven are of human clinical importance as

denoted by their presence in the top 15 serovars listed on the BEAM Dashboard (CDC, 2024), including serovar Saintpaul which was MDR.

CRISPR-SeroSeq was applied to all *Salmonella*-positive samples. A subset of these failed to amplify (n = 56/314), though the majority of these samples (45/56, 80%) were only positive in non-selective enrichment media (BPW). Multiserovar populations were identified in 89% (229/258) of samples, with an average of 3.7 serovars per sample (range 1-13 serovars) and a total of 37 serovars detected (Figure S3.4). On average, Creek C had the highest number of serovars per sample (4.1) and the most samples containing multiple serovars (90/95, 95%), while Creek A had the lowest (3.4; 55/71, 77%) (Figure 3.1B). Concordant with *Salmonella* prevalence, the greatest number of multiserovar samples were detected in spring (81/86, 94%), with significant differences in complexity when comparing based on creek (C to A), and season (Spring to Fall and Winter) ($p < 0.05$, Kruskal-Wallis test with Dunn post-hoc). Additionally, the Shannon diversity indices of *Salmonella* populations across sites following the main stem of each creek revealed that serovar complexity increased in downstream collection sites within creeks A and D (Figure 3.1C).

When comparing all serovars isolated from at least five samples across the whole dataset, serovars Give I, Muenchen I, Rubislaw, and Typhimurium were shared amongst all creeks (Figure S3.5A). Alternatively, some serovars were unique to a creek, such as serovars Agbeni and Oranienburg in Creek B and Anatum in Creek C. All serovars identified in more than five samples from Creek D were also found in other creeks. Creek A contained the greatest number of serovars (n = 30), while Creek D contained the least (n = 18). Additionally, differences in populations between sampling events were

analyzed by comparing the populations with a principal coordinates analysis based on Jaccard distance in each sample. This analysis showed that *Salmonella* populations were most consistent in Creek A, identified by measuring the average distance to the centroid point within each creek (Figure S3.5B). The most common serovar in creeks A, B, C, and D were Give I (n = 32), Rubislaw (n = 23), Montevideo II (n = 72), and Rubislaw (n = 46), respectively. The relative abundances of these serovars differ over time and within creeks, as one serovar may be highly abundant in one site and then lower in the next site (Figure 3.2). For example, serovar Infantis was almost exclusively isolated from sites 4 – 6 in Creek A. In Creek C, serovar Agbeni was only found at site 2 in the first six months of *Salmonella*-positive samples. Of the serovars identified 10 or more times across the creeks, Infantis, when present, comprised the largest average relative frequency (45.1%) within a sample. This was followed by Montevideo II and Braenderup at 42.1% and 40.7%, respectively. Serovar Rubislaw, although present in the most samples, had an average proportion of 32.6%.

USDA-FSIS conducts regular surveillance of animal agriculture systems, including poultry (turkey and chicken), pork, and beef. Utilizing current and historical data going back to 2016, with the exception of pork which only included datasets from 2019 to present day, the top 10 serovars by prevalence were identified for each commodity group and compared to water samples (Figure 3.3). The reported serovars for the former dataset do not include polyphyletic designations. The most commonly identified serovar for turkey, chicken, pork, and cattle was Reading (24%), Kentucky (33%), Anatum (18%), and Montevideo (23%), respectively. In contrast, serovar Rubislaw was most often found within the creek samples (22%), which contained

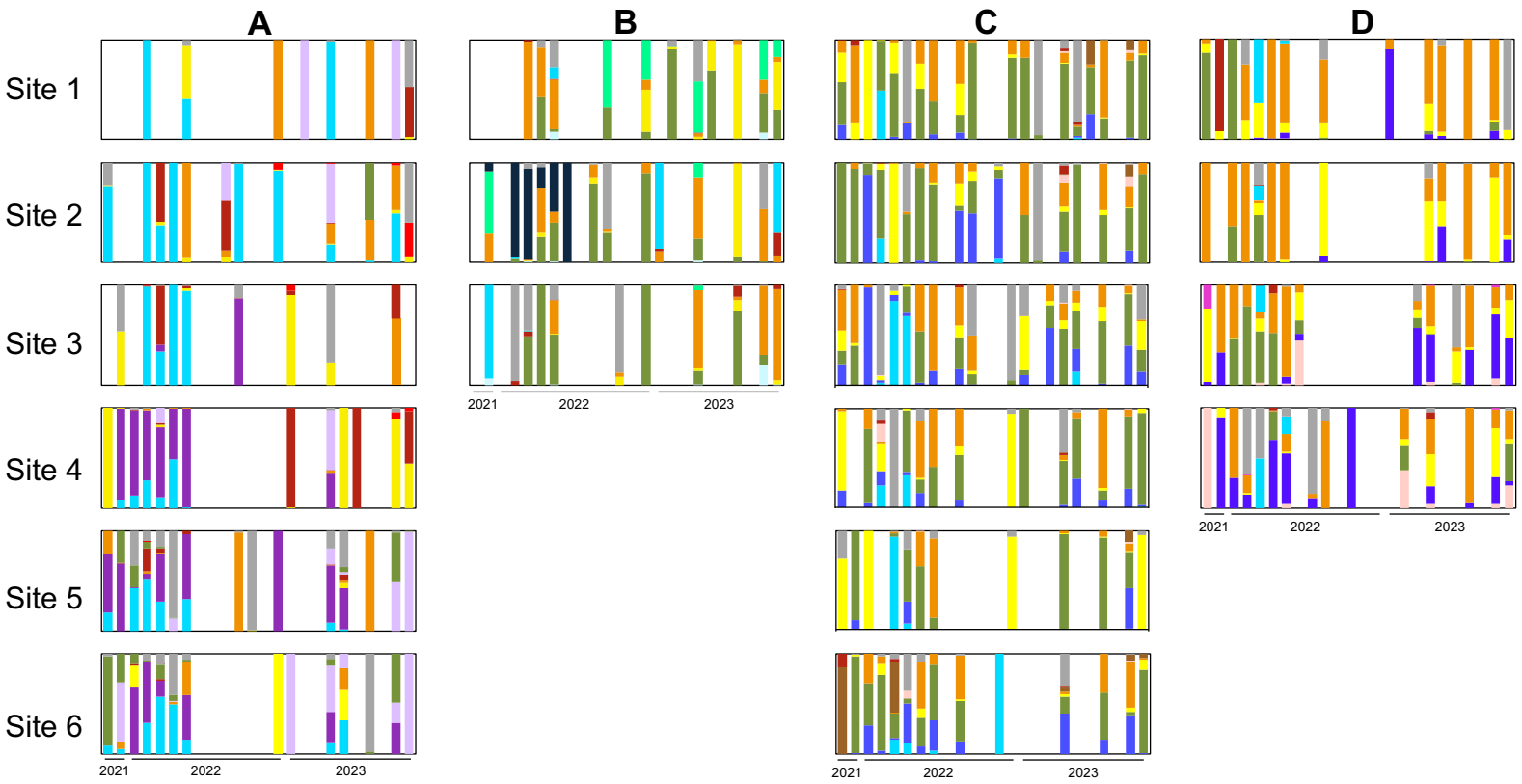
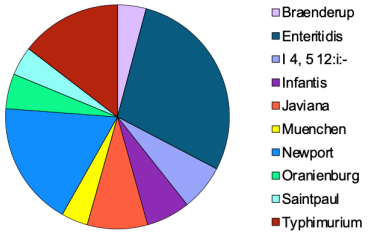
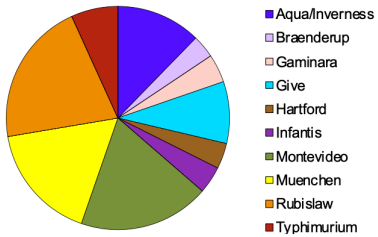

A
B
C
D
Site 1
Site 2
Site 3
Site 4
Site 5
Site 6
2021
2022
2023
Agbeni
Alabama/Bareilly III
Aqua/Inverness
Braenderup
Gaminara
Give I
Hartford
Infantis
Brazil I
Mississippi II
Montevideo II
Muenchen I
Oranienburg
Rubislaw
Typhimurium
Other

Figure 3.2. Top eight serovars present in each creek. Deep serotyping results are organized by site and creek. Each box represents the 24 months across a site, where colored bars show the proportion of each serovar present in that sample. Months with no bar represent a negative sample or a sample where deep serotyping was unsuccessful. Serovars outside of the top eight are classified as "other" and represented by a gray bar. The suffixes (-I, -II, -III) for some serovars refer to polyphyletic lineages.

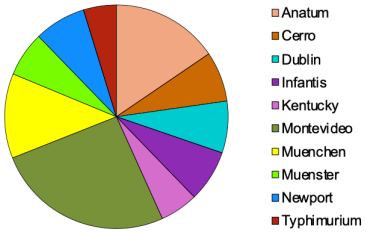
CDC-BEAM



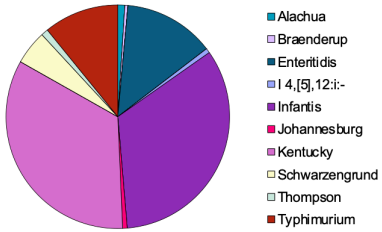
Creek



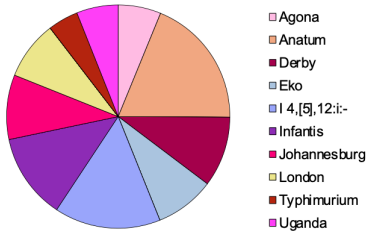
Beef



Chicken



Pork



Turkey

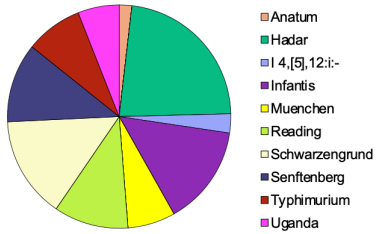


Figure 3.3. *Salmonella* serovars differ between animal agriculture, surface water, and clinical cases. The top 10 most common serovars for each food animal monitored by USDA-FSIS, clinical cases reported to CDC, and creek samples from this study are displayed. Serovar data for the commodity groups was accessed from the publicly available USDA-FSIS sampling repository (USDA-FSIS, 2024) and included samples that were collected between November 2021 – October 2023. Clinical isolates were accessed through the CDC BEAM Dashboard (CDC, 2024) and included cases for the entirety of 2021 - 2023. Polyphyletic serovars are not delineated by USDA-FSIS or CDC, so the suffixes were removed from serovars identified by deep serotyping and both results from both Montevideo lineages were combined for the creek pie chart. Serovar Typhimurium and its monophasic variant, I 4, [5], 12:i:-, cannot be distinguished by the CRISPR arrays.

additional serovars which were not present in the animal agriculture data, including Aqua/Inverness, Gaminara, Give I, Hartford, and Mississippi II.

Serovars Infantis and Typhimurium were the only serovars collected from this study that were also routinely identified in all four primary domestic food animals. Therefore, we completed a phylogenetic comparison of the study isolates belonging to these two serovars against publicly available genomes on NCBI-Pathogen Detection, which hosts human clinical and USDA-FSIS whole genome sequences, among others (Figures 3.4-3.5, S3.6-3.7, Tables S3.4-3.9). This revealed that the study isolates were most closely related to each other (they all fell within the same HC5 group) than to other isolates from human clinical, environmental, or food animal sources. For serovar Infantis, four of the five most closely related genomes were from human clinical isolates and the other was from a market swine in the midwest (Table S3.4). Importantly, the phylogenetic analyses (Figure 3.4A) showed that these five most closely related genomes did not share an HC20 type with the creek genomes, indicating that the creek isolates are genetically quite distinct from those on NCBI, which limits source attribution in this instance. Additional analysis using the CFSAN SNP Pipeline revealed that these creek isolates are between 53 – 104 SNPs apart from the NCBI isolates (Figure 3.4B). Similarly, for serovar Typhimurium, 91/95 of the most closely related isolates were clinical, and the remaining four were environmental, collected from almonds, white-tailed deer and emu feces, and a dairy cow from the northeast (Figure 3.5A, Table S3.7). There was more variation in the SNP differences between the serovar Typhimurium study isolates and NCBI isolates (Figure 3.5B), but only a select number of the human clinical

B

	2A-5	2B-1	5A-4	3A-4	4A-4	5A-6	11A-5	14A-6	SAMN25608903	SAMN25608883	SAMN24114991	SAMN09444132	SAMN22215935
2A-4	1	1	2	2	1	3	7	4	97	93	53	58	57
2A-5		2	3	3	2	4	8	5	98	94	54	59	58
2B-1			3	3	2	4	8	5	98	94	54	59	58
5A-4				4	3	1	5	2	99	95	54	60	58
3A-4					1	5	9	6	99	95	55	60	59
4A-4						4	8	5	98	94	54	59	58
5A-6							4	3	100	96	56	61	60
11A-5								7	104	100	60	65	64
14A-6									101	97	57	62	61
SAMN25608903										10	208	137	136
SAMN25608883											126	133	132
SAMN24114991												31	28
SAMN09444132													33

Figure 3.4. Serovar Infantis isolates collected in this study are not closely related to other isolates from mixed sources. Study isolates are bolded, with the label format of month-creek-site, and NCBI isolates are listed by sample ID with branch color corresponding to isolation type (human clinical: red, environmental/other: green, creek: blue). Isolation type was provided by NCBI metadata, and the HierCC schemes were determined through Enterobase (Zhao, 2021). Schemes were only reported if they are shared between two or more isolates. The phylogenies are rooted at the midpoint, and include all isolates within the two SNP clusters most closely related to study isolates. HC20 and HC5 values were added to reflect that the study isolates were greater than 20 and 5 allelic differences from the NCBI isolates, respectively. A) Core genome phylogeny of study isolates and NCBI isolates. B) SNP matrix comparing the study isolates and NCBI isolates. Darker shading indicates a greater SNP distance.

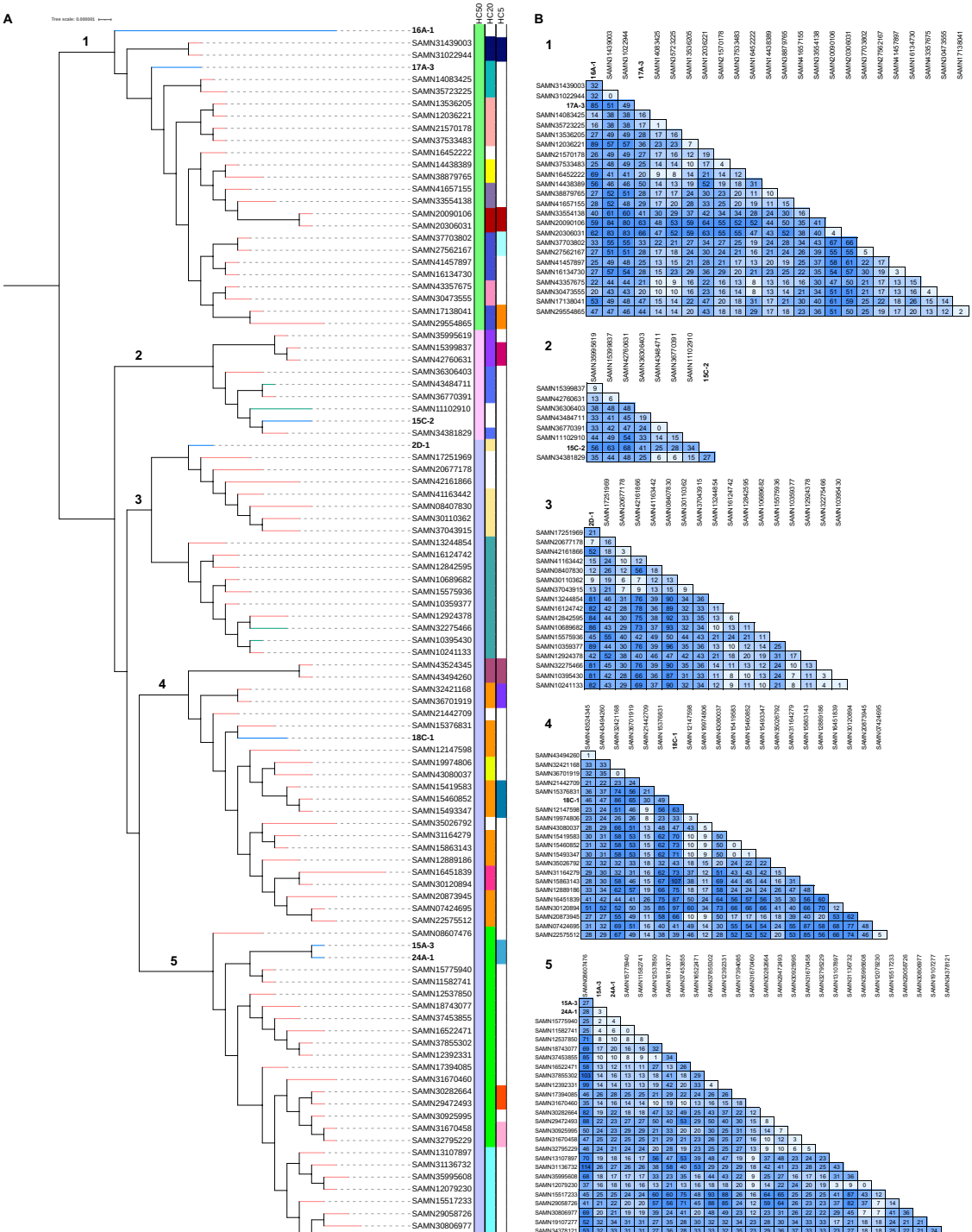

A
HC50
HC20
HC5
16A-1
SAMN31439003
SAMN31022944
17A-3
SAMN14083425
SAMN35723225
SAMN13536205
SAMN12036221
SAMN21570178
SAMN37533483
SAMN16452222
SAMN14438389
SAMN38879765
SAMN41657155
SAMN33554138
SAMN20090106
SAMN20306031
SAMN37703802
SAMN27562167
SAMN41457897
SAMN16134730
SAMN43357675
SAMN30473555
SAMN17138041
SAMN29554865
SAMN35995619
SAMN15399837
SAMN42760631
SAMN36306403
SAMN43484711
SAMN36770391
SAMN11102910
15C-2
SAMN34381829
2D-1
SAMN17251969
SAMN20677178
SAMN42161866
SAMN41163442
SAMN08407830
SAMN30110362
SAMN37043915
SAMN13244854
SAMN16124742
SAMN12842595
SAMN10689682
SAMN15575936
SAMN10359377
SAMN12924378
SAMN32275466
SAMN10395430
SAMN10241133
SAMN43524345
SAMN43494260
SAMN32421168
SAMN36701919
SAMN21442709
SAMN15376831
18C-1
SAMN12147598
SAMN19974806
SAMN43080037
SAMN15419583
SAMN15460852
SAMN15493347
SAMN35026792
SAMN31164279
SAMN15863143
SAMN12889186
SAMN16451839
SAMN30120894
SAMN20873945
SAMN07424695
SAMN22575512
SAMN08607476
15A-3
24A-1
SAMN15775940
SAMN11582741
SAMN12537850
SAMN18743077
SAMN37453855
SAMN16522471
SAMN37855302
SAMN12392331
SAMN17394085
SAMN31670460
SAMN30282664
SAMN29472493
SAMN30925995
SAMN31670458
SAMN32795229
SAMN13107897
SAMN31136732
SAMN35995608
SAMN12079230
SAMN15517233
SAMN29058726
SAMN30806977
SAMN19107277
SAMN34378121
SAMN43527257
B
1
2
3
4
5

Figure 3.5. Serovar Typhimurium isolates collected in this study are more closely related to human clinical isolates than animal sources. Study isolates are bolded, with the label format of month-creek-site, and NCBI isolates are listed by sample ID with branch color corresponding to isolation type (human clinical: red, environmental/other: green, creek: blue). Isolation type was provided by NCBI metadata, and the HierCC schemes were determined through Enterobase (Zhao, 2021). Schemes were only reported if they are shared between two or more isolates. The phylogenies are rooted at the midpoint, and include all isolates within the two SNP clusters most closely related to study isolates. HC20 and HC5 values were added to reflect that the study isolates were greater than 20 and 5 allelic differences from the NCBI isolates, respectively. A) Core genome phylogeny of study isolates and NCBI isolates. B) SNP matrices comparing the study isolates and NCBI isolates based on corresponding phylogenetic clades (1-5). Darker shading indicates a greater SNP distance.

isolates (n = 6) were within 10 SNPs of the study isolates, and these were shared between two SNP clusters, as defined by NCBI. Further, hierarchical clustering based on cgMLST values determined that there were more than 20 and 50 allelic differences between serovar Infantis and Typhimurium study isolates and the most closely related isolates, respectively (Figs. 3.4A, 3.5A).

3.5 Discussion

Each day, 198 billion gallons of surface water are used in the US, where 60.9 billion gallons of that are used for irrigation alone, accounting for the largest proportion of this source (Dieter et al., 2018). For this reason, it is pertinent to investigate *Salmonella* in surface water. While significant progress has been made in global surveillance studies (McConn et al., 2024), much is still unknown about what factors influence survival and persistence in this environment.

Our data show that the *Salmonella* strains we recovered do not match to food animal isolates, suggesting alternative reservoirs. In particular, more research is required to understand the contribution of wildlife to *Salmonella* populations within freshwater, since these hosts may be introducing pathogenic serovars and contributing to the AMR identified in the environment (Hernandez et al., 2021; Maurer et al., 2015b). This is further supported by the identification of AMR and MDR isolates in this study. Applying a One Health approach will be necessary to mitigate the impacts of *Salmonella* found in the environment, since there is opportunity for cyclical transmission between animals and humans via shared environmental reservoirs, such as surface water. To this end, a large, multiyear study was recently completed as part of a collaboration between federal, academic, industry, and local groups in southwest Arizona to map introduction,

transmission, and persistence of foodborne pathogens (https://www.fda.gov/food/environmental-studies/southwest-agricultural-region-environmental-microbiology-study-2019-2024). This study collected a wide variety of samples from irrigation waters, soil, sediment, air/dust, animal fecal material, wildlife scat, and other sources, and the resulting data will help inform management decisions and create a foundation for future surveillance studies.

As surface water is commonly used for agricultural water (i.e., irrigation), the Food Safety Modernization Act – Produce Safety Rule (FSMA-PSR) details its impact on food safety risks and provides guidance on best practices (FSMA Final Rule on Produce Safety, FDA 2024). In-field, antimicrobial treatments of irrigation water have limitations, since not all growers have access to the necessary resources and size of farm can greatly impact the efficacy of these treatments (Karp et al., 2015; Baur et al., 2017; Adalja and Lichtenberg, 2018; Devarajan et al. 2023). The extensive application of surface water and variability of sources also complicates risk assessment as *Salmonella* testing can be lengthy and costly, and there is likely not a one-size-fits-all control strategy. Many previous studies have sought to find an indicator organism that is easier to identify in the field and suggests the presence of *Salmonella* contamination. While *E. coli* has been considered an indicator of *Salmonella*, due to its association with fecal contamination and niche as enteric bacteria, some work has highlighted inconsistencies in the applicability of this organism (McEgan et al., 2013; Weller et al., 2020; Chung et al., 2023).

Salmonella prevalence throughout this study was variable, resulting in an overall prevalence of 69% (314/456), which is comparable to other studies in this region (Haley et al., 2009; McEgan et al., 2013; Cho et al., 2020) For example, a 3-month period during

2023 exhibited incidence of 100% (19/19) of sites to 0% (0/19), and back to 100% (19/19).

Salmonella prevalence was lower in the summer months. This could have been due to reduced rain and increased temperature that could have contributed to reduced nutrient availability and *Salmonella* influx to the environment. Previous work has shown a decrease in *Salmonella* prevalence in summer months (Deaven et al., 2021), while others have shown an increase during the same period (Liu et al., 2018; Haley et al., 2009), indicating high inter-creek variability. Similarly, precipitation has been associated with *Salmonella* in some studies (Strawn et al. 2012; Weller et al. 2020) while others found no relationship (Murphy et al. 2022; McEgan et al. 2013).

These findings were further supported by the GLMM output which suggested that *Salmonella* was more likely to be detected during spring, along with increased wind and precipitation. Increased proximity of hay pastures and mixed forests also increased the odds of detection, indicating that surrounding land attributes can contribute to *Salmonella* status. Alternatively, serovar complexity was associated with spring and winter, along with humidity and previously mentioned land use variables identified in the GLMM for prevalence. These findings can be used to support informed management decisions, as it could be more cost-effective to screen surface water for bacterial contamination after major precipitation events. However, more work is required to measure the contribution of different attribution sources to *Salmonella* surface water contamination, especially the wholly understudied role of wildlife.

Developing predictive models for likelihood of *Salmonella* detection in the environment, similar to work completed in this project, is a complex problem due to the

abundance of external interactions, along with regional differences in weather and creek composition, including nutrient availability and bacterial populations. Further, a model is only as good as the input data so cultural limitations of *Salmonella* identification and characterization, as well as variance in sample collection methods, may impact model development. One recent study demonstrated the importance of detection methods as both *Salmonella* prevalence and complexity differed in water samples that were processed with separate approaches (Murphy et al., 2024). Our study highlights the need for robust enrichment techniques to allow for the recovery of *Salmonella* while also trying to maintain original population dynamics, and high-resolution approaches to identify all serovars present within a sample.

When this study began, two different selective enrichment media, Rappaport-Vassiliadis (RV) and tetrathionate (TT) broths, were utilized and enrichments were plated onto xylose lysine tergitol-4 (XLT-4) agar. While the overall prevalence in the study was relatively high, there were several months when the prevalence was lower, particularly during the summer, and the total number of observed colonies present per plate during these times decreased as well. In the first 12 months, when *Salmonella* was not recovered following TT or RV enrichment, the pre-enrichment (buffered peptone water, BPW) culture was plated directly onto XLT-4 and, in some cases, yielded culturable isolates. It is possible that this is indicative of the stress upon cells in an aquatic environment, from which the non-selective enrichment for 24 hours prior was not able to allow for full recovery and thus, *Salmonella* were not able to proliferate in either RV or TT. It was noted that the inability to recover *Salmonella* in some of these samples was not due to overwhelming background as the plates were generally free of all growth. In the second

year of the study, a semisolid RV agar (MSRV) was included in addition to TT and RV as an alternate selective enrichment method, since the media components prevent growth of gram-positive bacteria. Importantly, MSRV plates serve as a motility test, such that *Salmonella* can use flagella to "swim" to the edge of the plate following culture inoculation in the middle and this provides a cleaner isolation (i.e., limited background bacteria) (Gorski et al., 2011). A previous study demonstrated that the parallel use of MSRV and a selective enrichment broth increased *Salmonella* recovery from water samples (Gorski, 2022), demonstrating the robustness of this approach and its applicability for *Salmonella* isolation from environmental samples.

Surface water harbors complex mixed-serovar *Salmonella* populations (Deaven et al., 2021), with an average of three serovars per sample identified in a large river (range: 1-10). Findings here show even higher complexity in small creeks (average of 3.7 serovars per sample, range 1-13). This is significant, demonstrating that these complex populations can form early in a creeks and are relevant to food safety. A previous study utilizing 16S analyses showed higher microbial community complexity upstream, with populations becoming more stable and less complex further downstream in creeks (Teachey et al., 2019; Liu et al., 2023). Focusing on a single species, the *Salmonella* data presented here is inverse; in two creeks, serovar populations increased in complexity as they flowed downstream, while the remaining two did not show complexity differences between up- and downstream sites (Figure 3.1C). A potential explanation for increasing complexity in some downstream sites is that the salmonellae are moving in the creek stem, and those populations are augmented by additional salmonellae that enter at later points, collectively accumulating downstream. Additionally, *Salmonella* can invade and

live in amoebas, ciliates, and biofilms which promote long-term survival in aquatic environments (Brandl et al., 2005; Byappanahalli et al., 2009; Gaertner et al., 2011; Sha et al., 2013).

The *Salmonella* populations identified here in surface water are more complex than those observed in food animals or in wild birds (Figure 3.3) (Siceloff et al. 2022; Obe et al., 2023; Siceloff et al., 2021; Cason et al., 2024; Smith et al. 2023). This is likely due to surface water acting as a catch-all for salmonellae from multiple different sources. Importantly, many serovars identified here are not commonly found in food animals or human isolates, suggesting that sources not traditionally surveyed may contribute to these populations. Additional research is needed to identify the factors that influence *Salmonella* complexity within all three components of a One Health scheme: animals, humans, and the environment. Meanwhile, surface water surveillance may serve as an indicator of salmonellae that are circulating within different ecosystems. Further, the expansion of environmental surveillance would help to develop a robust database that could begin to identify potential sources of sporadic illnesses not attributed to outbreaks. Subsequently, any generated whole genome sequences could provide new genomic insights on *Salmonella* transmission, including virulence factors needed for host colonization, environmental persistence, or stress response (Lipman et al., 2024). Previous work has been able to identify outbreak strains that had not previously been reported by using wastewater to test for serovar presence (M'ikanatha et al., 2024; Diemert et al., 2019; Vincent et al., 2007). Similarly to the concern that surface water acts as a reservoir for contamination by humans, agriculture, and wildlife, wastewater can potentially be used to measure burden of *Salmonella* illness or identify cases that would

otherwise go unreported (Berge et al., 2006; M'ikanatha et al., 2024). Since salmonellosis is often self-limiting within 7-10 days, it is significantly underrepresented in clinical cases, with an estimated 30 cases not reported for each lab-confirmed case, thus making wastewater surveillance a potential way to identify the presence and persistence of clinically relevant *Salmonella* serovars in human populations.

Human salmonellosis is often attributed, directly, or indirectly, to food animal production via consumption or improper storage of contaminated meat. While some serovars that are often found in food animals were also found in our study (e.g., Typhimurium, Infantis, Braenderup, Muenchen, Montevideo), the creeks were dominated by serovars that are not typical of these production systems. Serovars Give (Creek A) and Rubislaw (Creeks B-D) have been commonly associated with surface water environments, where reptiles are considered a reservoir for serovar Rubislaw and have been responsible for some human salmonellosis outbreaks (Waltenburg et al. 2022. Cho et al., 2020; Haley et al., 2009; Gorski et al., 2022; Pees et al., 2023; Lima Rocha et al., 2022; Rajabi et al., 2011). Notably, serovar Rubislaw contributes to more illnesses in Georgia than the national average, accounting for almost one-tenth of the illnesses caused by Enteritidis; the leading serovar in human cases (BEAM, 2025). Of these cases, nearly 13% come from blood or urine, suggesting this serovar is more likely to contribute to more serious illness compared to other serovars. Serovar Aqua/Inverness was frequently detected in Creeks C and D, and this also is not associated with food animals, based on data from the USDA-FSIS *Salmonella* verification program (USDA-FSIS, 2023). The current study did not include source attribution of fecal contamination within these creeks, but the Infantis isolates from this study are not related to the clonal lineage

responsible for the recent *Salmonella* outbreak in raw chicken products as determined by phylogenetic analyses and lack of pESI megaplasmid within the sequenced isolates (McMillan et al., 2020).

Serovar Agbeni was detected over six consecutive months at Site 2 in Creek B, and found once in Creek D. Interestingly, after these six months in Creek B, this serovar was never detected again for the remainder of the study, despite the depth provided by deep serotyping. While in a suburban area, this site is immediately downstream of a wooded area separating sites 1 and 2 with high incidence of wildlife (e.g., deer, raccoons, wild birds), and a sewage pipe and multiple manholes are within 500 ft of the sample collection. As it is unlikely that this serovar was shed from a human source for such a length of time, it is most likely linked to a wildlife reservoir(s) that repeatedly introduces it to the water (Langholz & Jay-Russell, 2013). However, previous work has yet to identify a food animal or wildlife source of serovar Agbeni. This illustrates the importance of further investigation into wildlife sources of clinically relevant *Salmonella* serovars. *Salmonella* surveillance data is dominated by that made available by federal agencies such as the U.S. Food and Drug Administration, the Centers for Disease Control and Prevention and USDA-FSIS. Under a One Health umbrella, understanding *Salmonella* in the environment and in wildlife is considerably understudied and there is a critical need to close this loop. While more work is needed to attribute serovars within these highly variable aquatic environments to likely hosts, the surveillance framework developed in this study can be helpful in identifying the salmonellae that are being circulated in respective environments.

The prevalence of AMR in this study was 11%. This is slightly greater than a recent study performed in Georgia (Cho et al., 2020), where isolates collected from water in the Middle Oconee River contained AMR phenotypes in 4.4% of samples. Our results indicate that antimicrobial resistance genes are being circulated even when human impact is limited but wildlife may be abundant, such as in the case of samples collected in a national forest (Creeek D). The presence of MDR in these isolates raises concern over the dissemination of antimicrobial resistance genes (ARGs) via various pathways, including direct and indirect wildlife interactions, horizonal gene transfer from bacterial communities, or human activity. As surveillance of wildlife has remained limited, an accurate assessment of the prevalence of both foodborne pathogens and ARGs is currently not within reach but this information can be supplemented with routine creek sampling. Otherwise, this limitation will continue to be a barrier to effective risk assessment as wildlife interaction in agricultural water and produce fields is a multifaceted problem with complex solutions (Gruszynski et al., 2014b).

While this work resulted in GLMMs to predict factors that influence the prevalence and complexity of *Salmonella* in creeks, the implications are limited to the effect of local climate and may not be applicable to other regions. The data used in these models can serve as a guide for research performed elsewhere to measure the variable effect of weather and land use on *Salmonella* in surface water and contribute to the growing body of work on *Salmonella* dynamics in the environment. Due to direct access restriction at some sites, water quality metrics, such as turbidity, dissolved oxygen, pH, conductivity, and salinity, were not collected and therefore not considered in modeling. However, previous work has demonstrated that the relationship between turbidity levels

and bacterial populations in water is dynamic (McEgan et al., 2013; Kim et al., 2023; Murphy et al., 2022), which further emphasizes that there is a complex network of biotic and abiotic factors that contributes to *Salmonella* abundance in water and there is not likely one main driver. Further, the sampling sites do not exist within a vacuum; for example, there may be human activity affecting *Salmonella* populations in the creek located within a national forest, and this is one of the limitations in modeling. *Salmonella* isolation from environmental samples can be difficult if there is an overwhelming amount of background bacteria competing for the same resources during growth and if the cells are unable to recover following any environmental stresses. As such, we had to modify our methods throughout the study as needed to increase *Salmonella* recovery (i.e., isolation directly from primary non-selective enrichments and inclusion of a primary mobility enrichment); this, in turn, limited next-generation sequencing results due to a decreased abundance of the target organism in the non-selectively enriched cultures. While we recorded the media for each *Salmonella* isolate (Figure S3.1B) and we continued to use traditional methods alongside alternative approaches, we acknowledge the variability added into our dataset through the use of multiple enrichment media and accounted for that by normalizing the relative serovar abundances across each media type for each sample. Our results demonstrate that most samples contained multiserovar populations. This continues to demonstrate that selecting multiple colonies is necessary to reflect the *Salmonella* resistome within each sample (Cason J. et al., 2011).

In conclusion, this study highlights the complexity and variability of *Salmonella* populations within creeks influenced by diverse land uses and climatic factors. Our findings demonstrate that multiserovar populations are prevalent within surface water and

suggest that environmental factors such as precipitation and humidity significantly influence *Salmonella* prevalence and complexity, although it is a multifaceted relationship. Population analyses showed increasing complexity further downstream, as well as higher serovar complexity in the spring compared to fall and winter. The identification of 37 serovars, including those of human concern with antimicrobial resistance, highlights the need for enhanced surveillance strategies that integrate environmental and wildlife monitoring. In particular, our serotyping data and subsequent whole genome sequence analysis revealed that there is a significant discordance between environmental, clinical, and animal agriculture isolates. These insights emphasize the critical importance of a One Health approach to address the interconnected risks posed by *Salmonella* in the environment, wildlife, and human systems. Future work should prioritize understanding the transmission pathways between these reservoirs and improving predictive modeling to inform targeted interventions and public health policies.

3.6 Materials and methods

Sample site selection

Four distinct creeks in the Southeastern United States were selected based on their surrounding land attribution, including animal agriculture, national forest, and suburban communities. The number of sites within each creek varied, based on size of the creek and accessibility of sites from the road. For each creek, at least one sample was collected from within 0.5 miles of the headwater. All sampling locations were within first or second order channels and were upstream of any wastewater or drinking water treatment facilities.

Salmonella population analysis

To identify the populations of *Salmonella* within water samples, enrichments resulting in *Salmonella* culture positive samples were processed individually by centrifuging 1 mL of each positive selective enrichment at 18,000 rcf for three minutes. Total genomic DNA was isolated from the resulting pellet using a Promega Genome Wizard kit (Madison, WI) according to the manufacturer's instructions and resuspended in 200uL of molecular-grade water. A total of 2 µL of this template was used in the PCR for CRISPR-SeroSeq with primers targeting the conserved direct repeat sequences within *Salmonella* CRISPR arrays (Thompson et al., 2018; Siceloff et al., 2022). Primers also included index sequences, which facilitated multiplexed, high-throughput sequencing. PCR products were purified using the AMPure system (Beckman Coulter, Indianapolis, IN) and pooled in approximate equimolar ratios. Pooled libraries were sequenced using the Illumina NextSeq 550 platform (Illumina, California, USA) mid-output 150 cycle v2.5 kit with single-end reads. A water control and a positive control containing *Salmonella* serovar Enteritidis genomic DNA with a known CRISPR profile was included in the library. Sequence reads were parsed and matched in a local BLAST search to a lab-curated database of over 160 serovars (Siceloff et al., 2022). Serovars were called only if they contained multiple CRISPR spacers that were unique to that serovar. Where there were sufficient *Salmonella* sequence reads (>1,000 reads) for both the TT and RV enrichments, the relative frequency of each serovar was normalized across both enrichments to provide a single serovar profile. Within this dataset, serovars Alabama and Bareilly III, Aqua and Inverness, Johannesburg, and Urbana are not able to be distinguished based on CRISPR arrays, respectively.

Calculating land attribution for each sampling site

Land cover data was obtained from USGS national land cover database (NLCD) (https://www.usgs.gov/centers/eros/science/national-land-cover-database). Creek data was obtained from U.S. Geological Survey (USGS) national hydrography database (https://www.usgs.gov/national-hydrography/access-national-hydrography-products). Inverse-distance weights (IDW) as described in King et al. (2005) were used to characterize land cover within creeks. The IDW proportions for each land cover class were calculated for the following distance intervals (m): 0-100, 100-250, 250-500, 500-1,000, 1,000-2,000, and 2,000-5,000 upstream of the sampling sites. Land cover data for each creek was extracted using ArcGIS Pro version 3.1 (https://www.mrlc.gov/data/legends/national-land-cover-database-class-legend-and-description).

Statistical testing

All analyses were performed in R version 4.3.3. PcoA analysis was performed using the Vegan package in R. The deep serotyping results were normalized using the DESeq2 package (http://www.bioconductor.org/packages/release/bioc/html/DESeq2.html) to adjust the read counts per sample based on the size factors present.

Weather data collection

Meteorological data was collected using nearby weather stations. Measurements included maximum and minimum air temperature, relative humidity, soil temperature at two, four, and eight inches, average wind speed, total solar radiation and total

precipitation (Tables S10-12). These were measured between 12:15 am to 11:59pm each day. Creeks A and B had unique weather stations, while Creeks C and D shared the same weather station, as this was closer in proximity than any other station.

Predictive modeling for *Salmonella* prevalence and complexity

Preliminary exploratory data analysis (EDA) was performed to determine the modeling input. Analysis of variance inflation factors (VIFs) was used to assess the multicollinearity among the weather and land attribution variables, with a cutoff VIF value of 4. Additionally, all land attributes variables which comprised less than 5% of the total land cover per creek were removed. Generalized linear mixed models (GLMMs) were developed using lme4 package in R to investigate how the weather, land attribution, and temporal factors affected the likelihood of *Salmonella* detection and population complexity. In these models, all variables were considered fixed effects, while the creek was included as a random effect to account for pseudo-replication. The final GLMM was chosen following forward selection to determine if any quadratic or interaction effects should be added, along with backwards selection to remove any insignificant variables from the model.

Phylogenetic analyses

Due to the overlap of serovars Infantis and Typhimurium in both water and food animal sources, we compared the study isolates against publicly available genomes. For these phylogenetic analyses, we used iterative steps to reduce computational demands and to increase the resolution of relatedness in the final tree. Metadata, including accession numbers, for all isolates with computed (sero)type matching serovar Infantis or Typhimurium were downloaded from NCBI Pathogen Detection on October 10th, 2024

(Table S6-7). The following steps were completed in parallel for both serovars of interest. To reduce selection bias, the first isolate with an assembly was chosen as the representative for each single nucleotide polymorphism (SNP) cluster, as organized on Pathogen Detection. Isolates within a SNP cluster are considered highly related (NCBI, 2024). Some SNP clusters were excluded from further analyses due to isolates lacking a GenBank assembly accession, for a total of 650 and 3,057 representative genomes/SNP clusters of serovars Infantis and Typhimurium, respectively. For each serovar, a core genome alignment was generated with the NCBI and creek genomes using roary (Page et al., 2015). The resulting alignment, consisting of 3,124 and 3,511 genes for serovars Infantis and Typhimurium, respectively, was used to generate a phylogenetic tree with Very Fast Tree (Piñeiro et al., 2020). All trees were visualized with iTOL (Letunic & Bork, 2021). To increase the phylogenetic resolution, the clades containing the study isolates and all representative isolates within two most recent common ancestors were selected to generate a core genome alignment (yellow highlight, Table S3.5 and S3.8, Figure S3.6-7). As the decreased number of isolates allowed for more computationally intensive algorithms, RAxML-NG (Kozlov et al., 2019) (1,000 bootstrap replicates, GTR+G model) was used to infer a maximum-likelihood phylogeny for the subset clades. Based on the resulting tree, we determined which two SNP clusters contained the most closely related for each creek isolate (Infantis: n = 2 SNP clusters; Typhimurium: n = 14 SNP clusters) (blue highlight, Table S3.6-S3.9, Figure S3.6-7) and analyzed all annotated assemblies within those clusters (Infantis: n = 5 isolates; Typhimurium: n = 95 isolates) to create a final phylogeny of the most closely related isolates on NCBI to the study isolates, using roary, RAxML-NG, and iTol as described above. To determine the

hierarchical clustering scheme (HierCC) for all isolates included in the final phylogeny, the corresponding raw reads were transferred from NCBI Short Read Archive to Enterobase (Zhou et al., 2018, 2020a, 2021a). The HierCC 50, 20, and 5 categories were included to demonstrate the genetic distance between study isolates and NCBI isolates, based on the clusters calculated with the distance between core genome multilocus sequence types (cgMLST). Additionally, the CFSAN SNP Pipeline was used to generate a pairwise comparison matrix of each isolate included in the final phylogenies (Davis et al., 2015).

3.7 Data availability statement

The datasets presented in this study can be found in online repositories. The names of the repository/repositories and accession number(s) can be found in the article.

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3.10 Competing Interest Statement

NWS has a licensing agreement with Ancera Inc.

3.11 Author Contributions

JCS and ATS performed the experiments, completed the analysis and wrote the first draft of the paper. SMS contributed to statistical analysis. RLB contributed to the design of the study and WGS of isolates. All authors reviewed, edited, and approved the paper.

3.12 Reference

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CHAPTER 4

SALMONELLA TRANSMISSION AND PERSISTENCE IN TWO RIVERS IN PICHINCHA, ECUADOR[1]

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4.1 Abstract

Salmonella enterica is a leading global cause of bacterial foodborne illness, often transmitted through contaminated food and water. *Salmonella* transmission dynamics in freshwater systems remain poorly understood. In this study, we collected samples from two rivers in Pichinca province, Ecuador daily over four days to determine *Salmonella* prevalence and characterize any genetic relationship between isolates at different points downstream and between rivers as well as repeated isolation of isolates at the same location on different days. These rivers, including an urban river and an agricultural river, contained four sites each. At each site, 10L water samples were collected using modified Moore swabs (n = 32) and cultured for *Salmonella*. All recovered isolates were analyzed by whole genome sequencing (WGS). Overall prevalence was 75% (24/32), where the urban river, Rìo Machángara, had a higher prevalence (87.5%; 14/16) than the agricultural river, Rìo San Pedro (62.5%; 10/16). Serotyping with WGS identified eight serovars, four of which had a high degree of genetic relatedness based on multilocus sequence typing and suggested possible transmission, persistence, or reintroduction events across sampling sites and days. Additionally, in positive samples where two or more isolates were recovered, 71.4% (10/14) contained multiple serovars. Importantly, both rivers contained clinically relevant serovars, which underscores the need for more robust environmental surveillance efforts to identify routes of foodborne pathogen transmission and reduce *Salmonella* illnesses.

4.2 Significance statement

Surface water contamination is a significant contributor to *Salmonella* illnesses worldwide; however, little is known about the ecology of this pathogen following

introduction to a water source. Thus, a knowledge gap exists in the understanding of diversity and long-term survivability of *Salmonella* within surface water sources. This study aimed to resolve these questions by analyzing *Salmonella* prevalence and serovar diversity in surface water samples collected in two important rivers in a highly populated region of Ecuador. Importantly, whole genome sequencing identified significant serovar overlap was identified between these rivers; however, more serovars were found in the urban river including those more commonly associated with human illness, demonstrating that human health risk is unequally distributed. Additionally, close WGS relationships between isolates downstream and across multiple days of sampling suggest potential transmission or persistence of *Salmonella* in these rivers. Overall, these results underscore the need to develop a One Health environmental monitoring approach that can be used to improve *Salmonella* control.

4.3 Introduction

Salmonella enterica is a leading cause of bacterial foodborne illness worldwide. This species is categorized into over 2,600 distinct serovars, which are defined by the combination of their somatic (O) and flagellar (H) antigens, according to the White-Kauffmann-LeMinor classification scheme (Grimont & Weill, 2007; Issenhuth-Jeanjean et al., 2014). Importantly, serovar-specific differences such as host restriction (Uzzau et al., 2000), virulence factors (Cheng et al., 2019), antimicrobial resistance (D. H. Shah et al., 2017), and stress response (Gorski & Noriega, 2023) can significantly influence the clinical relevance of a serovar. Subsequently, 100 serovars within *Salmonella enterica* subspecies *enterica* account for the majority of global human infections (Hendriksen et al., 2011; McVey et al., 2022; Park et al., 2009).

Subspecies *enterica* can be divided into two categories that separate typhoidal from non-typhoidal salmonellae (Crump et al., 2004). Serovars Typhi, Sendai, and Paratyphi (A, B, and C), are considered typhoidal serovars and the causative agents of typhoid (enteric) fever and paratyphoid fever (Gal-Mor et al., 2014). In Latin America, Sub-Saharan Africa, and Southeast Asia, where enteric fever is more common, illnesses are often attributed to ingestion of food or water contaminated with fecal material (IHME, 2021b, 2021a). Non-typhoidal serovars (NTS) are the causative agent of salmonellosis, a self-limiting gastroenteritis. This bacterium causes an estimated 150 million global illnesses and 60,000 deaths annually (CDC, 2024b).

As an enteric pathogen, *Salmonella* is often found in the gastrointestinal tract of animal hosts; however, *Salmonella* is also ubiquitous in the environment, and has been identified in various abiotic samples, especially in surface water such as creeks, rivers, ponds, and canals (Deaven et al., 2021; Haley et al., 2009; Khouja et al., 2024; You et al., 2006), but also soil and dust (Bardsley et al., 2021; Fahimipour et al., 2018; Jechalke et al., 2019b; Pal et al., 2021).

Salmonella can be introduced to water by direct or indirect fecal contamination by humans or animals (Casanova et al., 2020; Li et al., 2015; Topalcengiz et al., 2019). Direct contamination includes defecation into the water, or sewage release into the water, while indirect includes run-off from the land following rain. Once introduced, *Salmonella* can survive within biofilms, free-living protozoa, or free-floating in planktonic or viable but non-culturable (VBNC) states (Brandl et al., 2005; Byappanahalli et al., 2009; Gaertner et al., 2011; Oguadinma et al., 2022). Supporting this, a study showed long-term viability of *Salmonella* in a lab experiment where it was recovered in freshwater after

over 300 days post-inoculation. There is the potential for even longer survival in an open system when nutrients are regularly cycled through, which would occur with frequent introduction of fecal material into surface water (Topalcengiz et al., 2019). This ability to survive long periods in non-host environments likely explains why *Salmonella* has been regularly isolated from various aquatic sources such as urban wastewater, runoff caused by rain, and contaminated water due to agricultural waste (Berge et al., 2006; Casanova et al., 2020; Magana-Arachchi & Wanigatunge, 2020; Murphy et al., 2022; Santiago et al., 2018; Toro et al., 2022).

Food outbreak traceback capabilities were improved drastically with the development and implementation of whole genome sequencing (WGS) technology as a highly discriminatory typing tool (Allard et al., 2012, 2016; Timme et al., 2013). While individual whole genome sequences provide important genetic information, a robust database, such as GenomeTrakr, generates the capacity to assess isolates within the wider context of almost 750,000 *Salmonella* genomes (FDA, 2025). Broadly, WGS analysis utilizes single nucleotide polymorphisms (SNPs) to compare the sequence fidelity between a set of isolates. One form of this comparison is core genome multilocus sequence typing (cgMLST), which assesses the genomic relationship of isolates based on the sequence of genes present in 95% of a given dataset (Zhou et al., 2021b).

Pichincha lies in the Northern Sierra of Ecuador and, with a population of over three million, is the second most populated province in Ecuador (City Populations, 2024). This region is home to the Ecuadorian capital of Quito; the second highest capital in the world at 2,850m. As such, the population has increased by more than 60% and urban expansion by 7.1% since 2000 (Atlas of Urban Expansion, 2016; Carrión & Erazo

Espinosa, 2012). These changes are followed by additional stress on the environment, contributing to increases in chemical and microbial intrusion to water sources (Adler Miserendino et al., 2013; Benítez et al., 2019). In addition to direct human contributions to water pollution such as sewage, indirect runoff has the potential to collect bacteria and nutrients that contribute to unsafe water conditions. Importantly, these conditions can be exacerbated when runoff occurs over land where higher concentrations of *Salmonella* are present. The common use of rivers throughout this region for livestock, agricultural, and domestic activities, including livestock interactions, crop irrigation, washing clothes, and bathing in contaminated waters can produce severe health effects for the inhabitants (Borja-Serrano et al., 2020).

The Rìo Machángara is considered one of the main rivers in Pichincha; its headwaters lie at the base of Atacazo volcano near the southern tip of Quito, where it flows for 40km before entering Rìo Guayllabamba. This river in turn is a tributary to the Rìo Esmeraldas that flows into the Pacific Ocean. From Quito, water from the Rìo Machángara crosses three different Ecuadorian provinces before reaching the ocean. The Rìo San Pedro is also in Pichincha province. It begins high in the volcanic region of Iliniza Sur and flows into the Rìo Machángara just east of Quito. Along its 73 km course the Rìo San Pedro flows through agricultural land, including dense dairy cattle production in the Mejía canton, which includes the towns of El Chaupi (population 1,425) and Machachi (population 32,814), which lie at 3,386m and 2,933m above sea level, respectively.

The Rìo Machángara acts as the destination for untreated wastewater, collecting approximately 70% of the city's waste, including effluent and contaminants (Campaña et

al., 2017). Wastewater treatment facilities present at the city exit prevent untreated waste from extending beyond the heavily-populated area; however, this does little to prevent contamination within the city. For this reason, this river has been studied in multiple projects to measure chemical and microbiological traces (Alomía Herrera & Carrera Burneo, 2017; Méndez et al., 2022; Ortega-Paredes et al., 2020; Vinueza et al., 2021; Vizcaíno et al., 2016). These studies have recently led to a monumental impact: following Ecuador becoming the first country in the world to recognize the Rights of Nature in its constitution, in July 2024, an Ecuadorian judge ruled that pollution has violated the rights of the Rìo Machángara, and called for the city of Quito to clean the river (Altamirano Cardenas & Cervantes Galván, 2024).

Although several investigations have been completed regarding the presence of *Salmonella* in water sources, further characterization and investigation of transmission within rivers is understudied. The comparable sizes and contrasting creeks of the Rios Machángara and San Pedro in the Pichincha province lend themselves to a useful study to compare *Salmonella* across two individual rivers situated in the same region but with very different surrounding land-use. This study aimed to determine the presence and diversity of *Salmonella* over four sites in each river that were sampled repeatedly over four days. Using WGS, we sought to assess whether *Salmonella* was being re-introduced to the river daily and whether it was being transmitted downstream based on the genetic relatedness of isolates.

4.4 Materials and methods

4.4.1 Site selection

Two distinct rivers were chosen as the subject of this study based on land attribution characteristics associated with each river and both lie in the Pichincha province (Figure 4.1A). Four sites were selected along each river based on different land use and ease of access. The four sites along the Rìo Machángara in Quito were chosen to reflect the river just after entering the city (site 1), two points within the city (sites 2 and 3), and a final site after the river exits the city (site 4) (Figure 4.1B).

