FAST BUT RIGHT: OUTBREAK SURVEILLANCE AND FOODBORNE KNOWLEDGE INFRASTRUCTURE

A Dissertation
Presented to the Faculty of the Graduate School of Cornell University in Partial Fulfillment of the Requirements for the Degree of Doctor of Philosophy

by
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This dissertation examines knowledge infrastructures for detecting and investigating national outbreaks of foodborne disease. Drawing on archival and ethnographic material from US public health and regulatory agencies, I investigate how officials have built and used surveillance systems to make foodborne outbreaks visible, reflecting the shape of the industrialized food supply. I describe how, in the course of conducting outbreak investigation work, officials confront the challenges of a “balancing act” of needing to be fast but right, facing dilemmas associated with wanting to protect the public health yet minimize economic impact to commercial entities, while grappling with the highly distributed nature of both the food system and a federalist system of public health governance.

In the dissertation, I make three core arguments. First, during foodborne outbreak investigations, public health and regulatory officials manage time and uncertainty through systematization. Second, systematization has helped make visible a new kind of public health problem, rooted in the post World War II industrialization of the US food supply—national, diffuse outbreaks caused by contaminated food moving through interstate commerce. Third, despite the importance of and emphasis on systematization in this domain, the numerous and persistent challenges associated with needing to be fast but right preserves a need for expert judgment amidst formal systematization efforts.
In addition to examining broader public health infrastructure, the dissertation features analyses of two surveillance systems for foodborne disease: an historical examination of the National Salmonella Surveillance Program from 1962-1976, and an historical and ethnographic study of the current early-warning, real-time system based on molecular subtyping. Through these analyses, I demonstrate how these systems made outbreaks visible not only from a technical perspective, but also from social, political, and economic perspectives as well.
BIOGRAPHICAL SKETCH

Angie Boyce grew up in Bovill, Idaho. From 1999-2003, she attended Harvard College. There, she pursued an AB degree in History and Science, and completed an interdisciplinary certificate program in Mind, Brain and Behavior. She graduated magna cum laude, and received the Thomas T. Hoopes Prize for her undergraduate thesis on the history of the biology of aggression. Prior to entering the Science and Technology Studies Ph.D. program at Cornell University in 2008, she conducted research at the Boston Museum of Science, on creating effective programs for public engagement with science and technology in museum settings; and at the Stanford Center for Biomedical Ethics, on developing institutional mechanisms for increasing social accountability and ethical reflection in the life sciences. While at Cornell University, she discovered her enduring interests in food, public health, regulation, and infrastructure. During graduate school, she was awarded a National Science Foundation Graduate Research Fellowship, completed an internship at the Food and Drug Administration, and served as a visiting student researcher at the Centers for Disease Control and Prevention. In 2011, she received the department’s Jasanoff Prize for best graduate student paper, for her paper on food standards and consumer activism. She is publishing this research in a forthcoming article in Technology and Culture, and has also had a co-authored publication on microbiome science accepted in Science, Technology and Human Values. Following the completion of her Ph.D., Angie will begin a position as a Robert Wood Johnson Health & Society Scholar at Harvard University (2014-2016).
ACKNOWLEDGEMENTS

The dissertation I ended up writing is a much different one than I had initially envisioned, and has been vastly enriched by the opportunity learn so much from the public health and regulatory officials who stand watch over our food. I am grateful to each and every one of the numerous professionals at the federal, state, and local levels who have shared their thoughts and experiences with me. I would have to write a separate appendix to acknowledge everyone, which would be quite fitting as a reflection of just how many people, from many different places, have to work together to solve outbreaks and put all of the heterogeneous puzzle pieces together.

This project would not be what it is without the wisdom of Robert Tauxe; without his openness and reflexivity this study would not have been possible. The robust system of early warning outbreak detection chronicled here would not be as it is without Bala Swaminathan’s vision and work to build PulseNet; Efrain Ribot in keeping it running; and John Besser, Peter Gerner-Smidt, and Eija Trees in ensuring its future. Ian Williams and Casey Barton-Behravesh, head puzzle-solvers, were generous with their time and access to investigations, helping me understand their “disease detective” work. Patricia Griffin helped me gain historical perspective, especially on the interfaces between public health, medical research, and consumer advocacy communities. All of these leaders were not only generous with their own time and thoughts, but graciously allowed me to hang out with many of the top-notch scientists in their branches and units shadowing work and meetings, and I owe each and every one of these staff members many, many thanks (particularly the PulseNet lab and database teams, and the MLVA team). Susan Lance helped me see the importance of the theme of the “grey area,” and I admire her and all the other inter-agency liaisons who try to make the public health-regulatory interface smoother, a perennial challenge. To Molly Freeman in particular, thank you for being a wonderful friend and
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<th>Description</th>
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<tbody>
<tr>
<td>AES</td>
<td>Agricultural Experiment Station</td>
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<td>AMS</td>
<td>Agricultural Marketing Service</td>
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<td>APHA</td>
<td>American Public Health Association</td>
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<td>APHIS</td>
<td>Animal and Plant Health Inspection Service</td>
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<td>APHL</td>
<td>American Public Health Laboratories</td>
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<td>BAI</td>
<td>Bureau of Animal Industry</td>
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<td>CDC</td>
<td>Centers for Disease Control and Prevention</td>
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<td>EBLU</td>
<td>Enteric Bacteriology Laboratory Unit</td>
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<td>EDEB</td>
<td>Enteric Diseases Epidemiology Branch</td>
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<td>EDLB</td>
<td>Enteric Diseases Laboratory Branch</td>
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<td>EIS</td>
<td>Epidemic Intelligence Service</td>
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<td>ELC</td>
<td>Epidemiology and Laboratory Capacity for Infectious Diseases Cooperative Agreement</td>
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<td>FDA</td>
<td>Food and Drug Administration</td>
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<td>FDDB</td>
<td>Foodborne and Diarrheal Diseases Branch</td>
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<td>FMIA</td>
<td>Federal Meat Inspection Act</td>
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<td>FoodNet</td>
<td>Foodborne Diseases Active Surveillance Network</td>
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<td>EPA</td>
<td>Environmental Protection Agency</td>
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<td>FSIS</td>
<td>Food Safety Inspection Service</td>
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<td>FSMA</td>
<td>Food Safety Modernization Act</td>
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<tr>
<td>HACCP</td>
<td>Hazard Analysis and Critical Control Point</td>
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<td>HIPAA</td>
<td>Health Insurance Portability and Accountability Act</td>
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<td>Acronym</td>
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<tr>
<td>HUS</td>
<td>Hemolytic Uremic Syndrome</td>
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<td>INDM</td>
<td>Instant Non-fat Dried Milk</td>
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<td>MLVA</td>
<td>Multiple Locus Variable-number Tandem Repeat Analysis</td>
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<td>MSPB</td>
<td>Meningitis and Special Pathogens Branch</td>
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<tr>
<td>NASA</td>
<td>National Aeronautics and Space Administration</td>
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<td>NGSMDP</td>
<td>Next Generation Sequencing and Methods Development Program</td>
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<td>NORS</td>
<td>National Outbreak Reporting System</td>
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<td>NRC</td>
<td>National Research Council</td>
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<td>NSSP</td>
<td>National Salmonella Surveillance Program</td>
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<td>ORPB</td>
<td>Outbreak Response and Prevention Branch</td>
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<tr>
<td>OutbreakNet</td>
<td>A collaborative network of federal, state, and local public health officials involved in the detection and surveillance of, and response to, outbreaks of foodborne, waterborne, and other intestinal tract diseases.</td>
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<tr>
<td>PCA</td>
<td>Peanut Corporation of America</td>
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<td>PFGE</td>
<td>Pulsed Field Gel Electrophoresis</td>
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<tr>
<td>PHI</td>
<td>Protected Health Information</td>
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<td>PHS</td>
<td>Public Health Service</td>
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<td>PPIA</td>
<td>Poultry Products Inspection Act</td>
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<td>PTI</td>
<td>Produce Traceability Initiative</td>
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<tr>
<td>PulseNet</td>
<td>The national molecular subtyping network for foodborne disease surveillance</td>
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<tr>
<td>SEDRIC</td>
<td>System for Enteric Disease Response, Investigation, and Coordination</td>
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<tr>
<td>SMAC</td>
<td>Sorbitol Mac Conkey Agar</td>
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STOP    Safe Tables Our Priority

TTP    Thrombotic thrombocytopenic purpura

USDA    US Department of Agriculture
Prologue: From Everyday Life to Medical Crisis

Linda and I met at a café, and sat in a quiet corner over lattés.¹ Our mutual friend, who knows that I research foodborne disease, had mentioned that Linda’s daughter Sophia had been exposed to *E. coli* O157:H7, and developed hemolytic uremic syndrome (HUS), a serious illness that causes kidney failure and is often life-threatening, especially in young children. Though public health officials never identified what made Sophia sick, it was likely from a contaminated food, as food is the main way that people get exposed to *E. coli* O157:H7. Our mutual friend suggested that I speak with Linda, and Linda kindly agreed to share her story with me. We decided to meet in the morning, before a lunch date we had arranged with our mutual friend.

“When did you notice something was wrong?” I asked her. Until the end of the story, I did not have to ask her much more.

It was Christmas. Sophia was three years old. The young child was complaining about abdominal pain, and then developed diarrhea. Since Linda is an emergency doctor, she knew that toddlers often develop diarrhea, and it’s rarely a big deal. That was Sunday night. On Monday, Sophia was up all night. Linda would second-guess herself later, about her Sunday night assessment of Sophia’s condition. Blood appeared in Sophia’s diarrhea. Now, this seemed like a problem. Linda took Sophia to the ER.

This was such a big thing in their lives. Linda remembers the tiniest of details. They drew fluids and blood from Sophia (Linda could remember the specific volumes), and performed several ultrasounds. They had to move from one ER to a different ER for children. It was a “long parade” of occurrences. The next day, she found out that her daughter’s illness was caused

¹ Interview 3/8/2013.
by a shiga-toxin producing pathogen. That’s what she was told, and so initially she thought the pathogen was *Shigella*. She started pulling up medical papers on *Shigella*, and wondered how in the world Sophia got *Shigella*.

Twelve hours later they told her the pathogen was *E. coli* O157:H7. Over the next 48 hours, more clinical information started to filter in, Sophia’s white blood cell and platelet counts. Linda told me that she didn’t understand how bad the situation was at the time. Her husband was away on a trip, and she had been continuously debating whether to tell him to fly home, as Sophia could recover at any moment.

However, at that point, Sophia started to go “crazy” from the effects of the bacterial toxin in her body. She needed a blood transfusion. Linda fought hard to make sure that it came from a single donor and was not pooled blood, as pooled blood poses greater risk of transfusion-transmitted disease. Linda talked about feeling that she could control the situation through fighting for single donor blood. It was Thursday. She didn’t know what bad would look like.

The doctors measured Sophia’s creatine levels. They were at 6 or 7, an incredibly high number that meant that she would require dialysis. Then, Linda found out that the blood transfusion hadn’t gone well. After the procedure, the doctors couldn’t wake Sophia up from the anesthesia; during the transfusion, Sophia had stopped breathing. To revive her, the doctors had performed CPR. Linda was extremely upset upon hearing this. She explained that this upset her because many doctors hate CPR, the success rate is low and the risk of brain damage is high.

While Sophia was still unconscious, the doctors took an MRI. When the results of the MRI came in, there were possible “stroke like changes” indicated in the image, but the neurologist told Linda that this was uncertain, and it might be nothing at all.
Linda looked up a relevant medical study on 20 children. The study had identified different predictors associated with different outcomes. With certain predictors, 3-4 children died, and with other predictors, 3-4 were disabled. Sophia did not fit either of the two predictor categories—maybe she dodged a bullet. Sophia was unconscious for 12 hours, and then when she woke up, she needed dialysis again.

After this crisis within a crisis, Sophia and Linda rested; Linda fell asleep in the chair next to Sophia’s hospital bed. However, during their nap, Sophia’s catheter fell out. It hadn’t been secured well by the nurse. When Linda opened her eyes, she saw that Sophia had bled all over the bed, a scene she still sees in her nightmares. Sophia needed dialysis over and over again, and then the doctors put her on an oscillating ventilator. At that point, Linda had lost the control she was trying to maintain, as a doctor herself, trying to evaluate the decisions of the clinicians treating her daughter. She finally gave up the control she had been fighting for, and just let the other doctors handle the emergency.

Luckily, that ended up being the peak of the crisis. Sophia began to recover. The doctors administered Sophia a new drug (eculizimab) that usually used for atypical HUS, a genetic condition. However, the doctors thought that the drug might work for typical HUS (bacterial infection), based on a paper written by German clinicians who had treated HUS patients from a 2011 *E.coli* O104:H4 outbreak in Europe that involved more than 4000 cases in 16 countries. At $24,000-30,000 per dose, it was then the most expensive drug in America. Linda and her husband weren’t sure that their insurance company would cover the cost of the drug, but said to go ahead and give it to Sophia, they could pull out their 401Ks if they had to. As Sophia got better, and eventually went off of dialysis, Linda remembers what a miracle it was for Sophia to use the bathroom for the first time, for her kidneys to “wake up.” And thankfully, Sophia is a
“shiny penny,” and so far, there have been no signs that her *E.coli* infection has had any lasting effects.
Chapter 1: Introduction

Fast

Every person who develops a foodborne illness has their own unique story, though thankfully, not all of the stories are as traumatic as Linda and Sophia’s. In fact, one of the characteristics making foodborne illness a particularly interesting disease to study is that its range of symptoms is so wide. Many foodborne illness “cases” (public health term for ill persons) never reach the view of clinical professionals, let alone public health authorities, because sufferers experience mild and acute symptoms like upset stomach or diarrhea. They typically recover quickly, and so they do not seek medical care. Indeed, I myself suspect that I have had a foodborne illness at least three or four times in my life, though I never sought treatment for any of those instances. People like me are what public health officials call “unrecognized” or “under-reported” cases. While the Centers for Disease Control and Prevention (CDC) counted 19,531 cases of foodborne infection (confirmed with a laboratory diagnosis) in 2012, the agency estimates that each year, roughly 1 in 6 Americans (48 million people) get sick from a foodborne illness.¹

But other cases, like Sophia, experience severe and even life-threatening illnesses, such as hemolytic uremic syndrome. A subset of those cases experience chronic complications such as kidney failure; diabetes; and autoimmune, cardiac, and neurological disorders. The CDC estimates that annually, around 3,000 people die from foodborne disease. Consumer advocate Carol Tucker Foreman argued that one of the reasons it had been historically difficult to collect good national statistics on foodborne illness is that many thought of salmonellosis as “just a

bellyache.”² Foreman pointed to a 1993 outbreak of *E. coli* O157:H7 in Jack-in-the-Box restaurants as seminal in changing perceptions of the seriousness of foodborne disease, “when suddenly you had children dying terrible deaths” from eating hamburgers.

With the goal of helping to prevent illnesses like Sophia’s, in the 1990s, US public health officials at the CDC began building a real-time, early warning foodborne outbreak detection system, comprised of standardized molecular DNA fingerprinting techniques and a computer network consisting of members at the state level (public health laboratories and departments) and the federal level (CDC, Food and Drug Administration [FDA], and US Department of Agriculture [USDA]). Members of the network would work together to detect and solve potential national outbreaks of foodborne disease, with the goal of finding outbreaks as early as possible, and end them before they grew larger by finding the contaminated food causing the illnesses.

A key goal of the early warning detection system, which federal officials called PulseNet, was to use molecular methods of DNA fingerprinting to connect individual cases spread across the nation. In making these connections, officials could show how the shape of outbreaks mirrored the wide distribution of (contaminated) food in interstate commerce. They could demonstrate how a person from Los Angeles, California and a person from Providence, Rhode Island, though they live thousands of miles apart, could have eaten a common food contaminated with the same bacterial pathogen, and thus be members of the same outbreak. Figure 1 is a diagram of the network from 2002.

Builders of the national molecular computer network stated that PulseNet’s goal was to help identify national outbreaks earlier and faster, so that these emergencies could be stopped, and future illnesses prevented. The system’s founder, Bala Swaminathan, attributes PulseNet’s success to a vision articulated in “simple terms” – “PulseNet Saves Lives.” While Sophia’s illness was not linked to an outbreak (the public health term for a non-outbreak associated case is a “sporadic” case), a DNA fingerprint from the bacteria that made Sophia sick lives in the PulseNet database, along with a few data points about her, such as her age, sex, county of residence, and date that the bacteria that caused her illness was transformed into an “isolate”—a single, purified bacterial kind. Though Sophia’s bacterial DNA fingerprint is not likely to be

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4 Ibid.
part of an identified outbreak in the future, because years have elapsed since her original illness, her associated case data may provide hints or clues useful for future outbreak investigations.

It is not difficult to understand the importance of detecting national outbreaks faster so that young children like Sophia are sickened less often. However, what does “faster” mean in practice? How fast is the system? How and why was outbreak detection “slow” prior to the establishment of the computer network? Why can’t the system be faster than it is? All of these questions are about time. Intense management of time is central to the early warning system, and in fact, central to food safety. “Time is everything with food safety,” proclaims a biotech marketing campaign for new 1-day pathogen detection kits, boasting that it provides much more rapid results than “traditional” tests which take up to four days.\(^5\)

To illustrate how and why time matters so much in the real-time, early warning system, here I make a central point animating this dissertation, repeating what Robert Tauxe, the CDC’s Deputy Director for the Division of Foodborne, Waterborne, and Environmental Diseases emphasized to me in an early interview; many outbreaks do not “occur” unless they are detected.\(^6\) To understand this point, it is useful to think of an outbreak in the following manner. Individual illnesses, like Sophia’s, are individual occurrences. Without some sort of method of discernment to draw individual illnesses together as an outbreak, they “exist” as unconnected incidents. Prior to the establishment of the early warning system, many of these unconnected incidents were never discerned. The “traditional” method of discerning problems, which still exists, relied upon an alert physician happening to notice a group of unusual illnesses, triggering an investigation. In the traditional method, the single case in Rhode Island and the single case in California would never be drawn together as part of the same event.

\(^6\) Informal communication.
I call unconnected incidents an *event of disease occurrence*. When public health officials detect and investigate outbreaks, and turn materials into data and information, during the process, they represent the event of disease occurrence, and turn the outbreak into an *event of public health detection*. While they conduct outbreak investigations, officials collect heterogeneous materials about the event of disease occurrence, gathering evidence from humans, foods, localities, companies, and animals. Officials often use the simple metaphor of “connecting the dots” to explain the work involved in outbreak investigations. I borrow this metaphor to point out that outbreak investigations involve finding stuff, turning that stuff into dots, and then connecting those dots together as an outbreak, identifying interconnected relationships of disease transmission. As they connect the dots, officials turn the event of disease occurrence into the event of public health detection.

The event of public health detection aims to represent the event of disease occurrence as closely as possible, but one reason why the two events remain distinct is that there is a built-in time lag between an outbreak occurring “out there” and the machinery officials use to detect and investigate the outbreak. A historical joke at the CDC helps illuminate this point further. Federal epidemiologists used to speak of “sliding down the epi curve to glory,” because they would usually arrive in the field to assist local or state officials with investigations, outbreaks had almost finished running their course; however, federal officials would receive “glory” for taking public health actions to end the outbreak.⁷

What is interesting to consider is that, in the present moment, this old joke is not as resonant. The early warning system has significantly shortened the time lag between the two events. In fact, during the course of an outbreak investigation, if it is a successful one, these two

⁷ Pendergrast (2010).
events converge, when enough evidence has been collected to warrant the taking of public health actions to intervene upon, and stop the outbreak.

This is why public health officials call the current outbreak detection system a “real-time” one, because by using the system, they are able to not only represent the outbreak, but intervene on it as it occurs in the world. A comment that a county environmental health specialist made to me is also relevant to this “real-time” discussion.⁸ Hedging its plausibility, he stated that a “Star Wars,” science-fiction vision of a far-off future was that outbreak investigations would involve metagenomic analysis of a sample from the content of a person’s stomach identifying not only the disease causing agent but perhaps even the causative food, or a test providing instant genomic information about bacteria in contaminated food samples. While these futuristic technologies would indeed be even closer to a “true” real-time, officials feel that the current system of representing outbreaks gets them close enough to real-time to intervene on outbreaks, saving lives in the process. Time is a key life-saving currency in foodborne illness; saving time in outbreak investigations means saving lives.

…But Right

Outside of the German consulate in Valencia, Spain, on an afternoon in June 2011, Spanish produce farmers dumped 700 pounds of fruits and vegetables outside of the building’s entrance.⁹ The farmers were protesting a public health hazard announcement made by the German government, associated with a major, multi-country foodborne outbreak of *E.coli* O104:H4, involving what would eventually be confirmed as 3,816 cases of illness and 54 deaths

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⁸ Informal communication.
across Europe. The Spanish farmers were engaging in protest because the German government had issued warnings to consumers not to eat Spanish cucumbers, then lettuce, and tomatoes. Later, German officials had to retract their initial advice. Investigators eventually determined that the causative food “vehicle” (public health term for a food carrying bacteria) of the outbreak was fenugreek sprouts grown by an organic farm in the northern German region of Lower Saxony, and whose sprout seeds were eventually traced to Egypt where the initial contamination was thought to have occurred.

The initial warning issued by the German government had massive effects on the European trade of produce, and in particular, cucumbers from Spain. As an illustration of the cascading effects, in the middle of the outbreak, Russia banned all produce imports from Germany and Spain; the Czech Republic and France removed Spanish cucumbers from their national marketplaces; and Austria and Belgium restricted imports on Spanish and German cucumbers. After the outbreak was over, the European Commission eventually decided to compensate European vegetable growers 227 million Euros, a small percentage of the total estimate of economic losses.

As this example demonstrates, money is another currency in foodborne illness. Saving lives means removing contaminated food from the marketplace, which ultimately costs money. The Spanish produce farmers were angry because their wares were wrongly impugned as contaminated. During the outbreak, thousands of cases accumulated across Europe as public health officials actively pursued finding them, and in northern Germany, so many patients developed HUS, the region’s medical systems experienced shortages of blood and dialysis machines for patients with failing kidneys.

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10 Frank et. al (2011)
11 Willis (2011)
A German media publication, *The National*, criticized the German government’s “sluggish response” to the crisis, blaming its fragmented federal structure, and lack of coordination and centralization to overcome political fragmentation slowing the collation of information.\(^1\) “The German epidemic response system is slower than other developed nations such as the United States and Japan,” the article commented. The latter nations, the article pointed out, had “set up early warning mechanisms to help speed up the response to outbreaks such as *E. coli* infections.” The friction between the currencies of time and money, with lives and economies on the line, without a coordinated system to mediate that friction, helped to produce a major continental crisis.

One way that US public health and regulatory officials describe this friction in their work is as a “balancing act.”\(^1\) What is being balanced? John Guzewich, a former FDA official, coined a “pet phrase”\(^1\) (which I reference in the title of the dissertation) to explain how this balancing act feels to the officials performing it – “we have to be fast and we have to be right.” “That’s the dilemma,” he explained, a quandary present in “every investigation.” During investigations, officials feel a “tremendous sense of urgency,” he pointed out; protecting the public health and doing a “good job” were linked, “you [officials] want to get there as fast as you possibly can.” However, as he pointed out, being fast but wrong has huge economic impacts, as well as implications for professional standing. “The amount of credibility lost in these things is amazing,” he observed.

Officials have to be fast, because people’s health and sometimes even their lives are on the line, and the sooner they can identify the contaminated food vehicle, the sooner they can


\(^{14}\) Interview, Ian Williams, 3/1/2012.

\(^{15}\) Interview, John Guzewich, 10/30/2013.
remove it from the marketplace. The longer outbreaks remain unsolved, the longer contaminated food can remain in the marketplace, and sometimes in consumer cupboards and refrigerators, sickening more people, especially if those foods have a long shelf-life.

But officials need to be right, as the announcement of contaminated products has vast economic consequences for individual commercial entities, sectors, and at times, entire industries, as the European fenugreek sprout outbreak, and many others that will be discussed in this dissertation, demonstrate. In negotiating their balancing act, officials are interested in maintaining their status as credible and trusted public health advisors. The phrase “fast but right” emphasizes that the stakes are high—foodborne outbreaks are a meeting place for confluent tensions between protecting the public health and having an economic impact on food production. In a real-time, early warning system, officials must intensely manage time and uncertainty.

The metaphor of the balancing act is often illustrated with a two-sided scale. To achieve balance between the two sides, an equal amount of weight must be placed on each. The two sides are interdependent; if one side is weighed down too heavily, the other side rises up in response. Applying this to the fast but right balancing act, if being fast is weighed too heavily, it can come at the expense of being right. However, if being right is weighed too heavily, it can come at the expense of being fast. Hanging in the balance are people’s health and lives, professional credibility, economic stakes, commercial reputations, and political legitimacy.

Complicating the balancing act is the fact that national outbreaks are highly distributed phenomena in many dimensions: space, time, jurisdiction, sector, and level, just to name a few. What cannot be overstated is the importance of understanding that foodborne outbreak investigation work is thus distributed as well, requiring the participation, collaboration, and
coordination of dozens or even hundreds of workers across multiple locales, jurisdictions, and agencies, with different disciplinary backgrounds and roles. Because of this distributed nature, investigations typically involve grappling with an array of (sometimes differing or conflicting) municipal, county, state and federal legal constraints on collecting and sharing patient health data and confidential commercial information. Additionally, as is the case with any form of widely distributed work with multiple workers from different organizations responsible for different pieces of a larger picture, maintaining the accuracy and coherence of information takes ongoing, careful, and active work. The children’s game of “telephone” is a clear example of how information is at risk of being corrupted as it passes through many ears.

Outbreaks are time-sensitive not only because of risk to human health, but also because of the ephemerality of evidence: varying shelf-life of foods, varying survival of pathogens, variations in food sourcing and distribution, and limits on length of patient memory of consumed foods. Evidence can be not only ephemeral, but is at times ambiguous; in describing some of the complexities involved in making judgments during epidemiologic field investigations, CDC official Richard Goodman and colleagues cite epidemiologist George Comstock, who said, “The art of epidemiologic reasoning is to draw sensible conclusions from imperfect data.”

This dissertation makes three core, overarching arguments. First, it argues that during foodborne outbreak investigations, public health and regulatory officials manage time and uncertainty through systematization, a term I use to encompass standardization, classification, and ordering practices, as well as the building of formal systems. It is important to use an encompassing term because I am interested not only in formal systems and the artifacts which

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16 Goodman et al. (1990).
17 Martha Lampland prefers the catch-all phrase “formalizing practices,” (2010) to encompass quantification, standardization, and formalization. In my context, I prefer the term systematization, because formal systems are at the heart of what I study, yet I am trying to encompass something broader about the dynamic work people conduct to make systems work.
comprise them, such as laboratory techniques, protocols, and databases, but also the people who make those systems work, and the broader organizational, natural and political environments in which artifacts and people are embedded. Second, it argues that systematization has helped make visible a new kind of public health problem, rooted in the post World War II industrialization of the US food supply—national, diffuse outbreaks caused by contaminated food moving through interstate commerce. Third, it argues that despite the importance of and emphasis on systematization in this domain, the numerous and persistent challenges associated with needing to be fast but right preserves a need for expert judgment amidst formal systematization.\textsuperscript{18}

\textbf{Research Questions and Themes}

The three core arguments of the dissertation frame three broad research questions this dissertation asks. First, when, how, and why did systematization become important in public health surveillance and foodborne outbreak investigations, and in what ways do public health and regulatory officials manage time and uncertainty through systematization? Second, how has systematization helped make visible the public health problem of the national, diffuse outbreak caused by contaminated food moving through interstate commerce, what does such visibility reflect about the industrialization of the US food supply, and what effect does this visibility have on the regulatory politics of prevention in the food safety arena? Third, how is expert judgment enacted amidst systematic practices? In this introduction, I give an overview of ideas and findings from relevant literatures I use to answer these questions and explore the main themes of

\textsuperscript{18} For an excellent look at expert judgment in the history of quantification and the pursuit of objectivity, see Porter (1996).
interest, but in each individual empirical chapter, I will draw more specifically on pertinent ideas to support the more local arguments each chapter makes.

*Systematization 1: Systems, Infrastructures, Layering, and Localization*

**Systems and Infrastructures.** By using the term systematization in an encompassing manner, I aim to include both the building of formal systems with well-defined boundaries, and the development of systematized practices, which can be much more diffuse. In doing so, I am indebted to the large technical systems approach, historical and social studies of infrastructure and information, the sociology of classifications, and historical and social studies of standardization, and I note that these literatures have significant overlaps, reflecting the ties between systems, infrastructure, classifications, and standardization. On the one hand, making the argument that officials manage time and uncertainty through systematization is unsurprising. Managing time and managing uncertainty are difficult things to do, so systematization seems like a logical solution experts would take to solve challenging problems. However, when and how systematization occurs, and to what effect, are important questions to answer in specific contexts.

Scholars who are thinking about infrastructure have acknowledged the foundational role that Thomas Hughes’ work has played to the study of this topic. When I began my fieldwork, I was in fact was only nominally familiar with it, but after a few weeks, I picked up *Networks of Power* (1993), and in those pages, found much resonance with the sociotechnical world I was learning about. On retrospect, the connections are obvious; Hughes was writing about the historical process of building electrical systems, which are large-scale, distributed, complex, and
made of heterogeneous components. All of these characteristics hold for public health surveillance systems.

What especially resonated with me was that Hughes made the problem-solving activities of system-builders so central to his narrative, and problem-solving, I soon observed, was an activity central in public health surveillance. Hughes’ ideas have been widely influential in part because he proposed a general pattern by which large technological systems evolve, and has developed many helpful heuristic categories for studying that pattern. However, Hughes’ work was focused primarily on how one system developed, incorporating more and more elements of the system’s broader environment into the system’s boundaries, whereas what I was observing was much more multi-sectoral and segmented, and focused on the production of evidence to inform action, rather than on the generation of an output like electricity.

Another book I picked up during my fieldwork was Paul Edwards’ *A Vast Machine* (2010), and in it, saw many parallels between the history of climate science and the history of foodborne disease surveillance. For example, Edwards begins his book by making a central point; “without models, there are no data.”

19 We “see” the climate, he argues, through global systems of data modeling, with weather and climate observation systems that cover the whole world, knitted together by data analysis.

20 Similarly, I would argue that we “see” foodborne disease through national systems of public health surveillance, that interlink with hospitals, state laboratories, and sites in the food chain, knitted together by data analysis. Edwards’ point about the relationship between models and data echoes Tauxe’s point above, that outbreaks do not “occur” unless they are detected.

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19 Edwards (2010), xiii.
20 Also relevant here is James Scott’s concept of legibility (1999).
Edwards bolsters his central point with a concept he calls “knowledge infrastructures,” and defines as “robust networks of people, artifacts, and institutions that generate, share, and maintain specific knowledge about the human and natural worlds.” Interestingly, Edwards and colleagues (2013) have actually identified the CDC as a specific example of a knowledge infrastructure. They distinguish an infrastructure perspective from a systems perspective in that the former is concerned with many layers, modularity, multiple systems, and ecologies. By contrast, systems, they argue, are more coherent, engineered, and rationalized from end-to-end.

Rather than replacing a systems perspective with an infrastructures perspective, my dissertation borrows from both, because used together, they help elucidate how and why officials have built public health surveillance systems to help solve outbreak investigations, and how those systems exist within and link to a broader knowledge infrastructure. To create public health surveillance systems, officials have had to develop significant interlinkages between different sectors, attaching their surveillance systems to clinical, regulatory, and food production sectors.

Ingo Braun and Bernward Joerges (1994) have studied what they call “second-order systems,” that involve “networking parts of different first-order systems for specific, macro-level social domains,” and are either “superimposing” or “parasitic” on existing infrastructures. Indeed, one public health scientist even jokingly called their early warning system a “parasite” on the clinical sector, because the primary input to the early warning system comes from there—a bacteria from a sick person. The early warning system is also parasitic in a different manner in its relation to the food production sector. To identify contaminated food in the food supply, officials must attempt to reconstruct the past movement of food through interstate commerce.

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22 Summerton (1994).
They link patient’s illness experiences to complex industrialized food chains, trying to gather traces of food’s prior travels, working backwards from fork back to farm. This is always a challenging prospect, as food chains are designed to move quickly food quickly from farm-to-fork, without a consistent identity as food passes through many, many hands.\footnote{Lezaun and Groenleer (2006) have examined European responses to food control emergencies and efforts to build traceability into food chains.}

Outbreaks are events that involve frequent novelty, contingency, and surprise, as contamination can occur at any point along the farm-to-fork continuum, and involve any kind of food. While officials use many systematic practices, they often emphasize the uniqueness of every outbreak. Thus, their investigation work involves what they call a balance of art and science. They have defended the importance of professional judgment for managing the exigencies of different situations, though they do use many rubrics and routines. In this context, while emergency management has many routine elements because of systematization, it retains a contingent, improvisational, and dynamic character. This unique character is expressed by a frequent metaphor used to describe outbreak investigation work—disease detective work.

To connect the dots during outbreak investigations, disease detectives turn stuff—bacteria, people, foods, animals, places, states, brands, companies—into data, and assembling them into the heterogeneous web that comprises any individual outbreak. Choose any food vehicle – cantaloupe, chicken, peanut butter – and every web is different. Investigators have scrutinized cantaloupe fields in Colorado in the heat of the summer, chicken plants where poultry move on factory lines at 140 birds per minute, and peanut butter factories where peanuts were
roasted and ground into a paste loaded into a tanker truck carrying over 40,000 pounds to suppliers.\textsuperscript{24}

This is not to downplay the importance of systematized patterns and practices. Though the dots vary, they come from the locales of health care provision as well as the modern food marketplace, such as doctor’s offices, hospitals, restaurants, supermarkets, refrigerators, home kitchens, farms, distribution centers, and factories. Human stools, environmental swabs, and food samples are taken from these fieldsites and sent to laboratory scientists, who are tasked with trying to find elusive pathogens from sometimes unyielding matter. Information about symptoms and foods eaten is collected from people via questionnaires; these “food history” interviews usually take place over the phone, though in small counties some are still conducted face-to-face. Increasingly, investigators try to collect shopper card information from companies to help access the purchase records of consumers, to help fill in for the inevitably fuzzy human memory about the foods people ate a week or more before the food history interview. In foodborne outbreak detection and response, investigators use a mix of art and science to conduct their work.

While I have provided my rationale for using a systems/infrastructures approach to study public health surveillance, it is important to note that this topic could be studied using other research traditions. Rather than Hughes’ large technical systems approach to study distributed and complex phenomena, many STS scholars have used Actor-Network Theory (ANT).\textsuperscript{25} In fact, there are some affinities between the two approaches, as Hughes (1986) has described.

\textsuperscript{24} I wish to note that in most cases, the investigator who collects evidence from a field site (e.g. local regulatory inspector) is not the investigator who engages in sensemaking (epidemiologists and regulatory officials in headquarters).

\textsuperscript{25} This literature is voluminous so here I cite only the classic works: Latour (1987, 1991, 1993); Callon (1986); Law (1992, 2009). Latour (1988) is the most relevant of his works to this dissertation (\textit{The Pasteurization of France}).
However, because I am interested in highlighting the epistemologies of molecular epidemiology from a more emic perspective, I do not use ANT as a primary framework, with its “neologisms” and “abstractions of interaction.” Furthermore, an ANT approach would tend to give more agency to bacteria than I would like to in my work, because I am more focused on how bacteria are defined through technologies of classification.

**Layering.** One useful analytic concept to help expand on the importance of multiple systems linking together with some of them existing on different “orders” is layering. Geoffrey Bowker (2000) has argued that STS scholars need to further “engage the complexity and historicity of data within the sciences so that social, political and organizational context is interwoven with statistics, classification systems and observational results.” In the dissertation, I delve into the historicity of the layers of foodborne knowledge infrastructure, to understand the social, political, organizational aspects that interweave with the scientific and technical. Janet Abbate (2000) used a metaphor of layering when examining the history of a US defense computer network that laid the technical groundwork for the internet; she argued that layering helps capture how levels of the network were arranged in a hierarchy from concrete (lower-level) to abstract (higher-level).

One benefit of thinking about layers in knowledge infrastructure is that it helps shed light on the shaping effect that previously lain layers have on subsequently lain layers; as Susan Leigh Star (1999) states, “infrastructure does not grow *de novo*; it wrestles with the inertia of the installed base and inherits strengths and limitations from that base.”

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In the multi-level federalist public health system, where counties, states, and the federal government share authority, it is useful to see the federal layer as superimposed upon the installed base of counties and states. Especially key players making up the “installed base” in foodborne disease surveillance are state departments of public health and state public health laboratories. The Institute of Medicine (IOM) has called the relationships in the multi-level federalist system “complex and hotly contested” (2002). Highlighting the continual negotiation involved in federalism, the IOM called federalism a “sorting device” for determining which level of government should respond to a particular public health threat, emphasizing that it was always necessary to have “good communication and cooperation” amongst multiple levels.

Infrastructure has major implications for the ease and quality of communication and cooperation amongst levels. The relationship between state and local levels varies considerably across different states, ranging from extremely powerful local government authority in “home rule” jurisdictions, to weaker local government in favor of more centralization at the state level. Some states, particularly in the Midwest, follow the latter model because of a doctrine called “Dillon’s Rule,” a 19th century framework which gave local governments only the authority and power expressly granted to them by the legislature.27 States that use Dillon’s rule typically have more centralization at the state level, which tends to enable better coordination and aggregation of multi-county information.

At the state level, control of public health threats typically falls under state “police power,” though the national government can “preempt” state public health regulation if the threat is deemed major enough. Public health police power grants states the right to “pass and enforce isolation and quarantine, health, and inspection laws to interrupt or prevent the spread of

27 Goodman et al. (2007).
disease.”28 State can invoke police power to meet four objectives: (1) promote the public health, morals, or safety, and the general well being of the community; (2) enact and enforce laws for the promotion of the general welfare; (3) regulate private rights in the public interest; and (4) extend measures to all great public needs.29 Given the complexity and contestation involved in multi-level federalism, the IOM has strongly emphasized the importance of collaborative relationships between levels and need for partnership-building to achieve inter-level “alignment” in public health protection.30

One strength of having institutions like county and state departments of health and state public health laboratories having served as an “installed base” for many years is that these institutions have continued to play an important role in enacting the day-to-day functions of foodborne disease surveillance, despite changing technologies. Having a stable institutional platform has been crucial for the building of public health surveillance systems. That said, the capacities of these organizations not only enable, but constrain future possibilities.

To illustrate, I will summarize two brief examples that I will explain in more detail in the empirical chapters of the dissertation. Before the current molecular-based early warning system was put into place in the 1990s, in the 1960s, public health officials built a second-order surveillance system based on a typing technology called serotyping. This system strengthened and intensified the relationship between states in the installed base and the federal level, and helped officials develop epidemiologic knowledge and tools to identify and investigate multi-state common-source outbreaks, which turned out to be benefits for the current system. However, it is important to understand that the system based on serotyping was built for its own

29 Ibid.
30 Ibid.
purposes, and that its builders did not necessarily envision that their system would contribute to the development of a later system. Furthermore, the two systems are distinct, as the serotype-based system is still in operation. Some attributes of the first system became resources for later system-builders, but some attributes were limitations and constraints. A key one was technical; serotyping worked for *Salmonella*, but could not be developed into an epidemiologically useful cross-pathogen tool.

A strength of the current system is that it was established and standardized decades ago and has amassed an enormous amount of molecular data out of which officials can detect foodborne outbreaks on an early warning basis. However, that same strength is also its weakness in light of future requirements. Genomic technologies have been evolving at an increasingly rapid pace, requiring officials to shift from the current molecular system to build not only a new system, but to build a novel informatic infrastructure within the public health system. This is not only a major technical challenge, but a vast economic one. Many expert bodies have pointed out that the public health system has been “chronically underfunded for decades.”\(^{31}\) Between 2008-2012, state and local public health budgets have been cut more than $1.15 billion.\(^{32}\)

Another benefit of thinking about layering is to better understand the relationship between outbreak surveillance and the “community,” a term public health scientists use to refer to human populations at large. For us community members, food is an infrastructural layer for our everyday life. We eat to survive, and to provide sustenance and energy for the myriad activities of living.

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\(^{31}\) [http://healthyamericans.org/assets/files/TFAH2013InvstgAmrcsHlth05%20FINAL.pdf](http://healthyamericans.org/assets/files/TFAH2013InvstgAmrcsHlth05%20FINAL.pdf).

\(^{32}\) Ibid.
In the “community,” the infrastructure of everyday life is also dense with social meaning. Trying to capture this social meaning is one of the central goals of ethnography, to produce “thick descriptions” of cultures, how communities live in a particular time and place. By contrast, since outbreaks are acute events that briefly perturb everyday life in particular times and places, outbreak investigations have a more short-term, instrumental goal—solve the outbreak. The goal of investigating outbreaks is to solve them as quickly as possible; in the process, investigations rapidly generate what Theodore Porter (2012) calls “thin descriptions,” the “least common denominator[s] for a large and heterogeneous world that must be made commensurable to be regulated uniformly.”

Thin descriptions can also be seen as simplifications. James Scott (1999) says that simplification has two meanings; facts must appear as simplified forms and members of a class of facts so that officials can gain synoptic views, and the grouping of facts means collapsing or ignoring distinctions that might be relevant in other contexts. Susan Leigh Star (1983) has identified several forces that press scientists to simplify their work, such as the need to communicate with specialists in other fields, exigencies of limited time or resources, pressures from funding agencies, or pressures to popularize their results. Indeed, I observed that (proper) simplification is a CDC ethos and aspiration. In 2012, a poster displayed at the entryway of a CDC building floor on “Explaining CDC’s Food Safety Story” features a quote from Albert Einstein, who said, “Make everything as simple as possible, but not simpler.” Outbreak surveillance systems superimpose a layer of detection and investigation onto communities, and the thin descriptions investigations produce are a result of simplification, sifting out the density of everyday life and the social meaning of food within communities.

33 Geertz (1973).
A straightforward definition of epidemiology is that it is the core field of public health, and is the science of disease in populations. Many epidemiologic findings “potentiate” large-scale, population-level interventions in a variety of domains, from food production and marketing and air quality to automobile safety (McMichael 2005). Going back to the Greek roots of the term epidemiology (epi, demos, logia), demonstrates that layering is a particularly evocative concept for analyzing the science of epidemiology; epidemiology means the “study of what is upon the people.”

Similarly, Tauxe has called outbreaks “disturbances” or “tears” in a “covering fabric,” a “heightened reality” amplified by the meeting of underlying causal mechanisms.

Studying the heightened reality upon the people means layering a variety of concepts onto the world, so that epidemiologists can generate thin descriptions from the thickness of everyday life, thin descriptions that help them solve outbreaks and take public health actions. Persons are divided up into “ills” and “wells.” Sick persons are “cases.” Cases are either outbreak-related or “sporadic.” Multiple cases with some “commonality” are defined as a “cluster.” Contaminated foods are “exposures” (risks to human health) and “vehicles” (carriers of bacteria). Once a vehicle has been identified, a cluster becomes an outbreak. An outbreak is a kind of epidemic, and an epidemic is an “increase, often sudden, in the number of cases of disease above what is normally expected in that population in that area.”

Localization. Embedded within the concept of an epidemic is that there is a normal expectation of disease, which epidemiologists call a “baseline” or “endemic” level of disease. An outbreak is

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34 Webb & Bain (2011)
also defined as a sudden increase in disease above baseline levels, but is more “localized” in some dimension, such as within a village, town, event, or sub-population. Alfredo Morabia (2004) argues that there are two underlying principles of epidemiology: population thinking (concerns with the measurement of the health of whole groups, e.g. prevalence, incidence) and group comparisons (concerns with measuring contrasts between groups). Those can be seen directly in the definition of an outbreak, concerned with the disease of a population in different areas, with those areas divided up further into smaller units (village, town, etc).

The term “localized” reflects how outbreak investigations involve honing in on a more specific population where illness is occurring; a key way to define the situation and study the populations involved is to consider the classic “epidemiologic triad” of person, place, and time. To figure out how a group was sickened, important fundamental questions to answer are who got ill, where, and when? While person, place, and time become important means to help define the groups for calculating group comparisons, each component of the epidemiologic triad is a thing “good to think.” Person, place, and time are each complex, active, and lively sites of enactment and coordination. But outbreak investigations represent only brief encounters with community lifeworlds and foodways.

To illustrate how localization reflects brief encounters with community lifeworlds and foodways, I draw upon a textbook example of how the source of a *D. Latum* tapeworm outbreak among Jewish housewives in the 1940s was successfully identified. Investigators collected information on the ethnic and demographic characteristics of the sick population, queried this population’s specific eating habits, and were able to pinpoint how the outbreak had occurred.

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37 Quinlisk (2010)
38 Lilienfeld & Stolley (1994)
39 Levi-Strauss (1964)
40 Mol (2002)
41 Lilenfeld and Stolley (1994)
The housewives became infected with tapeworm because they were testing the seasoning of uncooked gefilte fish while they were preparing it. This sort of “localization” happens in every single outbreak, as a confluence of the modern food marketplace, the diverse identities of its eaters, and contaminated food. The tapeworm outbreak is a small window into a decidedly domestic picture of home cooking from the 1940s, when gefilte fish was a primarily homemade food. Today, gefilte fish is typically sold as a processed, ready-to-eat product, packed in a can or a jar, and is (hopefully) tapeworm-free.

During the research and writing of this dissertation, I have heard about outbreaks associated with a number of social events (weddings, national conventions, company picnics, and family reunions) or in particular kinds of institutions (prisons, nursing homes, hospitals, resorts, and cruise ships). I have heard about outbreaks associated with what one case called “kid crack” (Veggie Booty, a snack food popular with small children), North Carolinian vegetarian tempeh-eaters, and live-animal slaughter markets in New York City.

Foodborne outbreaks (and their investigations) surface particular foods, their eaters, and the places where they live, showing how their eating practices combined with food system structures placed them at-risk. In thinking about localization, I quote Sidney Mintz and Christine Dubois (2002):

> In-depth studies of food systems remind us of the pervasive role of food in human life. Next to breathing, eating is perhaps the most essential of all human activities, and one with which much of social life is entwined.

Eating is entwined with social life, and since foodborne outbreaks are entwined with eating, they are also entwined with social life, as mediated by the surveillance systems which help to thinly describe how what “we” eat places “us” at risk—meat-lovers, party-goers, warehouse shoppers,

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42 Desowitz (1978).
kids in daycare, men, women, and all consumers reliant on the food supply. Underneath thin
descriptions, the entwining of eating and social life thickly transpires.

_Systematization 2: Interfaces, Time Management, and Uncertainty Management_

Interfaces. Layering and interlinkage are so important for foodborne knowledge infrastructure, because multi-level, cross-sector relationships must be built to facilitate the creation and flow of the information necessary to solve national outbreak investigations. One widely-used metaphor that emphasizes the need for teamwork and collaboration between groups involved in foodborne disease outbreak investigations is a “three-legged stool,” comprised of epidemiologists, laboratory scientists, environmental health specialists, and regulatory officials, who have different responsibilities and expertise, but must work together to coordinate outbreak response.\(^4^3\) I argue that a useful idea for understanding these interactions and the enactment of teamwork is the interface, which I define as a zone where different entities meet, communicate, and influence each other as they interdependently create and share information. That an interface is a computational and informational metaphor is to its advantage, since what is happening within each “leg” is a transformation of an artifact of everyday life into data, afforded by a layer of computational and informational technologies. Interfaces are particularly important and interesting spaces to observe within and between foodborne knowledge infrastructure and public health surveillance systems.

Epidemiologists interview patients about their clinical symptoms and the foods they recently ate using questionnaires, collecting case and possible exposure data. Laboratory scientists extract pathogens from feces and foods to identify subtypes of bacteria using

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\(^{43}\) Van Bendenen and de Val (2013)
standardized protocols. Environmental health specialists turn food samples and records from restaurants, factories, and farms into data. Data circulate through and email, telephone, and list-servs, and are organized and analyzed in databases. All of these activities happen in and across multiple jurisdictions and organizations. While there are some shared spaces of information management, such as collaborative databases, there are many spaces that are not shared, often due to legal restrictions, fragmenting the creation, aggregation, and dissemination of information by the design of the political system. For example, since the privacy of Protected Health Information (PHI) is regulated by the Health Insurance Portability and Accountability Act (HIPAA), patient names are only allowed to be seen and managed by county or state health workers, who transform them into cases with case ID identifiers and house this data within their own disease surveillance systems. Federal officials have no access to PHI, so the case data they work with are de-identified with personal information, but linked and retrievable through the case ID number.

My interest in interfaces bears some relationship to Robert Kohler’s study of the history of the lab-field border in biology (2002); he uses the concept of a “border” to emphasize that the two groups of scientists do not live in entirely separate and different worlds, but engage in a permeable “zone of active interaction and exchange.” However, while borders and interfaces are somewhat similar concepts, my goals contrast with Kohler’s. His was to capture “what it was like to do fieldwork in a world that took laboratory methods as universally the best for all sciences,” and he was particularly concerned with how the laboratory imposed its own version of objectivity on field biology, to what he argues is the detriment of field biology.

Mervyn Susser (1998) makes a similar argument to Kohler in talking about the impact that bacteriology had on epidemiology with the rise of germ theory in the 19th century. Susser
argues that germ theory’s specific cause model disrupted the environmentally oriented epidemiology animated by miasma theory, and that after germ theory, epidemiologists began to serve as “handmaidens” who simply applied their bacteriological colleagues work, moving to a more “secondary” public health role.\(^4\) Alfredo Morabia (1998) disagrees with Susser’s contention, and argues that it is perhaps scholarly inattention to epidemiology during the bacteriological revolution that creates this distorted view.

I argue that analytic attention to interfaces is a way to step out of the competitive frame of who was “handmaiden” to whom, and instead consider questions about how and why coordinated relationships of information creation and sharing have been established, and how they have evolved over time, to become increasingly intensive and interdependent as outbreaks are detected at earlier stages in new surveillance systems. To go from disease at an epidemic level lasting months or even years, to events that last weeks and days and involve only a handful of people, has drastically shaped the relationships and practices of epidemiology, laboratory science, environmental health, and regulation alike. To me, the compelling questions are when, how, and why epidemiology, laboratory science, environmental health, and regulation have negotiated relationships of information-sharing, created interfaces of information exchange, and began to work together to constitute coherent surveillance systems around the shared work object of a distributed national outbreak.

Additionally, the interface concept is relevant not only for understanding information-sharing relationships between experts, but also more broadly, the relationships between the surveillance system and broader society. Creating thin descriptions of the world involves the transformation of society into information—people must become cases, foods must become

\(^4\) Judith Leavitt (1992) has similarly suggested that earlier historiographic takes on the effect of bacteriological science on public health in the 20th century have overestimated the degree to which reductionism actually limited the scope of public health activities.
exposures, companies must become sources. Outbreaks have a social life beyond their detection and investigation, as regulators make policy changes, as cases sue companies, as companies recall foods and change their sanitary practices.

Managing Time: Real-Time. Today, operating a real-time system means intensely managing time, and interfaces are particularly salient and interesting spaces to observe the dynamics of time management. Without monitoring and managing the connections between heterogeneous components and layers of the system, information cannot move quickly through the system. Data have friction. When the first iteration of the current early warning outbreak surveillance system was established in the 1990s, much of the system-building work involved computerization and digitization, to turn the molecular DNA fingerprint of the bacteria into a digital file that is uploaded, stored, and analyzed in a database accessible to the distributed set of actors in the network. The pattern of evolution for foodborne outbreak surveillance systems, like many other systems, has shifted from systems to networks to webs.

Today, network members use a variety of bioinformatic databases, online collaboration platforms, teleconferencing, and email communications to rapidly share information about outbreaks. Communicating across laboratory-epidemiology-environmental health interfaces involves a mix of formal practices of information exchange (e.g. standardized protocols, data entry, web-board alerts), and lively collaborative sensemaking practices.

One simplified holistic vision of the early warning system uses time as its central organizing principle to track the journey of a person being transformed into a case in an outbreak. Below, I include a CDC timeline that diagrams the steps between a patient eating contaminated food and a case being confirmed as part of an outbreak. The following timeline

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46 Ibid.
(Figure 2) illustrates why it takes up to 2-3 weeks between those steps for an outbreak-related case to be confirmed.

Figure 2. Timeline for Reporting Salmonella Cases

Included in this timeline are multiple system steps and the interfaces between them: the interface between the patient and the health care system (illness, contact, diagnosis); the interface between the health care system and the public health sector (shipping time); and incorporation into the system via the bacterial subtyping that enables outbreak detection (serotyping and DNA fingerprinting).

Because every step has a time window associated with it, and the work associated with each step is interdependent, this helps create accountability for distributed participants in the real-time system. However, I must emphasize, it is only the latter steps where participants in the system have the most control. It is only when a pathogen is identified as a *Salmonella* in the
clinical laboratory, and is shipped to the state public health laboratory, that it is solidly inside of the early warning system. Even then, I wish to emphasize that bacterial subtyping happens in every state public health laboratory, in distributed locales; it is the shared PulseNet database that brings distributed cases together in one “place,” to enable national outbreak detection. As Donald Mackenzie (2012) argues, hardware and place matter enormously in the configuration and operation of high-speed systems.

To study time management, I find three concepts especially relevant: temporal structures, temporal rhythms, and temporal thinking. Wanda Orlikowski & JoAnne Yates (2002) have developed the concept of temporal structures as part of a practice-based perspective that emphasizes the important role enactment plays in shaping how people experience time and take action in the world. Through enactment, people produce and reproduce temporal structures that guide, orient, and coordinate their activities, and temporal structures both shape and are shaped by human actions. “Real time” outbreak detection is usefully understood as an enacted temporal structure that is the outcome of systematized, coordinated actions within a sociotechnical system. An even better concept than “real time” is “real enough time,” to underscore that people’s work processes and technologies must be designed to accommodate variable and contingent demands, as well as material and organizational constraints.47 Real time is perhaps best seen as an idealized concept; would even the Star Wars metagenomic example referred to above be a “true” real time, when in that scenario it still involves a person getting sick, as opposed to a contaminated food being identified before it leaves the manufacturer’s hands? By understanding real time as an idealized concept, the dynamics of systems development become clearer; each system sows the seeds for its own destabilization by making visible what the system cannot do, in addition to what it can do.

47 Orlikowski & Yates cite Bennett & Weill (1997) for an original mention of “real enough time.”
Steven Jackson and colleagues (2011) have argued that collaborative scientific work is organized around four different temporal rhythms: organizational (e.g. semesterly academic calendars, weekly lab group meetings), infrastructural (e.g. equipment replacement schedules, time to build interoperability between systems), biographical (e.g. career trajectories, life-course events), and phenomenal (e.g. deriving from objects or phenomena under study). The latter temporal rhythm (phenomenal) plays an especially strong role in organizing the scientific work of outbreak investigations, because the outbreak is a disease event unfolding in the community, as mediated by the surveillance system designed to detect it. The way in which this event unfolds, and the ability of the investigation to map the unfolding event, is a key temporal rhythm shaping the flow of outbreak investigation work.

That said, other temporal rhythms are still important. In fact, the emergency has in some sense become the ordinary, through the development of organizational routines for outbreak work; for example, every week, PulseNet database managers routinely scan the PulseNet database for to identify clusters of disease (defined as 3 or more human isolates above the normal pattern baseline frequency).48 In addition to rhythm, I find other musical metaphors useful in helping explain the dynamic temporalities involved in outbreak investigation work, such as tempo, acceleration, and improvisation.

I propose the concept of temporal thinking as a way of capturing the centrality of time in problem-solving during outbreak investigations. Time, after all, is one of the three members of the epidemiologic triad. Temporal thinking is complementary to population thinking and group comparisons. Outbreak detection and response work entails asking and answering many different kinds of questions about time. Some of these are questions about the timeliness of the surveillance system. For example, how long does it take for an isolate to be mailed from the surveillance system designed to detect it. The way in which this event unfolds, and the ability of the investigation to map the unfolding event, is a key temporal rhythm shaping the flow of outbreak investigation work.

