Separating the Chaff from the Wheat:
How to determine the strength of a foodborne illness claim

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William Marler is the managing partner of Marler Clark LLP, PS. Since 1993, Mr. Marler has represented thousands of persons who have become ill with *E. coli*, *Salmonella*, Hepatitis A, *Listeria*, *Shigella*, *Campylobacter* or Norovirus illnesses in over thirty states. As a trial lawyer, Mr. Marler has been involved in several cases of national importance. He represented a nine-year-old girl in her $15.6 million *E. coli* settlement with Jack-in-the-Box. This settlement created a Washington State record for an individual personal injury action. Mr. Marler resolved several other Jack-in-the-Box *E. coli* cases for more than $2.5 million each.

In May of 1998, Mr. Marler settled claims on behalf of the families of several children who were severely injured after consuming *E. coli* O157:H7-contaminated Odwalla apple juice for a reported $12 million. In 2001 he successfully tried to verdict an *E. coli* case involving a school lunch program in Washington State. The jury returned a verdict of $4.75 million. He also resolved dozens of *E. coli* cases in 2003 related to one of the largest meat recalls in United States history. Mr. Marler recently settled an *E. coli* case for a young girl for $11 million. In addition, Mr. Marler has been lead counsel in litigation stemming from the 2002 Chili's *Salmonella* Outbreak, 2003 Pat & Oscar’s *E. coli* Outbreak, 2003 Chi Chi's Hepatitis A Outbreak, and the 2004 Sheetz *Salmonella* Outbreak.

Mr. Marler speaks frequently on issues of safe food and is a principal in Outbreak, Inc., a non-profit business dedicated to training companies on how to avoid foodborne illness outbreaks (see http://www.outbreakinc.com).
I. The Chaff - Cases We Turn Away Every Day

Unfortunately, some people make suspect and unsupportable foodborne illness claims. It is therefore important to develop a reliable method of identifying suspect, unsupportable, or illegitimate claims. In our experience, food industry corporations over-emphasize, and thus over react to, the presence of such claims. Such a strategy can lead to the denial of legitimate claims. Denying legitimate claims increases the likelihood of missing important measures to improve food safety. Not improving food safety increases the risk of poisoning consumers and resulting litigation. Litigation not only carries its own expenses, but the threat of public relations headaches as well.

What, then, are some reliable methods for recognizing suspect food poisoning claims?

A. Incubation Period

Although incubation periods – the time between ingestion of a foodborne pathogen and the onset of symptoms – are only ranges, and wide ones at that, they can still be used to identify an improper claim. The claimant who insists that her E. coli O157:H7 illness was sparked by the hamburger she ate an hour before she got sick does not have a winnable case regardless of the damages because the incubation period of E. coli O157:H7 is one to ten days, typically two to five days.

<table>
<thead>
<tr>
<th>PATHOGEN</th>
<th>INCUBATION PERIOD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Staphylococcus aureus</td>
<td>1 to 8 hours, typically 2 to 4 hours.</td>
</tr>
<tr>
<td>Campylobacter</td>
<td>2 to 7 days, typically 3 to 5 days.</td>
</tr>
<tr>
<td>E. coli O157:H7</td>
<td>1 to 10 days, typically 2 to 5 days.</td>
</tr>
<tr>
<td>Salmonella</td>
<td>6 to 72 hours, typically 18-36 hours.</td>
</tr>
<tr>
<td>Shigella</td>
<td>12 hours to 7 days, typically 1-3 days.</td>
</tr>
<tr>
<td>Hepatitis A</td>
<td>15 to 50 days, typically 25-30 days.</td>
</tr>
<tr>
<td>Listeria</td>
<td>3 to 20 days, typically 21 days</td>
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<tr>
<td>Norovirus</td>
<td>24 to 72 hours, typically 36 hours.</td>
</tr>
</tbody>
</table>

A case we turned away:

“After getting out of church yesterday morning, Sunday, December 12, 2004, I stopped at [a restaurant] to grab a sandwich, just a double cheese [sandwich], and a small Dr. Pepper at 12:02 pm. I still have my receipt. I had not eaten anything prior to eating the sandwich, and I still am unable. Within two hours of eating that sandwich I became very ill. My fever went up from 98.6 to 100.2; I got diarrhea, stomach cramps, headache and chills. I am still very sick, I'm very weak, I can't really eat anything, and I'm having chills. I'm at work trying to work and I feel like crap...I don't know what to do, I called the restaurant and the
manager is supposed to be calling me back when he gets in. Can you please help me?"

A quick consultation of the chart above, which is not exhaustive, reveals that this person’s lunch from this restaurant is most likely not the source of his illness. The incubation period is too short. A diagnosis of *Salmonella*, *Shigella*, *Campylobacter*, or *E. coli* O157:H7, for example, all of which have incubation periods longer than two hours, would effectively rule out the meal as a source of the illness. It is possible that the person became ill after ingesting *Staphylococcus aureus*, but given the prevalence of the bug, and without knowledge of multiple ill persons, this is a very difficult causation case.