For the Rìo San Pedro, the four sites were selected to cover areas including upstream (site 1), within (site 2) and downstream (site 3) of the major agriculturally concentrated areas as well as just prior to entering urban populations (site 4) (Figure 4.1C). The Rìo San Pedro flows into the Rìo Machángara; the last sampling site on the Rìo Machángara is upstream of where the Rìo San Pedro enters.

4.4.2 Sample collection and *Salmonella* isolation

A modified Moore swab and peristaltic pump were used to collect 10 L of creek water from each site (n = 32 samples) as described (Deaven et al., 2021). The swabs were stored on ice until returning to the lab, where 100 ml of buffered peptone water (BPW; BD Difco, Franklin Lakes, NJ) and novobiocin (40 mg/L; Thermo Scientific Chemicals, Waltham, MA) were added. Swabs were each hand-massaged for 1 minute, then incubated at 42°C for 20-24 hours. Following incubation, 1 and 0.1 mL of BPW were transferred into 12.2 mL of tetrathionate (TT; BD Difco Franklin Lakes, NJ) and 9.9mL Rappaport-Vassiliadis (RV; BD Difco Franklin Lakes, NJ) broth, respectively, and incubated at 37°C for 20-24 hours. Iodine-iodide solution was not readily available when

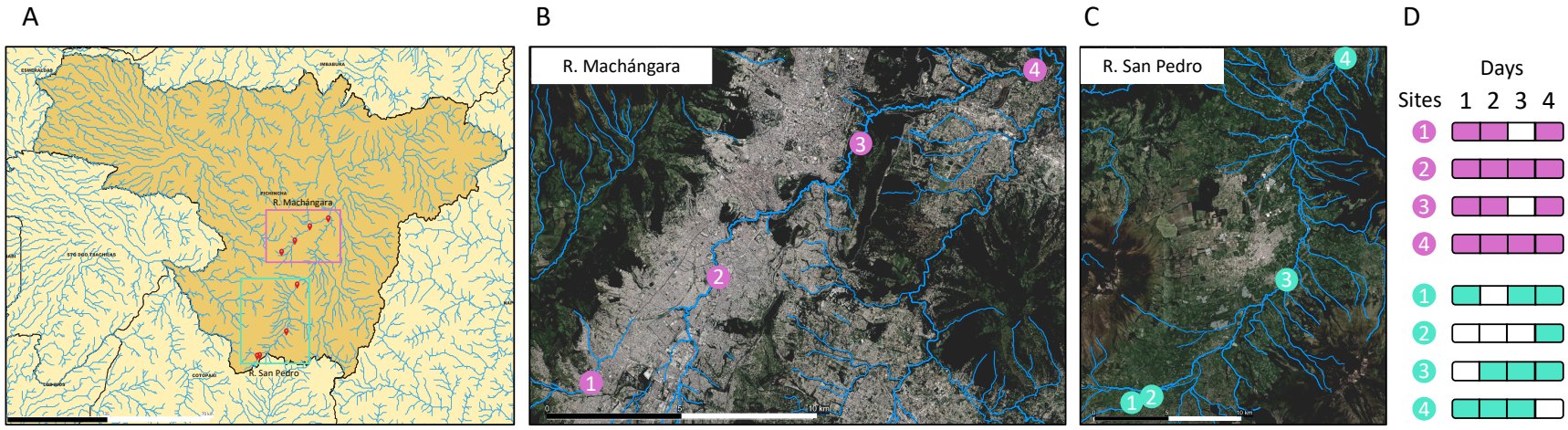

Figure 4.1. Sampling site distribution and *Salmonella* prevalence of two rivers in Pichincha, Ecuador. Regional map of Pichincha (A) indicates the relative location of both rivers sampled in this study (Rìo Machángara in purple and Rìo San Pedro in green). Sampling sites of Rìo Machángara (B) and Rìo San Pedro (C) are shown along with respective tributaries. *Salmonella* prevalence (D) is shown as colored boxes representing positive samples for Rìo Machángara (purple, top) and Rìo San Pedro (green, bottom).

we were performing this work. Therefore, prior to the study, we tested multiple commercially available iodine products as potential replacements including potassium triiodide, povidone, and an iodine tincture. In a comparison of *Salmonella* recovery in TT broth between traditional iodine-iodide solution and these alternatives, povidone demonstrated consistent and comparable recovery resulting in its use during this study (data not shown). To achieve the same concentration as is typically used, 3.2 ml of 10% povidone (1% free iodine) were added to 9 ml of TT for a final volume of 12.2 ml. Additionally, 0.1 mL of the pre-enriched BPW culture was inoculated into modified semisolid Rappaport-Vassiliadis (MSRV; BD Difco Franklin Lakes, NJ) and incubated at 37°C for 24-48 hours. *Salmonella* presence was tested by streaking the TT and RV enrichments, along with any presumptive *Salmonella* growth from the MSRV plates, onto xylose lysine deoxycholate (XLD; BD Difco Franklin Lakes, NJ) plates with novobiocin (40mg/L) followed by incubation at 37°C for 24-48 hours. Up to three colonies were selected (based on differing morphologies) from each plate and restreaked for isolation onto fresh XLD plates. After incubation at 37°C for 24-48 hours, a single colony was streaked onto Luria-Bertani (LB; Hardy Diagnostics, Santa Maria, CA) agar. These were confirmed as *Salmonella* using poly A-I serum agglutination (BD Difco, Franklin Lakes, NJ). All confirmed *Salmonella* isolates were grown overnight in LB broth and then stored at -80°C in 20% glycerol.

4.4.3 Whole genome sequencing

Stored glycerol stocks were used to propagate *Salmonella* isolates. Stocks were streaked onto LB agar and grown overnight. A single colony was picked from the LB plate and used to start an overnight growth. DNA was isolated from overnight growth and

shipped to the FDA Center for Food Safety and Applied Nutrition (FDA-CFSAN) for library preparation and sequencing. Enterobase was used to analyze the whole genome sequences for serotyping and core genome MLST analysis (Zhou et al., 2020b, 2021b). SeqSero and SISTR (as part of Enterobase) were used for serovar identification (Yoshida et al., 2016; Zhang et al., 2019).

4.5 Results

Over four consecutive days, water samples were collected at eight sites across two rivers in the Pichincha region in Ecuador (n = 32). The Rìo Machángara and Rìo San Pedro contained four sites each, where the rivers primarily flowed through a dense urban environment and animal agriculture, respectively (Figure 4.1). In total, 75% (24/32) of samples were positive, where the Rìo Machángara and Rìo San Pedro had an overall prevalence of 87.5% (14/16) and 62.5% (10/16), respectively. Daily incidence ranged from 75% (3/4) to 100% (4/4) in the Rìo Machángara and 25% (1/4) to 75% (3/4) in the Rìo San Pedro.

Whole genome sequencing (WGS) was completed on 54 isolates, ranging from one to six isolates per sample. A total of eight serovars were identified, where serovars Uganda, Typhimurium, I 4,[5],12:i:-, Infantis, and Anatum were identified in both rivers (Figure 4.2). Serovars Amager and Derby were unique to the Rìo Machángara, and serovar Manhattan was unique to the Rìo San Pedro. Serovar Uganda was the most frequent serovar isolated and was present in 50% (13/24) of positive samples, including 71% (10/14) and 30% (3/10) of samples from the Rìo Machángara and Rìo San Pedro, respectively. Serovar Typhimurium and its monophasic variant I 4,[5],12:I:-, were also common, with isolation from 25% (6/24) and 21% (5/24) of positive samples,

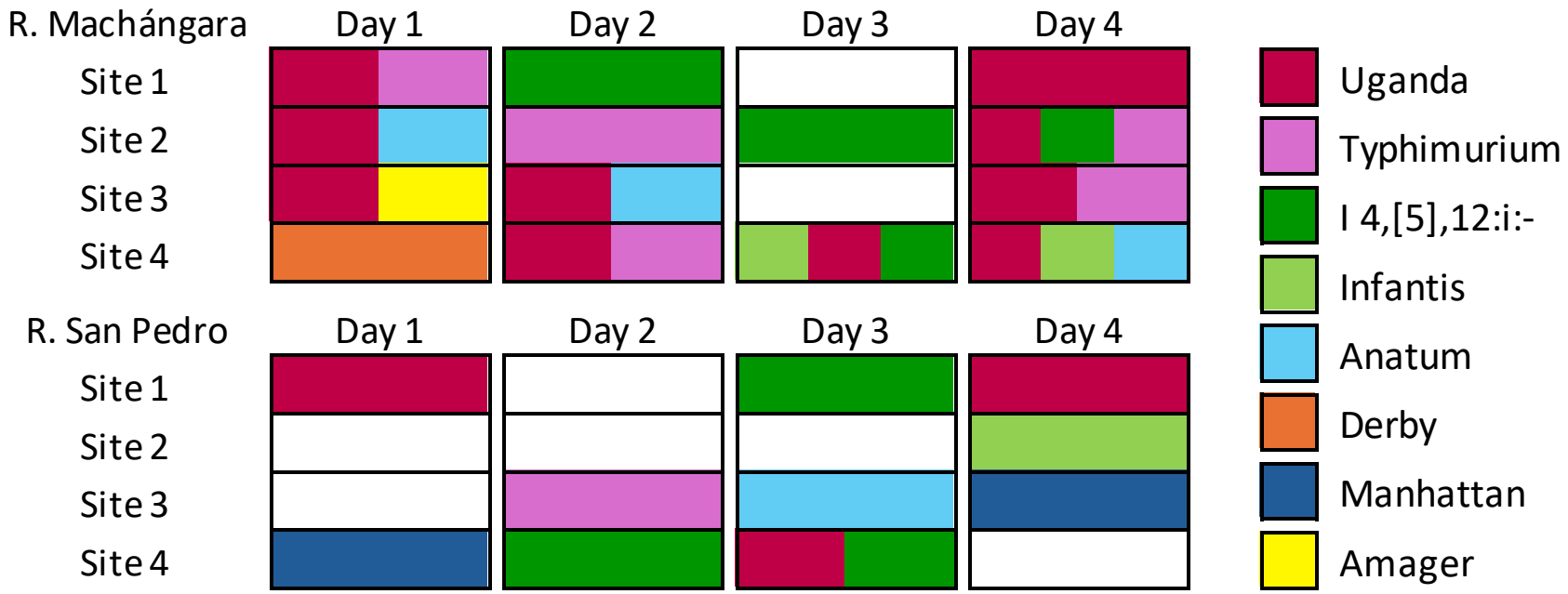

Figure 4.2. Serovar identification from whole genome sequencing of river isolates. Rìo Machángara (top) and Rìo San Pedro (Bottom) positive samples are shown as colored squares. Samples are represented as one, two, or three colors showing total number of serovars identified (right). Negative samples are shown as white squares.

respectively. In 14 positive samples, two or more isolates were recovered, resulting in the identification of multiple serovars in 71.4% (10/14) of samples, including 64% (9/14) of samples from the Rìo Machángara and 10% (1/10) of samples in the Rìo San Pedro.

Enterobase was used to designate a cgMLST to each isolate (Zhou et al., 2020b). Additionally, hierarchical clustering (HC) was used to group related isolates based on allelic differences. Isolates within two allelic differences (i.e. sharing the same HC2 cgMLST) are considered highly related (Zhou et al., 2021b). We assigned HC2 sequence types to isolates belonging to serovars that were identified in three or more samples and used this to look at relationships between different isolates in our study. Serovar Uganda isolates were categorized into three sequence types, where 54% (7/13) of samples containing this serovar belonged to HC2 472322 type (Figure 4.3). Less closely related, HC2 472380 had between 5 and 10 allelic differences from the prominent HC2 472322 type. Interestingly, the remaining serovar Uganda cluster, HC2 472372, had a distant relationship of more than 50 allelic differences from the other clusters. Isolates within this sequence type were recovered from all four sites of the Rìo Machángara and in three out of four days in this river. Notably, all isolates of serovars Anatum and I 4,[5],12:I:- belonged to a single sequence type, respectively. Patterns of related isolated between sampling sites or across days at the same site were marked with arrows or circles, respectively (Figure 4.3). For example, this pattern was identified for HC2 472322 was recovered in the first three three sites of the Rìo Machángara on day one and on site four of day two. Additionally, this same cluster was found in site one on days one and four in the Rìo Machángara.

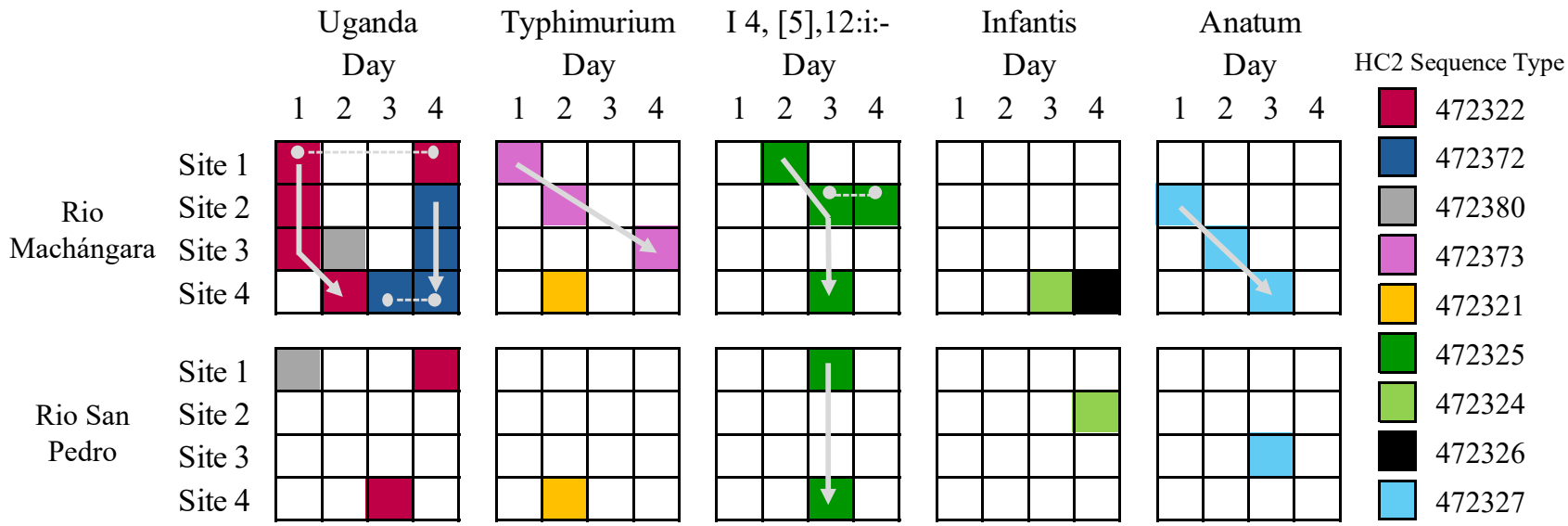

Figure 4.3. cgMLST highlights trends of *Salmonella* persistence and transmission. Study isolates sharing core genomes within two allelic differences (HC2) are shown by color-coded squares. Possible connections of transmission and persistence are indicated with solid gray arrows and dashed gray lines with circles.

4.6 Discussion

Several studies have been conducted on both Rìo San Pedro and Rìo Machángara to identify various pathogenic microorganisms, including studies that specifically looked for *Salmonella* (Borja-Serrano et al., 2020; Campaña et al., 2017; Ortega-Paredes et al., 2020; Vinueza et al., 2021). Interestingly, these studies were unable to identify the presence of *Salmonella* in their samples, however, high levels of *E. coli* and fecal coliforms were detected. Both studies utilized membrane filtration, a method consisting of collecting and filtering water from an 800 mL grab sample through a nitrocellulose membrane (Entis, 1990; Meinersmann et al., 2008). A previous study has successfully identified *Salmonella* prevalence using DNA extracted from resuspended sediment in Bangladesh following membrane filtration with no enrichment (Amin et al., 2020). The use of the modified Moore swabs in this study provided an advantage of filtering 10L of water, increasing the likelihood of detecting *Salmonella* when present at low concentrations (Sharma et al., 2020). The use of selective enrichment is advantageous for *Salmonella* recovery as the growth of nontarget organisms is suppressed, which may explain why *Salmonella* was not detected in previous studies. Therefore, this current study represents the first to recover and characterize *Salmonella* in these rivers.

Recently, a collaborative research effort, known as the Joint Institute for Food Safety and Applied Nutrition (JIFSAN), has brought together research groups throughout Latin America, the University of Maryland, and the US Food and Drug Administration. This group has carried out surveillance *Salmonella* testing in surface water in Chile, Brazil, and Mexico, utilizing similar sampling and culturing methods as those described in this study (Toro et al., 2022). One of these studies similarly compares rates of

Salmonella incidence in urban areas (29.1%) compared to agricultural areas (27.0%) (Toro et al., 2022). That study found that seasonality and water type (creek or canal versus river or pond) were the most significant contributors to *Salmonella* presence. In another integrated study conducted in Chile, Brazil, and Mexico, samples were taken from surface waters and showed variable *Salmonella* prevalence: 33%, 63%, and 68%, respectively (Chen et al., 2024). Notably, serovars Infantis and Typhimurium were frequently identified in both the integrated study and this current work. Additional work in Mexico has surveyed *Salmonella* prevalence and serovar diversity in multiple projects (González-López et al., 2022; Jiménez et al., 2014). These studies have identified 24 and 27 serovar from samples collected in 2008-2009 and 2018-2019, respectively. An additional study compared isolates of serovar Oranienburg collected in these studies and found close genomic relationships, regardless of the year of isolation (González-Torres et al., 2023).

Previous studies in Ecuador identifying *Salmonella* serovars have been limited to those in animal agriculture. These studies have reported the presence of many serovars identified in this work, including Infantis, Uganda, I 4,[5],12:i:-, Typhimurium, Manhattan, and Derby (Medina-Santana et al., 2022; Vinueza-Burgos et al., 2016, 2024). Importantly, serovar Amager was identified for the first time in Ecuador in this study; however, this serovar has been isolated from chicken carcasses in Venezuela (Boscán-Duque et al., 2007) and pigs in Colombia (Chamorro-Tobar et al., 2024). The largest *Salmonella* sequence repositories are the National Center for Biological information (NCBI) and Enterobase. While they provide a global repository for whole genome sequences, they contain significantly fewer *Salmonella* isolates from Ecuador and other

countries in Latin America, which limits possible comparative analyses between studies. This highlights a need for greater equitable pathogen surveillance across the world to improve food safety on a global level.

WGS results demonstrated that some serovars that were collected from the same river were represented by up to three different cgMLSTs. Interestingly, highly related isolates of serovars Typhimurium, Infantis, and Anatum were found in both rivers. While these rivers eventually merge, the sampling sites were upstream of this location, suggesting that a shared source of transmission may be responsible for these findings such as wildlife or other animal activities, or via humans (Kagambèga et al., 2017). Additionally, evidence of persistence and transmission were identified throughout this study. Identical cgMLSTs were found at the same sampling points on different days, suggesting the reintroduction or persistence of the same strain over multiple days. As the river flows downstream, we would expect any persistent *Salmonella* to be somewhat stationary, such as within biofilms on sediment (Gaertner et al., 2011; Sha et al., 2013). Given that sampling was conducted in moving water and not the sediment at the base, additional serovars or potentially persistent strains could be identified with alternative sampling methods. Evidence of *Salmonella* transmission was supported by finding identical or highly related cgMLSTs at different sampling locations on the same day. This could also be explained by regular contamination events from a common host.

This study demonstrates the importance of routine environmental *Salmonella* surveillance to better understand exposure to pathogenic bacteria, such as *Salmonella*. During the week we sampled, *Salmonella* prevalence was high and we also isolated multiple clinically relevant serovars. In many cases, more than one serovar was identified

from a sample, highlighting the need to more fully elucidate multiserovar *Salmonella* populations within environmental samples. While limited to four sequential sampling days, this study establishes a potential framework for more routine sampling of surface water sources. Longitudinal studies would be helpful in determining whether any seasonal or weather patterns influence *Salmonella* in these rivers. Patterns of persistence and transmission were also identified throughout this study, suggesting a need to identify routes of *Salmonella* contamination in these rivers and apply management strategies to reduce human health risks associated with contaminated surface water.

4.7 Data availability statement

Whole genome sequences are available on NCBI under the following isolate names: CFSAN137150-CFSAN137204.

4.8 Funding

This work was supported through funds from the University of Georgia to NWS. Sequencing for this project was funded through the University of Georgia Graduate School summer research grant awarded to JCS.

4.9 Acknowledgements

We thank members of the Vinueza lab for their technical assistance and Dr. Chris Grim at the FDA-CFSAN for sequencing support.

4.10 Competing interest statement

NWS has a licensing agreement with Ancera Inc.

4.11 Author Contributions

JCS, DAV, and NWS conceived and planned the study. JCS and DAV performed the experiments, completed the analysis and wrote the first draft of the paper. CVB

contributed laboratory support in Ecuador. All authors reviewed, edited, and approved the paper.

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CHAPTER 5

CONCLUSIONS

5.1 Conclusions

Salmonella enterica is a significant human pathogen, causing an estimated 176 million global human illnesses and 290,000 deaths annually (CDC, 2024). In the United States, it is a leading bacterial foodborne pathogen, contributing to 1.35 million cases each year. In addition to being commonly identified in food animals, this pathogen is ubiquitous in the environment, including wildlife and water. Illness often occurs following consumption of contaminated food, including meat and poultry, which contribute to 43.7% of estimated illnesses from foodborne outbreaks (IFSAC, 2024). This is expected, as sources such as poultry, cattle, and swine can carry *Salmonella* in their gastrointestinal tract as part of their natural microbiomes. Surprisingly, produce accounts for 43.4% of estimated human illnesses, suggesting that transmission from contaminated animal feces occurs during the growing, harvesting, or packing stages of production. As the preparation of produce often does not include cooking, pathogenic contaminants are more likely to remain viable, leading to a greater potential for human illness (Devarajan et al., 2023). Because of this, it is critical to prevent initial contamination by reducing the risk of transmission from various environmental routes. Relative to meat and poultry, these vectors are significantly understudied. The work presented in this dissertation advances our understanding of *Salmonella* ecology within an animal reservoir (wild birds) and in the environment (surface water) by evaluating risk factors that influence

Salmonella viability, identifying complex serovar populations, and establishing foundations of environmental monitoring in these environments.

Wild birds present a unique food safety challenge as traditional wildlife mitigation, such as fences and netting, do not prevent intrusion of this potential contamination source (Rivadeneira et al., 2018). Previous work has identified *Salmonella* and other foodborne pathogens in wild birds near crop fields, suggesting a potential role in transmission to produce (Gorski et al., 2011; Kirk et al., 2002; Navarro-Gonzalez et al., 2020; Rivadeneira et al., 2016; O. M. Smith, Edworthy, et al., 2020; O. M. Smith et al., 2022). In the second chapter of this dissertation, the role of wild birds in *Salmonella* transmission to produce fields in the Southeastern United States is detailed. Prior work investigated this role on the West Coast, where leafy greens such as lettuce, kale, and broccoli are the predominant crop. Alternatively, produce in the Southeast is represented by peppers, tomatoes, and cucumbers. In the Southeast study presented in this dissertation, wild bird feces were sampled from produce fields and cultured for the presence of *Salmonella*. Prevalence was low in this sample type, identifying viable *Salmonella* in 2.1% (16/773) of samples. This suggests that wild birds likely have a limited role in pathogen transmission in this region. *Salmonella* was significantly more likely to be recovered from fresh feces, indicating that this matrix is particularly susceptible to desiccation. This is a contrast from studies showing long-term recovery of *Salmonella* in composited bird feces up to 291 days (Oni et al., 2015; Topalcengiz et al., 2020). The results of the model were further supported by GLMM models from weather data that identified decreased precipitation as a significant factor in reduced *Salmonella* prevalence, suggesting that weather may play a critical role in risk assessment and

pathogen transmission. Molecular detection of *Salmonella* by PCR of the *invA* gene identified 59 additional samples containing non-culturable *Salmonella* signatures. This demonstrates the relevance of culture-based surveillance to avoid overestimating risk attributed to wild birds, and is a current dispute in food safety (O. M. Smith, Snyder, et al., 2020). Additionally, *Salmonella* serovar populations were assessed in culture-positive samples and the majority of these samples were found to contain multiple serovars. Results from this data showed that while the presence of viable *Salmonella* is low, these populations can include clinically relevant serovars such as Enteritidis, Typhimurium, Newport, Saintpaul, Infantis, and Muenchen. Finally, this work aimed to identify wild bird species associated with higher transmission risk. To accomplish this, a combination approach of in-field point counts and molecular identification via PCR of the gene encoding the cytochrome C oxidase subunit I identified 57 bird species, including 10 species from feces containing *Salmonella*. Species associated with on-farm structures and direct crop interaction were assigned the highest risk category due to their increased likelihood of transmitting *Salmonella* to produce. This suggests that targeted mitigation towards these species would be the most effective and efficient in reducing food safety risks from wild birds. While weather factors vary between regions, bird species associated with large monoculture farms, along with those found in West Coast studies, were similar to species identified here (Flohre et al., 2011; Gonthier et al., 2019; Olimpi et al., 2024; O. M. Smith, Edworthy, et al., 2020). This congruence of commonly identified bird species suggests that improved risk management could have a widespread impact on food safety.

Conclusions from this study showed that *Salmonella* transmission through defecation in produce fields is low, suggesting that risk management strategies should be targeted towards one or more alternative sources. Limitations of this study included species identification, as point counts identified species interacting in and around crop fields but not necessarily those contributing to fecal contamination on the produce. Molecular species identification demonstrated a direct assessment of species defecating in the fields; however, this method failed in a majority of samples, likely due to DNA degradation of the target sequence (Hebert et al., 2003; IVANOVA et al., 2007; Joo & Park, 2012; KERR et al., 2007). This method did provide an advantage over studies that trap and collect samples from live birds: while those studies were able to identify species, it was not necessarily indicative of the birds defecating on the produce. Future work on this topic is needed to better categorize *Salmonella* prevalence and multiserovar populations in wild birds. By continuing to collect both sporadic fecal samples and direct collections from wild birds, the development of a robust *Salmonella* database from this source would improve risk assessment. This would facilitate more thorough source attribution investigations if wild birds are involved in future outbreaks. Additionally, further analysis is needed to evaluate human activities associated with increased *Salmonella* in wild birds, such as urbanization and agricultural intensification. While these activities have been assessed in a few studies (Hernandez et al., 2016; O. M. Smith, Edworthy, et al., 2020), regional differences may contribute to variable influence by wild birds, suggesting a need for more widespread research in this field.

Surface water has been regularly identified as a source of *Salmonella* around the world (Casanova et al., 2020; Chen et al., 2024; Gorski et al., 2022; Haley et al., 2009;

Jokinen et al., 2015; Mahagamage et al., 2020; Micallef et al., 2012; Ruiz et al., 1987; Somda et al., 2021). Additionally, a wide range of serovars and incidence of AMR have been reported, demonstrating a need to further evaluate this *Salmonella* reservoir as a food safety concern (Berge et al., 2006; Cho et al., 2020; Li et al., 2014; Micallef et al., 2012). Chapter three introduces a study that investigated *Salmonella* prevalence, serovar complexity, and antibiotic resistance in four distinct creeks in the Southeastern United States. Prevalence was high in each creek, ranging from 61% to 78%, where the lowest *Salmonella* prevalence was identified in the urban creek and the highest in a rural creek. Seasonality significantly influenced *Salmonella* prevalence, for example, the lowest prevalence was found in summer months, in contrast with prior work (Deaven et al., 2021; Haley et al., 2009). Deep serotyping identified multiserovar populations in almost 90% of positive samples; a proportion higher than that of previously studied subjects, including poultry, cattle, and wild birds (Cason et al., 2024; Obe et al., 2023; Siceloff et al., 2021, 2022; J. C. Smith et al., 2023). These populations contained high complexity, averaging almost four serovars per sample and ranging up to 13 serovars in a single sample. As this complexity has not regularly been identified from other sources, it is likely that surface water can act as a collection point for serovars being shed by multiple sources in the environment. Importantly, many clinically relevant serovars were identified, demonstrating the importance of water surveillance to identify previously understudied routes of *Salmonella* transmission. Land attribution characteristics and weather variables were modeled to identify predictive factors for *Salmonella* risk, finding that increased precipitation and humidity were significantly associated with higher prevalence and complexity. This agreed with prior studies (Deaven et al., 2021; Strawn et

al., 2013; Weller et al., 2020); however, proximal land use was less significantly associated with any *Salmonella* trends. This was surprising, considering these creeks were each enriched for a unique land use, including a national forest, a suburban population, and animal agriculture. Limited access by humans and the absence of animal agriculture would likely prevent the introduction of antibiotic resistance to this creek; however, results suggest a natural transmission of AMR genes. This finding is supported by other studies where AMR transmission was identified in natural environments (Cho et al., 2022; Hwengwere et al., 2022).

Overall, the results of this study demonstrate a high prevalence and complexity of pathogenic serovars in various surface water sources. The dissimilarity between serovars found in this source and those recovered from food animals underscores the need for a One Health approach for *Salmonella* surveillance to identify food safety risks and implement effective management strategies. Further surveillance of environmental and wildlife sources is needed to better understand the transmission of *Salmonella* between the agricultural and environmental interface. Future work is needed to identify *Salmonella* dynamics that influence survival in aquatic environments, such as adaptations to overcome limited nutrient availability or resistance to osmotic stress. These genomic adaptations that promote viability in water may allow a strain to outcompete others in the source, leading to its expansion within the niche.

As previously identified, a variety of contamination sources threaten surface water, making these sources of freshwater unsafe for human use. While many countries around the world have established water sanitization practices, the United Nations estimates that 2.2 billion people in the world do not have access to clean water (Burden

of Disease Attributable to Unsafe Drinking Water, Sanitation and Hygiene, n.d.). This results in the use of unsafe water, which leads to higher rates of typhoid fever and other diseases. To differentiate characteristics that influence water safety in agricultural and urban rivers, chapter four discusses a study that measured *Salmonella* prevalence and serovar diversity of two rivers in Pichincha province, Ecuador. In this investigation, three-quarters of samples were positive for *Salmonella,* where the urban river was found to have higher prevalence and complexity compared to the agricultural river. Whole genome sequencing of 54 isolates identified eight serovars, where nearly half of positive samples contained two or more serovars. Importantly, analysis of the core genome between isolates of the same serovar identified characteristics of transmission in multiple strains, including serovar Typhimurium and its monophasic variant, I 4,[5],12:i:-. The presence of highly related isolates consistently found in the same site for multiple days also suggests potential reintroduction events or persistence within these rivers. This suggests that persistent strains may be maintained for extended periods, contributing to increased safety concerns for these rivers.

Data from this study demonstrated, for the first time, high *Salmonella* serovar prevalence that was also reflected in many serovars being isolated in two rivers in Ecuador. This reinforces the need to protect public water sources and the need for regular surveillance of surface water for public health risks. As a result of previous studies that identified unsafe levels of chemical and microbial water quality in rivers including the Rìo Machángara (Borja-Serrano et al., 2020; Ortega-Paredes et al., 2020; Vinueza et al., 2021), an Ecuadorian court ruled that protection of this urban river is a legal requirement. This is an encouraging first step for making publicly accessible water sources safe. Future

work is needed to assess the efficacy of water treatment to reduce the prevalence and complexity of *Salmonella* populations in contaminated rivers. To further support this aim, more thorough surveillance of rivers, including upstream sites and points of convergence, would provide a system of tracking contamination routes in these rivers.

Regulations in the United States, including FSMA and GAPs, seek to reduce food safety concerns from various routes of transmission. To do so, risk assessment strategies for irrigation water, soil amendments, and worker hygiene are clearly described; however, wildlife management is uniquely non-specific. This is, in part, due to the variety of animal species that may contribute to risk, making an all-encompassing mitigation approach difficult (Gordus, 2011; Langholz & Jay-Russell, 2013). Wild bird exclusion strategies are unrealistic compared to other wildlife, as fences and other barriers do little to prevent avian movement into fields (Rivadeneira et al., 2018; Varriano et al., 2025). As a result, many research groups have characterized *Salmonella* prevalence within wild birds to assess food safety risks (Kirk et al., 2002; Maurer et al., 2015a; Navarro-Gonzalez et al., 2020; Olimpi et al., 2024; O. M. Smith, Edworthy, et al., 2020; O. M. Smith, Olimpi, Navarro-Gonzalez, et al., 2022). Research in this dissertation further characterized prevalence; however, deep serotyping set this work apart by identifying multiserovar populations within wild birds. This demonstrated a potential limitation in previous studies, where serovars may not have been detected. While the incidence of *Salmonella* was low in this sample type, the presence of clinically relevant serovars within positive samples highlights the need to identify the entire population rather than only the most abundant serovar. This research found an association between the freshness of feces and viable *Salmonella*, where the pathogen was significantly less

likely to be cultured from dried feces. Additionally, no incidence of movement from the feces to produce on the plant or to neighboring plants was identified. These findings suggest that the risk associated with wild bird feces in produce fields is low, and this risk is further reduced as feces become desiccated. The results of these findings could be used to support arguments for limiting no-harvest zones following fecal contamination on produce and reducing pressure to interfere with wild bird movement (Hamilton et al., 2015; Olimpi et al., 2024). Studies testing *Salmonella* viability and transmission in controlled environments will be needed to further support these findings.

Surface water is a critical component in food systems for the irrigation of produce and as collection points for runoff. As such, *Salmonella* prevalence has been previously characterized in many studies, both within the United States and internationally (Chen et al., 2024; Haley et al., 2009; Mahagamage et al., 2020; McEgan et al., 2014; Micallef et al., 2012; Murphy et al., 2022; Somda et al., 2021; Vadde et al., 2019). This dissertation highlights two surface water studies: one in the Southeastern United States and one in the Pichincha region of Ecuador. The use of deep serotyping technology advanced the understanding of *Salmonella* populations in the United States, where only one prior study had assessed these complex populations (Deaven et al., 2021). This study resolved a limitation of the previous investigation, where smaller creeks were sampled instead of a large river. This difference allowed for a more comprehensive analysis of the entire *Salmonella* population in the water source. Additionally, monthly sampling over two years allowed for more robust modeling of factors that influence *Salmonella* prevalence and complexity. Modeling suggested that season and weather conditions, including precipitation and humidity, influenced *Salmonella* more than neighboring land

attribution. This finding may be helpful for establishing surface water management efforts, where these risk factors can be implemented to determine when surface water is likely to be unsafe for agricultural use. The study in Ecuador isolated multiple serotypes in single samples and analyzed these using whole genome sequencing for the first time. While rivers in this region have been evaluated for water quality in previous work (Borja-Serrano et al., 2020; Ortega-Paredes et al., 2020; Vinueza et al., 2021), the characterization of *Salmonella* was unclear, and potentially underestimated. Genomic analysis suggested potential downstream transmission of highly related isolates; however, further studies are needed to evaluate both multiserovar populations and transmission of *Salmonella* in surface water, as these features likely play a critical role food safety.

The body of work presented in this dissertation addresses significant knowledge gaps in human health and food safety. These studies identified environmental sources of complex *Salmonella* populations in wild birds and surface water containing clinically relevant serovars. Additionally, factors contributing to food safety risks in these reservoirs were identified, which can be utilized in farm-specific risk management strategies, including species-specific mitigation of wild birds and the identification of weather conditions that increase the likelihood of irrigation water contamination. Finally, this research establishes a framework for a One Health approach to *Salmonella* surveillance, which can be utilized to reduce the incidence of *Salmonella* in food safety.

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APPENDIX A

Supplemental Material for Chapter 2

Supplemental Table 2.1. ΔAICc and Akaike weight *(ω)* for detection of *Salmonella* and weather effect models.

Model	*β* Value (SE, p)			ΔAICc	ω
	Precipitation	Humidity	Wind		
Humidity		-0.2898 (0.179, 0.105)		0	0.379
Precipitation + Humidity	0.2348 (0.190, 0.215)	-0.3375 (0.183, 0.065)		0.45	0.289
Precipitation + Humidity + Wind	0.2788 (0.197, 0.158)	-0.2833 (0.190, 0.129)	-0.2006 (0.208, 0.335)	1.54	0.169
Precipitation	0.165 (0.189, 0.381)			1.88	0.139

Supplemental Table 2.2. Genome assembly statistics

	Genome Size (Mb)	Number of contigs	N50 (Kb) Value
JSBird1	4.95	132	103.59
JSBird2	4.99	193	66.87
JSBird3	4.74	129	114.65
JSBird4	4.74	122	115.51
JSBird5	4.75	40	735.89
JSBird6	4.77	101	140.49
JSBird7	4.77	102	159.79
JSBird8	4.77	137	98.89
JSBird9	4.77	122	127.85
JSBird10	4.74	121	94.07
JSBird11	4.67	37	613.89
JSBird12	5.03	129	101.89
JSBird13	4.84	184	86.61
JSBird14	4.91	177	71.96
JSBird15	4.84	149	102.96
JSBird16	4.61	268	36.06
JSBird19	4.80	55	405.57
JSBird20	4.85	57	430.46
JSBird21	4.92	59	417.46

North Georgia	May	June	July	August	September	October
Temperature (°F)	3.88	4.59	2.05	-0.83	0.56	-1.71
Precipitation (in)	-1.41	-3.05	0.12	-2.69	-0.96	-5.49
Humidity (%)	4.61	-8.84	-1.18	2.95	-8.58	-13.71
Wind (mph)	-0.09	0.19	0.45	-0.55	1.37	0.93

*Sampling in 2021 took place between June 13th-October 14th
*Sampling in 2022 took place between May 31st-August 24th

South Georgia/Florida	May	June	July	August	September	October
Temperature (°F)	2.12	4.32	2.38	1.00	0.11	-0.48
Precipitation (in)	4.82	-2.94	-0.89	-3.08	-2.34	-1.10
Humidity (%)	9.48	-3.68	-2.43	-2.79	-6.06	-9.24
Wind (mph)	-0.87	-0.19	0.11	-1.27	1.86	0.59

*Sampling in 2021 took place between May 26th-September 18th
*Sampling in 2022 took place between May 23rd-June 29th

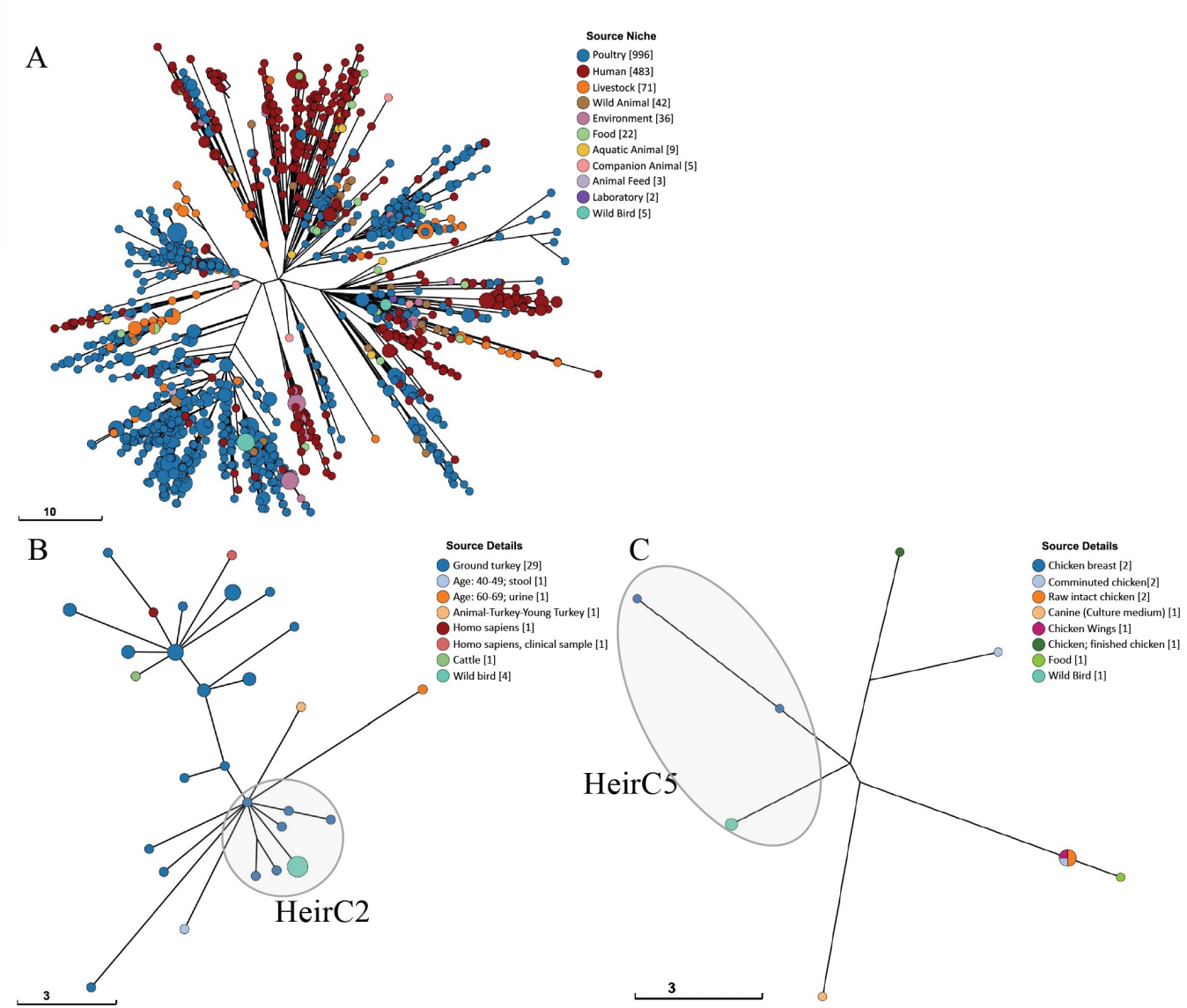
Tennessee	May	June	July	August	September	October
Temperature (°F)	4.13	3.23	0.97	-1.06	0.17	-2.87
Precipitation (in)	0.04	-0.03	0.20	-0.24	0.03	-0.08
Humidity (%)	2.91	-8.19	4.02	0.16	-7.25	-19.50
Wind (mph)	0.64	0.47	0.63	0.23	1.12	0.81

*Sampling in 2021 took place between June 23rd-October 1st
*Sampling in 2022 took place between June 22nd-August 30th

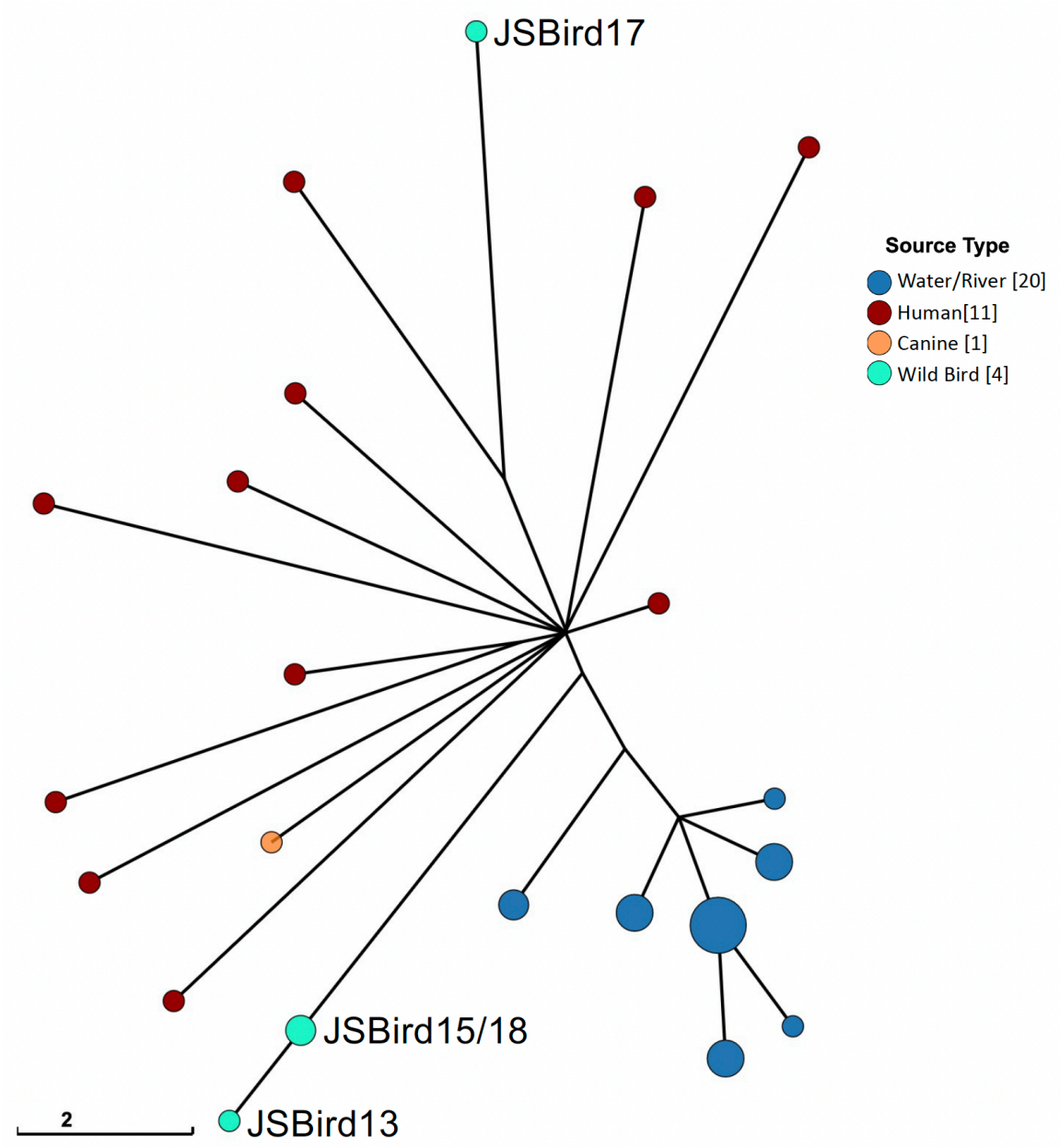
South Carolina	May	June	July	August	September	October
Temperature (°F)	2.69	5.00	2.55	-1.05	-1.57	-4.48
Precipitation (in)	0.30	-1.50	0.94	-0.28	-1.10	-1.42
Humidity (%)	4.40	-7.37	2.41	0.04	-2.27	-6.38
Wind (mph)	0.62	-0.83	0.86	0.16	1.32	-0.01

*Sampling in 2021 took place between August 3rd-October 21st
*Sampling in 2022 took place between June 9th-August 10th

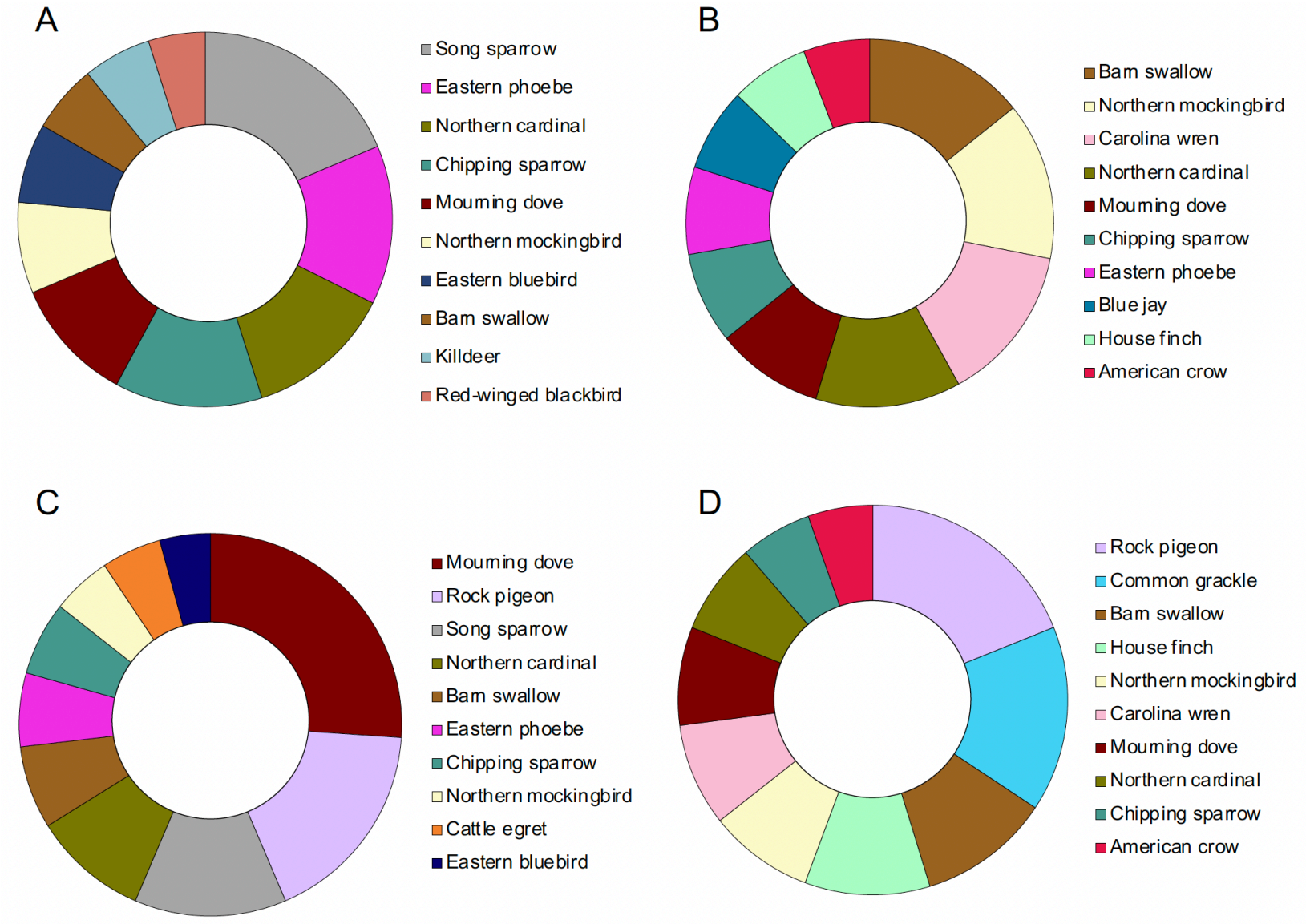
Supplemental Figure 2.1. Comparison of weather conditions for sampling regions. Temperature, precipitation, humidity, and wind were compared between 2021 and 2022 collection seasons. Averages for each variable (except precipitation where cumulative rainfall was calculated) are shown in under the respective month for each region. Negative numbers (red boxes) indicate a lower value in 2022 and positive numbers (blue boxes) indicate a higher value in 2022. Dates of the first and last sampling in each region for both years are listed to the right of the weather values.


A
Source Niche
Poultry [996]
Human [483]
Livestock [71]
Wild Animal [42]
Environment [36]
Food [22]
Aquatic Animal [9]
Companion Animal [5]
Animal Feed [3]
Laboratory [2]
Wild Bird [5]
10
B
Source Details
Ground turkey [29]
Age: 40-49; stool [1]
Age: 60-69; urine [1]
Animal-Turkey-Young Turkey [1]
Homo sapiens [1]
Homo sapiens, clinical sample [1]
Cattle [1]
Wild bird [4]
HeirC2
3
C
Source Details
Chicken breast [2]
Comminuted chicken[2]
Raw intact chicken [2]
Canine (Culture medium) [1]
Chicken Wings [1]
Chicken; finished chicken [1]
Food [1]
Wild Bird [1]
HeirC5
3

Supplemental Figure 2.2. Serovar Hadar isolates include variable poultry relationships between turkey and chicken isolates. (A) Phylogenetic relationships between serovar Hadar isolates from wild bird feces in this study (turquoise circles) to isolates on Enterobase shown on a GrapeTree plot. (B) JSBird3, 4, 5, and 10 isolates and Enterobase isolates with up to 10 cgMLST allelic differences. (C) JSBird11 and Enterobase isolates with up to 10 cgMLST allelic differences. (Scale bar) Number of cgMLST allelic differences.



Supplemental Figure 2.3. Serovar Newport isolates are similar to human and environmental isolates. Serovar Newport isolates from wild bird feces (turquise circles) with phylogenetic relationships to isolates within Enterobase shown on a GrapeTree plot. (Scale bar) Number of cgMLST allelic differences.