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clinical laboratory to the public health laboratory? When will the confirmatory DNA testing be
done on a newly detected case? Other questions query the event of disease occurrence. For
example, how long is the average incubation period for *E.coli* O104? What is the shelf-life of
cherry tomatoes? When is the peak of the epidemic curve? Asking and answering such
questions not only helps officials keep the system running in real time speed, but also to make
logical inferences about the dynamics of the event of disease occurrence, and solve the outbreak.

**Managing Uncertainty: Early-Warning.** In addition to managing time to keep the surveillance
system running efficiently in “real-time,” officials must manage the amplified uncertainty
entailed with an “early warning” system. PulseNet helps alert officials to “suspected” outbreaks
– clusters of disease initially defined by matching DNA fingerprint patterns. While it is possible
that clusters of cases with matching patterns are an outbreak (caused by a common source), it is
also possible that they are not, and happen to share the same pattern by chance. It is more likely
that clusters are real outbreaks when they are caused by new or rare patterns within the PulseNet
database. Some patterns are extremely common, making it difficult to tease out outbreaks within
the mass. During outbreak investigations, officials must collect other kinds of data and conduct
other kinds of analyses to determine whether those clusters are, in fact, outbreaks.

One thing that this dissertation charts is that multi-level coordinated practices and
collaborative relationships between different fields and organizations around the early warning
system had to be negotiated. Scientific agencies and regulatory agencies had to arbitrate their
conflicting orientations to uncertainty. The CDC as a scientific agency with a pragmatic and
problem-solving approach with more tolerance for ambiguity had to negotiate coordinated
actions with the FDA and USDA, whose authorities as regulatory agencies drew heavily from
making certified evidence to be used in adversarial legal contexts. I argue that systematization helped mitigate some of these organizational differences. Resorting to “trust in numbers” signals conditions of social distrust, and standardization should be seen as an outcome of social struggle and historical process.\textsuperscript{49}

To help explicate the systematization involved in the early warning system, I argue that its “network form”\textsuperscript{50} is best understood as what I call a \textit{confirmatory pathway for provisional knowledge}. A key insight from STS is that in different settings, uncertainty has different qualitative forms.\textsuperscript{51} To understand early warning, real time outbreak detection, I argue that the form uncertainty takes is provisional knowledge. What is uncertain at first is temporary. Because there is a confirmatory pathway for knowledge to travel, provisional knowledge promises that better knowledge is soon-to-follow. The future that provisional knowledge envisions is in the near-term, and will be determined by the results of confirmatory activities in the present.\textsuperscript{52} While provisional knowledge will not definitely be converted into more certain knowledge, workers inside of the confirmatory pathway will try their best to achieve that goal.

Investigational activities are designed to transform provisional knowledge into confirmed knowledge, determining whether suspected outbreaks are indeed outbreaks, or not, through the use of standardized tools such as bacterial typing, epidemiologic studies, and food tracing. A cluster is a suspected outbreak, and becomes a confirmed outbreak when a source is identified.\textsuperscript{53} But exactly when a cluster or pre-outbreak becomes an outbreak can be a “grey area,” as one epidemiologist in a state pointed out to me; if there are a number of cases, and a specific source

\textsuperscript{50} Podolny & Page (1998)
\textsuperscript{51} Shackley & Wynne (1996), Timmermans and Buchbinder (2012), MacPhail (2010).
\textsuperscript{52} An outbreak is a way to temporally bound disease but outbreaks can have a seasonality and recur every year; a pattern can be endemic to a site.
\textsuperscript{53} Another uncertainty management term that officials use is a “pre-outbreak,” DNA fingerprint matches between human isolates and food/animal isolates.
is suspected but not definitively identified during the investigation, it seems to be an outbreak, yet it would not be counted as such in official reporting unless a source was definitively identified.\textsuperscript{54}

I borrow the term pathway from neurology, where pathways are defined as neural circuitry driving how information is transmitted and travels through the nervous system. I argue that this is an apt analogy for detected outbreaks, which are made of information and travel through a multi-organizational, multi-jurisdictional, multi-disciplinary system. Yet information does not simply flow through the pathway, which is captured by the term confirmatory. That the pathway is a confirmatory one highlights the central importance that tools and techniques for uncertainty management play in this domain. While initial outbreak data are uncertain (is a cluster or pre-outbreak an outbreak?) as heterogeneous bits of data are collected, they move along the pathway, and are made increasing sense of through the application of laboratory and epidemiologic tools and techniques designed to make the data less uncertain, and become the basis for taking actions in the world. To observe outbreaks moving along the confirmatory pathway is to observe the gradual transformation of data from a preliminary state into a more confirmed state, justifying the taking of actions in the world, which in turn produce more data, in a kind of feedback loop. Feedback loops are hallmarks of a system.

While the confirmatory pathway for provisional knowledge is immediately oriented to the near-term future, in solving outbreaks and stopping more cases, it has a longer term purview and interest in producing information that can help alter food system practices to make them safer and prevent future illnesses. Robert Tauxe (2006) argues that foodborne surveillance plays a driving role in what he calls a “cycle of prevention.” The detection and investigation of outbreaks, he states, helps point to “pathways that are the most problematic” within the food

\textsuperscript{54} Field notes 6/5/2013.
safety system, and identify “new challenges and gaps” in it. Within the cycle of prevention, each outbreak is not an single event in itself, but has greater implications, providing an “opportunity” for improving illness prevention efforts through “lessons learned” from each outbreak episode. Figure 3 depicts the cycle of public health prevention.

![Figure 3. The Cycle of Public Health Prevention](image)

That the real-time, early-warning system aims to intervene in both the short-term and long-term future suggests that it is machinery for anticipatory knowledge, or knowledge oriented towards possible futures.\(^{55}\) Anticipatory knowledge has the goal of not only describing, but shaping the future and enabling interventions in it. The kind of anticipatory knowledge needed is not always what is produced or producible. Indeed, it is important to understand that the confirmatory pathway for provisional knowledge was built under historically specific conditions of possibility. The temporalities related to anticipatory knowledge are complex, and that machineries for producing anticipatory knowledge strongly shape the content of the knowledge produced.\(^{56}\) Policy bodies are important producers and users of anticipatory knowledge, exemplified by forms like forecasts, models, scenarios, and risk assessments.

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56 Mahajan (2008) writes of the “foreknowledge” about HIV/AIDS carried within epidemiological modeling as it moved between national contexts.

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Within the real-time, early-warning system, a number of systematized knowledge practices and tools assist officials in confirming the provisional knowledge they create. Confirmation is nicely explicated as analogous to testing, a technical process enabling the tester to make a valid inference that one thing and another are similar.\textsuperscript{57} One key thing being tested or confirmed in outbreak investigations is whether bacteria are the “same” in people, food, and places. Bacterial identities become the data keys that enable officials to connect the dots to each other.

Once a pathogen has been isolated from a sick person, it can be examined in the laboratory to determine its type. This establishes a similarity relationship between the particular pathogen in a person, and a general category of pathogen as defined by a scientific classification system. Once a reasonable number of case interviews have been conducted, statistical evaluations of food exposures can be made, establishing a similarity relationship between those who ate particular foods, or those who did not. Once a food sample from a producer who manufactured the contaminated product yields a DNA fingerprint match to the cases from the cluster under investigation, the product can be implicated. This establishes the ultimate inference public health and regulatory officials wish to confirm as true—identifying the source of the outbreak, and thus definitively transforming the cluster into a confirmed outbreak.

One thing that I have found particularly interesting in my research is that when looking at the development of techniques for identifying microorganisms over time, the “sameness” of the “germ” fragments into a disunity, bringing to the fore how technique-dependent, purpose-laden, and complex the task of defining and classifying bacteria has been to the various groups involved in identifying and controlling infectious disease. “What is a pathogen?” asks a recent microbiology article, referencing a long debate in the field about the uncertainties involved with

\textsuperscript{57} Mackenzie (1989)
distinguishing pathogens from non-pathogens; drawing clear lines between the two categories has long been considered to be biologically problematic.\textsuperscript{58}

Though creating stable and static classification systems for dynamic bacterial entities may be biologically problematic, stable and static classification systems have long been key to making clinical and public health interventions. Many scholars have shown how interventionist aims have shaped the representational techniques of scientists.\textsuperscript{59} Bacteria are excellent model organisms for understanding the intertwined practices of representation and intervention in different domains. As this dissertation will show, there are long-standing frictions and alignments between classification systems for bacteria aimed at clinical relevance, public health utility, and evolutionary taxonomy.\textsuperscript{60}

To quickly describe this friction, clinically relevant diagnostics tend to emphasize the creation of information that is of maximum utility to the doctor treating the patient. For example, from a physician’s perspective, it is enough to know that an \textit{E.coli} produces shiga-toxin to be on heightened alert for the patient developing hemolytic uremic syndrome. The \textit{E.coli}’s specific subtype is not of interest to the physician treating an individual patient, but hugely important for public health officials interested in detecting diffuse outbreaks by connecting distributed cases. Though subtypes have enormous public health utility, they are not necessarily categories that help illuminate bacterial evolution.

\textsuperscript{58} Pirofski & Casadevall (2012).
\textsuperscript{60} I examine this topic in more depth in Chapter 3, related to \textit{Salmonella}.  

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Contaminated Food in Interstate Commerce

In *Fear of Food: A History of Why We Worry About What We Eat*, Harvey Levenstein (2012) roots the source of Americans’ anxieties about their food supply with the vast changes occurring at the end of the 19th century. Food production was undergoing fundamental changes. Food networks expanded beyond the home and small farm to a larger mode of industrial production to support American urbanization. Americans and their food began to travel more and across longer distances. Large companies became chiefly responsible for making food, which passed through lengthy supply chains and many hands as it was processed. These changes, Levenstein argues, fueled Americans’ increasing distrust of food production.

By the 1920s, most families, both rural and urban, were heavy users of commercially canned food, though during WWII, American women were re-enrolled in home canning to free up the supply of commercially canned foods for soldiers.\(^6^1\) The US food system had many industrial elements before WWII, but the war played a significant role in increasing the level of industrialization in food production. Military research and procurement, new regulations, and standardization and rationing of food, were the major factors helping to create a postwar context in which Americans were eating more processed products produced by bigger companies than before the war.\(^6^2\)

To some degree, however, food (and its microorganismic hitchhikers) has been travelling globally for centuries, within colonial and then capitalist networks.\(^6^3\) For example, sugar has been global for over 400 years.\(^6^4\) However, there have of course been significant changes in

\(^{61}\) Bentley (1998)
\(^{62}\) Backer (2012)
\(^{63}\) Ochoa (2012)
\(^{64}\) Mintz (1985)
global food networks over time. Overall, the level of intensity has skyrocketed in the past several decades.

Food scholars root the mass globalization of food in the post 1960s era, emphasizing the importance of increased processing and distribution technologies, such as mechanization, preservation, refrigeration, and long-haul trucking.65 As a key example of how these technologies have shaped and changed the character of global trade, in 2011, the US imported over $18 billion dollars worth of fruits and vegetables; while in the 1970s the US was a net exporter of produce, today it is a net importer, exporting only $7 billion dollars worth of fruits and vegetables to trade partners.66 The mass globalization of produce began in the 1990s, and while some firms are transnational, they are typically much smaller than large-scale industries like the automobile industry, and it is the distribution aspect of the produce industry that is seeing the most globalization.67

Within global public health communities, it has become commonplace to say that infectious diseases do not respect international borders, especially when calling for cross-national cooperative control efforts. Foodborne disease is a global problem, because the trade of food is a global one, as the fenugreek sprout outbreak discussed above demonstrates, with its linkages between European consumers, a German sprouter, and an Egyptian fenugreek seed company. Structural changes in the food supply intertwine with foodborne disease. As food production and distribution chains have lengthened, so have the routes for infectious agents to travel and manifest within longer networks.

However, political jurisdiction shapes the way we see and act on outbreaks in powerful ways. The commerce clause of the US Constitution provides the authority for the federal

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67 Friedland (1994)
government to regulate affairs involving the movement of commerce between states. “Interstate commerce” is a national concept, highlighting how political jurisdiction shapes how we represent and intervene on public health problems. Within the borders of the US, there has been significant integration and coordination of national efforts around detecting and controlling outbreaks of foodborne disease, through systematization. Though international cooperation efforts around multi-country foodborne outbreaks exist, the early warning system’s intensity of surveillance fades outside US borders, because of the limits of national political jurisdiction.\(^{68}\)

This is not to say that international outbreaks are not perceived; some are picked up, but usually through the identification of an outbreak attributed to an imported food consumed domestically. For example, in 2012, state and federal officials investigated a multistate outbreak with a large concentration of cases in California, small clusters in 5 states, and single cases in 10 states, eventually identifying mangoes imported from Mexico as the source.\(^{69}\) From a technical perspective, it would be possible to expand the current early warning system’s molecular and computer infrastructure around the globe. In principle, foodborne knowledge infrastructure could be just as global as climate knowledge infrastructure. However, it is important to recognize that technical possibility does not determine reality.

*Outbreaks and the Social Significance of Disease Prevention*

Between 2006-2010, a series of major multi-state outbreaks in foods as diverse as spinach, peanut butter, refrigerated cookie dough, and eggs attracted major public and political

\(^{68}\) PulseNet International is the global version of the network, and while there is some information-sharing between network members, it does not have the same early warning, real-time structure as PulseNet USA.

\(^{69}\) [http://www.cdc.gov/salmonella/braenderup-08-12/index.html?s_cid=fb1785](http://www.cdc.gov/salmonella/braenderup-08-12/index.html?s_cid=fb1785)

[http://www.fda.gov/food/recallsoutbreaksemergencies/outbreaks/ucm317337.htm](http://www.fda.gov/food/recallsoutbreaksemergencies/outbreaks/ucm317337.htm).
Due in major part to these food safety issues, on January 4, 2011, President Obama signed the FDA Food Safety Modernization Act (FSMA) into law. FSMA entailed a major shift in regulatory framework for the agency, from a reactive mode, or responding to problems after they already occurred, to a prevention-oriented mode, or seeking to stop illnesses before they happen in the first place. While preventing illnesses before they occur is a laudable goal, what kind of social significance does this prevention-oriented mode have, and how is it being enacted?

I briefly draw on a different example—expanded newborn genetic screening in the US—to illustrate why these are important questions to ask. Stefan Timmermans and Mara Buchbinder (2012) examined the social consequences of this prevention-oriented program, aimed at saving more newborns’ lives by employing genetic testing to identify asymptomatic infants who may develop severe metabolic conditions and offer them preventive treatment. While the program certainly has a commendable aim, it also has unintended consequences. Since the test has a high positive rate, it identifies patients with abnormal levels, instead of providing a clear-cut diagnosis. This test creates what Timmermans and Buchbinder call “patients-in-waiting,” who are neither sick nor healthy, but somewhere in between. This in-between state creates difficult and often emotionally painful uncertainties for the patient’s families to bear, as they wait to see whether their baby is truly ill or not.

Confirming whether all cases with DNA fingerprint matches are all related to an outbreak does not hold the same level of personal and emotional consequences for patients, in large part because most foodborne illnesses are fleeting, acute events. However, there are useful

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comparisons to be made between the two domains. Both have worthy objectives—saving lives. Both involve the use of genetic technology to provide “early warning” surveillance information in the service of taking actions to intervene on health problems before they worsen. Both involve the intensified management of uncertainty in the context of taking actions and making interventions.

Early warning newborn screening and foodborne outbreak surveillance are both examples of existing large-scale prevention programs, however, a widespread sentiment within public health communities is that it is challenging to “sell” prevention, even though it is often easier, cheaper, and ultimately more health promoting to stop health problems from developing in the first place, or from getting so bad that they cannot easily be fixed. Harvey Fineberg (2013) calls this the “paradox of disease prevention,” pointing out the contrast between how much our society says it values prevention, and how rarely prevention programs are put into practice. He points to multiple reasons as to why the paradox of disease prevention exists:

the success of prevention is invisible, lacks drama, often requires persistent behavior change, and may be long delayed; statistical lives have little emotional effect, and benefits often do not accrue to the payer; avoidable harm is accepted as normal, preventive advice may be inconsistent, and bias against errors of commission may deter action; prevention is expected to produce a net financial return, whereas treatment is expected only to be worth its cost; and commercial interests as well as personal, religious, or cultural beliefs may conflict with disease prevention.

These reasons provide a useful starting point for explaining why early warning foodborne outbreak surveillance has been able to, at least partially, overcome the paradox of prevention.

The existence of large-scale prevention programs should not be attributed to solely technological causes; as Timmermans and Buchbinder argue, in addition to technologies, new realities for
diseases need vast social movements and enormous infrastructure to turn disease categories into practical clinical entities and attach patients to regimes of diagnosis and treatment.\textsuperscript{72}

Over the past several decades, a great deal of work has been conducted to quantify foodborne illness and assess its economic burdens on society, to help justify the cost of prevention efforts. An estimated 48 million Americans suffer from a foodborne illness each year, and cost-of-illness modeling estimates the average cost per case at $1,626, which totals to over $77 billion in medical costs, productivity losses, and illness-related mortalities.\textsuperscript{73} Today it is possible to generate these numbers because a massive statistical infrastructure has been built for doing so.\textsuperscript{74}

Far from lacking drama, outbreaks are highly dramatic events, particularly when they are large, caused by emerging and/or highly virulent pathogens, or are associated with novel food vehicles, because they herald new kinds of threats or identify threats in unexpected places.\textsuperscript{75} They are newsworthy incidents that capture public attention, especially given current popular interest in food more generally. An outbreak is not unlike a disaster, in that it is an emergency situation that involves threats to human and/or animal health and life. An outbreak is a breakdown of social order, and can interrupt the ordinary consumption of food. Stephen Hilgarter (2007) calls disasters “profoundly disturbing collective experiences” that have the tendency to “challenge the managerial vision of orderly systems.”

Disasters often generate public inquiries which assess cause and blame, typically starting with the media, and then moving to formal political inquiries. When playing out on public stages, inquiries become “social dramas” where a normative “breach” creates a community

\textsuperscript{72} See also Star & Bowker (1999).
\textsuperscript{73} Scharff (2012).
\textsuperscript{74} http://www.cdc.gov/foodborneburden/2011-methods.html.
\textsuperscript{75} For more on social drama, see Turner (1980) and Hilgarter (2000, 2007). I discuss this more in depth in Chapter 7.
“schism,” moving to stages of “crisis,” “redress,” and then either “reintegration” if redress is effective, or continued schism if it is not. Outbreaks are used as evidence to highlight the fallibility of regulatory control systems over food production practices. In this realm, statistical lives often do have emotional effect, because media and public inquiry forums have been forged for ill persons behind the statistics to share their harrowing illness narratives. Charles Rosenberg (2008) points out that in the modern world, though epidemics are no longer interpreted as punishment from God as in 18th century New England, we retain our own unique “rituals of invocation, propitiation, and jeremiad,” which any media article or congressional hearing would confirm.

There is a great deal of social debate about how foodborne disease should be controlled. What constitutes an avoidable harm that can and should be mitigated? What risks should be accepted as normal? For example, while the USDA has a “zero-tolerance” policy around shiga-toxin producing strains of *E. coli* in raw ground beef, it defines *Salmonella* in chicken as a normal avoidable harm that can be addressed through proper consumer food handling. Many consumer groups have argued that this *Salmonella* policy places undue responsibility for control of the problem on the vulnerable consumer. This debate illustrates the importance of understanding that definitions of risk involve the social distribution of responsibilities for control.76 That risk definitions also distribute responsibility for control helps illustrate why preventive advice can be inconsistent in food safety; since prevention can be enacted any place between farm and fork in multiple ways, different social groups often hold different opinions on where emphasis for control should be placed, and who should be held responsible.

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76 Hilgartner (1992); Wetmore (2004).
Foodborne outbreaks are a site where the social anxieties around the US’s modern industrialized (and increasingly globalizing) food supply play out. In the wake of outbreaks, public inquiries are filled with debates about how safe the food supply is, what safety itself means, who is responsible for making and keeping it safe, with which benefits and at what cost, reflecting whose definitions and values, and involving what kinds of tradeoffs. Because foodborne outbreaks perturb our collective sense of the food supply’s safety, they are excellent “sites” for understanding how social order is constituted, what happens when it breaks down, and how it is repaired (or not).77

Throughout the course of my fieldwork, I have often heard consensus-building sentiments that emphasize that “safe food” is a universal value and even a fundamental right. “Everyone wants safe food.” “Food safety is not a competitive advantage.” “We are all consumers.” On its face, it is rhetorically difficult to argue with the idea that food should be safe. As Fairchild et al have noted, in contrast to other forms of public health surveillance, the “well-accepted staple” of investigating common-source outbreaks of foodborne disease has not tended to provoke public controversy around privacy issues.78 While not true in every single case, for the most part, individuals are willing to share their names, health information, and food histories with public health officials in the interest of making the food supply safer. However, what has provoked public controversy within food safety is the tension between protection of public health, and the privacy of the information of commercial entities who make food, a tension made more significant within common public health practices through the improvement of outbreak surveillance technologies and network-building.

77 Similar things happen in scientific controversies. For an overview of the voluminous STS literature on controversy studies, see Chapter 11 of Sismondo (2010).
Recently, Jasanoff and colleagues (2011:15) have argued for a bioconstitutionalist approach, that is, to examine the interactions between law and the life sciences to understand the “basic building blocks of rights,” and ask questions such as, what are our “social commitments concerning what is worth protecting and why, for and against whom, through which kinds of social and institutional agency, by what means, to what extent, and through what processes?”

The existence of such a mechanism reflects a vision of an ideal sociopolitical order; the state is expected to care for and protect its citizens, and needs to build machinery for accomplishing these purposes. Citizens are imagined as vulnerable beings that cannot solve their problems alone, but need public health protection.

How has the notion of a right to safe food been transformed by new technological possibilities in the public health surveillance of national outbreaks? As Michel Foucault and his interlocutors have articulated with the concept of “governmentality,” human life at the population-level has been an increasing concern of state administration and object of scientific and medical knowledge production, creating new forms of authority, subjectivity, and transforming the politics of health around biology and biomedicine. 79 One of the more influential ideas from Foucault’s work on surveillance is the Panopticon, a concept he drew from 18th century philosopher Jeremy Bentham’s design for a circular prison with an observation tower in it, to create a more general theory of modern surveillance. 80 Foucault’s interpretation of Bentham’s panoptic prison theorized the operations of surveillance and control in the following manner. The innovation of the design was that the prison was built such that the guards could observe the prisoners, yet the prisoners could not see the guards. Because the prisoners were

79 This is an enormous literature, but for key works, see Foucault (1975, 2007), Franklin (2000), Rabinow (1999), and Rose (2007).
80 Foucault (1975).
uncertain about precisely when they were being monitored, this created the constant sense that the prisoners were being observed. Foucault argued that this was not only a technique of observation, but worked by internalizing in the inmates the idea that their conduct was always being monitored; this would create, he posited, a disciplining effect on the subjectivities of the inmates, so that they would internalize surveillance and monitor their own conduct. This, Foucault proposed, was a modern form of power that, throughout the 18th and 19th centuries, extended beyond the prison to many other institutions, such as schools, hospitals and factories.

The panoptic theory holds some relevance for understanding the issues surrounding commercial compliance with food safety regulations. That new surveillance technologies can help detect food safety problems, and that new regulatory requirements for self-monitoring practices have been passed, have indeed resulted in more widespread internal monitoring practices in companies as compared to an earlier period. However, as the continued prevalence of foodborne disease demonstrates, in practice, compliance and control are elusive and highly complex issues. The panoptic theory is a general one, too generic to help understand the complex relationships between the array of social actors involved in safeguarding the food supply from farm-to-fork, a configuration which enormously complicates the power dynamics of surveillance and control. Furthermore, Kevin Haggerty and Richard Ericson (2000) note, the development of new technologies, in particular computerized databases, require rethinking panoptic theory. My study uses archival and ethnographic methods to understand the historical development and operations of specific systems, to shed light on how system builders have managed the challenges and dilemmas their fast but right balancing act presents.

81 James Fairhead and Melissa Leach (2007) also note this limitation in their book on the politics of vaccination. 82 Haggerty and Ericson build on ideas from Gilles Deleuze and Félix Guattari to examine newer technologies of surveillance.
Charting these challenges and dilemmas helps open “safety” up for social study. Sarah Vogel (2012) states that questions of safety are often put in black in white terms, however, as she shows in her study of the regulatory politics of bisphenol-A, safety is not an “absolute.” Instead, safety is a “dynamic and often elusive concept,” filled with “layers of meaning,” involving not just scientific questions, but politics, power, and values. Different social groups have different beliefs about how food should be made safe, which aspects of safety are more important than others, what it takes to produce safe food, how much those disease prevention efforts would cost, whether everyone can afford to put those measures into place, should that ability matter, and also, how much safety should be valued over other aspects of food, such as its quality, taste, or sustainability.

While quality, taste, sustainability, and safety seem like separate aspects of food, at times, these values can directly conflict with one other. For example, if valuing safety, a consumer should cook a hamburger to an internal temperature of 160 degrees Fahrenheit to kill pathogens, but if valuing taste, one should cook it to a lower temperature to keep it as rare, medium-rare, or medium, depending on one’s preference for the doneness of meat. Another example of value conflicts in food is the irradiation of lettuce, which could be an effective method for killing pathogens, but conflicts with USDA National Organic Program standards and could lead to textural changes in the leafy green, making it unacceptable to consumers. Though they are often presented as value-free, standards are thoroughly value-laden. As Lawrence Busch (2011) says, “Even as standards are technical rules, they are also compromises among diverse values, themselves drawn from different worlds or orders of worth. Standards are attempts to fix values, to embed them in particular products, processes, persons, practices and organizations.”

Methods
I used a variety of qualitative methods to collect and analyze data for this dissertation. First, I conducted an ethnographic analysis of the early warning outbreak detection and response system. Over the spring and fall of 2012, I visited the CDC three times for a total duration of six months, conducting observation of outbreak-related work, meetings, conferences and training sessions (lab, database, epidemiologic investigation); ongoing interviews with participants; and informal conversations and interactions. Initially I had been planning a month-long visit to conduct oral history interviews, but when I was given the opportunity to observe a couple of meetings and observe some of the work practices, I decided to include an ethnographic component to the project. I was both challenged and fascinated by the fact that it was such a specialized, technology and information-intensive workplace, but soon came to the realization that the CDC’s role as the center of coordination for national outbreak detection and response would give me an analytic foothold. It is difficult if not impossible to go everywhere in a large network or system, so many scholars have gotten traction on understanding large-scale distributed phenomena by “strategically-situating” themselves within a particular place in the network, to understand how participants within that place are themselves trying to manage issues around scale and distribution.

Though I strategically situated myself within one place in the network, because outbreak investigations are so profoundly multi-level, inter-organizational, and cross-jurisdictional, between Fall 2011 and Spring 2014 I elicited perspectives from other vantage points through a week-long site visit at a state department of public health and laboratory, and in-person and phone interviews with public health and regulatory officials from local, state, and federal levels.

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83 I discuss this in more detail in Chapter 2, and draw this concept from Suchman (1997), who derived it from Latour’s concept of a “center of calculation” (1987).
84 Geiger & Ribes (2011).
(FDA and USDA). In many of the interviews, I adopted an oral history approach, asking the interviewee to describe the evolution of outbreak surveillance in terms of their own career trajectory, focusing on the time period from the 1970s to the present.

What came to the fore early as I began interviewing public health scientists when I first arrived at the CDC, was that problem-solving was a central activity they were continually engaged in, in maintaining and operating surveillance systems, and creating new ones, from the vantage point of the center of coordination in a federalist network of other federal agencies and state public health laboratories and departments of health and agriculture. Interviews provided me with much more dynamic accounts of past problem-solving than the scientific literature, though the literature was helpful when I required more detailed information about techniques, procedures, dates, or events. Collectors of oral history accounts must of course acknowledge the fallibility of human memory; one way to reckon with this issue is to not use oral histories as to reconstruct detailed sequences of events. With oral history interviews, I could delve into aspects of organizational culture and change, and contingencies and aspects that were not published in the scientific literature. Finally, since no archival material is yet available for the 1974–2008 time period, oral history interviews and scientific literature provide two of the best sources of material to represent this history.

Some interviews were less oral history-oriented, and more focused on specific topics of interest, however, my interview approach remained open-ended, to accommodate discussion of issues that participants deemed relevant and important. Interviews ranged from 20-120 minutes in length, and when permission was granted, digitally recorded. I conducted interviews place in-

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86 Starting from around the 2000s, an increasingly prolific amount of information can be found online. I give oral history accounts primacy in this chapter, so have chosen not to include too much online material from agency websites in this chapter.
person and by telephone. Most interviewees were identified through either targeted or snowball sampling. One key methodological challenge that shows how profoundly large-scale and collaborative this arena is, was that each interviewee would recommend multiple colleagues for me to speak with, creating significant scoping issues for the project.

I used many “documents” for the analysis, a bulk of them electronic in nature. Literature from public health, medical, scientific, and social scientific fields was of course a significant resource. Agency websites contained a wealth of information of various types, such as regulations, guidances, program descriptions, reports, press releases, standard operating procedures, protocols, and forms. Because outbreaks, especially major ones, attract media and political attention, I also incorporate information from news stories and Congressional and regulatory hearings in the project. While at the CDC, I received access to copies of select outbreak-related records (e.g. correspondence, summary data), most of which had already been collected for others’ Freedom of Information Act (FOIA) requests.

As I conducted the ethnographic fieldwork, collected oral history interviews, and analyzed documents, I began to realize how much I wanted to historicize the work that I was observing, even beyond the 1990s context in which the early warning system was first put into place. Tauxe, who has served as a key contact for the dissertation and has himself published in the history of public health literature, 87 emphasized the importance of a National Conference on Salmonellosis organized by the CDC in March of 1964. As I read the proceedings of this conference, the lively presentations and intense discussions I read in the transcript sparked my interest in historicizing the current outbreak detection and response system with an examination of the dynamics of building an earlier one—the National Salmonella Surveillance Program (NSSP) from the 1960s. In addition to collecting documents about the NSSP from the CDC, I

also consulted archival records from the Atlanta branch of the National Archives, which houses the CDC’s official historical records, as well as the Adelphi branch of the National Archives, which houses FDA records.

As an outgrowth of strategically situating myself in such a distributed and intricate phenomenon, I have had to make choices about which aspects to pursue at a deeper resolution than others, and which lines of inquiry to pursue and not pursue. As a result, this dissertation focuses more on the coordinating center of the CDC, somewhat on the FDA, and less on the USDA. Much of this is an artifact of this particular historical moment—a lot of the social action I observed during my fieldwork was happening in FDA-regulated foods.

The dissertation is also focused on the federal level, and thus does not capture the full multi-level aspect of the phenomenon. I would like to have conducted more work at the state and local levels, because the majority of foodborne outbreaks, and thus the investigations, remain at the “front-line”, though I have tried to address this in part with a site visit at the Wisconsin State Public Health Department. However, I did not reach the local level in any significant way, though county public health nurses are often the ones conducting food history interviews, and environmental health inspectors are often the ones who collect evidence from firms. Reaching the front-line would be a good direction for future work. In addition, I only scratch the surface on consumer, producer, and legal perspectives, which are all important components of foodborne outbreaks. There are many more interesting vantage points in this phenomenon, and many different stories could be told from different locations in the “farm-to-fork continuum.” However, the dissertation does help to explain how the “farm-to-fork continuum” became a holistic policy frame for conceptualizing how we eat together as a society.
Chapter Outline

The rest of the dissertation proceeds as follows.

Chapter 2 takes an ethnographic look at how officials engaged in collaborative sensemaking inside of the confirmatory pathway for provisional knowledge in the year 2012, to coordinate their inter-jurisdictional, multi-disciplinary outbreak investigation work. The chapter proceeds by discussing the infrastructure around the early warning system; the makeup of the system within the broader infrastructure; and provides ethnographic windows into the sociotechnical work and collaborative sensemaking throughout the confirmatory pathway, from patient to isolate, patient to case, isolate to pattern, pattern to cluster, and cluster to outbreak.

Chapter 3 begins the project of historicizing the ethnographic snapshot provided in the previous chapter, by first sketching out a broader history important for understanding disease surveillance infrastructure, explaining three key elements: disease reporting; the rise of the CDC; and identifying, measuring, and classifying Salmonella. The chapter then begins to tell a more detailed history of how a fractional picture of “large scale food infection” in the US first began to appear in the 1940s, partially illuminated by growing surveillance efforts focused on Salmonella. I chart the rise of what I call the “fowl problem,” a cycle of Salmonella disease transmission intensified by the industrialization of the US poultry industry, and conclude the chapter with an examination of a first major national policy debate on the fowl problem.

Chapter 4 continues the historical examination of foodborne knowledge infrastructure by analyzing the building and evolution of the National Salmonella Surveillance Program (NSSP). I describe the NSSP’s role in helping to support the investigation of two contamination issues (one, a different manifestation of the fowl problem, a hospital-based epidemic associated with
shell eggs; and two, contamination of nationally distributed instant non-fat dried powdered milk).

Finally, I examine debates about *Salmonella* control between the 1950s-1970s. This chapter argues that the NSSP was established as a second-order system, building upon previously lain infrastructure to help strengthen *Salmonella* surveillance, helping fill in the fractional picture of salmonellosis from the 1940s-1950s and making the disease more visible at a population level.

Chapter 5 examines foodborne knowledge infrastructure building in the period between the 1970s-1990s, as the NSSP stabilized but shifted in its utility. While public health officials would not build the early warning, real-time system of outbreak detection and response until the 1990s, the interim period between the NSSP and the early warning system was an active one. In particular, the fowl problem resurged, in the form of a *Salmonella* Enteriditis epidemic caused by shell eggs. In the 1990s, *E.coli* O157:H7 radically shifted the politics of consumer advocacy in the food safety arena.

Chapter 6 chronicles how the early warning, real-time system for foodborne outbreak detection and response was built. I explain how PFGE became a salient for PulseNet to incorporate into the system; PulseNet’s genesis and early development; and the concurrent creation of FoodNet. Then, I describe how system-builders attacked a set of critical problems all related to standardization: shortening protocols, formalizing a testing system, and implementing information technology. After that, I chronicle how standardization began to payoff as data in the system accumulated, and the new, diffuse outbreaks became more visible in the database. The last empirical section of the chapter examines how the epidemiology and regulatory interface became more integrated over time, aimed at attacking the (long-standing) critical problem of tracing the food back to the source.
Chapter 7 follows one major multi-state outbreak from start to “finish,” with the aim of capturing the dramatic way in which a single investigation unfolded. I chronicle the drama involved with uncertainty management and dynamic situations as they coalesce. I do not stop the journey at the boundary of the technical investigation, but examine the public inquiry provoked by this major crisis. Using an anthropological model, I argue that the phases of the outbreak can be understood as an unfolding social drama, involving breach, crisis, and redress. Though the outbreak occurred between 2008-2009, the social drama continues to unfold, in policy and legal arenas, continuing to express a dynamic tension between overflow and containment.

Chapter 8 concludes the dissertation.
Chapter 2. Inside the Confirmatory Pathway for Provisional Knowledge: Collaborative Sensemaking in Foodborne Outbreak Detection and Response

Hypotheses are nets: only he who casts will catch. –Novalis, as quoted by Karl Popper, *The Logic of Scientific Discovery*

So all life is a great chain, the nature of which is known whenever we are shown a single link of it. –Sherlock Holmes in Arthur Conan Doyle’s *A Study in Scarlet*

Chapter Introduction

On my first day as a visiting student researcher at the CDC, in February 2012, Tauxe brought me into the office of Ian Williams, the Outbreak Response and Prevention (ORPB) Branch Chief. There, a group of five epidemiologists were gathered around his desk, listening in to a conference call about an emerging multi-state outbreak of *E.coli* serogroup O26. Tauxe started jotting notes for me to help me make sense of the situation. During the call, participants (from multiple states and agencies) discussed the evidence they had gathered about the emerging outbreak, examining sandwich ingredients, pending case demographic data, and the design of new questionnaires. As they talked, I fixed my eyes on Tauxe’s pen and paper to help ground the information I was seeing and hearing. From his notes, I learned that *E.coli* O26 is a shiga-toxin producing pathogen, and in this outbreak there might be up to six pulsed field gel electrophoresis (PFGE) patterns involved, according to preliminary matches in the PulseNet database. Three question marks followed the number six that he jotted down on the paper.

The cases were all female, with one hospitalization. Investigators suspected some type of sandwiches with lettuce as the possible exposure. Someone on the line asked, “what kind of lettuce? Romaine, iceberg?” Other sandwich items in common were sprouts and tomatoes. “Name the restaurant chain?” Tauxe wrote on the paper, underlining the question, and looked at me pointedly. I nodded, weighing the dilemma; “when was the information right enough to take action?” I wondered.
Participants on the call touched on that issue briefly, and together, quickly agreed that the feeling was not quite yet—this investigation was still “ongoing.” At the moment, they knew the restaurant chain of interest, but there were still questions to answer about what the specific vehicle was. There would be a follow-up call later in the afternoon to discuss incoming data that would be filtering in from the state and county-level investigation work, from the “front-line” professionals who had their “boots on the ground”—more patient interviews, more distribution records. “More to come, folks,” Williams said, a phrase I would hear him utter often over the next few months.¹

“More to come” is an especially resonant phrase because it hints at the special dynamics of making knowledge and gathering evidence in this domain. During foodborne outbreak investigations, there is a primary reason public health officials produce knowledge— to guide the taking of actions to find and end outbreaks, and prevent further and future illnesses. Outbreaks are emergencies. They are time-pressured situations that involve a coalescing investigation process, one characterized by what Karl Weick (1988) calls enacted sensemaking. As Weick states, “To sort out a crisis as it unfolds often requires action which simultaneously generates the raw material that is used for sensemaking and affects the unfolding crisis itself.” Broadly, officials involved in disease control are highly concerned with maintaining situational awareness, defined as “the ability to utilize detailed, real-time health data to confirm or refute and provide an effective response to an outbreak.”²

To help them maintain situational awareness, sort out unfolding crises, and manage time and uncertainty, investigators use a collaborative model of sensemaking. To be fast and right,

¹ Field notes 2/7/12
² Olson et al (2013). These scholars actually point out that while this term is frequently used in public health, it is not defined in operational terms. The main “working definition” they cite is “real-time analysis and display of health data to monitor the location, magnitude, and spread of an outbreak.”
they work together to enact a goal-directed process of collecting and interpreting data, and making decisions and taking actions which themselves produce more data, until their ultimate goal is reached—identify the source of the outbreak. While inside of the confirmatory pipeline for provisional knowledge during fieldwork, I wrote in my fieldnotes, “to be inside of this fieldsite is to feel movement.” As a particular kind of network, the confirmatory pipeline is what Manuel Castells (2000) calls a “space of flows,” the “material organization of time-sharing social practices,” which are “purposeful, repetitive, programmable sequences of exchange and interaction between physically disjointed positions held by social actors.” During outbreak investigations, making knowledge and taking action are tightly intertwined.

One way to understand the tight relationship between making knowledge and taking action is through what Brian Campbell (1985) calls “the problem of adequate knowledge,” or what is reasonable to know for practical purposes. In his study of the uncertainty management of scientists participating in a government inquiry on the social, economic, and environmental impacts of a proposed natural gas pipeline, he found that scientists often discussed the problem of adequate knowledge. In foodborne outbreak investigations, the problem of adequate knowledge often confronts officials, particularly in relation to the scope and expense of the strategies they use, which must be accountable to a resource-limited policy environment. For example, during an outbreak where cheese was a suspected source, but a specific type had not yet come to the fore as the most likely candidate, public health and regulatory officials had to decide on a limited subset of cheese types to sample—which cheeses were most likely to harbor
the pathogen, based on their material characteristics? It was deemed too expensive and time-intensive to sample every possible cheese that could be contaminated.\(^3\)

In this chapter, I take an ethnographic look at how officials engage in collaborative sensemaking, as they coordinate inter-jurisdictional, multi-disciplinary outbreak investigation work to advance multi-state outbreaks along the confirmatory pathway for provisional knowledge. I focus on the practical actions that constitute everyday aspects of “disease detective” work. This chapter proceeds by describing the infrastructure around the early warning system; the makeup of the system within the broader infrastructure; and provides ethnographic windows into the sociotechnical work and collaborative sensemaking throughout the confirmatory pathway, from patient to isolate, patient to case, isolate to pattern, pattern to cluster, and cluster to outbreak.

**The Infrastructure Around the System**

As I discussed in the first chapter of the dissertation, I combine system and infrastructure approaches by looking at how a coherent public health surveillance system (the confirmatory pathway for provisional knowledge) interlinks and interfaces with broader infrastructures. Infrastructures are made up of multiple, layered systems and networks, and it is this sensibility I would like to bring to describe early-warning, real-time foodborne outbreak detection and response.

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\(^3\) During the outbreak, one epidemiologist ruefully commented that enumeration studies to quantify contamination (and shed light on infective doses) are not done often enough; they must be conducted in real-time to gain accurate measures, but do not directly contribute to solving outbreak investigations (Field Notes, 9/9/2012).
Early in my fieldwork, I mistakenly used “PulseNet” as an encompassing term for early-warning, real-time foodborne outbreak detection and response. This was before I had shifted to an infrastructure-oriented approach. However, in making this error, I learned that to officials, “PulseNet” is limited to the outbreak detection component of the system, a network comprised of state and federal (CDC, FDA, USDA) public health laboratories.\(^4\) In each state, PulseNet members use the same standardized PFGE protocols to subtype pathogens from each case of foodborne illness residing in their respective state, and upload the PFGE and associated case demographic information into a shared database. But the investigation of outbreaks required not just the laboratory network, but an array of other epidemiologic, regulatory, and environmental health networks as well.

A key goal for keeping the system “real-time” is for state public health laboratories to perform PFGE within 48 hours of receiving the pathogen. PulseNet leaders call the network “decentralized,” since state public health laboratory technicians perform PFGE and input the results from their own labs, and thus require equipment to perform PFGE, as well as the requisite computer hardware and software.\(^5\) PulseNet members all use the same software analysis program, Bionumerics, to provide an initial analysis of the PFGE data. At the CDC, the PulseNet Bionumerics database is coordinated by a database unit at “PulseNet Central.” The database unit is responsible for curating the inputted subtype data, confirming its validity, and continuing to maintain the interface between state public health laboratory network members and the CDC.

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\(^4\) PulseNet is primarily a domestic network, although there is a separate international network (PulseNet International).

\(^5\) Personal computer equipped with an Intel Pentium CPU or better, 256 MB RAM or more, 65 K color graphics or better and Windows 98, Windows NT 4.0, or higher, and a high resolution screen 1024x768 or higher; true color
Also housed at PulseNet Central are two other units, a reference unit and a methods development and validation unit. The reference unit is largely a system-maintaining entity focused on the present, helping state lab network members troubleshoot PFGE protocol problems, conducting training activities, updating protocols, managing relationships with PFGE equipment vendors, and other miscellaneous administrative activities to keep the network running. The methods development unit is responsible for coordinating the development and use of typing methods beyond PFGE, focused on the future of the network. All the PulseNet units are housed within the Enteric Diseases Laboratory Branch (EDLB), a CDC organizational entity responsible for a broad range of laboratory-based work related to enteric pathogens.

Since PulseNet is the outbreak detection piece, another network called OutbreakNet is the response piece. OutbreakNet is a network of epidemiologists in state public health departments who either specialize in or have duties related to foodborne disease surveillance. These duties include conducting follow-up interviews with cases during multi-state outbreaks, and maintaining responsibility for gathering and processing investigational information, and keeping it organized. However, it is important to note that because of the multi-level federalist system, state public health department duties often involve coordinating local activities they do not directly perform. For example, county public health nurses are typically responsible for conducting patient interviews or collecting food samples from patient homes, and state and local environmental health and regulatory staff are typically responsible for collecting distribution records and environmental samples from food producers and distributors.

Both on the detection and response sides, the CDC plays a similar organizational role. It operates as what Lucy Suchman (1997) calls a “center of coordination,” responsible for coordinating the “deployment of people and equipment across distances according to a canonical
time table or the emergent requirements of rapid response to a time-critical situation.” The center of coordination concept is useful because it moves away from an artifact-centered view, and instead sees technical workers and the artifacts they use as constituting a holistic “working information system.”

At the center of coordination, every week, database managers send detected “early warning” PFGE clusters to epidemiologic teams within the Outbreak Response and Prevention Branch (ORPB), which operates as the coordinating entity for the OutbreakNet network. ORPB is responsible for “cluster management,” and coordinating the distributed investigation activities conducted by members of OutbreakNet, who are themselves coordinating on-the-ground investigation activities, some of which are conducted by local public health workers. In addition, ORPB is a key coordinating entity for the public health-regulatory interface, responsible for organizing and running routine cluster management meetings also attended by regulatory agencies. The FDA and USDA have liaisons housed at ORPB, but also have investigatory teams that participate remotely in cluster management meetings.

Federal regulatory officials also coordinate separate investigatory activities through their field-based networks. These networks must interface with PulseNet and OutbreakNet, but are distinct, because of the differences in agency roles. The CDC is categorized as a scientific, non-regulatory agency, its procedural chains are subject to different restrictions and considerations than those of regulatory agencies, which are, in short, more rigid because of their more legal context. For example, during outbreak investigations, if food is taken from a patient’s home as evidence, if the food has been opened, it is sent to the CDC. In theory, this is because the sample could have been cross-contaminated, though the likelihood of this is variable, and testing open containers can still provide good “leads” in the investigation. If the food is sealed, it is either
sent to the FDA or USDA (depending on which agency regulates the food in question), or state labs must perform rigid chain-of-custody procedures recognized as equivalent to regulatory agency practices. A sealed food can often provide the “smoking gun” helping to soundly implicate a contaminated product.  

While there are distinct roles for different experts in foodborne disease, it is important not to view detection and response as mutually exclusive. Instead, outbreak investigations have a multi-level, overlapping, iterative character. Though the laboratory detection of clusters is an initiating step, outbreak investigations always involve ongoing laboratory and database work to process and examine incoming and/or additional evidence. Every outbreak investigation involves back-and-forth communication and interdependent work between scientists in the laboratory, in the database, and in the epidemiologic and regulatory interfaces of the investigation. Throughout the course of an investigation, new information is continuously generated and exchanged, through online platforms, emails, conference calls, and phone calls.

The System within the Infrastructure

Now that I have described some of the infrastructure around the system, I will now discuss the makeup of the system within the broader infrastructure—the confirmatory pathway for provisional knowledge. To assist me in this discussion, I have created a schematic diagram of how and where data are created, enter, and move through the confirmatory pathway. I map sequences of actions and the creation and transformation of a cluster into an outbreak in six arenas: the clinic, clinical lab, public health lab, PulseNet Database, Outbreak Investigation, and public health action.

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6 Javier Lezaun (2009) has written about the European Union’s efforts to make genetically modified organisms traceable, which has also involved the creation of rigid chain-of-custody procedures.
I start the confirmatory pathway at the step where a person who eats contaminated food and seeks clinical care becomes a patient (red). The key way that a patient is diagnosed with a foodborne illness is through a procedure called culture—taking a sample from the patient (stool, blood, urine, or even cerebrospinal fluid) and placing the sample in a special medium that encourages particular kinds of bacteria to grow. This takes place in a clinical laboratory. After bacteria are cultured, the pathogen of interest is isolated; that is, a pure strain of a single pathogen is separated from other bacteria (e.g. *Salmonella*, *Campylobacter*). This step is always necessary because in the human body and in the broader environment, bacteria exist in multi-member communities. The pure strain, or isolate (orange), is then shipped to a state public health laboratory. There, technicians use standardized PFGE protocols to “subtype” isolates, giving them more granular information than a pathogen’s strain.

PFGE is a molecular biology technique developed in the 1980s, standardized by public health officials in the 1990s, and works in the following manner, as portrayed by a CDC explanation of the process (Figure 5).
Once a pure bacterial culture is shipped to the state public health laboratory, the scientist takes bacterial cells from the agar plate (step 1), and preps the DNA for subtyping (step 2). Since bacteria have circular DNA, laboratory scientists must use restriction enzymes to cut this DNA into different sized linear fragments (step 3). The fragments are loaded into a gel (step 4), which is placed in an electrical field created by an electrophoresis machine that separates the fragments according to their size (measured in base pairs). The following photograph depicts an electrophoresis system, with a power source that alters the direction of the current on the left, and the chamber for the gel filled with a buffer solution on the right, so that the current can travel, helping separate the DNA fragments.

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When a gel has finished running, the resulting separation of fragments creates a pattern that will correspond to a subtype when it is analyzed. To prepare the gel for analysis, state public health laboratory scientists stain the gel, photograph it, turn the gel into a digital image, upload it into an organism-specific PulseNet database, and perform an initial analysis step called band-marking, which involves clicking on the bands of the gel (step 5).

Illustration 1. PFGE Equipment

8 http://aphl.smugmug.com/Other/Miscellaneous/14495013_8Gr2Vm#!i=3153155874&k=hdVgBTX&lb=1&s=A.
While the Bionumerics software has an automatic band search (Figure 6), the protocol requires the laboratory scientist to check the gel and look for bands that are not properly marked or are unmarked. After a gel is marked, the software automatically assigns the gel an unconfirmed pattern name, which has three components: serotype, enzyme used, and pattern number. While the automatic assignment of a pattern name is useful, since it is not foolproof, PulseNet does not treat an automatically named pattern as a confirmed pattern until it undergoes verification by a database manager at PulseNet Central.

Cluster searches occur at both the local and national level, usually on a weekly basis. The data that can be seen at the local level differs from the national view. While PulseNet databases are shared in that local sites can upload PFGE data to the national level, local sites do not have viewing access to all of the data. Interestingly, this means that the local and national levels see entirely different clusters. Clusters are defined as three or more matching isolates above the baseline, or expected normal level of disease, within the last 60 days.\(^\text{10}\) That local views involve scanning a smaller subset of the total population vastly increases the amount of clusters that can be detected. Clusters of rare patterns, new to the PulseNet database or not


\(^10\) Except for *Listeria*; the pathogen has a longer incubation period, so database managers scan within the last 120 days for *Listeria* clusters.
highly represented, are more obvious. However, some patterns are highly common, making clusters harder to discern. PulseNet Central uses the following graphs of pattern uploads as training examples to illustrate the difference between cluster detection of rare patterns (left) versus common patterns (right) in the PulseNet database (Figure 7).

![Figure 7. Rare and Common Pattern Clusters](http://www.aphl.org/conferences/proceedings/Documents/2008_12th_Annual_PulseNet_Update_Meeting/13-LifePulseNetClusStroikaWilliams.pdf)

Officials are careful to emphasize that PulseNet is a cluster detection tool, not an outbreak detection system. Clusters are only possible outbreaks. Only through an investigation, with consideration of epidemiologic and regulatory data, can officials determine whether a cluster is truly an outbreak. To determine this, they use a variety of systematized laboratory and epidemiologic tools and procedures to produce, analyze, and share information during outbreak investigations. Once a cluster has been detected, outbreak investigation work involves further case interviewing; analytic studies; and product and environment tracing, sampling, and testing (dark blue). Identifying a source is what definitively transforms a cluster into a confirmed outbreak, but since source identification is an ongoing process rather than a single moment, during outbreak investigations, officials will often take public health actions such as hazard messaging to the public, or regulatory actions such as asking companies to initiate recalls or market withdrawals, (purple).

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Now that I have given a high level overview of the confirmatory pathway for provisional knowledge, the next section provides ethnographic windows into the sociotechnical work and collaborative sensemaking that takes place inside of the pathway, following the general flow of the schematic presented above. I provide thick descriptions of the making of thin descriptions, which help illuminate the organizational, material, and social worlds from which data are derived. Thin descriptions should be understood as thinned descriptions, products of winnowing, sifting, and simplifying practices.

From Patient to Isolate

As I mentioned earlier, when ill patients go to the doctor, during diagnosis a pathogen is isolated from a sample (typically stool) at the clinical laboratory either in a hospital or in a regional clinical laboratory used by a hospital.¹² The first step for processing stool is to dilute and enrich it in a broth, which is then added to a plate with specific growth medium to cultivate bacteria. After the plate is incubated for a day so that bacteria can grow, the laboratory scientist performing the culturing conducts “colony picks” to identify “suspicious growth.”

While shadowing a laboratory scientist performing culturing, I asked her how she identifies “suspicious growth.” She told me, “Training!” Suspicious colonies of *Salmonella*, which is what she happened to be isolating at the time, are fairly straightforward bacteria from a visual perspective, because the colonies are distinctive, colorless with a black “fish eye” in the middle.¹³ As I shadowed her, the technician mentioned that she found the work to be

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¹² I did not view this step directly, but gained insight into the isolation process from shadowing a CDC lab technician who performs isolations to support outbreak investigations when necessary.

fundamentally “exponential,” an aspect of the work she felt was not always widely understood. I was interested in what she meant by that, and asked her to expand. She said:

“First you have 35 direct plates, then 35 enrichments, then 3-5 colony picks, then a second enrichment. Soon you are up to over 200 [entities]. 5 samples just turned into 40 [entities]… Everything builds on each other… it’s exponential. I say this as a scientist. You go from 5 to 50 plates on day two. You hope some selective media won’t have growth so you can roll in or roll out. We do everything possible to make sure we’re going down the right path.”

The process of isolating a pathogen from stool, while technical, is not absolutely mechanistic; the process must involve the trained human eye to identify what is “suspicious” based on the phenotypic appearance of the bacterial colony—commonly called a subjective technique because of this human element. This step also reveals an important microbiological aspect of foodborne disease—pathogens do not exist singularly out there in the world, but are members of teeming and diverse microbial communities, and are separated or thinned from environments and communities through technical procedures explicitly designed to do so. This process is not only subjective in terms of requiring a trained human eye to work, but it also involves the human performance of technical work experienced as “exponential,” involving the responsibility for cultivating and monitoring a rapidly increasing number of material entities.

Once an isolate reaches the state public health laboratory, the laboratory scientist there has a number of responsibilities to perform on it. Before they perform PFGE, in many labs the isolate undergoes a form of fast identification of strain or serotype via biochemical testing or enzyme immunoassays. The fast identification serves a couple of purposes. It is a kind of check on the clinical laboratory’s work, and also helps determine if the isolate is a “presumptive positive.” That is, a fast identification gives preliminary results, which can be helpful for keeping those further downstream in the confirmatory pathway on heightened alert. The
confirmatory test, PFGE, takes two or more days for the state laboratory scientist to perform from start to finish. In instances where a detected outbreak is being actively investigated, having preliminary information like a presumptive positive status can be helpful for prioritizing and fast-tracking work.

**From Patient to Case**

When public health workers (primarily county public health nurses) call patients on the telephone to conduct food history interviews varies significantly. In an ideal world, patients would be phoned as quickly as possible, which would mean as soon as a clinical lab notifies a state public health lab that a patient’s isolate is being shipped. Time is of the essence in contacting a patient. Already, the food that caused a patient’s illness was ingested long before the patient’s onset of illness; this time period is called the incubation period, and can vary significantly, depending on the organism or even strain in question. For example, the incubation period for *Salmonella* can range between 6 and 72 hours.\(^{14}\) Several days (often times, more) can pass before first contact between a public health worker and the patient, because the patient has to ingest food, undergo the incubation period, get sick enough to merit a visit to the doctor, have a culture performed, and have that culture sent to the state public health lab (or at least have some communication between the clinical lab and public health lab that includes the patient’s contact information).