A
Song sparrow
Eastern phoebe
Northern cardinal
Chipping sparrow
Mourning dove
Northern mockingbird
Eastern bluebird
Barn swallow
Killdeer
Red-winged blackbird
B
Barn swallow
Northern mockingbird
Carolina wren
Northern cardinal
Mourning dove
Chipping sparrow
Eastern phoebe
Blue jay
House finch
American crow
C
Mourning dove
Rock pigeon
Song sparrow
Northern cardinal
Barn swallow
Eastern phoebe
Chipping sparrow
Northern mockingbird
Cattle egret
Eastern bluebird
D
Rock pigeon
Common grackle
Barn swallow
House finch
Northern mockingbird
Carolina wren
Mourning dove
Northern cardinal
Chipping sparrow
American crow

Supplemental Figure 2.4. Wild bird point counts. Top 10 species for the following categories: (A) Visits with species observation in-field, (B) visits with species observation off-field, (C) in-field individual observations, and (D) off-field individual observations.

APPENDIX B

Supplemental Material for Chapter 3

Supplementary Materials and Methods

Sample collection and *Salmonella* isolation

A modified Moore swab and peristaltic pump were used to collect 10 L of creek water from each site (n = 456 samples)(Figure S1A). Modified Moore swabs were placed at approximately midchannel resting on the sediment for the duration of pumping. The swabs were stored on ice until returning to the lab, where 100 ml of buffered peptone water (BPW; Neogen, Lansing, MI) and novobiocin (40 mg/L; Thermo Scientific Chemicals, Waltham, MA) were added. The swabs were hand-massaged for 1 minute, then incubated, shaking at 42°C for 20-24 hours. Following incubation, 1 and 0.1 mL of BPW were transferred into 10 mL of tetrathionate (TT; Hardy Diagnostics, Santa Maria, CA) and Rappaport-Vassiliadis (RV; Hardy Diagnostics, Santa Maria, CA) broth, respectively, and statically incubated at 37°C for 20-24 hours. Starting at month 13, 0.1 mL of enriched BPW was inoculated into a modified semisolid Rappaport-Vassiliadis (MSRV; Hardy Diagnostics, Santa Maria, CA) plate and incubated at 37°C for 20-24 hours. *Salmonella* presence was tested by streaking the TT and RV cultures, along with any presumptive *Salmonella* growth from the MSRV plates, onto xylose lysine tergitol-4 (XLT-4; Hardy Diagnostics, Santa Maria, CA) plates, followed by incubation at 37°C for 24-48 hours. During the first 12 months, if the TT and RV enrichments did not result in presumptive *Salmonella* colonies, then the enriched BPW culture was directly plated onto XLT-4 (Figure S1B). Three colonies were selected and restreaked for isolation onto new XLT-4 plates. After incubation at 37°C for 24-48 hours, a single colony was streaked onto Luria-Bertani (LB; Hardy Diagnostics, Santa Maria, CA) agar, then confirmed with serum agglutination (BD Difco, Franklin Lakes, NJ). All confirmed *Salmonella* isolates

were saved via glycerol stocks in a -80°C freezer. From all enrichments, 1 mL was removed and centrifuged at 18,000 rcf for 3 minutes, then the supernatant was removed, and the pellets were stored at -20°C for later use.

Antimicrobial susceptibility testing

One *Salmonella*-positive isolate from each site in each month was selected for antimicrobial susceptibility testing (AST). When selecting isolates, preference was given to the first colony recovered from TT, followed by RV and BPW. To measure the susceptibility of the *Salmonella* isolates, the Kirby-Bauer disk diffusion assay was used to test ten different antibiotics: ampicillin (10 μg), amoxicillin-clavulanic acid (20/10 μg), ceftriaxone (30 μg), gentamicin (10 μg), streptomycin (10 μg), tetracycline (30 μg), ciprofloxacin (5 μg), sulfamethoxazole-trimethoprim (25 μg), nalidixic acid (30 μg), and chloramphenicol (30 μg) (BD Diagnostics, Franklin Lakes, NJ). Bacterial overnight cultures were standardized to 0.5 McFarland (ThermoScientific Remel, Lenexa, KS) and spread plated onto Mueller-Hinton agar (BD BBL, Franklin Lakes, NJ). Antibiotic disks were placed on the plates, followed by incubation for 18-22 hours at 37°C. Zones of inhibition were measured and the results were interpreted with the CSLI standards to determine if isolates were susceptible, intermediate, or resistant (CLSI, 2024). Isolates were categorized as multidrug resistant (MDR) if they were resistant to three or more classes of antibiotics. For subsequent analyses, an isolate with intermediate resistance was considered as “susceptible”.

Whole genome sequencing

To confirm the serovar of each isolate with antimicrobial resistance and compare the relatedness to publicly available *Salmonella* genomes, a single colony was picked from

the LB plate used for *Salmonella* confirmation, inoculated in an LB agar stab, and shipped to the FDA Center for Food Safety and Applied Nutrition (FDA-CFSAN, now Human Food Program) for library preparation and sequencing. Raw Illumina sequence reads were uploaded to NCBI (BioProject PRJNA186035) for further processing through the Prokaryotic Genome Annotation Pipeline and the resulting assemblies were used for further analysis (Serovar Infantis: BioSample ; Serovar Typhimurium: BioSample).

Supplemental Table 3.1. Final generalized linear mixed model for *Salmonella* prevalence.

	Estimate	Std. Error	z value	Pr(>|z|)	OddsRatio	LowerCI	UpperCI
(Intercept)	0.15033	4.522179	0.033243	0.973481	1.162	0	8210.097
Spring	0.739343	0.259482	2.849307	0.004381	2.094	1.26	3.479
Summer	-2.772454	0.300477	-9.226852	2.79E-20	0.063	0.035	0.113
Winter	0.352735	0.261828	1.347204	0.177915	1.423	0.852	2.379
Temp	-0.05905	0.070748	-0.834652	0.403913	0.943	0.82	1.083
Humidity	-0.132516	0.104371	-1.269668	0.204203	0.875	0.714	1.073
Wind	0.18682	0.046893	3.983976	6.78E-05	1.206	1.1	1.322
Precip	0.188953	0.033516	5.637634	1.72E-08	1.208	1.13	1.291
Hay pasture	0.033582	0.005133	6.542097	6.07E-11	1.035	1.024	1.045
Mixed forest	0.115867	0.020004	5.792277	6.94E-09	1.123	1.08	1.168
I(Temp^2)	0.000896	0.000494	1.81315	0.069809	1.001	1.001	1.001
I(Humidity^2)	0.00143	0.000733	1.949504	0.051235	1.001	0.999	1.003

Supplemental Table 3.2. Final generalized linear mixed model for *Salmonella* population complexity.

	Estimate	Std. Error	df	t value	Pr(>|t|)	OddsRatio	LowerCI	UpperCI
(Intercept)	-3.5375163	1.27479562	106.278724	-2.7749674	0.00652395	0.029	0.002	0.354
Spring	0.74102359	0.30118276	248.021969	2.46037848	0.01456161	2.098	1.163	3.785
Summer	-0.507279	0.35467614	248.931894	-1.4302598	0.15389642	0.602	0.3	1.208
Winter	-0.7368902	0.3038573	247.200975	-2.4251194	0.01602059	0.479	0.264	0.868
Humidity	0.05412746	0.01270287	248.944623	4.26104181	2.89E-05	1.055	1.029	1.083
Wind	0.13041264	0.05581676	247.079948	2.33644212	0.02026832	1.139	1.02	1.271
Hay pasture	0.02960848	0.00755701	9.32887179	3.91801513	0.00328558	1.03	1.014	1.047
Mixed forest	0.14233459	0.03648874	4.71875035	3.90078175	0.0127588	1.153	1.074	1.237

Supplemental Table 3.3. Full list of serovars identified by USDA-FSIS in food animal samples and clinical cases, along with the serovars identified in samples from this study.

BEAM top 10	
Enteritidis	24780
Newport	15610
Typhimurium	12661
Javiana	7565
I 4, 5 12:i:-	5755
Infantis	5506
Oranienburg	4386
Saintpaul	3634
Braenderup	3595
Muenchen	3330

Beef	
Montevideo	103
Anatum	62
Muenchen	49
Dublin	30
Infantis	30
Newport	30
Cerro	29
Muenster	26
Kentucky	22
Typhimurium	19
Brandenburg	13
Agona	12
Uganda	12
Altona	9
Give	9
I 4,[5],12:i:-	9
Mbandaka	9
Meleagridis	7
Schwarzengrund	7
Other	6
Enteritidis	6
Eko	5
London	5
Barranquilla	4
Derby	4
Lubbock	4
Senftenberg	4
Thompson	4
Adelaide	3
Braenderup	3
Amsterdam	2
Bovismorbificans	2
Bredeney	2
Idikan	2
Johannesburg	2
Kiambu	2
Litchfield	2
Ohio	2
Orion	2
Reading	2
Saintpaul	2
Albany	1
Apapa	1
Bareilly	1
Berta	1
Blockley	1
Cannstatt	1
Corvallis	1
Eastbourne	1
Fresno	1
Gaminara	1
Heidelberg	1
Manhattan	1
Minnesota	1
Panama	1
Paratyphi B var. L(+) tartrate+	1
Poona	1
Takoradi	1

Chicken	
Kentucky	2615
Infantis	2578
Enteritidis	1015
Typhimurium	839
Schwarzengrund	381
Alachua	78
Thompson	78
I 4,[5],12:i:-	51
Johannesburg	51
Other	37
Braenderup	34
Hadar	27
Heidelberg	25
Mbandaka	20
Blockley	17
Senftenberg	17
Anatum	15
Montevideo	11
Agona	10
Muenchen	10
Newport	8
Cerro	7
Berta	6
Litchfield	6
Uganda	4
Eko	3
Muenster	3
Reading	3
Tennessee	3
Albany	2
Derby	2
Elomrane	2
Fresno	2
Liverpool	2
Oranienburg	2
Ouakam	2
Widemarsh	2
Barranquilla	1
Brandenburg	1
Chailey	1
Dublin	1
Gateshead	1
Give	1
Havana	1
Livingstone	1
London	1
Minnesota	1
Ohio	1
Orion	1
Oslo	1
Putten	1
Rissen	1
Roodepoort	1
Soerenga	1
Worthington	1

Pork	
Anatum	335
I 4,[5],12:i:-	273
Infantis	220
Derby	181
Johannesburg	166
Eko	153
London	151
Agona	110
Uganda	107
Typhimurium	78
Muenchen	62
Adelaide	57
Ohio	56
Worthington	56
Berta	51
Rissen	37
Schwarzengrund	37
Chailey	33
Brandenburg	28
Senftenberg	28
Mbandaka	21
Manhattan	20
Panama	20
Saintpaul	20
Litchfield	19
Reading	19
Soerenga	18
Bovismorbificans	17
Alachua	15
Cerro	14
Give	14
Montevideo	14
Newport	14
Other	13
Heidelberg	9
Kentucky	9
Kiambu	9
Braenderup	8
Muenster	8
Bredeney	6
Orion	6
Hadar	5
Liverpool	5
Altona	4
Barranquilla	4
Havana	4
Livingstone	4
Kedougou	3
Thompson	3
Cannstatt	2
Choleraesuis	2
Enteritidis	2
Indiana	2
Krefeld	2
Meleagridis	2
Minnesota	2
Tennessee	2
Gatuni	1
Hartford	1
Idikan	1
Poona	1

Turkey	
Hadar	99
Schwarzengrund	64
Infantis	63
Other	54
Senftenberg	50
Reading	47
Typhimurium	36
Muenchen	30
Uganda	26
I 4,[5],12:i:-	12
Anatum	8
Brandenburg	8
Montevideo	8
Albany	7
Newport	5
Worthington	5
Enteritidis	4
Kentucky	4
Orion	4
Ouakam	4
Cubana	3
Litchfield	3
Bovismorbificans	2
Heidelberg	2
Johannesburg	2
Liverpool	2
London	2
Muenster	2
Amsterdam	1
Barranquilla	1
Gateshead	1
Give	1
Olten	1
Rissen	1
Ruiru	1

Creek	
Rubislaw	163
Muenchen I	133
Montevideo II	126
Aqua/Inverness	96
Give I	70
Typhimurium	53
Gaminara	32
Infantis	31
Hartford	29
Braenderup	26
Mississippi II	23
Brazil I	22
Montevideo I	22
Alabama/Bareilly III	13
Oranienburg	13
Anatum	12
Thompson	12
Muenchen II	9
Newport II	9
Hadar	8
Agbeni	7
Kentucky I	7
Enteriditis	5
Kiambu	5
Luciana	5
Untypeable	5
Johannesburg/Urbana	2
Rissen	2
Senftenberg I	2
Uganda	2
Agona	1
Cubana	1
Derby III	1
Dublin	1
Java	1
Mbandaka	1
Poona	1
Saintpaul I	1
Saintpaul II	1
Senftenberg II	1

Supplemental Table 3.4. Select metadata downloaded from NCBI for serovar Infantis isolates included in Figure 3.4A.

BioSample	Collected by	Collection date	Location	Isolation type	Isolation source	SNP cluster	Assembly	Run	Isolate	Strain	Computed types
SAMN09444132	USDA-FSIS	2018	USA:NE	environmental/other	animal-swine-market swine	PDS000097345.3	GCA_008447705.1	SRR7358414	PDT000332042.1	FSIS11810649	antigen_formula=7:r:1,5,serotype=Infantis
SAMN22215935		2021-08	USA	clinical		PDS000097345.3	GCA_020465275.1	SRR16292359	PDT001148831.1	PNUSAS235240	antigen_formula=7:r:1,5,serotype=Infantis
SAMN24114991	Not Collected	2017	USA	clinical	Not Collected	PDS000097345.3	GCA_021719155.1	SRR17247291	PDT001232544.1	18-038875-057	antigen_formula=7:r:1,5,serotype=Infantis
SAMN25608883		2014	Slovenia	clinical		PDS000116014.1	GCA_024995025.1	SRR17880716	PDT001389277.1	S105	antigen_formula=7:r:1,5,serotype=Infantis
SAMN25608903		2015	Slovenia	clinical		PDS000116014.1	GCA_024992195.1	SRR17880860	PDT001389419.1	S125	antigen_formula=7:r:1,5,serotype=Infantis

Supplemental Table 3.5. Select metadata downloaded from NCBI for serovar Infantis isolates included in Figure S3.6A.

Yellow highlight indicates samples chosen for Figure S3.6B.

BioSample	Collected by	Collection date	Location	Isolation type	Isolation source	SNP cluster	Assembly	Run	Isolate	Strain	Computed types
SAMD00019585		2000	Japan:Kyusyu			PDS000116010.1	GCA_024958125.1	DRR022723	PDT001391554.1	1037	antigen_formula=7:r:1,5:serotype=Infantis
SAMD00019594		2000	Japan:Kyusyu			PDS000116035.2	GCA_024957805.1	DRR022740	PDT001391571.1	1082	antigen_formula=7:r:1,5:serotype=Infantis
SAMD00019610		1996	Japan:Kyusyu			PDS000116011.2	GCA_024957445.1	DRR022728	PDT001391559.1	407	antigen_formula=7:r:1,5:serotype=Infantis
SAMD00019612		1996	Japan:Kyusyu			PDS000116172.1	GCA_024958065.1	DRR022730	PDT001391561.1	425	antigen_formula=7:r:1,5:serotype=Infantis
SAMD00019617		1999	Japan:Kyusyu			PDS000116013.2	GCA_024958245.1	DRR022717	PDT001391548.1	900	antigen_formula=7:r:1,5:serotype=Infantis
SAMD00019623		2000	Japan:Kyusyu			PDS000116034.2	GCA_024957485.1	DRR022716	PDT001391547.1	G117	antigen_formula=7:r:1,5:serotype=Infantis
SAMD00019628		2000	Japan:Kyusyu			PDS000116032.1	GCA_024957465.1	DRR022758	PDT001391588.1	G60	antigen_formula=7:r:1,5:serotype=Infantis
SAMD00019643		2000	Japan:Kyusyu			PDS000116159.1	GCA_024956985.1	DRR022780	PDT001391610.1	R116	antigen_formula=7:r:1,5:serotype=Infantis
SAMD00019646		2000	Japan:Kyusyu			PDS000116163.1	GCA_024956945.1	DRR022774	PDT001391604.1	R63	antigen_formula=7:r:1,5:serotype=Infantis
SAMEA104349014		2017	Ireland	clinical	human	PDS000117330.1	GCA_025075735.1	ERR2173672	PDT000452099.1		antigen_formula=7:r:1,5:serotype=Infantis
SAMEA104394951		2011	Germany		food	PDS000027773.3	GCA_010155115.1	ERR2200349	PDT000277278.3		antigen_formula=7:r:1,5:serotype=Infantis
SAMEA104394959		2015	Germany		food	PDS000018208.4	GCA_010417045.1	ERR2200357	PDT000277286.3		antigen_formula=7:r:1,5:serotype=Infantis
SAMEA104394974		2016	Germany		feed	PDS000027745.7	GCA_010461465.1	ERR2200372	PDT000277301.3		antigen_formula=7:r:1,5:serotype=Infantis
SAMEA104452261	DTU	1/3/18	Denmark			PDS000073471.158	GCA_010142175.1	ERR2233202	PDT000276305.2	Infantis	antigen_formula=7:r:1,5:serotype=Infantis
SAMEA111505873	Hospital of The Social Welfare Institute	11/19/20	Paraguay	clinical	feces	PDS000125084.1	GCA_026352875.1	ERR10432297	PDT001493329.1		antigen_formula=7:r:1,5:serotype=Infantis
SAMEA112357034		2008	Spain		Pigs	PDS000026845.91	GCA_033185015.1	ERR10799944	PDT001961605.1	VISAVET_VED8_029555SM2	antigen_formula=7:r:1,5:serotype=Infantis
SAMEA11580037	National Institute for Communicable Disease, South Africa	3/29/04	South Africa	clinical	Human Rectal Swab	PDS000109857.5	GCA_023492265.1	ERR7658218	PDT001310290.1		antigen_formula=7:r:1,5:serotype=Infantis
SAMEA11580041	National Institute for Communicable Disease, South Africa	11/29/04	South Africa	clinical	Human Stool	PDS000109886.5	GCA_023492275.1	ERR7658222	PDT001310294.1		antigen_formula=7:r:1,5:serotype=Infantis
SAMEA11580042	National Institute for Communicable Disease, South Africa	11/29/04	South Africa	clinical	Human Rectal Swab	PDS000109864.9	GCA_023492025.1	ERR7658223	PDT001310295.1		antigen_formula=7:r:1,5:serotype=Infantis
SAMEA11580050	National Institute for Communicable Disease, South Africa	12/15/06	South Africa	clinical	Human Stool	PDS000109881.5	GCA_023491805.1	ERR7658236	PDT001310308.1		antigen_formula=7:r:1,5:serotype=Infantis
SAMEA11580163	National Institute for Communicable Disease, South Africa	5/7/12	South Africa	clinical	Human Stool	PDS000113776.2	GCA_024225515.1	ERR7671393	PDT001357659.1		antigen_formula=7:r:1,5:serotype=Infantis
SAMEA11580170	National Institute for Communicable Disease, South Africa	12/14/12	South Africa	clinical	Human Blood culture	PDS000114840.1	GCA_024479325.1	ERR7671403	PDT001370691.1		antigen_formula=7:r:1,5:serotype=Infantis
SAMEA11580171	National Institute for Communicable Disease, South Africa	1/16/13	South Africa	clinical	Human Stool	PDS000077667.3	GCA_024267295.1	ERR7671404	PDT001358639.1		antigen_formula=7:r:1,5:serotype=Infantis
SAMEA11580191	National Institute for Communicable Disease, South Africa	10/15/13	South Africa	clinical	Human Stool	PDS000113888.3	GCA_024271165.1	ERR7671436	PDT001359668.1		antigen_formula=7:r:1,5:serotype=Infantis
SAMEA11580200	National Institute for Communicable Disease, South Africa	6/18/14	South Africa	clinical	Human Stool	PDS000114825.1	GCA_024440375.1	ERR7671471	PDT001365695.1		antigen_formula=7:r:1,5:serotype=Infantis
SAMEA11580253	National Institute for Communicable Disease, South Africa	11/25/08	South Africa	clinical	Human Stool	PDS000109873.5	GCA_023486465.1	ERR7658235	PDT001310317.1		antigen_formula=7:r:1,5:serotype=Infantis
SAMEA11580260	National Institute for Communicable Disease, South Africa	2/18/07	South Africa	clinical	Human Stool	PDS000109861.7	GCA_023491725.1	ERR7658245	PDT001310317.1		antigen_formula=7:r:1,5:serotype=Infantis
SAMEA11580266	National Institute for Communicable Disease, South Africa	7/19/07	South Africa	clinical	Human Stool	PDS000109862.3	GCA_023498345.1	ERR7658272	PDT001310322.1		antigen_formula=7:r:1,5:serotype=Infantis
SAMEA11580268	National Institute for Communicable Disease, South Africa	12/11/07	South Africa	clinical	Human Stool	PDS000109867.2	GCA_023498285.1	ERR7658277	PDT001310327.1		antigen_formula=7:r:1,5:serotype=Infantis
SAMEA11580273	National Institute for Communicable Disease, South Africa	4/23/09	South Africa	clinical	Human Stool	PDS000113782.7	GCA_024225815.1	ERR7658320	PDT001357657.1		antigen_formula=7:r:1,5:serotype=Infantis
SAMEA11580301	National Institute for Communicable Disease, South Africa	10/19/13	South Africa	clinical	Human Blood culture	PDS000114212.1	GCA_024263185.1	ERR7671439	PDT001358551.1		antigen_formula=7:r:1,5:serotype=Infantis
SAMEA11580323	National Institute for Communicable Disease, South Africa	1/15/11	South Africa	clinical	Human Stool	PDS000114228.2	GCA_024366255.1	ERR7658470	PDT001365205.1		antigen_formula=7:r:1,5:serotype=Infantis
SAMEA11580396	National Institute for Communicable Disease, South Africa	2/7/14	South Africa	clinical	Human Stool	PDS000113879.1	GCA_024270775.1	ERR7671453	PDT001370914.1		antigen_formula=7:r:1,5:serotype=Infantis
SAMEA11580399	National Institute for Communicable Disease, South Africa	1/13/14	South Africa	clinical	Human Stool	PDS000154029.1	GCA_024478585.1	ERR7671450	PDT001370707.1		antigen_formula=7:r:1,5:serotype=Infantis
SAMEA3538994	Istituto Zooprofilattico Sperimentale delle Regioni Lazio e Toscana, Via Appia Nuova 1411, 00178, Rome, Italy	2007	Italy			PDS000085352.6	GCA_010797915.1	ERR1014109	PDT000123840.2		antigen_formula=7:r:1,5:serotype=Infantis
SAMEA3538995	Istituto Zooprofilattico Sperimentale delle Regioni Lazio e Toscana, Via Appia Nuova 1411, 00178, Rome, Italy	2009	Italy			PDS000085372.1	GCA_011103495.1	ERR1014110	PDT000123841.2		antigen_formula=7:r:1,5:serotype=Infantis
SAMEA4668128		2016	Germany		animal		GCA_010181655.1	ERR2586092	PDT000343770.1		antigen_formula=7:r:1,5:serotype=Infantis
SAMEA5039313						PDS000056247.1	GCA_011283665.1	ERR3843733	PDT000674492.1	MA.RM_119	antigen_formula=7:r:1,5:serotype=Infantis
SAMEA5556920						PDS000077402.1	GCA_013640815.1	ERR4289588	PDT000733261.1	MA.NK07.023	antigen_formula=7:r:1,5:serotype=Infantis
SAMEA5670929						PDS000085606.9	GCA_013559675.1	ERR4338231	PDT000789717.1	MA.CE08.063	antigen_formula=7:r:1,5:serotype=Infantis
SAMEA5978362	IZSLT	2016	Italy	clinical		PDS000085370.1	GCA_015857625.1	ERR3562266	PDT000905850.1		antigen_formula=7:r:1,5:serotype=Infantis
SAMEA6057638		2018	Germany		food	PDS000051895.5	GCA_010294345.1	ERR3580973	PDT000606482.1		antigen_formula=7:r:1,5:serotype=Infantis
SAMEA6058020		2013	Germany		animal	PDS000051882.1	GCA_009618595.1	ERR3581355	PDT000606851.1		antigen_formula=7:r:1,5:serotype=Infantis
SAMEA6058279		2018	Germany		animal	PDS000051922.5	GCA_010295885.1	ERR3581613	PDT000607107.1		antigen_formula=7:r:1,5:serotype=Infantis
SAMEA6262611		7/5/17	Turkey		chicken wing	PDS000099909.1	GCA_020994405.1	ERR3697221	PDT001186441.1		antigen_formula=7:r:1,5:serotype=Infantis
SAMEA6262612		10/29/17	Turkey		chicken wing	PDS000099890.1	GCA_020994425.1	ERR3697222	PDT001186442.1		antigen_formula=7:r:1,5:serotype=Infantis
SAMEA6262625		8/10/17	Turkey		chicken leg	PDS000099881.1	GCA_020994105.1	ERR3697235	PDT001186455.1		antigen_formula=7:r:1,5:serotype=Infantis
SAMEA6942530		1995	Germany		broiler	PDS000077667.3	GCA_016111585.1	ERR4233826	PDT000916645.1		antigen_formula=7:r:1,5:serotype=Infantis
SAMEA7540849		2018	Germany	clinical	stool	PDS000076389.2	GCA_020141375.1	ERR4832839	PDT001137335.1		antigen_formula=7:r:1,5:serotype=Infantis
SAMEA78283918		2016	Ireland	clinical	human	PDS000117335.1	GCA_025084235.1	ERR1816827	PDT000451530.1		antigen_formula=7:r:1,5:serotype=Infantis
SAMEA8036918		2015	Ireland	clinical	human	PDS000114393.1	GCA_024298245.1	ERR1823516	PDT000451704.2		antigen_formula=7:r:1,5:serotype=Infantis
SAMN01902298	NYSDOH	8/16/07	USA:NY	environmental/other	food	PDS000108885.140	GCA_011455395.1	SRR949438	PDT000000178.3	NY_BAC07000006871	antigen_formula=7:r:1,5:serotype=Infantis
SAMN01902343	NYSDOH	8/21/09	USA:NY	environmental/other	chicken	PDS000032398.53	GCA_011455905.1	SRR1158047	PDT000003207.5	NY_BAC09000003911	antigen_formula=7:r:1,5:serotype=Infantis
SAMN01902347	NYSDOH	8/17/09	USA:NY	environmental/other	green onion	PDS000032022.4	GCA_011455885.1	SRR1106258	PDT000002555.3	NY_BAC09000004010	antigen_formula=7:r:1,5:serotype=Infantis
SAMN02253058	FL	12/21/10	USA:FL	environmental/other	cilantro	PDS000085033.550	GCA_011460595.1	SRR1041496	PDT000001635.3	FL_FLDACS-08003	antigen_formula=7:r:1,5:serotype=Infantis
SAMN02261146	New Mexico Public Health Lab	2010	USA:NM	environmental/other	ground turkey	PDS000003971.75	GCA_008809675.1	SRR1171476	PDT000001025.6	AZ_TG68244	antigen_formula=7:r:1,5:serotype=Infantis
SAMN02265314	FDA Contracted Laboratory	10/12/07	USA:SD	environmental/other	bovine carcass	PDS000032397.259	GCA_006839415.1	SRR949876	PDT000004812	CFSAN005687	antigen_formula=7:r:1,5:serotype=Infantis
SAMN02345107	United States Food and Drug Administration| Office of Regulatory Affairs| New York Human and Animal Food Laboratory	2/17/11	USA	environmental/other	snail	GCA_010702915.1	SRR1164504	PDT000003330.3	FNE00073	FNE00073	antigen_formula=7:r:1,5:serotype=Infantis
SAMN02345192	United States Food and Drug Administration| Office of Regulatory Affairs| Atlanta Human and Animal Food Laboratory	5/13/11	USA	environmental/other	frozen calamari ring	PDS000032456.133	GCA_011633805.1	SRR1212330	PDT000025179.3	FSE0064	antigen_formula=7:r:1,5:serotype=Infantis
SAMN02345571	United States Food and Drug Administration| Office of Regulatory Affairs| Winchester Engineering Analytical Center	5/26/12	USA	environmental/other	papaya	PDS000029861.9	GCA_010906165.1	SRR1175787	PDT000003556.3	FMA0050	antigen_formula=7:r:1,5:serotype=Infantis
SAMN02367727	FDA		Senegal	clinical		PDS000001365.4	GCA_010633205.1	SRR1060649	PDT000002227.3	IVB 385/72	antigen_formula=7:r:1,5:serotype=Infantis
SAMN02367946	FDA: Shaohua Zhao	2011	USA:CA	environmental/other	chicken breast	PDS000027076.841	GCA_010636605.1	SRR1122618	PDT000002888.3	N29304	antigen_formula=7:r:1,5:serotype=Infantis
SAMN02470974		2008-11	Israel	clinical	stool sample	PDS000032399.10	GCA_000506925.1		PDT000020091.2	119944	antigen_formula=7:r:1,5:serotype=Infantis
SAMN02483567	United States Food and Drug Administration| Office of Regulatory Affairs| San Francisco Human and Animal Food Laboratory	3/23/11	USA	environmental/other	cat claw powder herbal product	PDS000193341.2	GCA_011626645.1	SRR1157945	PDT000003302.3	FSF0091	antigen_formula=7:r:1,5:serotype=Infantis
SAMN02646824	Minnesota Department Of Health	9/16/03	USA:MN	environmental/other	feces	PDS000032436.5	GCA_006752825.1	SRR1284729	PDT000030278.4	MDH-2014-00050	antigen_formula=7:r:1,5:serotype=Infantis
SAMN02646832	Minnesota Department Of Health	9/19/03	USA:MN	environmental/other		PDS000030006.9	GCA_011080965.1	SRR1282397	PDT000029788.3	MDH-2014-00058	antigen_formula=7:r:1,5:serotype=Infantis
SAMN02678547	United States Food and Drug Administration	2/1/10	USA	environmental/other	environmental swab	PDS000026846.6	GCA_006630545.1	SRR1481663	PDT000034037.4	FCC0141	antigen_formula=7:r:1,5:serotype=Infantis
SAMN02678643	United States Food and Drug Administration	2/8/13	USA	environmental/other	brewer yeast	PDS000032262.2	GCA_006690205.1	SRR1346247	PDT000031888.4	FDN0122	antigen_formula=7:r:1,5:serotype=Infantis
SAMN02678851	United States Food and Drug Administration	5/31/13	USA	environmental/other	dried parsley	PDS000003946.17	GCA_011080545.1	SRR1258567	PDT000026781.3	FSW0124	antigen_formula=7:r:1,5:serotype=Infantis
SAMN02678883	United States Food and Drug Administration	11/5/13	USA	environmental/other	pumpkin seed powder	PDS000032243.3	GCA_007743175.1	SRR1264975	PDT000027684.3	FSW0156	antigen_formula=7:r:1,5:serotype=Infantis
SAMN02698446	United States Food and Drug Administration	9/8/10	USA	environmental/other	meat bone meal	PDS000031702.5	GCA_010505935.1	SRR1503322	PDT000034347.3	FSE0192	antigen_formula=7:r:1,5:serotype=Infantis
SAMN02699512	Minnesota Department of Health	11/15/02	USA:NC	environmental/other	tissue	PDS000043301.3	GCA_011448675.1	SRR1461812	PDT000033698.3	MDH-2014-00346	antigen_formula=7:r:1,5:serotype=Infantis
SAMN02699691	Minnesota Department of Health	8/1/03	USA:MN	environmental/other	liver	PDS000032186.7	GCA_006629545.1	SRR1586570	PDT000039706.4	MDH-2014-00525	antigen_formula=7:r:1,5:serotype=Infantis
SAMN02742080	FDA Contracted Laboratory	11/8/06	USA:WA	environmental/other	porcine colon	PDS000032430.2	GCA_011632405.1	SRR1292254	PDT000030502.3	CFSAN012647	antigen_formula=7:r:1,5:serotype=Infantis
SAMN02845479	United States Food and Drug Administration|Office of Regulatory Affairs|Pacific Northwest Laboratory	6/7/04	USA	environmental/other	coriander	PDS000047465.1	GCA_007756535.1	SRR3027072	PDT000982053.3	FDA286791-2	antigen_formula=7:r:1,5:serotype=Infantis
SAMN02845486	United States Food and Drug Administration	6/17/04	USA	environmental/other	frozen iguana meat	PDS000111461.1	GCA_024107855.1	SRR9697638	PDT001330427.1	FDA288328 B	antigen_formula=7:r:1,5:serotype=Infantis
SAMN02845835	United States Food and Drug Administration| Office of Regulatory Affairs| Arkansas Human and Animal Food Laboratory	11/14/06	USA	environmental/other	soy lecithin fluid	PDS000031930.2	GCA_008115525.1	SRR2058144	PDT000066962.2	FDA395623 1-1	antigen_formula=7:r:1,5:serotype=Infantis
SAMN02846127	United States Food and Drug Administration|Office of Regulatory Affairs|Southeast Food and Feed Laboratory	6/25/08	USA	environmental/other	environmental	PDS000031894.8	GCA_008915945.2	SRR3457701	PDT000127759.4	FDA474819 sub14 isolate3	antigen_formula=7:r:1,5:serotype=Infantis
SAMN02847097	United States Food and Drug Administration| Office of Regulatory Affairs| Irvine Human and Animal Food Laboratory	10/25/10	USA	environmental/other	frozen cut crab	PDS000031805.4	GCA_007743975.1	SRR1658074	PDT000041344.6	FDA654627 5-1	antigen_formula=7:r:1,5:serotype=Infantis
SAMN02847795	United States Food and Drug Administration| Office of Regulatory Affairs| New York Human and Animal Food Laboratory	2/28/01	USA	environmental/other	animal feed	PDS000120908.4	GCA_006757045.1	SRR2075155	PDT000685403.3	FDA82581-C	antigen_formula=7:r:1,5:serotype=Infantis
SAMN02849840	FDA Contracted Laboratory	1/9/07	USA:CO	environmental/other	feces bovine	PDS000032425.4	GCA_011452475.1	SRR1505456	PDT000034455.3	CFSAN018572	antigen_formula=7:r:1,5:serotype=Infantis
SAMN02867156	VA	4/24/14	USA:VA	environmental/other	Not Provided	PDS000027077.489	GCA_008070535.1	SRR1573090	PDT000038660.3	VA-WGS-00278	antigen_formula=7:r:1,5:serotype=Infantis
SAMN02894095	FDA	2005	USA:MD	environmental/other	pork chop	PDS000047443.1	GCA_007744195.1	SRR1664264	PDT000044370.4	AZ.TG74536	antigen_formula=7:r:1,5:serotype=Infantis
SAMN02900341	Not Provided	5/14/12	USA	environmental/other	water	PDS000032400.84	GCA_006860505.1	SRR1566312	PDT000038128.3	CFSAN008910	antigen_formula=7:r:1,5:serotype=Infantis
SAMN02900552	United States Food and Drug Administration| Office of Regulatory Affairs| New York Human and Animal Food Laboratory	6/6/14	USA	environmental/other	natural pig ear	PDS000027717.1	GCA_006012685.1	SRR1614288	PDT000040798.2	FDA865717	antigen_formula=7:r:1,5:serotype=Infantis
SAMN02902678	FDA Contracted Laboratory	1/9/07	USA:WA	environmental/other	bovine necropsy	PDS000031652.1	GCA_006846065.1	SRR1528522	PDT000035947.2	CFSAN022438	antigen_formula=7:r:1,5:serotype=Infantis
SAMN02902775	FDA Contracted Laboratory	1/8/07	USA:WA	environmental/other	canine bronchoalveolar lavage	PDS000094101.2	GCA_010655515.1	SRR1544357	PDT000036631.2	CFSAN022535	antigen_formula=7:r:1,5:serotype=Infantis
SAMN02918683	United States Food and Drug Administration|Office of Regulatory Affairs|Southeast Food and Feed Laboratory	7/7/10	USA	environmental/other	cat food	PDS000036558.2	GCA_006741985.1	SRR5341494	PDT000192840.4	FDA630591	antigen_formula=7:r:1,5:serotype=Infantis
SAMN02918748	United States Food and Drug Administration|Office of Regulatory Affairs|Southeast Food and Feed Laboratory	9/14/10	USA	environmental/other	pet treat bully chew	PDS000074308.4	GCA_009351975.1	SRR5804744	PDT000226656.2	FDA646456	antigen_formula=7:r:1,5:serotype=Infantis
SAMN02918817	United States Food and Drug Administration|Office of Regulatory Affairs|Southeast Food and Feed Laboratory	8/22/11	USA	environmental/other	animal feed meat bone meal	PDS000078068.1	GCA_009557175.1	SRR8126784	PDT000247076.2	FDA706717	antigen_formula=7:r:1,5:serotype=Infantis

SAMN02919009	United States Food and Drug Administration|Office of Regulatory Affairs|Pacific Northwest Laboratory	4/30/13	USA	environmental/other	pet treat	PDS000163211.5	GCA_008996375.1	SRR4418762	PDT000181649.2	FDA809033 1-1	antigen_formula=7:r:1,5,serotype=Infantis
SAMN03018567	United States Food and Drug Administration| Office of Regulatory Affairs| New York Human and Animal Food Laboratory	7/22/14	USA	environmental/other	shelf stable grated parmesan cheese	PDS000031455.1	GCA_010533095.1	SRR1569593	PDT000038141.4	FDA827173	antigen_formula=7:r:1,5,serotype=Infantis
SAMN03102309	FDA Contracted Laboratory	3/10/13	USA:MO	environmental/other	environmental swab sponge	PDS000031546.3	GCA_006852905.1	SRR1614993	PDT000040875.2	CFSAN025186	antigen_formula=7:r:1,5,serotype=Infantis
SAMN03102331	FDA Contracted Laboratory	5/17/13	USA:CA	environmental/other	produce	PDS000032395.554	GCA_006851205.1	SRR1615207	PDT000040918.2	CFSAN025208	antigen_formula=7:r:1,5,serotype=Infantis
SAMN03152388	PHE	2012-06	United Kingdom: South of England	clinical	human	PDS000077501.61	GCA_006387635.1	SRR1635086	PDT000041899.4	H122460440	antigen_formula=7:r:1,5,serotype=Infantis
SAMN03168514	PHE	2012-09	United Kingdom: South of England	clinical	human	PDS000029259.40	GCA_008031155.1	SRR1645143	PDT000042801.4	H123640745	antigen_formula=7:r:1,5,serotype=Infantis
SAMN03168728	PHE	2012-05	United Kingdom: South of England	clinical	human	PDS000032463.148	GCA_011076895.1	SRR1645344	PDT000042952.4	H122040361	antigen_formula=7:r:1,5,serotype=Infantis
SAMN03168826	PHE	2012-10	United Kingdom: Midlands and East of England	clinical	human	PDS000003951.13	GCA_004262665.1	SRR1645468	PDT000043051.4	H124060483	antigen_formula=7:r:1,5,serotype=Infantis
SAMN03169120	PHE	2012-11	United Kingdom: South of England	clinical	human	PDS000029248.50	GCA_008003065.1	SRR1645854	PDT000043287.5	H124740809	antigen_formula=7:r:1,5,serotype=Infantis
SAMN03169191	PHE	2012-04	United Kingdom: North of England	clinical	human	PDS000030674.26	GCA_008002095.1	SRR1645903	PDT000043328.5	H121740568	antigen_formula=7:r:1,5,serotype=Infantis
SAMN03169192	PHE	2012-11	United Kingdom: Midlands and East of England	clinical	human	PDS000003938.55	GCA_008002085.1	SRR1645904	PDT000043329.5	H124700500	antigen_formula=7:r:1,5,serotype=Infantis
SAMN03169303	PHE	2012-09	United Kingdom: South of England	clinical	human	PDS000046454.79	GCA_008019075.1	SRR1645987	PDT000043412.5	H123800507	antigen_formula=7:r:1,5,serotype=Infantis
SAMN03169451	PHE	2013-01	United Kingdom: South of England	clinical	human	PDS000027464.61	GCA_008020475.1	SRR1646102	PDT000043504.5	H130260449	antigen_formula=7:r:1,5,serotype=Infantis
SAMN03177794	United States Food and Drug Administration| Office of Regulatory Affairs| New York Human and Animal Food Laboratory	11/4/14	USA	environmental/other	individually quick frozen scallop adductor	PDS000043225.8	GCA_006011925.1	SRR1659019	PDT000044193.2	FDA885223-1-1	antigen_formula=7:r:1,5,serotype=Infantis
SAMN03199677	FDA Contracted Laboratory	4/16/07	USA:SD	environmental/other	beef	PDS000032452.2	GCA_006856405.1	SRR1722878	PDT000045990.2	CFSAN027146	antigen_formula=7:r:1,5,serotype=Infantis
SAMN03218239	USDA-FSIS	2012	USA:PA	environmental/other	egg yolk	PDS000055687.3	GCA_008028105.1	SRR1745556	PDT000047090.3	CFSAN024812	antigen_formula=7:r:1,5,serotype=Infantis
SAMN03255335	Middle East Technical University| Food Engineering Department	7/18/12	Turkey	environmental/other	chicken meat	PDS000031430.1	GCA_004192975.1	SRR1745554	PDT000047088.3	CFSAN004222	antigen_formula=7:r:1,5,serotype=Infantis
SAMN03255336	Middle East Technical University| Food Engineering Department	7/18/12	Turkey	environmental/other	chicken meat	PDS000091376.27	GCA_007745235.1	SRR1745619	PDT000047152.4	CFSAN004223	antigen_formula=7:r:1,5,serotype=Infantis
SAMN03255371	Middle East Technical University| Food Engineering Department	11/28/12	Turkey	environmental/other	chicken meat	PDS000031424.3	GCA_006755385.1	SRR1812815	PDT000049924.3	CFSAN004258	antigen_formula=7:r:1,5,serotype=Infantis
SAMN03255375	Middle East Technical University| Food Engineering Department	12/7/12	Turkey	environmental/other	chicken meat	PDS000091393.1	GCA_011090745.1	SRR1812857	PDT000049956.2	CFSAN004262	antigen_formula=7:r:1,5,serotype=Infantis
SAMN03255390	Middle East Technical University| Food Engineering Department	12/19/12	Turkey	environmental/other	chicken meat	PDS000091404.1	GCA_010877105.1	SRR1916110	PDT000051454.2	CFSAN004277	antigen_formula=7:r:1,5,serotype=Infantis
SAMN03272229	USDA-FSIS	2012	USA:GA	environmental/other	egg raw yolk	PDS000032433.20	GCA_011093685.1	SRR1849368	PDT000050642.2	CFSAN027393	antigen_formula=7:r:1,5,serotype=Infantis
SAMN03291525	United States Food and Drug Administration	2006	USA	environmental/other	chicken breast	PDS000003947.4	GCA_006756565.1	SRR1822308	PDT000050326.3	WAPHL_SAL-A00696	antigen_formula=7:r:1,5,serotype=Infantis
SAMN03329207	UCD Dublin	5/4/07	Colombia	environmental/other	potato and meat bovine	PDS000074085.2	GCA_008910305.1	SRR3660562	PDT000134324.2	CFSAN006222	antigen_formula=7:r:1,5,serotype=Infantis
SAMN03463818		7/21/11	USA:VA	environmental/other	creek water	PDS000038968.1	GCA_002863785.1		PDT000276839.1	CFSAN003307	antigen_formula=7:r:1,5,serotype=Infantis
SAMN03464908	FDA Contracted Laboratory	5/5/14	USA:AR	environmental/other	chicken	PDS000031282.1	GCA_006874285.1	SRR2035396	PDT000065132.2	CFSAN031263	antigen_formula=7:r:1,5,serotype=Infantis
SAMN03465894	PHE	2014-10	United Kingdom: North of England	clinical	human	PDS000032426.13	GCA_011091665.1	SRR1957992	PDT000052975.2	57602	antigen_formula=7:r:1,5,serotype=Infantis
SAMN03466051	PHE	2014-09	United Kingdom: London	clinical	human	PDS000032435.26	GCA_011483845.1	SRR1958151	PDT000053132.2	46157	antigen_formula=7:r:1,5,serotype=Infantis
SAMN03466221	PHE	2014-08	United Kingdom: London	clinical	human	PDS000042428.20	GCA_011637295.1	SRR1958328	PDT000053300.2	46087	antigen_formula=7:r:1,5,serotype=Infantis
SAMN03466388	PHE	2014-07	United Kingdom: North of England	clinical	human	PDS000032447.4	GCA_011487025.1	SRR1958495	PDT000053466.2	46621	antigen_formula=7:r:1,5,serotype=Infantis
SAMN03466401	PHE	2014-10	United Kingdom: Midlands and East of England	clinical	human	PDS000037475.16	GCA_011095105.1	SRR1958508	PDT000053479.2	60408	antigen_formula=7:r:1,5,serotype=Infantis
SAMN03466516	PHE	2014-10	United Kingdom: None	clinical	human	PDS000032454.2	GCA_010876285.1	SRR1958623	PDT000053593.2	60103	antigen_formula=7:r:1,5,serotype=Infantis
SAMN03466543	PHE	2014-09	United Kingdom: South of England	clinical	human	PDS000003953.63	GCA_011095505.1	SRR1958650	PDT000053620.2	49717	antigen_formula=7:r:1,5,serotype=Infantis
SAMN03468504	PHE	2014-11	United Kingdom: South of England	clinical	human	PDS000121792.1	GCA_011101675.1	SRR1959299	PDT000053853.2	69721	antigen_formula=7:r:1,5,serotype=Infantis
SAMN03468515	PHE	2014-12	United Kingdom: Midlands and East of England	clinical	human	PDS000026854.11	GCA_011096785.1	SRR1959310	PDT000053864.2	78724	antigen_formula=7:r:1,5,serotype=Infantis
SAMN03468933	PHE	2014-12	United Kingdom: South of England	clinical	human	PDS000208495.4	GCA_010966995.1	SRR1960090	PDT000054077.2	78661	antigen_formula=7:r:1,5,serotype=Infantis
SAMN03468966	PHE	2014-12	United Kingdom: Midlands and East of England	clinical	human	PDS000003948.24	GCA_011097165.1	SRR1960146	PDT000054110.2	78650	antigen_formula=7:r:1,5,serotype=Infantis
SAMN03469660	PHE	2015-01	United Kingdom: North of England	clinical	human	PDS000028615.5	GCA_008034725.1	SRR1963110	PDT000054405.2	79055	antigen_formula=7:r:1,5,serotype=Infantis
SAMN03474046	PHE	2014-08	United Kingdom: North of England	clinical	human	PDS000032440.92	GCA_008050995.1	SRR1965152	PDT000054867.2	39491	antigen_formula=7:r:1,5,serotype=Infantis
SAMN03475350	PHE	2015-03	United Kingdom: London	clinical	human	PDS000031128.2	GCA_008051375.1	SRR1965218	PDT000054916.2	94227	antigen_formula=7:r:1,5,serotype=Infantis
SAMN03475503	PHE	2014-10	United Kingdom: London	clinical	human	PDS000031016.2	GCA_008053455.1	SRR1965380	PDT000055069.2	63715	antigen_formula=7:r:1,5,serotype=Infantis
SAMN03475623	PHE	2014-09	United Kingdom: North of England	clinical	human	PDS000030939.13	GCA_008053295.1	SRR1965502	PDT000055187.2	51289	antigen_formula=7:r:1,5,serotype=Infantis
SAMN03476132	PHE	2015-03	United Kingdom: Midlands and East of England	clinical	human	PDS000078465.3	GCA_008060255.1	SRR1966045	PDT000055666.2	99054	antigen_formula=7:r:1,5,serotype=Infantis
SAMN03476905	PHE	2014-08	United Kingdom: Midlands and East of England	clinical	human	PDS000028363.1	GCA_008074645.1	SRR1966827	PDT000056419.2	40237	antigen_formula=7:r:1,5,serotype=Infantis
SAMN03476985	PHE	2014-05	United Kingdom: South of England	clinical	human	PDS000032455.25	GCA_008112805.1	SRR1966907	PDT000059047.2	14903	antigen_formula=7:r:1,5,serotype=Infantis
SAMN03477234	PHE	2014-07	United Kingdom: North of England	clinical	human	PDS000028342.9	GCA_008078475.1	SRR1967144	PDT000056722.2	36472	antigen_formula=7:r:1,5,serotype=Infantis
SAMN03477358	PHE	2014-08	United Kingdom: South of England	clinical	human	PDS000028325.11	GCA_008092365.1	SRR1967268	PDT000056846.2	38383	antigen_formula=7:r:1,5,serotype=Infantis
SAMN03478097	PHE	2015-01	United Kingdom: North of England	clinical	human	PDS000028897.52	GCA_008100165.1	SRR1968006	PDT000057582.2	83688	antigen_formula=7:r:1,5,serotype=Infantis
SAMN03478189	PHE	2014-05	United Kingdom: North of England	clinical	human	PDS000091399.1	GCA_008106625.1	SRR1968098	PDT000058119.2	13417	antigen_formula=7:r:1,5,serotype=Infantis
SAMN03478213	PHE	2014-07	United Kingdom: London	clinical	human	PDS000029164.1	GCA_008106505.1	SRR1968122	PDT000058143.2	27964	antigen_formula=7:r:1,5,serotype=Infantis
SAMN03478249	PHE	2014-01	United Kingdom: North of England	clinical	human	PDS000003952.33	GCA_010921435.1	SRR1968158	PDT000058179.2	784	antigen_formula=7:r:1,5,serotype=Infantis
SAMN03478931	PHE	2015-02	United Kingdom: North of England	clinical	human	PDS000014139.5	GCA_008104485.1	SRR1968833	PDT000057854.2	84452	antigen_formula=7:r:1,5,serotype=Infantis
SAMN03479117	PHE	2014-04	United Kingdom: North of England	clinical	human	PDS000031025.3	GCA_008200285.1	SRR1969020	PDT000058040.2	5836	antigen_formula=7:r:1,5,serotype=Infantis
SAMN03479630	PHE	2014-07	United Kingdom: London	clinical	human	PDS000023228.5	GCA_008575205.1	SRR1969553	PDT000059177.2	27010	antigen_formula=7:r:1,5,serotype=Infantis
SAMN03479910	PHE	2014-08	United Kingdom: Midlands and East of England	clinical	human	PDS000028284.21	GCA_008577925.1	SRR1969833	PDT000059448.2	39489	antigen_formula=7:r:1,5,serotype=Infantis
SAMN03576987	Ohio State University	11/20/13	Kenya	environmental/other	feces swine	PDS000030886.1	GCA_006177845.1	SRR2847944	PDT000105349.2	CFSAN031454	antigen_formula=7:r:1,5,serotype=Infantis
SAMN03577087	Addis Ababa University	1/1/05	Ethiopia	environmental/other	camel spleen	PDS000047484.3	GCA_007763695.1	SRR4243100	PDT000148818.3	CFSAN031554	antigen_formula=7:r:1,5,serotype=Infantis
SAMN03577251	Ohio State University	9/23/05	Ethiopia	environmental/other	porcine meat	PDS000030866.2	GCA_006891675.1	SRR3066290	PDT000105466.2	CFSAN031718	antigen_formula=7:r:1,5,serotype=Infantis
SAMN03732551	United States Food and Drug Administration|Office of Regulatory Affairs|San Francisco Laboratory	5/11/15	USA	environmental/other	white pepper powder	PDS000025794.4	GCA_005524315.1	SRR2040619	PDT000065434.2	FDA908414-1	antigen_formula=7:r:1,5,serotype=Infantis
SAMN03894177	FDA	2013	USA: MD	environmental/other	Pork Chops	PDS000032428.4	GCA_001242545.1		PDT000078001.1	CVM N45943	antigen_formula=7:r:1,5,serotype=Infantis
SAMN03921963	USDA-FSIS	2015	USA:MS	environmental/other	NRTE (Not-Ready-to-Eat) Comminuted Poultry Exploratory Sampling - Chickens	PDS000032396.172	GCA_008613485.1	SRR2125853	PDT000072737.2	FSIS1501379	antigen_formula=7:r:1,5,serotype=Infantis
SAMN03988221	FDA	2014	USA:GA	environmental/other	Pork Chop	PDS000032437.60	GCA_008514055.1	SRR2407541	PDT000085839.2	CVM N52020	antigen_formula=7:r:1,5,serotype=Infantis
SAMN03988224	FDA	2014	USA:GA	environmental/other	Pork Chop	PDS000003964.7	GCA_008514025.1	SRR2407544	PDT000085842.2	CVM N52023	antigen_formula=7:r:1,5,serotype=Infantis
SAMN03988443	FDA	2014	USA:OR	environmental/other	Ground Beef	PDS000084305.4	GCA_008515565.1	SRR2407763	PDT000086061.2	CVM N54747	antigen_formula=7:r:1,5,serotype=Infantis
SAMN04054235	USDA-FSIS	2015	USA:WA	environmental/other	Animal-Swine-Roaster Swine	PDS000032458.97	GCA_008651375.1	SRR2353813	PDT000082883.2	FSIS1503893	antigen_formula=7:r:1,5,serotype=Infantis
SAMN04208145	USDA-FSIS	2015	USA:NJ	environmental/other	Animal-Calf-Formula-fed Veal	PDS000030658.18	GCA_008406325.1	SRR2774951	PDT000088586.2	FSIS1504743	antigen_formula=7:r:1,5,serotype=Infantis
SAMN04208146	USDA-FSIS	2015	USA:OH	environmental/other	comminuted beef	PDS000027079.13	GCA_008646575.1	SRR2775033	PDT000088594.2	FSIS1504744	antigen_formula=7:r:1,5,serotype=Infantis
SAMN04218219	FDA	2008	USA:GA	environmental/other	chicken breast	PDS000032407.2	GCA_007762035.1	SRR3242364	PDT000115403.3	CVM-N19784	antigen_formula=7:r:1,5,serotype=Infantis
SAMN04224261	FDA	2008	USA:MD	environmental/other	ground turkey	PDS000051934.3	GCA_007757345.1	SRR3055288	PDT000100313.3	NY-N19911	antigen_formula=7:r:1,5,serotype=Infantis
SAMN04240651	South Dakota State University| South Dakota Animal Disease Research and Diagnostic Laboratory	2015	USA	environmental/other	feces	PDS000027078.67	GCA_008657815.1	SRR2962658	PDT000095446.2	ADRDL-15-5024	antigen_formula=7:r:1,5,serotype=Infantis
SAMN04244006	USDA-FSIS	2015	USA:NE	environmental/other	comminuted chicken	PDS000005980.10	GCA_008470025.1	SRR2920602	PDT000094751.2	FSIS1504844	antigen_formula=7:r:1,5,serotype=Infantis
SAMN04256045	FDA	2010	USA:NM	environmental/other	chicken breast	PDS000032417.118	GCA_007229675.1	SRR3151800	PDT000102314.5	CVM-N23770	antigen_formula=7:r:1,5,serotype=Infantis
SAMN04311834	USDA-FSIS	2015	USA:PA	environmental/other	comminuted chicken	PDS000085035.33	GCA_008691545.1	SRR2970938	PDT000095668.2	FSIS1505056	antigen_formula=7:r:1,5,serotype=Infantis
SAMN04337197	USDA-FSIS	2015	USA:PA	environmental/other	animal-swine-sow	PDS000032459.168	GCA_008730515.1	SRR3000039	PDT000097431.2	FSIS1503917	antigen_formula=7:r:1,5,serotype=Infantis
SAMN04363695	PHE		United Kingdom: None	clinical	human	PDS000037747.4	GCA_008751035.1	SRR3049399	PDT000099550.2	40346	antigen_formula=7:r:1,5,serotype=Infantis
SAMN04370507	USDA-FSIS	2015	USA:MI	environmental/other	animal-swine-sow	PDS000030275.3	GCA_008769815.1	SRR3056931	PDT000100756.2	FSIS1502315	antigen_formula=7:r:1,5,serotype=Infantis
SAMN04437783	USDA-FSIS	2015	USA:IL	environmental/other	animal-swine-market swine	PDS000028697.3	GCA_008789685.1	SRR3115431	PDT000103323.2	FSIS1501185	antigen_formula=7:r:1,5,serotype=Infantis
SAMN04535470	FDA Contracted Laboratory	11/2/15	USA:WA	environmental/other	spinach	PDS000030401.1	GCA_007835085.1	SRR3372356	PDT000125171.2	CFSAN047706	antigen_formula=7:r:1,5,serotype=Infantis
SAMN04568546	USDA-FSIS	2016	USA:NE	environmental/other	animal-swine-sow	PDS000030405.1	GCA_008408935.1	SRR3241215	PDT000115241.2	FSIS1605969	antigen_formula=7:r:1,5,serotype=Infantis
SAMN04600407	PHE	2015-04	United Kingdom: United Kingdom	clinical	human	PDS000028362.2	GCA_008852785.1	SRR3322087	PDT000122482.2	107287	antigen_formula=7:r:1,5,serotype=Infantis
SAMN04600452	PHE	2015-09	United Kingdom: United Kingdom	clinical	human	PDS000028377.4	GCA_008878255.1	SRR3322121	PDT000122514.2	160847	antigen_formula=7:r:1,5,serotype=Infantis
SAMN04600635	PHE	2015-06	United Kingdom: United Kingdom	clinical	human	PDS000028371.2	GCA_008852415.1	SRR3322334	PDT000122611.2	126801	antigen_formula=7:r:1,5,serotype=Infantis
SAMN04600766	PHE	2015-08	United Kingdom: United Kingdom	clinical	human	PDS000028274.5	GCA_008879805.1	SRR3322625	PDT000122716.2	149301	antigen_formula=7:r:1,5,serotype=Infantis
SAMN04600788	PHE	2015-06	United Kingdom: United Kingdom	clinical	human	PDS000028372.1	GCA_008880015.1	SRR3322632	PDT000122722.2	122229	antigen_formula=7:r:1,5,serotype=Infantis
SAMN04600792	PHE	2015-05	United Kingdom: United Kingdom	clinical	human	PDS000052159.1	GCA_008836435.1	SRR3322637	PDT000122726.2	114077	antigen_formula=7:r:1,5,serotype=Infantis
SAMN04925735	CDC	2016-03	USA	clinical	stool	PDS000029506.4	GCA_011422125.1	SRR3475662	PDT000128636.2	PNUSAS002094	antigen_formula=7:r:1,5,serotype=Infantis
SAMN05396752		2016-06	USA	clinical	stool	PDS000028328.2	GCA_008926045.1	SRR3933556	PDT000139285.2	PNUSAS002972	antigen_formula=7:r:1,5,serotype=Infantis
SAMN05417410	Instituto Nacional de Salud Lima, Peru	2010	Peru	clinical	Not Provided	PDS000180602.1	GCA_005969485.1	SRR3931661	PDT000140584.2	CFSAN037646	antigen_formula=7:r:1,5,serotype=Infantis
SAMN05596693	CDC	2014-10	USA	clinical		PDS000054530.6	GCA_005976645.1	SRR4044650	PDT000161281.2	2014AM-2812	antigen_formula=7:r:1,5,serotype=Infantis
SAMN05726936	USDA-FSIS	2016	USA:AR	environmental/other	comminuted chicken	PDS000015609.4	GCA_008525305.1	SRR4125007	PDT000146675.2	FSIS1607511	antigen_formula=7:r:1,5,serotype=Infantis
SAMN05784370	USDA-FSIS	2016	USA:IN	environmental/other	animal-swine-market swine	PDS000198584.1	GCA_008949485.1	SRR4252433	PDT000149012.2	FSIS1607638	antigen_formula=7:r:1,5,serotype=Infantis
SAMN05859736	CDC	2016-08	USA	clinical	stool	PDS000028775.15	GCA_008980485.1	SRR4414081	PDT000151779.2	PNUSAS004377	antigen_formula=7:r:1,5,serotype=Infantis
SAMN05936853	CDC	2016-10	USA	clinical	stool	PDS000029125.32	GCA_008481365.1	SRR4454573	PDT000154439.2	PNUSAS004932	antigen_formula=7:r:1,5,serotype=Infantis
SAMN05957693	CDC	2016-09	USA	clinical	stool	PDS000029941.1	GCA_008987255.1	SRR4841720	PDT000156274.2	PNUSAS004862	antigen_formula=7:r:1,5,serotype=Infantis
SAMN06015755	USDA-FSIS	2016	USA:KS	environmental/other	animal-cattle-heifer	PDS000028662.1	GCA_009036005.1	SRR5019448	PDT000158993.2	FSIS1608231	antigen_formula=7:r:1,5,serotype=Infantis
SAMN06030045	NML		Canada	clinical		PDS000140912.1	GCA_009194745.1	SRR5053023	PDT000263313.4		antigen_formula=7:r:1,5,serotype=Infantis
SAMN06030141	NML		Canada	clinical		PDS000052155.4	GCA_009194585.1	SRR5055283	PDT000263458.4		antigen_formula=7:r:1,5,serotype=Infantis
SAMN06045809		2016-09	USA	clinical	stool	PDS000029534.35	GCA_009049345.1	SRR5057246	PDT000163163.3	PNUSAS005199	antigen_formula=7:r:1,5,serotype=Infantis
SAMN06113963	Pontifical Catholic University of Rio Grande do Sul| Immunology and Microbiology Laboratory	2008	Brazil	environmental/other	blood meal	PDS000109836.2	GCA_009100965.1	SRR5155620	PDT000176719.2	ADRDL-777	antigen_formula=7:r:1,5,serotype=Infantis
SAMN06113998	Pontifical Catholic University of Rio Grande do Sul| Immunology and Microbiology Laboratory	6/2/11	Brazil	environmental/other	drag swab	PDS000028822.7	GCA_009105985.1	SRR5173480	PDT000177693.2	ADRDL-798	antigen_formula=7:r:1,5,serotype=Infantis
SAMN06213880	FDA Contracted Laboratory	2016	USA:IL	environmental/other	meat feed	PDS000027418.10	GCA_006924595.1	SRR5239000	PDT000187251.2	CFSAN059148	antigen_formula=7:r:1,5,serotype=Infantis
SAMN06247757	PHE	2016-08	United Kingdom: United Kingdom	clinical	human	PDS000029743.3	GCA_007231415.1	SRR5193696	PDT000180017.2	286764	antigen_formula=7:r:1,5,serotype=Infantis
SAMN06248397	PHE	2015-10	United Kingdom: United Kingdom	clinical	human	PDS000028282.1	GCA_009033425.1	SRR5194337	PDT000180640.2	171291	antigen_formula=7:r:1,5,serotype=Infantis
SAMN06256185	USDA-FSIS	2016	USA:WA	environmental/other	raw intact chicken	PDS000029718.6	GCA_008527375.1	SRR5201510	PDT000181320.2	FSIS1609514	antigen_formula=7:r:1,5,serotype=Infantis
SAMN06270129	FDA Contracted Laboratory	2016	USA:GA	environmental/other	environmental swab sponge	PDS000027853.5	GCA_005987245.1	SRR5239004	PDT000187255.2	CFSAN059750	antigen_formula=7:r:1,5,serotype=Infantis
SAMN06278213	PHE	2015-10	United Kingdom: United Kingdom	clinical	human	PDS000026844.37	GCA_009062165.1	SRR5215640	PDT000182216.2	177443	antigen_formula=7:r:1,5,serotype=Infantis
SAMN06278239	PHE	2016-07	United Kingdom: United Kingdom	clinical	human	PDS000016779.5	GCA_009062485.1	SRR5215668	PDT000182244.2	272504	antigen_formula=7:r:1,5,serotype=Infantis
SAMN06278509	PHE	2015-09	United Kingdom: United Kingdom	clinical	human	PDS000029699.1	GCA_007231795.1	SRR5216101	PDT000182515.2	168332	antigen_formula=7:r:1,5,serotype=Infantis
SAMN06278718	PHE	2016-12	United Kingdom: United Kingdom	clinical	human	PDS000029695.7	GCA_007232155.1	SRR5216321	PDT000182729.2	323116	antigen_formula=7:r:1,5,serotype=Infantis
SAMN06298790	CDC	2016-12	USA	clinical	urine	PDS000190283.1	GCA_009080525.1	SRR5238307	PDT000187186.2	PNUSAS007222	antigen_formula=7:r:1,5,serotype=Infantis
SAMN06346072		2016-11	USA	clinical		PDS000032462.11	GCA_006635985.1	SRR5278637	PDT000189185.2	PNUSAS008690	antigen_formula=7:r:1,5,serotype=Infantis
SAMN06392768		2017-01	USA	clinical		PDS000029577.13	GCA_006106995.1	SRR5291636	PDT000190217.2	PNUSAS009093	antigen_formula=7:r:1,5,serotype=Infantis
SAMN06459278	USDA-FSIS	2017	USA:OH	environmental/other	animal-swine-sow	PDS000029569.19	GCA_008434565.1	SRR5297419	PDT000190632.2	FSIS1710548	antigen_formula=7:r:1,5,serotype=Infantis

SAMN06647493		2017-01	USA	clinical	stool	PDS000163311.2	GCA_009179885.1	SRR5396349	PDT000198437.2	PNUSAS009939	antigen_formula=7;r1,5,serotype=Infantis	
SAMN06658893	CDC	2017-02	USA	clinical		PDS000052604.1	GCA_006048055.1	SRR5399993	PDT000198931.2	PNUSAS010533	antigen_formula=7;r1,5,serotype=Infantis	
SAMN06672068	CDC	2017-03	USA	clinical		PDS000029200.15	GCA_006048415.1	SRR5408975	PDT000199079.2	PNUSAS010639	antigen_formula=7;r1,5,serotype=Infantis	
SAMN06693630	CDC	2017-03	USA	clinical	stool	PDS000028150.5	GCA_009194395.1	SRR5434463	PDT000201049.2	PNUSAS011316	antigen_formula=7;r1,5,serotype=Infantis	
SAMN06848175	CDC	2017-03	USA	clinical		PDS000032431.2	GCA_006762405.1	SRR5501151	PDT000206812.2	PNUSAS012037	antigen_formula=7;r1,5,serotype=Infantis	
SAMN06883646		2017-03	USA	clinical		PDS000027460.1	GCA_006968565.1	SRR5583082	PDT000211029.2		antigen_formula=7;r1,5,serotype=Infantis	
SAMN06929009		2017-03	USA	clinical		PDS000029301.8	GCA_006759145.1	SRR5584800	PDT000211495.2	PNUSAS013325	antigen_formula=7;r1,5,serotype=Infantis	
SAMN07135304	United States Food and Drug Administration	2009	USA	environmental/other	pig stool	PDS000127721.1	GCA_010617385.1	SRR6950215	PDT000303603.2	CFSAN064200	antigen_formula=7;r1,5,serotype=Infantis	
SAMN07152380	PHE	2017-01	United Kingdom: United Kingdom	clinical	human	PDS000117332.1	GCA_009247665.1	SRR5583203	PDT000211783.2	340264	antigen_formula=7;r1,5,serotype=Infantis	
SAMN07155338	PHE	2017-01	United Kingdom: United Kingdom	clinical	human	PDS000026853.1	GCA_009248405.1	SRR5583890	PDT000211864.2	335317	antigen_formula=7;r1,5,serotype=Infantis	
SAMN07155479	PHE	2017-05	United Kingdom: United Kingdom	clinical	human	PDS000029223.2	GCA_007233365.1	SRR5583958	PDT000211883.2	368286	antigen_formula=7;r1,5,serotype=Infantis	
SAMN07155558	PHE	2017-03	United Kingdom: United Kingdom	clinical	human	PDS000016766.28	GCA_009249185.1	SRR5584045	PDT000211904.2	352824	antigen_formula=7;r1,5,serotype=Infantis	
SAMN07155951	PHE	2017-01	United Kingdom: United Kingdom	clinical	human	PDS000032467.1	GCA_009283605.1	SRR5585298	PDT000212704.2	340252	antigen_formula=7;r1,5,serotype=Infantis	
SAMN07176328		2017-04	USA	clinical		PDS000029078.1	GCA_006560715.1	SRR5680621	PDT000218390.2	PNUSAS014844	antigen_formula=7;r1,5,serotype=Infantis	
SAMN07180133	USDA-FSIS	2017	USA:CA	environmental/other	Product-Raw-Intact-Pork	PDS000033050.3	GCA_008527765.1	SRR5632691	PDT000213903.2	FSIS1701443	antigen_formula=7;r1,5,serotype=Infantis	
SAMN07180327	PHE	2017-02	United Kingdom: United Kingdom	clinical	human	PDS000028364.5	GCA_009290585.1	SRR5632273	PDT000214373.2	342929	antigen_formula=7;r1,5,serotype=Infantis	
SAMN07180701	PHE	2017-02	United Kingdom: United Kingdom	clinical	human	PDS000028349.6	GCA_009296725.1	SRR5633125	PDT000214618.2	348709	antigen_formula=7;r1,5,serotype=Infantis	
SAMN07180843	PHE	2017-05	United Kingdom: United Kingdom	clinical	human	PDS000029140.2	GCA_006916555.1	SRR5633351	PDT000214734.2	369880	antigen_formula=7;r1,5,serotype=Infantis	
SAMN07189975		2017-04	USA	clinical		PDS000029135.1	GCA_006814645.1	SRR5655509	PDT000216486.2	PNUSAS014884	antigen_formula=7;r1,5,serotype=Infantis	
SAMN07198309		2017-04	USA	clinical		PDS000026139.21	GCA_006554195.1	SRR5659662	PDT000217248.2	PNUSAS015154	antigen_formula=7;r1,5,serotype=Infantis	
SAMN07209958	CDC	2017-05	USA	clinical	stool	PDS000029106.38	GCA_008485905.1	SRR5680622	PDT000218391.2	PNUSAS015257	antigen_formula=7;r1,5,serotype=Infantis	
SAMN07246803	MOD1	12/30/98	Peru	clinical	biological fluid or tissue	PDS000077666.4	GCA_005680475.1	SRR7456227	PDT000338737.1	MOD1_Per62	antigen_formula=7;r1,5,serotype=Infantis	
SAMN07249964	USDA-FSIS	2017	USA:MT	environmental/other	Product-Raw-Intact-Pork	PDS000029083.1	GCA_008552975.1	SRR5693827	PDT000218870.2	FSIS1701915	antigen_formula=7;r1,5,serotype=Infantis	
SAMN07279534	CDC	2017-04	USA	clinical		PDS000029270.6	GCA_006571405.1	SRR5799771	PDT000222534.2	PNUSAS014373	antigen_formula=7;r1,5,serotype=Infantis	
SAMN07410878	USDA-FSIS	2017	USA:NE	environmental/other	animal-swine-market swine	PDS000028790.15	GCA_009397125.1	SRR5865521	PDT000227134.2	FSIS1703083	antigen_formula=7;r1,5,serotype=Infantis	
SAMN07428689	CDC	2017-07	USA	clinical	stool	PDS000028821.1	GCA_008488205.1	SRR5908519	PDT000232063.2	PNUSAS019777	antigen_formula=7;r1,5,serotype=Infantis	
SAMN07460708		2017-07	USA	clinical		PDS000032451.1	GCA_006761645.1	SRR5929567	PDT000232884.2	PNUSAS020390	antigen_formula=7;r1,5,serotype=Infantis	
SAMN07490583		2017-07	USA	clinical		PDS000010535.1	GCA_007045105.1	SRR5932401	PDT000233407.2	PNUSAS020759	antigen_formula=7;r1,5,serotype=Infantis	
SAMN07499599		2017-06	USA	clinical		PDS000037193.2	GCA_006898285.1	SRR5947770	PDT000234827.2	PNUSAS019001	antigen_formula=7;r1,5,serotype=Infantis	
SAMN07571885	CDC	2017-06	USA	clinical		PDS000028630.12	GCA_006241875.1	SRR6019639	PDT000239640.2	PNUSAS018239	antigen_formula=7;r1,5,serotype=Infantis	
SAMN07605944	USDA-FSIS	2017	USA:IA	environmental/other	animal-swine-market swine	PDS000028599.2	GCA_008437305.1	SRR6006968	PDT000239107.2	FSIS1703746	antigen_formula=7;r1,5,serotype=Infantis	
SAMN07662460		2017-08	USA	clinical		PDS000028561.1	GCA_006573125.1	SRR6107362	PDT000244325.2		antigen_formula=7;r1,5,serotype=Infantis	
SAMN07672066	CDC	2017-09	USA	clinical		PDS000098009.1	GCA_006166765.1	SRR6107937	PDT000244608.2	PNUSAS023565	antigen_formula=7;r1,5,serotype=Infantis	
SAMN07684580		2017-07	USA	clinical		PDS000028532.6	GCA_008437205.1	SRR6082052	PDT000244110.2	PNUSAS022315	antigen_formula=7;r1,5,serotype=Infantis	
SAMN07735226	CDC	2015-03	USA	clinical		PDS000048168.2	GCA_006501335.1	SRR6128825	PDT000247229.2	2015AM-0847	antigen_formula=7;r1,5,serotype=Infantis	
SAMN07795020	CDC	2017-09	USA	clinical		PDS000543234	GCA_006169945.1	SRR6192496	PDT000254178.2	PNUSAS025967	antigen_formula=7;r1,5,serotype=Infantis	
SAMN07812114	PHE	2017-08	United Kingdom: United Kingdom	clinical	human	PDS000028383.1	GCA_004261205.1	SRR6190510	PDT000253594.2	414452	antigen_formula=7;r1,5,serotype=Infantis	
SAMN07812119	PHE	2016-11	United Kingdom: United Kingdom	clinical	human	PDS000088360.1	GCA_009517375.1	SRR6190531	PDT000253613.2	328648	antigen_formula=7;r1,5,serotype=Infantis	
SAMN07812126	PHE	2017-09	United Kingdom: United Kingdom	clinical	human	PDS000028285.9	GCA_009560555.1	SRR6190535	PDT000253617.2	427431	antigen_formula=7;r1,5,serotype=Infantis	
SAMN07812130	PHE	2015-12	United Kingdom: United Kingdom	clinical	human	PDS000028380.1	GCA_006917355.1	SRR6191085	PDT000253779.2	202261	antigen_formula=7;r1,5,serotype=Infantis	
SAMN07812136	PHE	2016-06	United Kingdom: United Kingdom	clinical	human	PDS000028347.1	GCA_009560155.1	SRR6190960	PDT000253622.2	261486	antigen_formula=7;r1,5,serotype=Infantis	
SAMN07812147	PHE	2016-06	United Kingdom: United Kingdom	clinical	human	PDS000082588.4	GCA_009560135.1	SRR6190975	PDT000253632.2	262862	antigen_formula=7;r1,5,serotype=Infantis	
SAMN07812155	PHE	2015-11	United Kingdom: United Kingdom	clinical	human	PDS000028369.3	GCA_009517835.1	SRR6190983	PDT000253713.2	184486	antigen_formula=7;r1,5,serotype=Infantis	
SAMN07812156	PHE	2015-09	United Kingdom: United Kingdom	clinical	human	PDS000028353.1	GCA_009517815.1	SRR6190982	PDT000253712.2	169652	antigen_formula=7;r1,5,serotype=Infantis	
SAMN07812163	PHE	2017-08	United Kingdom: United Kingdom	clinical	human	PDS000023024.8	GCA_009560725.1	SRR6190991	PDT000253721.2	402747	antigen_formula=7;r1,5,serotype=Infantis	
SAMN07812166	PHE	2016-01	United Kingdom: United Kingdom	clinical	human	PDS000026843.3	GCA_009560265.1	SRR6190996	PDT000253726.2	208754	antigen_formula=7;r1,5,serotype=Infantis	
SAMN07812169	PHE	2016-11	United Kingdom: United Kingdom	clinical	human	PDS000028379.1	GCA_007199595.1	SRR6190989	PDT000253719.2	320928	antigen_formula=7;r1,5,serotype=Infantis	
SAMN07812170	PHE	2015-03	United Kingdom: United Kingdom	environmental/other		PDS000028351.1	GCA_009560655.1	SRR6190992	PDT000253722.2	97247	antigen_formula=7;r1,5,serotype=Infantis	
SAMN07812249	PHE	2017-07	United Kingdom: United Kingdom	clinical	human	PDS000028376.5	GCA_007200155.1	SRR6191103	PDT000253797.2	389623	antigen_formula=7;r1,5,serotype=Infantis	
SAMN07812318	PHE	2016-08	United Kingdom: United Kingdom	clinical	human	PDS000028368.3	GCA_004264325.1	SRR6191115	PDT000253807.2	291736	antigen_formula=7;r1,5,serotype=Infantis	
SAMN07812321	PHE	2016-09	United Kingdom: United Kingdom	clinical	human	PDS000032421.1	GCA_004266005.1	SRR6191122	PDT000253814.2	305162	antigen_formula=7;r1,5,serotype=Infantis	
SAMN07812362	PHE	2016-08	United Kingdom: United Kingdom	clinical	human	PDS000082136.1	GCA_009518625.1	SRR6191152	PDT000253844.2	288896	antigen_formula=7;r1,5,serotype=Infantis	
SAMN07812365	PHE	2017-09	United Kingdom: United Kingdom	clinical	human	PDS000032420.5	GCA_007201115.1	SRR6191154	PDT000253846.2	425664	antigen_formula=7;r1,5,serotype=Infantis	
SAMN07812411	PHE	2016-08	United Kingdom: United Kingdom	clinical	human	PDS000053319.2	GCA_009560895.1	SRR6191169	PDT000253861.2	285843	antigen_formula=7;r1,5,serotype=Infantis	
SAMN07812434	PHE	2017-09	United Kingdom: United Kingdom	clinical	human	PDS000042637.1	GCA_007839895.1	SRR6191189	PDT000253879.2	423842	antigen_formula=7;r1,5,serotype=Infantis	
SAMN07812484	PHE	2016-08	United Kingdom: United Kingdom	clinical	human	PDS000101986.1	GCA_009561435.1	SRR6191336	PDT000253918.2	291745	antigen_formula=7;r1,5,serotype=Infantis	
SAMN07812495	PHE	2017-09	United Kingdom: United Kingdom	clinical	human	PDS000037053.16	GCA_007201215.1	SRR6191353	PDT000253927.2	421160	antigen_formula=7;r1,5,serotype=Infantis	
SAMN07812504	PHE	2016-08	United Kingdom: United Kingdom	clinical	human	PDS000076468.2	GCA_009518835.1	SRR6191364	PDT000253938.2	285536	antigen_formula=7;r1,5,serotype=Infantis	
SAMN07812614	PHE	2016-09	United Kingdom: United Kingdom	clinical	human	PDS000037140.4	GCA_007201275.1	SRR6191374	PDT000253946.2	294745	antigen_formula=7;r1,5,serotype=Infantis	
SAMN07812629	PHE	2016-11	United Kingdom: United Kingdom	clinical	human	PDS000028361.3	GCA_004253045.1	SRR6191385	PDT000253956.2	318523	antigen_formula=7;r1,5,serotype=Infantis	
SAMN07812635	PHE	2017-08	United Kingdom: United Kingdom	clinical	human	PDS000038543.1	GCA_007201295.1	SRR6191519	PDT000253986.2	413067	antigen_formula=7;r1,5,serotype=Infantis	
SAMN07812688	PHE	2017-09	United Kingdom: United Kingdom	clinical	human	PDS000016781.3	GCA_009520155.1	SRR6191564	PDT000254008.2	416398	antigen_formula=7;r1,5,serotype=Infantis	
SAMN07812695	PHE	2016-11	United Kingdom: United Kingdom	clinical	human	PDS000028345.1	GCA_009561785.1	SRR6191577	PDT000254021.2	317721	antigen_formula=7;r1,5,serotype=Infantis	
SAMN07812757	PHE	2017-07	United Kingdom: United Kingdom	clinical	human	PDS000032466.2	GCA_009561725.1	SRR6191584	PDT000254028.2	390047	antigen_formula=7;r1,5,serotype=Infantis	
SAMN07812791	PHE	2016-11	United Kingdom: United Kingdom	clinical	human	PDS000023642.5	GCA_007201435.1	SRR6191675	PDT000254076.2	314922	antigen_formula=7;r1,5,serotype=Infantis	
SAMN07812807	PHE	2015-09	United Kingdom: United Kingdom	clinical	human	PDS000042719.1	GCA_007839675.1	SRR6191693	PDT000254088.2	169631	antigen_formula=7;r1,5,serotype=Infantis	
SAMN07812812	PHE	2017-09	United Kingdom: United Kingdom	clinical	human	PDS000052160.3	GCA_007201615.1	SRR6191701	PDT000254096.2	417344	antigen_formula=7;r1,5,serotype=Infantis	
SAMN07812832	PHE	2017-09	United Kingdom: United Kingdom	clinical	human	PDS000028333.1	GCA_009562335.1	SRR6191705	PDT000254100.2	418891	antigen_formula=7;r1,5,serotype=Infantis	
SAMN07816144	PHE	2016-10	United Kingdom: United Kingdom	clinical	human	PDS000028340.1	GCA_007200755.1	SRR6192971	PDT000254488.2	307224	antigen_formula=7;r1,5,serotype=Infantis	
SAMN07816162	PHE	2017-06	United Kingdom: United Kingdom	clinical	human	PDS000028336.1	GCA_007200835.1	SRR6192986	PDT000254503.2	387198	antigen_formula=7;r1,5,serotype=Infantis	
SAMN07816206	PHE	2015-05	United Kingdom: United Kingdom	clinical	human	PDS000037060.1	GCA_009541435.1	SRR6193009	PDT000254526.2	117619	antigen_formula=7;r1,5,serotype=Infantis	
SAMN07816219	PHE	2014-06	United Kingdom: United Kingdom	environmental/other	food	PDS000028332.3	GCA_007200795.1	SRR6193018	PDT000254535.2	18581	antigen_formula=7;r1,5,serotype=Infantis	
SAMN07816324	PHE	2017-08	United Kingdom: United Kingdom	clinical	human	PDS000016775.7	GCA_009544235.1	SRR6193070	PDT000254587.2	400343	antigen_formula=7;r1,5,serotype=Infantis	
SAMN07822204		2017-09	USA	clinical		PDS000028312.21	GCA_006762805.1	SRR6217869	PDT000258118.2	PNUSAS025609	antigen_formula=7;r1,5,serotype=Infantis	
SAMN07967206		2017-07	USA	clinical		PDS000032423.2	GCA_008490105.1	SRR6286759	PDT000263933.2	PNUSAS025823	antigen_formula=7;r1,5,serotype=Infantis	
SAMN08032388		2017-10	USA	clinical	stool	PDS000028214.1	GCA_008490405.1	SRR6310548	PDT000265722.2	PNUSAS027782	antigen_formula=7;r1,5,serotype=Infantis	
SAMN08040085			USA	clinical		PDS000028208.1	GCA_007098875.1	SRR6313736	PDT000266186.2	PNUSAS028256	antigen_formula=7;r1,5,serotype=Infantis	
SAMN08114233	FDA	2016	USA:MD	environmental/other	Chicken wings	PDS000012025.3	GCA_010130165.1	SRR6351140	PDT000284440.2	CVM N163365	antigen_formula=7;r1,5,serotype=Infantis	
SAMN08168465		2017-11	USA	clinical		PDS000027980.55	GCA_006182805.1	SRR6386496	PDT000273208.2	PNUSAS030043	antigen_formula=7;r1,5,serotype=Infantis	
SAMN08211236	CDC	2017-12	USA	clinical	stool	PDS000019373.92	GCA_010134475.1	SRR6412926	PDT000274223.2	PNUSAS030555	antigen_formula=7;r1,5,serotype=Infantis	
SAMN08354556	CDC	2012	USA:WY	environmental/other		PDS000054116.4	GCA_006187685.1	SRR6475537	PDT000278540.2	PNUSAS031738	antigen_formula=7;r1,5,serotype=Infantis	
SAMN08355231	CDC	2017-11	USA	clinical	stool	PDS000025883.2	GCA_010163875.1	SRR6475563	PDT000278566.2		antigen_formula=7;r1,5,serotype=Infantis	
SAMN08376490	CDC	2015-03	USA	clinical		PDS000103799.1	GCA_006262915.1	SRR6480645	PDT000278896.2	2015AM-0283	antigen_formula=7;r1,5,serotype=Infantis	
SAMN08387011	Universidade de Sao Paulo| Ciencia e Tecnologia de Alimentos	Laboratorio Microbiologia de Alimentos	2015	Brazil	environmental/other	broiler chicken	PDS000018462.82	GCA_011625925.1	SRR6519508	PDT000282424.2	NC_SI08	antigen_formula=7;r1,5,serotype=Infantis	
SAMN08513839	CDC	2017-12	USA	clinical	stool	PDS000055450.1	GCA_011611385.1	SRR6703928	PDT000288398.2	PNUSAS030547	antigen_formula=7;r1,5,serotype=Infantis	
SAMN08514389	Texas A&M University	2011	USA: Texas	clinical	feces	PDS000032409.1	GCA_003118475.1		PDT000317971.1	175	antigen_formula=7;r1,5,serotype=Infantis	
SAMN08581681	USDA-FSIS	2017	USA:WI	environmental/other	Product-Raw-Ground\, Comminuted or Otherwise Nonintact-Pork	PDS000103802.1	GCA_010886755.1	SRR6764209	PDT000288648.2	FSIS11706682	antigen_formula=7;r1,5,serotype=Infantis	
SAMN08737160	Universidade de Sao Paulo| Ciencia e Tecnologia de Alimentos	Laboratorio Microbiologia de Alimentos	2013	Brazil	environmental/other	slaughterhouse	PDS000027719.1	GCA_011582685.1	SRR6881656	PDT000297507.2	NC_SI8	antigen_formula=7;r1,5,serotype=Infantis	
SAMN08772945	CDC	2018-02	USA	clinical		PDS000027705.1	GCA_008465045.1	SRR6979300	PDT000296484.2	PNUSAS035586	antigen_formula=7;r1,5,serotype=Infantis	
SAMN08885910	CDC	2018-01	USA	clinical		PDS000100243.2	GCA_011614065.1	SRR6956027	PDT000303881.2	PNUSAS037962	antigen_formula=7;r1,5,serotype=Infantis	
SAMN08888262	USDA-FSIS	2018	USA:MN	environmental/other	Product-Raw-Ground\, Comminuted or Otherwise Nonintact-Pork	PDS000032457.9	GCA_008491865.1	SRR6959499	PDT000304086.2	FSIS11808941	antigen_formula=7;r1,5,serotype=Infantis	
SAMN08923845	FDA Contracted Laboratory	2016	USA:PA	environmental/other	environmental swab	PDS000027521.4	GCA_005535275.1	SRR7012008	PDT000306727.2	CFSAN078666	antigen_formula=7;r1,5,serotype=Infantis	
SAMN08951109	Universidade de Sao Paulo| Ciencia e Tecnologia de Alimentos	Laboratorio Microbiologia de Alimentos	2016	Brazil	environmental/other	chicken cage cleaning	PDS000020042.7	GCA_011576445.1	SRR7128413	PDT000312221.2	NC_SI690	antigen_formula=7;r1,5,serotype=Infantis	
SAMN08951117	Universidade de Sao Paulo| Ciencia e Tecnologia de Alimentos	Laboratorio Microbiologia de Alimentos	2016	Brazil	environmental/other	broiler chicken	PDS000027497.1	GCA_006286655.1	SRR7130356	PDT000312244.2	NC_SI10124816	antigen_formula=7;r1,5,serotype=Infantis	
SAMN08951144	Universidade de Sao Paulo| Ciencia e Tecnologia de Alimentos	Laboratorio Microbiologia de Alimentos	2016	Brazil	environmental/other	broiler chicken	PDS000078471.1	GCA_011159075.1	SRR7130523	PDT000312270.2	NC_SI10088955	antigen_formula=7;r1,5,serotype=Infantis	
SAMN09198063	CDC	2018-03	USA	clinical		PDS000078087.1	GCA_007495568.1	SRR7153425	PDT000313293.2	PNUSAS038447	antigen_formula=7;r1,5,serotype=Infantis	
SAMN09203649	PHE	2017-10	United Kingdom: United Kingdom	clinical	human	PDS000042856.1	GCA_011554475.1	SRR7157754	PDT000313510.2	437979	antigen_formula=7;r1,5,serotype=Infantis	
SAMN09228894	PHE	2015-09	United Kingdom: United Kingdom	clinical	human	PDS000046252.1	GCA_006931495.1	SRR7188023	PDT000317401.1	164937	antigen_formula=7;r1,5,serotype=Infantis	
SAMN09231662	CDC	2018-04	USA	clinical		PDS000079754.3	GCA_006701385.1	SRR7189191	PDT000317454.1	PNUSAS040740	antigen_formula=7;r1,5,serotype=Infantis	
SAMN09277804	PHE	2018-04	United Kingdom: United Kingdom	clinical	human	PDS000109030.5	GCA_007147925.1	SRR7230725	PDT000320993.1	527489	antigen_formula=7;r1,5,serotype=Infantis	
SAMN09281951	CDC	2017-12	USA	clinical	stool	PDS000038639.33	GCA_008546935.1	SRR7234709	PDT000321254.1	PNUSAS041308	antigen_formula=7;r1,5,serotype=Infantis	
SAMN09294311	CDC	2018-04	USA	clinical		PDS000104180.5	GCA_006556525.1	SRR7247765	PDT000323631.1	PNUSAS041587	antigen_formula=7;r1,5,serotype=Infantis	
SAMN09386518	PHE	2017-12	United Kingdom: United Kingdom	clinical	human	PDS000032464.2	GCA_007157645.1	SRR7284478	PDT000325991.1	465535	antigen_formula=7;r1,5,serotype=Infantis	
SAMN09388708	PHE	2018-05	United Kingdom: United Kingdom	clinical	human	PDS000023641.1	GCA_007160545.1	SRR7286702	PDT000327409.1	539160	antigen_formula=7;r1,5,serotype=Infantis	
SAMN09399742	CDC	2018-05	USA	clinical		PDS000084335.1	GCA_006760085.1	SRR7294485	PDT000328564.1	PNUSAS042039	antigen_formula=7;r1,5,serotype=Infantis	
SAMN09430466	CDC	2018-05	USA	clinical		PDS000032408.4	GCA_006648885.1	SRR7348225	PDT000330193.1	PNUSAS042722	antigen_formula=7;r1,5,serotype=Infantis	
SAMN09444132	USDA-FSIS	2018	USA:NE	environmental/other	animal-swine-market swine	PDS000097345.3	GCA_008447705.1	SRR7358414	PDT000332042.1	FSIS11810649	antigen_formula=7;r1,5,serotype=Infantis	
SAMN09474947	PHE	2018-05	United Kingdom: United Kingdom	clinical	human	PDS000037749.1	GCA_004182605.1	SRR7415032	PDT000334756.1	549480	antigen_formula=7;r1,5,serotype=Infantis	
SAMN09519832	PHE	2017-08	United Kingdom: United Kingdom	clinical	human	PDS000114379.13	GCA_007244655.1	SRR7456720	PDT000338989.1	402844	antigen_formula=7;r1,5,serotype=Infantis	
SAMN09519843	PHE	2018-04	United Kingdom: United Kingdom	clinical	human	PDS000042664.1	GCA_007246655.1	SRR7456730	PDT000338999.1	520172	antigen_formula=7;r1,5,serotype=Infantis	
SAMN09520820	PHE	2018-05	United Kingdom: United Kingdom	clinical	human	PDS000123669.1	GCA_007247035.1	SRR7456942	PDT000339157.1	550784	antigen_formula=7;r1,5,serotype=Infantis	
SAMN09545083	CDC	2018-06	USA	clinical		PDS000043520.4	GCA_006799925.1	SRR7468567	PDT000340935.1	PNUSAS044092	antigen_formula=7;r1,5,serotype=Infantis	
SAMN09545747	CDC	2018-06	USA	clinical		PDS000192431.1	GCA_006270965.1	SRR7468892	PDT000341066.1	PNUSAS044709	antigen_formula=7;r1,5,serotype=Infantis	

SAMN09624126	PHE	2015-12	United Kingdom: United Kingdom	clinical	human	PDS000109863.3	GCA_003897055.1	SRR7480204	PDT000342437.1	195856	antigen_formula=7;r:1,5,serotype=Infantis
SAMN09624165	PHE	2018-06	United Kingdom: United Kingdom	clinical	human	PDS000037029.6	GCA_007261025.1	SRR7480297	PDT000342474.1	563031	antigen_formula=7;r:1,5,serotype=Infantis
SAMN09624562	USDA-FSIS	2018	USA:WA	environmental/other	animal-cattle-dairy cow	PDS000051049.4	GCA_008542115.1	SRR7494598	PDT000342394.1	FSIS11811380	antigen_formula=7;r:1,5,serotype=Infantis
SAMN09631556	Lebanese Agriculture Reseach Institute	3/20/17	Lebanon	environmental/other	poultry	PDS000039494.10	GCA_006294295.1	SRR7504356	PDT000344445.1	NC_WHO_S031	antigen_formula=7;r:1,5,serotype=Infantis
SAMN09631587	Tripoli Governmental Hospital	6/28/12	Lebanon	clinical	stool	PDS000043087.2	GCA_006074255.1	SRR7504234	PDT000344418.1	NC_WHO_S011	antigen_formula=7;r:1,5,serotype=Infantis
SAMN09634349	PHE	2016-06	United Kingdom: United Kingdom	environmental/other		PDS000025793.5	GCA_004261045.1	SRR7501507	PDT000344182.1	267023	antigen_formula=7;r:1,5,serotype=Infantis
SAMN09643883	PHE	2018-02	United Kingdom: United Kingdom	environmental/other	food	PDS000116037.2	GCA_003895575.1	SRR7511919	PDT000345309.1	492517	antigen_formula=7;r:1,5,serotype=Infantis
SAMN09652008	PHE	2017-11	United Kingdom: United Kingdom	clinical	human	PDS000037080.2	GCA_007273955.1	SRR7516662	PDT000347719.1	442654	antigen_formula=7;r:1,5,serotype=Infantis
SAMN09664804	CDC	2018-04	USA	clinical		PDS000044779.8	GCA_006774425.1	SRR7524811	PDT000350232.1	PNUSAS040517	antigen_formula=7;r:1,5,serotype=Infantis
SAMN09683752	PHE	2015-08	United Kingdom: United Kingdom	clinical	human	PDS000109847.3	GCA_007266855.1	SRR7533380	PDT000351179.1	146194	antigen_formula=7;r:1,5,serotype=Infantis
SAMN09754816	CDC	2018-07	USA	clinical		PDS000032793.2	GCA_006725865.1	SRR7633390	PDT000356473.1	PNUSAS047827	antigen_formula=7;r:1,5,serotype=Infantis
SAMN09763734	University for Development Studies	5/23/16	Ghana: Tamale	environmental/other	goat	PDS000144668.6	GCA_004310545.1		PDT000471925.1	NC6	antigen_formula=7;r:1,5,serotype=Infantis
SAMN09850715	UNIETAR	11/3/14	Ecuador	environmental/other	chicken feed raw material	PDS000038582.5	GCA_005539355.1	SRR7765194	PDT000368021.1	2CTA-058	antigen_formula=7;r:1,5,serotype=Infantis
SAMN09880434	CDC	2018-07	USA	clinical		PDS000089300.5	GCA_006735785.1	SRR7739574	PDT000366369.1	PNUSAS051409	antigen_formula=7;r:1,5,serotype=Infantis
SAMN09881742	CDC	2018-07	USA	clinical		PDS000056320.1	GCA_006673585.1	SRR7739840	PDT000366570.1	PNUSAS050848	antigen_formula=7;r:1,5,serotype=Infantis
SAMN09981108	CDC	2018-07	USA	clinical		PDS000643454.9	GCA_006793525.1	SRR7789807	PDT000372489.1	PNUSAS050900	antigen_formula=7;r:1,5,serotype=Infantis
SAMN10061287	CDC	2018-08	USA	clinical		PDS000036910.9	GCA_006804165.1	SRR7830508	PDT000376831.1	PNUSAS051675	antigen_formula=7;r:1,5,serotype=Infantis
SAMN10067780	PHE	2018-08	United Kingdom: United Kingdom	clinical	human	PDS000037032.2	GCA_007017725.1	SRR7841519	PDT000377580.1	591845	antigen_formula=7;r:1,5,serotype=Infantis
SAMN10093785	PHE	2018-09	United Kingdom: United Kingdom	clinical	human	PDS000038706.5	GCA_007301615.1	SRR7879354	PDT000381485.1	600753	antigen_formula=7;r:1,5,serotype=Infantis
SAMN10093994	PHE	2018-07	United Kingdom: United Kingdom	clinical	human	PDS000047118.1	GCA_007303655.1	SRR7879443	PDT000381515.1	568457	antigen_formula=7;r:1,5,serotype=Infantis
SAMN10097135	USDA-FSIS	2018	USA:WI	environmental/other	animal-swine-sow	PDS000037637.66	GCA_003893135.1	SRR7883779	PDT000382185.1	FSIS11814083	antigen_formula=7;r:1,5,serotype=Infantis
SAMN10097556	PHE	2018-09	United Kingdom: United Kingdom	clinical	human	PDS000051594.3	GCA_007304795.1	SRR7884620	PDT000382400.1	604880	antigen_formula=7;r:1,5,serotype=Infantis
SAMN10241329	Adolfo Lutz Institute	2014	Brazil	clinical	urine	PDS000026845.91	GCA_003726045.1		PDT000407491.1	345/14	antigen_formula=7;r:1,5,serotype=Infantis
SAMN10261404	Laboratorio Nacional para la Investigacion en Inocuidad Alimentaria	11/10/10	Mexico	environmental/other	river water	PDS000037789.1	GCA_005698495.1	SRR8100769	PDT000395879.1	277	antigen_formula=7;r:1,5,serotype=Infantis
SAMN10261412	Laboratorio Nacional para la Investigacion en Inocuidad Alimentaria	1/11/10	Mexico	environmental/other	river water	PDS000037784.1	GCA_005553215.1	SRR8100706	PDT000395816.1	288	antigen_formula=7;r:1,5,serotype=Infantis
SAMN10391360	CDC	2018-09	USA	clinical		PDS000075945.2	GCA_005456065.1	SRR8170271	PDT000403737.1	PNUSAS058597	antigen_formula=7;r:1,5,serotype=Infantis
SAMN10396998	United States Food and Drug Administration\ Office of Regulatory Affairs\ New York Human and Animal Food Laboratory	2018	USA	environmental/other	animal feed	PDS000049113.18	GCA_005418315.1	SRR8187241	PDT000407082.1	FDA1089114-C001-015	antigen_formula=7;r:1,5,serotype=Infantis
SAMN10432554	CDC	2008-08	USA	clinical		PDS000070048.7	GCA_005419015.1	SRR8193039	PDT000407893.1	PNUSAS060838	antigen_formula=7;r:1,5,serotype=Infantis
SAMN10438031	PHE	2018-10	United Kingdom: United Kingdom	clinical	human	PDS000051043.2	GCA_004267035.1	SRR8201849	PDT000408739.1	621510	antigen_formula=7;r:1,5,serotype=Infantis
SAMN10535894	PHE	2018-11	United Kingdom: United Kingdom	clinical	human	PDS000040917.1	GCA_006995435.1	SRR8293580	PDT000415258.1	649009	antigen_formula=7;r:1,5,serotype=Infantis
SAMN10689695	CDC	2018-11	USA	clinical		PDS000040261.8	GCA_005822905.1	SRR8389569	PDT000430239.1	PNUSAS063888	antigen_formula=7;r:1,5,serotype=Infantis
SAMN10719940	PHE	2018-10	United Kingdom: United Kingdom	clinical	human	PDS000042831.2	GCA_007395275.1	SRR8427262	PDT000433960.1	637674	antigen_formula=7;r:1,5,serotype=Infantis
SAMN10767248	PHE	2019-01	United Kingdom: United Kingdom	clinical	human	PDS000090615.1	GCA_007400325.1	SRR8452337	PDT000442827.1	668776	antigen_formula=7;r:1,5,serotype=Infantis
SAMN10787668	CDC	2019-01	USA	clinical		PDS000045748.2	GCA_005728355.1	SRR8469414	PDT000447500.1	PNUSAS066020	antigen_formula=7;r:1,5,serotype=Infantis
SAMN10823717	PHE	2018-07	United Kingdom: United Kingdom	clinical	human	PDS000193824.1	GCA_007402465.1	SRR8490603	PDT000452815.1	568283	antigen_formula=7;r:1,5,serotype=Infantis
SAMN10823854	PHE	2019-01	United Kingdom: United Kingdom	clinical	human	PDS000059917.12	GCA_007584065.1	SRR8490759	PDT000452953.1	666308	antigen_formula=7;r:1,5,serotype=Infantis
SAMN10823872	PHE	2019-01	United Kingdom: United Kingdom	clinical	human	PDS000042727.1	GCA_007406405.1	SRR8490778	PDT000452972.1	674076	antigen_formula=7;r:1,5,serotype=Infantis
SAMN10836415	PHE	2018-05	United Kingdom: United Kingdom	clinical	human	PDS000078747.1	GCA_007507225.1	SRR8499365	PDT000454738.1	548505	antigen_formula=7;r:1,5,serotype=Infantis
SAMN10843061	PHE	2018-03	United Kingdom: United Kingdom	clinical	human	PDS000085361.2	GCA_007653075.1	SRR8503770	PDT000455525.1	516856	antigen_formula=7;r:1,5,serotype=Infantis
SAMN10867986	PHE	2017-11	United Kingdom: United Kingdom	clinical	human	PDS000055140.7	GCA_007511325.1	SRR8526011	PDT000458602.1	443513	antigen_formula=7;r:1,5,serotype=Infantis
SAMN10938074	CDC	2019-01	USA	clinical		PDS000514555.10	GCA_005489205.1	SRR8575496	PDT000465797.1	PNUSAS067523	antigen_formula=7;r:1,5,serotype=Infantis
SAMN11116125	USDA-FSIS	2019	USA:CA	environmental/other	Product-Raw-Ground\, Comminuted or Otherwise Nonintact-Pork	PDS000158697.9	GCA_006748285.1	SRR8723164	PDT000471165.2	FSIS11918346	antigen_formula=7;r:1,5,serotype=Infantis
SAMN11132121	CDC	2018-12	USA	clinical		PDS000091389.1	GCA_005664275.1	SRR8731274	PDT000478341.1	PNUSAS069447	antigen_formula=7;r:1,5,serotype=Infantis
SAMN11191435	United States Food and Drug Administration	2005	USA	environmental/other	food	PDS000055150.1	GCA_009884755.1	SRR10846303	PDT000656032.1	CFSAN077748	antigen_formula=7;r:1,5,serotype=Infantis
SAMN11191436	United States Food and Drug Administration	2005	USA	environmental/other	Not Provided	PDS000055154.1	GCA_009884785.1	SRR10845411	PDT000656012.1	CFSAN077781	antigen_formula=7;r:1,5,serotype=Infantis
SAMN11357597	USDA-FSIS	2019	USA:NY	environmental/other	chicken carcass	PDS000187106.2	GCA_005564218.1	SRR8842128	PDT000480962.2	FSIS11919738	antigen_formula=7;r:1,5,serotype=Infantis
SAMN11358944	CDC		USA	clinical		PDS000050914.3	GCA_005595215.1	SRR8866214	PDT000487084.1	PNUSAS067473	antigen_formula=7;r:1,5,serotype=Infantis
SAMN11404274	CDC	2019-01	USA	clinical		PDS000074034.3	GCA_005747195.1	SRR8888389	PDT000489479.1	PNUSAS068143	antigen_formula=7;r:1,5,serotype=Infantis
SAMN11419486	CDC	2019-03	USA	clinical		PDS000049425.3	GCA_005747495.1	SRR8899117	PDT000490458.1	PNUSAS072594	antigen_formula=7;r:1,5,serotype=Infantis
SAMN11456511	USDA-FSIS	2019	USA:MO	environmental/other	Product-Raw-Ground\, Comminuted or Otherwise Nonintact-Pork	PDS000047115.2	GCA_006750655.1	SRR8924514	PDT000491520.2	FSIS11919903	antigen_formula=7;r:1,5,serotype=Infantis
SAMN11492695	PHE	2019-04	United Kingdom: United Kingdom	clinical	human	PDS000064811.2	GCA_007536085.1	SRR8952508	PDT000495456.1	728347	antigen_formula=7;r:1,5,serotype=Infantis
SAMN11553499	USDA-FSIS	2019	USA:PA	environmental/other	comminuted chicken	PDS000070128.10	GCA_006838345.1	SRR9000510	PDT000497158.2	FSIS11920355	antigen_formula=7;r:1,5,serotype=Infantis
SAMN11582745	CDC	2019-04	USA	clinical		PDS000183373.1	GCA_005517095.1	SRR9019100	PDT000498900.1	PNUSAS075018	antigen_formula=7;r:1,5,serotype=Infantis
SAMN11885466	PHE	2019-05	United Kingdom: United Kingdom	clinical	human	PDS000097077.1	GCA_006396095.1	SRR9153222	PDT000509819.1	745826	antigen_formula=7;r:1,5,serotype=Infantis
SAMN11940217	CDC	2019-04	USA	clinical		PDS000462442.2	GCA_006216085.1	SRR9184979	PDT000517408.1	PNUSAS076176	antigen_formula=7;r:1,5,serotype=Infantis
SAMN11962996	CDC	2019-05	USA	clinical		PDS000066423.1	GCA_006410245.1	SRR9267746	PDT000519993.1	PNUSAS077934	antigen_formula=7;r:1,5,serotype=Infantis
SAMN12009173	PHE	2019-05	United Kingdom: United Kingdom	clinical	human	PDS000137628.1	GCA_006403735.1	SRR9261123	PDT000519516.1	752144	antigen_formula=7;r:1,5,serotype=Infantis
SAMN12041584	USDA-FSIS	2019	USA:TX	environmental/other	Product-Raw-Intact-Siluriformes	PDS000046533.8	GCA_006429715.1	SRR9289518	PDT000522016.1	FSIS11921837	antigen_formula=7;r:1,5,serotype=Infantis
SAMN12097925	Hamidreza Sodagari, Murdoch University	2018-06	Australia: Perth	environmental/other	Retail table eggs	PDS000050596.2	GCA_008362965.1		PDT000584671.1	RS43-4	antigen_formula=7;r:1,5,serotype=Infantis
SAMN12137517	PHE	2019-06	United Kingdom: United Kingdom	clinical	human	PDS000116007.3	GCA_007537215.1	SRR9595252	PDT000532571.1	761617	antigen_formula=7;r:1,5,serotype=Infantis
SAMN12249832	CDC	2019-06	USA	clinical		PDS000075101.1	GCA_007620775.1	SRR9667689	PDT000540535.1	PNUSAS082678	antigen_formula=7;r:1,5,serotype=Infantis
SAMN12307545	USDA-FSIS	2019	USA:FL	environmental/other	animal-cattle-dairy cow	PDS000097635.2	GCA_007700985.1	SRR9715837	PDT000545985.1	FSIS11922859	antigen_formula=7;r:1,5,serotype=Infantis
SAMN12349821	CDC	2019-07	USA	clinical		PDS000048482.1	GCA_007731405.1	SRR9822750	PDT000548029.1	PNUSAS084542	antigen_formula=7;r:1,5,serotype=Infantis
SAMN12414030	CDC	2019-06	USA	clinical		PDS000115416.2	GCA_007875085.1	SRR9860581	PDT000551293.1	PNUSAS083448	antigen_formula=7;r:1,5,serotype=Infantis
SAMN12421777	USDA-FSIS	2019	USA:MD	environmental/other	animal-chicken-young chicken	PDS000079497.1	GCA_007886005.1	SRR9879923	PDT000553388.1	FSIS11922889	antigen_formula=7;r:1,5,serotype=Infantis
SAMN12573511	PHE	2019-07	United Kingdom: United Kingdom	clinical	human	PDS000051578.3	GCA_011635395.1	SRR9968531	PDT000564313.1	786635	antigen_formula=7;r:1,5,serotype=Infantis
SAMN12644096	CDC	2019-07	USA	clinical		PDS000054025.2	GCA_008194925.1	SRR10026518	PDT000574468.1	PNUSAS093508	antigen_formula=7;r:1,5,serotype=Infantis
SAMN12648175	CDC	2019-08	USA	clinical		PDS000051346.4	GCA_008241505.1	SRR10031163	PDT000575238.1	PNUSAS093866	antigen_formula=7;r:1,5,serotype=Infantis
SAMN12684481	USDA-FSIS	2019	USA:GA	environmental/other	chicken carcass	PDS000184419.7	GCA_008301405.1	SRR10061004	PDT000579230.1	FSIS11902555	antigen_formula=7;r:1,5,serotype=Infantis
SAMN12708773	CDC	2019-08	USA	clinical		PDS000050601.9	GCA_008355585.1	SRR10079564	PDT000581355.1	PNUSAS096264	antigen_formula=7;r:1,5,serotype=Infantis
SAMN12768400	USDA-FSIS	2019	USA:NC	environmental/other	animal-chicken-young chicken	PDS000095625.1	GCA_008564995.1	SRR10126655	PDT000587219.1	FSIS11924523	antigen_formula=7;r:1,5,serotype=Infantis
SAMN12772799	CDC	2019-08	USA	clinical		PDS000182268.1	GCA_008594225.1	SRR10130076	PDT000587643.1	PNUSAS098861	antigen_formula=7;r:1,5,serotype=Infantis
SAMN12868843	PHE	2019-09	United Kingdom: United Kingdom	clinical	human	PDS000113590.1	GCA_008766315.1	SRR10198891	PDT000596059.1	804352	antigen_formula=7;r:1,5,serotype=Infantis
SAMN12924292	PHE	2019-09	United Kingdom: United Kingdom	clinical	human	PDS000054748.2	GCA_008864225.1	SRR10236702	PDT000601995.1	812272	antigen_formula=7;r:1,5,serotype=Infantis
SAMN12984342	CDC	2019-09	USA	clinical		PDS000052593.1	GCA_008920655.1	SRR10239041	PDT000602182.1	PNUSAS105578	antigen_formula=7;r:1,5,serotype=Infantis
SAMN13002624	PHE	2019-09	United Kingdom: United Kingdom	clinical	human	PDS000108921.1	GCA_008999995.1	SRR10254201	PDT000603905.1	814850	antigen_formula=7;r:1,5,serotype=Infantis
SAMN13002663	PHE	2019-09	United Kingdom: United Kingdom	clinical	human	PDS000051836.3	GCA_009001665.1	SRR10254284	PDT000603977.1	815519	antigen_formula=7;r:1,5,serotype=Infantis
SAMN13003126	CDC	2019-09	USA	clinical		PDS000052870.1	GCA_008970905.1	SRR10254706	PDT000603707.1	PNUSAS107152	antigen_formula=7;r:1,5,serotype=Infantis
SAMN13020340	CDC	2019-09	USA	clinical		PDS000054622.7	GCA_009114445.1	SRR10269936	PDT000604934.1	PNUSAS108110	antigen_formula=7;r:1,5,serotype=Infantis
SAMN13153270	CDC	2019-10	USA	clinical		PDS000055192.1	GCA_009415065.1	SRR10360075	PDT000620101.1	PNUSAS112529	antigen_formula=7;r:1,5,serotype=Infantis
SAMN13186319	CDC	2019-10	USA	clinical		PDS000060874.2	GCA_009547795.1	SRR10389106	PDT000623204.1	PNUSAS113628	antigen_formula=7;r:1,5,serotype=Infantis
SAMN13293484	PHE	2019-10	United Kingdom: United Kingdom	clinical	human	PDS000056479.4	GCA_010886395.1	SRR10454579	PDT000628818.1	836993	antigen_formula=7;r:1,5,serotype=Infantis
SAMN13527233	Dr. Mariam Siala, Veterinary Research Center of Sfax, Sfax, Tunisia	2016	Tunisia	environmental/other	poultry	PDS000137623.1	GCA_010437295.1	SRR10665540	PDT000643474.1	CFSAN072811	antigen_formula=7;r:1,5,serotype=Infantis
SAMN13616076	PHE	2019-12	United Kingdom: United Kingdom	clinical	human	PDS000158718.1	GCA_010524295.1	SRR10718490	PDT000646872.1	854873	antigen_formula=7;r:1,5,serotype=Infantis
SAMN13672825	CDC	2019-10	USA	clinical		PDS000126582.2	GCA_010382705.1	SRR10759715	PDT000649624.1	PNUSAS124698	antigen_formula=7;r:1,5,serotype=Infantis
SAMN13719582	Universidad Nacional Autonoma de Mexico \ Facultad de Medicina Veterinaria y Zootecnia \ Departamento de Medicina Preventiva y Salud Publica	7/8/19	Mexico	environmental/other	lake	PDS000107936.1	GCA_010801765.1	SRR10824937	PDT000654588.1	MPSPSA1934-1	antigen_formula=7;r:1,5,serotype=Infantis
SAMN13832406	United States Food and Drug Administration\Office of Regulatory Affairs\Pacific Northwest Laboratory	11/2/99	USA	environmental/other	roasted roll up	PDS000078706.4	GCA_010708195.1	SRR10875923	PDT000660235.1	FDA38947	antigen_formula=7;r:1,5,serotype=Infantis
SAMN13870237	CDC	2019-11	USA	clinical		PDS000199592.1	GCA_011210845.1	SRR10901315	PDT000663867.1	PNUSAS129393	antigen_formula=7;r:1,5,serotype=Infantis
SAMN13871629	CDC	2016-12	USA	clinical		PDS000063868.1	GCA_011210565.1	SRR10902848	PDT000664121.1	2016AM-2798	antigen_formula=7;r:1,5,serotype=Infantis
SAMN13874146	CDC	2019-12	USA	clinical		PDS000084381.6	GCA_011263295.1	SRR10904826	PDT000664269.1	PNUSAS129553	antigen_formula=7;r:1,5,serotype=Infantis
SAMN13874238	CDC	2019-12	USA	clinical		PDS000131950.1	GCA_011211285.1	SRR10904953	PDT000664310.1	PNUSAS129634	antigen_formula=7;r:1,5,serotype=Infantis
SAMN13978551		2019	Serbia: Kaljevo		farm	PDS000074190.3	GCA_013378415.1		PDT000782193.1	84182948	antigen_formula=7;r:1,5,serotype=Infantis
SAMN14271278	USDA-FSIS	2020	USA:NE	environmental/other	Product-Raw-Ground\, Comminuted or Otherwise Nonintact-Pork	PDS000083027.2	GCA_011387575.1	SRR11232367	PDT000703375.1	FSIS12029039	antigen_formula=7;r:1,5,serotype=Infantis
SAMN14338032	CDC	2020-03	USA	clinical		PDS000602777.14	GCA_011468675.1	SRR11273409	PDT000707807.1	PNUSAS137832	antigen_formula=7;r:1,5,serotype=Infantis
SAMN14381824	USDA-FSIS	2020	USA:NC	environmental/other	Product-Raw-Ground\, Comminuted or Otherwise Nonintact-Pork	PDS000121021.1	GCA_011710535.1	SRR11311367	PDT000709736.1	FSIS32003448	antigen_formula=7;r:1,5,serotype=Infantis
SAMN14420840	PHE	2020-03	United Kingdom: United Kingdom	clinical	human	PDS000100677.12	GCA_011725995.1	SRR11363628	PDT000711530.1	903358	antigen_formula=7;r:1,5,serotype=Infantis
SAMN14470113	CDC	2020-03	USA	clinical		PDS000061492.1	GCA_011765115.1	SRR11434672	PDT000714856.1	PNUSAS140016	antigen_formula=7;r:1,5,serotype=Infantis
SAMN14485905	CDC	2020-03	USA	clinical		PDS000199259.1	GCA_011770635.1	SRR11448898	PDT000716764.1	PNUSAS140154	antigen_formula=7;r:1,5,serotype=Infantis
SAMN14535191	PHE	2020-03	United Kingdom: United Kingdom	clinical	human	PDS000157548.1	GCA_012042135.1	SRR11479089	PDT000718599.1	917839	antigen_formula=7;r:1,5,serotype=Infantis
SAMN14539426	PHE	2020-03	United Kingdom: United Kingdom	clinical	human	PDS000141973.1	GCA_012040055.1	SRR11483198	PDT000718734.1	918361	antigen_formula=7;r:1,5,serotype=Infantis
SAMN14591479	CDC	2020-03	USA	clinical		PDS000064145.1	GCA_012280735.1	SRR11534368	PDT000721962.1	PNUSAS141629	antigen_formula=7;r:1,5,serotype=Infantis
SAMN14681089	USDA-FSIS	2020	USA:VA	environmental/other	animal-chicken-young chicken	PDS000085091.5	GCA_014581275.1	SRR11601058	PDT000725573.1	FSIS12029972	antigen_formula=7;r:1,5,serotype=Infantis
SAMN14681161	USDA-FSIS	2020	USA:TX	environmental/other	Product-Raw-Ground\, Comminuted or Otherwise Nonintact-Pork	PDS000099334.1	GCA_014581475.1	SRR11600316	PDT000725401.1	FSIS12027859	antigen_formula=7;r:1,5,serotype=Infantis
SAMN14833471	CDC	2020-04	USA	clinical		PDS000085526.3	GCA_014562575.1	SRR11679719	PDT000730718.1	PNUSAS143257	antigen_formula=7;r:1,5,serotype=Infantis
SAMN14944013	USDA-FSIS	2020	USA:IL	environmental/other	Product-Raw-Ground\, Comminuted or Otherwise Nonintact-Pork	PDS000073454.2	GCA_013120695.1	SRR11805695	PDT000739559.1	FSIS22027972	antigen_formula=7;r:1,5,serotype=Infantis
SAMN14998598	CDC	2020-05	USA	clinical		PDS000065055.2	GCA_013159765.1	SRR11831609	PDT000744389.1	PNUSAS145087	antigen_formula=7;r:1,5,serotype=Infantis
SAMN15353773	CDC	2020-06	USA	clinical		PDS000065341.4	GCA_013663135.1	SRR12077264	PDT000770873.1	PNUSAS149109	antigen_formula=7;r:1,5,serotype=Infantis
SAMN15408326	CDC	2020-06	USA	clinical		PDS000084924.5	GCA_013613795.1	SRR12121576	PDT000774460.1	PNUSAS150624	antigen_formula=7;r:1,5,serotype=Infantis
SAMN15599886	CDC	2020-07	USA	clinical		PDS000075181.1	GCA_013553175.1	SRR12286922	PDT000794578.1	PNUSAS154816	antigen_formula=7;r:1,5,serotype=Infantis
SAMN15666302	CRIE		Russia	clinical		PDS000123605.1	GCA_014334195.1		PDT000826650.1	SLR1_7866	antigen_formula=7;r:1,5,serotype=Infantis
SAMN15690267	CDC	2020-07	USA	clinical		PDS000096051.2	GCA_014047305.1	SRR12362162	PDT000800042.1	PNUSAS157028	antigen_formula=7;r:1,5,serotype=Infantis
SAMN15731431	CDC	2020-06	USA	clinical		PDS000115651.2	GCA_014064555.1	SRR12389156	PDT000802201.1	PNUSAS158014	antigen_formula=7;r:1,5,serotype=Infantis
SAMN15739189	PHE	2020-07	United Kingdom: United Kingdom	clinical	human	PDS000066720.42	GCA_014095955.1	SRR12396153	PDT000803152.2	968771	antigen_formula=7;r:1,5,serotype=Infantis