In recent years, the CDC has created a program called FoodCORE to improve state and local response to foodborne disease outbreaks. As of 2013, there are seven participating

FoodCORE states. I conducted a site visit in one FoodCORE state, Wisconsin, and one of the objectives this site had developed to improve outbreak response was to achieve “timely and complete routine follow-up” for foodborne disease cases. In a home-rule state with 93 local health departments, county public health nurses performed most of the case patient interviews. However, as part of the FoodCORE program, the state public health department provided assistance to county public health nurses, conducting interviews on their behalf if needed. I observed one of these interviews, which took place over the phone, and excerpt an extended fieldnote here:

The epidemiologist called the patient for an interview, who public health knew had been infected with a shiga-toxin producing *E.coli* because a rapid diagnostic test had been used, but results from culturing the specific subtype by the public health laboratory had not yet been completed. The interview began as most of the interviews begin, with an inquiry into the patient’s illness experience, from a clinical perspective. When they first started feeling sick, what specific symptoms they experienced, what medications they had taken, whether they had any underlying medical conditions. Then, the interview shifted into querying about the patient’s exposures, during a one-week period before the onset of her illness. The patient laughed in surprise, and the epidemiologist quickly assured her “try your best, I know it’s a long time.” The epidemiologist asked the patient whether she had traveled and where, about her pets, her shopping habits, water supply (municipal or well). And then about food: where she normally shopped, whether she gardened or had a special or restricted diet. Whether she ate at a “commercial food establishment,” a cafeteria, fast food restaurant, or deli. Whether she went to a large gathering or party, and what she ate there. As the patient answered questions, the epidemiologist marked down the answers on a printed version of the questionnaire—it was easier to mark answers down on the paper while on the phone, especially in writing out more lengthy information, and to enter the data into the state’s disease database after the interview.

During the exposure period, the patient had gone to a family party, an informal supper after finishing a major do-it-yourself home renovation project. She listed what she had eaten, and emphasized that she had made a couple of the dishes, a banana dessert that was “awesome,” and a pasta salad, which she could rattle off the ingredients for easily, as she stated that she had the recipe right there in front of her. The patient was in her kitchen during the interview. As the epidemiologist went through questions about vegetables, meat, and then fruit, the sounds of the patient rummaging through her refrigerator to retrieve brand
information came through over the phone, as well as the intermittent sounds of a young child chattering in the background. The exposure list went on, yes or no questions. Did you eat mushrooms? Peapods? A pepper other than the one from the party? Radish? Jicama? Alfalfa? No, no, no. Other vegetables I didn’t mention? “Not that I can remember,” the patient replied. Hummus? Fresh salsa? Tofu? No, no, no. The “no” response to tofu had a bemused tone. Chocolate? “Oh yeah!” The patient exclaimed, laughing, “There’s not a candy bar I can remember, but I definitely had chocolate.” When the epidemiologist finished the interview, she thanked the patient for her time, hung up the phone, and entered the symptom and exposure data into the system, thus transforming the patient into a case (Field notes, 6/24/13).

Even though this interview was conducted as soon as possible, upon the state public health department learning that the clinical lab diagnosed this patient with a shiga-toxin positive \textit{E.coli} with a rapid test, the fact that the patient was asked to remember what she had eaten in the week prior to her onset of illness provoked her to laugh, likely because the task seemed challenging at first. As a testament to the infrastructural role that food plays in our everyday lives, most of us probably would not be able to remember a detailed list of all the foods we ate in the last week or two, myself included. However, because the interview was conducted early, days after her illness, the patient still had some of the relevant foods stored in her refrigerator. The FoodCORE program views timely and complete routine food history interviewing as a best practice, to increase the patient’s recall of consumed foods, and also increase the likelihood that they still have foods available for pathogen testing, if they become an outbreak-associated case.

At this intimate interface between the patient and the public health system, I was struck by the thickness of this eater’s social life as it entwined with food. I learned that her family had just completed a renovation project, and that she loved to cook. I learned that this eater does not enjoy tofu, and adores chocolate. Over the phone, I heard the sounds of her child’s voice echoing off her kitchen walls, and even the sound of her refrigerator opening and closing. I felt a conversational rapport between the public health worker and the patient, the former gently
encouraging the patient to try to remember what they ate and being thankful for the patient’s time, the latter trying to be helpful to a greater good and spending thirty minutes of her time answering hundreds of questions about what she ate last week, while busy managing a child who boisterously sought her mother’s attention. Through the technique of the food history interview, the thickness of social life and eating were thinned. As a result of this process, the patient became a case, constituted as a set of demographic variables and list of possible disease exposures, and recorded in the state’s electronic disease surveillance system, to be possibly linked to a future outbreak.

**From Isolate to Pattern**

As part of its center of coordination role, scientists at PulseNet Central hold frequent trainings for network members to learn how to perform PFGE and use BioNumerics, which are important mechanisms for keeping a standardized network operating. I shadowed this training, and provide an ethnographic account of laboratory and database work from that perspective.

Six state laboratory scientists filtered in to the CDC’s PFGE Reference Unit lab, to learn how to produce the “beautiful gels” of a well-performed standardized PFGE protocol. The training commenced with the lab’s unit chief asking everyone to go around and describe their roles and levels of PFGE experience. There was significant heterogeneity in both roles and experience. One laboratory scientist from a state public health lab, the most experienced there, was only responsible for performing PFGE and was there to thus brush up on her skills, while another had very little PFGE experience, but was preparing to take on the responsibilities of the person in her lab who performs PFGE and was about to retire. Jumping off on that point, the unit chief said that continuity in the lab was one of the most important aspects of good PFGE. While it is science, it is also an “art form.” As she said this, the training participants nodded vigorously in agreement.
The training consisted of everyone performing a PFGE protocol from start to finish—taking bacteria from the isolates, growing them up on an agar plate, preparing cell suspensions and PFGE plugs, setting up an electrophoretic gel and running it using proper conditions, then imaging the gel to create a TIFF image for uploading into the BioNumerics database. Like all molecular assays, PFGE is an intricate, detailed, multi-step process. Performing a PFGE protocol from beginning to end during the training allowed the state laboratory scientists to engage in hands-on learning, and ask questions along the way. While some had come to the training with specific questions and issues, during the training, some new questions emerged, provoked by slight differences between the state laboratory scientists’ usual practices and how the practices were demonstrated in the training. For example, during the step of using a wet swab to remove cells from the agar plate, the unit chief described how rolling the swab on the cells was better than scraping them. While *Listeria* was more robust and could handle a scraping motion, other pathogens may not. Seeing the rolling action provoked one trainee to laugh a bit; she remarked that she usually scraped the cells with the swab, but would know exactly how to roll the swab in the future (Field notes, 5/1/12).

Many of the themes illustrated by this field note have been widely discussed in STS lab ethnographies: visual language and representation are important components of lab science; practitioners often describe laboratory work using art as a metaphor; and in addition to theoretical knowledge, lab work requires tacit, bodily knowledge, which cannot only be conveyed in writing.\(^{15}\)

While these themes are not unique to the PulseNet network, what I wish to point out is the importance of setting, particularly as it relates to standardization. Most STS lab ethnographies have examined labs in academic settings. As Holmes (1990) and Shankar (2004) discuss, academic labs are different from government, corporate and clinical laboratories. Academic labs have fewer record-keeping and institutional accountability mandates, and are not as routinized as the other kinds of labs. In the PulseNet network, extensive standardization and reproducibility are central values, to achieve system goals. While researchers from different labs who are in the same field or use the same techniques may have some interest in pursuing

reproducible results, what is important to understand about the PulseNet context is that PFGE can only be considered a confirmatory technique certifying that indistinguishible patterns are part of the same outbreak if the labs that produced the patterns are known to produce reliable reproducible results.

Thus, PulseNet Central puts forth significant effort to keep the network standardized, not only holding trainings like the one described above, but developing extensively detailed manuals and protocols (which they call Standard Operating Procedures), and devoting extensive time to helping state laboratory scientists “troubleshoot” PFGE problems that arise. If PFGE gels do not run correctly, laboratory scientists have to figure out whether the problem relates to a mistake they made in the procedure, a laboratory-level issue with one of the ingredients, a malfunctioning piece of equipment, or sometimes even a network-level issue with a manufactured ingredient.16

The state public health laboratory scientist’s work does not end at the bench; after the gel is turned into a digital image file, the state public health laboratory scientist is responsible for band-marking the gel, a coding process that involves not only the computer, but also the human eye and hand, which I describe in the next ethnographic moment of how a gel becomes a pattern.

During the BioNumerics training, a laboratory scientist offered to share her computer with me, so that I could look over her shoulder. Each spot in the training was precious, so I was not actually taking the training, but simply watching it. A database manager started the session, and brought up an image of a PFGE gel on a Power Point slide. The laboratory scientist leaned over and whispered, “I’ve never seen a gel this beautiful! I should change labs.” We shared a smile. Later, I asked her later why she said that. She told me that she mostly works with “bot” (Clostridium botulinum). Because of bot’s large DNA molecules, if the samples are not very fresh, the bands will likely bunch up in the middle during electrophoresis, which creates smeary, blurry gels. Similar to the PFGE training, the BioNumerics training was hands-on, and took training participants through the uploading and marking processes from start to finish: fixing the color of the image, marking a few bands against the standard, normalizing the image, linking lanes to a database entry, marking the bands, adding the available isolate metadata (sex, age, species/serotype, source state,

16 Interview, Molly Freeman, 2/10/2012.
city, source type, source site). During the band-marking step, following the instructions, the laboratory scientist zoomed in on a lane, and began to mark its bands, rendering it into a pattern. To include me in her process, when she got to an ambiguous band she stopped, so we could puzzle together over a key question: was that band a fat singlet or a doublet? She looked at me quizzically, and I shrugged my shoulders in response. My untrained eyes could not tell. As the participants completed the band-marking exercise and moved on to conduct pattern comparisons and cluster analyses with the help of the software, another database manager emphasized, “The computer is just a tool. Your eyes always determine whether a pattern is similar” (Field notes, 5/2/2012).

The following figure, a training slide developed by PulseNet Central, helps illustrates the singlet vs. doublet interpretation issue.

![Figure 8. Band-marking instructions](http://www.pulsenetinternational.org/assets/PulseNet/uploads/bionumerics/15-PFGE_tips_tricks_interpretation2011_MF.pdf)

BioNumerics has an automatic band-marking feature, so the human process of band-marking is not usually a matter of manually clicking on all of the bands in a pattern, but rather, to check the automatically coded bands for accuracy. As part of the band-marking process, laboratory scientists are asked to zoom in on ambiguous bands, to look for indentations, differences in color, and separation. As a trainee, I found band-marking hard and was exceedingly slow at performing it. When I shadowed the PulseNet Central database managers doing their verification checks, I observed that their eyes could scan gels at a rapid pace. With experience,

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the database managers explained, they develop a “trained eye” that allows them to discern similarities and differences in bands very rapidly. As Charles Goodwin (1994) notes, members of a profession develop what he calls “professional vision,” or “socially organized ways of seeing and understanding events,” so that phenomena can be transformed objects of knowledge, through analysis within a expert perceptual field.

As I stated above in the overview section, once a gel is marked, BioNumerics automatically assigns the gel an unconfirmed pattern name, using a continuously running script that utilizes an algorithm to name patterns. However, patterns only become confirmed patterns when they receive an official name from the PulseNet Central database managers. Every week, database managers routinely scan their organism’s database to identify newly uploaded and named patterns from the states, conduct quality checks, and in verifying patterns, confirm their official names. After verification checks, the bacterial strain that came from a sick person now has a key data identity—e.g. JAXX01.0015.  

From Pattern to Cluster

As one database manager pointed out, their role was that of a “liaison” between the laboratory scientists performing PFGE and the epidemiologists conducting the outbreak investigation. They aimed to keep “everyone in the loop,” the “line of communication” open, and the information moving through the confirmatory pathway. The following CDC diagram highlights the liaison role played by the database managers.

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19 Field notes, 2/29/2012.
Every Monday, the entire PulseNet database team meets to go over their cluster searches together, after they have each respectively checked for new uploads from the state labs. Typically, they run searches by examining uploads from the past 60 days. They check to see if the pattern is above a “baseline” level. This means that they compare the observed amount of disease with what they have seen historically, during the same period of time in the previous year. If there are three or more matching patterns above a baseline level, those become a cluster. Database managers create a cluster report, which includes a line list of cases as well as a frequency graph of the pattern of interest. A line list is a core epidemiologic tool for organizing data concerning the epidemiologic triad—person, place, and time. It is usually presented in tabular form. If one of the isolates in a cluster is from a food source, this can provide an initial “indication of source” in a cluster.

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21 The number three was selected as a pragmatic rule of thumb, personal communication.
22 A database manager I shadowed gave the example that if a pattern has been associated with chicken, they would typically contact a USDA VetNet database manager to see if they have additional matches (Field notes, 2/29/2012).
After the database managers complete the cluster reports, they forward them to the epidemiologists in ORPB. They also post the cluster reports in a protected online collaboration space (SharePoint), where both PulseNet and OutbreakNet state members can access them.\textsuperscript{23} Even after they submit their weekly cluster reports, database managers continue to play a liaison role. For example, during outbreak investigations, ORPB may want the database managers to keep a heightened watch for states submitting new uploads of possibly matching patterns, and keep tabs on the results of additional lab testing often conducted for outbreak investigations.\textsuperscript{24}

**From Cluster to Outbreak**

Clusters can be detected at the state or national level. If a state detects a cluster, it will post key information about the cluster in SharePoint. The PulseNet database team will initiate a national search on the cluster’s pattern, which often reveals more clusters or at least, individual cases, from other states. If at the national level a cluster is detected, similarly, PulseNet Central will post the cluster on SharePoint and communicate with the states where the cases are located. At the national level, clusters can appear as quite geographically dispersed and thus not be discernable at the local level, or can link what appear to be local clusters occurring in different states but are likely caused by a common source.\textsuperscript{25}

If a cluster involves more than one state, federal officials then launch a multi-state cluster investigation. Cluster investigations are coordinated by epidemiologic teams in ORPB, who

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\textsuperscript{23} Members in the states can also perform cluster detection, but from a state vantage point, as opposed to the national vantage point taken by the CDC.

\textsuperscript{24} Examples of additional lab testing include running an additional PFGE on an isolate with a second enzyme, or performing Multiple Locus Variable Number Tandem Repeat Analysis (MLVA), a sequence-based DNA fingerprinting method more discriminatory than PFGE.

\textsuperscript{25} http://www.pulsenetinternational.org/networks/usa.
work with state OutbreakNet members, who are in turn responsible for coordinating and conducting local cluster and case investigations. When epidemiologic teams in ORPB receive cluster reports from the database managers, they determine which clusters to investigate in a process they call “triage.” During the busy summer months, when bacteria are more active, clusters come in a “firehose” of up to 40 at a time, they must prioritize which clusters to investigate, looking into the available evidence and identifying whether clusters have cases that look promising for “follow-up.” Summer is busy season, with the seasonality of bacterial activity shaping a phenomenal rhythm of work for outbreak investigators.

As I observed outbreak investigation work, I saw epidemiologic teams using a heterogeneous mix of highly technical language for outbreak work objects and generic investigatory language for making the connections between objects; outbreak investigations consist of making epidemiologic “links” between heterogeneous pieces of evidence, until those links can be confirmed, transforming a suspected outbreak into a confirmed outbreak by “implicating” the contaminated food as the outbreak’s source. Investigators often spoke of the importance of “hunches” and “getting to the right question.” At first, I attempted to separate the technical terminology from the generic in my questions. In one conversation, I asked Tauxe, “What do you call the evidence you gather, you know, like clues or something?” He responded, “Well, we call them clues.” ORPB’s Deputy Chief, Casey Barton-Behravesh, emphasized to me that “epidemiology is more than just statistics, it is puzzle-solving.”

As I gained more familiarity with outbreak investigations, I decided that the mix of the technical and investigatory language and puzzle-solving practices, and collaborative sensemaking, should be central to the analysis. In the media, popular culture, and educational

26 Interview, Casey Barton-Behravesh, 4/19/2012.
initiatives, CDC scientists are commonly described as “disease detectives,” or “gumshoe epidemiologists.” While at first I had thought of these categories as externally-focused, to promote public understanding of the CDC’s work, as I gained more ethnographic familiarity with outbreak investigation practices I realized that I could gain analytic purchase from the category of “disease detective” as constitutive of both their scientific identity and collaborative sensemaking work.  

Carlo Ginzburg (1980) wrote a creative essay comparing the investigatory work of Arthur Conan Doyle’s fictional detective Sherlock Holmes, art historian Giovanni Morelli, and psychoanalyst Sigmund Freud. All three investigators, he argued, had a common “heart” to their epistemology—the idea that “reality is opaque; but there are certain points—clues, signs—which allow us to decipher it.” One major connection between these three forms of investigatory work, he argues, is that they all arose during the late 19th century, in a broad paradigm he calls “medical semiotics.” Medical semiotics gained ascendance across a swath of the medical and human sciences.

Ginzburg defines medical semiotics as “the discipline which permits diagnosis, though the disease cannot be directly observed, on the basis of superficial symptoms or signs, often irrelevant to the eye of the laymen.” These signs, he states, are “tiny details [which] provide the key to a deeper reality, inaccessible by other methods.” Tiny details come to matter greatly in foodborne outbreak investigations. Precisely when did the case start feeling sick? What did the case eat for breakfast last Thursday? What does the food labeling say about when the product was manufactured? Are the roasted peanuts in the factory stored near the raw peanuts?

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27 I discuss this from a historical perspective in Chapter 3.
Sensemaking during outbreak investigations involves generating heterogeneous questions and interpreting heterogeneous clues to find the deeper reality—the source of the outbreak.

Claudio Rapezzi, Roberto Ferrari, and Angelo Branzi (2005) have examined investigative characteristics in a cross-section of detective literature to draw analogies to diagnostic reasoning in medicine. They identified six important characteristics: capacity for observation and logical reasoning; existing knowledge; ability to conduct an interview; ability to recognize inconsistencies; action, obstinacy, and initiative; and criticism of slavish adherence to institutional procedures. Their focus on observation resonates with Ginzburg’s description of medical semiotics. In describing the importance of logical reasoning, Rapezzi et al invoke philosopher Charles Peirce’s concept of abduction as a process of forming an explanatory hypothesis. Stefan Timmermans and Iddo Tavory (2012), also building on Peirce, describe abduction as discovering relationships between phenomena and observations to discern a hidden cause and effect, often informed by past insights, and to create new ones about the situation at hand. In contrast to the more static and linear deductive and inductive modes of reasoning, abductive reasoning is dynamic, oriented to problem-solving, improvisation, innovation, and a trial and error mode of work to create new knowledge about surprising events in the world.

Williams, who during his tenure as ORPB branch chief has worked to make organizational processes for making outbreak investigation methods and procedures more systematic, reflected on how “foreknowledge” and extensive hypothesis-generation played a role in foodborne outbreak investigation work, comparing different disease domains:

I used to work in Hepatitis. You know how it is transmitted. For example, Hepatitis C, it's blood to blood stuff. Very rarely through sex. So when you walk into an outbreak of Hepatitis C, your universe is right here already [places thumbs close together]. It's going to be these sorts of things that I'm looking at. When you walk into one of these [foodborne or zoonotic disease] outbreaks, it could be the
food, it could be the baby chicks, the baby turtles, well the universe is relatively smaller [holds hands out to the sides], it's not like somebody walked by you and sneezed kind of stuff [stretches hands out wider], it's just that you've got to take a little bit of a larger view and go through this process of really hypothesis generating in a way that you don't have to do when your universe is much more narrow.\textsuperscript{28}

It was common to talk of investigations as a process of narrowing the “universe” of exposures\textsuperscript{29}. The line between foodborne disease and zoonotic disease can be a blurry one, since ultimately, many enteric pathogens come from the guts of animals. When PulseNet detects an initial cluster, investigators must keep in mind that it could be either a foodborne or zoonotic outbreak. For example, a \textit{Salmonella} outbreak could be associated with a food, a pet turtle, a goat from a petting zoo, or pet food. According to Williams, foodborne disease sat somewhere in the middle of Hepatitis and airborne disease, in terms of the number of possible vehicles of exposure. This exemplifies one way in which uncertainty is bounded in foodborne outbreaks. Identifying a common source is a task made manageable through an investigative process involving specialized knowledge practices and ongoing data collection.

To turn clusters into outbreaks, investigators decipher and link heterogeneous clues. They create many thin descriptions and weave them together through abductive reasoning. ORPB uses a highly collaborative model to conduct their sensemaking. Every week they meet for “cluster management” meetings. This involves going through each cluster, discussing the status of each investigation, and collectively brainstorming about next steps. Before joint cluster management meetings, individual teams are responsible for organizing the cluster data by creating line lists and other summary data. While Power Point has been and remains a frequently used tool to pull summary data together, helping investigators maintain situation awareness, over

\textsuperscript{28} Interview, Ian Williams, 5/7/2012.
\textsuperscript{29} Enteric pathogens can be transmitted multiple ways, though mostly through foodborne or zoonotic modes of transmission.
the past couple of years, ORPB has been piloting a web-based “knowledge management platform,” with an array of data visualization tools built into it. Recently, they have called this platform SEDRIC (System for Enteric Disease Response, Investigation, and Coordination). During cluster management meetings, the branch sits in front of a screen which projects outbreak investigation data via SEDRIC, flipping through and discussing each cluster as summary data are displayed for all to see. The following photograph portrays a typical cluster management meeting:

Illustration 2. ORPB using SEDRIC in a cluster management meeting. Courtesy of CDC.

In this picture, the projected image uses PulseNet data to create a visual map of a multi-state cluster that portrays the different numbers of cases in the various states that are part of the cluster. This particular cluster is concentrated in the western and eastern parts of the country. Even from the limited information available in the picture, many questions could be asked.
Because the cases are concentrated in two different geographic areas, even if they share the same pattern, is this truly one outbreak, or is it two or more? Does temporal patterning of cases reveal any hints as to whether this is one outbreak or not? Why is there a high concentration of cases in one of the eastern states (as indicated by the red color)? In the western states, is the potential common source a product that is distributed only in the western part of the country?

Though specific teams take ownership of individual clusters and conduct the actual data organization work before and after the meetings, meetings are an important collaborative space where the entire branch puzzles through each cluster together, generating the kinds of questions I described above. In this section, I present a real slice of talk in a typical cluster management meeting.

Epidemiologist 1: Next on the hit parade is *Salmonella* [Serotype A].
Epidemiologist 2: There are 8 isolates in this cluster, it is a brand new pattern in PulseNet, 3-4 cases report drinking [Brand X] bottled water. From other cases, we have a [Brand Y] breaded chicken hypothesis which is interesting but uncertain.
Epidemiologist 3: This breaded chicken scenario echoes some scenarios from the past. People perceive the product as ready to eat. Is this a ready-to-eat product or a ready-to-cook product?
Epidemiologist 2: I think the product is flash-fried so it is brown, but it may be undercooked.
Epidemiologist 3: How consumers microwave and then have a thorough cook is an unresolved problem. Are instructions given to them adequate? Do the consumers follow the instructions?
Epidemiologist 1: It sounds like we need more information from a couple more cases.
Epidemiologist 4: I think [Brand X] is a regional water.
Epidemiologist 2: If it was nationally distributed you’d think we’d see more cases.
Epidemiologist 1: There’s not enough of a story here yet.
Epidemiologist 5: We’ve not seen this pattern in VetNet before, should we look at closely related [Serotype A] patterns from there? Would there be more cases for consideration?
Epidemiologist 2: I know there has been a [Serotype A] outbreak with blueberries in the past, but I’m not sure the pattern is closely related.

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30 Field notes, 8/15/2012.
Epidemiologist 6: How closely is Bln related on the second PFGE?
Epidemiologist 2: I will check with PulseNet; we usually only use the first enzyme on rare patterns.
Epidemiologist 1: Looks like on this one we definitely need more cases to get a better picture.

This is a very typical brainstorming session for several reasons. Multiple people participate at a rapid-fire pace, and usually ask questions which point to other data that should be pulled and analyzed from existing sources, or collected if it is not yet available. In a dynamic way, talk tacks back and forth between scales (molecular, individual, demographic, regional) and times (past, present, and future). More lab and database work would be necessary for moving the outbreak investigation forward.

The cluster I have chosen in this example is at a very early stage, which one epidemiologist points out throughout the discussion, suggesting that more cases would provide the additional information needed. From the food history interviews of individual cases, ORPB looks for commonalities in the exposures that people report having consumed, typically performing odds ratio calculations as a first step. Here, half of the cases report bottled water; however, earlier in the meeting, a different disease cluster was discussed, and since cases in that cluster had demographic, temporal, and geographic commonalities with this cluster and a breaded chicken hypothesis, for this cluster, they were also adopting the same hypothesized exposure. They also suspected that the clusters were in fact a multiple pattern outbreak, but were investigating the clusters as two separate work objects for the interim.

As one epidemiologist pointed out, public health labs typically only perform PFGE on rare serotypes using one enzyme. This is chiefly to reduce costs. It is in theory possible to perform PFGE using up to three enzymes, which would cut the DNA fragments differently and provide more information that would help determine whether one pathogen is the same as
another. The epidemiologists discussed going back to the PulseNet lab and database personnel, to consider running PFGE with additional enzymes on isolates in the cluster.

Population thinking, group comparisons, and temporal thinking are explicit here because the group is using a collaborative sensemaking model, puzzling together aloud, and using abductive reasoning in their process of generating good hypotheses. Hypotheses are probable exposures, and the hypothesis generation phase involves the collection and analysis of evidence about heterogeneous clues and their potential relationships.

In addition to projecting “backwards” in the pathway into the database and the lab to have more data created and confirmed, the discussion also involved making inferences which projected both “backwards” and “forwards,” by puzzling over the issue of consumer perception of a suspected contaminated product. The epidemiologists were aware from past outbreaks that consumers had previously been sickened when they perceived that ready-to-cook products were ready-to-eat products. One epidemiologist wondered whether different measures would be necessary to prevent future consumers from suffering the same fate, through better labeling or better microwave technology.

Projecting out to thinking about future consumers is a way of re-thickening thin descriptions of cases. Outbreak investigators do this frequently, to remind themselves that their work saves lives and has public health impact. On a day-to-day basis, they work with data, sitting at computers and performing statistics. But the data they work with are about the health of real people, an aspect of the work that attracts many individuals to the field of public health. During several outbreak investigations, I observed that during investigators’ deliberations about when to announce outbreaks to the public, they used a “grandmother standard.” That is, they asked themselves whether they would feel comfortable if their own grandmothers ate the foods
they were investigating, since older adults are an immune-compromised, vulnerable population.
The grandmother standard was a method they used to move out of the investigative data layer, back down into the personal layer of everyday life in the community they were responsible for safeguarding.

Another dimension of the work I wish to bring out is its sociality. Charles Bosk (1980) has examined the “occupational rituals” of doctors, who used forums such as meetings, conferences, and grand rounds, to express shared understandings about their work (“special and unique tensions, burdens, responsibilities, and joys”). Occupational rituals, Bosk argues, enable physicians “to dramatize, to teach, and to remind themselves and their colleagues of their sense of what it means to be a physician.” Furthermore, Bosk points out, occupational rituals help physicians manage uncertainty, an “endemic” feature of making diagnoses and treatment decisions. I argue that the cluster management meetings can be seen as an occupational ritual for epidemiologists, and wish to note that the similarities between the two settings are not coincidental—many epidemiologists at the CDC were originally trained as physicians.

In particular, two devices Bosk observed for managing uncertainty are also apparent in the collaborative sensemaking of cluster management in the excerpt above—Socratic dialogue (talk consisting of questioning each other, e.g. “is this X? how closely related are X and Y?”) and linguistic shields (hedged assertions, e.g. “I think,” “that’s interesting but uncertain”).

As Timmermans and Buchbinder (2012) have noted, to institutionalize abductive reasoning, groups may create “transposable problem-solving repertoires” and “standard way(s) of managing the unexpected.” One way that ORPB has worked on institutionalizing abductive

31 I discuss historical reasons for why medicine has been so emphasized in epidemiology at the CDC in Chapter 2, but wish to note that currently, epidemiologists may come from many other backgrounds besides medicine, such as statistics, veterinary science, laboratory science, and anthropology.
reasoning is to model the steps in its outbreak investigation process, which is captured in the following diagram produced by the branch.\(^{32}\)

**Steps in a Foodborne Outbreak Investigation**

![Diagram of Steps in a Foodborne Outbreak Investigation](image)

Figure 10. Model of the Steps in a Foodborne Outbreak Investigation

Earlier in this chapter, I have described cluster detection, case finding, and hypothesis generation from ethnographic perspectives. Here I pick up a more in-depth discussion of step 4, hypothesis testing. Both epidemiologic and laboratory tools and techniques can be used in the hypothesis testing, or confirmatory phase. The gold standard procedure for hypothesis testing in disease outbreak investigations has traditionally been the case-control study, which involves an analysis of cases with a particular outcome (e.g. disease) as compared to controls (a group free of the particular outcome), in regards to the differences in frequency of exposure(s) in question. However, case-control studies can be time-consuming and resource intensive.

In recent years, outbreak investigations have shifted to collecting information to help guide their selection of food samples to test, relying upon the confirmatory power held by the

\(^{32}\) [http://www.cdc.gov/outbreaknet/investigations/investigating.html](http://www.cdc.gov/outbreaknet/investigations/investigating.html), accessed 11/12/2013. Model has since been slightly changed to a more linear representation.
laboratory identification of a pathogen matching an outbreak strain found in a food sample. The CDC emphasizes that the relationship between hypothesis generation and hypothesis testing is an iterative one. If the investigation is not uncovering good associations between specific foods and outbreak illnesses, and people continue to get sick, they return to the hypothesis generation phase, in a feedback loop, “iterating” until they find the source, or the outbreak ends before they can find the source.

Indeed, that the outbreak can end before the investigation uncovers the source creates enormous time-pressure for investigators. In these instances, the outbreak becomes an “unsolved mystery.” However, the preferred outcome is to find good associations between specific foods and the outbreak illnesses, to investigate them to “drill-down” into how the contamination happened, so that the source can be removed from the marketplace. Emphasizing the materiality of sensemaking, Williams frequently refers to the outbreak investigation process as “getting your arms around the whole thing [the outbreak].”

While outbreak investigations are conducted with a sense of urgency, the fact that evidence is highly distributed and heterogeneous ultimately makes the investigation slower than many would ideally like. Furthermore, since some of the data is confidential commercial information, it must be collected by local and state regulatory officials who must adhere to rigid, legally-bound procedures. Regulatory officials use two methodologies to examine food flowing through the supply and distribution chain. The first is traceback, starting at a “point of service” (food from commercial entity to consumer), and obtaining distribution records to identify shipments and suppliers back through the chain until a common source is found (convergence).33

The second is traceforward, to identify the destinations of contaminated food in interstate commerce.

Identifying contaminated food is only the first step of many to companies removing it from the marketplace based on the direction of regulatory agencies, and preventing people from consuming it. Because contaminated food can often be in people’s refrigerators and cupboards, or on store shelves or warehouse distribution centers, or in many other places in the supply chain, the CDC issues public warnings identifying food hazards through their website, and their warnings are carried and amplified to the public by the media. Warnings are typically issued when public health and regulatory actions begin, and updates are posted as investigations proceed to a conclusion. These warnings include a description of the current status of the outbreak investigation and advice to consumers, as well as a chart of the outbreak, the epidemic curve, which visually depicts how many people get sick each day.

Officials typically do not wait until an outbreak investigation is fully solved to make public health warnings, not only because they have a duty to warn vulnerable members of the public, but that they are also trying to communicate transparently, and play a more direct role in creating and disseminating outbreak information broadcast by the media. Theresa MacPhail (2012) notes that during the 2009 H1N1 pandemic, the CDC used a form of uncertainty as a strategy to maintain credibility and institutional authority, framing the virus as “predictably unpredictable” in both internal scientific work as well as public communications. The “predictably unpredictable” frame is not used in foodborne disease, but the “investigation” itself provides a frame for communicating with the public: the investigation is ongoing, data are waiting to be confirmed, and more information will be coming soon.
Often times, public health officials must “decide” when an outbreak is over. They use their judgment to do so, because they are managing the time gap between the event of disease occurrence and the event of public health detection. The time gap between the two creates a time window around the possible end of an outbreak, a 2-3 week period that public health officials mark on the epidemic curve in gray and append the description “illnesses that began during this time period may not yet be reported,” and informally, refer to as a “window of uncertainty” (see below).34

![Epidemic curve with window of uncertainty](http://www.cdc.gov/salmonella/enteritidis/epi_curve.html)

**Figure 11. Epidemic curve with window of uncertainty**

Monitoring when the epidemic curve has reached its peak, falls, and ends is particularly important in understanding whether the actions that have been taken (hazard announcements, recalls) have been effective in preventing more cases and ending the outbreak. During cluster management meetings, I found the closing of clusters particularly interesting, as it did not necessarily feel like “closure.” Investigators typically have multiple multi-state clusters under

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34 For example, see [http://www.cdc.gov/salmonella/enteritidis/epi_curve.html](http://www.cdc.gov/salmonella/enteritidis/epi_curve.html)
investigation at any one time (in high season up to 40 - 50); only a small subset makes it all the way through the confirmatory pathway. Sometimes a cluster would “flag,” where the number of cases would go above baseline, and then when the number of cases went below baseline, investigators would “throw it back.” Sometimes, a cluster with the same PFGE pattern would appear, and if it exhibited a similar epidemiological pattern, might eventually be deemed as the same cluster. Many clusters had seasonality—a pattern would rise above baseline in a particular place around the same time every year. Even closing a cluster felt provisional, within the confirmatory pathway. Given the high burden of foodborne disease, and the subsequent volume of detected clusters, there was always another possible major multi-state outbreak around the bend.

Conclusion

This chapter has taken an ethnographic look at the sociotechnical work and collaborative sensemaking involved in outbreak investigations. I developed the concept of a confirmatory pathway for provisional knowledge to characterize the early warning, real-time system’s network form, and help capture the dynamics of knowledge production, evidence gathering, and public health action within it. The confirmatory pathway for provisional knowledge creates a protected space for uncertainty management, and a systematized space for time management, enabling distributed, multi-jurisdictional, multi-organizational, and multi-disciplinary participants to coordinate their complex and interdependent work.

By using an ethnographic perspective, I was able to show how various technical procedures and tools work to thin out material and social thicknesses and simplify them,
winnowing microbial communities into single strains, and winnowing patients/eaters to cases. Then, in chronicling how patterns are created, clusters are assembled, and outbreaks are investigated, I showed how many thin descriptions were aggregated and woven together to create the heterogeneous web that is an outbreak. I demonstrated that this work is conducted collaboratively, and relies heavily on abductive reasoning, population thinking, group comparisons, and temporal thinking. Meetings were not only a forum for collaborative sensemaking, but an occupational ritual that helped reinforce what it means to be an epidemiologist and collectively manage uncertainty. In recent years, outbreak investigation work has undergone increasing institutionalization and systematization, yet it retains a dynamic, contingent, and lively quality, due in major part to material diversity in the universe of foods.

This ethnographic account of foodborne outbreak detection and response inside of confirmatory pathway for provisional knowledge is for the most part a snapshot of how it looked in 2012. However, the confirmatory pathway did not always exist. As the remainder of this dissertation shows, it is deeply historical, growing out of layers of public health knowledge infrastructure, and became a reality only through historically specific conditions of possibility.
Chapter 3. Setting the Stage for “Large-Scale Food Infection”: Salmonellosis and the Fowl Problem

The previous chapter took an ethnographic snapshot from the year 2012, when investigators in the early warning, real-time, national molecular subtyping network were exceedingly busy, detecting and responding to multi-state outbreaks on a routine basis. While Chapter 5 will examine the 1990s rise of the early warning molecular system, this chapter begins by charting a broader history important for understanding the growth of disease surveillance infrastructure.

More specifically, the aim of the chapter is to describe how a situation arose in which building better disease surveillance systems seemed both necessary and possible. Importantly, I discuss how a fractional picture of “large scale food infection” in the US first began to appear in the 1940s, partially illuminated by growing surveillance efforts focused on Salmonella, the “foremost” of 20th century foodborne organisms (Hardy 2004). I focus on the rise of what I call the “fowl problem” – a cycle of Salmonella disease transmission intensified by the industrialization of the US poultry industry. The fowl problem was a key example of a larger phenomenon—the increasing industrialization of the US food system, which was reflected in the changing epidemiology of salmonellosis. The material I use in this chapter is based primarily on secondary literature, scientific publications, and transcripts from Congressional hearings.

Key Elements of Disease Surveillance Infrastructure

Before delving into the “fowl problem,” I wish to provide a historical overview of three key elements of disease surveillance infrastructure, that help establish important institutional and
technical context: disease reporting; the rise of the CDC; and identifying, measuring, and classifying *Salmonella*. These elements are not presented in chronological order, but come to overlap and interweave, as part of the installed base for subsequent infrastructural layers.

*Disease Reporting*. To understand the broad phenomenon of public health surveillance in the US, it is important to take into account the history of the idea of disease reporting, and how it arose first at the scale of community and the local municipality, before moving to the state, and then the national level, thus constituting the multi-level model of federalist governance in place today. As Amy Fairchild and colleagues (2007) discuss, the idea of disease reporting in the US is very old, with local statutes on the books as early as the mid 18th-century. These laws made the reporting of disease a public responsibility, requiring a diverse range of people such as doctors, shippers, lodging house proprietors, and family members to inform local officials of cases of communicable diseases of concern, which at the time were acute infectious diseases such as cholera, yellow fever, and smallpox. The idea behind these laws was to warn officials about potential epidemics of communicable disease. Fairchild et al emphasize that in colonial and preindustrial times, disease monitoring was a community endeavor, “embedded in a culture where people broadly monitored and controlled entry into and behavior within in the community.” Hand-in-hand with the idea of communicable disease being a community problem was that it was also treated as an episodic problem with ad hoc responses, rather than with special and sustained institutions.

It was not until the last few decades of the 19th century that a more stable and national system for “notifiable” disease reporting began to take shape in the US, growing out of late 18th-19th century developments, such as sanitary reform movements, and the growth of municipal and
state boards of health as governing bodies (Duffy 1992). Increased institutionalization at the federal level began to occur in the early 20th century, beginning when Congress authorized the US Marine Hospital Service1 to collect weekly morbidity reports from overseas US consuls on cholera, smallpox, plague, and yellow fever for informing quarantine policies, and expanded weekly reporting requirements to state and municipal levels. Disease reporting grew somewhat in the 1910s-20s, though largely on a voluntary basis; much of the growth that did occur was propelled by the Association of State and Territorial Health Officers (ASTHO), a professional association for local and state public health agencies and officials (Koo & Wetterhall 1996).

Foodborne outbreaks of disease had long received attention from local and later, state public health authorities. But the first significant federal attention foodborne outbreaks received was in the 1920s, when the US Public Health Service (PHS) opened a field station for the “investigation and study of all outbreaks of food poisoning occurring in the United States.”2 The PHS collected and reviewed all the records they could find from local and state departments of health, examining over 800 “suspected” food poisoning outbreaks. The PHS included the “suspected” caveat in their description, because they determined that in over 80 percent of the investigations, there were signs that the outbreaks were not caused by foodborne illnesses, or that the particular food that was blamed was wrongly impugned. One PHS epidemiologist, J.C. Geiger, bemoaned the inaccuracies of the clinical diagnosis of food poisoning, and complained about how difficult it was to get “reliable facts” about US food poisoning prevalence, because the disease was not nationally reportable.

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1 Precursor to the Public Health Service (PHS), named as such in 1912
The Rise of the CDC. The CDC originated as an entity with a specific role in WWII, as the MCWA (Malaria Control in War Areas), a unit of the PHS. As Elizabeth Etheridge (1992) documents, the MCWA expanded its original malaria control purview, first to typhus-control, and then, as the war came to a close, a mission to improve civilian health more generally. The new name chosen for the MCWA was the Communicable Disease Center (CDC), and establishing this new health agency meant strategically negotiating jurisdiction with the National Institutes of Health (NIH), so that CDC would not be seen as a rival. The CDC would focus on disease control and providing support and service to states, which left NIH freer to pursue basic research and to focus on chronic diseases. The new agency was officially established in 1946.

One key way that the new agency established itself was to ground its expertise in epidemiology. Early on, many of its scientists were specialists in entomology and parasitology, given the agency’s early focus on malaria and typhus control. In 1949, the CDC recruited Alexander Langmuir from Johns Hopkins University to develop epidemiology at CDC, a field that would, as Etheridge puts it, become the “hallmark of the institution.”

Langmuir, CDC’s first chief epidemiologist, was a key system-builder for the institution. As Thomas Hughes (2012) states, a system-builder has the “ability to construct or to force unity from diversity, centralization in the face of pluralism, and coherence from chaos.” In his post, Langmuir focused on developing ways to cultivate relationships between the states and the CDC necessary to improve national disease reporting and control. To help enact CDC’s institutional mandate to provide support and service to states, Langmuir promoted and professionalized field epidemiology, to create and spread a shared expert identity amongst federal and state public health officials. In fact, he drew strongly from the “disease detective” frame to do so, a frame
previously popularized by the PHS, associated with a national campaign to improve rural sanitation and control typhoid fever.³

Langmuir promoted the idea of “shoe-leather epidemiology,” which he likely drew from his time working with Edward S. Godfrey Jr., the commissioner of health in New York State, and one of the creators of the American Epidemiological Society. Though some publications attribute the term “shoe-leather epidemiology” to Langmuir, Godfrey had discussed it in 1941:

‘Shoe leather epidemiology’ in regard to tuberculosis case finding differs from ‘swivel chair epidemiology’ — which consists of attempting to solve a problem by prepared statistics — in that it means going out on the highways, byways, and alleys, climbing the stairs, walking the streets, or bumping over country roads in order to ferret out the sources of tuberculosis infection.⁴

While the shoe-leather epidemiologist became a key expert identity Langmuir promoted for the CDC scientist, he also began to espouse a “philosophy of surveillance” that would become the agency’s “cornerstone” activity.

Prior to Langmuir’s redefinition, the concept of surveillance within the field of public health referred to physicians actively monitoring individual patients with certain serious infectious diseases so they could be quarantined if necessary. Langmuir redefined surveillance to apply to diseases, not just individuals, and as a job for the epidemiologist, not just the physician. Surveillance, he stated:

when applied to a disease, means the continued watchfulness over the distribution and trends of incidence through the systematic collection, consolidation and evaluation of morbidity and mortality reports and other relevant data. Intrinsic in the concept is the regular dissemination of the basic data and interpretations to all who have contributed and to all others who need to know. The concept, however, does not encompass direct

⁴ Health News, Volume 18, New York State Department of Health, 1941.
responsibility for control activities. These traditionally have been and still remain with the state and local health authorities.\textsuperscript{5}

As an institutional program for operationalizing his concept of surveillance, Langmuir helped to found the Epidemic Intelligence Service (EIS), a CDC training program whose goal was to quickly turn young medical professionals into epidemiologists and send them out into the field to assist state and local health authorities with epidemic investigations, when state and locals requested help.\textsuperscript{6}

An early training exercise developed for EIS officers also plays a pivotal role in the history of systematization in foodborne outbreak investigations. In 1940, some of the residents of Lycoming, New York, a small village in Oswego County, held a supper at a local church. Unfortunately for supper attendees, one of the foods served at the event was contaminated, causing an outbreak of gastroenteritis. The village health officer’s investigation determined that the food culprit was homemade ice cream.

This small outbreak would become famous. It was first transformed into a textbook model problem at Albany Medical College in 1940, and would also be used in the US Navy Epidemiology Training Program in 1942, at the Johns Hopkins School of Hygiene and Public Health in 1946, at the CDC in 1949 (Morabia & Hardy 2005). In 1951, Langmuir used it to train the 1951 class of incoming EIS officers, to show them how to investigate outbreaks, building on previous iterations of the model problem.

The outbreak was not transformed into the Oswego problem because the investigation was particularly innovative, but rather, that the church supper was a clear-cut and well-defined outbreak that was amenable to being turned into an exemplar for training purposes, helping field


\textsuperscript{6} For more on EIS, see Pendergast (2010) and McKenna (2008).
epidemiologists learn the methods involved in outbreak investigations. It was especially important to develop a training program for EIS officers because many of them would be involved in outbreak investigations, jumping in to assist states whenever their services were requested.

Joseph McCormick (1999), co-author of a CDC thriller about virus hunting, wrote a detailed account of his first outbreak investigation as an EIS officer, which happened to be a foodborne outbreak. He traveled to a small town in Arizona, where attendees of a Fourth of July picnic at an Indian reservation had fallen ill with severe streptococcal sore throats.

McCormick used a “by the book” procedure from his training to investigate the outbreak, conducting a case control study on ills and wells who had attended the picnic. After dividing the population into two groups, he created a questionnaire to ascertain their food histories through interviewing. He emphasized the importance of being “careful” in the way questions were phrased, so that people would be more likely to give accurate answers. He noted that it was common that people might forget answers, give false answers trying to please the questioner, or hide sensitive information. He also collected throat swabs to send the bacteria to the CDC for laboratory testing.

One calculation conducted with food history data was attack rate, to quantify the percentages of ills and wells who ate or did not eat particular foods, signaling which foods should be suspect from the meal. The following example problem on how to calculate attack rate is excerpted from a field training manual prepared by the CDC for investigating foodborne outbreaks:

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7 Referring to cases and controls as ills and wells was much more common earlier in 20th century epidemiology.
8 National Archives, RG 442. Folder, “Information 3.” Food-Borne Disease Investigation: Analysis of Field Data. PHS Pub No 1186. Technically, this manual was produced by the CDC for local public health workers in the field responsible for investigating local outbreaks. It was published in 1964. I spoke to CDC museum staff, who could
The list of vulnerable foods listed in the attack rate table reflect typically consumed foods in the mid-20th century US: baked ham, custard, jello, cole slaw, baked beans, and potato salad. These foods were likely to grace a “church supper,” and indeed, referring to outbreaks as “church-supper” outbreaks has since become a colloquial metaphor for local outbreaks, events of disease transmission occurring in conjunction with social gatherings involving the sharing of (contaminated) food, whether it is at a family reunion, a professional conference, a dinner out with friends, or a catered wedding at a hotel.

not find original training materials from the early EIS trainings. Interestingly, this manual focused on outbreaks of Salmonellosis. It might be possible to find some material from one of the other places where the Oswego model problem was developed, but is out of scope for this project.
From McCormick’s case-control study, he identified potato salad as the likely culprit, since all those who had become sick at the picnic ate that dish. The last kind of evidence he collected before concluding the on-site field part of the investigation was leftover potato salad from picnic attendees’ houses, to send to the CDC for laboratory testing to find the outbreak strain.

Early on in the EIS program, the majority of trainees chosen were physicians, and as William Schaffner and F. Marc LaForce recounted in memories of their training (1996), the goal of the first month of EIS was to turn physicians into epidemiologists, to “transform our focus from individual patients to the consideration of groups of people.” They wrote, “the group to be analyzed, whether a community, school, or assemblage at a church supper, had become our ‘patient.’” Reflecting on the Oswego problem, Schaffner and LaForce argued that diagnostic power of the shoe-leather epidemiologic method could be demonstrated in such a bucolic setting without the need for high-technology assistance continues to be alluring to those who recently have left work in hospital intensive care units.

This passage demonstrates that technological style of public health was not only relevant to the laboratory side, but also applied to epidemiologic methods, in contrasting the “high-tech” intensive care unit with the “bucolic” church-supper.

Schaffner and LaForce’s account makes clear the ways in which Langmuir was using the clinical realm as a rhetorical frame for the EIS program. Not only would the groups of people in an outbreak be considered a “patient,” but the mechanism by which EIS trainees would be sent out into the field to assist states in outbreak investigations, the “epidemic aid,” used a model he called the “public health emergency room.” It was similar to the emergency room, where teaching hospitals would give interns “frontline” clinical responsibilities, but be subject to the guidance and supervision of more senior physicians.
To help cultivate the agency’s relationship with the broader public, in the 1950s, Langmuir invited Berton Roueché, a writer for the *New Yorker’s* “Annals of Medicine” who had become famous for a 1947 story called “Eleven Blue Men.” Roueché dramatized a New York City Health Department investigation of a curious outbreak, where over the course of one morning, eleven different “elderly and dilapidated” men filtered into the emergency room, exhibiting the same odd symptoms, especially their “sky blue” skin color.

The investigation, it turned out, ended up being a foodborne one, though the event was not bacterial, but caused by an accidental poisoning. Langmuir invited Roueché to write about, and help popularize, EIS investigations. Roueché’s stories were extremely popular, and helped turned Roueché into a “folk hero” within the field of epidemiology, with his stories sparking greater interest in the profession and its “captivating” disease-detective work. While others had commented that Roueché’s pieces were like “classic” detective stories, Roueché himself said that “Eleven Blue Men” led him to write a “new kind of detective story,” one harkening back to its “origins” in Sir Arthur Conan Doyle’s “Sherlock Holmesian” method.

Several of Roueché’s stories featured foodborne investigations, and brought out their localizing quality. In “A Game of Wild Indians,” which featured an investigation of a typhoid outbreak amongst an Armenian neighborhood in New York City, one of the investigators quipped, “My job was the recent social life of the Armenian colony.” “S. Miami” involved an outbreak of salmonellosis, where the unusual vehicle of watermelon turned out to be the culprit. Helping shed light on where the watermelon got contaminated was the serotyping conducted by

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the New York Salmonella Center, which identified the serotype S. Miami, originating from Florida.

Identifying, Measuring, and Classifying Salmonella. Like many other bacteria, Salmonella’s early biography comes out of the late 1800s, a time when the field of bacteriology was making dramatic discoveries about the nature of communicable disease, identifying their specific bacterial causes. As Nancy Tomes (1999) charts, germ theory brought about a number of major social transformations: a shift from notions of atmospheric infection (miasma theory) to a focus on transmission via persons, food, water, and insects; expansion of collective public health measures like municipal sewage, food inspection, and garbage collection; and the expansive reformation of hygiene amongst individuals and households, the latter of which she calls the “gospel of germs.” Before germ theory, food poisoning was attributed to ptomaines, “cadaveric” poisons produced by the decaying of animal proteins, which could spread through the air, water, and food supply.

The entity that came to be designated as Salmonella came out of research at the USDA’s Bureau of Animal Industry (BAI). The BAI got its start in 1884, under the direction of veterinarian Daniel Salmon, and became a centralized and powerful federal agency chiefly focused on monitoring and controlling major animal diseases, such as hog cholera and Texas cattle fever (Olmstead 2009). Theobald Smith, who would eventually become one of the US’s most pre-eminent bacteriologists, came to work for Salmon at the BAI, and soon after arriving there identified a new species of bacteria which he thought to be the cause of hog cholera. At
first this entity was deemed the “hog cholera bacillus,” but in 1900, it would be renamed in Salmon’s honor.¹⁰

As Anne Hardy (2003) discusses, between the 1880s and the 1920s, the classification of Salmonella was in “complete confusion.” Bacteriologists around the world were developing and using many new laboratory techniques, and researchers from different places were naming the same bacteria with a variety of names, or using the same name for different organisms. Classificatory order would be imposed through the stabilization of one particular technique (serological testing), and the development of an international classification system for Salmonella.

Serology, the study of specificity, sought to explain immune reactions in terms of chemical properties (Mazumdar 2002). Early on, it developed as an international science, because it was important for international disease control efforts. Key to serological typing of bacteria was the creation of serum, a substance made from blood (usually rabbit blood) injected with antigens from the bacteria to create antibodies. When a bacterium of unknown type needed identification, laboratory scientists could use different known serums containing specific antibodies to discern which one would produce agglutination (the clumping of bacterial cells evincing a match between serum antibodies and bacterial antigens in the unknown strain). Agglutination tests were conducted in either tubes or slides. The following picture portrays slide agglutination.

¹⁰ Scholars have examined a kind of credit dispute between Smith and Salmon, and most argue that Smith’s work was co-opted by Salmon, who was Smith’s supervisor and not actively involved in the research (Dolman and Wolfe 2003).
For *Salmonella*, there were two principal kinds of antigens: O-antigens, on the body of the bacteria, and H-antigens, on the flagella.

Serological typing was at the heart of an international classification system for *Salmonella*, the Kauffmann-White Scheme. In the early 1920s, a Scottish bacteriologist, Philip Bruce White, began targeted research on *Salmonella*, and would focus on the bacteria’s O antigen. In 1924, he went to the Lister Institute, where he set about to make antigenic formulas the basis of a classification scheme for *Salmonella*, work he continued when he moved to the National Institute of Medical Research in 1927. White conducted many studies to characterize variations in *Salmonella* and develop better methods for antigenic analysis. In 1929, he contributed a chapter on *Salmonella* to the MRC’s *System of Bacteriology*, which later became one of the bases for the Kauffmann-White scheme.

Around the time that White began his *Salmonella* work, Fritz Kauffmann, a German bacteriologist, began working on *Salmonella* at the Robert Koch Institute in Berlin, where he

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11 CDC bulletin, vol. IX, no. 5, May 1950
stayed through the 1920s.\textsuperscript{13} In 1930, he published his first antigenic schema for \textit{Salmonella}, and then in the early 1930s, left Germany to escape the Nazis, landing in Denmark’s famous State Serum Institute (SSI) to direct an “International \textit{Salmonella} Centre.” There, he would help to facilitate serological \textit{Salmonella} diagnoses internationally. By the 1930s, a global network of salmonella reference laboratories had been established, and Kauffmann, building significantly on White’s work, developed a standardized classification system for \textit{Salmonella}—the Kauffmann-White scheme, where newly identified \textit{Salmonellas} would be named after the place where they were found (Hardy 2003). While some scientists had already been using this practice (e.g. \textit{Salmonella} berlin, oranienburg, schwarzengrund), with the new scheme, it became the agreed-upon standard convention.

Kauffmann emphasized that the scheme was a pragmatic and simplified one, an “arbitrary delimitation” of the microbe enabled by serology. He gave the qualification that “no exact definition” of the \textit{Salmonella} group was possible, because there existed “no sharply delimited groups in nature.”\textsuperscript{14} The internationally recognized definition of \textit{Salmonella} in 1947 pulled together a variety of usual phenotypic traits to create the group:

A large genus of serologically related Gram-negative and non-sporing bacilli, 0.4-0.6\(\mu\) X 1-3\(\mu\) in usual dimensions, but occasionally forming short filaments; showing, with certain exceptions, a motile peritrichous phase in which they normally occur; in fact adhering to the pattern of S. Typhi in staining properties and morphology. Rarely fermenting lactose or sucrose, liquefying gelatin or producing indole, they regularly attack glucose with, but occasionally without gas production. All the known species are pathogenic for man, animals, or both.


\textsuperscript{14} Hardy’s forthcoming book (\textit{Salmonella Infections, Networks of Knowledge, and Public Health in Britain 1880-1975}, Oxford: Oxford University Press, 2015, in press) will examine the history of Kauffman’s work and the international network of scientists involved in standardizing \textit{Salmonella} in further detail, but for the purposes of this chapter what is important to understand about this standardized scheme is that it simplified the complexity of \textit{Salmonella} by establishing standardized methods and naming conventions.
Developing a uniform classification scheme for *Salmonella* facilitated increased communication and collaboration between laboratory scientists responsible for typing the microorganism and fostered an international scientific community. Also, I argue, it facilitated improved communication and collaboration between laboratory scientists and epidemiologists. Serotypes were much easier to handle as data than antigenic formulas. As mostly common or at least readable words, serotypes could be more easily written and spoken.

As an illustration of the communicative facility the uniform classification scheme created, one scientist later referred to it as the “Salmonella telephone book.” A portion of the Kauffmann-White Schema from 1949 is excerpted here as an illustration:

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*The Diagnosis of Salmonella Types*

**Table III**

*The Kauffmann-White Schema (1949)*

Diagnostic Antigenic Schema

<table>
<thead>
<tr>
<th>Type</th>
<th>O Antigen</th>
<th>H Antigen</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Phase 1</td>
</tr>
<tr>
<td>Group A</td>
<td></td>
<td></td>
</tr>
<tr>
<td>S. paratyphi A</td>
<td>I, II, XII</td>
<td>a</td>
</tr>
<tr>
<td>Group B</td>
<td></td>
<td></td>
</tr>
<tr>
<td>S. kirschneri</td>
<td>I, IV, V, XII</td>
<td>a</td>
</tr>
<tr>
<td>S. arnoldi</td>
<td>I, IV, XII</td>
<td>a</td>
</tr>
<tr>
<td>S. bispebjerg</td>
<td>I, IV, XII</td>
<td>a</td>
</tr>
<tr>
<td>S. abortus equi</td>
<td>IV, XII</td>
<td>—</td>
</tr>
</tbody>
</table>

Figure 13. The Kauffmann-White Schema

After a laboratory scientist conducted serotyping on an organism to determine properties of the O and H antigens, she could use the antigenic formula (e.g. IV, V, XII, a, 1, 7) to find its type (e.g. 15

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S. kisangani). The telephone book metaphor is apt, but is perhaps more similar to “reverse lookup,” in using numbers to find the name.