SAMN15739189	PHE	2020-07	United Kingdom: United Kingdom	clinical	human	PDS000066720.42	GCA_014095955.1	SRR12396153	PDT000803152.2	968771		antigen_formula=7;r1,5;serotype=Infantis
SAMN15800758	CDC	2020-07	USA	clinical		PDS000107940.1	GCA_014171115.1	SRR12435792	PDT000807709.1	PNUSAS160109		antigen_formula=7;r1,5;serotype=Infantis
SAMN15819199	CDC	2020-07	USA	clinical		PDS000070230.1	GCA_014206415.1	SRR12453816	PDT000810004.1	PNUSAS160458		antigen_formula=7;r1,5;serotype=Infantis
SAMN16055883	CDC	2020-08	USA	clinical		PDS000123661.2	GCA_014473755.1	SRR12586207	PDT000829726.1	PNUSAS165114		antigen_formula=7;r1,5;serotype=Infantis
SAMN16122018	USDA-FSIS	2020	USA:VA	environmental/other	comminuted beef	PDS000072087.4	GCA_014534895.1	SRR12630733	PDT000835692.1	FSIS22029258		antigen_formula=7;r1,5;serotype=Infantis
SAMN16272189	USDA-FSIS	2020	USA:MA	environmental/other	comminuted chicken	PDS000122790.1	GCA_014767695.1	SRR12717964	PDT000847647.1	FSIS22029291		antigen_formula=7;r1,5;serotype=Infantis
SAMN16337413	PHE	2020-09	United Kingdom: United Kingdom	clinical	human	PDS000108317.3	GCA_015190945.1	SRR12762325	PDT000852296.1	993678		antigen_formula=7;r1,5;serotype=Infantis
SAMN16364192	PHE	2020-08	United Kingdom: United Kingdom	environmental/other	animal	PDS000073011.2	GCA_015133135.1	SRR12770039	PDT000854949.1	980472		antigen_formula=7;r1,5;serotype=Infantis
SAMN16411109	PHE	2020-09	United Kingdom: United Kingdom	clinical	human	PDS000073469.9	GCA_014916095.1	SRR12806919	PDT000859487.1	997048		antigen_formula=7;r1,5;serotype=Infantis
SAMN16532588	USDA-FSIS	2020	USA:TX	environmental/other	comminuted beef	PDS000074153.3	GCA_014946475.1	SRR12884609	PDT000865573.1	FSIS12034721		antigen_formula=7;r1,5;serotype=Infantis
SAMN16559229	United States Food and Drug Administration	2014	USA	environmental/other	chicken feces	PDS000074309.4	GCA_015005255.1	SRR12901903	PDT000867972.1	CFSAN107146		antigen_formula=7;r1,5;serotype=Infantis
SAMN16559750	United States Food and Drug Administration	2013	USA	environmental/other	soy glycine max	PDS000106150.5	GCA_015004955.1	SRR12903412	PDT000868401.1	CFSAN107129		antigen_formula=7;r1,5;serotype=Infantis
SAMN16634374	United States Food and Drug Administration	2014	USA	environmental/other	pig snout	PDS000074994.3	GCA_015223515.1	SRR12971246	PDT000877378.1	CFSAN107167		antigen_formula=7;r1,5;serotype=Infantis
SAMN16729108	PHE	2020-11	United Kingdom: United Kingdom	environmental/other	other	PDS000075942.1	GCA_015448685.1	SRR13020572	PDT000888291.1	1021740		antigen_formula=7;r1,5;serotype=Infantis
SAMN16729110	PHE	2020-11	United Kingdom: United Kingdom	environmental/other	animal	PDS000075940.1	GCA_015448285.1	SRR13020587	PDT000888297.1	1021706		antigen_formula=7;r1,5;serotype=Infantis
SAMN16929635	PHAC	10/2/17	Canada	clinical	stool	PDS000103860.1	GCA_015868865.1	SRR13220791	PDT000906224.1	PNCS013645		antigen_formula=7;r1,5;serotype=Infantis
SAMN16955899	CDC	2020-10	USA	clinical		PDS000076988.2	GCA_015878545.1	SRR13166762	PDT000900999.1	PNUSAS182742		antigen_formula=7;r1,5;serotype=Infantis
SAMN16970700	PHAC	11/17/17	Canada	clinical	stool	PDS000101222.1	GCA_016021975.1	SRR13243600	PDT000912493.1	PNCS013746		antigen_formula=7;r1,5;serotype=Infantis
SAMN17043287	CDC	2020-10	USA	clinical		PDS000077142.2	GCA_015907785.1	SRR13234029	PDT000908164.1	PNUSAS184262		antigen_formula=7;r1,5;serotype=Infantis
SAMN17115240	CDC	2020-11	USA	clinical		PDS000079943.6	GCA_016133705.1	SRR13273149	PDT000919117.1	PNUSAS185106		antigen_formula=7;r1,5;serotype=Infantis
SAMN17128054	United States Food and Drug Administration	2017	USA	environmental/other	chicken carcass	PDS000078491.1	GCA_016220425.1	SRR13277022	PDT000919367.1	CFSAN107239		antigen_formula=7;r1,5;serotype=Infantis
SAMN17128497	United States Food and Drug Administration	2016	USA	clinical	feces	PDS000140572.1	GCA_016230965.1	SRR13277467	PDT000919645.1	CFSAN107229		antigen_formula=7;r1,5;serotype=Infantis
SAMN17153955	PHE	2020-12	United Kingdom: United Kingdom	clinical	human	PDS000078466.1	GCA_016438845.1	SRR13306476	PDT000925509.1	1052039		antigen_formula=7;r1,5;serotype=Infantis
SAMN17171074	United States Food and Drug Administration	2017	USA	clinical	fecal swab	PDS000078459.1	GCA_016437405.1	SRR13313289	PDT000925563.1	CFSAN107261		antigen_formula=7;r1,5;serotype=Infantis
SAMN17257874	PHAC	1/23/12	Canada	clinical		PDS000079390.1	GCA_017248655.1	SRR13497654	PDT000939079.1	PNCS007291		antigen_formula=7;r1,5;serotype=Infantis
SAMN17383779	CDC	2020-12	USA	clinical		PDS000125069.1	GCA_016726905.1	SRR13483593	PDT000938317.1	PNUSAS189349		antigen_formula=7;r1,5;serotype=Infantis
SAMN17505197	USDA-FSIS	2020	USA:GA	environmental/other	comminuted chicken	PDS000079450.1	GCA_017236405.1	SRR13510829	PDT000939807.1	FSIS32104559		antigen_formula=7;r1,5;serotype=Infantis
SAMN17516059	A. Szmolka, Center for Agricultural Research, Budapest; Hungary	2016	Hungary	environmental/other	broiler, faeces	PDS000082140.1	GCA_016943555.1	SRR13514232	PDT000972057.1			
SAMN17516060	A. Szmolka, Center for Agricultural Research, Budapest; Hungary	2016	Hungary	environmental/other	broiler, neckskin	PDS000082132.1	GCA_016944835.1	SRR13514175	PDT000972000.1			
SAMN17516063	A. Szmolka, Center for Agricultural Research, Budapest; Hungary	2016	Hungary	environmental/other	broiler, faeces	PDS000082137.3	GCA_016945475.1	SRR13514142	PDT000971967.1			antigen_formula=7;r1,5;serotype=Infantis
SAMN17516084	A. Szmolka, Center for Agricultural Research, Budapest; Hungary	2018	Hungary	environmental/other	broiler, caecum	PDS000082124.1	GCA_016945035.1	SRR13514168	PDT000971993.1			antigen_formula=7;r1,5;serotype=Infantis
SAMN17516087	A. Szmolka, Center for Agricultural Research, Budapest; Hungary	2018	Hungary	environmental/other	broiler, caecum	PDS000082138.1	GCA_016945055.1	SRR13514165	PDT000971990.1			
SAMN17516101	A. Szmolka, Center for Agricultural Research, Budapest; Hungary	2012	Hungary	clinical	human	PDS000018208.4	GCA_016945235.1	SRR13514149	PDT000971974.1			antigen_formula=7;r1,5;serotype=Infantis
SAMN17709534	USDA-FSIS	2021	USA:MA	environmental/other	comminuted chicken	PDS000113165.1	GCA_017182995.1	SRR13589110	PDT000944622.1	FSIS32104633		antigen_formula=7;r1,5;serotype=Infantis
SAMN17764451	PHE	2021-01	United Kingdom: United Kingdom	clinical	human	PDS000141967.1	GCA_017147695.1	SRR13614783	PDT000947298.1	1080526		antigen_formula=7;r1,5;serotype=Infantis
SAMN17897949	USDA-FSIS	2021	USA:NC	environmental/other	comminuted chicken	PDS000120913.1	GCA_017032155.1	SRR13697443	PDT000954831.1	FSIS12137750		antigen_formula=7;r1,5;serotype=Infantis
SAMN18051634	PHAC	2017	Canada	clinical	stool	PDS000091871.1	GCA_019007945.1	SRR14882919	PDT001075900.1	PNCS003079		antigen_formula=7;r1,5;serotype=Infantis
SAMN18076436	PHE	2021-02	United Kingdom: United Kingdom	clinical	human	PDS000091397.53	GCA_017102845.1	SRR13796438	PDT000974058.1	1108478		antigen_formula=7;r1,5;serotype=Infantis
SAMN18266709	CDC	2021-02	USA	clinical		PDS000083266.1	GCA_017333925.1	SRR13943446	PDT000983689.1	PNUSAS194677		antigen_formula=7;r1,5;serotype=Infantis
SAMN18527565	USDA-FSIS	2021	USA:GA	environmental/other	Product-Raw-Ground\, Comminuted or Otherwise Nonintact-Pork	PDS000083862.1	GCA_017612195.1	SRR14094605	PDT000993055.1	FSIS22130871		antigen_formula=7;r1,5;serotype=Infantis
SAMN18618424		2017	Australia	clinical		PDS000084313.4	GCA_017798665.1	SRR14143708	PDT000998910.1	AUSMDU00006635		antigen_formula=7;r1,5;serotype=Infantis
SAMN18618433		2017	Australia	clinical		PDS000084296.6	GCA_017867565.1	SRR14143698	PDT000998900.1	AUSMDU00007173		antigen_formula=7;r1,5;serotype=Infantis
SAMN18879982	CDC	2021-04	USA	clinical		PDS000085525.1	GCA_018186615.1	SRR14333439	PDT001015211.1	PNUSAS199475		antigen_formula=7;r1,5;serotype=Infantis
SAMN19012497	Pennsylvania State University	2018	Pakistan	environmental/other	stool	PDS000085537.1	GCA_018277845.1	SRR14421694	PDT001018665.1	FMBL10		antigen_formula=7;r1,5;serotype=Infantis
SAMN19012499	Pennsylvania State University	2018	Pakistan	environmental/other	stool	PDS000101017.1	GCA_018277225.1	SRR14421782	PDT001018697.1	FMBL19		antigen_formula=7;r1,5;serotype=Infantis
SAMN19289050	CDC	2021-04	USA	clinical		PDS000091223.3	GCA_018487225.1	SRR14608387	PDT001043729.1	PNUSAS202647		antigen_formula=7;r1,5;serotype=Infantis
SAMN19414275	USDA-FSIS	2021	USA:GA	environmental/other	raw intact chicken	PDS000098565.4	GCA_018601755.1	SRR14682907	PDT001049846.1	FSIS32105230		antigen_formula=7;r1,5;serotype=Infantis
SAMN19593215		2021-05	USA	clinical		PDS000091383.3	GCA_018774725.1	SRR14748472	PDT001062963.1	PNUSAS204958		antigen_formula=7;r1,5;serotype=Infantis
SAMN19652398	PHE	2007-11	United Kingdom	clinical	food	PDS000085358.2	GCA_018843675.1	SRR14774719	PDT001066399.1	hPHE_7		antigen_formula=7;r1,5;serotype=Infantis
SAMN19652412	PHE	2003-09	United Kingdom	clinical	food	PDS000091387.1	GCA_018843635.1	SRR14774721	PDT001066401.1	hPHE_60		antigen_formula=7;r1,5;serotype=Infantis
SAMN19652413	PHE	2004-05	United Kingdom	clinical	human	PDS000091390.1	GCA_018841215.1	SRR14774830	PDT001066510.1	hPHE_48		antigen_formula=7;r1,5;serotype=Infantis
SAMN19652416	PHE	2004-08	United Kingdom	clinical	human	PDS000074019.6	GCA_018841435.1	SRR14774821	PDT001066501.1	hPHE_58		antigen_formula=7;r1,5;serotype=Infantis
SAMN19652424	PHE	2006-08	United Kingdom	clinical	human	PDS000109846.4	GCA_018843175.1	SRR14774740	PDT001066420.1	hPHE_30		antigen_formula=7;r1,5;serotype=Infantis
SAMN19652447	PHE	2009-04	United Kingdom	clinical	human	PDS000091391.1	GCA_018841135.1	SRR14774832	PDT001066512.1	hPHE_210		antigen_formula=7;r1,5;serotype=Infantis
SAMN19652531	PHE	2013-09	United Kingdom	clinical	human	PDS000096922.1	GCA_018842255.1	SRR14774777	PDT001066457.1	hPHE_161		antigen_formula=7;r1,5;serotype=Infantis
SAMN19652550	PHE	2001-08	United Kingdom	clinical	human	PDS000091402.1	GCA_018841115.1	SRR14774835	PDT001066515.1	hPHE_102		antigen_formula=7;r1,5;serotype=Infantis
SAMN19652557	PHE	2002-10	United Kingdom	clinical	human	PDS000091398.1	GCA_018844595.1	SRR14774673	PDT001066353.1	hPHE_111		antigen_formula=7;r1,5;serotype=Infantis
SAMN19658827	PHE	2021-05	United Kingdom: United Kingdom	clinical	human	PDS000092557.1	GCA_018839195.1	SRR14782968	PDT001066655.1	1194862		antigen_formula=7;r1,5;serotype=Infantis
SAMN19697350	CDC	2021-06	USA	clinical		PDS000123022.1	GCA_018938755.1	SRR14810345	PDT001069811.1	PNUSAS206540		antigen_formula=7;r1,5;serotype=Infantis
SAMN19801305	CDC	2021-06	USA	clinical		PDS000094078.1	GCA_018987905.1	SRR14875741	PDT001074853.1	PNUSAS207772		antigen_formula=7;r1,5;serotype=Infantis
SAMN20304972	USDA-FSIS	2021	USA:NC	environmental/other	raw intact chicken	PDS000137700.3	GCA_019280955.1	SRR15192301	PDT001092503.1	FSIS22132003		antigen_formula=7;r1,5;serotype=Infantis
SAMN20396438	United States Food and Drug Administration\ Office of Regulatory Affairs\ Irvine Human and Animal Food Laboratory	2021	USA	environmental/other	environmental swab sponge	PDS000095989.2	GCA_019344695.1	SRR15245538	PDT001096170.1	FDA1165060-S095-001		antigen_formula=7;r1,5;serotype=Infantis
SAMN20527766	CDC	2021-07	USA	clinical		PDS000113504.2	GCA_019561215.1	SRR15324150	PDT001101731.1	PNUSAS216992		antigen_formula=7;r1,5;serotype=Infantis
SAMN20527825	CDC	2021-07	USA	clinical		PDS000170118.1	GCA_019559155.1	SRR15326439	PDT001101835.1	PNUSAS217051		antigen_formula=7;r1,5;serotype=Infantis
SAMN20571094	CDC	2021-07	USA	clinical		PDS000152465.1	GCA_019472505.1	SRR15345379	PDT001104377.1	PNUSAS217869		antigen_formula=7;r1,5;serotype=Infantis
SAMN20571099	CDC	2021-07	USA	clinical		PDS000097083.1	GCA_019472545.1	SRR15345833	PDT001104379.1	PNUSAS217867		antigen_formula=7;r1,5;serotype=Infantis
SAMN20603143	CDC	2021-07	USA	clinical		PDS000094065.9	GCA_019481105.1	SRR15361674	PDT001103815.1	PNUSAS218526		antigen_formula=7;r1,5;serotype=Infantis
SAMN20840725	CDC	2021-08	USA	clinical		PDS000192037.1	GCA_019892195.1	SRR15445874	PDT001110825.1	PNUSAS220357		antigen_formula=7;r1,5;serotype=Infantis
SAMN20934367		2021-07	USA	clinical		PDS000096415.2	GCA_019736515.1	SRR15571276	PDT001115471.1	PNUSAS222625		antigen_formula=7;r1,5;serotype=Infantis
SAMN20977398	USDA-FSIS	2021	USA:NC	environmental/other	comminuted chicken	PDS000095162.1	GCA_019772475.1	SRR15614015	PDT001116938.1	FSIS12142011		antigen_formula=7;r1,5;serotype=Infantis
SAMN20980434	USDA-FSIS	2021	USA:TX	environmental/other	comminuted beef	PDS000095158.2	GCA_019773935.1	SRR15613652	PDT001116863.1	FSIS12142105		antigen_formula=7;r1,5;serotype=Infantis
SAMN21161573	CDC	2021-08	USA	clinical		PDS000095287.1	GCA_019834305.1	SRR15680218	PDT001120754.1	PNUSAS224588		antigen_formula=7;r1,5;serotype=Infantis
SAMN21250567	USDA-FSIS	2021	USA:OH	environmental/other	product-raw-intact-beef	PDS000095565.3	GCA_019896415.1	SRR15736185	PDT001125085.1	FSIS32105527		antigen_formula=7;r1,5;serotype=Infantis
SAMN21371013	University of Ibadan \ Department of Microbiology	2012	Nigeria	clinical	feces	PDS000095875.2	GCA_019931995.1	SRR15833393	PDT001127614.1	LZ125_002		antigen_formula=7;r1,5;serotype=Infantis
SAMN21423939	CDC	2021-08	USA	clinical		PDS000189025.1	GCA_019995125.1	SRR15886064	PDT001130168.1	PNUSAS227926		antigen_formula=7;r1,5;serotype=Infantis
SAMN21569193	CDC	2021-09	USA	clinical		PDS000101939.1	GCA_020115295.1	SRR16021945	PDT001136341.1	PNUSAS230495		antigen_formula=7;r1,5;serotype=Infantis
SAMN22822631	USDA-FSIS	2021	USA:OK	environmental/other	Animal-Chicken-Young Chicken (cecal)	PDS000179719.1	GCA_020711735.1	SRR16680954	PDT001166872.1	FSIS12106541		antigen_formula=7;r1,5;serotype=Infantis
SAMN22824524	CDC	2021-10	USA	clinical		PDS000183122.1	GCA_020717765.1	SRR16684209	PDT001167112.1	PNUSAS241292		antigen_formula=7;r1,5;serotype=Infantis
SAMN23099042	USDA-FSIS	2021	USA:NC	environmental/other	Animal-Chicken-Young Chicken (cecal)	PDS000120610.2	GCA_020864745.1	SRR16931955	PDT001174088.1	FSIS12106729		antigen_formula=7;r1,5;serotype=Infantis
SAMN23168049		2021-10	USA	clinical		PDS000177313.1	GCA_020955335.1	SRR16952070	PDT001175566.1	PNUSAS244684		antigen_formula=7;r1,5;serotype=Infantis
SAMN23442717		2021-10	USA	clinical		PDS000119545.8	GCA_020986665.1	SRR17041150	PDT001184945.1	PNUSAS246893		antigen_formula=7;r1,5;serotype=Infantis
SAMN23704689		2021-11	USA	clinical		PDS000101021.1	GCA_021070505.1	SRR17143269	PDT001197199.1	PNUSAS248642		antigen_formula=7;r1,5;serotype=Infantis

Supplemental Table 3.6. Select metadata downloaded from NCBI for serovar Infantis isolates included in Figure S3.6B. Blue highlight indicates samples chosen for Figure 3.4A.

BioSample	Collected by	Collection date	Location	Isolation type	Isolation source	SNP cluster	Assembly	Run	Isolate	Strain	Computed types
SAMD00019623		2000	Japan:Kyusyu			PDS000116034.2	GCA_024957485.1	DRR022716	PDT001391547.1	G117	antigen_formula=7:r:1,5,serotype=Infantis
SAMEA11580171	National Institute for Communicable Disease, South Africa	1/16/13	South Africa	clinical	Human Stool	PDS000077667.3	GCA_024267295.1	ERR7671404	PDT001358639.1		antigen_formula=7:r:1,5,serotype=Infantis
SAMEA6942530		1995	Germany		broiler	PDS000077667.3	GCA_016111585.1	ERR4233826	PDT000916645.1		antigen_formula=7:r:1,5,serotype=Infantis
SAMEA7540849		2018	Germany	clinical	stool	PDS000076389.2	GCA_020141375.1	ERR4832839	PDT001137335.1		antigen_formula=7:r:1,5,serotype=Infantis
SAMN02345192	United States Food and Drug Administration\ Office of Regulatory Affairs\ Atlanta Human and Animal Food Laboratory	5/13/11	USA	environmental/other	frozen calamari ring	PDS000032456.133	GCA_011633805.1	SRR1212330	PDT000025179.3	FSE0064	antigen_formula=7:r:1,5,serotype=Infantis
SAMN02698446	United States Food and Drug Administration	9/8/10	USA	environmental/other	meat bone meal	PDS000031702.5	GCA_010505935.1	SRR1503322	PDT000034347.3	FSE0192	antigen_formula=7:r:1,5,serotype=Infantis
SAMN02699512	Minnesota Department of Health	11/15/02	USA:NC	environmental/other	tissue	PDS000043301.3	GCA_011448675.1	SRR1461812	PDT000033968.3	MDH-2014-00346	antigen_formula=7:r:1,5,serotype=Infantis
SAMN02699691	Minnesota Department of Health	8/1/03	USA:MN	environmental/other	liver	PDS000032186.7	GCA_006629545.1	SRR1586570	PDT000039706.4	MDH-2014-00525	antigen_formula=7:r:1,5,serotype=Infantis
SAMN02849840	FDA Contracted Laboratory	1/9/07	USA:CO	environmental/other	feces bovine	PDS000032425.4	GCA_011452475.1	SRR1505456	PDT000034455.3	CFSAN018572	antigen_formula=7:r:1,5,serotype=Infantis
SAMN02902678	FDA Contracted Laboratory	1/9/07	USA:WA	environmental/other	bovine necropsy	PDS000031652.1	GCA_006846065.1	SRR1528522	PDT000035947.2	CFSAN022438	antigen_formula=7:r:1,5,serotype=Infantis
SAMN03169192	PHE	2012-11	United Kingdom: Midlands and East of England	clinical	human	PDS000003938.55	GCA_008002085.1	SRR1645904	PDT000043329.5	H124700500	antigen_formula=7:r:1,5,serotype=Infantis
SAMN03199677	FDA Contracted Laboratory	4/16/07	USA:SD	environmental/other	beef	PDS000032452.2	GCA_006856405.1	SRR1722878	PDT000045990.2	CFSAN027146	antigen_formula=7:r:1,5,serotype=Infantis
SAMN03465894	PHE	2014-10	United Kingdom: North of England	clinical	human	PDS000032426.13	GCA_011091665.1	SRR1957992	PDT000052975.2	57602	antigen_formula=7:r:1,5,serotype=Infantis
SAMN04054235	USDA-FSIS	2015	USA:WA	environmental/other	Animal-Swine-Roaster Swine	PDS000032458.97	GCA_008651375.1	SRR2353813	PDT000082883.2	FSIS1503893	antigen_formula=7:r:1,5,serotype=Infantis
SAMN04337197	USDA-FSIS	2015	USA:PA	environmental/other	animal-swine-sow	PDS000032459.168	GCA_008730515.1	SRR3000039	PDT000097431.2	FSIS1503917	antigen_formula=7:r:1,5,serotype=Infantis
SAMN04600407	PHE	2015-04	United Kingdom: United Kingdom	clinical	human	PDS000028362.2	GCA_008852785.1	SRR3322087	PDT000122482.2	107287	antigen_formula=7:r:1,5,serotype=Infantis
SAMN06346072		2016-11	USA	clinical		PDS000032462.11	GCA_006635985.1	SRR5276637	PDT000189185.2	PNUSAS008690	antigen_formula=7:r:1,5,serotype=Infantis
SAMN07460708		2017-07	USA	clinical		PDS000032451.1	GCA_006761645.1	SRR5929567	PDT000232884.2	PNUSAS020390	antigen_formula=7:r:1,5,serotype=Infantis
SAMN08376490	CDC	2015-03	USA	clinical		PDS000103799.1	GCA_006262915.1	SRR6480645	PDT000278896.2	2015AM-0283	antigen_formula=7:r:1,5,serotype=Infantis
SAMN08581681	USDA-FSIS	2017	USA:WI	environmental/other	Product-Raw-Ground\, Comminuted or Otherwise Nonintact-Pork	PDS000103802.1	GCA_010886755.1	SRR6764209	PDT000288648.2	FSIS11706682	antigen_formula=7:r:1,5,serotype=Infantis
SAMN08888262	USDA-FSIS	2018	USA:MN	environmental/other	Product-Raw-Ground\, Comminuted or Otherwise Nonintact-Pork	PDS000032457.9	GCA_008491865.1	SRR6959499	PDT000304086.2	FSIS11808941	antigen_formula=7:r:1,5,serotype=Infantis
SAMN09444132	USDA-FSIS	2018	USA:NE	environmental/other	animal-swine-market swine	PDS000097345.3	GCA_008447705.1	SRR7358414	PDT000332042.1	FSIS11810649	antigen_formula=7:r:1,5,serotype=Infantis
SAMN11132121	CDC	2018-12	USA	clinical		PDS000091389.1	GCA_005664275.1	SRR8731274	PDT000478341.1	PNUSAS069447	antigen_formula=7:r:1,5,serotype=Infantis
SAMN15408326	CDC	2020-06	USA	clinical		PDS000084924.5	GCA_013613795.1	SRR12121576	PDT000774460.1	PNUSAS150624	antigen_formula=7:r:1,5,serotype=Infantis
SAMN16122018	USDA-FSIS	2020	USA:VA	environmental/other	comminuted beef	PDS000072087.4	GCA_014534895.1	SRR12630733	PDT000835692.1	FSIS22029258	antigen_formula=7:r:1,5,serotype=Infantis
SAMN19652416	PHE	2004-08	United Kingdom	clinical	human	PDS000074019.6	GCA_018841435.1	SRR14774821	PDT001066501.1	hPHE_58	antigen_formula=7:r:1,5,serotype=Infantis
SAMN20603143	CDC	2021-07	USA	clinical		PDS000094065.9	GCA_019481105.1	SRR15361674	PDT001103815.1	PNUSAS218526	antigen_formula=7:r:1,5,serotype=Infantis
SAMN25608883		2014	Slovenia	clinical		PDS000116014.1	GCA_024995025.1	SRR17880716	PDT001389277.1	S105	antigen_formula=7:r:1,5,serotype=Infantis
SAMN30385371		2022-08	USA	clinical		PDS000116131.2	GCA_024944465.1	SRR21120049	PDT001392917.1	PNUSAS292578	antigen_formula=7:r:1,5,serotype=Infantis
SAMN39330787				clinical		PDS000148192.2	GCA_035583875.1	SRR27478710	PDT002050087.1	PNUSAS413508	antigen_formula=7:r:1,5,serotype=Infantis

Supplemental Table 3.7. Select metadata downloaded from NCBI for serovar Typhimurium isolates included in Figure 3.5A.

BioSample	Collected by	Collection date	Location	Isolation source	Isolation type	SNP cluster	Run	Assembly	Isolate	Strain	Computed types
SAMN07424695	CDC	2017-06	USA	stool	clinical	PDS000051476.18	SRR5888672	GCA_008884465.1	PDT000230419.2	PNUSAS018095	antigen_formula=4:i:1,2,serotype=Typhimurium
SAMN08407830	CDC	2018-01	USA	stool	clinical	PDS000076391.10	SRR6729918	GCA_016381345.1	PDT000282937.4	PNUSAS032925	antigen_formula=4:i:1,2,serotype=Typhimurium
SAMN08607476	CDC	2017-09	USA		clinical	PDS000049109.27	SRR6782607	GCA_007561945.1	PDT000289579.2	PNUSAS033431	antigen_formula=4:i:1,2,serotype=Typhimurium
SAMN10241133	CDC	2018-09	USA		clinical	PDS000038355.20	SRR8054302	GCA_016300485.1	PDT000391315.1	PNUSAS056026	antigen_formula=4:i:1,2,serotype=Typhimurium
SAMN10359377	CDC	2018-10	USA		clinical	PDS000038355.20	SRR8144669	GCA_016294885.1	PDT000400298.1	PNUSAS059811	antigen_formula=4:i:1,2,serotype=Typhimurium
SAMN10395430	FDA Contracted Laboratory	2017	USA:CA	finished almond	environmental/other	PDS000038355.20	SRR8176610	GCA_005628135.1	PDT000405208.1	CFSAN087747	antigen_formula=4:i:1,2,serotype=Typhimurium
SAMN10689682	CDC	2018-12	USA		clinical	PDS000038355.20	SRR8389118	GCA_016275845.1	PDT000430161.1	PNUSAS064747	antigen_formula=4:i:1,2,serotype=Typhimurium
SAMN11102910	KY-M	7/9/18	USA:KY	Odocoileus virginianus Feces	environmental/other	PDS000044303.2	SRR8767784	GCA_006239385.1	PDT000481252.1	SAL-18-VL-LA-KY-0011	antigen_formula=4:i:1,2,serotype=Typhimurium
SAMN11582741	CDC	2019-04	USA		clinical	PDS000049109.27	SRR9019092	GCA_016267365.1	PDT000498892.1	PNUSAS075012	antigen_formula=4:i:1,2,serotype=Typhimurium
SAMN12036221	CDC	2019-05	USA		clinical	PDS000054358.20	SRR9283102	GCA_006425235.1	PDT000521479.1	PNUSAS077320	antigen_formula=4:i:1,2,serotype=Typhimurium
SAMN12079230	CDC	2019-06	USA		clinical	PDS000049109.27	SRR9312597	GCA_006467545.1	PDT000524007.1	PNUSAS079150	antigen_formula=4:i:1,2,serotype=Typhimurium
SAMN12147598	CDC	2019-05	USA		clinical	PDS000051476.18	SRR9610707	GCA_007615915.1	PDT000533382.1	PNUSAS079837	antigen_formula=4:i:1,2,serotype=Typhimurium
SAMN12392331	CDC	2019-06	USA		clinical	PDS000049109.27	SRR9856451	GCA_007869825.1	PDT000550967.1	PNUSAS085644	antigen_formula=4:i:1,2,serotype=Typhimurium
SAMN12537850	CDC	2019-06	USA		clinical	PDS000049109.27	SRR9932329	GCA_008202105.1	PDT000558638.1	PNUSAS088114	antigen_formula=4:i:1,2,serotype=Typhimurium
SAMN12842595	CDC	2019-09	USA		clinical	PDS000038355.20	SRR10179687	GCA_016241875.1	PDT000593792.1	PNUSAS102443	antigen_formula=4:i:1,2,serotype=Typhimurium
SAMN12889186	CDC	2019-09	USA		clinical	PDS000051476.18	SRR10215630	GCA_008825405.1	PDT000598887.1	PNUSAS104449	antigen_formula=4:i:1,2,serotype=Typhimurium
SAMN12924378	CDC	2019-10	USA		clinical	PDS000038355.20	SRR10236752	GCA_016230825.1	PDT000601905.1	PNUSAS105693	antigen_formula=4:i:1,2,serotype=Typhimurium
SAMN13107897	CDC	2019-09	USA		clinical	PDS000049109.27	SRR10340447	GCA_009375435.1	PDT000617246.1	PNUSAS110904	antigen_formula=4:i:1,2,serotype=Typhimurium
SAMN13244854	CDC	2019-10	USA		clinical	PDS000038355.20	SRR10420101	GCA_016225105.1	PDT000625882.1	PNUSAS115298	antigen_formula=4:i:1,2,serotype=Typhimurium
SAMN13536205	CDC	2019-11	USA		clinical	PDS000054358.20	SRR10664442	GCA_011182755.1	PDT000643427.1	PNUSAS121348	antigen_formula=4:i:1,2,serotype=Typhimurium
SAMN14083425	CDC	2020-01	USA		clinical	PDS000149796.1	SRR11067570	GCA_011489485.1	PDT000682507.1	PNUSAS134090	antigen_formula=4:i:1,2,serotype=Typhimurium
SAMN14438389	CDC	2020-03	USA		clinical	PDS000054358.20	SRR11401971	GCA_011742965.1	PDT000712723.1	PNUSAS139746	antigen_formula=4:i:1,2,serotype=Typhimurium
SAMN15376831	CDC	2020-06	USA		clinical	PDS000051476.18	SRR12097834	GCA_013651915.1	PDT000771998.1	PNUSAS149604	antigen_formula=4:i:1,2,serotype=Typhimurium
SAMN15399837	CDC	2020-06	USA		clinical	PDS000150767.2	SRR12109672	GCA_016167695.1	PDT000773851.1	PNUSAS149967	antigen_formula=4:i:1,2,serotype=Typhimurium
SAMN15419583	CDC	2020-06	USA		clinical	PDS000051476.18	SRR12129538	GCA_016167435.1	PDT000774770.1	PNUSAS150862	antigen_formula=4:i:1,2,serotype=Typhimurium
SAMN15460852	CDC	2020-06	USA		clinical	PDS000051476.18	SRR12160529	GCA_013522295.1	PDT000783830.1	PNUSAS151518	antigen_formula=4:i:1,2,serotype=Typhimurium
SAMN15493347	CDC	2020-06	USA		clinical	PDS000051476.18	SRR12185040	GCA_013494565.1	PDT000784644.1	PNUSAS151488	antigen_formula=4:i:1,2,serotype=Typhimurium
SAMN15517233	CDC	2020-06	USA		clinical	PDS000049109.27	SRR12207372	GCA_013526655.1	PDT000787128.1	PNUSAS152531	antigen_formula=4:i:1,2,serotype=Typhimurium
SAMN15575936	CDC	2020-07	USA		clinical	PDS000038355.20	SRR12263105	GCA_013545025.1	PDT000790069.1	PNUSAS153944	antigen_formula=4:i:1,2,serotype=Typhimurium
SAMN15775940	CDC	2020-07	USA		clinical	PDS000049109.27	SRR12423984	GCA_016160075.1	PDT000806632.1	PNUSAS159350	antigen_formula=4:i:1,2,serotype=Typhimurium
SAMN15863143	CDC	2020-08	USA		clinical	PDS000051476.18	SRR12481490	GCA_014257065.1	PDT000817064.1	PNUSAS161843	antigen_formula=4:i:1,2,serotype=Typhimurium
SAMN16124742	CDC	2020-08	USA		clinical	PDS000038355.20	SRR12633952	GCA_016158575.1	PDT000835764.1	PNUSAS166591	antigen_formula=4:i:1,2,serotype=Typhimurium
SAMN16134730	CDC	2020-08	USA		clinical	PDS000054358.20	SRR12643153	GCA_014604815.1	PDT000836650.1	PNUSAS167241	antigen_formula=4:i:1,2,serotype=Typhimurium
SAMN16451839	CDC	2020-09	USA		clinical	PDS000051476.18	SRR12827167	GCA_016154295.1	PDT000862761.1	PNUSAS175260	antigen_formula=4:i:1,2,serotype=Typhimurium
SAMN16452222	CDC	2020-08	USA		clinical	PDS000054358.20	SRR12826943	GCA_014953285.1	PDT000862571.1	PNUSAS173666	antigen_formula=4:i:1,2,serotype=Typhimurium
SAMN16522471	CDC	2020-09	USA		clinical	PDS000049109.27	SRR12883077	GCA_014927845.1	PDT000865377.1	PNUSAS176648	antigen_formula=4:i:1,2,serotype=Typhimurium
SAMN17138041	CDC	2020-11	USA		clinical	PDS000054358.20	SRR13289723	GCA_016308605.1	PDT000921969.1	PNUSAS186397	antigen_formula=4:i:1,2,serotype=Typhimurium
SAMN17251969	CDC	2020-12	USA		clinical	PDS000076391.10	SRR13380180	GCA_016589225.1	PDT000929858.1	PNUSAS187431	antigen_formula=4:i:1,2,serotype=Typhimurium
SAMN17394085	CDC	2020-12	USA		clinical	PDS000049109.27	SRR13495192	GCA_017249195.1	PDT000939029.1	PNUSAS189168	antigen_formula=4:i:1,2,serotype=Typhimurium
SAMN18743077	PHAC	2018	Canada	stool	clinical	PDS000049109.27	SRR14280642	GCA_018116045.1	PDT001009683.1	PNCS006221	antigen_formula=4:i:1,2,serotype=Typhimurium
SAMN19107277		2021-04	USA		clinical	PDS000049109.27	SRR14494720	GCA_018352055.1	PDT001024284.1	PNUSAS201133	antigen_formula=4:i:1,2,serotype=Typhimurium
SAMN19974806	CDC	2021-06	USA		clinical	PDS000192391.1	SRR15006564	GCA_019087325.1	PDT001080017.1	PNUSAS209577	antigen_formula=4:i:1,2,serotype=Typhimurium
SAMN20090106		2021-06	USA		clinical	PDS000093278.1	SRR15058485	GCA_019162375.1	PDT001083896.1	1019624001	antigen_formula=4:i:1,2,serotype=Typhimurium
SAMN20306031		2021-06	USA		clinical	PDS000093278.1	SRR15192434	GCA_019290995.1	PDT001092596.1	1021644001	antigen_formula=4:i:1,2,serotype=Typhimurium
SAMN20677178	CDC	2021-07	USA		clinical	PDS000076391.10	SRR15377064	GCA_019499845.1	PDT001106150.1	PNUSAS219220	antigen_formula=4:i:1,2,serotype=Typhimurium
SAMN20873945	CDC	2021-07	USA		clinical	PDS000051476.18	SRR15540741	GCA_019707935.1	PDT001113791.1	PNUSAS221923	antigen_formula=4:i:1,2,serotype=Typhimurium
SAMN21442709	CDC	2021-08	USA		clinical	PDS000051476.18	SRR15904945	GCA_020010025.1	PDT001130701.1	PNUSAS228169	antigen_formula=4:i:1,2,serotype=Typhimurium
SAMN21570178	CDC	2021-08	USA		clinical	PDS000054358.20	SRR16023372	GCA_020121535.1	PDT001136369.1	PNUSAS230456	antigen_formula=4:i:1,2,serotype=Typhimurium
SAMN22575512	CDC	2021-10	USA		clinical	PDS000051476.18	SRR16578854	GCA_020649135.1	PDT001162192.1	PNUSAS239477	antigen_formula=4:i:1,2,serotype=Typhimurium
SAMN27562167	CDC	2022-03	USA		clinical	PDS000054358.20	SRR18741443	GCA_022982875.1	PDT001286699.1	PNUSAS265900	antigen_formula=4:i:1,2,serotype=Typhimurium
SAMN29058726		2022-05	USA		clinical	PDS000049109.27	SRR19660945	GCA_023735575.1	PDT001329142.1	PNUSAS277091	antigen_formula=4:i:1,2,serotype=Typhimurium
SAMN29472493	CDC	2022-06	USA		clinical	PDS000049109.27	SRR19918648	GCA_024123825.1	PDT001351043.1	PNUSAS279926	antigen_formula=4:i:1,2,serotype=Typhimurium
SAMN29554865	CDC	2022-06	USA		clinical	PDS000054358.20	SRR20015298	GCA_024189215.1	PDT001354067.1	PNUSAS281387	antigen_formula=4:i:1,2,serotype=Typhimurium
SAMN30110362	CDC	2022-07	USA		clinical	PDS000076391.10	SRR20773590	GCA_024569865.1	PDT001377903.1	PNUSAS288206	antigen_formula=4:i:1,2,serotype=Typhimurium
SAMN30120894	CDC	2022-07	USA		clinical	PDS000051476.18	SRR20776032	GCA_024570545.1	PDT001377943.1	PNUSAS288117	antigen_formula=4:i:1,2,serotype=Typhimurium
SAMN30282664	CDC	2022-06	USA		clinical	PDS000049109.27	SRR21025949	GCA_025020505.1	PDT001386943.1	PNUSAS279148	antigen_formula=4:i:1,2,serotype=Typhimurium
SAMN30473555	CDC	2022-07	USA		clinical	PDS000054358.20	SRR21203170	GCA_024863145.1	PDT001398211.1	PNUSAS294577	antigen_formula=4:i:1,2,serotype=Typhimurium
SAMN30806977		2022-08	USA		clinical	PDS000049109.27	SRR21530791	GCA_025184945.1	PDT001414556.1	PNUSAS298977	antigen_formula=4:i:1,2,serotype=Typhimurium
SAMN30925995	CDC	2022-08	USA		clinical	PDS000049109.27	SRR21627096	GCA_025301155.1	PDT001424607.1	PNUSAS300557	antigen_formula=4:i:1,2,serotype=Typhimurium
SAMN31022944	CDC	2022-09	USA		clinical	PDS000121793.1	SRR21712787	GCA_025418035.1	PDT001434404.1	PNUSAS303535	antigen_formula=4:i:1,2,serotype=Typhimurium
SAMN31136732		2022-09	USA		clinical	PDS000049109.27	SRR21786390	GCA_025535175.1	PDT001440408.1	PNUSAS305274	antigen_formula=4:i:1,2,serotype=Typhimurium
SAMN31164279		2022-09	USA		clinical	PDS000051476.18	SRR21814042	GCA_025589815.1	PDT001443006.1	PNUSAS305912	antigen_formula=4:i:1,2,serotype=Typhimurium
SAMN31439003		2022-09	USA		clinical	PDS000121793.1	SRR22031776	GCA_025858515.1	PDT001465735.1	PNUSAS312148	antigen_formula=4:i:1,2,serotype=Typhimurium
SAMN31670458	CDC	2022-10	USA		clinical	PDS000049109.27	SRR22245970	GCA_026111315.1	PDT001482596.1	PNUSAS315606	antigen_formula=4:i:1,2,serotype=Typhimurium
SAMN31670460	CDC	2022-10	USA		clinical	PDS000049109.27	SRR22245971	GCA_026110595.1	PDT001482597.1	PNUSAS315614	antigen_formula=4:i:1,2,serotype=Typhimurium
SAMN32275466	Washington State University Washington Animal Disease Diagnostic Laboratory	1/6/22	USA:OH	feces (Dromaius novaehollandiae)	environmental/other	PDS000038355.20	SRR22766161	GCA_026962955.1	PDT001529903.1	SAL-22-VL-OH-WA-0003	antigen_formula=4:i:1,2,serotype=Typhimurium
SAMN32421168		2022-12	USA		clinical	PDS000051476.18	SRR22901383	GCA_027470025.1	PDT001549179.1	PNUSAS324236	antigen_formula=4:i:1,2,serotype=Typhimurium

SAMN32795229	CDC	2022-12	USA		clinical	PDS000049109.27	SRR23121872	GCA_028043485.1	PDT001581797.1	PNUSAS326947	antigen_formula=4:i:1,2,serotype=Typhimurium
SAMN33554138	CDC	2022	USA		clinical	PDS000054358.20	SRR23684026	GCA_028917265.1	PDT001649752.1	PNUSAS335255	antigen_formula=4:i:1,2,serotype=Typhimurium
SAMN34378121		2023	USA		clinical	PDS000049109.27	SRR24309521	GCA_030220445.1	PDT001707227.1	PNUSAS344545	antigen_formula=4:i:1,2,serotype=Typhimurium
SAMN34381829		2023	USA		clinical	PDS000154543.2	SRR24313502	GCA_032269965.1	PDT001708295.1	PNUSAS344096	antigen_formula=4:i:1,2,serotype=Typhimurium
SAMN35026792		2023	USA		clinical	PDS000051476.18	SRR24495916	GCA_033667145.1	PDT001728862.1	PNUSAS347886	antigen_formula=4:i:1,2,serotype=Typhimurium
SAMN35723225		2023	USA		clinical	PDS000149796.1	SRR24907085	GCA_032883215.1	PDT001778228.1	PNUSAS355697	antigen_formula=4:i:1,2,serotype=Typhimurium
SAMN35995608		2023	USA		clinical	PDS000049109.27	SRR25033789	GCA_032687805.1	PDT001791117.1	PNUSAS358588	antigen_formula=4:i:1,2,serotype=Typhimurium
SAMN35995619		2023	USA		clinical	PDS000150767.2	SRR25034504	GCA_032687305.1	PDT001791146.1	PNUSAS358607	antigen_formula=4:i:1,2,serotype=Typhimurium
SAMN36306403		2023	USA		clinical	PDS000154543.2	SRR25146543	GCA_032177825.1	PDT001809638.1	PNUSAS361102	antigen_formula=4:i:1,2,serotype=Typhimurium
SAMN36701919		2023	USA		clinical	PDS000051476.18	SRR25411884	GCA_032369635.1	PDT001827659.1	PNUSAS366057	antigen_formula=4:i:1,2,serotype=Typhimurium
SAMN36770391	CDC	2023	USA		clinical	PDS000154543.2	SRR25462192	GCA_032043915.1	PDT001835110.1	PNUSAS367477	antigen_formula=4:i:1,2,serotype=Typhimurium

Supplemental Table 3.8. Select metadata downloaded from NCBI for serovar Typhimurium isolates included in Figure S3.7A.