On the one hand, observed ST Cowan, a British bacteriologist, creating a Salmonella telephone book helped meet the needs of “workers in applied bacteriology,” the clinicians, veterinarians, medical officers of health, and sanitary inspectors. On the other hand, Cowan stated, “taxonomic purists” had difficulties with this common name simplification, particularly with the convention that the type was italicized and the place name began with a lower case initial, misleadingly indicating a specific Linnean binomial of genus and species, when Salmonella taxonomy was much more complex.

Indeed, illustrating these tensions, in 1953, the Enterobacteriaceae Subcommittee decided that henceforth, newly discovered serotypes would be described by formula only and not by common name. However, after the Subcommittee’s decision, some of its members later requested reconsideration of the decision, and in 1954, the members took a vote on the matter. In 1955, the results of the vote were formalized—a majority voted to cancel the 1953 decision, restoring the common name convention. Reversing this decision preserved the pragmatic use of the nomenclature system, maintaining communicative facility amongst a wide variety of users worldwide, if not conforming to scientific ideals of pure taxonomy.

In the early 1950s, US bacteriologists Philip Edwards and Joshua Lederberg had conducted a study of “the exchange of flagellar antigenic factors among various Salmonella

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17 Cowan explains that an early decision to designate S. cholerae-suis as a type species created problems down the line, when bacteriologists began to feel less comfortable calling as many combinations species as opposed to serotypes of a smaller number of species.
serotypes,” using genetic transduction. The proliferation of new *Salmonella* serotypes had made it widely believed, if not fully confirmed, that new types were created by evolutionary processes. The Edwards-Lederberg study identified a possible evolutionary mechanism. However, the Kauffmann-White scheme did not reflect evolutionary relationships between serotypes. Yet Lederberg & Edwards did not present their study to challenge the Kauffmann-White Scheme, and argued that their findings did not “detract in the least from the epidemiological and diagnostic applications” of the scheme. They wrote:

> The convention of assigning specific epithets to serological types must be adjudged according to its own practical advantages and disadvantages; the nomenclature in general use has not been proposed seriously as a taxonomic scheme, the substantiation of which would require far more detailed knowledge of the evolution of bacterial species than can now be claimed.

The careful conceptual work Lederberg & Edwards perform here is particularly interesting, distinguishing nomenclature from taxonomy. Because the Kauffmann-White Scheme was not designed to be a taxonomic scheme but a nomenclatural one, they argued, it retained its practical use. However, I argue, in helping to illuminate bacterial evolution, this work helped to feed a growing scientific desire to develop taxonomic classification schemes that would reflect evolutionary relationships between bacterial types.

**Salmonella in the US: Philip Edwards, Simplification, and Laboratory Service, 1925-1953**

The mere existence of a worldwide classification system for *Salmonella* was not sufficient for it to have significant influence in the US. In fact, the US had been a significant player in the international network of *Salmonella* researchers, chiefly through the work of Edwards, pictured below.

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Edwards had conducted his dissertation research on *Salmonella*, and after graduate school, worked at the University of Kentucky Agricultural Experiment Station (AES), from 1925-1948. While he worked on a variety of bacteriological studies, he continued his *Salmonella* serological and biochemical characterization research with a strong focus on poultry diseases. At the AES, he directed a National Salmonella Center from 1939-1948, and during WWII, was chiefly responsible for overseeing the production of needed *Salmonella* typing sera for use during the war, as salmonellosis (like all infectious diseases), was a “wartime problem” due to factors such as overcrowding and travel.  

In 1948, Edwards moved to the CDC, to serve as the Chief of the Enteric Bacteriology Laboratory Unit. In this position, his general responsibilities were two-fold—continue his long-
term research agenda on enteric bacteria (with a special focus on *Salmonella*), and, as a CDC leader, to “give service on a national and international scale to other diagnostic laboratories.” A core part of CDC’s founding mission was a service-based one, and has continued to profoundly shape both laboratory and epidemiologic science at it is practiced at the institution. For the CDC, a service-based role meant acting as a reference laboratory as well developing training materials and programs for other clinical and public health laboratories.

Service was not an entirely novel role for Edwards, who had already been working with laboratory scientists in a variety of state public health laboratories with his national serological work on *Salmonella* while at the AES. In 1948, the CDC collaborated with the Florida State Board of Health and Armed Forces Epidemiological Board to create a veterinary public health laboratory in Jacksonville, Florida to study salmonellosis in animals in Florida as well as contamination in human and animal foods. This laboratory was headed by bacteriologist Mildred Galton, who would join Edwards at the CDC in Atlanta in 1953 to head the agency’s new Veterinary Public Health Laboratory.

Edwards specialized in *Salmonella*, but as EBLU Chief, also conducted work on a variety of enteric bacteria. He collaborated frequently with William Ewing, a *Shigella* specialist at the CDC. Their early service work at CDC focused on developing simplified procedures for the selection of bacterial cultures and serologic identification, so that these activities could be “carried on even in the small diagnostic laboratory.”\(^2\) They disseminated their simplification work in the May 1950 CDC Bulletin, a publication aimed at technicians in state laboratories.

In the Bulletin, Edwards provided encouragement to this group to participate in *Salmonella* identification. Even though the Kauffmann-White scheme looked “very complicated,” with the “fast growing numbers of types add[ing] to the confusion of the average technician,” Edwards affirmed to the group the scheme was “the only sure basis” for recognizing the types of *Salmonella* “of greatest importance in the epidemiology of salmonelloses of man.”

The simplified laboratory routine Edwards published in May 1950 extended simplification work he and others had conducted previously. Through this work, they had established that between 98 – 99 percent of all *Salmonella* cultured from man fell into the first five somatic groups of the Kauffmann-White scheme. This meant that the average laboratory would only need to stock five of the O serums, for groups A through E, and be able to identify

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22 Later Edwards would diverge from Kauffmann in regards to the scheme, and the CDC would use a modified version of it. Hardy’s forthcoming book will discuss this in detail.
almost all *Salmonellas* then known to create illness in man (e.g. *S. typhi*, *S. paratyphi A*, *S. paratyphi B*, *S. paratyphi C*, *S. sendai*, *S. cholerae-suis*, *S. typhi-murium*). If the average laboratory came across a member of the 1-2 percent of types they could not identify, they could send it on to the CDC or another more advanced laboratory to conduct more exact characterization. Edwards’ goal was to identify the “minimal number of serums” a laboratory would need to have on hand, to encourage as many laboratories to adopt the routine as possible. He noted that most laboratories were not conducting O and H agglutination tests at all, which meant many delays and inaccuracies in *Salmonella* typing.

By making it simpler to participate in *Salmonella* serotyping, Edwards was not only enacting the CDC’s service mission, but providing a mechanism for improving public health surveillance of salmonellosis across the country. Using a simplified routine, more laboratories could type their salmonellas accurately and become information producers, and could help contribute to a better picture of the disease at the population level. Remarks from Geoffrey Edsall, the chairman of the Laboratory Section of the American Public Health Association (APHA), highlight why a simplified serotyping routine would be useful in the states. In a 1950 presentation at the APHA annual meeting, he talked about the enormous challenges facing the state public health laboratory. He pointed out that the laboratory worker in the public health laboratory was conducting an increasing amount of “routine repetitive maneuvers,” for example, handling up to 3000 specimens per day in a serological laboratory.  

Edsall commended laboratory workers for handling this “burden of routine performance” and facing this “threat of routine,” in service of the greater mission of advancing public health. A simplified routine could help ease some of the increasing burden on state public health laboratory workers.

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What Edwards had already helped establish before he came to the CDC in 1948 was to provide enough systematization of *Salmonella* to suggest that it was a major public health problem. In the 1940s, three US National Salmonella Centers—Edwards’ University of Kentucky Center (focused primarily on veterinary specimens), and two centers in New York, one in Albany and one in New York City (both focused primarily on clinical specimens)—serotyped thousands of specimens and cultures from all over the US, as many as they could get their hands on. The Kentucky Center examined cultures from both humans and animals, while the New York centers focused more on humans.

Researchers from the Kentucky Center noted that the number of avian cultures they serotyped far outnumbered cultures from other animal sources. They argued that this was a reflection of a few factors: how large the US chicken population was, how common it was to use bacteriological methods of diagnosis in poultry management, and how close the hatching and brooding quarters were, increasing the risk of communicable disease transmission. The New York City Salmonella Center sounded a more forceful alarm. They combined their human data with the Kentucky Center’s animal data, as well as another group’s study of *Salmonella* in meat products in retail markets\(^24\), to draw a broad and exceedingly disturbing conclusion; “the stage seems to be set,” they wrote, “for food infection on a large scale.”

But how large was large scale? In 1940, Erwin Neter, a bacteriologist from the University of Buffalo, called salmonellosis a “malady” but complained that its “source and mode of transmission… remains undetermined.” Neter noted that while many local outbreaks of salmonellosis had been traced to human carriers and to contaminated water and food, “the origin

of the infection in an extraordinary large number of sporadic cases has remained obscure.”

Massachusetts, long a leading state in the public health system, had made Salmonellosis reportable in 1937, and complained at the 1943 APHA meeting about the “complete lack of uniformity” within the states in regards to Salmonella reporting. At one extreme, where the disease was not reportable, officials investigated only outbreak-related cases. At another extreme, one state kept healthy carriers of paratyphoid organisms under close observation for a year. In the 1940s-1950s, state and federal public health officials continued to repeat the PHS’s refrain from the 1920s, about the lack of reliable facts about the prevalence of food poisoning in the United States.

A Partial Picture of Salmonellosis

Throughout the 1950s, Edwards and others were also working on creating as good an epidemiologic picture of Salmonella as they could on available data, publishing extensive review pieces. They identified myriad challenges of establishing true incidence of salmonellosis, in particular the “woefully inadequate reporting from a minority of the states.” While national cases of salmonellosis increased seven-fold between 1946-1955, and four-fold in Massachusetts between 1940-1955, Edwards argued that it was not clear as to how much this was an actual increase in disease versus better diagnosis and reporting. However, increased reporting demonstrated that public health labs and departments in the states were becoming more aware of, concerned about, and active around the problem of salmonellosis.

In a 1956 review, Edwards constructed a “long and varied history” of the topic, as well as assessing the current state of knowledge about each of the main sources of Salmonella infection—ruminants, swine, fowls, other animals, and man. In addition to mentioning the establishment of the Kauffmann-White scheme as important for the recognition of Salmonella’s diversity of serotypes and antigenic characteristics, Edwards boiled the history down to two “doctrines.”

First was the “Kiel doctrine,” which had stemmed primarily from the work of a German research group, establishing that S. Paratyphi B produced enteric fever and was primarily passed man-to-man, while S. typhimurium produced different diseases within animals, and in man was the cause of “mass outbreaks of food infection characterized by acute gastroenteritis.” The Kiel doctrine emphasized Salmonella as the cause of mass outbreaks.

Second was the “Montevideo doctrine,” which had more recently come from a group of Uruguyan researchers studying the causes of “infantile summer diarrhea.” They built on other research about the sporadic nature of Salmonella, and emphasized sporadic cases over outbreak cases, to better reflect the organism’s wide distribution throughout the animal kingdom and call attention to how young children were more susceptible to infection from the pathogen that other populations.

One important point Edwards emphasized in his 1956 review was that many animals “used widely as food” were reservoirs of infection; in particular he posited that fowls were likely the “largest single reservoir of Salmonella among animals,” given that they were so large in population. Eggs, egg products, handling of diseased fowl, and contaminated carcasses were all
mentioned as documented sources of infection. By 1956, Edwards and others had examined over 30,000 *Salmonella* cultures.

Edwards’ 1958 review paper provided an extensive discussion of how knowledge about *Salmonella* incidence pointed towards the need for particular control measures, for each source of infection (animals used as human food, human carriers, environment, and animal-to-man transmission). He repeated the point that fowls were the likely the largest *Salmonella* reservoir.²⁷ He argued a point that would become much more widely discussed in the next decade, that “food technology” and the “removal of food preparation from the family kitchen to the large establishment” as an important consideration, observing that the kinds of controls and their enforcement currently in place “have not kept pace with advances in food processing.”²⁸ Finally, Edwards emphasized a theme that was becoming more and more common—given the widely distributed and multi-faceted nature of salmonellosis, encompassing the animal kingdom (man included), control of the disease would only be possible through cooperation between the multiple governmental agencies working in health, agriculture, and food.

Also in 1958, several scientists at the CDC’s Enteric Disease Investigation Unit published a paper entitled “Widespread Salmonella reading infection of Undetermined Origin.”²⁹ In the paper, they described a new kind of “outbreak,” which had occurred between September 1956 - September 1957. During that year, 325 “acute sporadic cases” and “3 outbreaks” of salmonellosis due to *Salmonella reading* (a previously rare serotype) had occurred. To describe this new entity, they wrote:

²⁷ He cited his 1943 study with Bruner, a study of *Salmonella* cultures from avian outbreaks, and several microbiologic studies of various egg products.
²⁸ Edwards cited Savage in expressing some of these ideas.
This “outbreak” is an example of a phenomenon periodically noted by Salmonella typing laboratories when, within a few weeks, a relatively large number of cultures are received containing a serotype which has previously been encountered only rarely. This phenomenon is seldom explained except when circumscribed outbreaks of salmonellosis are clearly involved.

The paper went on to describe this “outbreak” in detail: how it was first noticed, what data were collected in an investigatory capacity, and how the investigation proceeded. The authors conveyed a slightly disappointed tone when they said that their investigation “failed to reveal the presumed common source responsible for this ‘outbreak.’” However, while they did not definitively identify the presumed common source, during the investigation, they did explore a “possible but unknown relationship between infected animal feeds and poultry.”

The scare quotes around the term “outbreak” demonstrate the newness of the phenomenon. Typically, outbreaks would come to light through the traditional means of observation by clinicians, or in an obvious manner associated with a church supper or other social gathering. This “outbreak” appeared in laboratory data. As more and more statistics were being collected on Salmonella serotype data, a new kind of outbreak came to light. However, the contours of its cause was not firmly established, but only suspected.

**The Fowl Problem of Salmonella**

As historian William Boyd (2001) documents, by the early 1960s, the chicken had become “one of the more thoroughly industrialized commodities in American agriculture.” To achieve chicken production at a large scale, producers used a strategy of intensive confinement, placing birds in close quarters to produce a continuous supply of chicks, and developing artificial incubation methods and environmental control measures to support the production of a continuous supply.
The industrialization of the egg industry began at the end of the 19th century. Important innovations for egg production at a large-scale were the invention of the incubator, to mimic hens’ hatching, and the trap nest to identify the more productive egg layers to become breeders. In the 1920s, egg production was vastly increased by the use of artificial lighting in chicken coops; lengthening daylight hours stimulated hens to lay more eggs. Both chick production and egg production required the same environmental condition for chickens—intensive confinement.

Because intensive confinement raised communicable disease risk to chicken populations, bacteriology became one of the most important expert fields in the chicken industry. Outbreaks of pullorum disease (PD) and fowl typhoid (FT) (caused by Salmonella Pullorum and Salmonella Gallinarium respectively) were major problems for commercial hatcheries, with young chicks particularly vulnerable to infection and death. Serological typing was important for helping to identify and remove infected birds and preventing contagion in flocks. However, it took a national program of coordination to control PD and FT in the poultry industry, the USDA BAI’s National Poultry Improvement Plan (NPIP). The NPIP was a partnership between the government (at federal and state levels) and the industry, to develop uniform standards and practices for testing, breeding, and inspecting poultry, which, between the 1950s–1970s, worked to virtually eradicate PD and FT from the US commercial poultry industry.

Within the poultry industry, Salmonella was managed as primarily as a problem of animal health, focused on PD and FT. However, public health officials were becoming increasingly concerned about the potential source of Salmonella risk to human health, as they were culturing many Salmonella serotypes pathogenic to man, particularly from fowl sources. Indeed, Edwards’ 1950s review papers posited that fowls were likely the biggest reservoir of

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30 Fran Gage, “Eggs,” The Oxford Encyclopedia of Food and Drink in America.
Salmonella risk to man, based on the frequency of isolations from that reservoir. Public health officials had other reasons to suspect fowls. In the 1940s, Salmonella contaminated dried egg powder was at the center of a trade issue between the US and the UK, when the UK isolated Salmonella from egg powder it had imported from the US. In that same decade, a PHS investigation of a salmonellosis outbreak on a merchant vessel implicated an egg salad that contained mayonnaise made from raw shell eggs. Edwards drew the egg powder and shell egg problem together, noting that if “cracked and soiled eggs” that could not be marketed as quality shell eggs were instead used in “dried and frozen egg products,” the logical inference would be that the latter would have a high incidence of Salmonella. He suspected that lower quality eggs were being diverted to make products like dried egg powder.

However, the two events that would ultimately push poultry safety into the national limelight came from media coverage. The deaths of several poultry workers in association with two psittacosis outbreaks (that had also sickened hundreds of workers and infected tens of thousands of turkeys) attracted national media attention. In August 1956, McCall’s Redbook published an investigation into the sanitary practices of poultry processors, called “How Safe is the Poultry You Eat?”

Redbook was a popular midcentury magazine aimed at middle-class American women. That the magazine would publish an investigative piece on poultry production was in keeping with the magazine’s frequent attention to health and food related pieces. However, the alarming message that American poultry presented danger stood in stark contrast to the rest of Redbook’s pages, chock-full of advertisements for food, modern kitchen gadgets, and other consumer

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32 Psittacosis is not caused by a Salmonella, but Chlamydophila psittaci, which sickens both birds and humans.
products. That such a piece would appear in *Redbook* reflected a deep and long-term cultural association between women, food, and political consumerism.\(^{33}\) Harvey W. Wiley, who had headed up the crusade to pass the Pure Food and Drug Act of 1906, joined *Good Housekeeping* in 1915 and opened a research institute for the magazine to test the safety of consumer products and disseminate the information to women consumers.\(^{34}\) The long-standing and powerful association between women, food, and political consumerism, I will show, continued to make its appearance in poultry safety debates.

In the wake of the media coverage, in 1956 and 1957, Congressional committees called five different hearings to consider the establishment of a compulsory federal poultry inspection program as the key means of controlling the fowl problem. Senator James Murray (D-MT), chairman of the “Subcommittee on Legislation Affecting the Food and Drug Administration,” argued strongly for compulsory federal inspection, citing the fact that the PHS reported that one-third of reported cases of human food poisoning were traced yearly to poultry or poultry diseases, as well as the fact that there had been periodic outbreaks of disease in poultry-plant workers.

One of the major questions debated in the hearings was whether the program should be under the jurisdiction of the USDA, responsible for beef inspection, or the responsibility of the FDA. Murray stated that his “strong belief” was that the FDA should run the compulsory program. He contrasted the “protection of consumers” as the “fundamental objective of inspection” at the FDA, with the USDA’s main mission of promoting American agriculture.


\(^{34}\) Coppin & High (1999).
Shirley Barker of the Amalgamated Meat Cutters and Butcher Workmen of North America provided in-depth testimony about the poultry plant-worker psittacosis disease outbreaks, beginning his remarks with the damning indictment that in many plants, it was not only the healthy bird that was processed, but the disease one as well, reflecting a “scandalous lack of sanitation.” Barker argued that the fast growth of the poultry industry had encouraged a number of “shoddy investors” who desired a “quick and easy profit no matter what dangers or consequences result to the public or industry.” These “chiselers,” he argued, made it difficult for “good and honest” processors to stay competitive and offer quality products.

In addition to the psittacosis outbreaks, Barker cited ten “typical outbreaks among consumers” that had been investigated by the CDC, including a prison outbreak of salmonellosis traced to Thanksgiving turkey, a restaurant outbreak traced to roast turkey, and a staphylococcus outbreak at a “state institution” traced to chicken salad. He used these outbreaks as evidence to “demonstrate the dangers posed to your family, mine, and all consumers,” as well as to cast doubt on the “old argument” that if the poultry were cooked adequately it would not cause disease.

Barker drew on the cultural association between women, food, and consumerism to make a counter-argument against the idea that foodborne illness should be controlled by consumer cooking. He argued:

…even if the theory about cooking poultry were true, there is no reason in the world why the housewife should get garbage for her money. She has the right and expects to be protected against disease whether she cooks her poultry well or not. All steps should be taken to assure the housewife that the chicken, turkey, or duck she brings into her kitchen is a healthy one, which offers no danger to her family.
One particularly interesting aspect of Barker’s statement was that he referred to this theory as an old argument, one that was likely to have been circulating in specialist circles, but now was entered into the public record in a Congressional hearing.

Another manifestation of the deep cultural association between women, food, and political consumerism was the fact that the consumer organizations in attendance at the hearing were primarily women’s groups, a constituency that had played a significant role in the sanitary movement and fight for pure foods and drugs. The women in these organizations were representatives of the ordinary housewife, the arbiter of food for her family. As housewives, they narrated a major societal shift in food production and its health implications to the end consumer through their gendered subject position. For instance, Genevive Oslund of the General Federation of Women’s Clubs commented:

In earlier days, when life in our country was less complex, the canny eye of the housewife could tell by looking at the color of the comb, the condition of the feet and feathers, whether the bird was healthy. Today, the busy housewife is glad to find her poultry already killed and dressed, but her clues for wholesomeness have been eliminated.

Frances Wright, of Housewives United, used a similar rhetorical strategy, contrasting her childhood memory of her grandmother cooking a chicken from her backyard flock for Sunday dinner with today’s “Mrs. Housewife” procuring her chicken from the supermarket. Wright further argued that the “blame” for contaminated chicken was “frequently hidden in anonymity” (229). Both Oslund and Wright used portrayed the housewife’s dilemma, that she could no longer directly discern the risks of her food as a tactic to argue in support of compulsory federal inspection. They supported the passage of a bill (S. 3176) to amend the FDCA to “prohibit the

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35 Goodwin (1999)
36 p. 217.
movement in interstate or foreign commerce of unsound, unhealthful, diseased, unwholesome, or adulterated poultry or poultry products.”

S. 3176 and the other bills related to poultry inspection collectively became known as the Poultry Products Inspection Act (PPIA), which was passed in 1957 and went into effect in 1959. While sponsors of S. 3176 had argued that the FDA should be responsible for compulsory poultry inspection, the PPIA awarded the responsibility to the USDA. Brent Riffel (1997) argues that the PPIA was a compromise, and ignored worker safety even though the issue had been key to attracting public and policy attention to poultry contamination. He argues that both large and small firms in the poultry industry had converged to support the USDA’s oversight of the inspection program, fearing that the FDA’s regulation would be too onerous. Furthermore, he contends that compulsory federal inspection furthered the mechanization and vertical integration of the industry, with small firms merging with and being acquired by larger ones. Ultimately, Riffel argues, this resulted in the increased acceleration of line speeds, to make poultry production one of the most dangerous jobs in the country.

Joe Atkinson, a PHS official, argued that the PPIA had major problems because it left regulatory gaps and placed undue burden on states and local agencies. For example, he noted that under the PPIA, only 1000 plants would be serviced by 1960, but there were approximately 2500 existing plants. He worried that this would cause the as-yet-uninspected plants to be a “dumping ground for diseased flocks,” unless state and local programs could assume adequate responsibility for the unserviced areas. The PPIA had no measures to support outbreak investigations, conducted by state and local public health officials. Atkinson also worried that since the PPIA had several exemptions, uninspected poultry could still be delivered to hotels,

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restaurants, and boarding houses. The Federal law focused heavily on the processing plant, even though many other spaces were involved in the poultry chain.

In his critique of the PPIA, Atkinson brought to the fore how central the distribution of responsibility was in the decision to craft control measures. “Where responsibility rests,” he argued, was a key issue to consider when assessing control measures for public health problems, and one that was not adequately acknowledged in the PPIA. He argued that since the consumer could not ensure the safety of food (herself), the consumer “looks to government for assurance and protection.” By not adopting a control measure that would address the full scope of the problem, by implication, the government was adopting a “let the buyer beware” position, one he felt was a morally unacceptable position to take in the domain of food oversight.

**Conclusion**

This chapter began the project of historicizing the phenomenon of foodborne disease surveillance, and how a situation arose in which building better disease surveillance systems seemed both necessary and possible. It first described three key elements of disease surveillance infrastructure particularly relevant to foodborne outbreaks: disease reporting; the rise of the CDC; and identifying, measuring, and classifying *Salmonella*. I showed that while these three elements of infrastructure were crucial ingredients for helping to make salmonellosis a more visible public health problem in post-WWII America, they were not sufficient. Making disease visible takes enormous infrastructure, and widespread social action. I documented the work conducted by Philip Edwards to simplify a *Salmonella* serotyping routine for the average state laboratory to increase the level of data production, as well as the partial picture of large scale food infection Edwards and other researchers had been able to create from studies of *Salmonella*
cultures, contamination issues, and outbreak investigations. One of the major foci of public health concern emerging from this partial picture was the fowl problem, one created by the increasing industrialization of the poultry industry and its association with higher risk of communicable disease, not only for birds but for people too.

However, what animated and fueled public and policy concern about the fowl problem, I showed, was media coverage of unsanitary poultry processing as well as the deaths of poultry workers. I examined this policy debate, calling attention to the relevance of the cultural association between women, food, and political consumerism in shaping discourse about consumer responsibility. While Congress passed a control measure for the fowl problem, with the USDA overseeing a compulsory federal inspection program, many social groups expressed dissatisfaction with its effects. Worker safety advocates felt their concerns erased from the discussion. Smaller firms could no longer afford to process poultry. Public health officials saw gaps in the regulation, and no additional support for outbreak investigations at the state or local level. Caveat emptor ruled the food marketplace.

My goal for the chapter was to document and describe the public health surveillance infrastructure and efforts made to render salmonellosis more visible as a public health problem, particularly in the 1940s-1950s, which culminated in a major policy debate on poultry safety. On the one hand, the machinery that was built did contribute to making salmonellosis more visible, but also made limits of surveillance more apparent, contributing to ongoing efforts to enhance the epidemiologic picture of salmonellosis. By the 1960s, a situation was in place that helped to create conditions of possibility for continual efforts to improve disease surveillance, and in particular, detection of national outbreaks. Since the fowl problem was not fully
controlled by the PPIA, it would make its appearance in other forms, as the next chapter discusses.
Chapter 4. “Salmonellae on the Grocery Shelf:” Industrial Food, the Interstate Outbreak, and the National Salmonella Surveillance Program, 1960s-1970s

“In the past,” Theodore Eickhoff penned in a 1967 editorial entitled “Salmonellae on the Grocery Shelf,” foodborne outbreaks of salmonellosis centered in the home or at a community gathering. However, he continued, in the past several decades, outbreaks had shifted drastically:

...a developing food technology has sharply changed patterns of food processing, distribution, and marketing. Complex patterns in the epidemiology of salmonellosis have simultaneously begun to unfold... these patterns relate, in many instances, to the presence of salmonellae in commercially processed and nationally distributed foodstuffs. The implications are broad and disturbing to both the consumer and the entire food industry.

Between the passage of the PPIA in 1957 and Eickhoff’s editorial in 1967, the complex patterns in the epidemiology of salmonellosis that had begun to unfold were made visible by increasing foodborne surveillance infrastructure, particularly, the creation of the National Salmonella Surveillance Program (NSSP) in 1962.

This chapter examines the building of the NSSP; the NSSP role’s in helping to support the investigation of two contamination issues (one, a different manifestation of the fowl problem, a hospital-based epidemic associated with shell eggs; and two, contamination of nationally distributed instant non-fat dried powdered milk); and finally, I examine debates about Salmonella control between the 1950s-1970s. In this chapter, I argue that the NSSP was established as a second-order system, building upon previously lain infrastructure to help strengthen Salmonella surveillance, making the disease more

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visible at a population level. The system was built within and interacted with an environment where epidemics of salmonellosis raised new and difficult dilemmas for public health, hospital, and regulatory officials around when, how, and what to communicate to which publics about food hazards, and the proper means and responsible entities for controlling food hazards. Making salmonellosis visible with new disease detection machinery was not only a technical achievement, but enabled other forms of visibility and social action: increased media coverage, public awareness, and higher stakes; a redefinition of the geographic locus of the problem; and new issues and directions for control and inter-sector communication.

**Initiating the NSSP, 1962-1963**

As I discussed in the previous chapter, Salmonella surveillance efforts in the 1950s created a disturbing but partial picture of large scale food infection. I argue that this is one of the key factors leading the CDC to initiate the NSSP in 1962. The NSSP would entail more intensive cooperation between the CDC and with state health departments to improve the quality of reporting, on a voluntary basis. In an introduction to a Public Health Reports symposium on salmonellosis, James Steele, chief of the CDC’s Veterinary Public Health Section, stated that the program’s goal was to “provide more adequate information as to the true prevalence of salmonellosis in man, animals, and their foods in the United States,” which would help “lead to the development of better methods to control the problem.” Two other CDC scientists, Philip Brachman and James Goldsby, articulated the goal of the program in the following way:

> The aim of our surveillance program has been to design a reporting system that would collect data, giving a clearer picture, a truer definition of the salmonella problems in the United States. Vital to the program has been the need for it to be

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sensitive enough to develop the endemic problems and yet acute enough to allow for the rapid identification of an epidemic or a potential epidemic, especially one with interstate movement.\(^3\)

The program targeted two foodborne disease problems—the need to elucidate endemic, or chronic issues (i.e. sporadic cases), and the need to quickly identify a potential epidemic associated with the movement of a food through interstate commerce.

One broad way that Brachman and Goldsby framed the NSSP was as a “blanket of surveillance” that could “cover all areas” contributing to the salmonella problem. This “blanket” metaphor helped signal the applicability of the theoretic concepts of layered, second-order systems, whose aims were to map and produce representations of a multi-component phenomenon. “Surveillance,” they argued, while not a “panacea of control,” could not only be a “beacon light, an alarm system, and chronicler of events.” It could also, they argue, be a “guide to better things for better living through surveillance.”

Concretely, creating a more comprehensive surveillance system would help answer a key question posed in the 1950s. How much of the observed increase in salmonellosis over time due to better reporting versus a true increase in incidence? Glenn Slocum, director of the Division of Microbiology at the FDA, was enthusiastic about the CDC’s NSSP. He pointed out that the number of cases of salmonellosis had risen from 882 in 1948 to 6,929 in 1960, and leaned toward it being a true rise in disease.\(^4\) However, since the source of infection was “rarely established” in most cases, and the reporting of foodborne cases was “grossly incomplete,” at the moment, a good answer to the question could not be given. What was needed, he argued, were “vastly improved systems for the epidemiologic investigation and reporting of salmonellosis to

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identify the food products and establishments in need of application of control measures.” The
NSSP would be “an important step in that direction.”

Slocum had been at the FDA for 30 years, and worked on foodborne disease issues for
much of it. In the 1950s, the FDA had taken regulatory actions to seize products in interstate
commerce associated with four salmonellosis outbreaks involving direct epidemiologic
implication of foods: canned egg-yolk powder for infants, a dry baby-formula product, yeast
powder, and hollandaise sauce. However, he noted that there were three salmonella outbreaks
where “possible interstate sources” were suspected, but “epidemiologic studies failed to discover
the origin of these episodes.” One was the 1957 S. reading outbreak mentioned in Chapter 3,
another was a 1956 Midwestern typhoid fever outbreak, and the third was a multistate increase in
S. Hartford infections detected by laboratory surveillance in 1962, suggesting but not proving
that an outbreak had occurred. The NSSP would benefit the FDA by helping inform its
development of general control measures, and also help find the FDA-regulated sources of multi-
state outbreaks like the three that had been suspected, but not pinpointed.

On May 29, 1962, the CDC’s Salmonella Surveillance Unit of the Investigations Section
of the Epidemiology Branch sent out its first report to its distribution list describing the
program.\footnote{National Archives, RG 442. USPHS CDC Salmonella Surveillance Reports (SSR), Nos. 1-19, 1962-1963. May
29, 1962.} The initial distribution list included state departments of public health; PHS regional
health directors; CDC leadership in the laboratory, bacteriology, and epidemiology branches;
Canadian public health officials; and a famous university researcher of foodborne disease, Gail
Dack at the Food Research Institute. The program started with increased Salmonella
surveillance in 8 states (CA, GA, IL, MA, NY, NC, WA, WI), one city laboratory (NYC), and
one hospital laboratory (NY Beth Israel). The program started with these sites because they had volunteered to do so, responding to the CDC’s “invitation” to submit their isolations of salmonella on a monthly basis, as well as “narrative material” about their own investigations. The NSSP’s first report provided a “summary” of the data that these ten “reporting centers” had shared, with primarily covered data from April.

I wish to point out that the information sharing occurring here was novel in several ways. Normally, disease reporting operated on a yearly or quarterly basis. Annual and quarterly reporting involved the calculation of summary data about outbreaks, sources, and seasonal and regional patterns. To share data on a more frequent basis, monthly, and data beyond summary data, would produce more actionable data relevant to possible disease epidemics occurring in the world. One thing marking the novelty of data sharing here was that the report was prominently labeled as “for administrative use only, not for publication,” and the distribution list was limited to a small audience of users. Much of the information shared would be much more provisional than quarterly or yearly statistics reports, especially because it would potentially be used to take administrative actions and implicate potential producers of contaminated food. While improving data sharing amongst reporting centers and creating more robust chains of cause was likely to improve the detection and investigation of outbreaks, it was important to create protected channels for sharing possibly sensitive information with major public health, legal, and economic implications.6

Another aspect of the program that is important to note is that the NSSP was a voluntary, not mandatory program. The language of the memo describing the report highlighted the program’s voluntary nature:

6 On the relationship between chains of cause and implication, see Gieryn and Figert (1990).
We hope this surveillance program will stimulate increased investigations of individual and contact cases. The concept that each reported case is, in reality, associated with additional cases which are not uncovered is an intriguing thought which needs further confirmation.

The language of the memo emphasized that each individual case was potentially part of a much bigger picture, one that states could work together to solve. The memo used language that invited state disease detectives to participate in solving a joint mystery, confirming the “intriguing thought” that individual cases actually constituted a multi-state outbreak. The NSSP would benefit workers in both the laboratory and field, across the country. Each laboratory could compare their isolation results with other laboratories, and feed this information into field investigations. The 10 reporting centers were only the beginning; the CDC had plans to expand the NSSP to “other interested states.”

The memo also highlighted the urgency of the *Salmonella* problem. It pointed out how “frightening” it was that *Salmonella* could be isolated so easily from food and animal sources. But the question was, how could the relationship between human disease and the salmonella that was isolated from food be confirmed? Through the NSSP, the CDC hoped that it would be able to further “develop interesting associations between nonhuman sources of salmonella and human susceptibles through a surveillance of all salmonella recoveries associated with appropriate investigations.”

How would these “interesting” associations be discovered? The memo highlighted the power of serotyping, and particular the rare serotype, as an epidemiologic tool. It stated:

The recovery of the rare serotype from any source is in some respects of greater potential significance than recovery of one serotype from multiple persons involved in a focal epidemic such as following a picnic supper. The rare serotype is like a *radioactive-tagged marker*, demanding every attempt to identify its source and its range of contamination (italics added).
The “radioactive-tagged marker” was a dramatic metaphor highlighting the importance of the rare serotype as an investigative tool. The rare serotype became a laboratory tool for localizing, and surfacing, distinct situations of making and eating contaminated food.

One of the investigations featured in the NSSP’s first report involved an outbreak of *S. thompson*, which had first come to light through a report from Canadian public health officials. They had noticed an increased incidence of human and animal salmonellosis cases of *S. thompson* in Newfoundland, around the time that one of its laboratories isolated *S. thompson* from many packaged cake mixes. In response, the CDC decided to round up all of its *S. thompson* isolates from 1961 (109 human cases). Of the 109 cases, since 21 were from Michigan, the Michigan State Department of Health decided to conduct an investigation. They figured out that many of the cases were from the same region in the greater Detroit area, and had eaten éclairs from the same small neighborhood bakery within 24 hours of the onset of their illness. An investigation of the ingredients of the éclair suggested that fresh eggs were a potential vehicle. Public health officials traced the source of the eggs back to a single large chicken breeder, who had lost many chickens in his flocks due to *S. thompson* infections. Fecal specimens from the breeder’s flock houses yielded *S. thompson*.

The report emphasized the significance of this investigation. It was an “affirmation of the epidemiologic approach to the human salmonella problem,” demonstrating that connecting distributed cases through epidemiologic and laboratory data, follow-up investigation, and source tracing with confirmatory laboratory testing could successfully lead to the identification of common source outbreaks. The report acknowledged that food history studies could sometimes be “tedious” and “often only minimally productive.” However, it pointed to the Thompson
outbreak as an example where food histories were the first step to help identify a “single common source.” The case interviews yielded the éclair exposure, which ultimately helped lead to the single large chicken breeder.

This investigation also led the CDC to review all of its *Salmonella* isolations between 1947-1958. Analysis of those 28,000 cultures led them to identify what looked like an S. thompson epidemic in 1955, showing a pattern with 3 distinct “blips” of 40, 10, and 5 cases. Disturbingly, it was possible that this epidemic was still occurring, since S. thompson cases were still popping up. According to the NSSP’s April data, 7 of the isolates were *S. thompson*.

One of the report’s mechanisms for facilitating more communication between federal and state officials was to include various “reports from states,” so that individual states could share information about anything related to salmonellosis in their state, whether it was a discussion of their recent isolations, in-state outbreak investigations, or applied research studies. After each report from a state, the CDC included an “editor’s comment,” highlighting what was most interesting about each report, or sharing additional information to help states interpret and understand what was going on.

For example, the Georgia Department of Public Health reported a study in progress analyzing the potential food poisoning hazards associated with commercially prepared and wrapped sandwiches. The sandwiches were typically kept at ambient temperature for up to 48 hours before they were sold, suggesting risk of increased bacterial growth. However, thus far they had not been associated with any outbreaks. Georgia sampled 800 sandwiches of different varieties and had not isolated any salmonella, therefore suggesting that commercially wrapped and prepared sandwiches were “not very likely to be incriminated in food poisoning.” The editor
called this “an encouragingly unexpected result,” but pointed out that other “caterer-induced”
salmonella outbreaks suggested that individual food handlers “may dramatically alter the
contamination rate.” By providing editorial commentary, the report aimed to serve as a
mechanism for community-building, knowledge-sharing, and collaborative sensemaking
between federal and state officials about a shared problem of nationwide human salmonellosis.

As the months went on, more states joined the program—TN and MD in May; NJ in
June; CO, DE, MN, OH, and RI in July. At the beginning of 1963, the CDC expanded the
NSSP to all 50 states, the District of Columbia, and the Virgin Islands, though it took a few more
months for most of the locations to implement the reporting fully. As the program expanded, the
CDC asked reporting centers to submit their isolate data on a weekly basis.

**Systematizing the NSSP**

As the program grew, CDC officials created a more formal workflow for reporting.
Every week, the reporting centers would create a report and mail it on Friday afternoon to the
CDC. The report would summarize all the salmonella isolates the reporting center had identified
in the past 7 days. The isolates were usually from ill persons or healthy carriers (likely food
handlers). The CDC created a reporting form to help structure the information the reporting
center needed to fill out and mail, including essential information such as: individual serotype
identified; name of patient, initials, or laboratory number; sex and age of patient; and country of
residence. For nonhuman isolations, the information requested was serotype, source, and
country. These data constituted the “basic keys to the epidemiology of salmonellosis.” To
illustrate, I excerpt the reporting form (Figure 15):

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Figure 15. Weekly Salmonella Surveillance Reporting Form

The form would create five copies, using carbon paper. The state would keep two of the copies. One would go to the department that filled out the form, and the other would go to either the laboratory or epidemiology chief in the state. The CDC would keep three copies. One was for the epidemiologist’s “scrutiny,” one was for the statistician’s “running record,” and one was for the “IBM punch card section,” as each isolate’s information was punched onto one card.

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The summary reports produced by the CDC would continue to be distributed on a monthly basis. However, the weekly data the states provided would be analyzed weekly, and any “unusual or urgent information” discovered would be “relayed promptly to the involved State or States by telephone.” Rapid communication by telephone or telegraph would be necessary if a possible epidemic or outbreak was occurring, and the NSSP was instrumental for discerning whether that was the case. As EIS Officer Charles McCall explained,

By definition, an epidemic involves a significant increase in the usual prevalence of a disease over a specified period of time and within a specific geographical area. Recognition of the large outbreak of salmonellosis, therefore, depends on a base line of specific information about the disease within the area involved, be it a State, region, or nation. This data must be developed by surveillance. The basic information presently in use to establish the norm is age, sex, date of isolation, geographical location, and serotype. Deviations of any one of these parameters from the norm within the area involved serve as indicators of an outbreak.9

McCall’s explanation of an epidemic’s definition emphasized that the weekly reporting states conducted contributed to creating a picture of the “base line,” out of which a localized epidemic could be identified. By consistently inputting core data, federal officials could monitor that data to identify deviations from normal patterns, indicating that an outbreak might be occurring.

In order to help them visualize the outbreaks from incoming NSSP data, the CDC created a “salmonella board,” a “large tally board” for monitoring the “most valuable parameters” for detecting outbreaks. The CDC found that the most valuable parameters for outbreak detection were incidence and serotype frequency, as portrayed in a picture of the tally board, below (Figure 16).

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9 Proceedings, p. 43.
Officials could find outbreaks on the tally board by listing state data separately and then grouping states within regions, as many multi-state outbreaks exhibited regional patterns, due to regional patterns in how common foods were distributed. They used fairly simple calculations in their analysis: cumulative totals of each serotype, and the percentage of total isolations represented by common serotype. When new isolates were reported, their number and location would “significantly alter prevalences portrayed by the board.” McCall called the board a “moving picture of the prevalence of salmonellosis throughout the country.”

As the NSSP expanded to more states, the distribution list for the monthly report continued to grow, even internationally, for instance, extending to public health officials at the World Health Organization, and in India and other nations. The reports detailed Salmonella data

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10 Proceedings, p. 45.
from an amazing variety of sources and places, and about incredibly diverse situations. Every month added to an ever-increasing list of multifarious sites where Salmonella made its home. For instance, the June 1962 report described “two unusual outbreaks,” one in children who had received chicks as Easter gifts, another associated with a vending machine distributing éclairs made in a “dilapidated and infested” bakery. As the report observed, Salmonella surveillance had an exceedingly “vicissitudinous character.”


The April 1963 report contained an account from a New York investigation that was initiated in March. On March 16, the Beth Israel Salmonella Center in NYC had isolated S. derby from one stool culture, a routine event until about two weeks later, when, on March 29, four more cultures yielded S. derby. All five cultures were from the same hospital, which triggered an NYC Health Department investigation. The five patients turned out to be post-operative surgical cases, and in examining their clinical symptoms and hospital experiences, the investigation revealed that the duration between their surgical procedures and infection was too long to be surgery-associated. What started as a hospital-associated outbreak soon shifted into a foodborne outbreak investigation.

Investigators examined all the spaces where food was prepared in the hospital. They found two primary preparation spaces: the main kitchen and the “feeding kitchen” (22). The main kitchen made food for all patients and employees. Investigators determined that since employees were not ill, they could eliminate the main kitchen as a source of the contamination. The feeding kitchen was responsible for creating liquid diets and food supplements primarily for

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12 SSR, April 26, 1963.
the post-operative patients. In the feeding kitchen, investigators found a number of “poor food handling” practices, and no salmonella in the stools of food handlers, indicating that the source of the trouble was a food that had come into the kitchen already contaminated.

The key suspect became eggs, because investigators noted that the eggs in the kitchen were “cracked and soiled.” But when they tried to “trace down” the source of the eggs, they had little luck. The eggs were supplied by four dairies that were in turn supplied by many different farms. The NYC report concluded with the observation that this was yet another “hospital acquired salmonella infection.” Previous NSSP reports had contained discussions of many other salmonella problems occurring in hospitals across the country. While “contamination from a single source” was “implicated,” what that single source was remained obscure, lost amongst the many farms that supplied eggs to the hospital.

Later, these five NYC cases would be marked as the beginning of an enormous *S. derby* epidemic, a disease event would last for over a year. In the next month, the Philadelphia Department of Public Health reported 20 cases of *S. derby* in one of their hospitals. Over the next couple of weeks, four other hospitals also reported outbreaks. By mid-June, there were 400 laboratory confirmed cases of *S. derby* illnesses.

Federal and state public health officials launched a major, large-scale investigation, which also included directors of multiple hospitals, as well as regulatory officials from many agencies and levels. The CDC assumed the role of “central clearinghouse” to help coordinate the investigation.13 Because *S. derby* cases appeared in multiple hospitals in different states, this “incriminated a common source of infection involved in interstate movement.”

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13 Proceedings, p. 111.
The uniquely vulnerable population involved in the outbreak helped officials determine that a common food was the source of the trouble. Food histories were easy to collect, since the patients were receiving such restricted diets controlled by the hospital. Analysis of the foods commonly eaten yielded one “almost universally” consumed food—“raw or undercooked eggs.” Upon identifying the “raw or undercooked egg” exposure from the food histories, officials launched an extensive field investigation. Tracing eggs from hospitals to large distributors, to wholesalers, to regional processors, and then to farms revealed that up to 12,000 poultry farms could have been the supplier of the *S. derby* contaminated eggs. The epidemic had such major public health implications that on July 11, 1963, the Surgeon General issued a public warning, recommending that the use of raw or undercooked eggs be discontinued in hospitals.

Public health officials continued to monitor the problem over the next months. Surveillance data suggested that the July warning had an effect, as the subsequent months showed on the epidemic curve that the June/July time period was the peak of the epidemic, eventually reducing downwards to a lower plateau. However, in December 1963, officials discerned a second peak, an uptick of cases, shown in the following graph.

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14 Proceedings, p. 113.
Three months later, the epidemic was still ongoing. The CDC had organized a “National Conference on Salmonellosis,” to discuss the “multiple interrelated facets of the problems associated with salmonellosis.”¹⁵ Over 200 attendees from different state and federal agencies, industry representatives, and university scientists participated. The *S. derby* epidemic was one of the key discussion topics, though many other topics were on the agenda. Though state and federal public health officials were already collaborating around the NSSP, the goal of the national conference was to create a forum for the “interchange of ideas” and “opening channels of communication” amongst a wider group, which would be “necessary to the development of a successful salmonella control program.” That is, while the NSSP had successfully improved *Salmonella* surveillance, *Salmonella* control was another matter, as the *S. derby* epidemic showed.

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¹⁵ Proceedings.
The CDC had organized a panel specifically focused on the *S. derby* epidemic. During the discussion period, some of the thornier issues encountered during the investigation came to the fore. The *S. derby* outbreak had raised new and difficult dilemmas for public health and hospital officials around when, how, and what to communicate to which publics about food hazards, and what kinds of control measures should be put into place, by whom.

Philip Levine, of the New York State Veterinary College in Ithaca, opened the discussion period with a strong critique of public health communications during the outbreak. He contended that the press releases sent out to the public announcing the derby outbreak should not have been sent out to the “nation at large,” because the problem was localized to hospitals. He criticized the “inferences drawn without absolute proof,” which ultimately caused “damage” to the egg industry.

Langmuir asked Levine to clarify his critique of the press releases. Levine argued that the first one was problematic because it contained a statement saying something to the effect that “it was presumed or it was suspected that eggs were the source of these infections,” and the second one was problematic because it “enjoined the population at large from eating cracked eggs.” Levine cited a complaint he made to the CDC over the phone, asking where he “could buy a dozen cracked eggs… I did not know that that was a marketable commodity of any significance.” Another attendee concurred with Levine’s critique. Don Turnbull of the American Poultry and Hatchery Federation criticized the press release for not making it clear that it was a “hospital situation and not a general public situation.”

The PHS’s actual press release contained the statement: “only grade A eggs should be purchased and only well-cooked eggs should be served to infants and the elderly in the family.”
James Goddard, the CDC’s director, defended this statement as “the same recommendations as made for hospital patients.” He argued that “at no time did we state eggs should not be eaten.”

As an example of what was cited in the press, the Chicago Tribune published an AP story on July 11, 1963 representing the PHS recommendation as, “There is sufficient epidemiological and bacteriological evidence to suggest that everyone should avoid buying and using cracked or unclean eggs.”

Langmuir replied at length to the critiques. He pointed out that the first release was prepared by the Philadelphia Department of Health, and focused on the “public in Philadelphia.” The second release was the Surgeon General release, and Langmuir talked about how carefully it had been drafted, cleared, and widely discussed in with many in the government, including with the FDA and the Assistant Secretary of Agriculture. He defended the document as a “thoroughly and carefully reviewed statement with all of the facts known,” not a “fly-by-night release.”

Furthermore, Langmuir emphasized that the problem of public communication was a challenging one for government officials. He said,

we in government face a very difficult and delicate issue whenever a problem reaches the point of sufficient public concern to involve the press. If the newspapers scent headlines while following a trail, then we really have a tough problem to handle.

The Philadelphia Department of Health official, John Hanlon, expanded upon Langmuir’s remarks to defend the public health response. He seconded what Langmuir said about those in “positions of public responsibility,” placing “stress” on the word “public.” Talking about the public health official’s moral dilemma, he stated that “we must operate with a somewhat awesome and frightening responsibility to the public at large and to all segments of it.” Part of

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what made this responsibility so frightening and challenging was that their work took place within a “fish bowl,” with media and the general public making it impossible for agency officials to “hide anything,” though Hanlon said this was “as it should be” within a democratic system.

Hanlon explained why it became difficult for agency officials to keep the epidemic from the press. He observed, “There is something very real about passage of information, correct or incorrect, warped or not, among the public at large.” Since a large-scale investigation had been initiated in multiple hospitals, a “grapevine” quickly developed, with discussions amongst patients, hospital staff, their families and neighbors about the problem. Though the Philadelphia Department of Health attempted to work with hospitals “quietly,” this was to no avail. The hospital and health department soon received a huge volume of calls from the press, television, and radio outlets, some of whom were accusing officials of “hiding things from the public.” Hanlon argued that it was important to take these issues head-on and not to “duck” them. To “protect the public at large,” officials had to “take whatever steps are necessary, based either upon presumptive or clear-cut evidence.” He stated, emphatically, “…one cannot afford to wait in the face of a potential community-wide epidemic. One must act. There is no other choice.”

Several others raised a critique not about public health communications, but of the “wild rumors” proliferating in the press about the epidemic. Cliff Carpenter of the Pacific Dairy and Poultry Association said the releases were “95 to 98 percent factual,” but he complained about the “multimillion dollar headlines” that damaged the industry, such as “Public Health Says Dallas Eggs are Safe to Eat,” and “Other Eggs Dangerous. Local Eggs Okay.” Carpenter defended the industry producers that were “fighting for their economic lives.” He wanted public health officials to pay more attention to “what happens to the releases when they get into the hands of the headline hunters.”
Wade Smith, of the Blanton Smith Company, agreed that the “popular, sensationalizing press” was mishandling the releases. He argued that public health officials should take more care with their releases, so a “nonsophisticated readership” would not misinterpret the announcement. To illustrate the pervasive problem of the “nonsophisticated readership,” he pointed out that on previous day of the conference, he heard an audience member (a “lady”) whisper to another, in response to the “magnitude of the salmonella problem in eggs and poultry,” the question, “Does this mean that we should stop eating eggs altogether?” Smith used the audience member example to argue that even amongst the conference attendees, who were supposed to be experts who knew how to keep the salmonella problem “in its own perspective,” made what Smith felt to be inappropriate inferences about egg safety.

Brachman, Langmuir, and Goddard launched into a detailed explanation and defense of public communications. Brachman argued that the PHS news release did in fact make it clear that this was a hospital problem, not a general public problem. Langmuir described how the Philadelphia “situation” unfolded and that health officers felt “constrained” to make a recommendation to local hospitals. This “hit” the Philadelphia papers, and then received wider coverage. As the epidemic continued, officials published updates on a weekly basis in the CDC’s publicly accessible journal *Morbidity and Mortality Weekly*, though they did not initially mention eggs. Langmuir emphasized how extended the process had been, that “we were aware that this was coming and over a period of some weeks kept briefing Dr. Goddard of the problem.” Driving the release of the PHS statement in July, he argued, was actually the press; “reporters in Washington were demanding statements from the Surgeon General, he was obligated to make some sort of reply.”
Goddard then described his interactions with the Surgeon General. Goddard and Langmuir reviewed the field investigation data that had been collected up to that point, created a draft news release, and Goddard reviewed the release with the Surgeon General, whose “public affairs man” suggested some changes. By describing this process, Goddard defended the credibility of the message since so many officials in Washington had reviewed it before it was disseminated. Another tactic he took to defend the credibility of the message was his own identity as a government official. He argued that in this matter, he was speaking as an “administrative person in this matter rather than as a scientist.”17 While scientists might be partial because “they have invested a lot of time and effort in a problem,” as a government official, he would ensure that the scientists would “remain impartial,” that the “facts are laid out,” that the facts would be presented to the public, and that they would make policy recommendations on the basis of those facts.

Responding to the criticism of the press and of the public, Goddard admitted that some reporters were sensationalistic, but defended most of the reporters he had interacted with as “responsible individuals.” While he noted that the public was “not always capable of proper interpretation of these facts,” it was the government’s responsibility to “objectively place the data in its proper perspective.” Goddard closed his explanation and defense with the statement that public health officials felt they enacted their responsibilities to the public to the “best of our knowledge and abilities,” and that he hoped the conference discussion had helped clear up the “misconceptions” that developed in the “publicity” given to the S. derby epidemic.

I have provided an in-depth discussion of the S. derby debate because it demonstrates themes that remain fundamentally important in understanding the issues surrounding the

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17 Proceedings, p. 121.
communication of information about food hazards to the public. Investigations are real events in the world, involving members of the public sickened by contaminated food. Hospital patients and staff gleaned that something major was going on from their involvement and proximity to the issue, and so it was easy for the press to discern that a health problem was occurring. Pressure from the media intensified the need to disclose information about the investigation, a need already present because public health officials were invested in protecting the public from harm, as part of their job. They attempted to craft a careful message grounded in solid facts and communicate with the public about a food hazard, disseminating the information via a press release. As the press carried this official message to the public, they portrayed the risks associated with eggs in many different ways, especially in the headlines. Furthermore, members of the public interpreted the risks associated with eggs in many different ways. The varying representations of the press and the many inferences made by members of the public had enormous economic implications for egg producers.

At the end of the meeting, Langmuir called one of the biggest achievements of the conference the opportunity it afforded for different parties involved with the Salmonellosis problem to get together and exchange information. He said, perhaps only half-jokingly:

Each has found that respectively the other does not have horns, cloven hoofs, and a forked tail…. All of us are better informed on the nature and extent of the problem and on the degree of activity that is under way, seeking for better definitions and solutions of the manifold problems.

Langmuir also commented that the S. derby epidemic demonstrated that society’s priorities related to health needed re-examination. The S. derby epidemic, he said,

piques our pride that in these days of heart surgery, artificial kidneys, and organ transplants, we cannot take dominance over a miniscule little bacillus with a tail on it that gets into our hospitals, causes no end of trouble, and has us stumped.
This whole situation is an indication of the extent to which fashion dictates where the money is spent and to what questions the brains of research workers are applied. In recent times, infectious diseases have been relatively ignored.

Langmuir’s remarks need to be interpreted in light of what is widely termed the “epidemiological transition,” the shift over the course of the 20th century in national disease patterns moving from primarily infectious diseases (tuberculosis, typhoid, syphilis, and diphtheria) to, in the mid-century onward, a transition to concerns about chronic diseases, such as heart disease and cancer. Salmonellosis, Langmuir argued, demonstrated that infectious diseases were still an important societal problem. In his view, the epidemiologic transition was not simply a story of conquering infectious disease, but also was a story of the de-emphasis of infectious disease in favor of high-technology medical problems.

In 1968, CDC officials published a five-year review on “Epidemic Salmonellosis in Hospitals and Institutions,” analyzing surveillance data from the NSSP. The review pointed out how important it was for hospitals to have an “alert” surveillance system, because hospitals posed a “unique and serious hazard;” populations in hospitals were more susceptible to pathogens than the general population, and the close quarters and medical procedures in the hospital environment made it easier to spread disease. That the review focused more on the space of the hospital as a control site, rather than the control of food contamination outside of the hospital, suggests that the S. derby epidemic was interpreted more as a hospital issue rather than a food issue. Indeed, the review article closed with the pessimistic prediction that the “persistent problem of nosocomial infections,” combined with the “ubiquity of salmonellas,” would continue to place hospital patients at risk for salmonellosis outbreaks.

18 Porter (2005).
One way to understand why the solution focused on the space of the hospital as a surveillance and control site rather than the problem of the ubiquitous Salmonella in the food supply is an observation made by medical sociologist Everett C. Hughes (1951)—“one man’s routine of work is made up of the emergencies of other people.” That is, different actors have different social roles and thus different perspectives on what constitutes an “emergency” and what makes up the “routine.” From a patient’s perspective, a medical crisis is an emergency not experienced everyday. From a medical professional’s perspective, since they see a number of patients, those emergencies are intensely managed through professional routines of work. Hospital professionals have the capacity and competency to control the space and practices within the hospital, but no such jurisdiction over the larger food supply.19

A Starlac Story: 1966-1968

Though the hospital epidemic associated with contaminated eggs attracted some public attention to Salmonella, another contaminated food would help make Salmonella more of a household name—instant non-fat dried milk (INDM). INDM was a food whose production expanded greatly in the WWII era, to help supply a form of milk with a long-shelf life and easy transportability to troops (Smith-Howard 2013). The war helped transform a formerly stigmatized skim milk from hog slop and chicken feed to human food.

In this section, I describe how the NSSP helped federal and state officials coordinate their investigation work related to the contamination of an INDM product. In many ways, this investigation bears similarities to modern investigations, which this description will show. But there are key differences as well. For example, the investigation lasted for almost a year; much

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19 This discussion relates to an extensive literature on social problems definition within sociology. For key works, see Blumer (1971), Kituse and Spector (1973), and Gusfield (1984).
communication was by mail (likely a great deal by phone, though phone conversations are rarely archived); and rapid communication of urgent official information took place by teletype. In addition, I wish to emphasize the novelty of the issue’s scope, and highlight that many of the communications that took place and the actions that were taken were unprecedented. This contrasts significantly with how, in modern outbreak detection and response, the emergency in many ways has become “routine.”

The INDM issue first came to light on January 19, 1966, when Donald Coohon, an epidemiologist from MI sent the CDC’s Salmonella Surveillance Unit a memo. The memo informed the CDC that MI had recently isolated *S. new-brunswick* from two infant isolates. While the infants were unrelated and lived in two different counties, the infant’s parents reported a common theme. Lately, the infants had been started on new powdered milk formulas. One brand was “Carnation brand instant non-fat skim milk,” and the other brand was “Food Club Brand.” The MI lab had purchased samples of these two brands and tested them for salmonella, but had gotten negative results.

*S. new-brunswick* was a rare serotype, but the young ages of the especially vulnerable cases especially concerned MI, which was likely why they had investigated the cases and conducted food testing in the lab. The CDC Salmonella Surveillance officer, James Goldsby, soon mailed MI a reply, as he had found the MI report “very interesting.” Goldsby looked into the demographics of all cases of *S. new-brunswick* from 1965, and saw a majority of cases between September and December were infants (5 of 9). Goldsby then sent letters to the other...

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20 I discuss this issue further later in the chapter.
21 RG 442. 73A1535: Box 3. As reported in a letter dated February 3, 1966, from Goldsby replying to Coohon.
22 Ibid.
five states who had reported *S. new-brunswick* cases since October, asking them to conduct food history interviews on those cases, and in particular, collect brand information.

In February, officials identified thirteen cases of *S. new-brunswick*, many of whom had been exposed to the Carnation brand milk, in multiple states.\(^\text{23}\) Twelve of the states were asked to collect samples of unopened product, and ship them to the CDC for microbiological analysis. During that month as well, Richard Collins, the Chief of the Salmonella Surveillance Unit, sent a telegram, (likely to all the state laboratories and departments of health) telling them to consider all isolations of *Salmonella* with somatic antigen 3, 15 (E2) to be considered as “suspect” for *S. new-brunswick* (antigenic formula = E2; 1, V, 1, 7) and to investigate those isolations for “possible” association with a dried milk product, without waiting for laboratory confirmation to do so.

Collins’ instruction suggests that either the state laboratories did not have the sera to characterize *S. new-brunswick* exactly, or complete characterization would be too lengthy, so the “suspect” strategy was a form of uncertainty management that would help move the investigation forward more quickly. Collins also mailed a copy of the text of the teletype in a “*Salmonella new-brunswick* Alert!” to “all recipients of the Salmonella Surveillance Report,” to keep all members of the distribution list informed of what was looking like an important unfolding issue.

State and federal laboratories worked to try to isolate *S. new-brunswick* from powdered milk samples. In March, the FDA isolated *S. new-brunswick* from shelf samples of three lots from one brand that had been packaged and labeled by a distributor. Investigators identified a specific plant in Plainsview, MN, which became the site of an extensive investigation, lasting for

months. In April, the investigation identified *S. new-brunswick* isolates yielded from “large dried milk particles marketed as coarse animal feed,” and “fine particle powdered milk used as animal feed.” The plant produced 11 million pounds of dried milk yearly, and 1,500 farms supplied the plant with milk.

The Plainsview investigation indicated that the problem could be enormous in scope, so in May the CDC convened a meeting on “Salmonella Contamination of Instant Non-Fat Dried Milk,” to discuss the situation and determine how to coordinate “further investigations and control.” In attendance were officials from the CDC, PHS, FDA, USDA, some state laboratory directors, epidemiologists, and veterinarians, and industry representatives from the American Dry Milk Institute, National Milk Producers Federation, and Milk Industry Foundation. While the archival information did not contain proceedings of the meeting, some post-meeting correspondence over the next couple of months indicated some of the outcomes of the meeting. The American Dry Milk Institute had shared a list of all known dry milk processors with meeting attendees, which the CDC forwarded to state epidemiologists, state health officers, and state agriculture officials, asking them to initiate state-based inspections, and to share results of those studies with the CDC.

The Plainview investigation became even more extensive. The CDC developed a six point objective plan of attack:

1) relating the Plainview plant to as many brands as they could identify as associated with human cases of *S. new-brunswick* through tracing Plainview’s distribution as well as its distribution of (contaminated) animal feeds to farms which in turn would have supplied milk for other brands

2) ruling out human contamination by examining the plant employees’ carrier status

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24 RG 442. 73A1535: Box 3. “Objectives Concerning the Problem of Salmonella New-Brunswick in Instant Non-Fat Dry Milk.”
3) examining how the contamination happened in the plant (through maintaining connections with the MN state agricultural officials)

4) Trying to identify the farm(s) that supplied the original contaminated milk to Plainview (through long term field surveys, veterinarian surveillance of herd health, raw milk sampling)

5) looking at “cycling phenomena” of *S. New-brunswick* in cattle; and

6) finding the original source of cattle contamination.

Because animal feed was involved, the CDC worked with the USDA to analyze cattle fecal samples from Plainview-area farms to look for *S. new-brunswick* more broadly. MN created a “total Salmonella program” at the state-level to “integrate and coordinate the activities of the other agencies,” initiating “intensive epidemiology and follow up of positive findings from the Department of Health laboratories and from laboratories of the other agencies.” MN officials would continue intensive study at the Plainview plant from a processing perspective, as well as of the state’s milk, feed, and cattle population.

The issue widened in scope even further. By June, there were seven more human *S. New-brunswick* cases, two of them employees from the Plainview plant. Results began to filter in from state investigations, uncovering 11 additional *Salmonella* serotypes that were isolated from dry milk products or from plant environments, involving interstate exchange between 6 different states. The state investigations continued.

The USDA’s Dairy Division of its Consumer and Marketing Service began a “Salmonella Surveillance Program,” folding salmonella testing into its quarterly surveys of dry milk plants. The FDA intensified its own investigation of dried milk plants, between May and July asking various district offices to inspect dry milk firms and look at interstate distribution of the product.

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25 RG 442. 73A1535: Box 3. June 23 memo. As of April 13 the USDA had already taken some action, by notifying all plants that they should be in compliance with a particular USDA guideline (3-A practices) and if they were not, they would receive a “limited probationary” categorization. See September 20, 1966, “Joint Investigation of Salmonella New Brunswick in Nonfat Dry Milk Produced by Plainview Milk Products Association, Plainview, MN”
For example, one issue involved microbial evaluation of frozen stocks of “Safeway Instantized Powdered Milk,” which was partially drawn from dry milk from the Plainview plant, but further processed, raising questions about whether it was processed enough to eliminate possible contamination.26

Indeed, as control measures were implemented, thorny questions came to the fore at the Plainview plant. Around 900,000 pounds of dry milk powder was being held for possible contamination. In July, the plant, the MN Department of Agriculture, and the FDA negotiated the best course of action. MN and FDA officials met with an attorney for the Plainview plant to discuss what to do. The plant wanted to know whether the FDA would allow it to sell the product to an ice cream manufacturer in CA. The FDA said if they did allow it, they would have to know the CA manufacturer, have assurance of its processes, supervise the milk loading, and have a detailed plan from the Plainview plant about the whole process.

One FDA official emphasized that Plainview must ensure that the CA firm would “handle this product properly as they will have no control over it as it leaves their warehouse,” and that Plainview’s legal responsibility would not end even with the passage of product. While the Plainview attorney did not divulge the CA firm’s name in the meeting, he emphasized that this was a national ice cream distributor with a “good reputation,” and the Plainview plant had gotten an offer from an ice cream plant in MN that it was not considering because of reputational issues. The unsaid implication here was that dealing with the MN ice cream plant would be within the state, not interstate commerce, calling the FDA’s jurisdiction into question. In response, FDA emphasized how many parties were involved with the Plainview issue and the

26 National Archives. RG 88. Box 3839. May 12, 1966 Memorandum of Telephone Conversation between Safeway Stores and Kenneth Lennington.
question of jurisdiction was complex. This aspect of the negotiation demonstrates how tightly federal political authority is tied to the regulatory category of “interstate commerce.”

The FDA mentioned that the plant also had the option of reconstituting and repasteurizing the product itself, if it took proper assurances to “safeguard the public,” allowing MN Department of Agriculture’s supervision and sampling of product during processing, and conducting continued monitoring of the plant environment with “no additional positive Salmonella samples.” The PHS had told one of the FDA officials that other recommended safeguards would be a “complete plant cleanup,” conversion of the material into a high heat powder for use in bakeries and candy making, and separation of reconditioned processing from normal processing. The plant stated that they could not handle reconditioning the whole 900,000 pound lot for financial reasons, but could likely reprocess a portion, if they purchased an additional pasteurizer.

Between August and September, 3 more confirmed S. New-brunswick cases with exposure to dried milk were identified in different states, two infants and one adult male. While Salmonella positives were still being identified through state investigations of various plants, post-cleanup samples were showing encouraging results, suggesting that clean-ups were “very successful.” An epidemiologic curve constructed for a 1968 publication examining the outbreak showed that September was in fact the peak time for cases, with no cases of S. New-brunswick in October.

However, the countrywide investigation of INDM had uncovered another Salmonella issue. Since August, the FDA had been investigating a plant in Dixon, IL, which supplied the
Borden Company, who manufactured an INDM product with the brand name Starlac. In October, FDA identified *Salmonella* contamination in Starlac. The FDA’s analyses identified 9 lots of INDM that tested positive for two serotypes, *S. binza* and *S. worthington*. The FDA asked the Borden Company to voluntarily recall its product from the marketplace, and the company complied.

An FDA official, Kenneth Lennington, sent out a public advisory, that all lots of the powdered milk brand Starlac manufactured by the Borden company should be considered as “potentially contaminated.” One unique dilemma the issue created for the state of Alaska was that “isolated Alaska villages and field hospitals” had a full year’s supply of the product on hand. An Alaskan public health official asked the CDC for advice, and the CDC informed Alaska that its surveillance indicated “very few” cases associated with this product, but that since contamination of the product “may escape laboratory detection,” all lots should be considered “suspect.”

National media coverage of the Starlac recall painted a slightly different picture. A November 2 New York Times article publicizing the recall stated that “no illness has been attributed to Starlac,” in contrast to the “very few” cases the CDC had described to the Alaskan official. Public health surveillance identified a few human cases of *S. binza* and *S. worthington*, but these cases were not reported in the media as associated with the Starlac contamination. Coverage of the Starlac recall did not mention the Plainsview issue. All of Borden’s Starlac’s stocks were being recalled, which had a huge economic impact on the company. Sales of Starlac averaged $4 million dollars annually, representing 1 percent of Borden’s revenue.

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27 http://dash.harvard.edu/bitstream/handle/1/8963880/Curatolo05.html?sequence=2.
The *New York Times* also quoted Lennington as targeting the health warning messages to a particular consumer, the housewife. The housewife was “urged” not to use any Starlac she had on hand. Non-fat dried milk was not only used as infant formula, but also as a general ingredient in cooking; as Kendra Smith-Howard (2013) has documented, by 1951, Americans bought 60 million pounds of the product, which was advertised as an emergency staple to stock in case of a nuclear attack. Lennington’s targeting reflected the targeted way Borden had previously advertised Starlac to women; “Modern homemakers drink Starlac and use it in all their made-with-milk recipes.” 28 The visual imagery Borden used in its advertisements conveyed the message that food was love, and by using Starlac, women were providing love to their families, a common advertising tactic used for food at the time. 29

Other media articles carried messages highly targeted to women. For example, one article carried the headline, “Recall Starlac Milk as Health Hazard” and quoted Lennington as worried that the product may be in “hundreds of thousands of kitchens.” Two days later the *New York Times* ran another story about the Starlac issue, as well as the risk of *Salmonella* contamination from any product “of animal origin,” with the headline, “Housewives Warned About Salmonella.” The article contained quotes from an FDA bacteriologist giving advice to housewives that they should cook meat thoroughly to kill bacteria and not to slice both raw and cooked foods on the same cutting board.

The brand “Starlac” did not survive the bad publicity. Tina Curatolo argues that the bad publicity was “more effective than any seizure or recall action alone to get the products out of the hands of consumers at to kill the Starlac brand name.” 30 She also contends that the

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28 http://dash.harvard.edu/bitstream/handle/1/8963880/Curatolo05.html?sequence=2.
30 http://dash.harvard.edu/bitstream/handle/1/8963880/Curatolo05.html?sequence=2.
salmonella contamination issue had lasting negative impacts on the entire powdered milk industry. A 1968 *JAMA* manuscript examining the outbreak stated that the ultimate source of the INDM outbreak was unknown, though studies of the Minnesota milkshed were still underway.31

**Salmonella as a Household Name and Nationally Important Problem, 1966-1968**

The year that the INDM crisis played out, 1966, was the year that the FDA said that “the American public first became aware of the Salmonella problem,” in a planning document.32 Dry milk was only one of many products the FDA was examining through an extensive *Salmonella* testing program, and most contaminated foods were “voluntarily recalled by the processors in the interest of consumer safety.” Beyond dry milk, some of the other products included egg products, inactive dry yeast, coconut, gelatin, cocoa, carmine red dye, frozen pies, and smoked fish. The “widespread publicity” of those recalls, like the Starlac one, not only created greater awareness amongst the consuming public, but awareness amongst the “entire food processing industry,” who considered the issue not only a “potential danger to consumers,” but also a “serious threat to the financial well being of any company which inadvertently marketed a product contaminated with the organism.”

This awareness, the FDA argued, had translated into industry’s “earnest effort” to control the *Salmonella* problem. Industry made increased investments in processing infrastructure, sanitation programs, and internal laboratory surveillance of ingredients and finished products. However, while industry improvements had been made, what more needed to be done?

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This planning document cited the recommendations of a 1969 National Research Council (NRC) Report ("An Evaluation of the Salmonella Problem"), which the FDA and USDA had jointly requested that the National Academy of Sciences produce to advise them on the strategies they should take to help control “the Salmonella Problem.” The FDA cited one of the key questions that the NRC had posed as a policy issue. Because certain processed foods posed a “potential hazard to the consumer” due to risk of contamination during manufacture, how would manufacturers and regulatory agencies assess this potential hazard? This was a challenge, the agency argued, because “how does one prove the absence of an organism”? Even if a sample of ingredient or product was negative, that did not mean that there was no contamination of the lot of product in question. The agency cited its past experience that many samples had come back negative, with a single positive, and that the more material that was examined, the more likely it was that Salmonella would be found. Ensuring there was no Salmonella in the lot would mean testing the entire lot, an “impractical” tactic. However, this meant that the agency would have to “settle for less than 100% certainty,” raising the question, “when should we [processor and regulatory agency] quit testing?”

Thus, to help answer the question, the agency decided to adopt and implement NRC recommendations to consider a food as having a higher likelihood of Salmonella hazard if it had been previously identified as a “significant potential source,” that the manufacturing process did not include a control step for destroying the bacteria, and that there was great chance the microbial growth would ensue if the product was mishandled or abused during distribution or use by the consumer. In addition, due to their “high degree of susceptibility,” “infants, the aged, and the infirm” would be particular considered as a “special hazard category.”

Combining all of these characteristics, the FDA adopted the NRC scheme to categorize foods in five bins, from highest risk (use by a susceptible population and with *Salmonella* hazard) to lowest risk (products with none of the aforementioned “hazard characteristics.”) The plan created testing thresholds for each category. For example, Category I foods (highest risk) would require 60 25-gram samples tested with no positives, versus Category III-V requiring 15 negative samples. Underlying these thresholds were calculations of 95 percent confidence levels about *Salmonella* non-presence per varying amounts of product. Through this testing regime, *Salmonella* was enacted as a fundamentally probabilistic entity, a conception for the entity that contrasted greatly with public awareness of *Salmonella* as a stable health hazard related to food.

As another example of *Salmonella* becoming more of a household name around this time, unfolding in the political realm, was that on June 26, 1967, Senator Warren Magnuson (D-WA) introduced bill S. 2019, which proposed to redesignate “salmonella” as “sanella,” and the disease “salmonellosis” as “sanellosis.” Magnuson, a Congressman from a salmon-industry state, gave a lengthy rationale for introducing the bill. He argued that he had been advised that the “wide publicity” given to the bacteria was having an adverse effect on salmon sales in the country, even though *Salmonella* was not related to salmon. He argued that the “constant use of the term” in the papers and other media was detrimental to the industry, because consumers were making an “erroneous association” between *Salmonella* and the “wholesome food product” of salmon. He defended the proposed redesignation as one that had undergone “considerable thought,” with the following benefits. Sanella would not be confused with the “fine foodfish” salmon, it was similar to salmonella, and it sounded like the word “sanitation.”

Magnuson claimed that his staff contacted the agencies as well as the fishing industry about his proposal, and that there was consensus around its adoption, or, at least amongst the
latter, that a term was needed to avoid the “present unfavorable association with a specie of wholesome fish.” Earle Borman, Director of the Connecticut State Department of Health, wrote to Senator Lister Hill (AL), the chairman of the Senate Committee on Labor and Public Welfare, to criticize the bill as “unacceptable to bacteriologists,” as it would conflict with the international system for classifying bacteria and thus be “entirely unenforceable.” US bacteriologists, Borman argued, would be seen as “scofflaws,” Congress would be seen as “incompetent,” and, invoking Cold War rhetoric, he called this kind of congressional action “Lysenkoism” for its “governmental dictation of scientific thought.” In March of 1968, the Washington Post reported, in an article headlined ‘Salmonella’ Gives Fish Bad Name, that the Alaska House Resources Committee also introduced a congressional resolution not to use the terms “salmonella” or “salmonellosis.”

Neither the bill nor the resolution appeared to gain traction, but both are striking examples of the greater public and political awareness surrounding Salmonella as a public health hazard around and after 1966. Additionally, this evinced that both Congress and industry considered Salmonella not just a public health problem to control, but a public relations problem with vast economic implications.

**Debating Salmonella Control: From Feed to Consumer Education, 1958-1974**

Much of this chapter has focused on surveillance, outbreak detection and investigation, and the greater public awareness of Salmonella that resulted from publicity on contaminated foods presenting public health hazards. I conclude this chapter with an examination of two debates about Salmonella control, and how they shifted between the 1950s-1970s. While earlier in this period, contaminated animal feed seemed an important control point, over this two-decade

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period, views on feed control shifted and were de-emphasized, as consumer education rose in emphasis and prominence.

Contaminated animal feed made a brief appearance in the INDM contamination issue I described earlier, as that was one facet of the Plainview investigation, when investigators became concerned that contaminated INDM was being used in animal feed and distributed widely to farms. Contamination of animal feed had been an active concern of public health and regulatory officials since the 1950s. In 1958, Edwards had voiced concerns about fish meal used as a supplement in animal feeds, because this substance was, as Edwards stated, “known to be a fertile source of salmonellae,” based on his and others’ studies of Salmonella cultures coming from animal feed. He argued that if “eradication” of Salmonella in herds and flocks was the ultimate goal, officials should “take into consideration the continuous seeding of the population through infected feedstuffs.”

Whether or not controlling Salmonella contaminated animal feed would ultimately help lead to eradication of Salmonella in animals was a hotly debated question during the 1964 National Conference on Salmonellosis. Some felt that salmonella-free feed was an important contributing source to the problem that could be attacked, while others expressed doubts about its feasibility, expense, and ultimate efficacy in controlling the entire salmonella problem. During the discussion on Salmonella control after a panel focused on the topic, an audience member posed a question about feeds to John Walker, a USDA official. The question began with the statement, “The domestic animal salmonella cycle must be broken in order to reasonable influence human disease.” The questioner found it “difficult to accept” that “salmonella-infected

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35 He was not the only one, there was a 1956 paper he cited from Germany, and he also cited Galton’s research involving isolating Salmonellas from fish meal.
feeding stuffs” were currently continually distributed to animals. “Why,” the questioner queried, “should not the first step be a requirement that noninfected feeds be clearly labeled as such?”

Walker replied that while this was a point the USDA had been actively considering, the agency divided the question into two separate parts. One was the treatment of feed to make it salmonella-free, and currently, Walker argued, the agency did not think there was a “practical, surefire way” of treating feed on a “large scale.” The second, which the USDA felt was more “attainable and practical,” was to prevent future contamination through increased sanitary guidelines for feed producers.

Charles Darby, of the Ralston Purina Company, added to Walker’s answer, emphasizing to the group that the feed industry produced 40 million tons of commercial feeds, and that farms likely produced an additional 40 million tons, making the “chances of contamination” “very great.” Irradiation, Darby added, was felt to be too costly. Kevin Shea of the US Atomic Energy Commission put the cost of irradiation at approximately ½ - 1½ cents per pound.

CF Niven, of the American Meat Institute Foundation, went “out on a limb” to posit that even if animals were all fed sterile feed, he felt that the salmonella problem would continue, even if it was lessened. “I think we are going to have to contend with salmonellae for a long time but yet learn to live with them,” Niven observed. Steele’s view contrasted strongly with Niven’s, as he argued that all must “work to eliminate the treat of these ubiquitous agents.” He urged attendees to consider that in the future, the human and animal population would continue to grow, and Salmonella contamination would be “compounded if it is not tackled today.” “Today,” he pressed, “is almost too late to begin.”
Putting Walker’s view into practice, in 1967, the FDA and USDA initiated a joint program, along with states, to “reduce [the] risk” that animal by-products used in animal feeds ultimately posed to humans, given that “red meat and poultry” were recognized by the agencies as the “most prevalent cause” of human illness. The two regulatory agencies created a joint program, because the USDA was in charge of a voluntary program which almost 800 rendering plants joined, so that they could be registered on a USDA-approved list that they were producing a “Salmonella-free product,” while the FDA agreed to inspect the 125 plants who did not volunteer to be in the program.

However, over the next few years, views on the control of the Salmonella problem began to shift. The 1969 NRC Report played a strong role in defining the Salmonella problem and solution space. The NRC Report shared the established view of the Salmonella problem as one of a chain or cycle of infection, and created a holistic model of all the possible routes of salmonellae transmission, depicted in the following diagram.

![Figure 18. Possible Routes of Salmonella Transmission](image)

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37 NRC, p. 3.
The NRC Report also shifted the solution space, in developing a consensus view on what should be done about the problem. “Control of salmonellosis,” the report argued, “is not simply a matter of interrupting a cycle in nature.” While creating salmonella-free feed could reduce animal infections, the NRC believed that it would not result in elimination of the disease. There was “no simple or easy solution” to the problem. The strategy of control, the NRC argued, was to “attack at many points along a broad front” to achieve a “substantial reduction in incidence.” The NRC “reluctantly” was “forced to recognize the infeasibility of eradicating salmonellosis at this time.”

One likely outgrowth of the NRC recommendations was that in 1972, the USDA decided to terminate its involvement in the Salmonella-free feed program. According to an FDA report summarizing the USDA’s rationale, the USDA felt that cleaning up the rendering plants would cost several hundred million dollars, and would not significantly reduce the amount of human illness caused by *Salmonella*, because there were so many other non-feed environmental sources.  They decided that efforts and dollars would be better placed in improving slaughter sanitation; consumer and food handler education, and more research on improved processing methods. The NRC had recommended these measures, arguing that controlling contamination in meat and poultry products, and reducing food mishandling, were two of the arenas where control efforts would “do the most good” (3). Indeed, the NRC pointed out, there had been a “misplacing of emphasis,” focused on processed foods regulated by the FDA, which while potentially dangerous, were involved in “relatively few” disease outbreaks.

The USDA’s decision created a quandary for the FDA. The FDA debated whether to continue pursuing the animal feed program, and gathered an internal task force together to

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conduct a cost/benefit analysis to help the agency decide on a course of action. The agency was clear that they did not have the inspectional resources to surveill all 900 rendering plants, and felt it “dubious” to keep examining only 125. The FDA decided on a strategy to encourage the states and the rendering industry to keep pursuing a voluntary program, and to hone in on inspecting 40 facilities that “blended” products, who processed ~60% of animal by-products for animal feeds. The FDA also decided to maintain all of its other various Salmonella control programs, as well as increasing their efforts to improve the Food Code (model regulations for retail and food service), and educate food handlers and end consumers on “careless handling and preparation of food” in restaurants and home kitchens.

A major, contentious debate about Salmonella and consumer responsibility took place in the courts in the 1970s, centered on government inspection and labeling of poultry products. In 1971, the APHA wrote a letter to USDA Secretary Earl Butz, asking him to require a label on raw meat and poultry for human consumption, warning consumers:

Caution: Improper handling and inadequate cooking of this product may be hazardous to your health. Despite careful government inspection, some disease-producing organisms may be present. Consult your local health department for information on the safe handling and preparation of this product.

The APHA was concerned that the existing labels on raw meat and poultry indicating that it was “inspected and passed” by the USDA was a “false and misleading” claim in violation of the PPIA and Federal Meat Inspection Act (FMIA). Consumers, the APHA worried, thought that government-inspected raw meat and poultry were safe, even though they potentially contained pathogens such as Salmonella. In 1974, the dispute escalated to the US Court of Appeals.39

Judge Robb, author of the judgment, highlighted several aspects of the USDA’s letter in response to the APHA’s letter. The USDA quoted the NRC statement that controlling salmonella was complicated due to its widespread distribution and multiple ways to reach the human host. Additionally, the USDA cited the fact that many processed foods like egg products and dried milk were implicated in salmonellosis outbreaks. Since many products and sources were involved in the problem, the USDA argued that it would be “unjustified to single out the meat industry.” The agency used the fact that egg products and dried milk had caused major *Salmonella* outbreaks to argue that meat was not the only pathogenic health hazard to consumers. Robb also noted that salmonella “may be inherent in the meat,” and did not legally constitute an adulterant.40

Additionally, the housewife reappeared in Robb’s judgment, as she had in the 1950s PPIA hearings, and the Starlac publicity. Robb re-interpreted the USDA’s point that the American consumer was aware that meat and poultry could cause illness if handled improperly as saying that “American housewives and cooks normally are not ignorant or stupid and their methods of preparing and cooking of food do not ordinarily result in salmonellosis.”

Robb’s framing of the housewife as smart enough to properly handle food contrasted significantly with previous framings of the gendered figure. During the PPIA hearings, representatives from women’s groups had argued that while the housewife was “canny,” in an industrialized food system, she could no longer discern the quality of her food with her own

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40 Nestle argues that there was ambiguity on the meaning of the ruling, because there were dissenting judges who argued that more information would be helpful on the “understanding of consumers” in interpreting the “inspected and passed” label, and that the presiding judge would consider an additional challenge if consumer education programs did not work. She contends that the USDA chose to interpret the ruling in the following way; “because Salmonella and other pathogens are inherent properties of raw meat, the law prohibits the department from doing anything to control them.” This interpretation, she points out, provided the meat industry with a line of defense in a 2001 court proceeding, but interestingly, that there was ambiguity also left leeway for the USDA in 1994 to announce that it considered *E.coli* O157:H7 an adulterant in raw ground beef.
eyes. Indeed, in the 1960s, the FDA had also taken up this rhetorical frame in characterizing their own duty to protect the consumer. As FDA Commissioner George Larrick said in his remarks to the National Canners Association in 1962: 41

> As our society becomes more complex, the evolution of technology requires more safeguards for the consumer. As much as we might admire the rugged individualist, when you have 90,000 firms dealing in over $82 billion worth of food each year, you can't have each going his own merry way. Processors who are hundreds of miles from the point at which their product will be consumed have to have standards of operation to live up to and somebody has to see that the processor does in fact live up to them. We believe that you and we together have to do the job the individual housewife would do if she were preparing a product in her own kitchen. And really the food plant is just an extension of the home kitchen. Since the housewife can't go several hundred miles or more to assure herself of the quality of raw products used, the sanitary conditions of the commercial kitchen, the methods of handling and preparing the food, and the additives that are employed in its preparation, we are supposed to do this job for her.

The housewife was a mutable consumer experts could ventriloquize because she was an idealized figure they could talk about and speak for. Thomas Stapleford (2004) calls the 20th century American political discourse “schizophrenic” in its framing of the housewife, because at times experts called her a “rational, domestic manager,” and at other times they called her a “confused woman desperately in need of expert guidance.” While in the 1950s-60s women’s groups and the FDA framed the housewife as unable to discern the quality of industrialized food herself, requiring the assistance of industry and government, in 1974 Robb said that the housewife was smart enough to know that raw meat and poultry had Salmonella and required care in cooking. Following changing views on animal feed and the figure of the housewife through this two-decade period helps document a shifting paradigm of Salmonella control. This paradigm moved from viewing eradication as a contested yet possible goal to an infeasible objective, and adopted an increased policy focus on risk reduction and consumer education.

41 Larrick, George P. “FDA and Consumer Protection, the.” *Food Drug Cosm. LJ* 17 (1962): 266.
Conclusion

This chapter examined the building of the NSSP as a second-order system and its role in helping to support the investigation of major contamination issues in shell eggs and powdered milk. To help control these food contamination problems, officials had to issue warnings to the public. While public warnings were important for controlling both problems, they also raised public and political awareness of *Salmonella*, helping to make the pathogen a household name. As I documented, *Salmonella* was seen not only as a public health problem, but a public relations problem with vast economic implications.

Public health surveillance helped facilitate growing awareness and knowledge about the *Salmonella* problem, but did not drive consensus toward control measures. Indeed, *Salmonella* control issues were hotly debated through the 1950s-1960s. The 1969 NRC Report forged expert consensus around the Salmonella problem, defining its problem and solution space. While earlier in this two-decade period, eradication was a contested yet possible goal, by the end of the 1960s, it was agreed to be an infeasible objective. Afterwards, spurred on the APHA v. Butz judgment, both regulatory agencies adopted an increased policy focus on risk reduction and consumer education.
Throughout the 1970s, the NSSP remained a fairly stable entity. All of its federal and state members continued to mail the standardized form with carbon copies to the CDC on a weekly basis, to report all laboratory isolations of Salmonella. In addition to the states, some cities, and the FDA, state animal diagnostic labs and the USDA reference laboratories also joined the program. Overall, NSSP helped to demonstrate that individual cases of salmonellosis could usually be associated with additional and related cases.¹

However, while the NSSP improved understanding of the aggregate picture of salmonellosis, gaps in the picture remained. The 1969 NRC Report pointed out that there remained a “natural reluctance to report ‘minor’ diseases,” and “significant underreporting” remained in Salmonella surveillance.² The program was not “totally inclusive,” for several reasons.³ Existing laboratory facilities were too limited, the quality of laboratory work needed to be improved, and more local laboratories were necessary in cities and hospitals to improve routine culturing for Salmonella and send on more cultures to state labs.

Outside of a still fragmented total picture of salmonellosis, the program had another major problem, according to CDC official Eugene Gangarosa. In an essay examining the question, “What have we learned from 15 years of Salmonella surveillance?” Gangarosa opened the document recounting the high expectations officials had held when the program started.⁴ The hope was that by adopting the surveillance approach the agency had taken to control malaria and

¹ NRC, 163.
² NRC, 164.
³ NRC, 165.
poliomyelitis, they would also be able to control salmonellosis. By the “one measurement” of salmonellosis control, Gangarosa said, “perhaps the most important,” the NSSP “has been a failure.”

In 1976, over 23,000 isolates were reported, a mean that had not changed significantly over the past 15 years. Officials estimated that only 1 percent of salmonellosis cases were reported, totaling a total estimate of 2.5 million cases per year, with 500,000 hospitalizations and 9,000 deaths, at an estimated cost of 1.2 billion dollars annually. “We think these figures are conservative,” Gangarosa qualified. “The problem is persistent and serious.”

By leading with the notion that the program was a “failure,” Gangarosa emphasized the gravity of the Salmonella control problem. However, the rest of his essay detailed the many ways that the NSSP had aided the increased understanding of salmonellosis, knowledge about host-parasite relationships, identification of high-risk foods, and the detection and investigation of outbreaks. Indeed, he argued, in regards to the latter, the NSSP was particularly strong. Yet in that strength, paradoxically, it was weak. He wrote:

> Surveillance has accomplished a good deal to control epidemics, but it has done little to control the endemic problem from which epidemics emerge. Our surveillance effort has been a fire-fighting operation; we have dealt with each outbreak as if it were a separate problem. As fast as we could put out a fire in one place another would appear.

Instead of fire-fighting, Gangarosa argued, a better approach would be to attack the “root issue” and break the “chain of transmission”—“the vast reservoir in the animals man depends on for his food.” Gangarosa’s recommendation contrasted with the NRC view, that interrupting the Salmonella cycle was not sufficient to eliminate the problem. However, he did concur with another of the NRC’s recommendations, that a “closer working relationship” between federal,
state, and local agencies, and industry and academia would be needed to make any progress on control of salmonellosis.

This chapter examines the period between the 1970s-1990s, as the NSSP stabilized but shifted in utility. While public health officials would not build the early warning, real-time system of outbreak detection and response until the 1990s, the interim period between the NSSP and the early warning system was an active one. In particular, the fowl problem resurfaced, in the form of a *Salmonella* Enteriditis epidemic caused by shell eggs. In the 1990s, *E. coli* O157:H7 radically shifted the politics of consumer advocacy in the food safety arena.

**A Feedback Loop of Reverse Salients, Critical Problems, and Targets**

That Gangarosa rhetorically framed the NSSP as a “failure” because surveillance of the problem did not lead to control of the problem, its ultimate objective, is a point that can be usefully examined using Hughes’ large technical systems’ theory. As Hughes observed, a “crucial function” of the people building and operating large-scale systems is to “complete the feedback loop between system performance and system goal…to correct errors in system performance.” Using this point to re-frame Gangarosa’s argument, officials did not manage to enroll surveillance and control of salmonellosis into a feedback loop, where the surveillance system would perform the function of identifying problems and help achieve the goal of eliminating those problems, working on a circular basis to ultimately improve food safety.

Two concepts Thomas Hughes (1987) uses to explain how system-builders solve problems related to their system are reverse salients and critical problems. Reverse salients are lagging areas “holding up” the growth of a system. Hughes borrowed the term “reverse salient”
from military historians, who use it to describe how military front lines advance unevenly during battles, with some units moving ahead (salient), and some falling behind (reverse salient). He found the metaphor more useful than the conceptually related terms technological “bottleneck” or economic “sunk cost,” because it was more dynamic, and allowed for technological, social, and economic forces to play a role in the construction of reverse salients in large-scale systems. So applied to the NSSP, one reverse salient holding up its growth was that there weren’t enough local laboratories culturing *Salmonella* from patients to send on to state labs, contributing to under-reporting of salmonellosis. Hughes uses the concept of critical problems to understand the process of how system-builders turn reverse salients into puzzles they can solve. Turning the reverse salient of a lack of local laboratories culturing *Salmonella* into a set of critical problems would involve a process of coming up with solutions around how to support local laboratories, such as funneling federal funding into local efforts, or developing cheaper and easier methods of culturing, and so on. The reverse salient-critical problems pair is useful for helping to explain processes of invention, innovation, and the development of solutions.

An important point about reverse salient and critical problems Donald MacKenzie (1987) makes is that the direction of causality is not just from reverse salient to critical problems. The problems that are defined as critical are often the ones that a particular professional group is rewarded and equipped to solve. System builders must decide which critical problems are possible to solve at any given time, and different groups may hold different opinions on what is possible and where to best direct inventive effort.

Since surveillance systems like the NSSP are second-order, and have a layered quality and interlinked infrastructure that connects multiple sectors to produce a representation of problems in the food system, I find it useful to add a third concept to the reverse salient/critical
problems pair. I call this concept a “target” to capture how the central goal of a public health surveillance system is to ascertain the picture of a disease. This often involves divvying up aspects of the disease and how it is caused and transmitted, and translating those targets into critical problems. I use the term target to help explain how, while systems may have an overarching goal, to accomplish that goal, system-builders focus on a number of more specific ends in service of reaching their overarching goal.

To help explain what I mean by a target, I will use the “interstate outbreak” as an example. Prior to the establishment of the NSSP, public health officials knew that food was infected at a large scale, and that there were many cases of human salmonellosis a year caused by different serotypes of *Salmonella*. However, what relationships did these cases have to each other? Were there common source foods moving through interstate commerce? The building of the NSSP aimed to help answer these questions, turning the target of the interstate outbreak into a set of critical problems they could solve by building and operating a second-order surveillance system.

**Slow tempo as an NSSP Reverse Salient, 1970s**

Interviews with public health officials who worked on the NSSP in the 1970s revealed that the system had a slow tempo when it came to outbreak discernment. Paul Blake⁵ described his work in the NSSP on a practical level, when he started in the Enteric Diseases Branch in 1974. To briefly review the core workflow of the NSSP, it was a program focused on collecting five basic data elements, serotype, patient identifier (name, initials, or laboratory number), sex, age, and county of residence, from state public health labs. Each lab would submit this data

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⁵ Interview, Paul Blake, 3/26/13.
using standardized weekly isolation reports on carbon paper via mail to the CDC, so that the coordinating center could perform data entry and analysis of the epidemiology of salmonellosis.

If you were in charge of the salmonella surveillance program, all those things [isolation reports] would come first to you, and then your inbox would be a stack of pink carbons and if you were not on an outbreak, and weren’t totally snowed with other things, scan through them and see if there was anything that looked funny, like maybe a whole bunch of *Salmonella poonas*, or as you are looking through, I think I saw *poona* over here in this other place. But that was the extent, and so sometimes those things would stack up and nobody would look at them.

Surveillance officers were responsible for visually scanning the reports to see if there were clusters of a rare serotype, or anything else that looked suspicious, before sending the reports off to a clerk for tallying and official disease reporting, which would typically happen at a much later date.

I asked Blake to clarify how outbreak detection worked at the time, and he emphasized that it happened largely outside of the NSSP through traditional means. He pointed out that the system was “slow,” and that at times, it was even “months later” that they would see that an outbreak occurred, so only “rarely” did the NSSP help outbreak discernment. “Usually,” he recalled, the CDC would find out about outbreaks because a concerned physician would call about something unusual, or a state lab would call the CDC and say something like, “normally we just get one or two *typhis* a year, now we’ve had six in the last three weeks.” The NSSP, Blake emphasized, was “nothing at all like PulseNet.”

Since the NSSP was very occasionally helpful for outbreak discernment, “eye-ball-o-metric” scans continued to be employed as long as the NSSP remained paper-based. Martin Blaser recalled once spotting an outbreak of *Salmonella* Mbandaka in the “pile of green sheets,” when, over a period of a few months, he saw 10 cases, while previously this serotype had only

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6 Interview, Nancy Bean, 10/12/2012.
7 At some point the carbons went from pink to green.
been isolated once in the previous decade. An epidemiologic investigation was launched in the states where there were cases, and through it, public health officials saw that cases shared the commonality of East Indian surnames, and shopped at Indian grocery stores. However, they could not identify a specific exposure in their investigation, though they hypothesized that it might have been a spice. “This was the power of the system,” Blaser recalled, “to pick up the needle in the haystack.” He commented that the “precision” of the system made it a “great tool,” e.g. the value of the rare serotype as a marker. However, Blaser also concurred with Blake that the surveillance officer eyeing the isolation reports was not a primary outbreak detection mechanism by any means, recalling, “they sent the reports to me in the blind hope that I would spot something.”

The NSSP’s slow tempo can be seen as a reverse salient for outbreak discernment and the creation of a fast but right system. However, public health officials did not turn this reverse salient into a set of critical problems to solve by changing the NSSP. As Hughes has pointed out, sometimes reverse salients cannot be corrected within the confines of an existing system, and it takes the development of a new system to turn the previously unsolvable reverse salient into a set of critical problems. In the case of the NSSP, as its data accumulated, system-builders instead found it useful for other purposes, such as surveillance of long-term trends and elucidating the ecological aspects of different *Salmonella* serotypes, identifying unique patterns such as geographic distribution, seasonality, and relationships between types and animal/food reservoirs. Eventually, the CDC published this data in “atlases” of *Salmonella* serotypes. The following figure from an atlas published around 1989 portrays the national distribution of *S. derby* by state between 1968-1986.\(^8\)

\(^8\) Martin SM, Hargrett-Bean, N, Tauxe, R (1989?). “An Atlas of Salmonella in the United States; Serotype-Specific Surveillance 1968-1986.” The atlas states that since 1968, surveillance data have been stored on magnetic tape,
Outside of the strict bounds of the NSSP, but relevant to increasing the tempo of outbreak investigation work in the organization, was a major shift the agency undertook to tie the bureaus of epidemiology and laboratories within the organization together more tightly. Blake described how previously, the two administrative components of the organization were very separate. Epidemiologists would have difficulty working with the Bureau of Laboratories during outbreak investigations because it could take months to get requested results back. CDC laboratories were reference laboratories, conducting an array of careful characterization work on communicable disease agents.

During the 1970s, the agency established a new laboratory set up specifically for epidemiologic investigation, housed within the Bureau of Epidemiology. Blake recounted how much easier it became to set outbreak-related “priorities” for that lab. That said, priority-setting involved learning how to coordinate. Early on, he recalled, epidemiologists would “get food and dump it on the lab and say, test for everything.” However, over time, epidemiologists and laboratory scientists learned to work together and align their expectations; Blake discussed how having an investigation lab was an “educational” experience for epidemiologists, who learned which helps explain why earlier data were not included. The 1989 atlas was produced with the help of an IBM 3083 mainframe computer, SAS software, and a COMPAQ Deskpro 386 microcomputer.
how to collect specimens in a manner that facilitated quality lab analysis, and better tailor their testing requests to the lab. The new Enteric Investigations lab helped bench and epidemiologic scientists achieve alignment in their temporal rhythms of outbreak investigation work.

**Common serotype as target, 1970s-1980s**

I. Kaye Wachsmuth also emphasized the importance of this new lab. She started her CDC career in the Bureau of Laboratories, but soon shifted to the Bureau of Epidemiology to work more specifically on typing technique development for outbreak investigations. I asked her to talk about why the CDC began to look for other techniques beyond serotyping. She began by talking about “common serotypes” of *Salmonella* as presenting barriers to outbreak discernment. Outbreaks caused by common serotypes were “driving forces” for typing technique development. “You really couldn’t define your outbreak well,” she recalled. “You would have members of the community which might have *Salmonella* infections and you couldn’t separate them from the outbreak.”

Wachsmuth described the work that she and her colleagues conducted to develop new typing techniques to attack the common serotype target as “almost circular.” She said:

> I think it was almost circular. The single source outbreak helped us define the technology that we needed, and then we were able to help define the outbreak. Over time you were able to standardize your technology and with enough controls to show this was really the outbreak. But I think that was key in the beginning. To get the proper and right number of controls.

Turning the target of the common serotype into a set of critical problems entailed finding better laboratory techniques for typing bacteria, that could help differentiate between types within the

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9 Interview, I. Kaye Wachsmuth, 9/19/2013.
common serotype category. That she talked about the relationship between outbreaks and the development of tools for investigating them as “circular” again underscores that bacteria were represented to meet interventionist goals. Wachsmuth pointed to a kind of feedback loop between techniques for typing bacteria, detecting outbreaks as events of disease occurrence, and using past outbreaks to help refine the techniques. Particularly useful past outbreaks were well-characterized “single source” outbreaks where the contaminated food was definitively known, and the identification of clear controls to help confirm not only who was clearly in the outbreak, but who was outside of it.

Mitchell Cohen also talked about the common serotype problem with *Salmonella*, going into more detail about the implications that this issue had for the interpretation of epidemic curve data.\(^9\) He used *S. Typhimurium* as a particular example of a common serotyping, complaining that “…if you had a *Typhimurium*, you were up the creek” in trying to differentiate between them. He continued:

…when you would look in a community, you would see that there would be all of these, there was a secular trend that would occur with summer peaks in most *Salmonella*, but there were all of these, if you really looked at the curve in some detail, there were all of these peaks and valleys along the way. I had a couple of outbreaks I investigated, including one in Connecticut that was associated with a *Salmonella* Heidelberg that was transferred from animals, some subtherapeutic use of antibiotics, to humans. And looking at that outbreak, we were able to do some antibiograms and show that these were drug-resistant *Salmonellas*, and that there were different patterns, but people would say, why do you think they’re not different *Salmonellas* coming from different sources? So it became really obvious that we had a problem.

Since the 1950s, antibiotics had been used in feeds to promote increased growth in animals, and public health officials had, over the years, become increasingly concerned with the evolution of antibiotic-resistant strains in animal herds and flocks. Here, Cohen details an experimental

\(^9\) Interview, Mitchell Cohen, 11/9/12.
process where he employed the typing technique of the antibiogram to see whether the *Salmonella* Heidelberg outbreak isolates were antibiotic-resistant. While this was the case, the antibiograms characterized the outbreak isolates as different patterns. Cohen found this problematic because the isolates were epidemiologically related. While the antibiogram was a useful technique in some ways, it did not ultimately solve the problem of drawing the right connections between outbreak-related isolates from a technical perspective.

To help solve this critical problem of determining microbial relatedness for epidemiologic purposes, the CDC sent Cohen to work in the laboratory of Stanley Falkow, a microbiologist at the University of Washington who developed some of the first DNA probes for infectious disease diagnostics, and is regarded as the founder of the field of molecular epidemiology. Cohen brought organisms from the CDC that had been associated with several outbreaks, in order to learn more about molecular biology tools that could apply to epidemiology. During his time in the Falkow lab, Cohen became more interested in antimicrobial resistance as a public health problem, as well as the typing technique of plasmid profile analysis.

While Cohen was working in the Falkow laboratory, at the CDC, Wachsmuth also became interested in applying DNA-based analysis in outbreak settings, including the examination of plasmid profiles. She pointed to a 1981 multistate outbreak of *Salmonella Muenchen* associated with marijuana as the first time that the CDC used DNA to differentiate within serotypes, using plasmid fingerprinting. Muenchen, she pointed out, was an extremely common serotype. While this outbreak was notable in being the first one where the lab applied DNA-based analysis, she recalled how “cumbersome” it was for the lab to go through each step,

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isolating and digesting chromosomal DNA, spinning out chromosomal clumps, separating plasmids, and conducting ultra-centrifugation. This technique, she remembered, “took a long time and a lot of effort.”

However, she pointed out that while this would be seen as “very weak data today…at that point in time, it was all we had, and it worked, and it certainly helped, it agreed with the epidemiology, it gave us a little extra insight.” She concluded her account of the Muenchen outbreak with the following observation: “Since it was so common, it was all over the United States, marijuana (laugh) and *Salmonella Muenchen*, so it was good to be able to separate it.”

While plasmid profile analysis was a helpful technique over a period of five to ten years with various outbreak investigations, it exhibited some limitations beyond its cumbersome steps; not every strain had a plasmid, and plasmids were turning out to be a rather unstable biomarker. Wachsmuth recalled that because of this period of evaluation, they decided not to “put all our eggs in this one basket of plasmid DNA.” Though the Enteric Investigations lab was able to get the plasmid DNA pure enough to use restriction enzymes to digest it, as Wachsmuth recalled, restriction enzymes were also “limited at the time too.” She also recalled experimenting with ribotyping, but commented on the “tremendous amount” of work involved with that at the time, including blotting a gel, staining it, and hybridizing it. Wachsmuth summed up the experimentation process as “a lot of research for years trying to develop better methods and better markers, better technology.” During this period of research, outbreaks became, to use Hughes’ term, the key experimental or “test” environment for typing techniques.

**The Fowl Problem Resurges, 1980-1990s**
On April 8, 1988, many national media publications, including the *Washington Post*, publicized a public warning from public health scientists to residents of the Northeastern US, alerting them that “raw or undercooked eggs” were responsible for causing a major increase of salmonellosis, due to the strain *Salmonella* Enteritidis (SE). The region had seen 65 SE outbreaks, 27 with eggs definitely identified as the source, causing over 2000 cases of illness and 11 deaths.\(^{13}\) Though the egg industry used shell disinfection procedures, somehow, Grade A shell eggs were still becoming contaminated.

A *New York Times* piece on the SE epidemic noted the many dishes that Americans used raw eggs in: homemade mayonnaise, ice cream, eggnog, hollandaise sauce, and eggs sunny side up, to name a few.\(^{14}\) Americans also, the article noted, “like to lick batter from the bowl when cakes or cookies are prepared.” The SE epidemic localized traditional American foods and everyday food practices in kitchens, intersecting with taken for granted eating habits and customs, such as enjoying raw cake and cookie dough batter when baking a treat. These cultural habits and customs unfortunately met industrial practices in the US egg industry, as surveillance of the SE epidemic continued, and debates over its control escalated onto the national political stage.

In July 1988, New York traced three of its outbreaks to the Northeast’s largest egg producer, DeCoster, who shipped 4.5 million eggs a week from its farms in Maryland and Maine. Echoing the *S. derby* epidemic of 1963-1964, one of the outbreaks had taken place in a Manhattan hospital, sickening over 500 and killing 11.\(^{15}\) New York public health and regulatory

\(^{15}\) Salmonella poisoning in food: hearing before the Subcommittee on Oversight and Investigations of the Committee on Energy and Commerce, House of Representatives, One Hundred First Congress, second session, July 20, 1990., p 10.
authorities, unable to secure federal help, used their state-level authority to ban DeCoster from shipping eggs to New York through an embargo.\textsuperscript{16} By October, increased levels of SE-illnesses had been detected outside of the Northeast, in the Pacific and Mountain states.\textsuperscript{17} The next month, the \textit{Wall Street Journal} reported bad news for the $3 billion industry, that per capita egg consumption had hit an all time low in 1988, due to consumer concern about \textit{Salmonella}, as well as cholesterol. A \textit{Washington Post} article quoted an extension scientist blaming improper inferences from the general public about egg safety. Though some had suffered illness and a few had died, the scientist argued, “they were either elderly or infirm, people whose immunity systems couldn’t handle it.” However, the mass public was deciding, to the detriment of the egg industry, “all of a sudden, it’s not safe to eat an egg.”

The following year, in July 1989, the \textit{New York Times} published two stories that kept the fowl problem in the national spotlight. One article described the results of a study from the \textit{Lancet} which explained why the shell disinfection procedures used by the egg industry had not adequately controlled SE contamination.\textsuperscript{18} The study presented evidence for the “Trojan-egg theory.” Before the SE epidemic, it had been widely believed that \textit{Salmonella} was spread because of external contamination of eggs by chicken feces covering the shell. However, the Trojan-egg theory posited that SE had colonized the reproductive organs of the laying hens, allowing eggs to be infected before shell formation, and contaminating the inner contents of the egg. If the inner part of the egg were contaminated with \textit{Salmonella}, treating the shell would not get rid of the bacteria, leading to consumer illness when eating the egg in raw or undercooked preparations.

\textsuperscript{16} Ibid.
The second story, with the headline “Chicken Industry Called Unsafe and Uncontrolled,” covered a clash between a coalition of farmers, workers, and consumer groups, and the chicken industry. The coalition criticized the industry’s centralization and “excessive speed” of processing lines, the latter of which they blamed for worker injury as well as increased levels of *Salmonella* contamination. The industry fired back by questioning the coalition’s evidence, and contending that critics were labor organizers trying to unionize factory workers.

The SE epidemic continued on into 1990. What control efforts had been attempted, and why had they failed to stem human illness? A *Wall Street Journal* article pointed a finger at a “regulatory melee” between the USDA and FDA. According to the Government Accountability Office (GAO), there were many specific issues impeding coordination of control efforts, but more broadly argued that it was the regulatory structure of “split and concurrent jurisdictions” at fault. At the start of the epidemic, both the USDA and FDA were “unsure about their roles,” and played a game of passing the buck. Because SE was associated with eggs, the Agricultural Marketing Service (AMS) was at first the responsible agency (as the inspector of egg handlers and egg pasteurizing plants), but then, as SE became reframed as an animal health issue, the Animal Plant Health Inspection Service (APHIS) adopted responsibility. However, while APHIS had the regulatory power to control communicable disease in animals, it had no authority to control communicable disease in humans. Thus, the USDA decided that it could not act unless SE made chickens sick. While the FDA had food safety authority and a human health protection mandate, at first the agency argued that it did not have the adequate personnel or resources to address SE on the farm, and could not implement an SE control program.20

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20 This issue also came up with the feed program, discussed in Chapter 4.
According to the WSJ, because the USDA did not take action, and because Representative John Dingell (D-MI), chairman of the House Energy and Commerce Committee, had gotten involved, putting Congressional attention on the issue, the FDA decided to initiate a voluntary SE testing program for laying flocks. In laying flocks where SE positive results were identified, the flocks would be destroyed to prevent the spread of disease. When the FDA decided to make the testing program mandatory, the USDA initiated its own (“less draconian”) mandatory testing program, “prodded” by House members of the Agriculture Committee, who were “unhappy with the department ceding any turf to the FDA.”

“The response of Federal agencies responsible for combating the problem,” Dingell critiqued, “has been neither timely nor well coordinated.” One example that Dingell pursued aggressively in his questioning of USDA official Thomas Holt related to an information-sharing problem involving one of the outbreaks in New York. Dingell pointed out that hearing testimony had previously established that the FDA was “frustrated” because the USDA had not shared data about egg shipments from a producer that had been identified with SE positive flocks, because the FDA wanted to initiate a recall of the eggs. The FDA ended up contacting the New York State Department of Health to get the data.21 “Is this good cooperation?” Dingell asked Holt rhetorically. Dingell and Holt debated the fast-but-right issue, whether during the length of time between when New York State first notified the USDA of the outbreak and when the USDA considered the information officially confirmed (33 days), the USDA should have been taking actions.

21 1990 hearing, p. 113.
Roger Feldman emphasized that Congressional attention significantly shifted the “status quo” for inter-agency coordination amongst agencies in the food safety system.²² To illustrate the significance of the shift, Feldman shared an example of how inter-agency coordination typically operated prior to the changes brought about by the SE emergency:

…granted there were a lot of things that were separate, but every once in awhile, they weren’t separate, and to be prepared for this, may take some administrative finagling, and nobody did it. I know that I worked with guys at FDA and USDA, and I saw their lab data… There were people studying powdered milk, and I got on the job and said, I get a report every year about the number of salmonellas in powdered milk, what the hell am I supposed to do with that? And the answer was, where else would you want it to go? But, what are we supposed to do? How do we handle this? And the truth was, I never got any further than that.