Yellow highlight indicates samples chosen for Figure S3.7B.

BioSample	Collected by	Collection date	Location	Isolation type	Isolation source	SNP cluster	Run	Assembly
SAMD00097420		1992	Japan			PDS000076400.2	DRR106839	GCA_015650885.1
SAMD00097438		2012	Italy			PDS000098671.1	DRR106884	GCA_015650845.1
SAMD00097443		1999	Japan			PDS000050000.5	DRR106894	GCA_015651185.1
SAMD00097448		2006	Japan			PDS000139929.1	DRR106906	GCA_015651245.1
SAMD00097451		1980	Japan			PDS000076402.2	DRR106912	GCA_015649685.1
SAMD00097475		2013	Japan			PDS000076396.1	DRR106973	GCA_015650325.1
SAMEA1009558						PDS000041675.17	ERR044712	GCA_010121665.1
SAMEA1009580						PDS000049928.2	ERR044703	GCA_010122525.1
SAMEA1009599						PDS000041640.3	ERR044702	GCA_010121785.1
SAMEA1009601						PDS000042445.1	ERR044713	GCA_010121745.1
SAMEA1009630						PDS000049927.1	ERR044714	GCA_010122565.1
SAMEA1009638						PDS000077449.1	ERR044730	GCA_010121805.1
SAMEA1009664						PDS000042265.3	ERR044708	GCA_010121965.1
SAMEA1009684						PDS000065265.3	ERR044707	GCA_010122035.1
SAMEA1009689						PDS000077253.1	ERR044709	GCA_010122005.1
SAMEA1009700						PDS000040809.8	ERR044706	GCA_010122125.1
SAMEA1009744						PDS000201471.8	ERR044711	GCA_010122245.1
SAMEA1009747						PDS000065255.1	ERR044704	GCA_010122235.1
SAMEA1009750						PDS000077457.3	ERR044721	GCA_010122225.1
SAMEA1009766						PDS000077252.1	ERR044732	GCA_010132915.1
SAMEA1009801						PDS000041683.1	ERR044735	GCA_010121525.1
SAMEA1009853						PDS000065275.1	ERR044700	GCA_010122085.1
SAMEA1009867						PDS000041671.1	ERR044736	GCA_010122505.1
SAMEA1009868						PDS000065150.32	ERR044718	GCA_010132935.1
SAMEA1009873						PDS000002727.19	ERR044740	GCA_010122205.1
SAMEA1009891						PDS000042451.2	ERR044720	GCA_010122325.1
SAMEA104151237	DTU	2014	Denmark			PDS000027880.2	ERR2023545	GCA_010125785.1
SAMEA104157528		2017	Ireland	clinical	human	PDS000117337.1	ERR2030254	GCA_025077515.1
SAMEA104286012		2017	Ireland	clinical	human	PDS000117331.1	ERR2114795	GCA_025077375.1
SAMEA104398339		2010	Germany		animal	PDS000074136.5	ERR2202500	GCA_010379065.1
SAMEA104398348		2013	Germany		animal	PDS000077231.2	ERR2202509	GCA_011424785.1
SAMEA104398349		2015	Germany		animal	PDS000027706.1	ERR2202510	GCA_011128535.1
SAMEA104398350		2015	Germany		animal	PDS000027735.1	ERR2202511	GCA_010458305.1
SAMEA104398352		2015	Germany		animal	PDS000027713.2	ERR2202513	GCA_011401635.1
SAMEA104398354		2015	Germany		food	PDS000026616.11	ERR2202515	GCA_010614345.1
SAMEA104398364		2016	Germany		animal	PDS000027720.1	ERR2202525	GCA_010154675.1
SAMEA104411002	APHA	2015	United Kingdom			PDS000074192.3	ERR2208744	GCA_010189705.1
SAMEA104413273	NIPH-NIH	10/21/15	Poland			PDS000091078.1	ERR2210574	GCA_018337995.1
SAMEA111504959	Quelimane Central Hospital - CISM	3/12/20	Mozambique	clinical	punctate	PDS000125065.2	ERR10438741	GCA_026328495.1
SAMEA114307261		2010	China		pig	PDS000158216.2	ERR12019122	GCA_031330115.1
SAMEA114526782	A. Smith, National Institute for Communicable Diseases	6/8/23	South Africa	clinical	Human, Other	PDS000100611.11	ERR12142651	GCA_033124025.1
SAMEA114526790	A. Smith, National Institute for Communicable Diseases	5/24/23	South Africa	clinical	Human, Blood culture	PDS000109287.4	ERR12142659	GCA_033124005.1
SAMEA14288404		2008	France	clinical	human	PDS000110445.1		GCA_940677165.1
SAMEA1483990		2011	United Kingdom: Scotland	clinical	not known	PDS000026666.20		GCA_001217585.1
SAMEA1484011		1995	United Kingdom: Scotland	clinical	not known	PDS000026653.1		GCA_001216205.1
SAMEA1484114		1997	United Kingdom: Scotland	clinical	not known	PDS000026676.1		GCA_001222005.1
SAMEA1568486		2001	Canada	clinical	not known	PDS000026659.1		GCA_001116925.1
SAMEA1568512		2001	Canada	clinical	not known	PDS000100240.3		GCA_001096565.1
SAMEA1568522		2001	Canada	clinical	not known	PDS000026663.1		GCA_001161925.1
SAMEA1568541		2001	Canada	clinical	not known	PDS000026689.14		GCA_001127885.1
SAMEA1711387						PDS000009152.4	ERR230403	GCA_010122605.1
SAMEA1711417						PDS000013660.9	ERR230398	GCA_010633465.1
SAMEA1711464						PDS000037123.1	ERR230434	GCA_010122585.1
SAMEA20258668						PDS000041635.1	ERR1891220	GCA_010211645.1
SAMEA20322418						PDS000041685.1	ERR1891524	GCA_010228015.1
SAMEA20328418						PDS000041668.4	ERR1891552	GCA_010230855.1
SAMEA20331418						PDS000041660.1	ERR1891557	GCA_010229205.1
SAMEA20332168						PDS000041641.1	ERR1891561	GCA_010227895.1
SAMEA20337418						PDS000041669.1	ERR1891577	GCA_010229565.1
SAMEA2272227						PDS000065258.1		GCA_000493535.2
SAMEA2272777		2004	Malawi:Blantyre	clinical	blood	PDS000041999.125		GCA_000027025.1
SAMEA2493785	Statens Serum Institut (SSI)	8/23/05	Denmark	clinical		PDS000013655.11	ERR493461	GCA_010121845.1
SAMEA2625959						PDS000042042.1	ERR702724	GCA_010229995.1
SAMEA2625965						PDS000042322.2	ERR702798	GCA_010231575.1
SAMEA2626038						PDS000042057.1	ERR702896	GCA_010233175.1
SAMEA2626065						PDS000097742.1	ERR702837	GCA_010232485.1
SAMEA26869168	DTU	2014	Switzerland			PDS000094232.1	ERR1759212	GCA_017288335.1
SAMEA3269008	DANMAP	1/1/11	Denmark			PDS000026635.1	ERR769345	GCA_010123085.1
SAMEA3928631						PDS000042111.1	ERR1556162	GCA_010246135.1
SAMEA4349314		2013	Denmark	clinical	human	PDS000050005.2	ERR1540273	GCA_010194195.1
SAMEA4349328		2013	Denmark	clinical	human	PDS000027599.6	ERR1540287	GCA_010193865.1
SAMEA4349363		2013	Denmark	clinical	human	PDS000042397.12	ERR1540322	GCA_010194905.1
SAMEA4349405		2013	Denmark	clinical	human	PDS000040888.6	ERR1540364	GCA_010195905.1
SAMEA4349416		2014	Denmark	clinical	human	PDS000050042.4	ERR1540375	GCA_010196985.1
SAMEA4349465		2014	Denmark	clinical	human	PDS000042254.4	ERR1540424	GCA_010198385.1
SAMEA4349577		2014	Denmark	clinical	human	PDS000050016.3	ERR1540536	GCA_010199685.1
SAMEA4349592		2014	Denmark	clinical	human	PDS000027609.15	ERR1540551	GCA_010208885.1

SAMEA4390586	DTU	2/8/13	Denmark			PDS000042356.3	ERR1592513	GCA_022473075.1
SAMEA4390587	DTU	3/26/13	Denmark			PDS000105942.1	ERR1592526	GCA_022469875.1
SAMEA4390611	DTU	4/3/13	Denmark			PDS000026822.8	ERR1592530	GCA_022471475.1
SAMEA4390615	DTU	8/14/12	Denmark			PDS000105941.1	ERR1592384	GCA_022470115.1
SAMEA4390619	DTU	8/29/12	Denmark			PDS000042268.3	ERR1592389	GCA_024835425.1
SAMEA4390635	DTU	1/30/12	Germany			PDS000105937.1	ERR1592410	GCA_022473615.1
SAMEA4390644	DTU	12/18/12	Germany			PDS000105938.1	ERR1592418	GCA_022473485.1
SAMEA4390697	DTU	10/31/13	Denmark			PDS000045973.3	ERR1592488	GCA_024826695.1
SAMEA4625254	NIPH-NIH	9/14/16	Poland			PDS000088362.1	ERR2543061	GCA_010183345.1
SAMEA4715861		2016	Sweden	clinical	human	PDS000059956.3	ERR2617858	GCA_015879415.1
SAMEA5038452						PDS000065781.1	ERR3864679	GCA_011304605.1
SAMEA5038517						PDS000056421.1	ERR3864617	GCA_011242855.1
SAMEA5039443						PDS000056263.7	ERR3843480	GCA_011223795.1
SAMEA5130710						PDS000107522.2	ERR3967698	GCA_022223645.1
SAMEA5130713						PDS000104935.1	ERR3967664	GCA_022224325.1
SAMEA5130716						PDS000104923.1	ERR3967703	GCA_022223565.1
SAMEA53008168		2016	Ireland	clinical	human	PDS000175787.2	ERR1802430	GCA_025086485.1
SAMEA5423339						PDS000065783.1	ERR4303224	GCA_013536495.1
SAMEA5423396						PDS000099889.1	ERR4303403	GCA_013540495.1
SAMEA5556597						PDS000064969.1	ERR4289448	GCA_013642515.1
SAMEA5557066						PDS000065013.1	ERR4289759	GCA_013636475.1
SAMEA5671027						PDS000042410.3	ERR4338430	GCA_013596545.1
SAMEA5671056						PDS000065622.3	ERR4338542	GCA_013595835.1
SAMEA5671060						PDS000065599.1	ERR4338603	GCA_013600545.1
SAMEA5671104						PDS000042352.4	ERR4338355	GCA_013594035.1
SAMEA5671114						PDS000042403.3	ERR4338395	GCA_013530455.1
SAMEA5671316						PDS000042316.3	ERR4338424	GCA_013595625.1
SAMEA5671330						PDS000065788.1	ERR4338487	GCA_013530195.1
SAMEA5671356						PDS000065777.2	ERR4338581	GCA_013529135.1
SAMEA5671371						PDS000065795.1	ERR4338639	GCA_013528615.1
SAMEA5671393						PDS000065620.1	ERR4338728	GCA_013599915.1
SAMEA5671396						PDS000107959.1	ERR4338737	GCA_013487385.1
SAMEA5671408						PDS000042317.2	ERR4338774	GCA_013562855.1
SAMEA5671413						PDS000065595.4	ERR4338789	GCA_013562725.1
SAMEA5671418						PDS000065770.1	ERR4338804	GCA_013486525.1
SAMEA5671430						PDS000077419.3	ERR4338837	GCA_013486165.1
SAMEA5671433						PDS000065780.1	ERR4338846	GCA_013558625.1
SAMEA5671450						PDS000065774.1	ERR4338898	GCA_013562415.1
SAMEA5671454						PDS000065608.1	ERR4338910	GCA_013558415.1
SAMEA5671468						PDS000107923.1	ERR4338948	GCA_013561535.1
SAMEA5671473						PDS000065623.1	ERR4338961	GCA_013561275.1
SAMEA5671480						PDS000107922.1	ERR4338683	GCA_013600865.1
SAMEA5882646		2018	Germany		livestock	PDS000051888.2	ERR3489631	GCA_010264655.1
SAMEA6057492		2018	Germany		environment	PDS000051919.5	ERR3580827	GCA_009634055.1
SAMEA6057531		2019	Germany		animal	PDS000051917.1	ERR3580866	GCA_009634615.1
SAMEA6057847		2016	Germany		animal	PDS000051887.1	ERR3581182	GCA_009636795.1
SAMEA6057852		2017	Germany		animal	PDS000073725.1	ERR3581187	GCA_015044595.1
SAMEA6057960		2018	Germany		animal	PDS000051906.3	ERR3581295	GCA_009637655.1
SAMEA6058385		2018	Germany		animal	PDS000179743.1	ERR3581719	GCA_009641975.1
SAMEA6058528		2019	Germany		animal	PDS000153462.1	ERR3581862	GCA_009643575.1
SAMEA6514112						PDS000042504.4	ERR3901218	GCA_011798825.1
SAMEA6514128						PDS000056938.1	ERR3901229	GCA_011778265.1
SAMEA6514139						PDS000056907.1	ERR3901248	GCA_011541295.1
SAMEA6514145						PDS000027591.4	ERR3901254	GCA_011788745.1
SAMEA6514153						PDS000042298.5	ERR3901233	GCA_011778705.1
SAMEA6514157						PDS000042481.12	ERR3901235	GCA_011798785.1
SAMEA6514181						PDS000045969.2	ERR3901265	GCA_011541315.1
SAMEA6514185						PDS000056844.1	ERR3901344	GCA_011541395.1
SAMEA6514192						PDS000027583.4	ERR3901273	GCA_011585045.1
SAMEA6514199						PDS000057621.4	ERR3901275	GCA_011585305.1
SAMEA6514201						PDS000056871.2	ERR3901353	GCA_011779145.1
SAMEA6514220						PDS000056881.2	ERR3901358	GCA_011587985.1
SAMEA6514236						PDS000192791.1	ERR3901365	GCA_011790165.1
SAMEA6514251						PDS000178867.1	ERR3901378	GCA_011587665.1
SAMEA6514261						PDS000056902.1	ERR3901382	GCA_011585605.1
SAMEA6514264						PDS000459702	ERR3901383	GCA_011778525.1
SAMEA6514269						PDS000027429.6	ERR3901387	GCA_011584925.1
SAMEA6514285						PDS000042384.2	ERR3901398	GCA_011587565.1
SAMEA6514324						PDS000065130.9	ERR3901428	GCA_011587765.1
SAMEA6514364						PDS000065612.1	ERR3901459	GCA_011779945.1
SAMEA6514403						PDS000056942.1	ERR3901483	GCA_011798805.1
SAMEA6514404						PDS000056908.1	ERR3901485	GCA_011585325.1
SAMEA6514407						PDS000056868.4	ERR3901487	GCA_011789065.1
SAMEA6514468						PDS000042395.3	ERR3901535	GCA_011779325.1
SAMEA6514477						PDS000042259.2	ERR3901541	GCA_011782025.1
SAMEA6514483						PDS000056876.1	ERR3901547	GCA_011781945.1
SAMEA6514539						PDS000056943.3	ERR3901688	GCA_011791385.1
SAMEA6514553						PDS000056888.1	ERR3901704	GCA_014338825.1
SAMEA6514560						PDS000056941.1	ERR3901711	GCA_011799555.1
SAMEA6514583						PDS000056932.1	ERR3901732	GCA_011781905.1
SAMEA6514657						PDS000042479.3	ERR3901796	GCA_011783125.1
SAMEA6514709						PDS000042415.6	ERR3901857	GCA_011783355.1
SAMEA6514761						PDS000042315.3	ERR3901906	GCA_011786085.1
SAMEA6514802						PDS000042288.2	ERR3901574	GCA_011788805.1
SAMEA6514808						PDS000056857.1	ERR3901578	GCA_011786905.1
SAMEA6514836						PDS000056890.2	ERR3901606	GCA_011783985.1
SAMEA6514869						PDS000085367.1	ERR3902021	GCA_011784375.1
SAMEA6514876						PDS000056869.1	ERR3902029	GCA_011784225.1

SAMEA6514882						PDS000056934.1	ERR3902035	GCA_011586185.1
SAMEA6514942						PDS000056873.1	ERR3902094	GCA_011785825.1
SAMEA6514981						PDS000056915.1	ERR3902422	GCA_011792525.1
SAMEA6514996						PDS000056864.1	ERR3902436	GCA_011793045.1
SAMEA6515003						PDS000076485.1	ERR3902443	GCA_011792805.1
SAMEA6515018						PDS000056930.2	ERR3902459	GCA_011809425.1
SAMEA6515042						PDS000042408.4	ERR3902483	GCA_011809075.1
SAMEA6515275						PDS000056913.9	ERR3902237	GCA_011794025.1
SAMEA6515346						PDS000056901.1	ERR3902307	GCA_011796465.1
SAMEA6515454						PDS000056858.1	ERR3902605	GCA_011797805.1
SAMEA6845850		2002	Australia	clinical	feces	PDS000064098.3	ERR4159247	GCA_027052725.1
SAMEA6845855		2009	Australia	clinical	feces	PDS000129149.1	ERR4159252	GCA_027052575.1
SAMEA7114592	R. Kingsley (Sanger)		Mali	clinical	human	PDS000073994.5	ERR4401099	GCA_016028155.1
SAMEA72294418		2016	Ireland	clinical	human	PDS000056918.3	ERR1815492	GCA_025085215.1
SAMEA7501030		5/14/18	Belgium		UNK	PDS000013661.13	ERR4775356	GCA_021448865.1
SAMEA78282418		2016	Ireland	clinical	human	PDS000117323.1	ERR1816625	GCA_025084155.1
SAMEA78288418		2016	Ireland	clinical	human	PDS000043639.7	ERR1816633	GCA_025084175.1
SAMEA78594418		2016	Ireland	clinical	human	PDS000043899.2	ERR1816866	GCA_025082695.1
SAMEA78708418		2016	Ireland	clinical	human	PDS000117339.1	ERR1817109	GCA_025082755.1
SAMEA788597						PDS000065263.1	ERR024385	GCA_010192345.1
SAMEA788604		1946	USA: Colorado		not known	PDS000076154.1	ERR023808	GCA_010191865.1
SAMEA788607		1946	Switzerland		not known	PDS000077429.1	ERR023806	GCA_010192185.1
SAMEA788614		1946	Denmark		feces	PDS000076484.1	ERR023822	GCA_010192285.1
SAMEA788630		2002	Malawi	clinical	blood	PDS000050011.2	ERR028246	GCA_012172155.1
SAMEA788682						PDS000077388.1	ERR024753	GCA_010193615.1
SAMEA788699		2009	United Kingdom	clinical	feces	PDS000049932.2	ERR024402	GCA_010193105.1
SAMEA788716						PDS000077306.1	ERR024814	GCA_010194025.1
SAMEA788767		2009	United Kingdom	clinical	feces	PDS000049935.3	ERR024409	GCA_010193525.1
SAMEA788852						PDS000096100.1	ERR024362	GCA_010192795.1
SAMEA788906						PDS000077337.1	ERR024806	GCA_010193565.1
SAMEA788911						PDS000065272.1	ERR024379	GCA_009983035.1
SAMEA788918						PDS000077410.1	ERR024763	GCA_010194945.1
SAMEA81204418		2015	Ireland	clinical	human	PDS000114665.1	ERR1823933	GCA_024393595.1
SAMEA8226912		8/17/09	Gambia	clinical	human	PDS000108615.1	ERR5490318	GCA_023082225.1
SAMEA8226977		9/27/12	Gambia	clinical	human	PDS000108609.1	ERR5490253	GCA_023083905.1
SAMEA8947667		2020	Germany	clinical	human	PDS000092850.2	ERR6149450	GCA_019260485.1
SAMEA8983376		2000	United Kingdom		not known	PDS000042248.2	ERR6318193	GCA_028197885.1
SAMEA91507918		2014	Ireland	clinical	human	PDS000043170.4	ERR1840428	GCA_010283265.2
SAMN00811527						PDS000139932.2		GCA_000335935.1
SAMN01814034				environmental/other	swine intestine (Sus domesticus)	PDS000004580.22		GCA_000474555.1
SAMN01902240	NYSDOH	2/5/04	USA:NY	environmental/other	house sparrow	PDS000027369.1	SRR949381	GCA_007907145.1
SAMN01902246	NY	2/11/05	USA	environmental/other	chicken breast	PDS000180813.32	SRR988697	GCA_014448235.1
SAMN01902249	NYSDOH	5/2/05	USA:NY	environmental/other	house sparrow	PDS000027367.3	SRR949390	GCA_010702775.1
SAMN01902257	NYSDOH	8/22/05	USA:NY	environmental/other	ground beef	PDS000027924.102	SRR949398	GCA_011454405.1
SAMN01902266	NYSDOH	2/27/06	USA:NY	environmental/other	red billed gull	PDS000013825.117	SRR949406	GCA_011455475.1
SAMN01902313	NYSDOH	7/28/08	USA:NY	environmental/other	mung bean sprout	PDS000175012.65	SRR949458	GCA_011455775.1
SAMN01902346	NYSDOH	8/21/09	USA:NY	environmental/other	chicken breast	PDS000027365.1	SRR1272775	GCA_007742255.1
SAMN01902387	NYSDOH	5/9/11	USA:NY	environmental/other	chicken breast	PDS000027362.1	SRR1177598	GCA_007748815.1
SAMN01920889	FDA Contracted Laboratory	2006	USA:WA	environmental/other	bovine necropsy	PDS000026824.7	SRR949861	GCA_011457775.1
SAMN01920894	FDA Contracted Laboratory	2007	USA:NE	environmental/other	bovine swab	PDS000004509.59	SRR949855	GCA_010632525.1
SAMN01920898	FDA Contracted Laboratory	2006	USA:AK	environmental/other	canine feces	PDS000028149.6	SRR949851	GCA_006691125.1
SAMN01920929	FDA Contracted Laboratory	2007	USA:ID	environmental/other	intestine	PDS000013650.257	SRR949511	GCA_006862605.1
SAMN01920941	FDA Contracted Laboratory	2007	USA:WA	environmental/other	rodent feces	PDS000026749.231	SRR949521	GCA_011456875.1
SAMN01920945	FDA Contracted Laboratory	2012	USA:AZ	environmental/other	environmental swab production facility	PDS000013860.55	SRR958019	GCA_006863235.1
SAMN01924592	FL	11/21/11	USA:FL	environmental/other	stool (Alligator mississippiensis)	PDS000027352.2	SRR952373	GCA_005932055.1
SAMN02182869	Washington State Department of Health| Public Health Laboratories	5/13/02	USA	environmental/other	Not Provided	PDS000013845.477	SRR945120	GCA_011457295.1
SAMN02222912	NYSDOH	1/13/09	USA:CT	environmental/other	peanut butter	PDS000013830.75	SRR1036439	GCA_011460035.1
SAMN02225273		2005	Australia			PDS000013858.28		GCA_000314915.2
SAMN02253032	FL	10/1/09	USA:FL	environmental/other	bean sprout	PDS000042386.6	SRR1015729	GCA_006840365.1
SAMN02261106	New Mexico Public Health Lab	2008	USA:NM	environmental/other	chicken breast	PDS000036013.2	SRR1021745	GCA_006813085.1
SAMN02261151	New Mexico Public Health Lab	2010	USA:NM	environmental/other	pork chop	PDS000026674.7	SRR2082849	GCA_008116675.1
SAMN02261172	New Mexico Public Health Lab	2010	USA:NM	environmental/other	pork chop	PDS000144857.34	SRR1029572	GCA_006755665.1
SAMN02265290	FDA Contracted Laboratory	12/5/09	USA:WA	environmental/other	ground beef	PDS000032664.656	SRR961984	GCA_005931315.1
SAMN02265310	FDA Contracted Laboratory	10/12/07	USA:SD	environmental/other	bovine carcass	PDS000013885.45	SRR949872	GCA_006838325.1
SAMN02265324	FDA Contracted Laboratory	10/17/07	USA:SD	environmental/other	bovine carcass	PDS000027265.1	SRR950691	GCA_005678635.1
SAMN02344553	NYSDOH	8/27/13	USA:NY	clinical	stool	PDS000026656.42	SRR974687	GCA_006862945.1
SAMN02344807	United States Food and Drug Administration	4/23/10	USA	environmental/other	hot chili powder	PDS000023721.19	SRR9052492	GCA_006003925.1
SAMN02344929	United States Food and Drug Administration	8/23/10	USA	environmental/other	rubbed sage	PDS000013739.143	SRR1177622	GCA_010960115.1
SAMN02344931	United States Food and Drug Administration	8/25/10	USA	environmental/other	frozen amberjack fish portion	PDS000029638.98	SRR1177271	GCA_005934185.1
SAMN02345025	FMA	11/27/10	Portugal	environmental/other	coconut, shelled	PDS000013900.3	SRR1182724	GCA_003868895.1
SAMN02345102	United States Food and Drug Administration| Office of Regulatory Affairs| San Francisco Human and Animal Food Laboratory	2/12/11	USA	environmental/other	spice mixture indian cottage cheese buttered paneer makhanwala	PDS000013838.134	SRR1107478	GCA_011546085.1
SAMN02345147	United States Food and Drug Administration| Office of Regulatory Affairs| San Francisco Human and Animal Food Laboratory	3/29/11	USA	environmental/other	cashew mixture	PDS000013887.53	SRR1107438	GCA_010907305.1
SAMN02345308	United States Food and Drug Administration| Office of Regulatory Affairs| Irvine Human and Animal Food Laboratory	9/6/11	USA	environmental/other	frozen largemouth bass fish	PDS000026701.200	SRR1033509	GCA_010949235.1
SAMN02345593	United States Food and Drug Administration| Forensic Chemistry Center	6/17/12	Not Provided	environmental/other	macadamia nut	PDS000031835.3	SRR1068366	GCA_011637115.1
SAMN02367644	FDA		Mexico	clinical	human	PDS000027127.3	SRR1060693	GCA_010123015.1
SAMN02367645	FDA			environmental/other		PDS000013867.69	SRR1060692	GCA_010726345.1
SAMN02367647	FDA	1986	USA:IN	environmental/other	Rabbit	PDS000027317.4	SRR1060691	GCA_010506855.1
SAMN02367648	FDA		Mongolia	clinical		PDS000031186.4	SRR1060690	GCA_010663185.1
SAMN02367649	FDA		Norway	clinical		PDS000029340.6	SRR1060689	GCA_010489995.1
SAMN02367650	FDA		Finland	environmental/other		PDS000031185.5	SRR1060688	GCA_010632375.1
SAMN02367651	FDA	1987	USA:CA	environmental/other	Parrot	PDS000027316.6	SRR1060687	GCA_010490975.1
SAMN02367652	FDA	1987	USA:CA	environmental/other	Opposum	PDS000029339.5	SRR1060686	GCA_010717005.1
SAMN02367655	FDA		France	clinical		PDS000002607.16	SRR1060683	GCA_011443255.1
SAMN02367656	FDA		Panama	clinical		PDS000031184.5	SRR1122731	GCA_010714835.1
SAMN02367657	FDA		USA:NC	clinical	human	PDS000031183.37	SRR1060682	GCA_010388365.1
SAMN02367660	FDA		Mexico	clinical	human	PDS000027125.5	SRR1060681	GCA_010123305.1
SAMN02367661	FDA		France	environmental/other		PDS000027124.3	SRR1060680	GCA_010122825.1
SAMN02367662	FDA		USA:OR	environmental/other	Heron	PDS000027123.7	SRR1060679	GCA_010123175.1

SAMN02367912	FDA: Shaohua Zhao	2010	USA:CT	environmental/other	chicken breast	PDS000030628.1	SRR1840608	GCA_011103075.1
SAMN02368015	FDA: Shaohua Zhao	2011	USA:NY	environmental/other	chicken breast	PDS000032378.1	SRR1122596	GCA_010123745.1
SAMN02368163	FDA	1988	Senegal	clinical		PDS000005713.5	SRR1060516	GCA_010123225.1
SAMN02368368	NCTR-FDA	2000	USA	clinical		PDS000026825.464	SRR1122508	GCA_010714915.1
SAMN02368373	NCTR-FDA	1999	USA	environmental/other	Veterinary Diagnostic	PDS000042369.133	SRR1122503	GCA_010637105.1
SAMN02368708	Technical University Of Denmark			clinical		PDS000040405.7	SRR1106385	GCA_011108435.1
SAMN02378010	Minnesota Department Of Health	6/6/07	USA:MN	environmental/other	feces	PDS000013868.575	SRR1177569	GCA_010868365.1
SAMN02378013	Minnesota Department Of Health	6/26/07	USA:MN	environmental/other	feces	PDS000013878.168	SRR1177549	GCA_009996925.1
SAMN02378047	Minnesota Department Of Health	4/24/08	USA:MN	environmental/other	feces	PDS000032661.317	SRR1166604	GCA_011085505.1
SAMN02378117	Minnesota Department Of Health	6/11/10	USA:MN	environmental/other	tissue	PDS000038564.8	SRR1158059	GCA_006756005.1
SAMN02378118	Minnesota Department Of Health	7/15/10	USA:MN	environmental/other	feces	PDS000027817.12	SRR1158084	GCA_011638235.1
SAMN02378119	Minnesota Department Of Health	7/16/10	USA:MN	environmental/other	tissue	PDS000032076.9	SRR1158026	GCA_011485085.1
SAMN02378120	Minnesota Department Of Health	8/26/10	USA:MN	environmental/other	feces	PDS000032334.1	SRR1158202	GCA_011084105.1
SAMN02378122	Minnesota Department Of Health	8/17/10	USA:MN	environmental/other		PDS000113872.9	SRR1158206	GCA_006690745.1
SAMN02378123	Minnesota Department Of Health	8/31/10	USA:MN	environmental/other	tissue	PDS000032374.2	SRR1157597	GCA_006691785.1
SAMN02378125	Minnesota Department Of Health	9/8/10	USA:MN	environmental/other	liver	PDS000032373.2	SRR1090983	GCA_006691465.1
SAMN02378126	Minnesota Department Of Health	9/17/10	USA:MN	environmental/other	feces	PDS000065117.13	SRR1089590	GCA_011484805.1
SAMN02378143	Minnesota Department Of Health	4/12/11	USA:MN	environmental/other	lung	PDS000032654.261	SRR1089565	GCA_010868865.1
SAMN02378154	Minnesota Department Of Health	11/17/11	USA:MN	environmental/other	feces	PDS000029035.8	SRR1067639	GCA_009997745.1
SAMN02378155	Minnesota Department Of Health	6/30/11	USA:MN	environmental/other		PDS000144856.54	SRR1067620	GCA_010886335.1
SAMN02378169	Minnesota Department Of Health	3/21/12	USA:MN	environmental/other	feces	PDS000028842.3	SRR1166648	GCA_011084805.1
SAMN02378182	Minnesota Department Of Health	10/17/12	USA:MN	environmental/other	chicken kiev	PDS000026617.605	SRR1048617	GCA_010941895.1
SAMN02378194	Minnesota Department Of Health	2/11/13	USA:MN	environmental/other	chicken breast	PDS000180814.9	SRR1030355	GCA_011089645.1
SAMN02378199	Minnesota Department Of Health	4/29/13	USA:MN	environmental/other	raw milk	PDS000026974.4	SRR1029570	GCA_011089285.1
SAMN02403222	VA	7/1/09	USA:VA	environmental/other	Not Provided	PDS000032655.382	SRR1047602	GCA_010705445.1
SAMN02403226	VA	5/30/12	USA:VA	environmental/other	Not Provided	PDS000081815.110	SRR3474702	GCA_004185455.1
SAMN02403227	VA	7/24/13	USA:VA	environmental/other	Not Provided	PDS000026746.181	SRR3474703	GCA_006822585.1
SAMN02403293	VA	1/24/06	USA:VA	environmental/other	Not Provided	PDS000032362.2	SRR1177188	GCA_004224185.1
SAMN02403301	VA	12/29/05	USA:VA	environmental/other	Not Provided	PDS000032361.1	SRR1177544	GCA_007548275.1
SAMN02403303	VA	12/22/05	USA:VA	environmental/other	Not Provided	PDS000026762.119	SRR1177591	GCA_011083945.1
SAMN02403304	VA	7/25/06	USA:VA	environmental/other	Not Provided	PDS000111171.3	SRR1177577	GCA_010946135.1
SAMN02403317	VA	1/19/06	USA:VA	environmental/other	Not Provided	PDS000032357.2	SRR1177571	GCA_006690525.1
SAMN02403320	VA	1/11/06	USA:VA	environmental/other	Not Provided	PDS000026810.182	SRR1177194	GCA_010926375.1
SAMN02403321	VA	1/23/06	USA:VA	environmental/other	Not Provided	PDS000042368.5	SRR1177172	GCA_006862325.1
SAMN02403334	VA	2/28/06	USA:VA	environmental/other	Not Provided	PDS000026745.32	SRR1181523	GCA_010424205.1
SAMN02403343	VA	3/18/06	USA:VA	environmental/other	Not Provided	PDS000060956.1	SRR1177641	GCA_011083085.1
SAMN02403392	VA	2/26/07	USA:VA	environmental/other	Not Provided	PDS000013882.327	SRR1238777	GCA_004227565.1
SAMN02403412	VA	9/30/10	USA:VA	environmental/other	Not Provided	PDS000029655.22	SRR1258614	GCA_011446495.1
SAMN02403418	VA	4/2/12	USA:VA	environmental/other	tomato	PDS000013883.13	SRR1272528	GCA_011080665.1
SAMN02403427	VA	6/1/07	USA:VA	environmental/other	hamburger	PDS000029982.2	SRR1299353	GCA_006689945.1
SAMN02419062	FL	7/27/11	USA:GA	environmental/other	Trachemys scripta from irrigation pond	PDS000032344.1	SRR1067663	GCA_007817195.1
SAMN02419096	FL	2011-05	USA:GA	environmental/other	sediment pond	PDS000032336.1	SRR1105692	GCA_007469495.1
SAMN02483572	United States Food and Drug Administration| Office of Regulatory Affairs| San Francisco Human and Animal Food Laboratory	1/30/12	USA	environmental/other	hot smoked milkfish	PDS000039817.8	SRR1157599	GCA_011084825.1
SAMN02595031	Arizona Department of Health	7/18/13	USA:VA	environmental/other	boneless skinless chicken breast	PDS000032335.1	SRR1195648	GCA_007472575.1
SAMN02595811	Minnesota Department Of Health	10/30/07	USA:MN	environmental/other	cow stool	PDS000056091.9	SRR1177330	GCA_010424465.1
SAMN02595832	Minnesota Department Of Health	4/22/08	USA:MN	environmental/other	pig liver	PDS000054612.3	SRR1266087	GCA_010456805.1
SAMN02595835	Minnesota Department Of Health	5/9/08	USA:MN	environmental/other	pig liver	PDS000029630.1	SRR1266040	GCA_010456905.1
SAMN02595843	Minnesota Department Of Health	11/20/08	USA:MN	environmental/other	cow tissue composite	PDS000041663.5	SRR1266090	GCA_007834975.1
SAMN02595861	Minnesota Department Of Health	8/24/09	USA:MN	environmental/other	pig	PDS000015886.15	SRR1266027	GCA_010655725.1
SAMN02595872	Minnesota Department Of Health	4/6/10	USA:MN	environmental/other	bird liver	PDS000037478.20	SRR1266058	GCA_011414115.1
SAMN02595882	Minnesota Department Of Health	11/15/10	USA:MN	environmental/other	intestine raccoon	PDS000032333.1	SRR1266032	GCA_007547895.1
SAMN02595890	Minnesota Department Of Health	4/29/11	USA:MN	environmental/other	dog stool	PDS000026818.68	SRR1292271	GCA_010389445.1
SAMN02595904	Minnesota Department Of Health	8/3/12	USA:MN	environmental/other	cat liver	PDS000026800.437	SRR1346298	GCA_011417435.1
SAMN02602986						PDS000013874.388		GCA_000213635.1
SAMN02602988						PDS000013899.30		GCA_000188735.1
SAMN02640793	United States Food and Drug Administration	2002	USA	environmental/other	ground beef	PDS000101019.1	SRR1185790	GCA_006691245.1
SAMN02640815	United States Food and Drug Administration	2002	USA	environmental/other	chicken breast	PDS000026832.35	SRR1185842	GCA_006830725.1
SAMN02640878	United States Food and Drug Administration	2002	USA	environmental/other	ground beef	PDS000031535.11	SRR1203017	GCA_006690785.1
SAMN02640882	United States Food and Drug Administration	2002	USA	environmental/other	chicken breast	PDS000050108.12	SRR1202988	GCA_007748355.1
SAMN02640897	United States Food and Drug Administration	2002	USA	environmental/other	chicken breast	PDS000028787.49	SRR1220766	GCA_006690835.1
SAMN02640928	United States Food and Drug Administration	2003	USA	environmental/other	ground turkey	PDS000037294.51	SRR1257274	GCA_006822105.1
SAMN02645813	Pennsylvania Department of Health	1/25/10	USA	environmental/other	chicken breast	PDS000026727.1	SRR1548413	GCA_003867855.1
SAMN02646788	Minnesota Department Of Health	8/24/03	USA:MN	environmental/other	feces	PDS000032299.5	SRR1299391	GCA_007906785.1
SAMN02646793	Minnesota Department Of Health	8/13/03	USA:MN	environmental/other	tissue	PDS000032196.3	SRR1299313	GCA_011446855.1
SAMN02646799	Minnesota Department Of Health	8/27/03	USA:MN	environmental/other	tissue	PDS000026978.189	SRR1299298	GCA_011081725.1
SAMN02646843	Minnesota Department Of Health	10/2/03	USA:AR	environmental/other	tissue	PDS000060958.2	SRR1272873	GCA_011079805.1
SAMN02646948	Minnesota Department Of Health	4/10/07	USA:MN	environmental/other	spleen	PDS000078507.6	SRR1233935	GCA_011445675.1
SAMN02646952	Minnesota Department Of Health	7/5/07	USA:MN	environmental/other	puffed vegetable snack	PDS000032293.3	SRR1198924	GCA_006832025.1
SAMN02664748	University of Florida	2011-08	USA:FL	environmental/other	surface water	PDS000046671.2	SRR1207482	GCA_011406715.1
SAMN02664824	Food Safety Laboratory, Cornell University	12/12/07	USA:NY	environmental/other	feces (Bos taurus)	PDS000076645.2	SRR1238752	GCA_010424145.1
SAMN02664832	Food Safety Laboratory, Cornell University	1/18/08	USA:NY	environmental/other	feces (Bos taurus)	PDS000013886.14	SRR1213770	GCA_011447555.1
SAMN02678465	United States Food and Drug Administration	8/13/13	USA	environmental/other	cantaloupe	PDS000027050.110	SRR1269327	GCA_011080645.1
SAMN02678618	United States Food and Drug Administration	10/2/08	USA	environmental/other	tomatillo fresh	PDS000032264.2	SRR1613897	GCA_007816915.1
SAMN02678758	United States Food and Drug Administration	9/16/08	USA	environmental/other	field irrigation system	PDS000078032.25	SRR1509584	GCA_011448095.1
SAMN02678793	United States Food and Drug Administration|Office of Regulatory Affairs|Pacific Northwest Laboratory	10/6/09	USA	environmental/other	crab in shell	PDS000036001.27	SRR3156729	GCA_007762275.1
SAMN02678865	United States Food and Drug Administration	7/10/13	USA	environmental/other	frozen ostrich	PDS000031764.13	SRR1264983	GCA_005932495.1
SAMN02678902	United States Food and Drug Administration	4/21/11	USA	environmental/other	raw shrimp	PDS000028689.2	SRR1272529	GCA_011080765.1
SAMN02698258	United States Food and Drug Administration	10/27/11	USA	environmental/other	lentil vegetable seasoning curry madras	PDS000032220.2	SRR1411118	GCA_008090095.1
SAMN02698395	United States Food and Drug Administration	2/10/11	USA	environmental/other	frozen grouper fillet fish	PDS000004540.14	SRR1427103	GCA_016377225.1
SAMN02698411	United States Food and Drug Administration	4/12/11	USA	environmental/other	fresh coriander	PDS000026817.30	SRR1427716	GCA_011449395.1
SAMN02699343	FDA	2012	USA: CA	environmental/other	chicken breast	PDS000032791.6		GCA_001480485.1
SAMN02699357	FDA	2012	USA: NM	environmental/other	pork chop	PDS000026887.2		GCA_001481175.1
SAMN02699437	Minnesota Department of Health	5/25/01		environmental/other	feces	PDS000013890.5	SRR1333878	GCA_016377265.1
SAMN02699450	Minnesota Department of Health	1/28/02	USA:MN	environmental/other	lung	PDS000079933.2	SRR1346284	GCA_008089745.1
SAMN02699453	Minnesota Department of Health	4/29/02	USA:MN	environmental/other	kidney	PDS000032200.1	SRR1346262	GCA_010498335.1
SAMN02699463	Minnesota Department of Health	9/16/02	USA:MN	environmental/other	feces	PDS000032207.1	SRR1385147	GCA_004218485.1
SAMN02699501	Minnesota Department of Health	11/1/02	USA:MN	environmental/other	tissue	PDS000072789.45	SRR1461807	GCA_011448735.1
SAMN02699528	Minnesota Department of Health	12/12/02	USA:MN	environmental/other	lung	PDS000071837.31	SRR1511537	GCA_011450035.1

SAMN02699529	Minnesota Department of Health	12/6/02	USA:MN	environmental/other	tissue	PDS000032182.3	SRR1511508	GCA_008091565.1
SAMN02699574	Minnesota Department of Health	2/21/03	USA:MN	environmental/other	lung	PDS000085468.3	SRR1576580	GCA_008067975.1
SAMN02699667	Minnesota Department of Health	7/8/03	USA:NC	environmental/other	tissue	PDS000032187.1	SRR1574252	GCA_008118305.1
SAMN02699670	Minnesota Department of Health	7/11/03	USA:AR	environmental/other	lung	PDS000060974.1	SRR1574292	GCA_008088105.1
SAMN02699754	Minnesota Department of Health	5/3/04	USA:MN	environmental/other	feces	PDS000053408.29	SRR1649547	GCA_008023655.1
SAMN02699805	Minnesota Department of Health	3/16/05	USA:MN	environmental/other	feces	PDS000051475.1	SRR1752731	GCA_007742675.1
SAMN02699908	Minnesota Department of Health	1/28/10	USA:MN	environmental/other	tissue	PDS000049034.5	SRR3057274	GCA_008669455.1
SAMN02699912	Minnesota Department of Health	9/17/10	USA:MN	environmental/other	feces	PDS000027862.5	SRR3306272	GCA_008808815.1
SAMN02712198		2007-05	USA: United States BIFSCo Region 1			PDS000026739.38		GCA_000941015.2
SAMN02742061	FDA Contracted Laboratory	11/8/06	USA:OR	environmental/other	bovine colostrum	PDS000002633.9	SRR1292266	GCA_010906285.1
SAMN02742117	FDA Contracted Laboratory	1/9/07	USA:WA	environmental/other	environmental bovine	PDS000032155.2	SRR1300679	GCA_006630425.1
SAMN02742123	FDA Contracted Laboratory	1/9/07	USA:WA	environmental/other	equine environment	PDS000032154.4	SRR1302862	GCA_006690125.1
SAMN02777702	FDA	2003	USA:MD	environmental/other	chicken breast	PDS000032147.9	SRR1582184	GCA_006629885.1
SAMN02843556	United States Food and Drug Administration| Office of Regulatory Affairs| New York Human and Animal Food Laboratory	11/7/01	USA	environmental/other	squid	PDS000002635.9	SRR1639646	GCA_008032215.1
SAMN02843557	United States Food and Drug Administration| Office of Regulatory Affairs| Irvine Human and Animal Food Laboratory	11/8/01	USA	environmental/other	salted egg yolk	PDS000042198.18	SRR3885123	GCA_008913985.1
SAMN02843783	United States Food and Drug Administration|Office of Regulatory Affairs|Pacific Northwest Laboratory	3/5/03	USA	environmental/other	dog chew	PDS000023327.4	SRR2096599	GCA_006694585.1
SAMN02843985	United States Food and Drug Administration| Office of Regulatory Affairs| Irvine Human and Animal Food Laboratory	2/24/04	USA	environmental/other	kasubha safflower	PDS000192852.1	SRR2133189	GCA_008997815.1
SAMN02843993	United States Food and Drug Administration| Office of Regulatory Affairs| New York Human and Animal Food Laboratory	3/2/04	USA	environmental/other	pig ear	PDS000032068.16	SRR1744009	GCA_007817315.1
SAMN02844025	United States Food and Drug Administration| Office of Regulatory Affairs| Irvine Human and Animal Food Laboratory	4/21/04	USA	environmental/other	salted duck egg	PDS000042321.2	SRR2133344	GCA_008997885.1
SAMN02844088	United States Food and Drug Administration|Office of Regulatory Affairs|Pacific Northwest Laboratory	7/30/04	USA	environmental/other	chutney powder	PDS000032062.1	SRR5155705	GCA_006953605.1
SAMN02844355	United States Food and Drug Administration| Office of Regulatory Affairs| New York Human and Animal Food Laboratory	4/3/06	USA	environmental/other	mackerel fish	PDS000027910.23	SRR1778012	GCA_008005085.1
SAMN02844501	United States Food and Drug Administration| Office of Regulatory Affairs| New York Human and Animal Food Laboratory	6/11/07	USA	environmental/other	oyster	PDS000032040.3	SRR1946914	GCA_010876165.1
SAMN02844697	United States Food and Drug Administration|Office of Regulatory Affairs|Southeast Food and Feed Laboratory	3/10/09	USA	environmental/other	red oak leaf lettuce	PDS000046000.13	SRR3219070	GCA_007761495.1
SAMN02845327	United States Food and Drug Administration| Office of Regulatory Affairs| Arkansas Human and Animal Food Laboratory	8/20/03	USA	environmental/other	red snapper	PDS000031984.1	SRR2534080	GCA_003877935.1
SAMN02845594	United States Food and Drug Administration| Office of Regulatory Affairs| Irvine Human and Animal Food Laboratory	1/7/05	USA	environmental/other	coriander powder	PDS000036080.3	SRR2102426	GCA_006634565.1
SAMN02845857	United States Food and Drug Administration|Office of Regulatory Affairs|Southeast Food and Feed Laboratory	11/13/06	USA	environmental/other	ground tomato from field	PDS000042474.3	SRR2002711	GCA_010914375.1
SAMN02845893	United States Food and Drug Administration| Office of Regulatory Affairs| Arkansas Human and Animal Food Laboratory	2/21/07	USA	environmental/other	extra hot chili powder	PDS000030026.6	SRR2014672	GCA_006018415.1
SAMN02846067	United States Food and Drug Administration| Office of Regulatory Affairs| New York Human and Animal Food Laboratory	2/7/08	USA	environmental/other	garam masala spice mixture	PDS000055038.2	SRR5883567	GCA_009402185.1
SAMN02846093	United States Food and Drug Administration|Office of Regulatory Affairs|Southeast Food and Feed Laboratory	7/20/08	USA	environmental/other	feces animal	PDS000078046.5	SRR3092104	GCA_008677375.1
SAMN02846135	United States Food and Drug Administration|Office of Regulatory Affairs|Southeast Food and Feed Laboratory	9/15/09	USA	environmental/other	water	PDS000053291.39	SRR3654321	GCA_011409195.1
SAMN02846769	United States Food and Drug Administration| Office of Regulatory Affairs| Irvine Human and Animal Food Laboratory	2/17/10	USA	environmental/other	environmental swab	PDS000048257.21	SRR2125026	GCA_008117745.1
SAMN02846949	United States Food and Drug Administration| Office of Regulatory Affairs| Irvine Human and Animal Food Laboratory	7/15/10	USA	environmental/other	frozen crooked duck egg yolk	PDS000047482.1	SRR1646545	GCA_008023485.1
SAMN02846950	United States Food and Drug Administration| Office of Regulatory Affairs| Irvine Human and Animal Food Laboratory	7/15/10	USA	environmental/other	frozen crooked duck egg yolk	PDS000101103.34	SRR1646546	GCA_008023615.1
SAMN02847167	United States Food and Drug Administration| Office of Regulatory Affairs| Irvine Human and Animal Food Laboratory	12/8/10	USA	environmental/other	golden corvina fillet	PDS000030030.89	SRR1693289	GCA_010661765.1
SAMN02847752	United States Food and Drug Administration|Office of Regulatory Affairs|Southeast Food and Feed Laboratory	3/7/14	USA	environmental/other	chili flakes	PDS000037726.1	SRR6805497	GCA_005997325.1
SAMN02849754	FDA Contracted Laboratory	1/9/07	USA:WA	environmental/other	bovine feed	PDS000031708.3	SRR1501486	GCA_006629965.1
SAMN02849788	FDA Contracted Laboratory	1/9/07	USA:WA	environmental/other	feces equine	PDS000031704.1	SRR1501658	GCA_011451135.1
SAMN02849946	FDA Contracted Laboratory	2/2/07	USA:WA	environmental/other	avian carcass rinse water	PDS000013824.215	SRR1515026	GCA_011453675.1
SAMN02894087	FDA	2005	USA:MN	environmental/other	chicken breast	PDS000031698.10	SRR1534908	GCA_007546935.1
SAMN02894174	FDA	2005	USA:CT	environmental/other	chicken breast	PDS000031690.10	SRR1528508	GCA_006688885.1
SAMN02894178	FDA	2005	USA:GA	environmental/other	chicken breast	PDS000028568.60	SRR1528483	GCA_006629825.1
SAMN02894179	FDA	2005	USA:GA	environmental/other	ground turkey	PDS000054041.1	SRR1528526	GCA_007906755.1
SAMN02900050	Not Provided	6/13/13	USA	environmental/other	water stream	PDS000030145.1	SRR1566330	GCA_006853955.1
SAMN02900080	Not Provided	9/13/13	USA	environmental/other	sediment stream	PDS000031685.2	SRR1567297	GCA_008119625.1
SAMN02902665	FDA Contracted Laboratory	1/9/07	USA:WA	environmental/other	llama	PDS000031654.2	SRR1528533	GCA_006845325.1
SAMN02902721	FDA Contracted Laboratory	1/9/07	USA:WA	environmental/other	tortoise feces	PDS000076499.1	SRR1537514	GCA_011074565.1
SAMN02902756	FDA Contracted Laboratory	1/9/07	USA:WA	environmental/other	equine culture medium	PDS000062489.10	SRR1544175	GCA_016331065.1
SAMN02902789	FDA Contracted Laboratory	1/9/07	USA:WA	environmental/other	tortoise necropsy abdominal fluid liver	PDS000055091.3	SRR1576564	GCA_010424025.1
SAMN02910163	FDA Contracted Laboratory	2/2/07	USA:ID	environmental/other	feces bovine	PDS000042126.1	SRR1534843	GCA_007834895.1
SAMN02911914	CDC	2012	USA	clinical	clinical sample	PDS000026657.7		GCA_001473345.1
SAMN02911926	CDC	2012	USA	clinical	clinical sample	PDS000026827.50		GCA_001473385.1
SAMN02911962	CDC	2011	USA	clinical	clinical sample	PDS000038312.3		GCA_001473635.1
SAMN02911971	CDC	2011	USA	clinical	clinical sample	PDS000013853.325		GCA_001475245.1
SAMN02911984	CDC	2012	USA	clinical	clinical sample	PDS000013712.42		GCA_001475335.1
SAMN02918702	United States Food and Drug Administration| Office of Regulatory Affairs| Arkansas Human and Animal Food Laboratory	7/8/10	USA	environmental/other	frozen animal feed baby mouse	PDS000200046.2	SRR1735343	GCA_010438435.1
SAMN02927354	CDC	2012-07	USA	clinical		PDS000027051.280	SRR1577657	GCA_006850405.1
SAMN02952708	FDA Contracted Laboratory	1/9/07	USA:WA	environmental/other	feces avian	PDS000031605.1	SRR1554517	GCA_005524235.1
SAMN02952817	FDA Contracted Laboratory	1/9/07	USA:WA	environmental/other	porcine necropsy intestine	PDS000013879.106	SRR1564495	GCA_006847705.1
SAMN02989090	Minnesota Department of Health	4/14/13	USA:MN	clinical	feces	PDS000032657.39	SRR1569728	GCA_008067215.1
SAMN03002011	FDA Contracted Laboratory	1/9/07	USA:WA	environmental/other	feces equine	PDS000052609.2	SRR1569681	GCA_006010785.1
SAMN03002026	FDA Contracted Laboratory	1/9/07	USA:WA	environmental/other	Not Provided	PDS000031586.1	SRR1569632	GCA_005935125.1
SAMN03024018	Western Carolina University (WCU)	9/13/13	USA:NC	environmental/other	bird feces	PDS000031575.1	SRR1773589	GCA_005756135.1
SAMN03069683	Not Provided	2014-07	USA	environmental/other	water gallon	PDS000166581.3	SRR1576129	GCA_006010185.1
SAMN03097225	FERA	2006/2008	United Kingdom	environmental/other	retail meat	PDS000026629.23	SRR1604875	GCA_007177635.1
SAMN03097226	FERA	2006/2008	United Kingdom	environmental/other	retail meat	PDS000206393.4	SRR1604876	GCA_010411025.1
SAMN03097227	FERA	2006/2008	United Kingdom	environmental/other	retail meat	PDS000042461.99	SRR1604877	GCA_008009465.1
SAMN03112887	VA	6/20/12	USA:VA	environmental/other	Not Provided	PDS000074567.36	SRR1660417	GCA_010697625.1
SAMN03120578	Hoi-Shan Kwan's Lab	2007	Hong Kong	clinical	blood	PDS000026807.1		GCA_001295425.1
SAMN03121892	Hoi-Shan Kwan's Lab	2005	Hong Kong	clinical	stool	PDS000004586.4		GCA_001295665.1
SAMN03121897	Hoi-Shan Kwan's Lab	2006	Hong Kong	clinical	stool	PDS000004586.4		GCA_001295595.1
SAMN03121900	Hoi-Shan Kwan's Lab	2006	Hong Kong	clinical	stool	PDS000004607.3		GCA_001295635.1
SAMN03121904	Hoi-Shan Kwan's Lab	2006	Hong Kong	clinical	stool	PDS000004607.3		GCA_001295495.1
SAMN03135169	PHE	10/17/12	United Kingdom: South of England	clinical	human	PDS000002642.19	SRR1618673	GCA_006387065.1
SAMN03144979	the NSW Ministry of Health and the NSW Enteric Reference Laboratory	11/9/06	Australia: Sydney	clinical	faecal sample	PDS000031528.45	SRR1645473	GCA_010796815.1
SAMN03152378	PHE	2012-10	United Kingdom: North of England	clinical	human	PDS000002654.21	SRR1635077	GCA_006387395.1
SAMN03152397	PHE	2012-02	United Kingdom: North of England	clinical	human	PDS000042336.2	SRR1635099	GCA_006387975.1
SAMN03152398	PHE	2012-11	United Kingdom: Midlands and East of England	clinical	human	PDS000026708.89	SRR1635098	GCA_006387835.1
SAMN03152400	PHE	2012-12	United Kingdom: London	clinical	human	PDS000026812.319	SRR1635102	GCA_006387875.1
SAMN03152422	PHE	2012-10	United Kingdom: South of England	clinical	human	PDS000042450.95	SRR1635124	GCA_006388515.1
SAMN03152426	PHE	2012-01	United Kingdom: South of England	clinical	human	PDS000042247.14	SRR1635121	GCA_006388575.1
SAMN03152428	PHE	2012-04	United Kingdom: Midlands and East of England	clinical	human	PDS000030204.56	SRR1635127	GCA_006386635.1
SAMN03160320	PHE	2013-07	United Kingdom: Midlands and East of England	clinical	human	PDS000031472.1	SRR1638967	GCA_008010815.1
SAMN03160768	VA	5/30/12	USA:VA	environmental/other	Not Provided	PDS000026658.115	SRR1694063	GCA_008025735.1
SAMN03168520	PHE	2012-09	United Kingdom: South of England	clinical	human	PDS000030104.7	SRR1645148	GCA_008031115.1
SAMN03168554	PHE	2012-10	United Kingdom: London	clinical	human	PDS000032663.161	SRR1645174	GCA_008032065.1
SAMN03168564	PHE	2012-04	United Kingdom: North of England	clinical	human	PDS000042419.6	SRR1645184	GCA_007743875.1
SAMN03168693	PHE	2012-06	United Kingdom: South of England	clinical	human	PDS000026729.64	SRR1645265	GCA_011595525.1
SAMN03168721	PHE	2012-08	United Kingdom: North of England	clinical	human	PDS000013822.12	SRR1645332	GCA_011550655.1

SAMN03168731	PHE	2012-07		clinical	human	PDS000020392.12	SRR1645347	GCA_011078645.1
SAMN03168750	PHE	2012-04	United Kingdom: North of England	clinical	human	PDS000031487.3	SRR1645367	GCA_004280775.1
SAMN03168771	PHE	2012-11	United Kingdom: London	clinical	human	PDS000030226.6	SRR1645387	GCA_011624845.1
SAMN03168788	PHE	2012-01	United Kingdom: South of England	clinical	human	PDS000004621.51	SRR1645430	GCA_011641475.1
SAMN03168804	PHE	2012-07	United Kingdom: South of England	clinical	human	PDS000013823.51	SRR1645446	GCA_010878365.1
SAMN03168853	PHE	2012-07	United Kingdom: South of England	clinical	human	PDS000026747.304	SRR1645553	GCA_006629685.1
SAMN03168856	PHE	2012-06	United Kingdom: South of England	clinical	human	PDS000027000.3	SRR1645555	GCA_011624905.1
SAMN03168892	PHE	2012-12	United Kingdom: Midlands and East of England	clinical	human	PDS000028712.6	SRR1645583	GCA_008043455.1
SAMN03168903	PHE	2012-08	United Kingdom: London	clinical	human	PDS000042439.11	SRR1645601	GCA_008043635.1
SAMN03168956	PHE	2012-01		clinical	human	PDS000056861.1	SRR1645606	GCA_008043675.1
SAMN03168959	PHE	2012-10	United Kingdom: North of England	clinical	human	PDS000056878.5	SRR1645611	GCA_008042975.1
SAMN03168960	PHE	2012-09	United Kingdom: South of England	clinical	human	PDS000206414.8	SRR1645608	GCA_008035085.1
SAMN03168981	PHE	2012-04	United Kingdom: South of England	clinical	human	PDS000031480.6	SRR1645717	GCA_006627165.1
SAMN03169013	PHE	2012-04	United Kingdom: London	clinical	human	PDS000042323.4	SRR1645748	GCA_008043835.1
SAMN03169028	PHE	2012-08	United Kingdom: South of England	environmental/other	animal	PDS000013764.14	SRR1645761	GCA_008043915.1
SAMN03169052	PHE	2012-08	United Kingdom: North of England	clinical	human	PDS000059907.10	SRR1645787	GCA_011553195.1
SAMN03169074	PHE	2012-03	United Kingdom: North of England	clinical	human	PDS000013762.90	SRR1645810	GCA_008042855.1
SAMN03169122	PHE	2012-09	United Kingdom: South of England	clinical	human	PDS000002738.143	SRR1645855	GCA_008006325.1
SAMN03169164	PHE	2012-04	United Kingdom: Midlands and East of England	clinical	human	PDS000314755.6	SRR1645876	GCA_007742795.1
SAMN03169190	PHE	2012-08	United Kingdom: North of England	clinical	human	PDS000080434.1	SRR1645902	GCA_008002145.1
SAMN03169216	PHE	2012-12	United Kingdom: Midlands and East of England	clinical	human	PDS000040805.2	SRR1645930	GCA_008018355.1
SAMN03169244	PHE	2012-01	United Kingdom: North of England	clinical	human	PDS000049449.12	SRR1645942	GCA_008018675.1
SAMN03169274	PHE	2012-03	United Kingdom: North of England	clinical	human	PDS000047449.1	SRR1645963	GCA_007743455.1
SAMN03169279	PHE	2012-10	United Kingdom: North of England	clinical	human	PDS000043142.5	SRR1645966	GCA_008018735.1
SAMN03169325	PHE	2012-06	United Kingdom: North of England	clinical	human	PDS000065764.1	SRR1646010	GCA_008018855.1
SAMN03169402	PHE	2012-11		clinical	human	PDS000039585.14	SRR1646070	GCA_008021475.1
SAMN03169443	PHE	2012-11	United Kingdom: Midlands and East of England	clinical	human	PDS000031464.1	SRR1646094	GCA_004192755.1
SAMN03169477	PHE	2012-10	United Kingdom: South of England	clinical	human	PDS000026614.75	SRR1646112	GCA_008020535.1
SAMN03169484	PHE	2012-06	United Kingdom: South of England	clinical	human	PDS000026707.2	SRR1646121	GCA_008020515.1
SAMN03169485	PHE	2012-09	United Kingdom: North of England	clinical	human	PDS000023053.2	SRR1646119	GCA_008020735.1
SAMN03169511	PHE	2012-08	United Kingdom: Midlands and East of England	clinical	human	PDS000030182.5	SRR1646146	GCA_008020015.1
SAMN03169527	PHE	2012-12	United Kingdom: London	clinical	human	PDS000074049.3	SRR1646162	GCA_008020915.1
SAMN03169551	PHE	2012-07	United Kingdom: North of England	clinical	human	PDS000066060.1	SRR1646198	GCA_008021295.1
SAMN03169568	PHE	2012-10	United Kingdom: Midlands and East of England	clinical	human	PDS000013849.68	SRR1646230	GCA_008021395.1
SAMN03169574	PHE	2012-11	United Kingdom: Midlands and East of England	clinical	human	PDS000026643.11	SRR1646238	GCA_008022175.1
SAMN03169627	PHE	2012-07	United Kingdom: South of England	clinical	human	PDS000056921.4	SRR1646285	GCA_008021845.1
SAMN03169682	PHE	2012-08	United Kingdom: London	clinical	human	PDS000031144.2	SRR1646378	GCA_008022995.1
SAMN03255380	Middle East Technical University| Food Engineering Department	12/7/12	Turkey	environmental/other	offal	PDS000013796.19	SRR1849305	GCA_006631245.1
SAMN03275998	FDA	2007	USA:TN	environmental/other	ground beef	PDS000031397.8	SRR1967103	GCA_006632545.1
SAMN03276090	FDA	2007	USA:CA	environmental/other	chicken breast	PDS000038752.6	SRR2070990	GCA_006634385.1
SAMN03276111	FDA	2007	USA:NY	environmental/other	chicken breast	PDS000031391.2	SRR2102423	GCA_006693865.1
SAMN03276121	FDA	2007	USA:CO	environmental/other	ground beef	PDS000092567.2	SRR2533574	GCA_007816355.1
SAMN03282125	FDA Contracted Laboratory	3/26/14	USA:GA	environmental/other	chicken	PDS000029417.21	SRR1812842	GCA_006016175.1
SAMN03285122	United States Food and Drug Administration	12/12/14	USA	environmental/other	hazelnut	PDS000028975.1	SRR1774093	GCA_006859685.1
SAMN03359716					culture	PDS000026652.51	SRR1814903	GCA_009256405.1
SAMN03464090	United States Food and Drug Administration	8/13/14	USA	environmental/other	water	PDS000111260.2	SRR2054219	GCA_006878805.1
SAMN03465619	PHE	2014-09	United Kingdom: Midlands and East of England	clinical	human	PDS000026809.31	SRR1957724	GCA_010536655.1
SAMN03465697	PHE	2014-09	United Kingdom: South of England	clinical	human	PDS000004546.13	SRR1957801	GCA_011624665.1
SAMN03465728	PHE	2014-11	United Kingdom: London	clinical	human	PDS000085353.2	SRR1957832	GCA_011085325.1
SAMN03465747	PHE	2014-07	United Kingdom: North of England	clinical	human	PDS000175001.7	SRR1957845	GCA_010907905.1
SAMN03465757	PHE	2014-08	United Kingdom: Midlands and East of England	clinical	human	PDS000029205.1	SRR1957855	GCA_011407815.1
SAMN03465786	PHE	2014-10	United Kingdom: Midlands and East of England	clinical	human	PDS000030975.3	SRR1957884	GCA_011086175.1
SAMN03465810	PHE	2014-08	United Kingdom: South of England	clinical	human	PDS000119524.2	SRR1957908	GCA_011093325.1
SAMN03465896	PHE	2014-09	United Kingdom: Midlands and East of England	clinical	human	PDS000013784.23	SRR1957994	GCA_011087425.1
SAMN03465914	PHE	2014-09	United Kingdom: Midlands and East of England	clinical	human	PDS000013717.8	SRR1958012	GCA_011638155.1
SAMN03465943	PHE	2014-10	United Kingdom: North of England	clinical	human	PDS000360017.5	SRR1958042	GCA_011087145.1
SAMN03466136	PHE	2014-08	United Kingdom: North of England	clinical	human	PDS000013747.38	SRR1958243	GCA_011087445.1
SAMN03466160	PHE	2014-08	United Kingdom: North of England	clinical	human	PDS000013840.10	SRR1958267	GCA_011094505.1
SAMN03466213	PHE	2014-10	United Kingdom: North of England	clinical	human	PDS000031258.5	SRR1958320	GCA_006632425.1
SAMN03466246	PHE	2014-10	United Kingdom: Midlands and East of England	clinical	human	PDS000013790.4	SRR1958353	GCA_010964125.1
SAMN03466269	PHE	2014-09	United Kingdom: London	clinical	human	PDS000031255.1	SRR1958376	GCA_004270105.1
SAMN03466279	PHE	2014-10	United Kingdom: London	clinical	human	PDS000030985.3	SRR1958386	GCA_011546975.1
SAMN03466287	PHE	2014-10	United Kingdom: North of England	clinical	human	PDS000065754.24	SRR1958394	GCA_010949275.1
SAMN03466288	PHE	2014-09	United Kingdom: London	clinical	human	PDS000194632.1	SRR1958395	GCA_011483165.1
SAMN03466298	PHE	2014-10	United Kingdom: London	clinical	human	PDS000026757.393	SRR1958405	GCA_011484025.1
SAMN03466300	PHE	2014-10	United Kingdom: North of England	clinical	human	PDS000026672.5	SRR1958407	GCA_011094805.1
SAMN03466369	PHE	2014-10	United Kingdom: South of England	clinical	human	PDS000050023.2	SRR1958476	GCA_011094925.1
SAMN03466374	PHE	2014-09	United Kingdom: South of England	clinical	human	PDS000031105.2	SRR1958481	GCA_011094865.1
SAMN03466456	PHE	2014-09	United Kingdom: South of England	clinical	human	PDS000030327.1	SRR1958563	GCA_011633225.1
SAMN03466568	PHE	2014-09	United Kingdom: South of England	clinical	human	PDS000051729.2	SRR1958675	GCA_010907205.1
SAMN03468422	PHE	2012-11	United Kingdom: None	clinical	human	PDS000030932.2	SRR1959219	GCA_011101575.1
SAMN03468479	PHE	2014-12	United Kingdom: South of England	clinical	human	PDS000047100.3	SRR1959274	GCA_011102175.1
SAMN03468483	PHE	2014-12	United Kingdom: London	clinical	human	PDS000013771.3	SRR1959278	GCA_010960215.1
SAMN03468489	PHE	2014-09	United Kingdom: North of England	clinical	human	PDS000031228.4	SRR1959284	GCA_006756325.1
SAMN03468532	PHE	2015-01	United Kingdom: Midlands and East of England	clinical	human	PDS000138059.1	SRR1959363	GCA_011534585.1
SAMN03468568	PHE	2015-01	United Kingdom: North of England	clinical	human	PDS000013788.10	SRR1959400	GCA_011097465.1
SAMN03468607	PHE	2014-12	United Kingdom: South of England	clinical	human	PDS000074030.6	SRR1959425	GCA_011096485.1
SAMN03468611	PHE	2012-11	United Kingdom: None	clinical	human	PDS000030391.4	SRR1959429	GCA_011107635.1
SAMN03468645	PHE	2014-04	United Kingdom: North of England	clinical	human	PDS000031220.12	SRR1959465	GCA_007748295.1
SAMN03468703	PHE	2014-12	United Kingdom: Wales	clinical	human	PDS000028519.2	SRR1959499	GCA_011640355.1
SAMN03468716	PHE	2014-12	United Kingdom: None	clinical	human	PDS000046543.1	SRR1959502	GCA_010966855.1
SAMN03468768	PHE	2014-12	United Kingdom: South of England	clinical	human	PDS000093289.4	SRR1960036	GCA_011109995.1

Supplemental Table 3.9. Select metadata downloaded from NCBI for serovar Typhimurium isolates included in Figure S3.7B.

Blue highlight indicates samples chosen for Figure 3.5A.

BioSample	Collected by	Collection date	Location	Isolation type	Isolation source	SNP cluster	Assembly	Run	Isolate	Strain	Computed types
SAMN07424695	CDC	2017-06	USA	clinical	stool	PDS000051476.18	GCA_008884465.1	SRR5888672	PDT000230419.2	PNUSAS018095	antigen_formula=4:i:1,2,serotype=Typhimurium
SAMN07455300		2017-03	USA	clinical	stool	PDS000064404.12	GCA_008435845.1	SRR5921488	PDT000232449.2	PNUSAS017228	antigen_formula=4:i:1,2,serotype=Typhimurium
SAMN07694680		2017-06	USA	clinical	stool	PDS000181137.1	GCA_009553555.1	SRR6107687	PDT000244544.2	PNUSAS024036	antigen_formula=4:i:1,2,serotype=Typhimurium
SAMN08407830	CDC	2018-01	USA	clinical	stool	PDS000076391.11	GCA_016381345.1	SRR6729918	PDT000282937.4	PNUSAS032925	antigen_formula=4:i:1,2,serotype=Typhimurium
SAMN08607476	CDC	2017-09	USA	clinical		PDS000049109.27	GCA_007561945.1	SRR6782607	PDT000289579.2	PNUSAS033431	antigen_formula=4:i:1,2,serotype=Typhimurium
SAMN08773726	CDC	2017-12	USA	clinical	stool	PDS000043305.4	GCA_007202195.1	SRR6880186	PDT000297011.3	PNUSAS033206	antigen_formula=4:i:1,2,serotype=Typhimurium
SAMN09437554	CDC	2017-11	USA	clinical		PDS000126891.2	GCA_006647405.1	SRR7352850	PDT000331872.1	PNUSAS042448	antigen_formula=4:i:1,2,serotype=Typhimurium
SAMN10241133	CDC	2018-09	USA	clinical		PDS000038355.20	GCA_016300485.1	SRR8054302	PDT000391315.1	PNUSAS056026	antigen_formula=4:i:1,2,serotype=Typhimurium
SAMN11102910	KY-M	7/9/18	USA:KY	environmental/other	Odocoileus virginianus Feces	PDS000044303.2	GCA_006239385.1	SRR8767784	PDT000481252.1	SAL-18-VL-LA-KY-0011	antigen_formula=4:i:1,2,serotype=Typhimurium
SAMN11526594	CDC	2019-03	USA	clinical		PDS000101797.2	GCA_005602395.1	SRR8984755	PDT000496339.1	PNUSAS070747	antigen_formula=4:i:1,2,serotype=Typhimurium
SAMN12036221	CDC	2019-05	USA	clinical		PDS000054358.20	GCA_006425235.1	SRR9283102	PDT000521479.1	PNUSAS077320	antigen_formula=4:i:1,2,serotype=Typhimurium
SAMN14083425	CDC	2020-01	USA	clinical		PDS000149796.1	GCA_011489485.1	SRR11067570	PDT000682507.1	PNUSAS134090	antigen_formula=4:i:1,2,serotype=Typhimurium
SAMN15399837	CDC	2020-06	USA	clinical		PDS000150767.2	GCA_016167695.1	SRR12109672	PDT000773851.1	PNUSAS149967	antigen_formula=4:i:1,2,serotype=Typhimurium
SAMN19974806	CDC	2021-06	USA	clinical		PDS000192391.1	GCA_019087325.1	SRR15006564	PDT001080017.1	PNUSAS209577	antigen_formula=4:i:1,2,serotype=Typhimurium
SAMN20090106		2021-06	USA	clinical		PDS000093278.1	GCA_019162375.1	SRR15058485	PDT001083896.1	1019624001	antigen_formula=4:i:1,2,serotype=Typhimurium
SAMN29376357	CDC	2022-05	USA	clinical		PDS000113229.3	GCA_023972455.1	SRR19866112	PDT001347919.1	PNUSAS277622	antigen_formula=4:i:1,2,serotype=Typhimurium
SAMN31022944	CDC	2022-09	USA	clinical		PDS000121793.1	GCA_025418035.1	SRR21712787	PDT001434404.1	PNUSAS303535	antigen_formula=4:i:1,2,serotype=Typhimurium
SAMN34381829		2023	USA	clinical		PDS000154543.2	GCA_032269965.1	SRR24313502	PDT001708295.1	PNUSAS344096	antigen_formula=4:i:1,2,serotype=Typhimurium
SAMN42161866				clinical		PDS000187829.1	GCA_040425175.1	SRR29644432	PDT002234490.1	PNUSAS447913	antigen_formula=4:i:1,2,serotype=Typhimurium
SAMN43494260				clinical		PDS000197424.1	GCA_041764295.1	SRR30541472	PDT002362326.1	PNUSAS470360	antigen_formula=4:i:1,2,serotype=Typhimurium

Supplemental Table S3.10 Weather metadata for day of sampling and two days prior from weather station 1 for Creek A.

Year	Julian Day	Max Air Temperature(F)	Min Air Temperature(F)	Avg Relative Humidity(%)	Avg Wind Speed(mph)	Max Wind Speed (m/s)	Total Solar Radiation(MJ/m^2)	Total Rain(in)
2021	18-Nov	70.9	44.3	74.0	3.4	9.1	9.1	0.0
2021	19-Nov	65.9	35.9	48.5	2.8	5.1	13.6	0.0
2021	20-Nov	53.5	39.0	49.3	4.7	9.1	13.3	0.0
2021	16-Dec	65.8	50.1	68.7	1.5	3.5	5.7	0.0
2021	17-Dec	68.6	55.0	91.5	0.9	2.7	5.1	0.0
2021	18-Dec	63.8	56.8	98.0	1.9	5.1	1.7	0.6
2022	13-Jan	53.3	32.8	68.5	3.8	3.5	7.9	0.0
2022	14-Jan	56.6	35.6	69.6	2.6	2.7	11.8	0.0
2022	15-Jan	43.3	36.6	67.2	6.6	5.1	3.8	0.0
2022	17-Feb	65.8	52.4	88.7	4.9	14.7	2.2	1.4
2022	18-Feb	59.9	32.9	68.3	7.2	9.1	17.0	0.1
2022	19-Feb	57.4	28.9	51.4	4.8	9.9	17.7	0.0
2022	17-Mar	71.4	51.8	75.7	2.8	7.5	20.2	0.0
2022	18-Mar	56.1	47.4	94.3	3.3	12.3	1.8	0.7
2022	19-Mar	62.2	48.2	70.7	6.4	10.7	20.6	0.1
2022	14-Apr	73.8	52.6	65.2	3.6	8.3	16.2	0.0
2022	15-Apr	72.4	42.4	51.3	4.0	9.1	25.3	0.0
2022	16-Apr	65.1	53.1	85.6	2.7	5.1	8.0	0.8
2022	19-May	88.9	65.6	67.9	2.9	7.5	23.8	0.0
2022	20-May	86.8	68.9	69.0	3.0	9.1	24.8	0.0
2022	21-May	86.2	68.0	73.7	3.0	11.5	19.1	0.0
2022	16-Jun	98.2	75.8	68.6	2.2	5.9	25.3	0.0
2022	17-Jun	92.2	73.7	75.7	3.7	11.5	18.8	0.1
2022	18-Jun	90.4	70.0	58.9	3.2	8.3	26.5	0.0
2022	14-Jul	89.8	71.9	70.7	2.3	5.1	22.6	0.0
2022	15-Jul	92.6	67.0	71.5	1.8	5.9	23.9	0.1
2022	16-Jul	90.1	69.4	76.3	2.1	5.1	19.3	0.0
2022	18-Aug	81.8	66.6	82.6	2.3	5.1	17.1	0.2
2022	19-Aug	78.3	69.6	88.7	3.3	6.7	9.1	0.0
2022	20-Aug	80.1	70.1	91.5	2.1	5.1	8.7	0.2
2022	22-Sep	90.4	65.0	69.4	4.3	14.7	17.0	0.0
2022	23-Sep	78.2	53.9	61.6	2.6	5.9	21.6	0.0
2022	24-Sep	84.0	50.9	63.5	2.1	5.1	20.2	0.0
2022	20-Oct	62.3	34.0	52.4	3.2	9.1	17.3	0.0
2022	21-Oct	70.7	36.4	56.8	2.1	5.1	16.9	0.0
2022	22-Oct	76.7	38.1	60.4	2.0	5.1	16.4	0.0
2022	17-Nov	47.9	34.1	46.6	5.3	6.7	13.7	0.0
2022	18-Nov	54.6	28.8	47.3	3.5	7.5	13.2	0.0
2022	19-Nov	51.4	35.3	54.9	5.6	8.3	10.6	0.0
2022	12-Dec	58.0	48.3	88.1	2.9	5.9	8.8	0.0
2022	13-Dec	49.8	42.5	76.2	5.8	9.9	8.4	0.0
2022	14-Dec	45.8	40.3	91.5	7.2	9.1	0.8	0.9
2023	19-Jan	69.1	55.7	67.2	5.6	11.5	8.4	0.1
2023	20-Jan	56.6	41.5	48.6	7.5	9.9	13.1	0.0
2023	21-Jan	49.2	35.1	59.2	4.5	6.7	8.3	0.0
2023	16-Feb	66.7	49.7	89.2	2.2	6.7	6.0	0.0
2023	17-Feb	64.2	33.6	74.8	8.2	12.3	12.3	0.8
2023	18-Feb	58.9	28.6	54.8	2.6	5.1	17.6	0.0
2023	16-Mar	67.9	31.1	49.2	3.2	6.7	20.2	0.0
2023	17-Mar	60.6	43.7	84.6	3.7	8.3	2.1	0.5
2023	18-Mar	53.2	37.3	50.2	6.7	9.1	20.1	0.0
2023	13-Apr	69.4	46.7	76.5	3.2	9.9	12.3	0.3
2023	14-Apr	69.6	57.8	91.3	2.4	5.9	10.2	0.8
2023	15-Apr	77.5	58.0	75.8	2.7	8.3	22.3	0.0
2023	18-May	69.2	60.8	86.3	5.3	8.3	5.9	0.0
2023	19-May	73.4	59.1	77.4	4.3	6.7	16.6	0.0
2023	20-May	79.1	64.1	83.2	3.0	5.1	12.6	0.4
2023	15-Jun	81.5	65.6	81.2	4.1	7.5	20.7	0.4
2023	16-Jun	84.7	65.5	74.4	4.5	9.9	23.8	0.7
2023	17-Jun	87.1	66.6	66.2	3.0	5.9	23.8	0.0
2023	13-Jul	91.5	71.5	72.5	2.9	8.3	24.5	0.0
2023	14-Jul	91.1	74.0	77.4	3.1	8.3	21.0	0.0
2023	15-Jul	89.3	72.8	76.5	3.2	8.3	19.3	0.1
2023	17-Aug	84.5	64.4	70.2	3.5	7.5	23.1	0.0
2023	18-Aug	89.3	68.6	64.4	3.6	6.7	22.6	0.0
2023	19-Aug	84.3	63.1	70.6	3.0	5.9	23.3	0.0
2023	14-Sep	76.8	68.3	79.4	3.1	5.9	7.0	0.0
2023	15-Sep	75.3	65.5	78.2	3.8	5.9	10.3	0.0
2023	16-Sep	72.4	62.7	87.4	2.5	5.1	6.6	0.1
2023	20-Oct	70.0	55.4	79.1	4.0	9.9	11.5	0.3
2023	21-Oct	72.2	50.8	56.0	4.2	8.3	17.0	0.0
2023	22-Oct	75.2	50.8	48.4	4.2	7.5	16.5	0.0

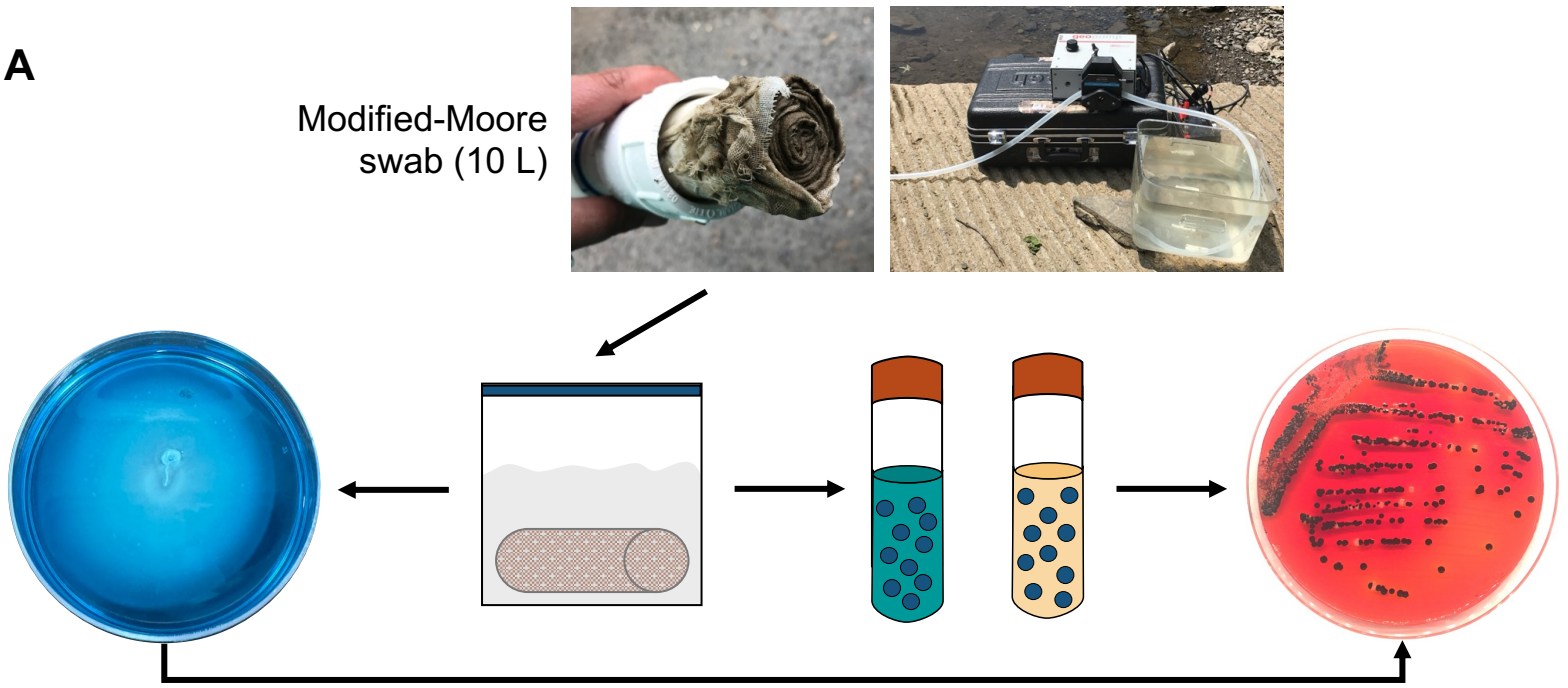
Supplemental Table S3.11 Weather metadata for day of sampling and two days prior from weather station 2 for Creek B.

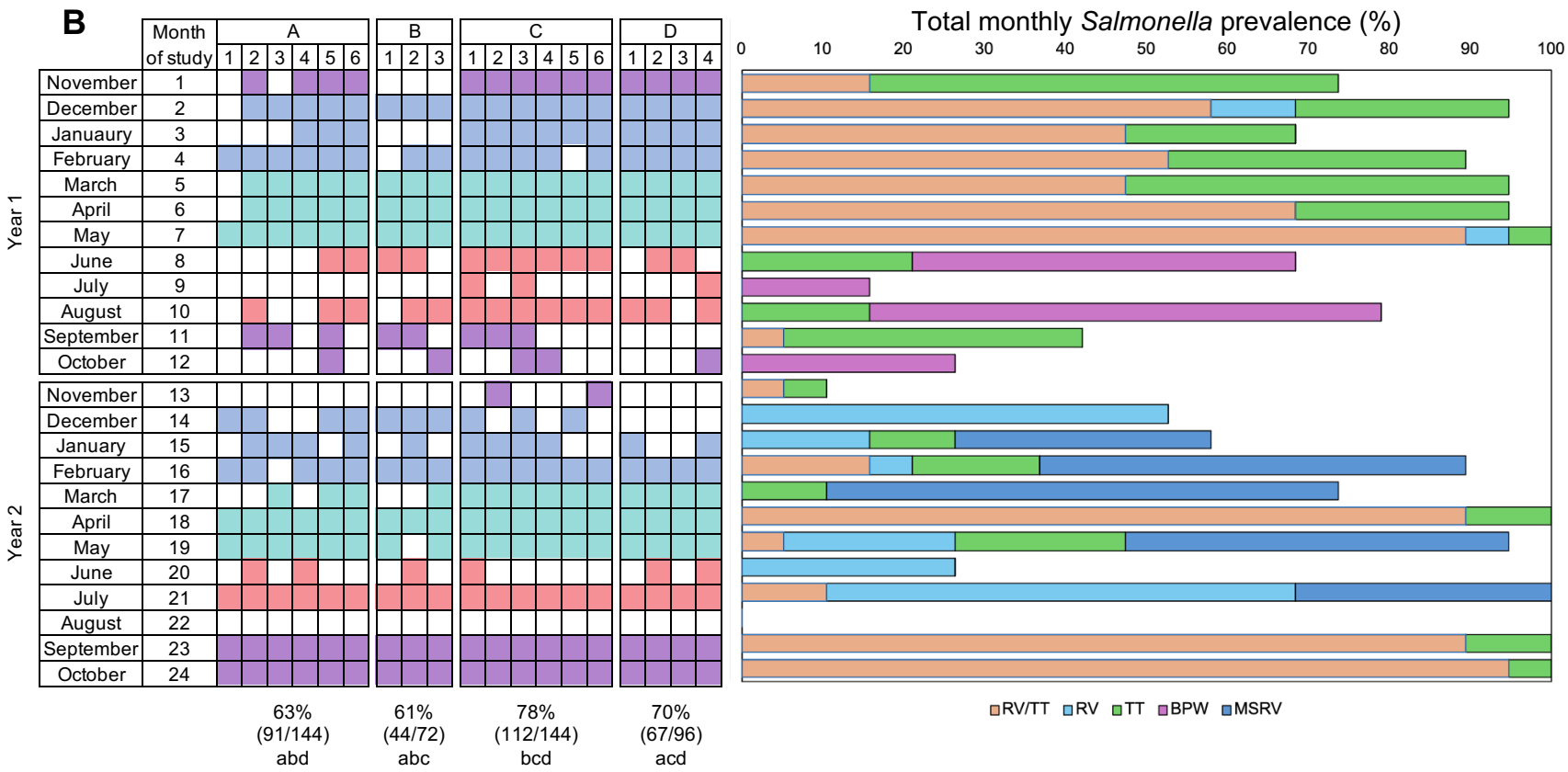
Year	Julian Day	Max Air Temperature(F)	Min Air Temperature(F)	Avg Relative Humidity(%)	Avg Wind Speed(mph)	Max Wind Speed (m/s)	Total Solar Radiation(MJ/m^2)	Total Rain(in)
2021	18-Nov	75.2	42.7	78.4	3.0	7.5	10.7	0.0
2021	19-Nov	61.3	33.9	43.5	2.4	4.3	14.8	0.0
2021	20-Nov	55.4	34.7	52.5	4.5	7.5	14.1	0.0
2021	16-Dec	63.1	44.0	80.7	2.1	4.3	5.3	0.0
2021	17-Dec	67.3	53.9	94.1	1.9	5.1	6.0	0.0
2021	18-Dec	65.6	57.3	99.3	3.2	6.7	2.0	0.8
2022	13-Jan	57.7	29.4	60.3	3.7	8.3	9.6	0.0
2022	14-Jan	56.4	36.1	64.7	2.6	5.9	12.9	0.0
2022	15-Jan	44.1	35.4	69.3	4.2	7.5	3.9	0.0
2022	17-Feb	72.1	52.2	78.9	6.8	13.9	5.9	0.0
2022	18-Feb	67.7	32.3	74.8	5.6	9.9	16.7	1.0
2022	19-Feb	58.4	27.6	52.7	4.2	6.7	18.6	0.0
2022	17-Mar	73.5	52.6	76.5	2.4	5.1	20.5	0.0
2022	18-Mar	58.1	50.3	91.7	3.0	11.5	2.5	0.7
2022	19-Mar	67.9	48.5	69.3	7.1	9.9	20.6	0.0
2022	14-Apr	77.6	56.0	64.3	4.9	9.9	17.1	0.0
2022	15-Apr	75.6	41.8	46.9	3.2	7.5	26.0	0.0
2022	16-Apr	64.0	53.0	89.2	2.8	4.3	7.2	0.6
2022	19-May	93.0	63.4	60.8	3.3	7.5	24.2	0.0
2022	20-May	89.1	67.5	62.4	4.1	9.1	26.6	0.0
2022	21-May	89.2	66.2	71.3	3.7	7.5	19.8	0.0
2022	16-Jun	97.3	73.4	64.2	2.0	5.9	22.3	0.0
2022	17-Jun	95.3	73.4	65.9	2.8	7.5	24.8	0.0
2022	18-Jun	92.4	68.0	57.7	2.2	5.1	28.1	0.0
2022	14-Jul	86.8	71.2	80.7	1.6	5.1	16.6	0.0
2022	15-Jul	90.7	68.8	71.3	1.8	5.1	25.4	0.0
2022	16-Jul	90.2	68.5	75.0	2.2	4.3	24.9	0.0
2022	18-Aug	82.7	69.1	81.1	1.5	5.1	14.3	0.0
2022	19-Aug	76.6	70.1	93.8	1.5	4.3	6.5	0.1
2022	20-Aug	85.4	71.5	84.6	0.9	5.9	14.6	0.5
2022	22-Sep	93.0	64.0	71.4	1.9	11.5	17.9	0.1
2022	23-Sep	78.5	53.0	57.8	2.3	6.7	21.5	0.0
2022	24-Sep	80.7	47.9	64.4	1.4	5.1	20.5	0.0
2022	20-Oct	63.5	31.1	53.6	1.7	5.9	17.4	0.0
2022	21-Oct	69.7	33.6	56.8	1.2	4.3	17.6	0.0
2022	22-Oct	74.3	35.3	62.2	1.3	4.3	17.3	0.0
2022	17-Nov	50.1	30.5	53.7	2.2	5.1	14.6	0.0
2022	18-Nov	55.3	27.2	50.0	1.7	6.7	14.3	0.0
2022	19-Nov	55.0	32.1	55.7	2.5	6.7	11.1	0.0
2022	12-Dec	55.9	43.5	94.9	1.9	3.5	4.0	0.0
2022	13-Dec	50.7	40.4	83.1	3.5	5.9	7.6	0.0
2022	14-Dec	44.9	41.0	89.4	3.8	5.9	0.9	0.5
2023	19-Jan	71.3	56.2	68.8	7.0	10.7	7.5	0.0
2023	20-Jan	60.3	43.1	43.8	5.6	9.1	13.5	0.0
2023	21-Jan	47.7	37.0	60.5	3.4	5.9	5.2	0.0
2023	16-Feb	73.0	43.8	82.6	3.7	7.5	8.7	0.0
2023	17-Feb	66.0	33.9	70.9	8.4	12.3	13.0	0.5
2023	18-Feb	56.7	26.7	51.9	3.0	5.1	18.3	0.0
2023	16-Mar	67.5	29.2	46.1	3.8	6.7	21.9	0.0
2023	17-Mar	60.1	47.9	82.0	4.8	9.9	3.8	0.6
2023	18-Mar	54.6	36.4	52.8	6.0	9.9	19.7	0.0
2023	13-Apr	70.9	44.2	85.1	2.7	10.7	10.5	0.6
2023	14-Apr	68.3	59.2	93.8	2.0	7.5	11.9	0.3
2023	15-Apr	78.7	57.1	74.2	2.8	6.7	23.8	0.0
2023	18-May	68.6	60.4	93.5	4.2	8.3	4.9	0.8
2023	19-May	71.2	58.6	83.8	3.6	7.5	12.0	0.0
2023	20-May	78.4	63.9	81.9	2.4	5.1	15.1	0.1
2023	15-Jun	80.8	66.0	83.0	4.0	5.9	16.3	0.0
2023	16-Jun	86.7	65.6	71.1	2.6	7.5	24.9	0.3
2023	17-Jun	88.9	66.7	64.0	1.7	5.1	26.9	0.0
2023	13-Jul	91.4	72.9	75.6	2.5	5.9	25.8	0.0
2023	14-Jul	92.0	73.1	78.6	2.2	4.3	23.4	0.0
2023	15-Jul	92.9	71.9	74.3	2.8	7.5	24.9	0.0
2023	17-Aug	86.0	64.9	72.8	1.5	5.9	25.3	0.0
2023	18-Aug	88.3	67.1	70.4	1.1	4.3	23.7	0.0
2023	19-Aug	86.5	64.2	75.3	1.6	5.1	24.5	0.0
2023	14-Sep	82.3	66.7	82.8	2.9	6.7	11.7	0.2
2023	15-Sep	77.5	65.3	82.0	2.6	5.1	10.3	0.1
2023	16-Sep	74.6	63.3	88.7	0.9	5.1	7.5	0.1
2023	20-Oct	74.2	53.5	76.0	2.9	9.1	10.9	0.1
2023	21-Oct	74.9	47.4	52.5	3.1	6.7	17.9	0.0
2023	22-Oct	77.9	50.9	50.5	1.9	5.9	17.8	0.0

Supplemental Table S3.12 Weather metadata for day of sampling and two days prior from weather station 3 for Creeks C and D.