In this example, Feldman was drawing out the issue that information produced by different agencies could and should be combined to inform action. The regulatory agencies had information on contaminated powdered milk, and the CDC had information on human illnesses from powdered milk. While they shared this information to a degree, they did not coordinate action around it. The powdered milk example, Feldman stated, illustrated “a problem for which a solution was needed… we didn’t know what the solution was or how we were going to deal with it.” Interestingly, that powdered milk got special attention from both agencies is most likely an artifact of the powdered milk contamination issue I described in the previous chapter, but as Feldman’s statements suggest, once the fire was stopped, the inter-agency coordination of action eventually stopped as well. As a result of the SE egg epidemic, Congress gave all of the involved agencies a mandate to increase their coordination and prevent problems like this from happening in the future. But Congress did not determine how the agencies should coordinate. It would be up to the agencies to operationalize how inter-agency coordination would be achieved in practice.

From an SE control perspective, the main regulation in place was the USDA’s 1990 rule requiring that when eggs were detected to be the cause of human illness, eggs must be traced back to source flocks, and SE positive flocks must have their eggs diverted for pasteurized or cooked use.23 Flocks did not have to be eliminated, but their housing was required to be cleaned and disinfected. SE regulation took a blow in 1995 when Congress discontinued funding for USDA enforcement of the rule, and subsequently, responsibility transferred to the FDA, continuing the regulatory melee. By 1997, public health surveillance documented that, from a national perspective, control measures were failing to reduce human illness from SE as well as the amount of SE in flocks or liquid egg products.

However, even though the SE control efforts were not successful at reducing human illness, the intense and high-stakes negotiation around SE control in the 1990s helped to cohere a shared policy frame across agencies for broadly delimiting the critical problems of foodborne disease—an approach of “risk-reduction” that would seek to create “barriers to the introduction and multiplication of the pathogen throughout the farm-to-table continuum.”24

This policy frame was not an entirely new product of the inter-agency debates around SE. It had derived from the Hazard Analysis and Critical Control Point System (HACCP), a control program originally developed in the 1960s as a collaboration between NASA, the Army Quartermaster Natick Labs, and the Pillsbury Company. In the intervening years, HACCP had seen sporadic uptake in microbiological standards setting-bodies and specific industries, prior to its elevation with SE (Ross-Nazzal 2009). In its original manifestation, HACCP was a modification of NASA’s mode-of-failure model, to apply to the management of food hazards, especially microbiological ones. While not newly created by the SE debate in the 1990s, SE was

24 Ibid.
the start of a push for HACCP to be more widely adopted as shared US food safety policy frame, with a concept of risk reduction across the farm-to-table continuum as the holistic vision of the entire system. It would gain significant momentum with a soon-to-follow *E.coli* O157:H7 in ground beef outbreak.

The “risk reduction across the farm-to-table continuum” concept played a significant role in shaping the discourse around who held responsibility for addressing the problem of foodborne disease. It distributed responsibility to everyone in the continuum; industry needed to reduce risk in processing, government needed to ensure that industry did so through regulations and guidances, government also needed to help educate consumers, and consumers needed to learn how to handle and cook their food properly and be careful in the kitchen.


When Tauxe first came to the CDC as an Epidemic Intelligence Service (EIS) officer in 1983, *E.coli* O157:H7 had been “discovered” just the previous year, in two outbreak investigations of hamburger restaurant outbreaks in Oregon and Michigan led by epidemiologist Lee Riley. The pathogen became a new target for public health officials; how the microorganism had evolved and exactly where it was located in the food supply or in the environment was unknown. Both of the O157 outbreaks had initially been discerned by clinicians, who noticed that more than one of their patients were experiencing the unusual symptoms of severe stomach cramps and bloody diarrhea, or public health officials quoted vividly from one case’s

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25 Interview, Robert Tauxe, 4/12/12.
interview, “all blood and no stool.” The severity of bloody diarrhea was particularly concerning to physicians and public health officials. These outbreaks initiated what Riley later recalled as an exceedingly “interesting” and “exciting” time, associated with the “process of going through with identifying a new pathogen for a new disease entity.” The pathogen became a target and public health officials began to turn its “identification” into a set of critical problems.

One critical problem was finding out just how rare this serotype was. Was 1982 its first emergence, or had it appeared before? At the CDC, laboratory scientist Joy Wells “orchestrated” a retrospective laboratory investigation. The goal of the investigation was to determine whether this serotype had been isolated previously, anywhere in the country, from both animal and human sources. To look for animal isolations, Wells checked the serotyping records of the USDA Animal Laboratories in Ames, IA, as well as the Pennsylvania State University Veterinary Research Laboratory. To look human isolations, she consulted the collections of the CDC Enteric Reference Laboratories. These collections contained over 3000 E. coli isolates starting in and since 1973. From this collection, E. coli O157:H7 had only been isolated once—from a single patient from California in 1975.

The reference laboratory had serotyped the isolate and had basic clinical information from the case. However, Riley needed to track down more information about the patient. The patient had been an employee of the Alameda Naval Air Station. The patient’s medical chart

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28 Lee Riley oral history, collected by UCLA, 12/30/1997.
29 The “orchestrated” term comes from Riley’s oral history.
ended up being stored in a Kansas facility, and had to be located, and taken out of storage and eventually mailed to Riley.31 When getting the records, Riley found out that, interestingly, that patient had also experienced bloody diarrhea. Solving the critical problem of microbial identification involved tracking down, fleshing out, and drawing new significance from past evidence.

Hugh Pennington (2003) points out that these two *E. coli* O157:H7 outbreaks had little public and policy effect. During and immediately after the events, neither investigation had attracted national press attention. However, after the events, in the fall of 1982, the investigations were presented at a scientific meeting. Public health officials had developed a confidentiality policy to help them share scientific information about outbreaks, by generifying the names of the commercial “sources” of contamination after the emergencies had faded. Somehow, the press found out the “Chain A” source was McDonalds. The day after the story broke, the corporation’s stock fell 1.5 points. However, as Pennington argues, the publicity had short-lived effects, as the stock price soon rebounded and consumers continued to eat hamburgers with “confidence,” which he attributes in part to the fact that none of the cases had died, or gotten severely ill.

Identifying *E. coli* O157:H7 continued to be an object of intense public health interest after the Oregon and Michigan outbreaks. Tauxe remembered a 1986 investigation of two individual sporadic cases of hemolytic uremic syndrome in babies as a particularly important step in establishing that cattle were a reservoir of the pathogen. Because the babies only consumed milk, and the milk was raw, it was easy to identify the food that had caused them to develop HUS. Initiating an investigation on individual cases, an unusual but not unheard of

measure, was likely due to the combination of the young ages of the cases, the severity of HUS, and increased public health interest in the issue. Furthermore, because the farm exposures were clear, animals from the two specific farms could be examined closely for *E. coli* O157:H7. Tauxe characterized the importance of this study as one that documented that cattle were an O157 reservoir; “Cattle gets turned into ground beef, so that all made sense.”

Tauxe shared that public health officials spent much of the time during the 1980s “feeling like Cassandra,” drawing from the Greek mythology tale as an explanatory resource. Cassandra, the daughter of King Priam of Troy, angered the gods, and in response, was cursed, Tauxe related. She was given the ability to see the future accurately, but was stymied by the fact that no one would believe her. Her predictions about the Trojan war, though telling, went unheeded. “We felt a little like that,” he said, continuing:

There’s some bad stuff out there, there’s some game-changing stuff out there. This *E. coli* O157, it kills kids. It’s not just bad diarrhea, or dehydration, it’s going to kill kids, with renal failure. And it’s in our cattle. And no one’s doing anything about it.

However, a major outbreak had not occurred yet. Throughout the 1980s, CDC scientists kept up their intense scrutiny of O157. Another critical problem to solve during the 1986 farm investigation was to develop a cheaper and more rapid screening procedure to process the hundreds of animal samples. Culturing and serotyping every single sample would be too expensive and time-consuming, making the screening undoable.

Since the early 20th century, bacteriologists had been using differential media to distinguish different types of bacteria from each other, by providing growth conditions that encouraged some kinds of bacteria to grow and discouraged others. An English bacteriologist, Alfred MacConkey, developed the first solid “differential media” that could separate lactose-
fermenting organisms from non-lactose fermenting organisms. By including the sugar lactose in the media, lactose-fermenting organisms would do just that, producing acid in the process and reducing the pH of the agar. MacConkey agar also contained a neutral red pH indicator, so that when the pH level dropped, the bacteria would absorb the neutral red, making the growing bacterial colonies appear bright pink or red.

Wells’ laboratory investigation of the 1982 outbreaks involved experimenting with differential media that would select for O157. She and her colleagues noted that in 1952, researchers had used a sorbitol-base MacConkey agar to differentiate some E.coli strains, and Wells confirmed this finding in the O157 investigation. O157 exhibited an unusual behavior making it different from many other E.coli. Most E.coli fermented lactose. By using a MacConkey agar that replaced lactose with sorbitol, scientists could differentiate O157 from non-O157 E.coli. “For laboratories that cannot serotype E.coli,” Wells et al recommended, “screening for sorbitol fermentation is a valuable and simple tool.” As with Edwards’ development of the simple serotyping routine in the 1950s, the CDC continued its service mission to develop simple technologies that would work in the average laboratory.32

In 1985, two other CDC laboratory scientists created a “single tube screening medium” for simply differentiating O157, not only taking advantage of the sorbitol-fermentation, but also that O157 had a flagellar antigen H7.33 Using the “single tube screening medium,” a laboratory could then identify “presumptive positives” of O157. This would allow laboratories to screen for a smaller subset of positives to then run the more expensive and time-consuming confirmatory

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testing with exact serotype characterization. Creating simple, “presumptive positive” screening techniques solved both the technical problem of identifying O157, and the economic problems of cost-effectiveness and volume.

In 1986, two Canadian researchers conducted a field trial of sorbitol MacConkey agar (shortened to SMAC), to test its real-world efficacy as a screening tool for O157. They found SMAC to be efficacious, reliable, and especially helpful to the laboratory technician for visual reasons. The bright pink colonies SMAC produced enabled laboratory technicians in public health labs to easily choose good suspect colonies to isolate. Another benefit was that SMAC was available commercially. SMAC medium was simple, inexpensive, and fast, resonating with a sentiment Falkow had expressed during concluding remarks at a 1983 conference on “Rapid Detection and Identification of Infectious Agents.”

He said:

The choice of technology… has its roots in the realities of the clinical laboratory. Shelf-life of material and economics may dictate against the adoption of even the most elegant of reagents.

However, technical and economic possibility were not sufficient for getting clinical laboratories to use SMAC on a widespread basis. It took political momentum, from more outbreaks, to drive wider dissemination of SMAC, and wider awareness of O157 as a public health problem.

**Substantiating the Target, 1986-1992**

Between the mid-to-late 1980s, increasing clinical awareness was not simply an issue of disseminating a proven fact of disease causation to physicians, but conducting and connecting the necessary scientific work to fully substantiate that O157:H7 was one of the major causes of

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hemorrhagic colitis. Riley recalled that during this time, there was high “intensity of disbelief” with many “skeptics” in the wider research community. However, over time, collaborations between epidemiologic, clinical, and laboratory investigators together led to understandings of the mechanisms underlying O157:H7’s virulence as distinct from other pathogenic *E.coli*. He explained:

So there is an explanation, but you couldn’t get to that point without the epidemiology being done. The epidemiology helped pinpoint that this was an organism to work with, and now you have to understand what the mechanism is (117).

Riley’s point recalled Wachsmuth’s, referring to a feedback loop of between epidemiologic and laboratory work, in this instance, organized around the process of identifying the pathogen.

I asked Patricia Griffin to recount the process of proving O157:H7 was the main cause of HUS. She began with a story of when she first arrived at the CDC as an EIS officer, and being told within a week of starting that “we [public health officials] know what causes hemolytic uremic syndrome.” As a gastroenterologist trained at Brigham and Women’s Hospital in Boston, a premiere institution, she recalled thinking that she “should have heard of” *E.coli* O157:H7, if that were the case.

I asked her to expand on that issue. The fact that as a gastroenterologist such important information had not been on her radar screen got her “very interested in the diffusion of scientific information,” since it seemed to be a problem not only with O157, but also with

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37 Interview, Patricia Griffin, 9/20/2012.
38 When Griffin mentioned the term diffusion, it made me recall that I had noticed that one of the textbooks on the shelf of many of the CDC scientists I had interviewed is sociologist Everett Rogers’ *Diffusion of Innovations*, and represented an interesting intersection of our epistemic repertoires for understanding the relationship between technology and society. Rogers, Everett M. *Diffusion of innovations*. Simon and Schuster, 2010.
Helicobacter pylori, where the CDC felt that its role in causing ulcers was more established than a “skeptical” GI community. O157 and H. pylori, she said,

…really impressed on me that it can take awhile for information to diffuse, certainly into the level of clinical practice, and that when you are in a position like the one I was entering into at CDC, you can get early and very interesting information about what might be the sources of important health problems.

Griffin did, like Riley, reference the skepticism of the broader medical community around O157:H7’s causal role. While some literature had incorrectly attributed cases to varying bacteria and viruses because those were the agents they happened to isolate, Griffin recalled that there were significant debates around the question of whether O157:H7 was “the major predominant cause” of HUS, or “one of many causes.” She cited papers from Mohamed Karmali’s group in Toronto\(^39\) as the evidence that “should have convinced everyone that E. coli were an important cause of HUS.” However, she recalled that while she was collecting data for a national surveillance study of HUS in 1985-1986, she would frequently hear from the pediatric nephrologists in her study sentiments such as, “yeah, yeah, we’ve heard that line before, people think they have the cause but they never really figure out the cause.” Perhaps even more so than scientific publications, Griffin cited outbreaks themselves as drivers of information diffusion, not just to the lay public, but to clinicians. CDC officials typically would respond to media calls about any E. coli outbreak, or even any serious individual cases, because “we know that the press can be really effective at spreading information even to the medical community.”

In November 1986, the state of Washington experienced a multi-county O157 outbreak.\(^40\)

The outbreak was initially detected by a concerned physician who noticed three clustered cases

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of thrombotic thrombocytopenic purpura (TTP), a rare blood clotting disorder. The following year, Washington state made O157 a reportable disease in its state. A 1990 paper in the American Journal of Epidemiology used the Washington outbreak as an example of why better national surveillance of O157 was needed. “The true magnitude of the outbreak,” the paper speculated, “may have been quite large.” O157 was a high-risk, “rapidly emerging pathogen” in the US food supply. Without the clinician who happened to notice the three TTP cases, the paper argued, this outbreak would likely gone undetected. This suggested the alarming notion that there were other undetected outbreaks out there. Awareness of the pathogen in clinical settings was low; most clinical labs were performing routine stool cultures with MacConkey agar, not SMAC, which would leave O157 undetected.

Echoing the state of confusion for the salmonellas in the 1920s-1940s, a proliferating literature throughout the 1980s about the newly discovered E.coli was messy, especially as it related to the toxins the bacteria produced. Fully thirteen classes of E.coli had been proposed in the literature, creating an enormous state of confusion, especially for key users of these categories, clinicians and clinical microbiologists. As an illustration of this confusion, a 1991 review paper by Griffin and Tauxe offered some suggestions for terms. They suggested that the term “Shiga-like toxin-producing E.coli” should be used for the strains of E.coli that elaborated Shiga-like toxin, or verotoxin. While Shiga-like toxin and verotoxin were to be

equivalent terms, they expressed a preference for “Shiga-like” because it emphasized the close connection between verotoxins and shiga toxins. “Enterohemorrhagic” *E. coli* should be considered a “defined subset” of the broader category, defined as serotypes that cause a clinical illness similar to the one caused by O157:H7 and produce one or more Shiga-like toxins.

Calling attention to the pathogen’s importance not only in and of itself, but as heralding a broader issue, Griffin and Tauxe called O157:H7 the “prototype” not only for the new enterohemorrhagic class, but for “new and emerging bacterial pathogens in general.” Calling the pathogen a “prototype” emphasized how it deserved special attention as a public health target. Furthermore, as King (2004) discusses, in the 1990s, “emerging diseases” became a “coherent concept” anchoring a broad public health campaign to garner support for increased control over the risks presented by globalization and modernity.

As a target, *E. coli* O157:H7 needed mitigation from multiple fronts. On the clinical side, Griffin and Tauxe pointed out while SMAC had been developed in the mid-1980s, SMAC was not widely adopted by clinical laboratories. However, SMAC had limitations, too; it would only identify O157 on the first week of a patient’s illness, and would not screen for other serotypes of Shiga-like toxin-producing *E. coli*. To make Shiga-like toxin-producing more visible, new screening methods needed to be developed and adopted in clinical laboratories.

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45 The CDC lab had been using the term “Shiga-like,” personal communication.
46 Ewing and Lindberg (1984) pointed out that shigellae and escherichiae were known to be very similar organisms, especially in terms of their O-antigen groups, and in the 1970s, DNA-based research confirmed that they were “so intimately related” that “conceivably,” one species could include both shigellae and *E. coli*. However, Ewing and Lindberg called this unified classification “impractical,” because of the need to differentiate *Shigella* and *E. coli*, as well as different serotypes of *E. coli*, for both clinical and epidemiological purposes. The relatedness of the two kinds of organisms in nature and the need to differentiate the two epidemiologically required a practical management technique; before performing serotyping, it was important for technicians to figure out whether an unknown culture was *Shigella* or *Escherichia* beforehand, through various biochemical tests measuring phenotypic characteristics of the two organisms. *Serology of Shigella*. Academic Press, 1984.
On the public health front, there needed to be better surveillance of both outbreak-related and sporadic cases. More needed to be understood about the pathogen, the illnesses it caused, the populations it placed at risk, the geographic spread of the organisms, and the different food vehicles where it tended to make its home. “Critically needed,” they emphasized strongly, were more on-farm and in-slaughterhouse studies to learn how to reduce contamination in those spaces.

Regulators, they argued, should make sure policies ensured that “cooked hamburger patties and other meats be sufficiently precooked to kill pathogens.” The paper warned that both food service personnel and consumers needed to be aware that unless a hamburger were extremely “well-cooked,” it could still contain O157, and cause serious illness, especially in young and old populations-at-risk.

Griffin shared a memory of a personal sentiment about consumer awareness she held at the time, but could not include as data in a scientific paper:

…it was clear just from talking to friends and family that it couldn’t be fixed just by telling people to cook their ground beef or their hamburgers. My own family wouldn’t do that… Unless you know someone that’s gotten horribly horribly sick, you’ve been cooking hamburgers like that your whole life, why would you change? It was pretty clear to me personally, that telling people to cook their hamburgers better, or to use a meat thermometer, wouldn’t in any way fix this problem.

Griffin could not have used personal conversations with her friends and family as evidence for making policy recommendations in a publication, but in her oral history interview, added depth and caveat to the consumer cooking warning so commonly repeated throughout the 1980s - 1990s, pointing out its inadequacies.
Who could speak for the consumer, and push back against the responsibilization of a vulnerable entity? Griffin recounted that Alabama housewife Mary Heersink contacted her in 1992. Heersink called the CDC to ask Griffin if her son’s case of severe HUS might be associated with O157. Heersink had been conducting extensive searches of the medical literature in an attempt to gain more information about her son’s sudden severe illness. The housewife would go on to author a book about her son’s illness experience, and become one of the spokespersons of Safe Tables Our Priority (STOP), a grass-roots victim advocacy group that formed after the game-changing O157 outbreak of 1993. In her book, Heersink recounted her conversations with CDC scientists:

They told me they had been battling this demon for years, investigating outbreaks and sounding alarms. No one appeared to be listening. What was needed was a grass-roots effort of affected people world wide to demand changes in the way meat is produced and inspected. They told me nothing would change until that occurred.

While CDC scientists sounded alarms about an “emerging” pathogen in the medical literature, Heersink could frame O157 with the rhetorically potent name “demon” endangering children like her son for years without social action to prevent other illnesses from occurring. Heersink’s 1992 visit to the CDC was one of the events that helped catalyze her shift from housewife and mother to victim advocate during the 1990s.

Heersink’s status as a housewife connected her to a long history of gendered advocacy in the food safety arena. However, her position was distinct in that she was the mother of a young victim. Victim advocacy was of course not unprecedented in the history of consumer protection. In the 1960s, victims of the drug thalidomide, which caused over 10,000 serious birth defects

47 http://articles.latimes.com/2001/jun/06/food/fo-6863
worldwide, organized and helped press for public health protection reforms at the FDA.\textsuperscript{49} The 1990s saw a proliferation of advocacy groups that formed identities around disease categories, and made political claims based on those disease identities, in arenas such as AIDS and cancer (Epstein 1995). The arena of food safety expressed these dynamics of change as well. It was less Heersink’s housewife identity, and more her identity as the mother of a young victim, that helped form the basis of her claims-making in the 1990s food safety arena.

\textbf{The Game Changer, 1993-1994}

One of STOP’s co-founders, Kathi Allen, an aunt of an O157 victim, painted a harrowing picture of the Seattle Children’s Intensive Care Unit during January 1993, during the major outbreak public health officials had thought they might encounter. Allen wrote:

I entered the ICU waiting room, the scene was beyond comprehension. Families huddled on plastic mats on the floor. Everywhere you looked you saw the same expression—haunting, frightened faces as family members clung to each other and waited for word on their critically ill children. At times, there were as many as 60 people huddled on the floor or in the few coveted chairs. It looked like a war zone—and it was.

I watched as a woman collapsed when the doctor gathered with her family and said, ‘I’m sorry, we have to take your son’s colon out.’ Their son was two. Days later, they would be burying him. I heard the audible gasps as the helicopter whipped the air overhead. They all knew what it meant, and I would learn, another victim was arriving. I tried to look away as families cried into pay phones mounted on walls at each end of the room. Then, I made the long walk down halls where countless children lay battling for their lives...all because of a hamburger.\textsuperscript{50}

As I described in previous chapters, salmonellosis had sickened vulnerable populations in the past, such as hospital patients in the \textit{S. derby} epidemic, and young children in the INDM outbreak. However, HUS was truly life-threatening. Rhetorically, it had been possible to

\footnotesize{\textsuperscript{49} Timmermans (2000), Carpenter (2010).} \\
\footnotesize{\textsuperscript{50} http://www.stopfoodborneillness.org/sites/default/files/pdfs/STOP%2010%20Year%20Report%20Why%20Are%20People%20Still%20Dying%20From%20Contaminated%20Food.pdf.}
downplay the seriousness of salmonellosis as mere diarrhea, an argument not often directly expressed, but tacitly lurking in the background. As Allen’s account demonstrates, victim advocates would not allow HUS to be dismissed in such a manner.

Many in depth analyses of the Jack-in-the-Box outbreak have been published, from a wide variety of perspectives.\(^5\) Given that so many other accounts of the outbreak exist, here, I only gloss some major features of the crisis. My goal is to help set the stage for the next chapter, where I demonstrate how outbreaks like the Jack-in-the-Box crisis became a target for public health officials to build an early warning, real-time system to help prevent such events from occurring in the future.

In Washington state, O157 was a reportable disease. Clinical and public health professionals had surveillance infrastructure in place, as a result of the state-wide outbreak of 1986. On January 13, 1993, a Seattle physician noticed a temporal cluster of young HUS cases, as well as an increase in the number of ER visits for patients with bloody diarrhea.\(^5\) The outbreak investigation would in total identify over 500 cases from multiple western states, 45 with HUS, and 3 deaths (two of them children).\(^5\) The outbreak was announced to the general public on January 18. That was when the CDC was able to link its epidemiologic evidence (many cases reported eating at Jack-in-the-Box) with a suspected link to contaminated hamburger patties supplied to Jack-in-the-Box, and report the link to the USDA.\(^5\) Jack-in-the-Box voluntarily recalled 28,000 pounds of its contaminated hamburger patties.\(^5\)

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\(^5\) Besides multiple scientific reports of the outbreak, it has been examined in various genres of journalism (Benedict 2011; Trum-Hunter 2009; Schlosser 2001; Booth & Brown 2014); food studies (Nestle 2010); and risk communication (Powell et al 1997).


\(^5\) Ibid.

\(^5\) Ibid.
Public attention to the Jack-in-the-Box outbreak would reach a new intensity, not only because of the disease event itself, and the medical plight of individual cases, but also the outbreak’s legal ramifications. Victims sued Jack-in-the-Box, Jack-in-the-Box sued its suppliers, and its suppliers sued their suppliers.\footnote{http://articles.latimes.com/2001/jun/06/food/fo-6863} From a political perspective, this outbreak catapulted food safety to a high priority agenda item for the newly elected Clinton administration.

Marion Nestle (2010) argues that major policy consequences swiftly followed the Jack-in-the-Box outbreak, because it was “especially difficult for the public to accept.” Children had died, and hamburger was an “American food icon” (74). However, as she charts, the creation of reform measures involved heated political battles between government, industry, legal counsel, and consumers, particularly over the fraught question of responsibility. She contends that the “usual line of reasoning” that pathogens were ubiquitous and could be addressed by end consumers cooking meat properly was troubled, especially by the newly formed victim advocacy groups.

The battles occurred in the media, Congressional hearings, regulatory hearings, and in the courts. To gloss, the outcomes of the battles were four key policy reforms:

1) FDA raising the internal temperature recommendation for cooked hamburgers to 155 degrees Fahrenheit (matching an already higher WA standard)
2) Consumer-oriented safe food handling labels on raw meat and poultry (shifting USDA interpretation of APHA v. Butz)
3) USDA announcing that it would view \textit{E.coli} O157:H7 as an adulterant in raw ground beef under its already existing authority from the FMIA
4) USDA’s passage of the “Megareg,” or Pathogen Reduction Act/HACCP rule, in 1996, which in short required raw meat and poultry plants to monitor their production with “performance standards” for \textit{Salmonella} (using indicator organisms and statistical sampling plans, see Wallace et al), and a zero-tolerance policy for O157 in ground beef.\footnote{This is a complex issue, but legal counsel Bob Hibbert has a nice encapsulated description of what this did from a legal standpoint: Established food safety law, as enforced in this country by both FSIS and FDA, and in large measure around the world as well, can be boiled down to a single sentence: Products that are considered adulterated...}
Regulatory reform for food safety became a major priority for government officials, spurred on by public interest and victim advocacy groups, lawsuits, and the gaze of a national media limelight. However, as the crisis faded, public fear of hamburgers largely abated. For much of the consuming public, the outbreak had “no discernable effect on consumption” of hamburgers, with 5.2 billion sold in the US in 1994.\(^{58}\)

To the scientific community, the O157:H7 outbreak was cast as a focusing event to call attention to another issue—a “crumbling foundation” for infectious disease surveillance.\(^{59}\) A policy forum article in *Science* argued that the 1993 outbreak illustrated the value of active public health surveillance for protecting the public health, but surveillance itself was at risk. While making O157 nationally reportable would be ultimately help control the disease, the paper noted that state public health agencies would be “reluctant” to add new diseases to the list of reportable diseases. Due to lack of funds and personnel, even currently reportable diseases were “significantly underreported,” implying that newly added diseases would add more burden, without more resources for the infrastructure of surveillance.

That public health infrastructure was “crumbling” in the 1990s is an important piece of historical context for understanding the genesis of PulseNet and FoodNet, one that would come up often during oral history interviews. The *Science* article had also lauded the CDC’s implementation of the “new molecular technique” of PFGE in the Jack-in-the-Box outbreak, to confirm that the bacteria from the patients in the outbreak matched the contaminated ground beef supplied to the fast food chain. The article drew rhetorical force to marshal more resources for

\(^{58}\) Levenstein, 55.

the public health system, by contrasting the need for new technologies to be implemented in a moment when more basic public health infrastructure was in free fall.

“The point at which that outbreak hit,” Griffin recalled, “I felt like I was ready because I knew that this was an opportunity that was presenting itself to me to grab national attention to try to get change. And that there was a very short window of time when something like that happens, when you can use it to make change happen.” That Griffin saw the 1993 Jack-in-the-Box outbreak as a creating a short window of opportunity to catalyze attention and stimulate changes is consistent with Thomas Birkland (1997) has called focusing events, or sudden events, such as crises, disasters, or catastrophes, that cause increased attention to policy issues or problems related to and revealed by the event. The next chapter will show how the Jack-in-the-Box outbreak became the target of a new real-time, early warning system, that public health officials would build with the aims of finding major multi-state outbreaks sooner, stopping them before they grew larger, and helping prevent illnesses from occurring in the future.

Conclusion

This chapter examined the 1970s-1990s, a time period that saw a shift in the perceived utility of the NSSP. While the system was established in the 1960s for the twin goals of helping to detect interstate outbreaks and reveal more about the epidemiologic patterns of salmonellosis, the system ended up being much more useful for the latter goal than the former. As data accumulated, public health officials began to learn more about long-term trends in the ecology of Salmonella serotypes. Though officials saw slow tempo and incomplete reporting as reverse salients of the system, they did not change the NSSP significantly, nor build a system in its place,
during the 1970s-1980s. But this period saw public health officials experimenting with a variety of new typing techniques, choosing the common serotype as a target so that they could identify more outbreaks in the community. Outside of the surveillance system, but relevant to the broader CDC infrastructure was a significant institutional innovation—the development of the Enteric Investigations laboratory, to align the tempos of laboratory and epidemiological work for outbreak investigations.

In the 1980s, the fowl problem resurfaced. While officials had managed to construct a better total picture of salmonellosis from the NSSP, control of the problem was an entirely different matter. Towards the latter part of the decade, SE caused a major epidemic associated with Grade A shell eggs, one that continued for years, as the federal government failed to organize a coordinated effort to control the problem. This lack of coordination and action garnered major critique from Congress, which gave all of the involved agencies a new mandate to significantly improve their coordination and control foodborne disease. Almost on the heels of the SE epidemic, an emerging pathogen, *E.coli* O157:H7, began to cause life-threatening illnesses, especially in the vulnerable population of young children.

Identifying the pathogen entailed much laboratory and epidemiologic work, but the problems were not simply technical. They were economic, social, and political as well, entailing the development and dissemination of rapid and cheap screening techniques, a number of smaller outbreak investigations, and finally, the game-changing Jack-in-the-Box outbreak of 1993-1994. Because of the outbreak, grass-roots victim advocacy groups began to form, creating a new disease identity as the basis for making political claims about food safety reforms.
As the next chapter examining the building of the early warning, real-time system will show, in addition to technical possibility, economic, social, and political factors played an important role in this system-building story.
“[PulseNet is] our Hubble Space Telescope,” Tauxe explained to a science journalist in 2009. “When the Hubble opened up and started taking pictures of the deep sky, the tiny points of light that appeared turned out to be constellations, entire clusters, giant congregations of stars that people hadn’t known were there. PulseNet is the CDC’s clustering. This is our opening of the scope.” In one way, the Hubble analogy explained PulseNet well. Stars were to cases as constellations were to clusters of disease, and the Hubble was a technology that allowed scientists to see the previously unseen. In another way, however, the Hubble analogy was misleading. While a scope could be opened in a single moment, it took much longer for the PulseNet database to be populated with data and thus create the stars in the sky.

PulseNet’s origin story is rooted in the Jack-in-the-Box outbreak. Timothy Barrett worked as a scientist in the Enteric Investigations lab during the Jack-in-the-Box outbreak, and recounted how, after the outbreak, the CDC was “getting hundreds of [O157] isolates from all over the country, from people wanting to know if it [their isolate] was related.” However, Barrett shared, “we didn’t really have a good way to look at that.”

While PFGE would become PulseNet’s centerpiece typing technique, Barrett shared that “it was by chance, almost, that I used it for O157 and the Jack-in-the-Box outbreak.” The Enteric Investigations lab happened to have PFGE equipment, but, Barrett recalled, “it must have been for something else that we were doing, I can’t remember why we even had it now.” When I interviewed him, I had never encountered that facet of the story, and I was intrigued. He

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1 Dybas (2009).
explained, “it had already been published that PFGE wasn’t especially useful for O157… I decided, well I’m going to take another look at this because we need other tools.”

I had not encountered the paper that Barrett mentioned, reporting that PFGE was not especially useful for O157, and I expressed my surprise to him. He continued by explaining that the group who had published this result did not have access to a “sufficiently large group of characterized strains,” so in his view, they really could not say, “these are epidemiologically linked and they look the same, and these are different.” That group, he recounted, found low variability in the strains they examined. The CDC also found a similar result of low variability. However, because such the Jack-in-the-Box outbreak made such a large group of clearly epidemiologically-related O157 strains available, the low variability had a different meaning. The group had a large set of members that were related, which did not mean that PFGE could not discriminate between O157 isolates. Instead, “by chance,” Barrett discovered that PFGE worked well for subtyping O157.

This chapter examines how the early warning, real-time system for foodborne outbreak detection and response was built, including the various networks of PulseNet, FoodNet, OutbreakNet, FoodCORE, and the FDA’s Coordinated Outbreak Response and Evaluation Network (CORE). I proceed by explaining how PulseNet incorporated PFGE into the system;

2 Tauxe pointed out to me (e-mail communication) that in 1986, Wachsmuth had been experimenting with restriction endonuclease DNA analysis, using restriction enzymes of 4-base and 6-base cutters, but found that these both cut the chromosome up into too many small pieces. In 1992, Karch developed an 8-base cutter, which the CDC began to use in their lab later that year (Cieslak et al). While this does not get at the very specific reason why the lab had the particular instrumentation that it did when Barrett worked on the O157 outbreak, that the Enteric Investigations lab was interested in the technique was established in the 1980s-1990s.


4 Barrett said that “in retrospect” this was a stable strain.
PulseNet’s genesis and early development; and the concurrent creation of FoodNet. Then, I describe how system-builders attacked a set of critical problems all related to standardization: shortening protocols, formalizing a testing system, and implementing information technology. After that, I chronicle how standardization began to pay off as data in the system accumulated, and the new, diffuse outbreaks became more visible in the database. The last empirical section of the chapter examines how the epidemiology and regulatory interface became more integrated over time, aimed at attacking the (long-standing) critical problem of tracing the food back to the source.

**PFGE: Solving Critical Problems to Create a System Component, 1984-1992**

This section briefly describes PFGE’s development in academic research and then its commercialization in the biotechnology sector, so that it could become a system component for public health surveillance. After its initial development in academia and biotech, it was a technique that was adapted for public health use through standardization. Because I am more interested in telling the latter story, I emphasize that I only gloss PFGE’s history as here to set up my narrative, though a much fuller history of PFGE’s biography could be written.

Electrophoresis was a technique first developed in the 1930s by chemist Arne Tiselius to separate colloids by moving charged particles through a liquid solution under the influence of an electric field, and one of the key tools through which biology was “molecularized” in the 1930s-1940s, defining the study of life at the submicroscopic level (Kay 1996). During the 1950s-1960s, scientists improved upon the previous methods, characterizing older techniques as “moving boundary” electrophoresis, and new techniques as “zone” electrophoresis (Chiang
2009). Zone electrophoresis, as Chiang discusses, enabled more complete separation and visualization of molecules, with A.H. Gordon’s group finding that agar jelly was the best medium to use for electrophoresing large molecules. Post-1960s, gel electrophoresis was a pervasive technique in molecular biology and biochemistry.

A major innovation for gel electrophoresis was the pulsed field technique. In 1984, David Schwartz and Charles Cantor, molecular biologists and human geneticists at Columbia University, published a paper describing their innovation.\(^5\) Large chromosomal DNA molecules could not be adequately separated by gel electrophoresis itself. Previous to the pulsed field innovation, scientists would previously manually reconstruct linkages between DNA fragments, “allowing a chromosomal view as complete as one has the patience to construct” (67). With the pulsed field technique, or, “perpendicularly oriented, non-uniform, alternately pulsed, electrical fields,” scientists could separate much higher molecular weight DNA molecules with a greater resolution than previously available. Schwartz and Cantor had filed a patent for their innovation in 1982, which was published in 1984.\(^6\)

Schwartz and Cantor’s original paper applied PFGE to yeast chromosomes. However, as Smith and Cantor\(^7\) pointed out, PFGE could be applied to bacterial chromosomes by converting their circular DNA into linear fragments with restriction enzymes that would cut the circles at rare restriction sites. Cutting would create linear “fingerprints” of the bacteria. Smith and Cantor ran a gel which included both *Salmonella typhimurium* and *E.coli*, revealing the “surprising result” that the two bacteria had “quite different” PFGE fingerprints.

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\(^6\) Electrophoresis using alternating transverse electric fields, US 4473452 A.

Schwartz and Cantor’s technique set off a subsequent “flurry of innovation in instrumentation” within molecular biology, so that by 1992, one geneticist remarked that “in a few years, PFGE has gone from being fairly esoteric technology… to being routine technology” in every molecular biology laboratory.\(^8\) PFGE was noted to be especially impactful for the study of microorganisms, because of their low complexity genomes. According to the National Institutes of Health (NIH), PFGE “enabled rapid early progress” for the Human Genome Project (HGP), before the development of more powerful sequence-based technologies.

When I asked Cohen what made PFGE the “right tool for the job”\(^9\) for PulseNet, he pointed to PFGE’s wide uptake in molecular biology, as well as the fact that many laboratory technicians would also have familiarity in working with restriction enzymes. The key to adapting PFGE for large-scale public health use would be in standardizing the technique. The “level of complexity” of some other methods (e.g. Western blots, sequencing), he recalled, “was perhaps a little too much for thinking about standardizing things.” He continued:

We were really at a point in time where the concept of doing even limited sequencing was beyond the scope of what you could really do as part of an outbreak investigation. It’s sort of humorous now, thinking about how rapidly one can sequence whole eukaryotes as opposed to prokaryotes but it’s something that didn’t seem like it make a great deal of sense to pursue. So pulsed field gel electrophoresis was something that was fairly straightforward, could be easily taught, could be relatively easily standardized, was one of a number of techniques people were familiar with, so it had a lot of appeal to it for those reasons.\(^{10}\)

Cohen mentions that limited sequencing was available, but that it could not be done as part of an outbreak investigation, a highly time-pressured activity. As Hilgartner (2004) points out, in the

\(^8\) http://www.the-scientist.com/?articles.view/articleNo/12490/title/Pulsed-Field-Electrophoresis-Enhances-Genome-Effort/.

\(^9\) I borrow this phrasing from Clarke & Fujimura (1992) and Casper & Clarke (1998)

\(^{10}\) Cohen did not specify and I did not ask what kind of sequencing was available at the time (in CDC’s view) but according to a history of sequencing created by Science, from 1987-1995 sequencing was done by hybridization (which would have been manually intensive), and it was in 1996 that pyrosequencing was commercialized, with the systems Roche Applied Science/454 Life Sciences (Genome Sequencer 20; Genome Sequencer FLX) http://www.sciencemag.org/site/products/posters/SequencingPoster.pdf.
early 1990s, at the outset of the ambitious Human Genome Project, capabilities did not match vision; mapping the genome was “painstaking, manual work,” requiring a great number of laboratory workers and as yet little automation. Cohen points out that PFGE, compared to other methods of the day, seemed more amenable to standardization, several other interviewees mentioned to me, at the time, that some members of the scientific community felt that standardizing PFGE would be too difficult.\textsuperscript{11} In the next section, I will describe PulseNet’s genesis, and how public health scientists at the CDC identified and tackled initial critical problems, and worked to winnow broad issues to finer-grained, specific questions, navigating scientific, organizational, political, and social dynamics in the process.

**System Genesis and Early Development: 1993-1999**

Bala Swaminathan, whom many other scientists have informally deemed the “father of PulseNet,” recounted a number of reverse salient, targets, and critical problems in his oral history narrative about the genesis of the National Molecular Subtyping Network for Foodborne Disease Surveillance, which would only later acquire the snappier name PulseNet.\textsuperscript{12} To explain to me what, exactly, PulseNet innovated, Swaminathan emphasized that in the Jack-in-the-Box outbreak, the laboratory investigation was all conducted “after the fact, after the outbreak had been investigated, everything had been done.” He recalled that after Barrett had completed this work, and brought him a draft of the paper to review, that this was the stimulus for an idea for a new system. As he reviewed the manuscript, he

\begin{quote}
…started thinking, why is this happening, it doesn’t make sense to me that all of this typing work, which really is critical to unraveling the outbreak, is being done
\end{quote}

\textsuperscript{11} See page 228.

\textsuperscript{12} Interview, Bala Swaminathan, 2/15/12.
later, as a confirmation of everything the epidemiologists have done. So you are really putting the cart before the horse here.

What Swaminathan selected as the reverse salient in this problem-solving moment was the timing of the typing, which typically happened after outbreak investigations were finished, to confirm epidemiologic findings. He recounted that he and Barrett had had extensive discussions about whether it was possible to speed up the timing of the typing, so that it could occur during outbreak investigations rather than after the fact.

Swaminathan remembered Barrett arguing that it was “impossible to do the typing in a time-sensitive way, within a time-frame that would be relevant to the investigation,” due to the fact that the CDC and only a couple of other state public health laboratories were doing PFGE, and could not handle a large number of isolates in their labs. Swaminathan rooted the “genesis of the idea” for PulseNet in these discussions, convincing him that “we really needed to do something to change things around.” “If you could put yourself in 1993,” he invited me to retrospect with him:

the epidemiologists had all the cards in their hands, because they were informed about the cluster of cases, then they would look into it, and formulate the hypothesis and all of that stuff. And the laboratory essentially played a supporting role in getting the samples and isolating the organism and confirming things. In 1994 then, we had this idea that we needed to do the typing as close to real-time as possible.

What they would eventually achieve, he argued, was shifting PFGE from a “confirmatory” tool to one that could be used within the time-window of an ongoing outbreak investigation—“real-time.” I will discuss this key critical problem of “real-time” a bit later, as public health scientists worked on it more extensively as they built the system up.

In the above narrative, Swaminathan used what Nicole Nelson (2012) calls a “localized” interpretive repertoire to narrate the innovation process, highlighting defined moments and key
individuals. However, he also used what Nelson calls a “distributed” repertoire for narrating innovation, that emphasized a longer time frame, the process of technical implementation, and a broader network of collaborators, which I describe below. Nelson studied technology transfer, where localized repertoires emphasized the patentability of the technology, and distributed repertoires emphasized the utility of the technology. In the context I discuss here, Swaminathan used the localized repertoire to capture the crystallization of a new public health target, and the distributed repertoire to detail its actual organizational implementation.

From 1986-1994, Swaminathan worked in the Meningitis and Special Pathogens Branch (MSPB), as the Chief of their Epidemic Investigations Lab. He emphasized the “collegial atmosphere” of the MSPB, where epidemiologists and microbiologists worked “very closely” with each other, and recalled that this “really formed the way I would tackle problems in foodborne diseases,” when he moved to the newly formed Foodborne and Diarrheal Diseases Branch (FDDB) in 1994.

Additionally, while in the MSPB, one of his foci was *Listeria monocytogenes* (LM), a foodborne pathogen of growing concern in the 1990s; LM could cause bacterial meningitis. Prior to PulseNet, Swaminathan recalled using PFGE for an extensive laboratory investigation associated with an Oklahoma investigation of a single case of LM.\(^\text{13}\) In 1988, a cancer patient had been hospitalized for sepsis caused by LM. The food history interview for the patient localized her singular eating practice; everyday, the patient microwaved and ate a single turkey frank. The laboratory investigation was able to connect isolates from the patient, an opened package of franks from her refrigerator, and two unopened packages from a store. All shared the

same PFGE pattern, demonstrating the power of the technique.

The MSPB also played a role in the genesis of another foodborne surveillance system, focused on sporadic cases of illness—FoodNet. Cohen pointed to the MSPB’s work on the “concept of active surveillance” as influential for FoodNet, particularly Claire Broome’s laboratory surveillance of meningitis in sentinel sites. The methodology of sentinel site surveillance involves selecting a smaller sample of reporting sites, conducting more intensive follow-up on information collection to improve data quality, and, if possible, generalize from cases to a larger population.\textsuperscript{14} The meningitis program consisted of active attempts to gather complete case information, monitor compliance, and resolve inconsistent or missing information between selected sites.\textsuperscript{15}

From a programmatic perspective, Broome’s “statistical and structural basis” fit nicely with what other people wanted to do with other active surveillance programs, Cohen recounted, and in FDDB, would help inform the FoodNet’s design. Active surveillance was “in the air” at the institution, Tauxe recalled. He also shared that while an initial proposal for a sentinel site surveillance system to study sporadic cases of disease was developed internally at the CDC in 1993, Fred Angulo’s oral history account covered why the system was not created until 1995.\textsuperscript{16}

Angulo shared that it was after the 1995 Megareg was passed that CDC saw another “opportunity” to build a system, and facilitate inter-agency coordination around it. After the Megareg was developed, he recalled, the meat industry asked the USDA, “how will you know this [regulation] works? Or, results in less illness?” As a result, the USDA contacted the CDC,

\begin{flushleft}
\textsuperscript{14} MacDonald (2012).
\textsuperscript{16} Interview, Fred Angulo, 11/27/12.
\end{flushleft}
and the CDC proposed to design a program to “measure precisely” the human illness in order to track the “progress” of the rule. To do so, Angulo narrated, “would require more intense surveillance than we currently have… it would require sentinel site surveillance.”

While initially launched to evaluate the public health impact of the Megareg, public health officials developed a holistic vision for FoodNet as a “platform” for launching analytic studies, such as case-control studies, or population surveys. The metaphor of “tip of the iceberg” had been used for decades to lament the fact that sporadic cases were not well-characterized or understood, and that foodborne disease had always been severely under-reported. They adapted the iceberg metaphor and modified it into a pyramid, with various layers to substantiate the middle and base of the iceberg, each layer representing a subsequent step between the base (well population) and the tip (sick population reported to public health). See the figure below:

Figure 20. FoodNet pyramid

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The pyramid helped represent how, by actively contacting physicians and clinical laboratories (via telephone) to improve the interface between health care and public health, and conducting survey studies with labs, physicians, and the sentinel populations, public health officials could able to quantify underreported cases at each step, and produce more statistically grounded estimates of overall foodborne disease burden.

With FoodNet, the long elusive reverse salient of underreported sporadic cases had finally been turned into a set of more actionable critical problems. While creators of the NSSP had initially hoped their system would be able to make both outbreaks and sporadic cases visible, PulseNet and FoodNet meant that there would be two different systems specifically tailored to each problem.

Swaminathan recalled that it was a small amount of initial funding that drove him to “decentralize” the performance of PFGE on a regional basis, to enroll some state public health laboratories to pulse isolates. As the hundreds of isolates sent to the CDC after the Jack-in-the-Box outbreak had demonstrated, it would be exceedingly difficult for the CDC to handle all of the nation’s isolates on a routine basis.

Cohen gave Swaminathan 150K of initial funding, from internal CDC funds, to evaluate whether it would be possible to create this decentralized bacterial subtyping system around PFGE. Swaminathan emphasized the small size of the grant, a “shoestring” budget, that led him to engage in negotiations with equipment manufacturers to sell him the equipment for as low a price as possible. The CDC selected 4 initial laboratories, “strategically located” in different geographic regions of the US (WA, MN, NY, TX), with the thought that if the CDC provided training, initial equipment, and some funds for personnel to these laboratories, other state laboratories could send their isolates to a lab in their area for PFGE typing.
Another initial critical problem to “tackle” was how to make patterns more comparable with each other. At the time, Swaminathan recounted, comparing patterns with each other was a process married to the physical gel on which the samples were run. “Comparison,” he pointed out, “could only be made on one particular run that was done in a laboratory that day on one gel.” Solving this problem would be achieved through digitizing PFGE gels, computerizing the typing system, and creating a database for storing and analyzing the data. To underscore the import of this decision, Swaminathan shared that this digital undertaking was a “far cry” from then-present practices at the CDC.

One interesting implication arising from Swaminathan’s latter point is that it affirms an argument Peter Chow-White & Miguel García-Sancho (2012) have made about the convergence of biology and computing, that it is far from being a “natural marriage” where biology and computing were “predestined to coalesce,” as it sometimes framed. Instead, convergences of biology and computing are dependent not just on technical capacity, but on an array of scientific and social factors unique to different contexts (Hine 2006). Technical capacity plays an influencing role, but does not determine why connections are built, nor how connections are developed.

In PulseNet’s first fifteen months, the group worked on PFGE standardization (protocol development and identifying a standard O157 strain); evaluating and then setting up analysis software (BioRad’s Molecular Analyst Fingerprinting); determining uploading procedures (at first, state labs would email patterns to the CDC, or upload them to an FTP site); pattern normalization (around the standard O157); and creating database infrastructure. During this initial period of development, Swaminathan shared, they encountered two major issues with the regional decentralization strategy they had chosen. One issue was that while there was an
established administrative and material “pipeline” where state health departments could send their samples to the CDC, there was “absolutely no mechanism for transferring samples from one state to another.” The second was that other state public health labs besides the four initially selected complained that a “superlaboratory” approach would create “disparity” amongst labs. State public health labs countered that all labs should have the same “basic capabilities.”

The solution for these two problems was the same—to expand the program beyond the four initial laboratories, decentralizing not just on a regional, but a national basis. Barrett pointed out that after the Jack-in-the-Box outbreak, many states had asked the CDC to pulse their O157 isolates, because of increased interest in the public health problem, so interest in network membership was already established. Swaminathan recounted a process of marshalling financial support for PulseNet expansion through an NGO partnership with the Association of Public Health Laboratories (APHL), as well as the CDC’s new (as of 1995) Epidemiology and Laboratory Capacity for Infectious Diseases Cooperative Agreement (ELC). The ELC, Swaminathan mentioned, had been established after the Institute of Medicine had issued a report decrying how the US’s public health capacity and infrastructure had reached an “alarmingly poor level.”

Network membership offered state labs some equipment and personnel to help deal with crumbling public health infrastructure in the states, and thus made joining the program attractive. Indeed, Barrett shared, “we [CDC] probably bought it [equipment] in most states, that was probably the key reason it [PulseNet] expanded rapidly.”

Another source of expansion came from another major E.coli O157:H7 outbreak in WA in 1996, which kept the pathogen in the national limelight. Additionally, since the source was contaminated apple juice manufactured by Odwalla, this new vehicle expanded public concern

beyond meat and poultry to the safety of the entire US food supply.\textsuperscript{19} If a nationally-distributed juice brand was not safe, what else was not safe? Was the whole US food supply tainted? From PulseNet’s perspective, because the outbreak had been detected in WA, one of initial 4 PulseNet sites, the state laboratory could PFGE type isolates and samples, further demonstrating and making an argument that the program was “valuable.”

Clinton used the apple juice outbreak as an example to support the need for his Food Safety Initiative (FSI), to make sure that the government was “using the world’s best science to help prevent food contamination tragedies before they happen, to make sure our supply of food is safe as it can be,” which he officially announced on October 2, 1997. The FSI injected $43M into the development of broad food safety policy reforms. In his announcement, Clinton lauded the US food supply as the world’s safest, but admitted that the Odwalla outbreak was evidence that it should be made even safer. This \textit{safe but could be safer} frame is usefully interpreted in light of an observation Stephen Hilgartner (2007) has made, that controlling disasters involves not only material, on the ground activities, but “discursive” containment, framing the situation in ways that help restore public confidence that social institutions have the capacity to protect the citizenry.

The FSI was broad in scope, with multiple agencies (FDA, CDC, USDA, EPA) involved in the process of making policy changes to try to make the food supply safer, and developing many new inter-agency coordination programs and practices in the process.\textsuperscript{20} PulseNet and FoodNet received additional support through the FSI, and were also held up as prominent examples of how the government was working proactively to make the food supply safer for

\textsuperscript{19} http://edition.cnn.com/HEALTH/9611/01/e.coli.poisoning/.
\textsuperscript{20} Food Safety from Farm to Table, A Report to the President, May 1997.
Americans. Swaminathan pointed out that one of PulseNet’s ingredients for “success” was that public health officials could articulate a “clear vision” for the program, in “simple terms”—“PulseNet Saves Lives.”

An AP article with the headline “Food Safety Now Entering Computer Age” from May 23, 1998 reported that on the previous day, Vice President Al Gore “unveiled the new computer web” during a White House event, calling it an “an early warning system for danger at the dinner table,” which, “by saving crucial time…will save lives.” In 1998, the DHHS sent out a press release about CDC’s PulseNet with the headline, “National Computer Network in Place to Combat Foodborne Illness: Detects and Traces E.coli Strains Up to Five Times Faster.”

According to the press release, as of 1998, twelve states were “on-line,” as well as the USDA and FDA. In 1999, PulseNet won a $100,000 “Innovations in American Government” award, funded by the Ford Foundation and administered by the Harvard Kennedy School of Government. In the public arena, the how part of safe but could be safer could now also be framed almost as simply; since the government was building a computer web to detect outbreaks faster, Americans would be safer at the dinner table.

Delineating and Narrowing the Myriad Critical Problems of Standardization, 1996-2013


23 Since the 1980s, Gore had taken an “intellectual and legislative interest in promoting high-speed data networks” in the US, though in 1999 would be publicly ridiculed for saying that he “took the initiative in creating the Internet,” which was widely interpreted as a claim that he invented the internet (Wiggins 2000).


25 The award criteria were: novel, effective, solve a significant problem, and be replicable by other government entities. http://www.cdc.gov/media/pressrel/r991015.htm.

http://govinfo.library.unt.edu/accessamerica/docs/pulsenet.html
In the public arena, the simple frame for the network’s configuration and purpose highlighted two characteristics; its configuration was computational/Internet-based, and its key purpose was to make outbreak detection speedier. However, one characteristic that was not as emphasized publicly, but for public health scientists, was its core, overarching critical problem—it had to be a standardized system. And standardizing the system would be anything but simple.

In this section, I pay closer attention to how public health scientists approached delineating, tackling, and narrowing the myriad critical problems of standardization, in the life course of the network. Furthermore, as I show, ongoing management work is required to keep standards standardized in the network, and to also accommodate and navigate change. Thus I bring this section into (very) recent history, though I will backtrack temporally to discuss different components of the sentinel subsystem.

Why standardization was the core critical problem for PulseNet is well-stated by Susan Rogers and Alberto Cambrosio (2007), who say that standards are a “sine qua non” of contemporary science. As they point out:

A machine as an isolated entity does not produce meaningful results, and debates about the factual status of a given result hinge upon successful claims about the reproducibility of the experiment that produces it, which in turn depends on establishing equivalences between the instruments and research materials used in different settings.

The answer to the question “why standardization” is thus a fairly simple one; it is the only way that the network could produce comparable, trustworthy knowledge across distributed space and unrestricted to a single point in time. However, the answers to the questions, “how specifically should standardization proceed,” and “what are the downsides and upsides of standardization in this context,” are much more complex ones, which the rest of this section takes up.
As I mentioned earlier, according to several interviewees, there was not widespread consensus in the scientific community in the 1990s that it would be possible to create a standardized system around PFGE. In discussing his group’s experience with a multi-center study evaluating PFGE reproducibility for *S. aureus*, Van Belkum pointed out that standardizing molecular typing methods is always an issue of debate, not only about what method to choose to standardize, but what constitutes appropriate use and interpretation of molecular data, and what database could support the import and analysis of information by multiple, distributed laboratories. He argued that one of the very few successful large-scale standardization efforts had been around IS6110 sequencing of *mycobacterium tuberculosis*, and that this effort required many years and the “concerted efforts of many individuals and institutions to achieve.” In other words, standardization of molecular typing systems was in theory possible and in practice rare, with involved and intricate work requiring the coordination of many players and many different elements.

Stefan Timmermans & Stephen Epstein (2010) define standardization as “a process of constructing uniformities across time and space, through the generation of agreed-upon rules.” Constructing uniformities, or standards, gain power when they are plugged into broader sociotechnical infrastructures, but as a result, they argue that makes them difficult to change; changes can introduce incompatibility and switching costs. Their use of the term “rules” is helpful, because, as they note, a core paradox of rules articulated by Wittgenstein is relevant; despite their purpose, rules do not necessarily determine courses of action. It is always helpful to examine the sociotechnical infrastructure keeping rules and actions bound together. How are

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standardization processes related to information balanced with the “anticipated and historically proven need to accommodate to as yet unknown changes and patterns of use” (Hanseth et al 1996)? Furthermore, as Geoffrey Bowker (2000) points out, those who have to conduct long-term data management have to confront a “Scylla and Charybdis”—keeping too much of the past versus not keeping enough of the past.