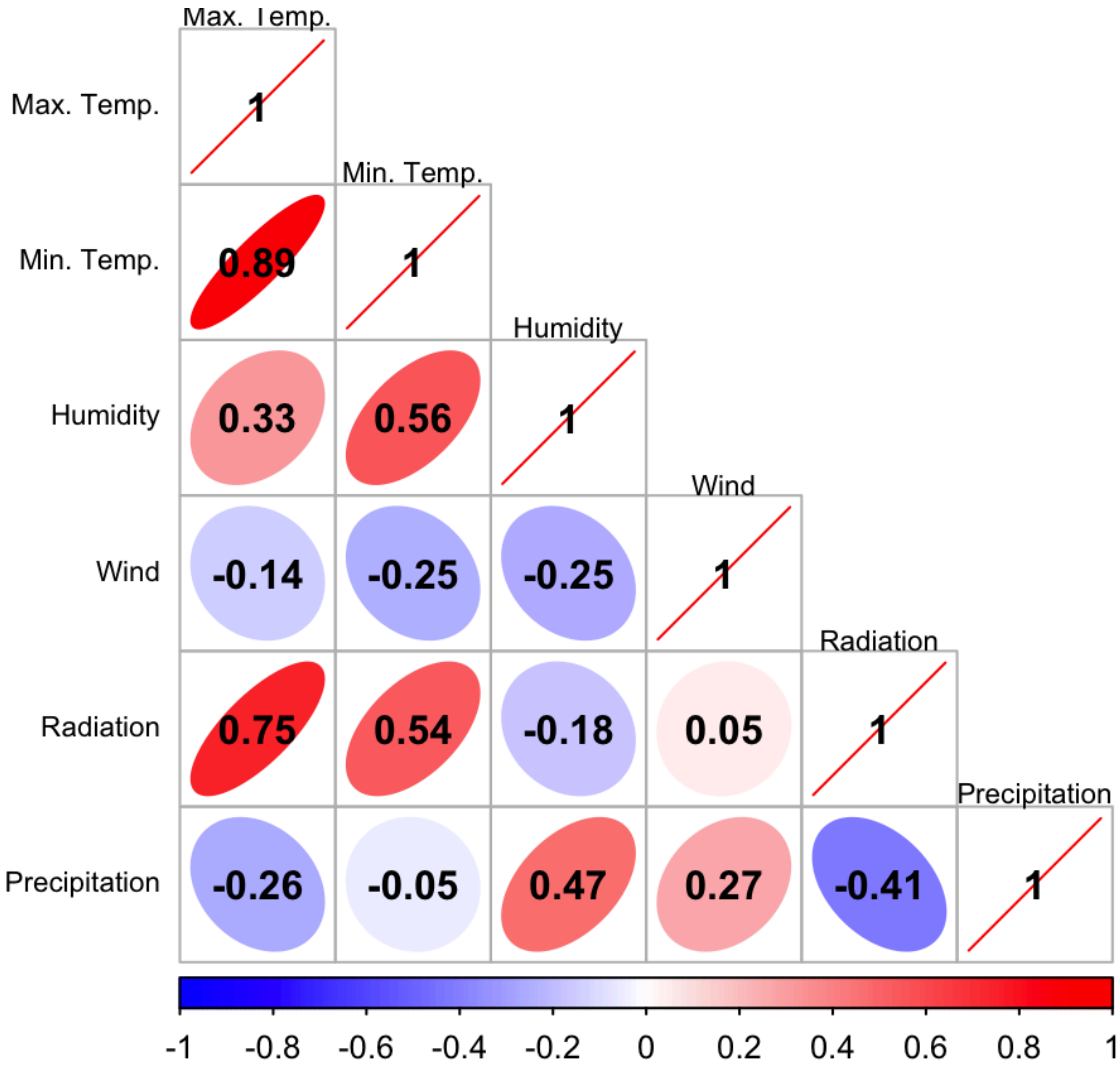
Year	Julian Day	Max Air Temperature(F)	Min Air Temperature(F)	Avg Relative Humidity(%)	Avg Wind Speed(mph)	Max Wind Speed (m/s)	Total Solar Radiation(MJ/m^2)	Total Rain(in)
2021	18-Nov	74.9	44.2	77.9	5.0	8.3	10.4	0.0
2021	19-Nov	61.9	33.7	48.2	3.0	6.7	13.8	0.0
2021	20-Nov	55.8	34.1	57.6	3.3	7.5	13.3	0.0
2021	16-Dec	63.2	44.8	83.0	2.0	4.3	5.0	0.0
2021	17-Dec	67.2	52.2	94.2	2.5	5.1	5.7	0.0
2021	18-Dec	65.8	56.6	99.3	4.7	6.7	2.0	0.8
2022	13-Jan	57.4	31.2	59.7	6.0	9.9	9.1	0.0
2022	14-Jan	57.1	36.2	65.7	4.1	6.7	12.0	0.0
2022	15-Jan	44.7	35.5	70.9	3.5	6.7	4.0	0.1
2022	17-Feb	72.3	52.2	79.4	8.0	17.1	6.1	0.0
2022	18-Feb	67.3	32.4	75.4	8.6	12.3	15.7	1.0
2022	19-Feb	57.9	27.9	52.6	6.6	9.9	17.8	0.0
2022	17-Mar	74.0	52.4	75.8	2.7	5.9	19.7	0.0
2022	18-Mar	58.2	50.5	91.1	3.3	20.3	2.4	0.7
2022	19-Mar	68.0	48.6	69.3	9.1	10.7	19.8	0.0
2022	14-Apr	77.0	54.8	65.4	6.7	9.1	16.9	0.0
2022	15-Apr	76.8	42.5	45.3	2.9	6.7	25.7	0.0
2022	16-Apr	64.2	53.1	89.6	3.0	4.3	7.3	0.6
2022	19-May	92.3	64.7	62.7	4.8	9.1	24.8	0.0
2022	20-May	89.3	66.9	64.6	4.7	9.1	26.9	0.0
2022	21-May	89.9	66.5	72.1	4.3	7.5	20.7	0.0
2022	16-Jun	98.6	73.3	65.2	1.7	4.3	24.1	0.0
2022	17-Jun	96.6	73.1	68.5	5.5	12.3	25.3	0.0
2022	18-Jun	92.9	68.7	58.8	5.1	9.9	28.2	0.0
2022	14-Jul	88.4	70.6	81.2	2.4	4.3	17.1	0.0
2022	15-Jul	92.0	69.1	72.0	2.3	4.3	24.3	0.0
2022	16-Jul	90.6	68.6	75.7	2.5	5.1	24.8	0.0
2022	18-Aug	83.4	69.5	80.6	0.9	4.3	13.8	0.0
2022	19-Aug	78.0	70.1	94.0	0.6	3.5	6.7	0.1
2022	20-Aug	86.1	71.4	84.9	1.0	7.5	16.0	0.2
2022	22-Sep	94.4	64.9	67.7	5.8	13.1	19.4	0.0
2022	23-Sep	79.9	53.3	55.9	1.4	5.1	22.5	0.0
2022	24-Sep	80.6	47.9	62.6	1.8	5.1	21.5	0.0
2022	20-Oct	63.5	31.8	52.4	2.5	6.7	17.3	0.0
2022	21-Oct	70.1	34.0	54.9	1.2	4.3	17.5	0.0
2022	22-Oct	74.6	36.7	60.3	1.1	3.5	17.0	0.0
2022	17-Nov	49.0	31.0	54.6	4.8	7.5	14.3	0.0
2022	18-Nov	55.1	27.5	49.9	3.2	5.9	13.8	0.0
2022	19-Nov	54.9	30.7	56.6	4.6	9.1	11.0	0.0
2022	12-Dec	56.3	43.0	95.4	1.3	3.5	4.3	0.0
2022	13-Dec	51.0	40.7	82.1	2.2	5.1	7.3	0.0
2022	14-Dec	45.2	41.1	90.5	2.5	5.1	0.9	0.6
2023	19-Jan	71.6	57.2	68.2	8.2	11.5	7.5	0.0
2023	20-Jan	59.4	43.2	45.0	8.9	10.7	13.2	0.0
2023	21-Jan	48.3	38.0	61.2	3.2	7.5	5.5	0.0
2023	16-Feb	73.0	44.8	82.5	3.6	7.5	9.2	0.0
2023	17-Feb	66.0	33.6	72.5	12.0	15.5	12.9	0.4
2023	18-Feb	57.3	27.3	51.5	3.0	5.9	18.3	0.0
2023	16-Mar	68.9	30.3	45.1	4.0	7.5	21.0	0.0
2023	17-Mar	60.2	47.4	84.1	5.9	12.3	3.5	0.7
2023	18-Mar	53.9	36.4	53.9	8.8	10.7	18.7	0.0
2023	13-Apr	72.4	44.7	87.6	1.7	8.3	10.6	0.6
2023	14-Apr	68.8	59.5	94.0	1.7	7.5	11.7	0.3
2023	15-Apr	78.3	56.6	74.6	3.5	8.3	23.4	0.0
2023	18-May	69.0	60.9	93.6	2.0	5.1	5.2	1.5
2023	19-May	72.6	58.8	83.3	1.6	4.3	12.2	0.0
2023	20-May	78.7	64.2	82.2	2.6	5.9	16.3	0.3
2023	15-Jun	80.7	65.2	85.0	4.9	9.1	16.8	0.0
2023	16-Jun	85.9	65.3	72.7	5.4	9.9	25.0	0.1
2023	17-Jun	88.2	65.9	66.0	3.3	7.5	25.5	0.0
2023	13-Jul	91.2	72.9	78.4	2.9	5.9	25.9	0.0
2023	14-Jul	90.9	72.4	80.7	2.6	5.9	22.6	0.0
2023	15-Jul	92.1	71.3	76.1	3.4	7.5	23.9	0.0
2023	17-Aug	85.2	64.6	74.3	2.9	6.7	23.9	0.0
2023	18-Aug	88.2	66.0	72.3	3.2	7.5	25.3	0.0
2023	19-Aug	87.1	65.3	73.7	1.2	4.3	23.7	0.0
2023	14-Sep	83.4	67.0	83.9	1.1	4.3	11.7	0.0
2023	15-Sep	78.6	65.1	83.9	1.4	4.3	11.2	0.1
2023	16-Sep	75.4	62.9	90.3	1.1	5.1	7.9	0.1
2023	20-Oct	73.3	52.7	77.7	5.5	9.9	10.3	0.1
2023	21-Oct	73.8	48.5	55.7	5.6	9.1	17.6	0.0
2023	22-Oct	76.6	53.1	52.4	4.8	9.1	17.4	0.0

A

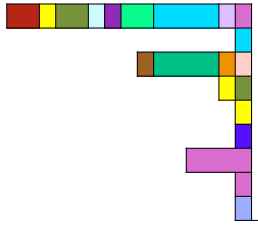

Modified-Moore swab (10 L)
Motility plate (MSRV)
Pre-enrichment (BPW)
Selective enrichment (RV and TT)
Selective plating (XLT-4)


B
Month of study
A
B
C
D
Year 1
Year 2
November
December
Januaury
February
March
April
May
June
July
August
September
October
January
63% (91/144) abd
61% (44/72) abc
78% (112/144) bcd
70% (67/96) acd
Total monthly Salmonella prevalence (%)
0
10
20
30
40
50
60
70
80
90
100
RV/TT
RV
TT
BPW
MSRV

Supplemental Figure 3.1. Sample collection and prevalence of *Salmonella* in creeks. A) Water samples were collected by pumping 10 liters through a modified Moore swab (MMS). The swab was returned to lab on ice and pre-enriched in buffered peptone water (BPW). Samples were transferred to selective enrichment media in parallel, including Rappaport-Vassiliadis (RV), Tetrathionate (TT), and modified semisolid RV (MSRV). Each enrichment was then plated onto an indicator plate, xylose lysine tergitol-4 (XLT-4) for confirmation. B) Prevalence is shown with colored squares reflecting positive samples and white squares are negative with overall sample number for each creeks shown at the bottom. The color of the square reflects the season of the sample collection, with observed differences in prevalence between them: fall (purple), winter (light blue), spring (light green), summer (light red) (Chi-squared test, $p < 0.001$). Additionally, *Salmonella* recovery varied by creek, with relationships denoted below prevalence values (Chi-squared test, $p < 0.05$). Isolated colonies were saved from XLT-4 plates for all positive enrichments, including from BPW when RV and TT cultures were negative. MSRV was included in the protocol at the beginning of Year 2 due to sustained low prevalence in RV and TT starting in June of Year 1, however, isolates only from this media were collected in five out of 12 months in Year 2.

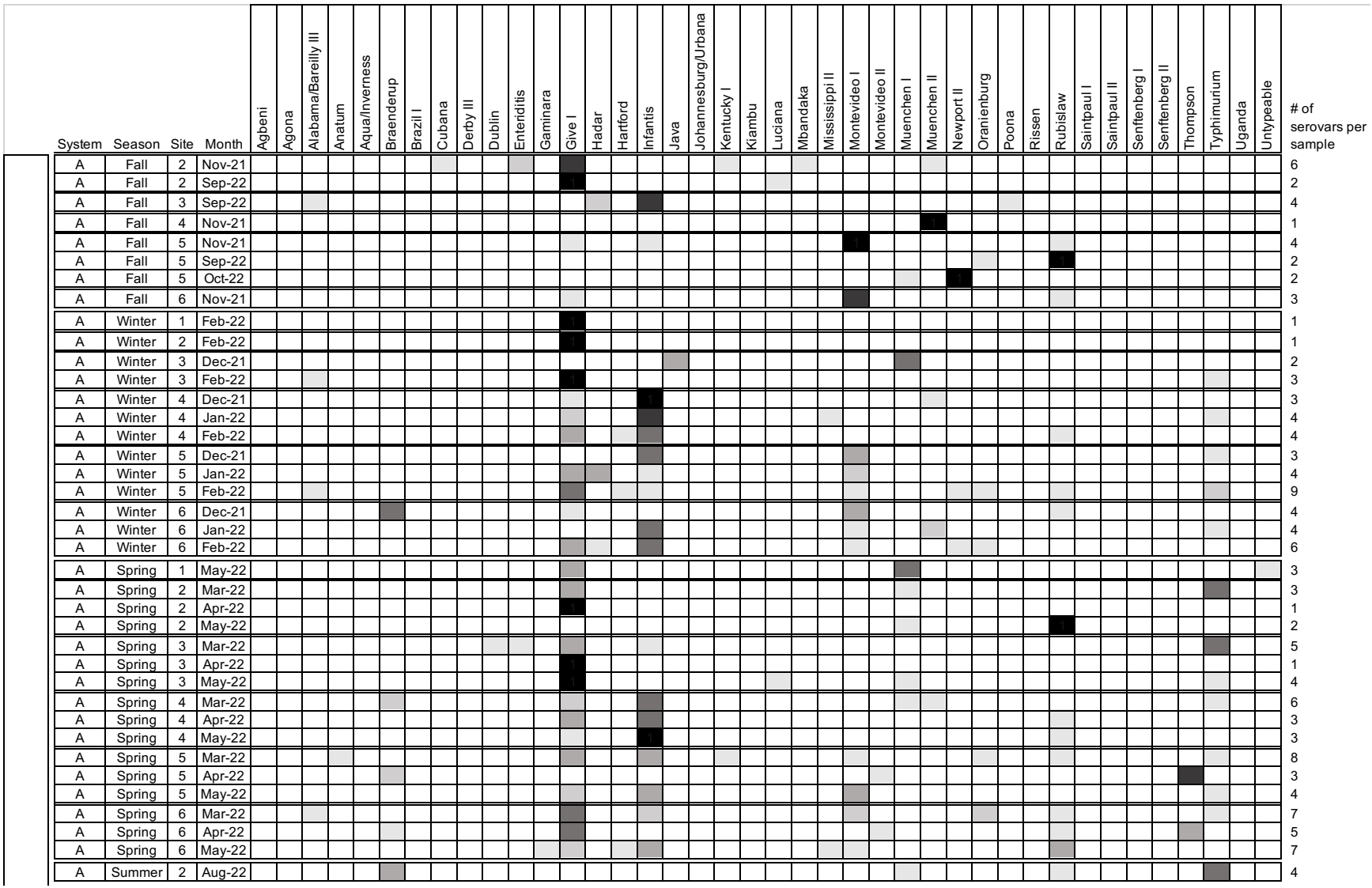


Supplemental Figure 3.2. Association between different weather variables. Pearson correlation coefficient was calculated for each pairwise combination of weather variables recorded in this study. The resulting coefficients are displayed within each respective box of the matrix, with the size of the circle indicating the strength and direction of association and the color denoting if it was positive (red) or negative (blue).



Watershed A	Watershed B	Watershed C	Watershed D	Antibiotic Resistance Profile
80	39	102	60	Pansusceptible (281)
7	2	4	2	Streptomycin (15)
1				Tetracycline (1)
3	2		2	Streptomycin,Tetracycline (7)
		1	1	Tetracycline, Chloramphenicol (2)
			1	Streptomycin,Tetracyclin, Nalidixic Acid (1)
			1	Tetracycline, Chloramphenicol, Amoxicillin/Clavulanic Acid (1)
		4		Ampicillin, Streptomycin, Tetracycline, Chloramphenicol (4)
		1		Ampicillin, Amoxicillin/Clavulanic Acid, Streptomycin, Tetracycline, Chloramphenicol (1)
	1			Ampicillin, Ceftriaxone, Gentamicin, Streptomycin, Tetracycline, Sulfamethoxazole-Trimethoprim, Chloramphenicol (1)
91	44	112	67	

Supplemental Figure 3.3. Assessment of antibiotic-resistant *Salmonella* isolates. Kirby-Bauer disk diffusion assays were used to assess resistance to ampicillin, amoxicillin-clavulanic acid, ceftriaxone, gentamicin, streptomycin, tetracycline, ciprofloxacin, sulfamethoxazole-trimethoprim, and chloramphenicol. The classification of resistance was determined by CLSI, and an intermediate result was considered as susceptible. Multidrug resistance was defined by resistance to three of more classes of antibiotics. Serovar identification was completed using SeqSero from whole genome sequencing data. Bolded serovars are of highest human clinical importance as denoted by their presence in the top 15 serovars listed on the BEAM Dashboard.



System	Season	Site	Month	Agbeni	Agona	Alabama/Bareilly III	Anatum	Aqua/Inverness	Braenderup	Brazil I	Cubana	Derby III	Dublin	Enteriditis	Gaminara	Give I	Hadar	Hartford	Infantis	Java	Johannesburg/Urbana	Kentucky I	Kiambu	Luciana	Mbandaka	Mississippi II	Montevideo I	Montevideo II	Muenchen I	Muenchen II	Newport II	Oranienburg	Poona	Rissen	Rubislaw	Saintpaul I	Saintpaul II	Senftenberg I	Senftenberg II	Thompson	Typhimurium	Uganda	Untypeable	# of serovars per sample
A	Fall	2	Nov-21																																									6
A	Fall	2	Sep-22																																									2
A	Fall	3	Sep-22																																									4
A	Fall	4	Nov-21																																									1
A	Fall	5	Nov-21																																									4
A	Fall	5	Sep-22																																									2
A	Fall	5	Oct-22																																									2
A	Fall	6	Nov-21																																									3
A	Winter	1	Feb-22																																									1
A	Winter	2	Feb-22																																									1
A	Winter	3	Dec-21																																									2
A	Winter	3	Feb-22																																									3
A	Winter	4	Dec-21																																									3
A	Winter	4	Jan-22																																									4
A	Winter	4	Feb-22																																									4
A	Winter	5	Dec-21																																									3
A	Winter	5	Jan-22																																									4
A	Winter	5	Feb-22																																									9
A	Winter	6	Dec-21																																									4
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A	Spring	1	May-22																																									3
A	Spring	2	Mar-22																																									3
A	Spring	2	Apr-22																																									1
A	Spring	2	May-22																																									2
A	Spring	3	Mar-22																																									5
A	Spring	3	Apr-22																																									1
A	Spring	3	May-22																																									4
A	Spring	4	Mar-22																																									6
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A	Spring	5	Mar-22																																									8
A	Spring	5	Apr-22																																									3
A	Spring	5	May-22																																									4
A	Spring	6	Mar-22																																									7
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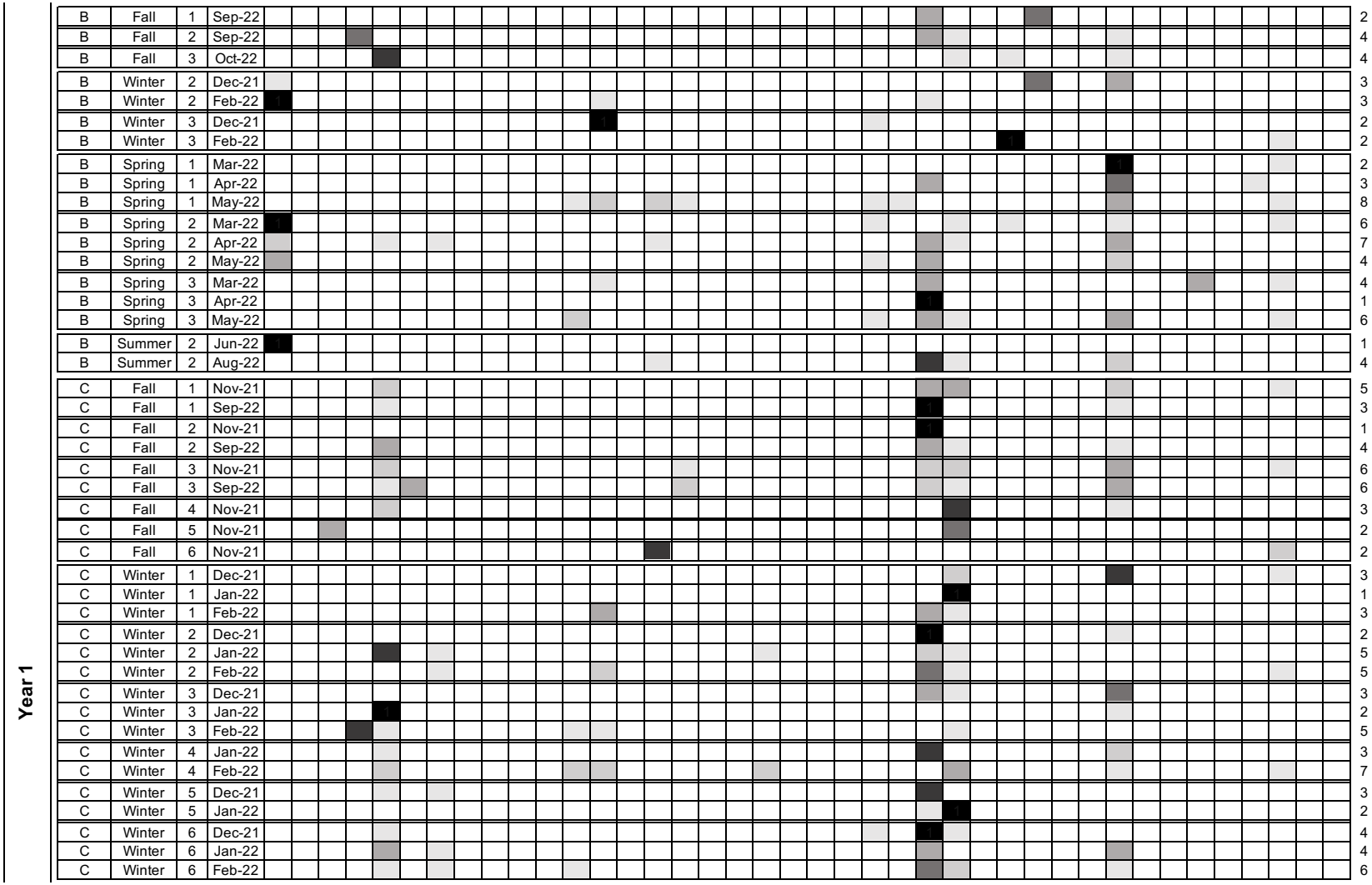
A	Spring	6	May-22																																									7
A	Summer	2	Aug-22																																									4


Year 1
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B Fall 2 Sep-22 4
B Fall 3 Oct-22 4
B Winter 2 Dec-21 3
B Winter 2 Feb-22 3
B Winter 3 Dec-21 2
B Winter 3 Feb-22 2
B Spring 1 Mar-22 2
B Spring 1 Apr-22 3
B Spring 1 May-22 8
B Spring 2 Mar-22 6
B Spring 2 Apr-22 7
B Spring 2 May-22 4
B Spring 3 Mar-22 4
B Spring 3 Apr-22 1
B Spring 3 May-22 6
B Summer 2 Jun-22 1
B Summer 2 Aug-22 4
C Fall 1 Nov-21 5
C Fall 1 Sep-22 3
C Fall 2 Nov-21 1
C Fall 2 Sep-22 4
C Fall 3 Nov-21 6
C Fall 3 Sep-22 6
C Fall 4 Nov-21 3
C Fall 5 Nov-21 2
C Fall 6 Nov-21 2
C Winter 1 Dec-21 3
C Winter 1 Jan-22 1
C Winter 1 Feb-22 3
C Winter 2 Dec-21 2
C Winter 2 Jan-22 5
C Winter 2 Feb-22 5
C Winter 3 Dec-21 3
C Winter 3 Jan-22 2
C Winter 3 Feb-22 5
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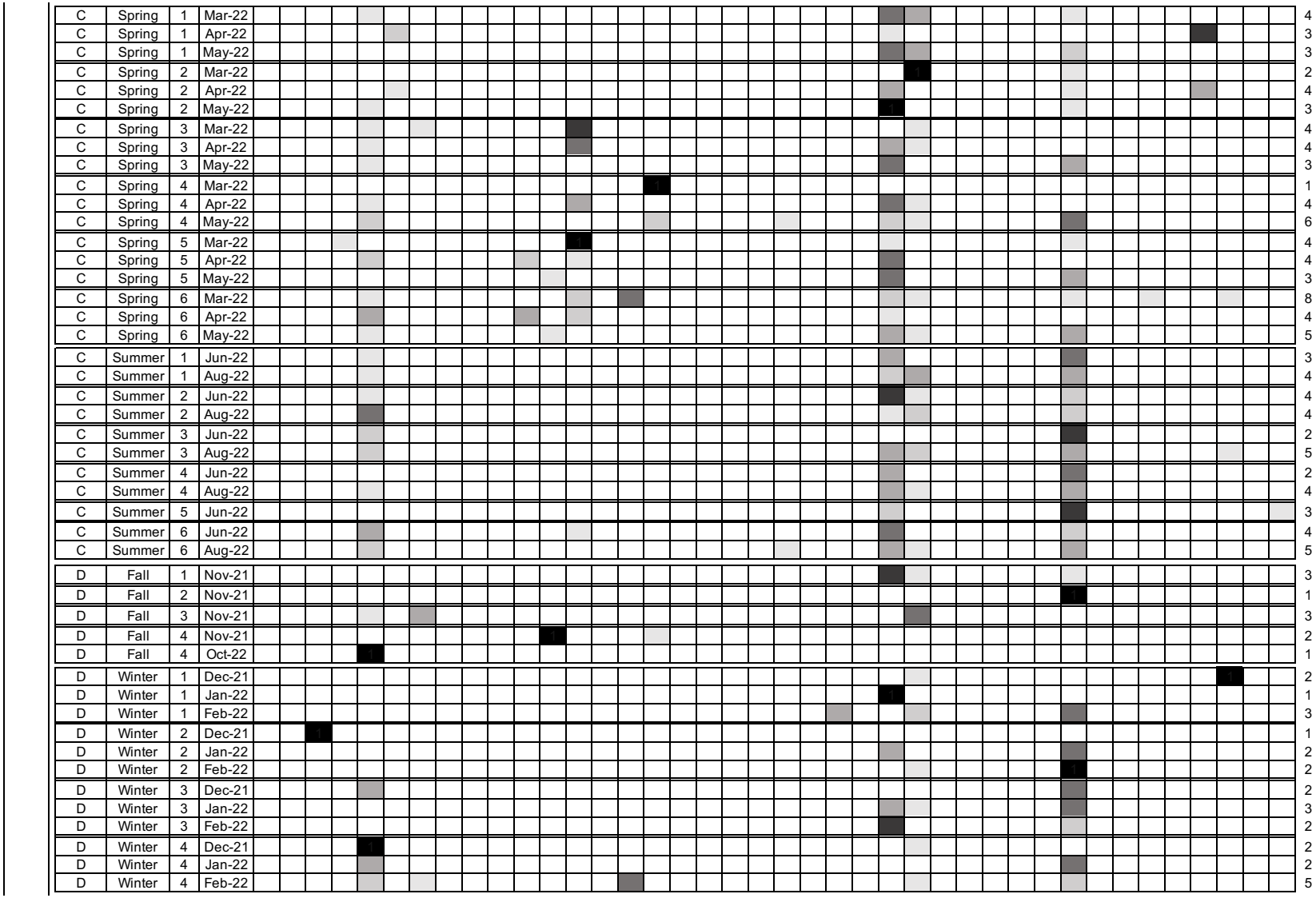

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C	Spring	5	May-22	3
C	Spring	6	Mar-22	8
C	Spring	6	Apr-22	4
C	Spring	6	May-22	5
C	Summer	1	Jun-22	3
C	Summer	1	Aug-22	4
C	Summer	2	Jun-22	4
C	Summer	2	Aug-22	4
C	Summer	3	Jun-22	2
C	Summer	3	Aug-22	5
C	Summer	4	Jun-22	2
C	Summer	4	Aug-22	4
C	Summer	5	Jun-22	3
C	Summer	6	Jun-22	4
C	Summer	6	Aug-22	5
D	Fall	1	Nov-21	3
D	Fall	2	Nov-21	1
D	Fall	3	Nov-21	3
D	Fall	4	Nov-21	2
D	Fall	4	Oct-22	1
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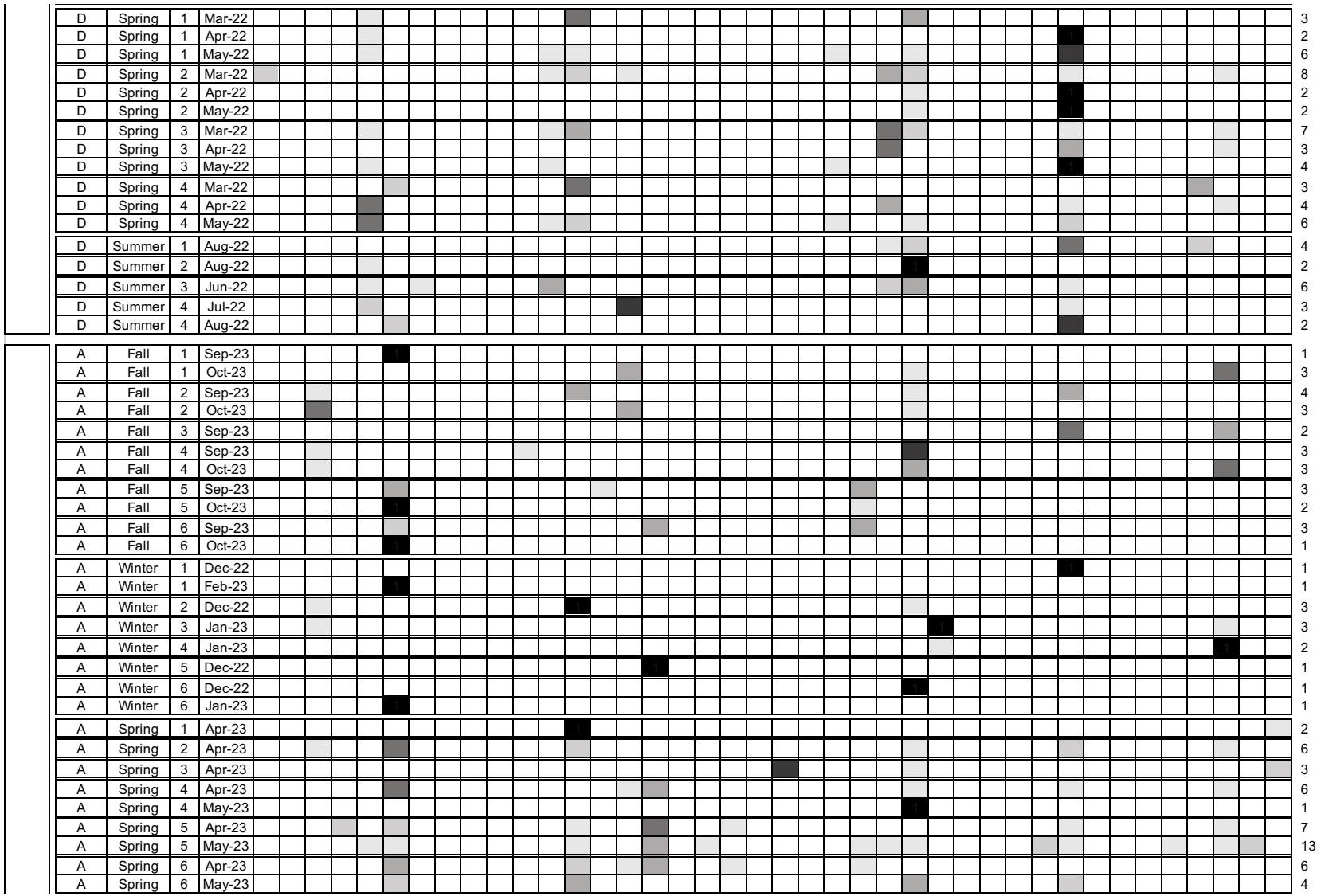
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D Spring 3 Apr-22 3
D Spring 3 May-22 4
D Spring 4 Mar-22 3
D Spring 4 Apr-22 4
D Spring 4 May-22 6
D Summer 1 Aug-22 4
D Summer 2 Aug-22 2
D Summer 3 Jun-22 6
D Summer 4 Jul-22 3
D Summer 4 Aug-22 2
A Fall 1 Sep-23 1
A Fall 1 Oct-23 3
A Fall 2 Sep-23 4
A Fall 2 Oct-23 3
A Fall 3 Sep-23 2
A Fall 4 Sep-23 3
A Fall 4 Oct-23 3
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A Fall 5 Oct-23 2
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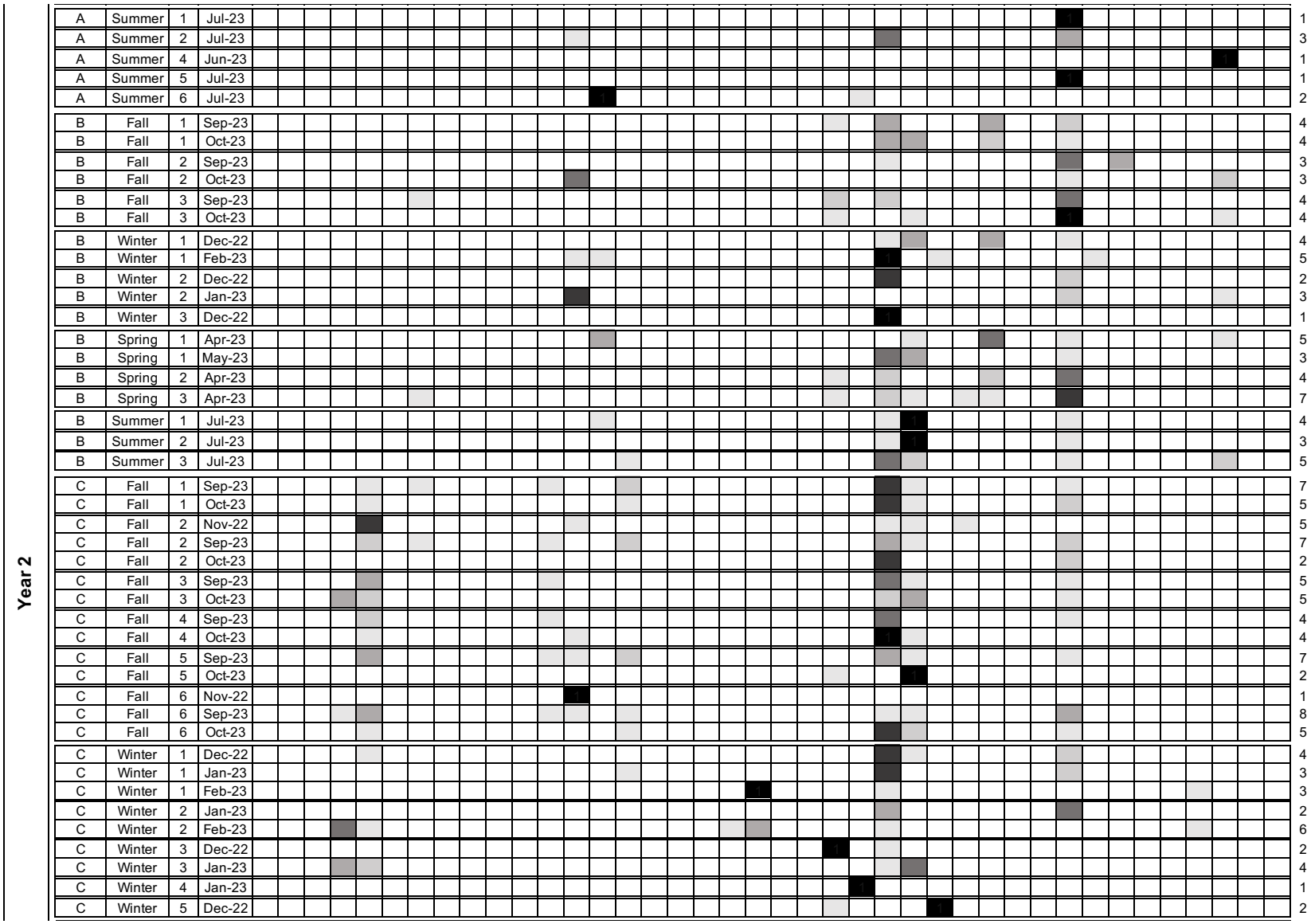
Year 2
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A Summer 2 Jul-23 3
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A Summer 6 Jul-23 2
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B Fall 2 Oct-23 3
B Fall 3 Sep-23 4
B Fall 3 Oct-23 4
B Winter 1 Dec-22 4
B Winter 1 Feb-23 5
B Winter 2 Dec-22 2
B Winter 2 Jan-23 3
B Winter 3 Dec-22 1
B Spring 1 Apr-23 5
B Spring 1 May-23 3
B Spring 2 Apr-23 4
B Spring 3 Apr-23 7
B Summer 1 Jul-23 4
B Summer 2 Jul-23 3
B Summer 3 Jul-23 5
C Fall 1 Sep-23 7
C Fall 1 Oct-23 5
C Fall 2 Nov-22 5
C Fall 2 Sep-23 7
C Fall 2 Oct-23 2
C Fall 3 Sep-23 5
C Fall 3 Oct-23 5
C Fall 4 Sep-23 4
C Fall 4 Oct-23 4
C Fall 5 Sep-23 7
C Fall 5 Oct-23 2
C Fall 6 Nov-22 1
C Fall 6 Sep-23 8
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C Winter 1 Dec-22 4
C Winter 1 Jan-23 3
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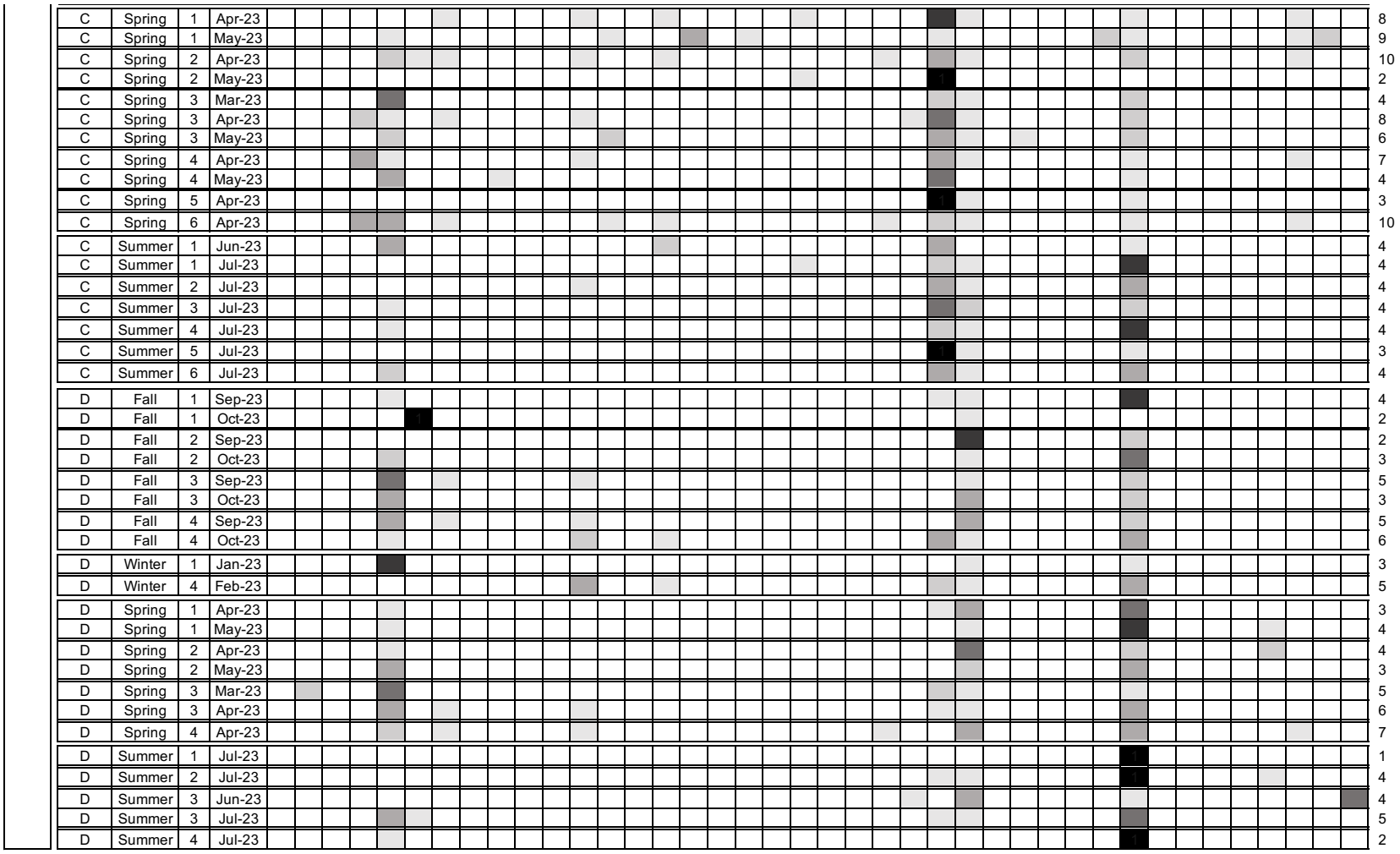

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C	Summer	5	Jul-23																																										3
C	Summer	6	Jul-23																																										4
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D	Fall	3	Sep-23																																										5
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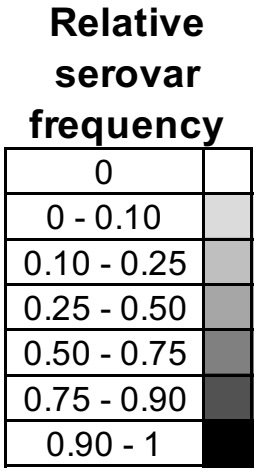
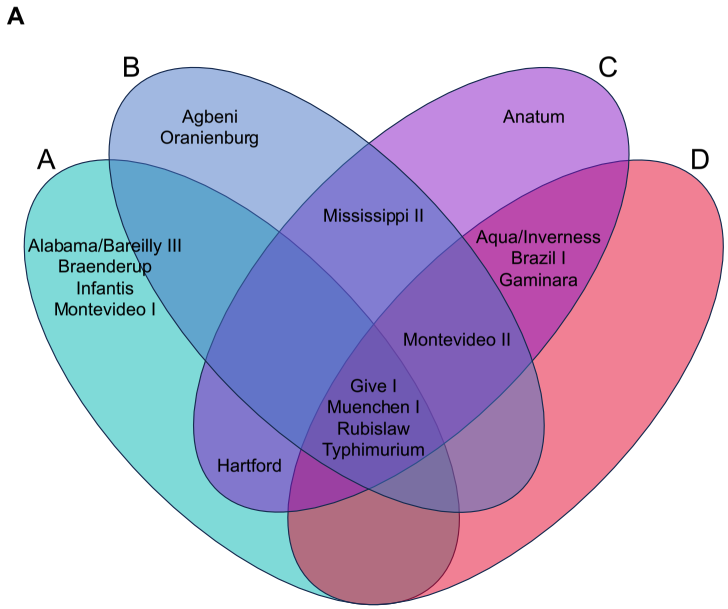
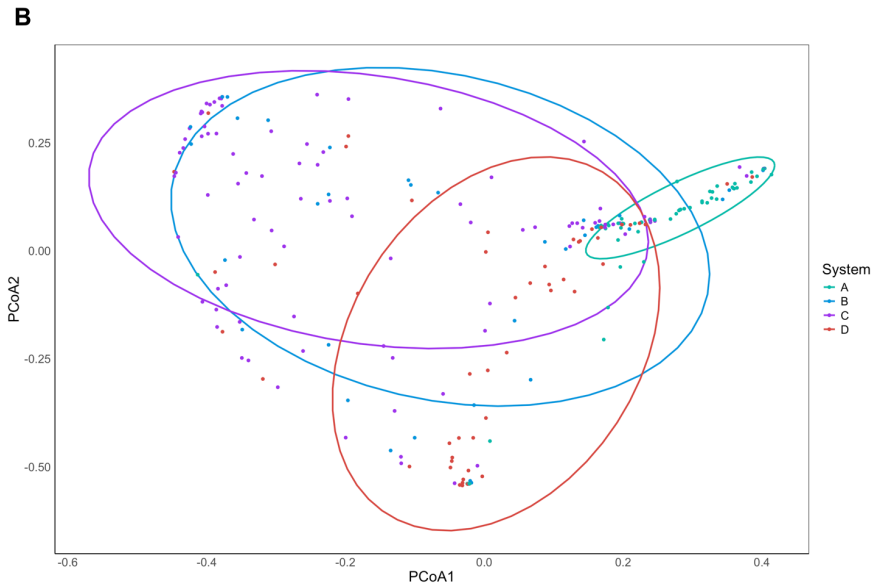
D	Fall	4	Sep-23																																										5
D	Fall	4	Oct-23																																										6
D	Winter	1	Jan-23																																										3
D	Winter	4	Feb-23																																										5
D	Spring	1	Apr-23																																										3
D	Spring	1	May-23																																										4
D	Spring	2	Apr-23																																										4
D	Spring	2	May-23																																										3
D	Spring	3	Mar-23																																										5
D	Spring	3	Apr-23																																										6
D	Spring	4	Apr-23																																										7
D	Summer	1	Jul-23																																										1
D	Summer	2	Jul-23																																										4
D	Summer	3	Jun-23																																										4
D	Summer	3	Jul-23																																										5
D	Summer	4	Jul-23																																										2

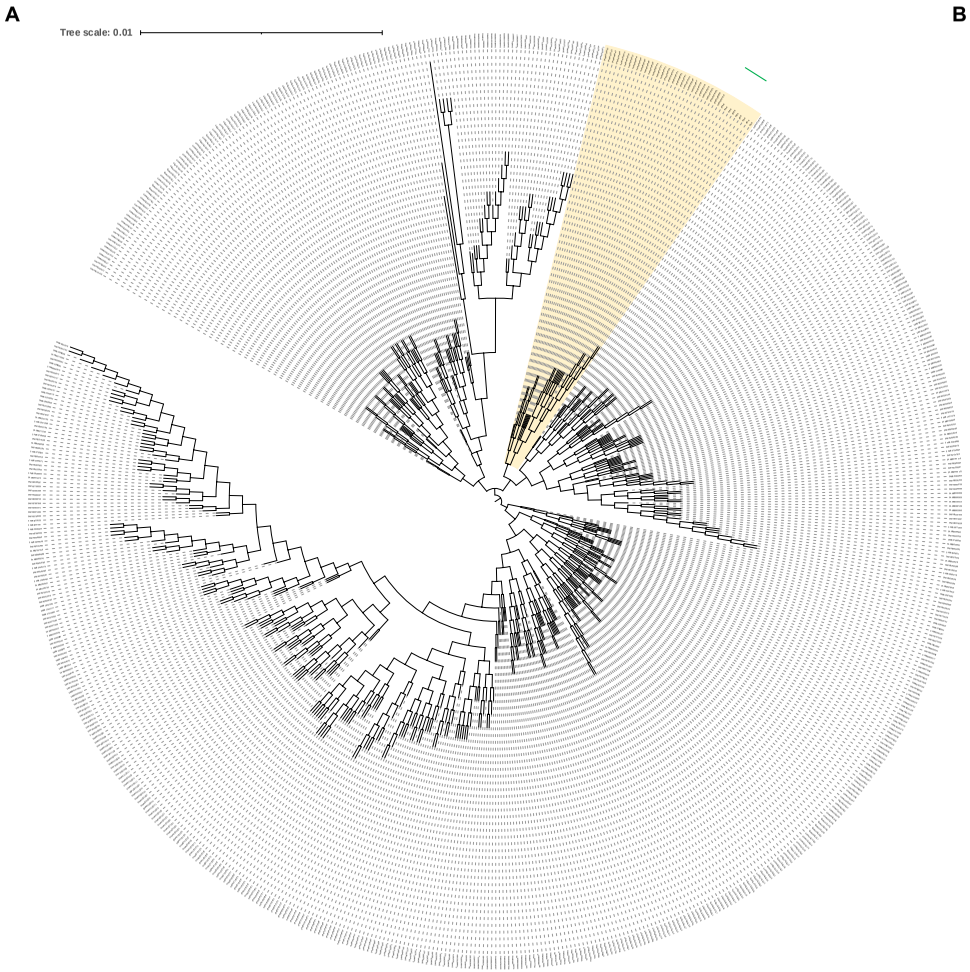


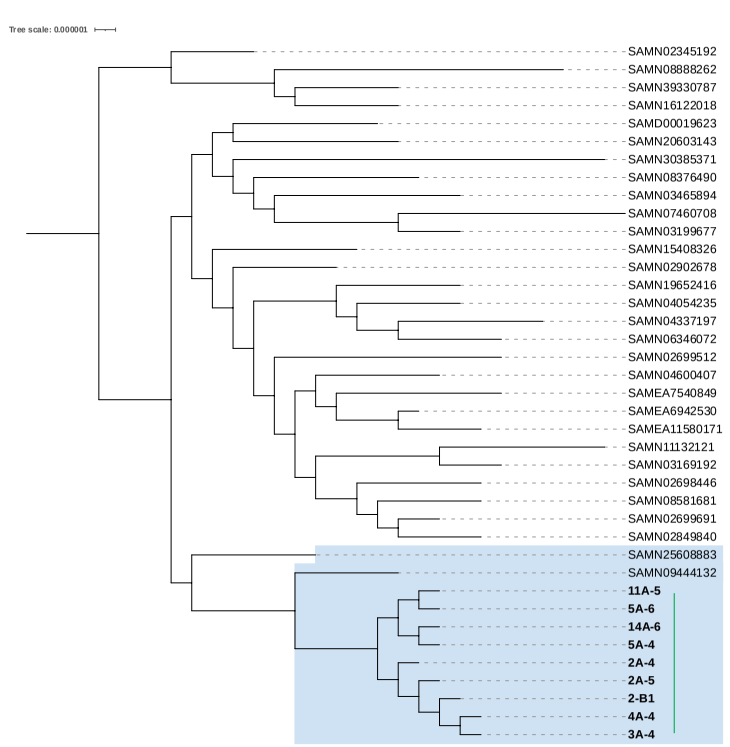
Supplemental Figure 3.4. *Salmonella*-positive water samples contain high serovar diversity. CRISPR-SeroSeq was used to determine the relative abundance of *Salmonella* serovars within each sample, where results from all positive enrichments (BPW, RV, TT) were normalized and combined. Samples are arranged according to the system, season, and site they were collected in, with fall in the first year of the study split between 2021 and 2022. The individual serovars are shown on the top, number of serovars identified per sample included on the right, and the heatmap shows the relative serovar abundance in each sample according to the key. Labels containing two serovars reflects that it is not possible to differentiate based on deep serotyping alone.


A
B
C
A
D
Agbeni
Oranienburg
Anatum
Mississippi II
Alabama/Bareilly III
Braenderup
Infantis
Montevideo I
Aqua/Inverness
Brazil I
Gaminara
Montevideo II
Give I
Muenchen I
Rubislaw
Typhimurium
Hartford

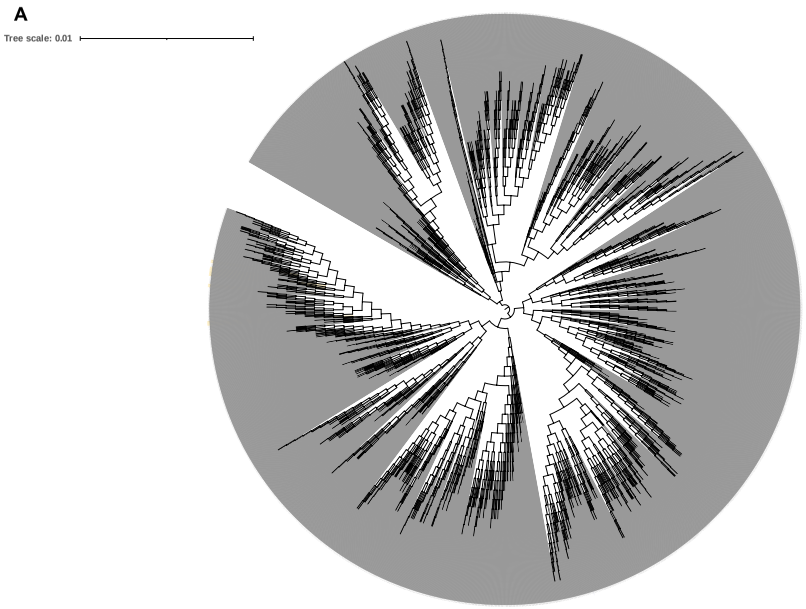

B
0.25
0.00
-0.25
-0.50
PCoA2
-0.6
-0.4
-0.2
0.0
0.2
0.4
PCoA1
System
A
B
C
D

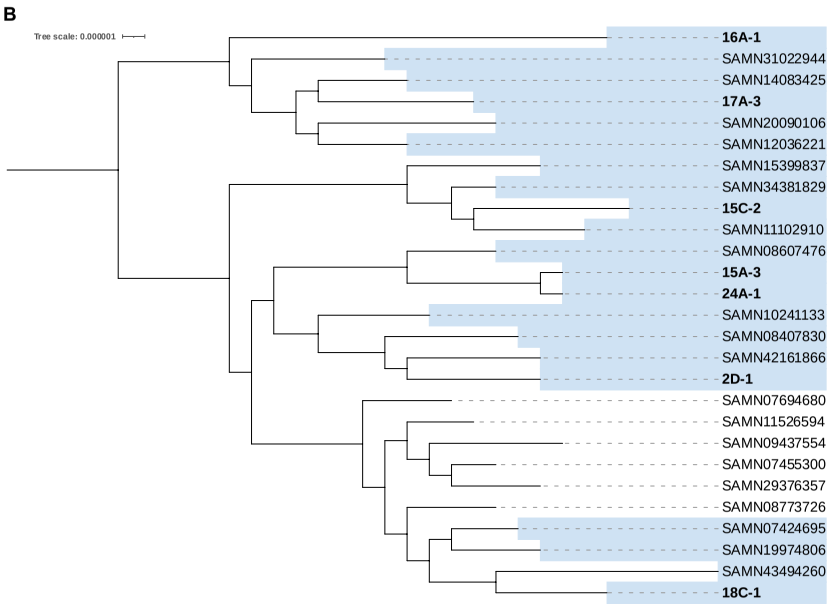
Supplemental Figure 3.5. Distribution of serovars and serovar populations across the creeks. A) Venn diagram displays the overlap of serovars found at least five times in each respective creek. Serovars located only within one ellipse were only found five or more times in that creek but could have been identified four or less times in any other creek. B) Principal component analysis of the Jaccard distance of samples within creeks is shown with ellipses containing 75% of each creek's samples. A larger ellipse indicates a more variable population, while a smaller ellipse indicates higher consistency.


A
Tree scale: 0.01


B
Tree scale: 0.000001
SAMN02345192
SAMN08888262
SAMN39330787
SAMN16122018
SAMD00019623
SAMN20603143
SAMN30385371
SAMN08376490
SAMN03465894
SAMN07460708
SAMN03199677
SAMN15408326
SAMN02902678
SAMN19652416
SAMN04054235
SAMN04337197
SAMN06346072
SAMN02699512
SAMN04600407
SAMEA7540849
SAMEA6942530
SAMEA11580171
SAMN11132121
SAMN03169192
SAMN02698446
SAMN08581681
SAMN02699691
SAMN02849840
SAMN25608883
SAMN09444132
11A-5
5A-6
14A-6
5A-4
2A-4
2A-5
2-B1
4A-4
3A-4

Supplemental Figure 3.6. Preliminary serovar Infantis phylogenies including nine isolates from this study and publicly available genomes from NCBI. Study isolates are indicated with a green bar, with the label format of month-creek-site, and NCBI isolates are listed by sample ID. The phylogenies are rooted at the midpoint. A) Phylogeny includes one representative isolate from each available SNP cluster belonging to serovar Infantis on NCBI (n = 650). Yellow highlight indicates clade selection for subsequent phylogeny. B) Phylogeny includes one representative isolate from the most closely related SNP clusters (n = 30). Blue highlight indicates clade selection for subsequent phylogeny.


A
Tree scale: 0.01


B
Tree scale: 0.000001
16A-1
SAMN31022944
SAMN14083425
17A-3
SAMN20090106
SAMN12036221
SAMN15399837
SAMN34381829
15C-2
SAMN11102910
SAMN08607476
15A-3
24A-1
SAMN10241133
SAMN08407830
SAMN42161866
2D-1
SAMN07694680
SAMN11526594
SAMN09437554
SAMN07455300
SAMN29376357
SAMN08773726
SAMN07424695
SAMN19974806
SAMN43494260
18C-1

Supplemental Figure 3.7. Preliminary serovar Typhimurium phylogenies including seven isolates from this study and publicly available genomes from NCBI. Study isolates are bolded, with the label format of month-creek-site, and NCBI isolates are listed by sample ID. The phylogenies are rooted at the midpoint. A) Phylogeny includes one representative isolate from each available SNP cluster belonging to serovar Typhimurium on NCBI (n = 3,057). Yellow highlight indicates clade selection for subsequent phylogeny. B) Phylogeny includes one representative isolate from the most closely related SNP clusters (n = 20). Blue highlight indicates clade selection for subsequent phylogeny.