**Getting to Real-Time: Shortening Protocols**

Efrain Ribot’s account captured how the process of making standardized PFGE protocols “real-time” was intertwined with making them doable in the network of state laboratory users.27 When he started working with PulseNet in 1996, he shared that one of his early tasks was to figure out a way to make the protocol for *E.coli* O157:H7 shorter, to make it work in the real-time context of an outbreak investigation. Existing protocols took between 3 and 7 or more days, using many different reagents, enzymes, and electrophoretic conditions.28 He narrated his thinking at the time:

…we need to shorten the protocol from 4 days to 1 day, 2 days, whatever the case may be. “Can we do that with the reagents we have now” is the first question obviously, right? Can we do it with the materials we have in the laboratory? Along those lines one of the first things that I did was to call a handful, I think it was either 5 or 7 state health departments that were already involved with PulseNet, and asked them the question, well what do you want to see in the protocol that is shorter? And one of the common themes was… well we don’t want to have to buy new reagents. So now you have your challenge. How do we push the envelope without specialized reagents? There was a state department that developed a 1-day protocol that required agarose in a buffer that was not commercially available, number one, and number two it was hard to make, because it pushed the limits of the buffering capacity of the buffer to begin with… neither of those two things I liked.

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27 Interview, Efrain Ribot, 2/21/2012.
The CDC had developed its first standard protocol for O157 in 1996, which it disseminated in a training manual. Ribot was referring to a 1-day protocol developed by Romesh Gautom in the WA state health department, published in 1997. Gautom argued that a 1-day protocol would make PFGE more useful for “monitoring the often rapid evolution of events during ongoing outbreaks,” shortening the then-current protocols’ “time-consuming, tedious procedures for the purification of intact genomic DNA trapped in agarose, lengthy restriction enzyme digests, and extended electrophoresis times.”

This debate over the question of just how much the different steps in PFGE protocols could be shortened reflects not just scientific and technical considerations, but economic and social ones as well. WA state had been one of the initial “superlaboratories” with a longer history with and infrastructure for surveilling O157, but other state laboratories were newer to the network and did not share same material and economic “realities” (to invoke Falkow’s phrasing). The push to expand the use of the technique widely shaped the negotiations over and decisions around what would eventually constitute a workable standardized “real-time” configuration. In 1998, the CDC and WA state health department collaborated to combine the longer and shorter protocols into one standardized solution for the network. While many steps were slightly shortened or tweaked, some of the major ones involved reducing lysis incubation time from overnight to 2 hours, and shortening restriction digestion incubation time from 4 - 16 hours to 2 hours.

In an ethnographic snapshot taken from the 2013 InFORM meeting, a related debate over the speed of PFGE protocols came up during a session where laboratory scientists were puzzling

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over how to solve an emerging reverse salient. One state had been experiencing a much longer shipping time for isolates between the clinical lab and the state public health lab, possibly heralding changes in the clinical lab. A laboratory scientist in the audience suggested a humorous solution. He mentioned how PFGE run times could be “frustrating” because they were long, and joked, “Turn up the temperature to make it warmer and it’ll go faster,” which caused chuckles in the audience. He used his joke, however, to lead into a serious question about the historical choice of temperature in the protocol.30 “Seriously,” he wondered, “how did we settle at 14 degrees as the magic number?”

To answer this question, Ribot explained that the PFGE manual from 1996 had standardized 14 degrees as the temperature for the protocol. He pointed out that when you initially standardize a protocol’s parameters, switch times, buffer composition, and temperature, and you then build a large data set based on that protocol, it makes it hard to go back and change those initially selected conditions. He recounted how PulseNet Central had conducted early studies on the effect of temperature changes on the protocol, and how that interacted with the seasonal stability of PFGE. This was a crucial factor, because they found that during the summer time, ambient temperatures in labs would increase and have a significant impact on the mobility of the DNA fragments. Thus 14 became the magic number, he recalled, because they had tried 16 and 18 degrees, finding that while the bands went faster, the gels lost resolution and consistency. Though 12 degrees would yield gels with “beautiful band sharpness,” the temperature change altered the resolution of individual bands, so that what would like a singlet using one temperature would look more like a doublet under the other temperature. This was a

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30 Pinch (1995) analyzed scientific meetings and found that humor is a rhetorical tool that helps convey and emphasize serious issues.
problem because it would make patterns produced under 12, 16, or 18 degrees, less comparable with patterns produced under the 14 degree standard temperature.

*From Method to Process: A Formalized Testing System to Get to Real-Time “Head-Hunting”*

Michael Lynch (2002) has examined the standardization of protocols in molecular biology, through an analysis of a biotechnology news group, where molecular biologists from other labs were offering advice to a scientist who had experienced some trouble with performing PCR. He points out that protocols are similar to recipes, and while recipes specify and instruct, they leave room for contingency, and can be underspecified in ways that require the judgment of the person following the protocol to implement the step correctly (e.g. rolling versus scraping the swab in Chapter 2). Lynch’s points help set up the fact that standardizing protocols is challenging, and to understand standardization, we should examine the negotiated relationships between protocols and scientific practices that help make protocols stable and reproducible.

Ribot emphasized to me that “he grew up in PulseNet” not just thinking about the “method” of PFGE, but also “thinking about the process,” “how it was going to be applied, and who was going to use it.” Trained as a molecular biologist, he brought what could be usefully characterized as more of an engineering mentality to PulseNet. This engineering mentality would be important for managing expansion of PulseNet’s purview beyond its initial focus on O157, to create standardized protocols for other enteric pathogens. Additionally, while the network initially adopted PFGE as its centerpiece typing technique, genetics was a growing and accelerating field, especially due to the influence of the Human Genome Project. From both Swaminathan and Ribot’s accounts, there was early thinking that the network would at some point shift to other typing methods, and it would be important to ensure that the methods were backwards compatible with PFGE.
One answer to the puzzle of how PulseNet standardized PFGE, I argue, is that PulseNet builders created a formalized testing system, using engineering-like principles to develop and validate protocols based on prospective, current, and retrospective testing (Pinch 1993). After hearing Ribot give a presentation emphasizing PulseNet’s process and how the network enabled “head-hunting” (early warning identification of laboratory clusters), I asked him to explain further:

…in order to arrive at the point where we could do real-time “head hunting” with laboratory tools/methods we had to identify a tool/method first and then (using retrospective isolates for which we had solid epi data) tested for epi relevance. If the method “held water,” in the epi context, then we could move forward with the validation phase (using a mixture of isolates, present and past) but focusing on the ability of the method to accurately segregate isolates “prospectively.” While head hunting is the final goal this phase is the last one in the process. That is, we only engage in head hunting after the method has been properly validated (both internally and externally—externally meaning that we always “test” the protocol in a handful (or more) of labs outside CDC (mostly public health labs).  

In the 1990s, there was a broad move towards formalizing the evaluation of microbial epidemiologic typing systems with process management and control principles. For example, in 1996, the European Society for Clinical Microbiology and Infectious Diseases (ESCMID) published consensus guidelines they had started developing in 1994, complaining that “new typing methods are often applied without critical evaluation of their performance characteristics.” ESCMID observed that many methods lacked standardization of technical procedures, reference material, quality assurance, interpretive criteria, and even basic terminology, such as “type,” “isolate,” or “outbreak.” The consensus guidelines offered definitions, criteria, and assessments of several phenotyping and genotyping methods.

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32 Personal communication.
In this chapter, I have chosen to focus on the criteria they identified, with special attention to two in particular. They identified five evaluative criteria for typing systems, each with mathematical formulas to produce quantitative measures of each criterion: typeability (proportion of strains assigned a type by a system), reproducibility (ability of a system to assign the same type to a strain typed by a separate assay), stability (ability of a system to recognize relatedness of strains despite phenotypic or genomic variation), discriminatory power (probability that the system would assign different types to two unrelated strains chosen at random), typing system concordance (similar results from different types of systems), and epidemiologic concordance (probability that epidemiologically related strains from a well-described outbreak are determined to be similar). The latter two capture my particular interest. I see “epidemiologic concordance” as a formal concept capturing the circular feedback relationship which began to link laboratory and epidemiologic science during the 1970s. “Typing system concordance” is also of interest to me, because it helps elucidate the strategy PulseNet used to try to expand and maintain the network by incorporating other typing systems into it.

In 2000, PulseNet started a new program, the Next Generation Sequencing and Methods Development Program (NGSMDP), with the aim, as Eija Trees said, “That one day we will be able to replace PFGE or complement PFGE.”34 Trees, who joined the NGSMP in 2004 as its head, recounted the program’s history for me. Initially NGSMDP gave grants to two (high performing) states to develop MLVA protocols for Salmonella Typhimurium (MN), and O157 (MA). The states conducted the initial development process, and Trees worked on “validating” those protocols.

34 Interview, Eija Trees, 2/24/2012.
However, Trees recalled, what the states felt was a “robust perfect protocol” differed from her view, which she attributed to the fact that each laboratory had its own perspective. Taking a national perspective, she had to create a “good enough” protocol that could “work in anybody’s hands.” In doing so, Trees had to negotiate the varying skill levels of different state labs. She pointed out that, due to infrastructure and funding problems, there was huge variance between labs, as well as what would become a perennial problem of high staff turnover in state public health labs. Thus, her protocol development/validation process took up a “ton of time,” in order to make the protocols “idiotproof.” The protocol development/validation process was extensive because it involved working with labs with different skill levels (low, medium, high) to extensively test whether the protocol was indeed working or not.

MLVA, it turned out, would become an “intermediate” technique for PulseNet, not a replacement for PFGE. Trees pointed out that PFGE did have the problems of being laborious, slow, and subjective, but as the protocol development and validation process for MLVA continued, they discovered that MLVA was too “serotype specific.” Trees explained:

O157 doesn’t work for any other shiga-toxin producing *E.coli*. MLVA will never replace PFGE. PFGE is universal, there is one protocol for all 2500 *Salmonella* serotypes and that works. MLVA will not work that way and we can’t develop a separate protocol for each serotype.

However, within PulseNet, MLVA became a “complementary” method to PFGE, helping them to further discriminate common serotypes. For example, Trees explained, 42 percent of the SE PFGE database consisted of the same SE pattern.

Both Trees and Ribot articulated another problem they were encountering with sequence-based technologies in general. Trees described the challenges that “fast moving” genetic/genomic technology created for her in the public health context. She said:
The companies come up with a new version of their sequencer every 4-5 years. And very often, when they come up with a new upgraded version, something’s changed. They may have new reagents, which means that we have to re-validate our protocols. Because the new reagents, for example a new type of polymer, can change the fragment sizing a little. Those are huge problems. I would prefer a technology, pretty much, staying the same. That’s why PFGE is so straightforward. There’s not a whole lot they can change.

Ribot said, similarly, that public health officials had gotten “lucky” with PFGE—the platform had not changed much in fifteen years while newer technologies’ platforms had been changing too fast; it was relatively inexpensive to perform; it was epidemiologically relevant; and was “versatile” in being applicable to multiple organisms.

Here is an interesting example of technology value conflicts between different sectors. In biotech, innovating upon and improving a sequencer is core to securing profit. While something like changing reagents in an upgraded version is to the developer an incremental improvement making their product better, to the NGSMDP, it was a reverse salient causing “huge problems.” The thing that made PFGE “straightforward,” the limit to how much you could improve electrophoresis equipment, was precisely what made it less appealing to biotech developers (as a mature technology whose improvement had been maximized), and more appealing to public health scientists operating a standardized system. While, as Ribot joked in 2011, PFGE had become a “dino-typing” method, especially when thinking about the modern sciences of whole genome sequencing, metagenomics, and bioinformatics that PulseNet would have to eventually adopt and adapt, PFGE was also like a “cockroach”—an ancient creature, but still surviving.

**Data Accumulation and the Emergence of New Targets, 1998-2008**

As PulseNet was established and expanded in the 1990s, one of the drivers of system-development was state participation, for example, the aforementioned collaboration between the
CDC and the WA health department. Another “superlaboratory,” MN, had a history of strong foodborne disease surveillance capacity and expertise, and in the early-to-mid 1990s, had been actively experimenting with both laboratory and epidemiologic techniques in the foodborne domain. John Besser recalled that early on, MN had experimented with PFGE for *Campylobacter* initially, on which the technique did not work well, but that when they “threw in” some *E.coli* O157:H7, it worked “spectacularly.”

After this, in MN, as soon as a case of O157 was reported to health officials, that would trigger two responses, one on the laboratory side and one on the epidemiologic side. The lab would pulse the case’s isolate as soon as it reached the public health laboratory, and the epidemiologists would use a “full trawling questionnaire” to interview the cases about possible disease exposures, to try to shorten the time between when the person ate contaminated food and when they were asked questions about their diet.

MN found PFGE useful not only for O157, but for *Salmonella* as well, especially in grappling with the reverse salient of the common serotype. He recalled:

We tried it [PFGE] for *Salmonella*, for some of the common serotypes… the goal here is to improve the signal-to-noise ratio. And we started, in the first three months of doing *Salmonella* PFGE, we found four or five Typhimurium outbreaks. We had never, in my memory at least, found Typhimurium outbreaks… So this was the beginning of mass subtype surveillance.

An important aspect of Besser’s demarcation of the beginning of mass subtype surveillance was not only the use of PFGE as a typing method, but also the focus on improving epidemiologic methods of case interviewing to collect better food exposure data.

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35 Interview, John Besser, 2/8/12.
Besser soon shifted into an explanation of the present-day system, as his current role is Deputy Chief of the CDC’s Enteric Diseases Laboratory Branch (EDLB). After describing a bit about how cases were pulsed and interviewed, he continued:

So all cases at the local level get uploaded, to here at CDC and the national database, and at the national level, we can compare patterns in different states. Just as they can look for clusters locally, we can look for clusters nationally, so there might be one case in each of 15 different states… It’s a resolution question. Sometimes the signal is strongest at a local level. Sometimes the signal is only apparent at a state level, or a national level. Each one of these levels is important.

Some of the concepts Besser used in his narrative reflect new informational metaphors that had been adopted within molecular epidemiology to reframe older versions of group comparison and population thinking—a language of signal-to-noise ratios, resolution, background, clusters, and levels.

In addition, not only could different “levels” be analyzed, but different kinds of data; Besser pointed out that “related programs” at the USDA, FDA, and in state departments of agriculture, that screened various sources from animals, meat, fruits, and vegetables, produced data that could be “fed, either directly or indirectly, into PulseNet.” One implication of including such heterogeneous data in the system was that preliminary matches between human and non-human isolates could provide clues as to what the source of newly detected outbreaks could be (e.g. if a newly detected cluster of human isolates matched the pattern of a food isolate, it might indicate that this was the food causing the outbreak—a “pre-outbreak”).

Besser tacked between present and past to explain PulseNet’s impact on the number of foodborne outbreaks that have been detected, before and after early warning surveillance was established, showing me the following slide of reported outbreaks between 1973-2008:
According to the graph, between 1973-1997, the average number of reported outbreaks was 500. Between 1998-2008, the total increased to 1200. Tauxe spoke of this increase in detection as PulseNet being “turn[ed] on,” saying:

PulseNet begins, and the number of detected outbreaks goes up, because we are detecting at least twice as many than without PulseNet. At least twice as many, and for the first time we are getting these broad multi-state outbreaks which we could never could see before.

In order to more fully decentralize the system and improve the management and analysis of accumulating data, in 2001 PulseNet shifted to a new database software, BioNumerics, developed by a then small start-up company called Applied Maths. One of the most important characteristics of the software that the company offered was customization. PulseNet became the “test-bed” for the company to develop subsequent versions of their software, according to

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Tauxe. BioNumerics, he said, was the “network-enabling” component for PulseNet. One significant issue that had encountered with the previous database was that, as more states wanted to have database access, it was challenging to add them because of security issues.\textsuperscript{37} By building a more extensive database system where states could not only submit, but analyze PFGE data, states could conduct cluster searches, and also for example, detect multi-county clusters. The CDC’s national perspective was an entirely different level of the population that would not “see” within-state or multi-county clusters as readily.

Accumulation of data and enhanced and decentralized cluster detection were the payoffs of standardizing the system. Not only were public health officials able to detect more possible outbreaks, they were eventually able to detect new kinds of “diffuse” clusters, with small numbers of cases in any individual state. Sometimes, a state involved in a national outbreak had just one case. New critical problems emerged.

One practical critical problem of detecting an increased number of clusters, Williams pointed out, was “workload.” Prior to 2010, outbreak response was housed along with all enteric disease surveillance in the Enteric Diseases Epidemiology Branch (EDEB), and members of the Outbreak Response Team were not only responsible for investigating outbreaks, but manning the National Outbreak Reporting System (NORS), a routine outbreak surveillance system. By 2010, the workload associated with cluster investigations was so large, the organizational entity reorganized, moving from a team-level entity to a branch level entity—Outbreak Response and

\textsuperscript{37} Hunter et al, “PulseNet: Software Changes and Improvements to Online \textit{E.coli} National Database,” Power Point presentation.
Prevention Branch. Between 2007-2010, the number of dedicated outbreak response personnel tripled.³⁸

Besser raised a critical problem that others responsible for control began to face, with the increased number of outbreaks being detected. Finding multiple outbreaks caused by the same vehicle pointed to bigger problems within that domain. He discussed sprouts as an example of a food type that, while “not yet a particularly safe food to eat,” was safer than before, because of PulseNet. “Part of the reason,” he said, “is that we’ve detected through PulseNet, over thirty outbreaks [implicating sprouts] in the last decade.” As a consequence, the sprout industry had been changing their practices. This was not only to try to avoid lawsuits and recalls, he explained, but importantly, regulatory agencies had responded to the group of outbreaks by placing more regulatory attention on that particular industry. As PulseNet accumulated more data and became more systematized between the 1990s-2000s, it became a significant shaper of new inter-sector communications around diffuse multi-state outbreaks as a shared responsibility of government, industry, and consumers.

“Consumer confidence is fragile,” food safety expert Gail Prince presented to the FDA Risk Communication Advisory Committee at a 2009 meeting.³⁹ Pulling data from a 2009 U.S. Grocery Shopper Trends study, he pointed out that 81 percent of consumers were aware of an increase in media coverage of food safety, and 73 percent of consumers reported being more knowledgeable about food safety than five years ago. Prince pointed to a number of underlying changes producing increased societal attention to food safety: concentration of food production,

³⁸ Personal communication, Ian Williams.
changes in food distribution, global trade. The consumer had changed, and science had changed, but most significantly, he argued, epidemiology had changed, with PulseNet detecting more outbreaks and new kinds of outbreaks.

PulseNet helped to significantly integrate what had been historically more fragmented activities of surveillance in the public health sector, and control in the regulatory and production sectors. In the next section, I follow one example of how sociotechnical networks and inter-sector communication became reconfigured, with a new public health target that began to emerge in the 1990s—contaminated produce.

The Epi-Regulatory Interface and the Critical Problems of Tracing Produce: 1996-2008

“Often, unfortunately,” Susan Lance told me in 2012, “with the big multi-state outbreaks, the ability to do a case-control study, or some other type of epi study is limited by a number of things.” To explain the limitations, she described “co-linearity in two dimensions.” She illustrated the first “dimension” with what she called the “Mexican food phenomenon,” a cuisine that could be particularly difficult to disentangle exposures within because “every ingredient [is] in every food.” One of the methods public health officials had developed in the past several years, to grapple with multi-state outbreaks, was to intensely focus on identified small clusters of cases associated with restaurants. Investigating these restaurant clusters raised this critical problem of disentangling exposures—“you can’t tease it out,” Lance said.

The second dimension, she explained, was the “physical part.” To illustrate this physical part, she described the issue of contaminated food in a sandwich shop, with multiple bins filled with different vegetables and other items. She pointed out that sandwich makers, if they did not

40 Interview, Susan Lance, 3/1/12.
wash their hands between handling vegetables in different bins, could cross-contaminate the sandwiches. So if the tomatoes were contaminated, she used as a for instance, but the sandwich maker cross-contaminated lettuce, even though the tomatoes were the source of contamination, some consumers who ate sandwiches without tomatoes, but with lettuce, could be sickened. Given these problems, she said, “what we’re left with is trying to narrow down with the epi… and then do what I consider distribution epidemiology, working from the point-of-service backwards.”

What Lance was referring to in her discussion was a reverse salient that had received significant policy attention in more recent years—the identification and removal of contaminated food in the food supply. The 2002 Bioterrorism Act had set in motion more intense policy focus on improving the record-keeping of food facilities, some of whom, beginning in 2005, were required by the FDA to:

- maintain records identifying the sources, recipients, and transporters of food products. The purpose of these records is to allow FDA to trace an article of food through each stage of the food supply chain—from a retail shelf back to a farm—if FDA has a reasonable belief that a food product is adulterated and presents a serious health threat. Traceability is the ability to follow the movement of a food product through the stages of production, processing, and distribution. Traceability includes both traceback and trace forward. Traceback is the ability to trace a food product from the retail shelf back to the farm. Conversely, trace forward is the ability to trace a food product from the farm forward to the retail shelf. Traceability is often needed to identify the sources of food contamination and the recipients of contaminated food in product recalls and seizures.41

Tracing contaminated food in the food supply and removing it from commerce has been a long existing and evolving public health/regulatory practice. However, I argue, it was in the 2000s when attention to and development of “traceability” as a policy focus and a systematized method

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41 http://oig.hhs.gov/oei/reports/oei-02-06-00210.pdf
intensified. Traceability evolved in a convergence of worries about unintentional and intentional contamination of food in the food supply.

This section draws on the oral history accounts of regulatory officials who described the creation of a more intensely coordinated epidemiology-regulatory interface around traceback and traceforward of contaminated food. As I stated in the introduction to the dissertation, the 1990s saw the beginnings of the mass globalization of produce. Thus, the 1990s also saw the emergence of global produce as the source of multi-state outbreaks, a target both public health and regulatory officials had to turn into critical problems.

Before coming to the FDA in 1997, John Guzewich was a career sanitarian in New York State.42 Because he had been trained in epidemiology and also worked in a regulatory capacity, at both the state and federal level, he could reflect on the lenses of each. In his oral history narrative, he began by describing his state work, which included setting up a state outbreak surveillance system. In describing this work, he recalled that he would often say, “I want to have an epi-driven food safety program.” Prior to establishing this system, he shared, he had long been impeded by another state sanitarian, who saw outbreak investigations as airing “dirty laundry.” Guzewich explained that sanitarians had inspections as a primary job, and outbreak investigations might call the competence of the inspector into question, suggesting that they did not complete adequate inspections of food facilities and restaurants.

This example, Guzewich explained, illustrated a general difference between epidemiologists and regulators, as ideal types. “Epidemiologists all want to be John Snow,” he said. “They all want to save the world.” Regulators, he contrasted, “have a different mandate.”

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42 Interview, John Guzewich, 10/30/13.
He pointed out that inspectional work was the primary focus of sanitarians, who would, for example, have to conduct a number of inspections within a certain time period to meet a “quota.” While of course both professional groups wanted food to be safe, he argued, their mandates and responsibilities differed. Additionally, he observed, epidemiologists and regulators inhabited a “different world.” The regulators’ world was very “structured,” with procedures, protocols, and legal restrictions shaping and constraining their actions. The epidemiologists’ world was much less structured, he said, using the John Snow reference again to illustrate:

When they are on the case... and they find a problem, they want to take the handle off the pump. Boom, boom! But our legal system doesn’t provide for it to be done that way. There’s this inherent friction between the two professions... The epis are out there finding associations and they want action taken, which is understandable, but our legal system doesn’t make that easy, for it to happen that way.

Guzewich then recounted the history of a 1995-1996 cyclosporiasis outbreak where the FDA would, for the first time, significantly confront global produce as a new vehicle of contamination.

In May 1996, eaters at a Houston restaurant, a Houston private clubhouse, and a Boca Raton dinner party, all fell ill with cyclosporiasis, a gastrointestinal illness caused by the rare parasite *Cyclospora*. County health officials in Texas and Florida investigated their clusters, and determined that strawberries were a key suspect food. While Florida health officials felt their evidence was still ambiguous, as the Boca Raton dinner party had featured a fruit salad with strawberries from California, red grapes from Chile, and raspberries and blackberries from Guatemala, Texas officials felt more sure. At the Houston restaurant, all of the ills had eaten a

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43 Stephanie Artero, “Cyclospora is showing scary affinity for strawberries,” *Palm Beach Post*, June 11, 1996.
dessert topped with a “signature strawberry.” Based on the epidemiologic evidence, Texas officials warned Houston residents not to eat strawberries, though no recall was issued.

By the end of June, the outbreak had grown to over 1,000 cases in 11 states. Furthermore, more recent evidence in the investigation pointed to raspberries, not strawberries as the culprit. Texas officials lifted their advisory on June 23. The California Strawberry Commission complained that the warning had cost the industry more than $20 million, and critiqued the Texas officials’ warning as “accusations without backup information.” Texas officials defended their warning, pointing out that testing for *Cyclospora* had been difficult and slow because no good test had been available, and that officials were protecting the health and safety of the citizenry. “It’s conceivable that we could be answering another question from the public right now: ‘Why didn’t you tell us?’”

At the Houston restaurant, Guzewich shared, the chef had reported that he only used strawberries to top his dessert. Part of the shift as the investigation proceeded was that traceback of distribution records, which took longer to conduct, revealed that the restaurant would sometimes use raspberries instead of strawberries.

Florida ended up having nine event-based clusters, eight in home meals and one at a catered event in a museum. They used the nine clusters to conduct a case-control study, which implicated fresh raspberries much more strongly than strawberries. Investigators conducted a traceback investigation with raspberries served at seven of the events to identify the country of origin for the raspberries. For six of the events, the raspberries definitely came from Guatemala;

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however, it was not possible to figure out which specific farm, as the exporter received berries from as many as 30 farms.

It was this outbreak that would bring Guzewich from NY State to the FDA, to help solve the critical problems that the outbreak had raised, particularly around the traceability challenges of produce. He narrated how this outbreak presented an entirely new set of challenges for the regulatory agency:

FDA, historically, was operating in the mold that it was comfortable with. It was a regulatory agency, okay? It did not really have much of a system where it got directly involved in the investigations, number one. Number two, when it got involved in investigations, they were manufactured foods, not produce. Different paradigm. Different experience than they had. Also, historically, FDA took actions based on a positive laboratory finding. If you had a Salmonella, you went out and sampled in until you found Salmonella, and so the action was being taken, as much as anything, because you found the pathogen in the food, not because of the epidemiologic association. Now they were being asked to take a regulatory action with a food commodity, that they really essentially, had no experience with, based on epidemiologic evidence, not on laboratory findings, with no prospect of having laboratory success. They were collecting all kinds of raspberries and doing microscopic exams trying to find Cyclospora, in this gemisch of mashed up raspberries. They were trying hard, but they just weren’t finding it. So, then you don’t know whether your method is bad, or you’re just not sampling the right berries. But either way they weren’t getting positives. So they were being asked to take action based on epi information, and there were a lot of legal people and historical regulatory people who wanted no part of that. So you had a tremendous amount of conflict.

When I asked Guzewich about an issue I had heard about frequently, that inspections were distinct from investigations, he expanded upon why there had “culture clash,” by contrasting the thought styles of epidemiologists and regulators. One specific example he gave was that FDA staff chafed at hypothesis-generating inquiry, a core norm for epidemiologists. He stated that the “typical” FDA investigator was trained specifically to not develop hypotheses, which were “poison” to these collectors of “findings of fact.”
FDA investigators, he told me, were not supposed to make hypothesis or conclusions, but simply make observations, take measurements, collect samples, and snap photographs, documenting their findings but making no inferences from the evidence they collected. Their job was to ensure that materials were being collected to withstand the “rules of evidence for a legal action.” He recalled “knock-down, drag out fights” in early investigations, around even the idea of thinking about hypotheses, where he would have upper level agency managers and lawyers “screaming” at him for introducing the idea.

Negotiating a way out of the agency conflict meant figuring out how to evaluate whether epidemiologic evidence could serve as evidence for taking a legal action. The following process was developed. Public health and regulatory officials created a “wise persons group,” which served as a “peer review mechanism” for outbreak investigations. This wise persons group consisted of some of the epidemiologists from the states.

In an outbreak investigation, the state who had the most cases in the outbreak would be responsible for collecting and sending out summary information (questionnaires, line lists, summary reports, pie charts, histograms, and epi curves). During a conference call, the states not heavily involved in the outbreak would then collectively evaluate the epidemiologic information, raising questions and issues to the main state in charge of the investigation.

After the peer review, the new summary report would be sent to the CDC, who would evaluate it, and then write an “endorsement” of the states’ investigation, recommending that the FDA take “appropriate” regulatory action, so that counsel could use this as the rationale for asking companies to initiate recalls, or taking other regulatory actions. This was “carving new ground at the time,” Guzewich remembered, distributing credit to Janice Oliver for playing a
This was not the only reverse salient that the agency encountered. At the time, Guzewich recalled, the FDA lacked a process for traceback, which he attributed to the agency’s past experience having focused on contamination in manufactured foods. He explained that manufactured foods were more simple to deal with because they had ready identifiers like lot codes, which would help them easily track down the manufacturer, requiring little investigation. “All of a sudden, along comes produce,” he said, presenting a number of challenges linked to its perishability, combinability, distribution, and lack of labeling. He continued by pointing out that “nothing in our food system” was ever “designed to facilitate traceback.” Rather, information was a “one way flow” to get foods from where they were manufactured to the “point of use…not to be able to figure out where it came from.”

Guzewich credited Michael Beach, at the CDC, for developing the FDA’s first version of its traceback procedure, a schematic for analyzing distribution records to identify the flow of contaminated foods. This schematic method was used to trace the Cyclospora outbreak to raspberries imported from Guatemala. With produce emerging as a food category of concern for the agency, a subsequent task Guzewich took on was to develop the first FDA’s process for investigating farms, as part of a new “paradigm” for the agency to move beyond manufactured food, and into the fields where produce was grown.

Sherri McGarry began to work on foodborne outbreaks in 1998 with Guzewich. She reflected that at the time, foodborne outbreaks were one of many crises that the same group at FDA was responsible for addressing—an “emergency response and coordination” group in the

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46 Interview, Sherri McGarry, 10/3/13.
CFSAN Office of Compliance, that would take on the “food angle” with diverse emergencies such as hurricanes and tsunamis. She noted that this group grew as a result of 9/11. When PulseNet began to detect more multi-state outbreaks, some of them in FDA-regulated products, her group got busier. However, her group did not receive more staff to address the increased workload associated with increased outbreak detection. By 2004, she recalled, the outbreaks “just kept going.” After a 2006 major multi-state outbreak of *E.coli* O157:H7 in spinach, she commented, “it just didn’t stop.”

According to *Food Safety Magazine*, a trade publication, the spinach outbreak “lit a rocket” under traceability efforts within the industry, helping to generate the Lettuce and Leafy Greens Marketing Agreement amongst California farmers, and the Produce Traceability Initiative (PTI) amongst over 50 companies to implement chain-wide electronic traceability. By standardizing identification of produce and storage of digital information, this would “narrow the impact of foodborne illness outbreaks and recalls,” by enabling the precise detection of exactly which units of food were contaminated. Through standardized electronic traceability, an industry white paper argued, companies could “react quickly and with surgical precision to recalls.”

While FDA officials were busy throughout the 2000s investigating outbreak after outbreak, as McGarry pointed out, a 2008 outbreak significantly shaped subsequent agency reforms in outbreak investigations and regulation of traceability. In the summer of 2008, a major multi-state outbreak of *Salmonella* Saintpaul resonated with the *Cyclospora* scenario of 1996.

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The Saintpaul outbreak was first associated with tomatoes, but later, jalapeño and serrano peppers were implicated. At the end of an exceedingly complex investigation, which vehicle it was, or whether it was both, could never be definitively disentangled, due to the many material and social complexities of food as it was prepared and eaten. For example, tomatoes were often eaten together with hot peppers in salsa and in Mexican dishes served at restaurants, and people might not recognize that hot peppers were an ingredient in the foods they were served.

McGarry, who designed the organizational model that FDA currently uses for foodborne outbreak investigations, the Coordinated Outbreak Response and Evaluation (CORE) network, pointed to the 2008 Saintpaul outbreak as heavily informing the design of CORE. The “key themes” of the network were to focus on foodborne outbreak detection, response, and post response, taking “signal detection from a systematic perspective,” and “post-response to translate lessons learned” to FDA’s “program” personnel. These themes were expressions of the agency’s broader focus on science-based, risk-based, prevention-oriented principles underlying the Food Safety Modernization Act.

I asked McGarry to discuss traceability in more detail, and some of the complexities the agency has encountered over time, with different produce items. She pointed out that the key factor was “what the web looks like for the commodity.” How many things were handled, how many people handled those things? To illustrate, she compared the complexity of traceback for a whole cantaloupe versus a diced tomato. The whole cantaloupe’s “network in the supply chain” was much smaller than the diced tomato’s. Cantaloupes have growers, packers, repackers, warehouses, distributors, and a grocery store, representing a “less complicated web.”

The diced tomato both had a more complicated web, and, importantly, a complex shelf-life, which significantly challenged the traceback. She described the tomato moving through the supply chain, starting with the whole tomato, which would ripen in transit, and as it ripened, each color would transform how the tomato was categorized at different points in the chain of distribution, for example, entering in one place as a “vine-ripe,” exiting as a “greenhouse,” and recorded at retail as a “red round bulk.”

David Acheson, then the FDA Associate Commissioner on Foods, pointed out that this “lack of consistency in nomenclature” made it more challenging and time-consuming to conduct traceback investigations and find the source. Also, because tomatoes go through such a complex supply chain, McGarry pointed out, there were “more nodes and places for things to go wrong from a records standpoint.”

The *Food Safety Magazine* article defended the industry traceback ability in the 2008 Saintpaul outbreak. It argued that the FDA could use industry trace-back information, but that the information pointed to many different farms, rather than a common source, contrasting with the CDC’s common source hypothesis. However, the article did admit proprietary limitations in the traceability system, confounded by limitations in FDA data-processing capacity:

Because chain-wide electronic traceability has yet to exist, the FDA, in attempt to follow a product through the supply chain from company to company, had to translate each produce company’s proprietary tracking system and try to make it correspond to other companies’ proprietary tracking systems. Given that the Bioterrorism Act does not mandate electronic record keeping and that FDA consequently is not equipped to process trace-back records electronically, all trace-back records were requested and submitted on paper. Horror stories exist of field staff faxing hundreds of pages of printed electronic records to FDA headquarters for someone to try to read through and connect the dots between multiple traceability systems of multiple produce companies throughout the supply chain to make sure that all the details—such as number of boxes, brand

names, lot codes and ship/receive dates—correlated exactly on invoices, bills of lading and other such documentation.

After the Saintpaul outbreak, debates about traceability reforms and control measures would continue over an extended period of time, between the produce industry, regulatory officials, and consumer groups. While the summer of 2008 had been busy with the Saintpaul outbreak, in the winter, PulseNet detected another major multi-state cluster.

**Conclusion**

This chapter examined how an early warning, real-time system for foodborne outbreak detection and response was built over time. I began by providing a brief biography of how PFGE became a resource for PulseNet to incorporate as the centerpiece bacterial typing technique. Because it had developed and stabilized outside of the public health sector, the main job of turning into a public health surveillance tool involved building a standardized system.

I chronicled PulseNet’s genesis and early development, where the aim was to turn PFGE from a confirmatory tool to a real-time tool. Swaminathan used a distributed repertoire to describe a wider context of innovation, how he drew from his work in the Meningitis and Special Pathogens Branch (MSPB). Work in the MSPB had also influenced the development of FoodNet, a sentinel site surveillance program aimed at sporadic cases. In contrast to the NSSP built in the 1960s, which sought to examine both outbreak and sporadic cases, in the 1990s, public health officials created two separate systems tailored to each problem.

Initially, Swaminathan chose an area-lab approach for the PulseNet network. However, this initial approach encountered two issues. One was social and infrastructural; other state labs did not like the disparity this created between labs, wanting to participate in the network. The
other was administrative; there was no mechanism for labs to send samples from one state to another, only from the states to the CDC. After negotiating additional funding streams, PulseNet shifted to a decentralized approach, where each state laboratory would pulse the isolates from ill citizens of its state.

Expansion of the network meant solving myriad critical problems of standardization. Achieving large-scale standardization was a challenging prospect. It would entail not only the standardization of a method, but use and interpretation of data, the creation of a shared database, and extensive coordination and rationalization of distributed work. I provided an in-depth description of how officials grappled with two critical problems related to getting to “real-time.” First, I described how they shortened protocols with the diversity of multiple state users in mind. Second, I discussed how they created a whole formalized testing system, and began to test new and complementary techniques.

Early on, public health officials thought that MLVA might replace PFGE, but they discovered the technique was too serotype-specific. So, it became a complementary method to PFGE. They also learned, through their work, that sequence-based technologies were presenting a major problem for a standardized system. While biotech developers improved their platforms through upgrades that may have seemed like small tweaks, from the perspective of the public health sector, those small tweaks had major implications, requiring them to conduct the extensive work of re-validating protocols.

While it took a few years for PulseNet to gain momentum and begin to accumulate data, as it did so, a new diffuse national outbreak began to come to the fore. New critical problems emerged along with this new outbreak. From a practical perspective, detecting more clusters
meant making changes in the organization to reflect increased workload and specialization.

Detecting multiple outbreaks in particular sectors created critical problems for regulators and industries who had to work on control efforts.

As it gained momentum, PulseNet became the center of new inter-sector communications, organized around the shared problem of the diffuse national outbreak.

Between the 1990s – 2000s, as the mass globalization of produce increased, this phenomenon began to appear as the source of big multi-state outbreaks. Dealing with this new target entailed negotiating a more coordinated, systematized epidemiology-regulatory interface, to speed up the traceability leg of the outbreak investigation. The O157 spinach outbreak and the Saintpaul raw produce outbreak demonstrated that the fragmented regulatory-industry interface was a reverse salient holding up rapid identification and removal of contaminated products from the food chain. Chain-wide, electronic traceability became a key, and ongoing critical problem, significantly reshaping the sociotechnical networks of food production, distribution, and regulation.
Chapter 7. *Salmonella* Typhimurium: Following a Social Drama, 2008-2013

Outbreaks are dramatic events, from start to finish. In his writings on epidemic investigations, Berton Roueché, the *New Yorker* journalist I mentioned in Chapter 3, aimed to capture this drama with his narrative method. “Conventional epidemiologic literature tends to be uncomfortable in the presence of drama,” he wrote.\(^1\) “Most epidemiologic reports…will typically begin: ‘This paper reports an outbreak of x cases of x centered at x in x.’” Roueché’s approach, by contrast, aimed to “reconstruct the investigation as it was actually carried out.” He wrote:

> I include the floundering up blind alleys and the stumbles down garden paths. I bring out the clinical ambiguities, the diagnostic alternatives, the evidential inconsistencies. And I withhold the source of the trouble, as it was withheld from the investigator himself, until it is discovered by the evidence.

This chapter borrows from Roueché’s method, to bring out the drama involved in outbreak investigations by following one outbreak of *Salmonella* Typhimurium from start to finish. I also use drama as an analytic by combining Roueché’s method with the processual anthropology of Victor Turner (1980), who developed a “social drama” model for describing the four “phases” through which many social crises move.

Social dramas, Turner argues, occur in social groups where people share values, interests, and history. Social dramas typically feature “star” groups, “whose fate is for us of the greatest personal concern.” Turner points out that all of us are members of many social groups, and may have different “star” groups; since who is considered a “star” group is can differ depending on who is doing the evaluation, these differences can be a source of tension in social dramas.

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\(^1\) *Annals of Epidemiology* (1967).
Turner argues that social dramas have four “processual units”: breach, crisis, redress, and either reintegration or recognition of schism. A breach involves an individual or social group’s “infraction” against a norm or rule of “morality, law, custom, or etiquette.” Breaches are followed by a “mounting crisis,” involving more conflict and antagonism between individuals and social groups; breaches have the tendency to become “contagious” and spread widely. To contain the spreading breach, redress mechanisms are usually put into place, ranging from informal management all the way to the formal “performance of public ritual.” After redress, the social group that caused the breach will either become reintegrated, or there will be social recognition that the breach between groups is irreparable and often result in social separation.

Stephen Hilgartner (2007) has used Turner’s social drama model to examine public inquiries on the aftermath of Hurricane Katrina. His take on public inquiries differs from Turner in that he points to the ways that breaches generate “contradictory potentials,” what he calls a “dynamic tension between overflow and containment that in principle can produce varying mixtures of reassurance and anxiety.”

Part of what makes the social drama concept useful for understanding the social dynamics of foodborne outbreaks is the centrality of food in social life, and thus the relevance of safety concerns to all consumers. Turner’s “star groups” concept is particularly applicable to foodborne disease, because enteric pathogens pose especial threats to the vulnerable in our society—the young, the old, the immune-compromised. As I will discuss later in the chapter, while few would refute the importance of protecting vulnerable children, because there are many constituencies in the food system with different values and social locations, some groups are occupied with the fate of different “star groups,” who are affected by whatever redress measures are adopted.
The four phases of a social drama map interestingly on to the public health concepts of surveillance and control. Outbreak investigations make “breaches” in the food chain visible (recall Tauxe’s metaphor of a tear in a covering fabric mentioned in Chapter 1), and quite literally track the “contagion” of crisis by surveilling and mapping the event of illness occurrence and determining its extent. The crisis mounts not only because the outbreak is growing, but also if the investigation is reported in the media as ongoing, the coalescing outbreak is in the public arena, and becomes a social drama even as it is still being investigated.

As the social drama heightens, more and more social groups become involved: the media, public interest groups, political bodies, and the public at large. Because outbreak investigations produce some information about the causes of breach, they produce “lessons learned” that are enrolled into the multiple processes and locations of redress, which in food safety, are largely the responsibility of regulatory agencies to craft standards that reshape the production practices that caused the breach.

Redress maps onto control. Just as I have shown that the interface between surveillance and control as it was negotiated by the CDC and FDA around PulseNet and outbreak investigation work was complex and historically contingent, I wish to make the same point about the four phases; breach, crisis, redress, and containment/overflow, overlap, bleed into each other, and exhibit patterns of feedback. In particular, if breaches are not contained with redress, the problems overflow back into breach.

While a bulk of this dissertation has shown how the confirmatory pathway involves the creation of thin descriptions, this chapter will show how thin descriptions are re-thickened as a social drama unfolds. Once anonymous cases participate in public inquiries and share their illness experiences, narrating the outbreak from their perspective. Social groups struggle over
the best means of producing safer food in the future, drawing examples from their own lives to make their claims. These struggles are not confined to a single moment, but are ongoing, as the social drama continues to unfold.

**Two Clusters or One?**

On November 10, 2008, PulseNet detected a multistate cluster of *Salmonella* Typhimurium, with 13 cases reported in 12 states sharing the same unusual PFGE pattern, JPXXO1.1818.² On November 24, PulseNet found a second multi-state cluster of *Salmonella* Typhimurium, with 27 cases reported in 14 states, with the unusual pattern JPXX01.0459. The next day, the CDC initiated a “epidemiologic assessment” of the first cluster, which by then had grown to 35 isolates, in 16 states. On December 2, the CDC began an assessment of the second cluster, which by then had grown to 41 cases from 17 states. Pattern 1818 and 0459 were “closely related,” exhibiting only a one band difference on Xba1 (the first enzyme), and indistinguishable by both the second enzyme and with MLVA. Since the patterns were related, that suggested that the clusters might be related as well.

What helped public health officials make that determination was the following spot map, created by William Keene, an epidemiologist from Oregon.³

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² Most of the detailed national investigation information comes from a Power Point I got from the CDC’s FOIA records, shared with me as discussed in Chapter 1. The narrative information comes from interviews, as designated.
³ I would like to have interviewed Keene also but he sadly passed away unexpectedly early this year.
Kirk Smith, an epidemiologist from Minnesota, distributed credit to Keene when I interviewed Smith about the outbreak investigation. Smith called the spot map “simple, yet brilliant.” He pointed out how the spot map visually illustrated how the two clusters were “probably all one outbreak.” The cases from one cluster were featured in red, from the other cluster, blue. In southern California, Massachusetts, and Rhode Island areas, the red and blue dots overlapped, suggesting that they might be one outbreak. As the CDC noted, additionally, cases in the two clusters had similar geographic and age distribution, with many cases under 18 years of age.

While the spot map helped officials see a connection and literally connect the dots, it also, Smith pointed out, helped illustrate what was not visible. At this point (December 3) there were over 100 total cases, but because most counties only perform a short food history interview

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4 Interview, Kirk Smith, 12/19/13. Smith shared a Power Point presentation with me, which contained this spot map graphic, as well as temporal anchors for other occurrences in the investigation I discuss in this chapter. Most of the information about Minnesota’s perspective came from Smith’s interview or Power Point.
with patients on a routine basis, he said, “we basically had no epi information, nothing useful at this time.” This outbreak, like others before it, Smith commented, “demonstrated how we needed to standardize outbreak investigation methods in this country.” Soon after this point, public health officials indeed merged the two cluster investigations into one outbreak investigation.

Multiple Possible Vehicles

Though at that point, Minnesota did not have many cases in the outbreak, with only 3 isolates coming in between November 17-24, because it is one of the states with the strongest foodborne surveillance programs, it was heavily involved in the investigation. On December 9, Ohio reported a restaurant cluster of Salmonella Typhimurium in its state that was “possibly linked to chicken” from a mall food court restaurant. Williams called this Ohio cluster an “interesting wrinkle,” and explained further.

Ohio’s investigation determined that the restaurant’s outbreak was definitely caused by chicken, due to poor raw chicken handling practices and other hygiene issues at the restaurant. There were three PFGE patterns associated with the outbreak, one of which matched one of the patterns in the national outbreak strain. “This made us think,” Williams narrated, “is the source of this outbreak actually chicken?” Also this opened up the question, how many patterns did the national outbreak involve? (The outbreak strain was currently comprised of two PFGE patterns). Since after Thanksgiving, more intense interviewing had been going on, many states were investigating their newly detected cases with the Standardized National Hypothesis Generating

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5 Interview, Ian Williams, 3/1/12.
Questionnaire, or open-ended interviewing, to trawl for food exposures and generate hypotheses.

Williams shared that the picture emerging from those interviews was:

> There were a lot of peanut butter eaters, a lot of processed food eaters, a lot of peanut butter… and a lot of people who said they had chicken. But lots of different types of chicken, no one piece.

Thus, the Ohio cluster seemed to point to chicken as a possible source for the multistate outbreak. While eventually, Smith said, the Ohio cluster would be determined to be a “red herring,” “at the time,” he remembered, “we heard this, and, it makes sense, chicken has *Salmonella* Typhimurium, as a matter of fact our chicken kiev outbreaks were due to *Typhimurium*.”

To support Ohio’s chicken hypothesis, Minnesota performed a traceback on chicken exposures from their state’s first four cases. However, it would turn out that none of the chicken traced back to the same source. Ohio had traced some of its chicken back to a source in Georgia. However, when FSIS, which began their traceback activities on December 12, looked at the chicken plant, they determined that the “distribution [of the suspected chicken] is such that it could not have really caused this coast-to-coast outbreak of this size.” By December 23, PFGE typing determined that none of 14 isolates from chicken samples from the Ohio restaurant matched the outbreak strain.

However, that did not mean that chicken was absolutely ruled out as a suspect vehicle. From a national perspective, the picture still seemed ambiguous, including the relationship between Ohio’s cluster and the multistate outbreak. On January 1, Ohio used the Epi-Aid mechanism to invite the CDC to collaborate on the investigation, and public health officials launched two studies, a restaurant cohort study and a multi-state case control study.
To backtrack a bit in time to capture the Minnesota perspective, December 10 - 19 was an interesting time period, because its lab received 8 more outbreak isolates. When those cases were interviewed with Minnesota’s trawling questionnaire, one of the food exposures in common was peanut butter. Minnesota found this peanut butter exposure “suspicious,” but did not see this as enough evidence to “implicate” peanut butter, or a particular product. It was possible that cases reporting peanut butter could have been a “proxy” for how much the cases enjoyed eating peanut butter. Peanut butter is a common, widely eaten food in the US. When you ask people about what they ate, they will often report what they like to eat. However, he shared, “I still remember one of my epidemiologists… said, ‘I think it’s peanut butter.’ You get a very good innate sense for these sorts of things.” Despite this hunch that it was peanut butter, however, there was not enough evidence to “prove” anything at that point.

**Big Break**

The “big break,” Smith said, came starting on December 22, when the Minnesota Department of Health was informed of a nursing home outbreak of *Salmonella*. After the lab performed PFGE on the nursing home isolates, they determined that all five cases in the nursing home outbreak matched the outbreak strain. Smith pointed out that the investigation response to the nursing home outbreak was standard for them, regardless of the multistate outbreak, he said, “*Salmonella* in nursing homes is a bad deal.” Also on December 22, Minnesota found out about a single case of *Salmonella* at a different nursing home, but from the same city as the first nursing home.
Smith offered some insight into the special challenges of doing food history interviews with nursing home residents.

Smith: We can’t really interview some of these patients, residents of the nursing home. They’re just not good historians, if they can communicate at all…
Boyce: That’s so sad!
Smith: Yeah… we looked at the institution as… the unit of observation.

In looking at the institution, investigators collected menus and food invoices from nursing home staff, as well as from an elementary school. On December 26-28, they had found 2 cases who went to the same school. The invoices from the nursing home and school indicated that all three institutions shared a common food distributor. Additionally, the only food that was common to the three institutions was “King Nut Creamy Peanut Butter.”

One of the nursing homes “happened” to still have an open tub of the peanut butter, and so the Minnesota Department of Agriculture “took a shot” at testing the peanut butter, using both rapid presumptive testing (an enzyme linked fluorescent assay) and more lengthy confirmatory testing using PFGE. Smith pointed out that his department “aggressively follows good leads,” which can be helpful for solving multi-state outbreak investigations. However, following good leads requires resources, a financial risk because it is always possible that leads could turn out to be “red herrings.” At this point in the investigation, also, he recalled, other “little bits” pointed to peanut butter: a coast-to-coast outbreak meant a widely distributed product and a long outbreak meant a long shelf-life, “ruling out” more perishable products.

Also, by January 5, the Ohio cluster had been ruled out as unrelated to the national outbreak. Williams shared how they teased out a particular ambiguity with the Ohio cluster with the restaurant cohort study, though the full “loop” was not fully explained until PCA was identified and the traceforward was conducted, which occurred later in the investigation. What was determined in the cohort study was that the onset dates of illness for the cases with one
PFGE pattern (the national outbreak strain) did not match the cases with the other patterns. Eventually, investigators would figure out that the reason that the Ohio cluster had multiple PFGE patterns was that some of the cases in the cluster not only ate at the restaurant, but had also consumed contaminated peanut butter. The Ohio town where the mall food court chicken joint was located was a college town, and some of the cases had eaten at a college cafeteria that served peanut butter produced by PCA. However, the epidemiologic data about differing onset dates with different patterns helped separate Ohio out from the national picture.

While Minnesota’s open jar was in the process of being tested by the MN Department of Agriculture’s lab scientists in the first week of January, on the 6th, another case was reported in a third nursing home in the same Minnesota city. This case, who died, had been suffering from terminal cancer, and for the weeks prior to onset of illness, had consumed an extremely limited diet, eating peanut butter and toast every day. As the interviewer hearing this story, I could not help but be emotionally affected by this case’s story; again, this was another moment of localization, a small window into an individual’s vulnerability and suffering.

Nursing home number three also served King Nut Creamy Peanut Butter. With lab testing pending, but strong epidemiologic information about this case, Smith shared that at that point, the sentiment was that the source was this peanut butter.

Going Public

When the presumptive test yielded a *Salmonella*-positive result, Minnesota decided to issue a press release, which came out on January 9. The issue received regional and state-oriented coverage, and two days later, the issue got a small amount of national news attention.
For example, the *New York Times* mentioned the outbreak in a one-paragraph “National Briefing” on page A14, labeled as an issue related to the “Midwest.” The paragraph indicated, however, that it was very early in an unfolding storyline; federal officials, the brief mentioned, had not yet identified the cause of a *Salmonella* outbreak in 42 states comprised of almost 400 people, but MN was reporting that “preliminary laboratory testing” identified *Salmonella* in a “five-pound container of King Nut brand creamy peanut butter.” The brief stated that the testing had not yet confirmed any link to the national outbreak, but that results would come in early next week.

Smith recalled, “we tried to make the wording like we didn’t necessarily know if it was because of the national outbreak.” While he said that others thought that MN was “going way out on a limb” in issuing the press release at this point, Smith asked me a rhetorical question to illustrate why they felt like that was the right moment. “Would any of us want our parents or grandparents, our loved ones in nursing homes, eating this peanut butter, knowing what we did?” Indeed, the nursing home resident with terminal cancer who ate peanut butter and toast everyday lingered in my mind, and still does, attesting to the moral power of the vulnerable image it presents.

Smith stated that Minnesota officials felt like they had “no choice” in announcing to the public not to eat King Nut peanut butter. Information continued to filter in, “strengthening” the evidence that this product was widely distributed, when five more cases came in that were related to institutions that had received King Nut peanut butter from that common distributor. The confirmatory PFGE testing results came in on January 12. Even then, however, the states continued their pursuit of food testing, especially on closed containers. Smith pointed out that a positive test from an open container was “less than ideal,” because nursing home staff could
theoretically have cross-contaminated the product. Yet counter-balancing this information was the fact that the sample from the open container had been taken from “subsamples deeper in the peanut butter” (where food handlers were less likely to have touched), and the samples yielded both PFGE patterns that were defined as the national outbreak strain.

On January 12 also, the first results from the national case-control study were calculated. 69% of case-patients ate peanut butter in the 7 days before their illness began, versus 48% of controls. No single national brand of peanut butter was coming to the fore. “Previously frozen chicken products” were also significantly associated with the cases, and like peanut butter, no individual product stood out. The results of the case-control study were ambiguous. Williams remembered that he had not been involved in the decision to initiate the study because he had been away during Christmas. He pointed out that while in late December the hypothesis had not yet “crystallized,” providing one rationale for initiating a case-control study, his own view was that monitoring incoming information was better than initiating a case-control study, for the purposes of crystallizing hypotheses.7

On January 13, USA Today reported that on the previous day, the MN Department of Health confirmed that the Salmonella from the King Nut jar matched the national outbreak strain. On January 16, Connecticut announced that it had found the outbreak strain in a sample from a closed tub of King Nut peanut butter, what many investigators would refer to as an example of a “smoking gun.” Yet this was not the end of the story, as the whole picture was not yet explained by King Nut peanut butter.

Before narrating more twists and turns in the outbreak investigation, I wish to briefly to point out that there is an interesting dynamic going on at this point because the investigation is

6 Matched odds ratio = 2.53, p=0.007.
7 A view he would continue to institutionalize after this outbreak, in his role as ORPB chief.
still ongoing, but the public inquiry has also begun in the media. This inevitably intensifies the “fast but right” dilemma for investigators, as they become more accountable to the public for solving an ongoing outbreak. Something that is out there is still making people sick. When “threats to social order” enter the public arena they demand a “social accounting,” as Stephen Hilgartner (2000) discusses in his dramaturgical study of science advice disputes in the diet/health domain.

While it raises the stakes of a situation to issue public warnings, as Minnesota’s accounting of their rationale for announcing demonstrates, only some of the information about the episode was fully confirmed at the time of public announcement. There was more provisional knowledge being created and moving through the confirmatory pathway. Though King Nut creamy peanut butter may or may not have explained all of the cases in the multistate outbreak, it explained Minnesota’s to the satisfaction of public health officials, who had the authority and felt the ethical imperative to make the outbreak (at least their piece of it) public.

What could also be playing a role is that, as Theresa MacPhail (2010) found, public health authorities who communicated uncertainty during the 2009 H1N1 pandemic could use uncertainty as an effective tool to retain scientific authority, because it signaled honesty and transparency in organizational risk communication. Sometimes being fast to communicate combines well with being right by admitting uncertainty. The flip side of announcing an ongoing investigation and only partial information, however, is that it could also raise questions about whether information was being confirmed fast enough, or if information was confirmed but not being shared with the public (e.g. the 1960s Salmonella Derby hospital egg epidemic). While communicating uncertainty can be a laudable organizational tactic, it is never a surefire one.
A Shift in Strategy

Minnesota’s identification of King Nut peanut butter triggered “traceback” of the product, which many pointed out to me as extremely simple in contrast to traceback in other investigations. As Guzewich pointed out, peanut butter was a packaged food, with a container that had a clear label on it. The traceback consisted of calling the King Nut company and finding out who manufactured their peanut butter, as King Nut was a distributor and not a manufacturer of peanut butter. The phone call revealed that King Nut peanut butter was manufactured by PCA’s plant in Blakely, Georgia. On January 9, the FDA began its investigation of the PCA plant, and on January 10, the King Nut peanut butter company issued a voluntary recall of its product, to help redress the outbreak. However, this redress measure would not stem the crisis, as the ultimate cause of the episode was much broader than King Nut; at the time, just how broad the problem was, was under investigation.

Before discussing the regulatory “leg” of the investigation, I would like to continue the epidemiologic thread. Because Minnesota had identified an institutional peanut butter (King Nut), that led the CDC and other states to focus more attention on trying to find other institutional clusters, for example, calling hospitals with clusters of cases and asking staff whether they served King Nut peanut butter. Williams commented that “asking the right question” yielded “amazing results,” when institutional clusters, one after one, showed associations with King Nut peanut butter.

Williams pointed to this institutional cluster strategy as being a major change for the CDC in terms of epidemiologic method, a “fundamental transformation” over the last four or

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8 Interview, John Guzewich, 12/16/13.
five years in the way they conducted outbreak investigations. The CDC had been investigating institutional clusters for years, but, according to Williams, they were “viewed very much in an epidemiologic perspective.” He explained this perspective using a hypothetical restaurant cluster. To identify the food that made people sick in the restaurant, the investigation would involve interviewing ill people at the restaurant as well as “meal companions” and other diners, and taking food histories for each group. Williams pointed out that this strategy was good if there were enough cases at that restaurant, however, “enough people” usually meant 15 or 20, while many PFGE clusters consisted of 2 or 3 cases.

“In retrospect,” Williams observed, the Salmonella Saintpaul outbreak reflected this prior “attitude” towards methods for institutional clusters. He recalled conducting extensive investigational activities such as getting receipts, names and phone numbers of restaurant patrons, and even, if the restaurant took reservations, to get a list of names of patrons who made reservations. Additionally, he recalled, they would ask questions about and construct case definitions around not just PFGE patterns, but extended clinical symptoms.

One of the sources the CDC used to navigate and implement a shift in strategy was to look to a technique Minnesota had developed.Williams recounted how Minnesota had created a strategy to leverage the small PFGE clusters and work around the smaller case counts in clusters. Minnesota would examine the food served in restaurant clusters, identify common foods, interview cases and ask them if they ate those foods. However, Williams pointed out, this was not statistical, “you can’t generate a p-value for that.” Thus, what they lost in statistical objectivity Minnesota supplemented with a tighter working relationship between and integration of evidence from the “three legs” of an outbreak investigation—epidemiology, laboratory, and traceback. By tacking back and forth between the three types of evidence, officials could move
from the traceback and “leapfrog to the product testing.” Williams characterized Minnesota’s strategy for finding the King Nut peanut butter in the following way:

So their approach was, we think it’s the peanut butter, the way we’re going to prove it is, I’m going to round up some of this peanut butter and test the hell out of it. And I’ll be smart about looking at what I’m going to test, I’m not testing any peanut butter, if I’m looking at peanut butter that’s served at these places at this time, I’m going to get lucky and find it.

Williams’ narration makes clear that part of navigating a shift in strategy around institutional clusters was related to navigating a shift in methodological credibility, from a statistically-grounded practice to one coupling an extension of epidemiologic reasoning and increased reliance on “smart” product testing and the ultimate authority of laboratory evidence. I argue that we can represent this shift as the epidemiologic triad turning into an epidemiologic “tetrad” of person, place, time, and thing, the latter representing the integration of product distribution records as a resource for sensemaking during the investigation, on par and used in conjunction with other forms of evidence.

However, the enactment of this shift, Williams emphasized, did not change underlying jurisdictional authority, but rather, it modified organizational practices of coordination. We discussed the issue:

Boyce: It makes sense to me, CDC’s old approach, where you have a lot of credibility, legitimacy, and know-how in talking to people and doing statistics. And so distribution records as a new source of epidemiological data, it needs new methods, that makes sense too…
Williams: It’s out of our control, is the other thing.
Boyce: That’s what I was going to say, exactly… they’re harder to get, right?
Williams: It’s not something that public health people do. We do not access and collect records, that’s the FDA, FSIS, state and locals. Part of it is, the analogy that was used, Morrie Potter’s way, in the old days, the way outbreak

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9 Examples of smart product testing: Using product information to find a lot code, using the lot code to find production windows, testing product manufactured in that time window. You can also test leftover product from a case patient’s home, if some is still available in their fridge or in their cupboards.
investigations were done, the public health people lit the fuse… and basically, when it got one inch away from the bomb going off, they handed it to the regulatory people and said, it’s peanut butter, good luck with that, boom!

By contrast, in recent years, increased inter-agency coordination entailed bringing regulatory officials “further ahead,” to give them more access to the “information as it develops.” Williams concretized the contrast between past and present by saying, previously the hand-off entailed passing on a statistically significant association between food and human illness, whereas in the present, the exchange was represented by the statement, “I think it could be peanut butter, and here’s why.” As I pointed out in Chapter 2, linguistic hedging is part of uncertainty management and sensemaking, and here it is a signal for the tightening of the public health/regulatory interface that this shift entailed.

A New Exposure

The growing amplification of the outbreak into crisis was heavily tied into untangling the widespread distribution of the underlying contaminated product, and both the regulatory and epidemiologic legs were seeing the underlying pattern manifest itself in different ways. Because of the increased focus on local institutional clusters, and heightened efforts in the states, another significant exposure of concern coalesced.

Prior to this outbreak, the CDC and the two regulatory agencies had been discussing mechanisms to increase overlap of their investigatory work, so that the CDC could “lean forward” into the traceforward and traceback process, and the regulatory agencies could “lean backward” into the illness investigation. Because the Blakely plant was only three hours from Atlanta, Williams shared, this outbreak gave him the opportunity to participate in the facility
investigation. The low cost of the travel (a three-hour drive) could be more easily justified, again demonstrating how cost is a huge shaper of investigatory practices in the confirmatory pathway. This was a major administrative contingency associated with the investigation.

When Williams arrived at the plant on January 10, one of the first things that he did was to share information about the current status of the outbreak investigation with the FDA team (comprised of investigators from the region), as well as with some of the plant’s personnel, who did not have the epidemiologic data and thus did not understand the full scope of the multistate outbreak being a national crisis. He stayed for a couple of days, and during that time, the new exposure coalesced.

Federal and state public health officials held a multi-state conference call on January 12 to discuss the ongoing investigation. At the time, though King Nut peanut butter did not seem to explain all the cases, another specific peanut butter hypothesis had not yet crystallized. Elizabeth Daly, an epidemiologist from New Hampshire, described the coalescing of the new significant exposure of concern.\textsuperscript{10} In her state, county public health nurses interview all of their \textit{Salmonella} cases with routine interviews, and when Daly was reviewing the interview data, she noticed that some of the nurses had happened to ask patients to list items they had eaten, beyond what was listed in the questionnaire. Some nurses wrote “Austin peanut butter crackers” on the sheets, or at least, “peanut butter crackers.”

Daly said that the nurses had not been prompted to ask cases about crackers. Instead, the nurses simply asked the patients whether they had eaten anything unusual. Daly noted that normally, nurses did not take extra notes on questionnaires, so that in itself was “kind of unusual.” On the January 12 conference call, Daly mentioned that two or three cases had mentioned a peanut butter cracker exposure. Then, other states began to chime in, saying that

\textsuperscript{10} Interview, Elizabeth Daly, 4/16/2014.
their cases had also mentioned peanut butter crackers. Interestingly, Daly remembered that she had felt slightly “sheepish” bringing up the peanut butter crackers, that states do not want to bring up irrelevant information. What turned out to be a very significant new exposure had come up in a “fluke-y” manner.

Williams recounted the process of making the link between the cracker exposure coming from case interviews reported on the conference call, and PCA’s manufacturing process, while having dinner with the investigators in the evening and discussing how PCA made and distributed its paste. The investigators explained PCA’s paste manufacturing process to Williams, in particular, the grinding and distribution process. Williams pulled up a picture on a Power Point slide to help explain this process. The slide displayed a bullet-shaped silver truck.

“This is a giant caulk gun,” he analogized, a tanker truck pressure-loaded with PCA’s peanut paste to carry to manufacturers who would use the paste as an ingredient for their peanut-flavored snack foods. Williams asked the investigators to name who PCA supplied its paste to, and the investigators offered that Kellogg’s was one of PCA’s major customers. “What???” Williams punctuated his narrative with, as the dots were connected in that moment; I laughed in surprise at this turn of events.

After he had this conversation with investigators, Williams called Barton-Behravesh on the phone that night. She immediately sent an email to the two states, New Hampshire and Michigan, who said they were testing packages of Austin crackers from a case household. Barton-Behravesh asked them to share details about the flavor of crackers, lot number, UPC code, best-by-date, and where purchased, on a follow-up call scheduled for the next day.

The “nail in the coffin” for the cracker exposure, Guzewich shared, was that Canada had a case in the outbreak. When Canadian officials investigated whether their case had eaten these
crackers, they found out that the case would drive across the border to a warehouse “buyers club” store in the US, purchase the crackers in bulk, bring them home to Canada, and consume them there.

The states continued to investigate, conducting follow-up interviews with cases to ask them about peanut butter crackers. Two brands came up in the interviews—Austin and Keebler—both manufactured by the parent company Kellogg’s. FDA officials began to investigate the North Carolina plant that manufactured Keebler’s peanut butter crackers, which revealed that the plant purchased a peanut paste ingredient from PCA. “When we realized now we didn’t just have people sick from peanut butter,” Guzewich narrated, “but people sick from peanut paste, that suggested that many products were coming out of a plant that could have contained Salmonella.”

**Traceforward “Nightmare”**

The paste discovery started what Guzewich called a “nightmare” of a traceforward process, to identify all the products that used the contaminated peanut paste ingredient. Before discussing that process, I wish to point out that the plant investigations triggered a quick sequence of events heightening the crisis and leading to further press updates on the investigation. On January 14, Kellogg placed a hold on its cracker products; on January 16, CT identified the outbreak strain in the closed jar, PCA announced a voluntary recall of its peanut butter and paste, and Kellogg voluntarily recalled its peanut butter crackers and a few other suspected products. On January 17, the media publicized some of these investigational developments. For example, the Atlanta Journal-Constitution reported that federal health
officials were “expanding” the outbreak investigation to include “peanut butter products that may have landed on store shelves,” and advised the public to “use caution in choosing products made with peanut butter.” The advisory was broad, because officials “said they could not identify products consumers should avoid at this time,” though the article also reported PCA and Kellogg’s recalls. Inside the confirmatory pathway, while there was still much provisional knowledge yet to be fully confirmed and more being gathered, the weight of evidence unquestionably pointed to the fact that there was an as-yet-unquantified-but-massive amount of contaminated peanut butter paste-containing products on store shelves presenting potential Salmonella risk to consumers. Peanut butter, with its long-shelf life, added to the difficulty of finding contaminated product not only in stores but in home kitchens.

While at PCA, investigators had encountered challenges in securing cooperation from the plant and access to its records to help determine where its peanut paste had traveled, and the FDA ended up invoking the 2002 Bioterrorism Act to compel the company to share its records. Guzewich saw these challenges as associated with issues around the plant personnel’s competence as well as some stonewalling of the investigation, which would come up later in the public arena during a Congressional hearing on the outbreak. However, the commercial challenges around the records had only just begun. Once an initial PCA customer was identified, the FDA had to work through its network of District offices (field investigators) to speak to that customer and determine what had been done with the contaminated ingredient, and whether it had traveled elsewhere. Guzewich emphasized that this involved not simply picking up the phone, but an FDA investigator going to each bakery, distributor, and store, to follow the contaminated ingredient, often through multiple steps in long distribution chains with many middlemen, all across the country. “It just kept rippling and rippling, over and over,” until
nearly all of the FDA’s district offices were involved in the traceforward investigation. The complexity of the peanut distribution chain is captured by the following FDA diagram:

Figure 23. PCA Distribution Chain

Traceforward was not just complicated from an investigational perspective, but from a recall perspective as well. Each entity needed to recall the product they made with the contaminated peanut ingredient, and this process involved varying amounts of negotiation between the commercial entity and the agency. Some commercial entities argued that their processing involved heat-treatment, but unless the entity used a “validated” heat-treatment process, the food could not be considered “microbiologically safe” by FDA definition. Guzewich argued that it

11 Originally from FDA.gov, no longer online.
was not widely understood just how heat-resistant *Salmonella* was, and that it could survive even in low-moisture foods. Over a period of several weeks, extending through January, investigation of the company, traceforward, and recall, which were all intricately related, became more and more complex. Investigators discovered that PCA had another peanut plant in Texas, which was unregistered with the FDA, increasing the amount of traceforward work.

More and more peanut-flavored products became associated with the outbreak, an array of snack foods, ice cream, and even pet food, expanding the recall significantly, and while early press coverage had focused on the ongoing status of the outbreak investigation, as the crisis snowballed, the media began to question whether the government was doing its job properly. The social drama was becoming more and more dramatic.

In a media teleconference held on January 28th, a journalist from *CNN* asked officials pointedly, “Some food safety experts blame the FDA and CDC for not properly regulating this plant and other plants that have had problems… do you take any responsibility for allowing this outbreak to happen? Do you think that the regulatory procedures have not protected the public?”

Embedded in this journalist’s question were bioconstitutional ideas about the idea that the state is expected to protect vulnerable citizens from a complex and risky food system.

The media examined all the information they could find about the situation, casting a wide net. Anything potentially related to PCA and the possible risk that peanut butter posed was of interest; for example, reporters broke a story based on FDA inspection records from April 2008, when Canada refused a shipment of PCA’s product because it was found to contain metal shavings. If not for the PCA outbreak and the intense scrutiny of the situation, these metal shavings would not have become newsworthy.

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Furthermore, the media pressed officials for as much information as possible, and also complained about the way that the FDA was disseminating its recall information to the public. For example, during the teleconference, the CNN journalist continued:

“… you don’t know or you’re not making it known what other companies are out there that manufacture these products. I mean not everybody goes to the FDA website to get this kind of information. Supermarkets – you say a lot of the PCA stuff isn’t sold in supermarkets, but it’s a lot harder to walk down the cookie aisle and sort out which and which isn’t sold including the PCA products. So is there a simpler way to do it or are you trying to protect the industry?”

The FDA and CDC emphasized that they had legal reasons for not revealing certain kinds of information, especially regarding specific details about the Blakely plant, as well as the names of all of the companies and products implicated in the recall. The FDA responded by emphasizing that the agency was “trying to be [as] accurate as possible,” and that they were “urging” people not to consume any product that may contain peanuts until “better information” was available.

As regulatory officials dug into PCA records in January, the investigation shifted from an “assessment,” to a mode that had more serious legal ramifications—a criminal investigation of the company, conducted jointly by the FDA and the Department of Justice, which restricted the flow of information out to the public as officials built their criminal case. On January 30, Reuters reported that officials had found evidence that the company had “knowingly shipped salmonella-tainted products.”13 The same article quoted Representative Bart Stupak (M), chairman of the House Energy and Commerce oversight subcommittee, as announcing that his subcommittee would hold a hearing on February 11 to seek answers from both the company and the FDA. On February 1, President Obama appeared on the NBC Today show to talk about the

13 http://mobile.reuters.com/article/healthNews/idUSTRE50Q6RC20090130
PCA outbreak and assure the public that the government was working to secure the safety of the food supply. He said:

…at bare minimum, we should be able to count on our government keeping our kids safe when they eat peanut butter ... that's what Sasha eats for — for lunch — probably three times a week. And, you know, I don't want to have to worry about whether she's going to get sick as a consequence to having her lunch.14

Public health and regulatory officials tried to contain the crisis and limit consumer inferences that all peanut butter was dangerous, saying on their websites and to journalists that there was no evidence that major brands of peanut butter were implicated in the outbreak.

However, as the New York Times reported on February 6, in a story with the headline “Fallout Widens as Buyers Shun Peanut Butter,” consumers were “disregarding the fine print” of agency websites and other consumer communications, and “swearing off all brands of peanut butter,” resulting in reduction of sales of national brands of jarred peanut butter by almost 25 percent. The J.M Smucker company (maker of Jif) and ConAgra Foods (maker of Peter Pan), ran advertisements in newspapers nationwide telling consumers that their companies did not buy PCA peanuts, and included coupons in the ads to incentivize customers to buy their peanut butter again. For the major national brands, their crisis was a public relations/financial crisis, stemming from the fact that the ever-expanding recall made consumers wary of all things peanut butter, despite public health and regulatory officials, as well as manufacturers, saying otherwise.

The community of Blakely, a center of peanut production, also experienced the situation as a local public relations/financial crisis. As one community member said to the New York Times, “We felt sort of maligned, like the media was lumping all peanuts together…one bad company does not a bad industry make.” The Early County News complained that PCA was not

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14 NBC TODAY show, February 1, 2009.
even locally owned, illustrating Turner’s point about schism and social separation. After the outbreak was over, to help repair the breach that it created for their community, Blakely organized a festival, “Peanut Proud,” which celebrated the making and eating of peanut products, and the role that peanuts played as the economic “lifeblood” of Southern Georgia. The PCA outbreak had localized and surfaced a regional economic infrastructure of peanut production.

To shift back to the crisis as it was ongoing, however, as the recall expanded and continued during February, the FDA ran into another problem. “Under the law,” Guzewich informed me, “recalls are supposed to be effected by the company.” It is the responsibility of companies whose products are contaminated to contact all of their “customers” to whom they supply their product to, since this information is typically classified as confidential commercial information, and also this places the onus on companies to conduct and fund the recall efforts (from a communications perspective and also in regards to removing contaminated product from commerce). As traceforward of the product kept snowballing, as the investigation added an additional layer (from regular outbreak investigation to a criminal investigation) and as the recall expanded more and more, PCA filed for Chapter 7 bankruptcy on February 13. The filing shows that the company had 98 creditors seeking claims against PCA, most of whom were either its customers who were seeking funds for their own recall efforts and lost sales, or Salmonella victims injured by PCA-associated products.

One of the many complications from PCA’s declaring bankruptcy was that the FDA had to take over responsibility for contacting PCA’s customers, a role it does not typically assume. Typically, after a company conducts a recall, the FDA field investigators do “recall effectiveness

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checks,” which Guzewich explained as spot checks on whether the contaminated product had been removed from commerce, because visiting every single location would be too onerous and expensive. One of the biggest challenges for the agency with this wrinkle, Guzewich recalled, was interpreting PCA’s “nebulous” records to actually identify who PCA’s customers were and take over customer contact. This investigational traceback and recall work continued for weeks more. As this extended on and on, Congressional oversight hearings, especially the House hearing on February 11, would attempt to redress the outbreak in a performance of public inquiry and political theater.

Public Inquiry

The overall structure of all House of Representatives hearings is codified in the House Rules, although each hearing has its own subcommittee dynamics. Congressional hearings are an important regulatory mechanism for the oversight of federal agencies by the legislative branch of the government to operationalize and enact democracy (Aberbach 1990). It was developed as a procedural mechanism for agency rulemaking to legitimate the delegation of power from the legislative branch to federal agencies, in keeping with the nondelegation doctrine of the Constitution, which bars excessive delegation of congressional power (Dripps 1988). The February 11 hearing’s title was “The Salmonella Outbreak: The Continued Failure to Protect the Food Supply.” Its overarching goal as defined by the House website was to “examine the recent

16 House of Representatives, 111th Congress. Hearing on “the salmonella outbreak: the continued failure to protect the food supply,” Subcommittee on Oversight and Investigation of the Committee on Energy and Commerce, HIF042.020, February 11, 2009.
salmonella outbreak associated with peanut products manufactured by the Peanut Corporation of America.”

The proceeding was highly stylized and scripted, as a public tribunal. As a public ritual, it involved the heavy use of symbolism. Stupak, as head of the subcommittee, ran the hearing, and read an opening statement about the salmonella outbreak. Each Representative on the committee, as well as other interested members of the House, prepared statements and/or a series of questions for different witnesses. Relevant documents were assembled and used as evidence during the hearing, such as inspection reports, incriminating emails by PCA executives, and the results of food testing. Some witnesses, such as afflicted family members, also gave testimony. Other witnesses were invited (agency officials and consumer advocate leaders), and two PCA executives had compelled by law through subpoena power to attend (the CEO and a senior manager). The latter group brought legal counsel with them to advise them on how to behave and what to say, and what not to say, at the hearing.

The overall framing of the hearing was a question as to whether this outbreak represented a larger problem in the food safety system, the instance of a “continued failure to protect the food supply.” However, in constructing the outbreak as an event of social drama, hearing attendees used different frames to identify what kind of activity the hearing was.

One frame for the hearing was centered around peanut butter and the domestic setting in which it normally lived as a taken-for-granted safe object, not a risk object. As Representative Jan Schakowsky (D-IL) extemporized, “Peanut butter. Peanut butter. Is there a kitchen that doesn’t have peanut butter, is there a lunch box that doesn’t have peanut butter sandwiches at some point? It is actually more American than apple pie.” That a food so symbolically representative of everyday American life (surpassing even apple pie!) was the cause of illness

and even death in the most vulnerable of citizens was repeatedly invoked as a moral standard that should not have been breached, that it violated what it meant to be an American to do so. Sally Moore & Barbara Myerhoff (1977) call ritual “a declaration of form against indeterminacy, therefore indeterminacy is always present in the background of any analysis of ritual.” The indeterminacy demonstrated by the complexity of peanut butter products distributed widely in the food chain was redressed by invoking the simple image of a peanut butter sandwich in a lunch box, as an American icon.

Another frame for the hearing was created by Representative Greg Walden, who made some remarks addressing the PCA CEO, during the initial portion of the hearing:

I wonder if Mr. Parnell is in the audience. Is Mr. Parnell in the audience? You know, I would think that the least he could have done was be here to hear your comments and to hear about your loved ones, like a victim impact panel, because that is really what this is today.

Walden’s frame for the hearing was that the proceeding was a victim impact panel, to create a forum for victims to share their illness narratives. The victims who testified tended to be representatives who spoke on behalf of their loved ones, the adult children of elderly cases, or parents of sickened children. That victims could not speak for themselves emphasized their vulnerability, as the “star groups” occupying societal concern. Jeffrey Almer, son of Shirley Almer, testified that “cancer couldn’t claim her, but peanut butter did.” Lou Toussignant, son of Clifford Toussignant, testified that his father had survived service in the Korean War, but “died because he ate peanut butter.” While during the outbreak investigation, Almer and Toussignant had been treated as anonymized cases, during the Congressional hearing, they were personified with compelling personal narratives; the adult children contrasted the seriousness of cancer and
war as threats to life with the contradiction that it was peanut butter, a food that should be safe, claiming the lives of their elderly parents that had survived much worse.

One parent of a sickened child testified about his son’s illness experience, using the brute facts of the outbreak investigation to weave a policy tragedy. Peter Hurley, whose 3-year-old son, Jacob, was a case in the outbreak, came to the hearing to tell “our story of how the Peanut Corporation of America poisoned our son.” Hurley used the fact that his son and PCA’s product were “genetically linked” (an epidemiologic brute fact) as a narrative starting point, and in doing so, adopted the (intentional) language of poison. He told legislators that he hoped their story would incite them to “take action to protect our food supply.” As he described how sick his son got, detailing some of his symptoms, and how he heard about the diagnosis of Salmonella, he asked a rhetorical question that emphasized how painful uncertainty was at the time: “What had we unknowingly given him that had given him Salmonella poisoning?” He then went on to narrate the awful and ironic implications of not knowing the food that had sickened his son:

As Jacob’s diarrhea continued, my wife was given the okay from our pediatrician’s office for Jacob to eat his favorite comfort food, Austin toasty crackers with peanut butter, the very food that we later found was the cause of his poisoning, so here we have a boy who is trying to get over food poisoning and one of the foods that was seen safe even to the people in the pediatric medical community is the exact product that is continuing to poison him. A week later, Dr. Bill Keene from Oregon's Office of Disease Prevention and Epidemiology came to our house at 5:00 on a Saturday night. As a friend said, this is like having the head of the FBI coming out to take fingerprints. On that Saturday night, Dr. Keene took custody of our supply of Austin toasty crackers with peanut butter manufactured by Kellogg's with a PCA product. One week later, Dr. Keene called us to say that Jacob and the crackers he had taken from our house had an exact DNA subtype match for Salmonella. Three out of the six packages of crackers he tested were positive, and that was all that we had left. The issue was no longer what had we done unknowingly but what had PCA done knowingly.
The ironic twist to the story was that the food that Jacob still wanted to eat while he was sick was the food that had caused his illness in the first place. This irony gave more weight Hurley’s argument that PCA wrongly acted *knowingly*, when consumers had to live with uncertainty. Hurley pointed out that PCA knew that it was shipping out tainted peanut products, but did not disclose and act on its *Salmonella*-positive testing results. Based on this family crisis, he made policy recommendations that the food safety system be improved with more rapid medical response, and increased FDA authority to halt food production and “criminally prosecute quickly and effectively.”

Indeed, another key frame for the hearing was as criminal proceeding for the executives who had knowingly poisoned children and nursing home residents with contaminated peanut butter. PCA CEO Stewart Parnell and senior plant manager Sammy Lightsey were not present during the victim statements. About an hour into the hearing, the two executives were escorted into the room, accompanied by their legal counsel. Stupak began to ask Parnell a series of questions, and Parnell refused to answer all of them, at times stumbling over the following pre-prepared, ritualistic statement: “Mr. Chairman and members of the committee, on the advice of my counsel, I respectfully decline to answer questions based on the protection afforded me under the United States Constitution.”

Given that the two executives were silent in the hearing, save for the repetition of their pre-prepared statements asserting their Fifth Amendment rights, Congressional Representatives created two “moments of high drama” (Scannell 1995) to assist them in questioning the two executives. One moment was when Stupak gave the floor to Walden to ask the executives a question. Walden held up a jar filled with contaminated peanut products and wrapped in yellow
caution tape, suggesting that the jar was a crime scene. As he held up the jar, he asked the two PCA executives:

   Mr. Parnell, Mr. Lightsey, let me just cut to the chase then. In this container are products that have your ingredients in them, some of which were on the recall list, some of which are probably contaminated. It seems like from what we read you are willing to send out that peanut base with these ingredients, and I just wonder, would either of you be willing to take the lid off and eat any of these products now like the people on the panel ahead of you, their relatives, their loved ones did?

Parnell repeated his statement, and Lightsey recited a similar, but slightly different one, saying, “I exercise my rights under the Fifth Amendment of the Constitution.”

During the second moment of high drama, at a time when the executives were absent, multiple legislators excoriated the executives’ actions, reading aloud passages from incriminating email exchanges exchanged between Parnell and Lightsey that were also displayed on two large screens in the hearing room, visually emphasizing proof of the CEO’s malfeasance.18 Using e-mail exchanges Parnell had made with other members of the company as well as a third-party safety auditing company, committee members turned Parnell’s words against him. The following email was displayed on the screen:

   Lightsey: We received final lab results from Deibel this morning and we have a positive for salmonella.
   Parnell: We need to discuss this… the time lapse, besides the cost is costing us huge $$$$$$ and causing obviously a huge lapse in time from the time we pick up peanuts until the time we can invoice.

Stupak called these emails “very disturbing,” positioning them as evidence that “this company cared more about its financial bottom line than it did about the safety of its customers.” It is a basic fact of capitalism that time is money, but it is interesting to see that Parnell’s bald statement of this point while being faced with a *Salmonella*-positive made for such powerful

18 See Lynch & Bogen (1996) on “proof notes.”
evidence that could be converted into a moment of high drama. These passages were displayed to demonstrate that Parnell had known about the presence of *Salmonella* in his products due to ongoing test results, and that he had sent products into commerce despite their contamination because of his profit motive.

While from a symbolic perspective this hearing was akin to a criminal trial, in procedural terms it was not. A federal criminal investigation was ongoing, and it would be many months before it concluded. The House hearing’s focus on proving Parnell’s criminal guilt can be read using Harold Garfinkel’s concept of the status degradation ceremony (1956), designed to “effect the ritual destruction of the person denounced.” Legislators and consumers “totally” identified Parnell as a greedy, money-hungry CEO; their denunciation through testimony effected Parnell’s movement into a lower order, especially since, in asserting his right not to self-incriminate in order to remain silent, only added to the widespread sentiment that he was guilty.

These moments of high drama, which were two of the most featured in print and television coverage of the hearing as “news icons” (Bennett and Lawrence 1995). Most viewers likely learned about the hearing from print and TV news coverage; while I do not have audience data, I will suggest that only a small audience watched the lengthy proceedings on C-Span. Instead, these two news icons could rapidly visually and symbolically communicate in a potent image to a broad audience that the government was doing something about the problem. As Stephen Hilgartner (2007) points out, “public inquiries serve as a device for managing the disorder and discord that disasters produce.”

Interestingly, the fact that the hearing considered a broad question about whether the PCA outbreak was evidence that the food safety system needed major reform, and that a specific party was a bad actor, did not seem to be in tension here, though the former was systemic and the
latter was individualistic. I argue that this is because the food system itself was framed as a vulnerable entity, and policy reforms needed to address how to protect the rest of the system against bad actors when their negligent actions could ramify throughout the supply chain, as they did with this outbreak. Because the hearing was lengthy, a number of policy reforms were suggested, and in their volume, added weight to the argument that the food safety system needed major repair, through the single act of Congress passing an omnibus bill to, as Representative Burgess put it, “modernize the FDA by giving them the money and the power they need to continue to protect our citizens.”

**Redress, Containment, Overflow**

Even after the event itself ended, the social drama of the PCA outbreak continued to unfold in three ways: major policy reform, another major peanut butter outbreak, and criminal prosecution of PCA officials. This section will briefly examine each, discussing how each domain continued to express the dynamic tension between containment and overflow.

While other major multi-state outbreaks also helped contribute to the notion that the US food safety system needed major reforms, the PCA outbreak was the most impactful. It had attracted an enormous amount of public and policy attention, deeply perturbing the mundane spaces of the supermarket and the lunchbox. In December of 2010, the House and the Senate passed the omnibus bill, the FDA Food Safety Modernization Act (FSMA), and in January 2011, Obama signed it into law.

The outbreak itself was of course not a sufficient cause of policy change. But it would be significant ammunition for the consumer advocates and lawmakers who had been working
towards food safety policy reform for years. It would be another dissertation in itself to examine this element of the story. But as a gloss, I quote some broad explanatory remarks from the keynote of William Marler, who had been heavily involved in lobbying for the passage of FSMA, at an October 2011 conference on Employee Rights and FSMA.\textsuperscript{19} He said:

\begin{quote}
Even though food safety has been talked about for decades, the Food and Drug Act hasn’t been revised for seventy-five to eighty years. But yet, we had a series of tragedies and difficult outbreaks that sickened hundreds and killed people—all coming together at the same time when Congress flipped parties. And where you’d never get hearings before, hearings started happening; things started moving and people started joining the fray—victims who had no political experience began taking dozens of trips to Washington D.C. in support of Nancy and Carolyn and the other people who pushed for food safety.\textsuperscript{20}
\end{quote}

Marler pointed out that PCA was one of several multi-state outbreaks that had associated public hearings, and while these outbreaks did not determine political action, they played an important shaping role in the passage of FSMA, with other elements of the political/advocacy environment in place. However, passing the law was only one step in a lengthy and contentious regulatory process that is still continuing to unfold, involving the writing of rules, notice-and-comment correspondence between the FDA and its publics, and the implementation phase for rules.

The PCA outbreak was not the first major multi-state outbreak in peanut butter, and it would not be the last. During 2007, a \textit{Salmonella} Tennessee outbreak sickened over 400 people from 44 states, resulting in a recall of Peter Pan peanut butter.\textsuperscript{21} In 2012, a \textit{Salmonella} breedeny outbreak resulted in an expanding recall, echoing PCA dynamics on a smaller scale. At first, Trader Joe’s recalled its Salted Valencia Peanut Butter; Sunland, Inc., the company which

\textsuperscript{19} Marler was a key litigator representing O157 victims in the Jack-in-the-Box outbreak, and has since remained as a prominent foodborne illness attorney and food safety policy advocate, who has also created and funded an online news outlet focused on food safety, \textit{Food Safety News}.

\textsuperscript{20} Nancy Donley and Carolyn Smith DeWaal are both consumer advocates; Donley was previously involved in STOP (is now a professor who researches food safety) and DeWaal is at the Center for Science in the Public Interest.

\textsuperscript{21} http://www.cdc.gov/ncidod/dbmd/diseaseinfo/salmonellosis_2007/outbreak_notice.htm
manufactured the product also manufactured a number of nut-based spreads for other companies. Commenting on the Sunland recall, Sandra Eskin, project director of the food safety campaign at the Pew Health Group, called peanut butter the “poster child” exemplifying the need for “prevention-based safety standards.” The lengthy process of changing the US food system was still ongoing.

In February 2013, it became public knowledge that federal prosecutors filed criminal charges against PCA executives. Parnell was charged with criminal fraud and conspiracy. Prosecutors, the New York Times reported, accused Parnell of developing a “scheme to ship peanut products known to be contaminated to customers in states across the country.” The trial is set for July 14, 2014. In the legal domain, the social drama of the PCA outbreak continues.

Conclusion

This chapter followed one outbreak from start to “finish,” with the aim of capturing the dramatic way in which a single outbreak investigation unfolded. I did not stop the journey at the boundary of the technical investigation, but examined the public inquiry provoked by this major crisis. I demonstrated that the phases of the outbreak could be understood as an unfolding social drama, involving breach, crisis, and redress. Though the outbreak occurred between 2008-2009, the social drama continues to unfold, in policy and legal arenas, continuing to express the dynamic tension between overflow and containment.

25 http://www.foodsafetynews.com/2014/06/pre-trial-evidentiary-hearings-for-criminal-trial-of-pca-executives/#.U5IFqI7iTRo
When PulseNet initially detected a possible breach, investigators faced an uncertainty right away. Were the two clusters with two different patterns, but sharing the same serotype, in fact one outbreak? Visualizing the two clusters with a spot map helped solidify the decision to merge the clusters.

During the first month of the investigation, multiple exposures seemed possible. In one state, Ohio, a restaurant cluster pointed to chicken. In another state, Minnesota, trawling food history interviews turned up the potential exposure, peanut butter. However, peanut butter could have been a red herring, after all, many Americans simply enjoy eating it.

A big break in the case came from a nursing home outbreak in Minnesota. The nursing home kitchen had an open jar of an institutional brand peanut butter called “King Nut,” and Minnesota took a shot at testing it for *Salmonella*. Its lab conducted an array of testing, ranging from rapid presumptive testing to the slower confirmatory testing. Upon receiving a presumptive positive, Minnesota officials decided to announce the outbreak to the public.

The identification of the King Nut peanut butter also triggered the traceback leg of the investigation, which simply involved finding out where King Nut was manufactured. The source ended up being a plant in Georgia, the Peanut Corporation of America (PCA). However, the investigation would soon grow exponentially more complex, as a significant new exposure came to the fore—peanut butter crackers.

This meant that PCA was the manufacturer not only of institutional peanut butter, but of a peanut paste ingredient used by many companies to manufacture their own peanut butter flavored products. Tracing the journey of the paste “forward” into the food chain was a complex process that kept on snowballing and ramifying.
Public inquiries heightened the drama, as media coverage of the investigation began to question what this outbreak said about the safety of the US food supply, and regulatory management of it. Even as the outbreak was still ongoing, a Congressional oversight inquiry began. That the investigation had shifted into a criminal one in the field was reflected in the dynamics of the Congressional hearing, with one of its key frames being about a criminal on trial. In addition, the hearing also served as a victim impact panel, as well as the typical examination of the ways in which the food safety system needed reform.

Even after the event itself ended, the social drama of the PCA outbreak continued to unfold in three ways, with ongoing activities in policy reform, another outbreak associated with peanut butter, and ongoing pursuit of criminal charges against PCA executives. Many reforms continue to be negotiated, with some put into place and others cast aside in what has long been an embattled political debate around controlling foodborne disease. In light of this, what seems a reasonably safe prediction to make is that as long as control measures for *Salmonella* keep failing, social dramas provoked by multi-state foodborne outbreaks will undoubtedly continue.
Chapter 8: Conclusion

Summary of Findings

This dissertation examined the building of foodborne knowledge infrastructure, and outbreak surveillance systems within it, focusing primarily on the post-WWII context. This time period saw the rise of a new public health problem in foodborne disease, as a result of the intensified industrialization of the US food supply—national, diffuse outbreaks caused by contaminated food moving through interstate commerce.

I chronicled how public health and regulatory officials used systematization as a key strategy for making this problem visible, and intervening on it. Systematization is an encompassing term that folds in the building of formal systems, and a broader array of rationalizing activities, such as standardization, classification, and ordering practices.

Managing time and managing uncertainty have always been key problems in foodborne outbreak investigations. Once a problem is discerned, getting to the root of it as quickly as possible is important to prevent future illnesses from occurring. Investigators always begin their inquiries not knowing what has caused the problem, and must convert uncertainty into certain enough to take actions in order to protect the public health. While the fast but right balancing act has been a persistent one, its specific qualitative form has been transformed by new technologies of detection, which have evolved in tandem with the changing food supply. I traced this evolution through the problem-solving work of surveillance system-builders, Even as new technologies have been created, and more complex systems built, and more systematization put into place, the persistent challenges of being fast but right have continued to create a need for expert judgment.
Investigating outbreaks at a large-scale, given the massively distributed nature of the US food supply, as well as the multi-level federalist public health system, has involved the building of vast infrastructure, and the creation of second-order surveillance systems within that broader infrastructure. In addition to building infrastructure and systems, as I have chronicled, it took major social action to make foodborne disease into a problem of national public health importance. Debates about the control of foodborne disease have involved many struggles over the fraught question of responsibility and how it should be distributed amongst food producers, consumers, and the government. As I showed, foodborne disease surveillance involves not only technical concerns, but political and moral ones, involving the relationship between the state and its citizens. Crucial to understanding disease surveillance are bioconstitutional ideas about the ideal social order: the state is expected to care for and protect its citizens, and needs to build machinery for accomplishing these purposes. Citizens are imagined as vulnerable beings that cannot solve their problems alone.

An STS lens has been invaluable for studying this large-scale phenomenon. I have combined an infrastructures and systems approach, and used historical and ethnographic methods. In Chapter 2, to take an ethnographic look at the sociotechnical work and collaborative sensemaking involved in outbreak investigations, I developed the concept of a confirmatory pathway for provisional knowledge. This concept helped me to characterize the early warning, real-time system’s network form, and help capture the dynamics of knowledge production, evidence gathering, and public health action within it.

Using an ethnographic perspective helped me to capture the intertwining of science and society. I was able to show how various technical procedures and tools work to thin out material and social thicknesses and simplify them, winnowing microbial communities into single strains,
and winnowing patients/eaters to cases. Then, in chronicling how patterns are created, clusters are assembled, and outbreaks are investigated, I showed how many thin descriptions were aggregated and woven together to create the heterogeneous web that is an outbreak.

I demonstrated that this work is conducted collaboratively, and relies heavily on abductive reasoning, population thinking, group comparisons, and temporal thinking. Meetings were not only a forum for collaborative sensemaking, but an occupational ritual that helped reinforce what it means to be an epidemiologist and collectively manage uncertainty. In recent years, outbreak investigation work has undergone increasing institutionalization and systematization, yet it retains a dynamic, contingent, and lively quality, due in major part to material diversity in the universe of foods.

Taking an ethnographic snapshot from 2012 helped me provide an important initial picture to work with, and use the rest of the dissertation to explain how an early warning, real-time system was built. The confirmatory pathway for provisional knowledge is deeply historical, growing out of layers of public health knowledge infrastructure, and became a reality only through historically specific conditions of possibility.

Chapter 3 began the project of historicizing the phenomenon of foodborne disease surveillance, and how important conditions of possibility for later system-building efforts arose and took shape. I described three key elements of disease surveillance infrastructure particularly relevant to foodborne outbreaks: disease reporting; the rise of the CDC; and identifying, measuring, and classifying *Salmonella*. These three elements of infrastructure were crucial ingredients for helping to make salmonellosis a more visible public health problem in post-
WWII America, however, they were not sufficient. Making disease visible takes enormous infrastructure, and widespread social action.

I documented the work conducted by Philip Edwards to simplify a *Salmonella* serotyping routine for the average state laboratory. During the 1940s-1950s, a partial picture of large scale food infection began to coalesce. One of the major foci of public health concern emerging from this partial picture was the fowl problem, one created by the increasing industrialization of the poultry industry and its association with higher risk of communicable disease, not only for birds but for people too.

What animated and fueled public and policy concern about the fowl problem was media coverage of unsanitary poultry processing as well as the deaths of poultry workers. I examined this policy debate, calling attention to the relevance of the cultural association between women, food, and political consumerism in shaping discourse about consumer responsibility. While Congress passed a control measure for the fowl problem, with the USDA overseeing a compulsory federal inspection program, many social groups expressed dissatisfaction with its effects. Since the fowl problem was not fully controlled in the 1950s, it would continue to make its appearance in other forms. The infrastructure that was built during the early part of the 20th century did contribute to making salmonellosis more visible, but also made limits of surveillance more apparent, animating ongoing efforts to enhance the epidemiologic picture of salmonellosis. By the 1960s, a situation was in place that helped to create the conditions of possibility for subsequent and continual efforts to improve disease surveillance, and in particular, detection of national outbreaks.
Chapter 4 moved from examining the growth of infrastructure and conditions of possibility, to looking at the dynamics of building of the National Salmonella Surveillance Program (NSSP) as a second-order system. A key goal was to improve the total picture of salmonellosis, one that had been only fractional in the 1940s-1950s. I described the NSSP’s role in supporting the investigation of major contamination issues in shell eggs and powdered milk. Controlling these problems entailed issuing warnings to the public. These warnings significantly raised public and political awareness of *Salmonella*.

While the 1960s was an important decade for improving public health surveillance, and contributed to widespread concern about and attention to *Salmonella*, salmonellosis control issues were hotly debated through the 1950s-1960s. Early on, eradication was discussed as contested yet possible goal, but by the end of the 1960s, due in large part to the NRC consensus, eradication was agreed to be an infeasible objective. As a result, the 1970s saw an increased policy focus on risk reduction and consumer education.

In Chapter 5, I continued to examine the NSSP through the 1970s. I documented a shift in its perceived utility, as it became more useful for elucidating long-term trends in the ecology of *Salmonella* serotypes. Officials did not change the NSSP significantly, nor build a system in its place during the 1970s-1980s, despite its acknowledged limitations of weak coupling to control efforts, under-reporting, and a slow tempo for outbreak discernment. But officials did experiment with a variety of new typing techniques, with the common serotype as a key target for innovative energy. Outside of the surveillance system but relevant to the infrastructure was the significant institutional innovation to create an outbreak investigation lab.

I continued examining the fowl problem and its resurgence in the 1980s. Towards the latter part of the decade, *Salmonella Enteriditis* caused a major epidemic associated with Grade
A shell eggs, one that continued for years. The federal government failed to organize a coordinated effort to control the problem. This lack of coordination and control garnered major critique from Congress, which gave all of the involved agencies a new mandate to significantly improve their coordination and control of foodborne disease. Almost on the heels of the SE epidemic, an emerging pathogen, *E.coli* O157:H7, began to cause life-threatening illnesses, especially in the vulnerable population of young children.

In confronting this target of *E.coli* O157:H7, public health officials faced not just technical problems, but economic, social, and political ones as well, to create and spread rapid and cheap screening techniques, investigate a number of smaller scale outbreaks, and finally, confront the game-changing Jack-in-the-Box outbreak of 1993-1994 as a new type of target. This outbreak brought with it the formation of new grass-roots victim advocacy groups who spoke on behalf of vulnerable children.

The Jack-in-the-Box outbreak was also an important stimulus for the early warning, real-time system. Chapter 6 told this system-building story, starting with an account of how PFGE became a salient for PulseNet to incorporate as the centerpiece bacterial typing technique. Because it had developed and stabilized outside of the public health sector, the main job of turning into a public health surveillance tool involved building a standardized system around it. I chronicled PulseNet’s genesis and early development, where the aim was to turn PFGE from a confirmatory tool to a real-time tool. As it expanded, PulseNet adopted a decentralized approach, where each state laboratory would pulse the isolates from ill citizens of its state.

Expansion of the network meant solving myriad critical problems of standardization. I provided an in-depth description of how officials grappled with two critical problems related to getting to “real-time,” shortening protocols and creating a formalized testing system. They also
began to test new and complementary techniques, thinking about the future of the network.

Early on, public health officials thought that MLVA might replace PFGE, but they discovered the technique was too serotype-specific. So, it became a complementary method to PFGE. They also learned, through their work, that sequence-based technologies were presenting a major problem for a standardized system. What biotech developers saw as upgrades that improved the appeal of their products, public health officials saw as reverse salient requiring them to continually re-validate standardized protocols.

When PulseNet gained momentum and begin to accumulate data, as it did so, a new diffuse national outbreak became visible, bringing new critical problems with it: increased workload and more control efforts. As it gained momentum, PulseNet became the center of a new, more intense inter-sector communications organized around the shared problem of the diffuse national outbreak. As the mass globalization of produce increased in the 1990s, this phenomenon began to appear as the source of big multi-state outbreaks.

Dealing with this new target entailed developing a tighter epidemiology-regulatory interface to attempt to trace back foods more quickly during the investigation. The O157 spinach outbreak and the Saintpaul raw produce outbreak demonstrated that regulatory-industry interface was a reverse salient holding up rapid identification and removal of contaminated products from the food chain. Chain-wide, electronic traceability became a key, and ongoing critical problem, significantly reshaping the sociotechnical networks of food production, distribution, and regulation.

Much of the dissertation focused on the infrastructures and systems helping make outbreaks visible. The final empirical chapter, Chapter 7, took a different tack, by following one major multi-state outbreak from start to “finish.” My goal was to capture the dramatic way in
which a single outbreak investigation unfolded, and examine the outbreak as a social drama going through the phases of breach, crisis, redress.

When the breach was first detected, a key uncertainty faced investigators right away. They had to decide if two different clusters were actually one outbreak. Multiple exposures seemed possible initially, and each required extensive follow up. Chicken seemed plausible at first, but turned out to be a red herring. Peanut butter also came to the fore fairly early, but was not seen as a solid hypothesis until a big break in the investigation came from a nursing home outbreak in Minnesota. This investigation revealed the institutional peanut butter “King Nut,” which triggered a fairly simple traceback to PCA. Yet the traceforward would soon grow exceedingly more complex, when PCA was revealed to be the manufacturer of a peanut paste ingredient used in thousands of products all over the country.

Public inquiries amplified the drama of the outbreak, first in the media, and then in an Congressional oversight hearing. That the investigation had shifted into a criminal one in the field was reflected in the dynamics of the Congressional hearing, with one of its key frames being about a criminal on trial. In addition, the hearing also served as a victim impact panel, as well as the typical examination of the ways in which the food safety system needed reform. Even after the event itself ended, the social drama of the PCA outbreak continued to unfold, expressing an ongoing dynamic tension between containment and overflow of the long-standing problem of controlling foodborne disease.

The Future of the System

My dissertation ends just as a significant new phase for the early warning, real-time
system has begun. The bulk of my data collection occurred in 2011-2013, and while I took an ethnographic snapshot of 2012, I was focused on telling a retrospective story of infrastructure and system-building.

In the introduction and in Chapter 6, I touched on how system-builders were looking to preserve the future of the system, in light of major technological changes in bacterial typing. “We are in a kind of transition,” Trees told me, when I talked to her in March 2014 to get a PulseNet update.1 “This is a huge paradigm shift for public health labs.” While I was visiting the CDC in 2012, the agency had been significantly gearing up for a whole genome sequencing (WGS)/bioinformatics/metagenomics transition, but their activities only started to get off the ground in 2013, and would ramp up significantly in 2014.

The reason, she told me, was that the CDC had been awarded $30M for its Advanced Molecular Detection Initiative (AMD) for fiscal year (FY) 2014. While good news for the CDC, given that the agency had been warned by a Blue Ribbon Panel that if it did not invest in bioinformatics, and it would run the “risk of going from outdated to obsolete, and then to irrelevant,”2 there were some caveats, she informed me. “A lot of hands” were reaching for a piece of the $30M, with $10M alone going to build CDC bioinformatic infrastructure for the new paradigm; as an example, even simply storing the coming flood of sequence data, she said, would cost $5M over the next 3 years. To conform to government funding cycle requirements, the money would have to be spent by June. They were hoping to receive another $30M award for FY 2015, to then help build up state infrastructure after solving critical problems about how the new configuration of the system was to take shape.

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1 Interview, Eija Trees, 3/5/14.
2014 would be a year bringing a series of enormous changes, raising new reverse salients that require translation into critical problems, to keep the early warning, real-time system running. Clinical laboratories were beginning to use culture-independent diagnostics more routinely, challenging the standardized interface built between clinical labs and state public health labs. Should state labs start isolating bacteria from stools? It seems odd for state labs to have to maintain such basic wet work capacity when they will also have to shift to the genomic/bioinformatics paradigm.

High-throughput sequencing technologies had finally dropped enough in price to start being implemented at a larger scale (e.g. personal genomics). When would they be ready for prime-time in public health? How would the public health system build such radically different infrastructure?

From a typing perspective, the major targets would no longer be common serotypes or clonal PFGE patterns; it would be developing bioinformatic capacity (hardware, software, data storage, smarts) to pull epidemiologically relevant “types” from masses of sequence data, and getting the new system to work in the network of state laboratory users.

All this within the context of a public health system with state budgets in free-fall, where, as a state laboratory associate director put it, “I believe public health is about 20 years behind, with the primary cause being lack of cash to build and maintain our business infrastructures.”

Would it make sense for state labs to take on hands-on, traditional isolation work while also ramping up genomic and bioinformatic capacity? Did the same kind of decentralized model PulseNet used make sense within the new world being created by WGS/bioinformatics/metagenomics?

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In many respects, this new world is an exciting one. One major multi-state *Salmonella* outbreak that occurred in 2012 that I did not discuss in the dissertation was caused by imported tuna sushi from India. In a retrospective study, FDA researchers used whole genome sequencing to link the outbreak strain geographically to the area of Southwest India where the contaminated tuna came from.\(^4\) It is widely hoped that new technologies and new infrastructure will be able to help address reverse salients in current outbreak detection systems, not only speeding up outbreak investigations, but making global outbreaks more visible. But as this dissertation has shown, solving technical problems is not sufficient. Economic, social, and political problems will continue to matter greatly in the project of surveilling and controlling foodborne disease.

Jane Summerton (2004) has keenly observed that “systems and networks are dynamic entities” that can “seldom be black-boxed for good.” What the future will hold for the early-warning, real-time outbreak detection and response system is still uncertain. But today it still operates robustly, detecting multi-state foodborne outbreaks at an early stage and on a frequent basis. As a result, foodborne disease is a frequent claimant of media attention in a crowded public agenda of social problems. Officials are even worried about “recall fatigue” in the consuming public, given the rate of detected outbreaks and subsequent recalls.

Increasingly, foodborne disease and food safety are not siloed topics, but are interacting more with other societal discourses about food—its sustainability, healthfulness, quality, affordability, taste, and the food system’s scale. My dissertation has shown the importance of understanding that risks do not simply exist in the world, but are *made visible* through specific configurations of knowledge infrastructure and sociotechnical systems. Whether we have these kinds of mechanisms in the first place, in what domains we have or lack knowledge

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\(^4\) [http://www.fda.gov/ForConsumers/ConsumerUpdates/ucm397287.htm](http://www.fda.gov/ForConsumers/ConsumerUpdates/ucm397287.htm)
infrastructure, and how robust or weak that knowledge infrastructure is, is a powerful shaper of how we live with risks, foodborne disease, and otherwise.
Epilogue

I return to the prologue of the dissertation to revisit my interview with Linda and share more of its content as an epilogue.

When Linda finished her narrative, we were both crying. I asked Linda what she would say should be done differently to improve the system for preventing foodborne disease as a result of her experience. Linda drew a line between public health and medicine. To her, most of her anger, and her recommendations for change have to do with the medical emergency care. She talked about her feelings of lack of control over the situation, and the eventual realization that just because she was a doctor, it did not mean that she could control the situation.

I pressed again, even though it almost felt beside the point, and not the right questions. What should the public health system do differently?

Linda said that her experience was mostly fine, public health called to collect Sophia’s food history in January. It was a pure coincidence that during the exposure period they hadn’t gone out to eat a lot. There really wasn’t much information to go on. There were no known outbreaks going on in the area at the time, except related to raw milk, and there was no way that Linda would expose Sophia to raw milk. Public health thought the pattern was connected to a case in the neighboring county but the other case did not shop at the same stores. No one else in the family was sick. The one issue Linda thought could be improved was that public health did not warn her that Sophia could be a carrier for multiple weeks, even after she was healthy; Sophia transmitted \textit{E.coli} to a friend’s child (probably when they were bathed together), but, luckily, the other child experienced only mild symptoms.

Sophia’s strain matched a case from last September. Maybe it was something from the freezer, or with a long shelf-life? It was disconcerting to hear that it was something that was
maybe still in the house. Linda threw away all the open food in the house. It was frustrating to not know what it was. They had had meatloaf recently, was it an issue of kitchen hygiene?

She felt guilty, she wasn’t always perfect in the kitchen, but why should she feel guilty? She also said that there was an aspect of this that didn’t feel fair, some people get settlements with just a day of diarrhea. It sounds greedy, but if Sophia were part of a known outbreak she could have gotten a settlement to help cover Sophia’s enormous medical costs, but she wasn’t part of a known outbreak. Public health couldn’t pin it on something, and so she doesn’t know where the fault was.

Linda told me that when she has shared this story with others, people really want to know what the cause was. Linda has a theory that it might be some frozen peas. Sophia was the only one in the household who loved to eat the frozen peas raw. But this was only a speculative theory. Linda felt like people needed to hear that it was something, so that it would never happen to them. Now, for example, one of Linda’s friends never feeds her children frozen peas.

I asked if Linda now does anything differently herself, and her answer was, “not really.” She’s no germaphobe, and while not perfect in the kitchen (who is?), she argued forcefully, “you can’t sterilize everything.” Furthermore, she asked whether it wasn’t the companies’ responsibility to produce safe food? Yet, her relationship to the food system has not changed. Linda likens this experience to a “lightening bolt”; no matter what she did, it could have happened anyway.

While I see that there could be “lessons” learned from this story to help change policy and improve prevention measures, this was not the reason why Linda shared this story with me. Linda is not trying to reshape the food system because of her daughter’s foodborne illness. This moment was about sharing, in itself; one person who had gone through a traumatic experience
sharing a recollection of that experience with another. It was about being human, and talking about life as it was lived.

But different things emerge from this story, than I have written about in the preceding pages. For me personally, Linda and Sophia’s story has laid much barer the intensely moralized site the kitchen still is. As Linda’s interview crystallizes, the broader policy discourse of consumer “responsibility” and “carelessness” can create (unnecessary) feelings of guilt that add to a parent’s pain and trauma when their child’s health is in the balance, and this issue has not been given enough visibility.

Yet, the “lessons” that are learned from this story have to be drawn. For the story in itself, the human suffering was clear, but there is ambiguity in what it “means,” because “lessons learned” were not the point of the story. As humans we may want resolutions, but don’t always get them; the bad guy might not be the frozen peas, and even if it were, it wouldn’t erase the trauma of the experience.

“Taking on a medical or scientific perspective… doesn’t help us to deal with the problem of suffering,” Arthur Kleinman (1988) says. “…there is no teleological perspective on illness that can address the components of suffering relating to problems of bafflement, order, and evil, which appear to be intrinsic to the human condition.”

After Linda and I finished our conversation, we dried our tears, finished our lattés, and met our other friend for lunch. How do we live with food risks? Most of the time, hopefully, we just live.
BIBLIOGRAPHY