**B. “The Food Looked/Smelled/Tasted Funny”**

In most situations, bacteria will be undetectable by the consumer, which is an intrinsic risk to consumers in the first place. Therefore, customers who complain that they got a foodborne illness from a particular meal because the food tasted funny are, very likely, wrong. Be careful, though; many consumers with legitimate complaints tend to retroactively assign a negative connotation to a meal once the health department has identified it as a source of an outbreak. This common instinct should not tear down an otherwise viable claim. But a claim that something tasted funny, without other proof linking a particular food to illness, remains suspicious.

A case we turned away:

“*I have recently read articles and lawsuits that you have pursued regarding contaminated food. I am hoping that you may be able to give me your professional advice or recommendation. My husband recently opened a bottle of salsa and smelled an unusual odor but chose to eat it regardless, thinking that it was just his nose. After taking two bites and tasting rather badly, he found what appeared to be a rather large piece (approx. the size of the back of an adult's fist) of human or animal flesh. Even though he didn't seek medical attention, he did become very nauseated. I do feel that the manufacturer should be held responsible for this mishap. Thank you for your time and consideration.”*”

**C. “Gross-Out” Claims**

While certainly not the type of thing a food provider may want on the news, claims centered on finding, but not eating, some undesirable agent in food rarely have value.

A case we turned away:

“*I opened a box of Buffalo wings and dumped them out on a plate to be cooked in the microwave. An unusually shaped piece caught my eye and I picked it up.*
When I saw that the "piece" had a beak, I got sick to my stomach. My lunch and
diet coke came up and I managed to christen my carpet, bedding and clothing. I
want them to at least pay for cleaning my carpet etc. What do you think?”

II. In Between the Quickly Dismissed and the Clearly Compelling, How
Does One Evaluate a Foodborne Illness Claim?

The tools we use:

- Health Department Investigation of the Incident
- Prior Health Department Inspections
- Medical Records
- Lab Results

III. The Health Department Investigation of an Outbreak

A. What Does the Health Department Do?

While statutes and regulations vary from state to state, there are a number of
bacterial and viral illnesses associated with food consumption that are monitored
by health departments, including *E. coli* O157:H7, *Campylobacter*, *Salmonella*,
*Shigella*, *Listeria*, Norovirus, and Hepatitis A. For most of these pathogens, a
positive lab result from a human sample (blood or stool), triggers a mandatory
report to the local health authority and some type of follow-up investigation. The
length, breadth, and paperwork involved in any investigation varies depending on
the pathogen involved, the type of food, the number of persons who are or may be
sick, the local jurisdiction, and other factors.

In most situations, the results of the investigation are either made public by the
health authorities or can be obtained through public records acts like the Freedom
of Information Act (5 U.S.C. §552 et. seq.).

B. Very Difficult to Dispute a Health Department Confirmed
Outbreak or Even an Isolated Case

In litigating thousands of food poisoning claims arising out of dozens of
outbreaks, many defendants have taken issue with some or all of the health
department’s conclusions regarding the outbreak. None of these defendants,
however, have yet successfully avoided liability where the health department
concluded that the defendant’s food was the source of a given outbreak. One
likely reason for this is that, in general, health departments do good and careful
work. Despite the occasional disagreement of the pinpointed member of the food
service industry, most would agree that health departments are rather cautious and
conservative. In our experience, health departments do not prematurely label an entity as the source of an outbreak.

In addition, health departments are operating with a much higher burden of proof than the civil justice system. Most epidemiologists will not confirm an outbreak without 95% confidence in a particular conclusion. Contrast this with the difficulty of bringing the jury's confidence in the source of an outbreak below 51% in the same scenario.

Finally, it has also been our experience that the jury is simply more likely to accept the “neutral” determinations of the health department over paid experts.

1. Finley School District *E. coli* O157:H7 Outbreak


The *E. coli* O157:H7 outbreak was investigated by local and state health officials who concluded that the source of the outbreak was a ground-beef taco meal prepared and served by the Finley School District at Finley Elementary School. The eleven minor plaintiffs were all identified as either “confirmed” or “probable” members of the outbreak. All but one of the plaintiffs attended Finley Elementary School. Four of the children developed hemolytic uremic syndrome (“HUS”), which resulted in varying degrees of permanent kidney damage.

Interestingly, the child with the most severe injuries (F.M.) was the only plaintiff who did not attend the school and did not eat the implicated meal. F.M.’s older sister, however, did attend Finley Elementary and had eaten the taco meal. It was the plaintiffs’ position that this non-student had acquired an *E. coli* O157:H7 infection through exposure to her sister or another student she had been in contact with. This phenomenon is known as “secondary infection.”

The defendant took issue with nearly every facet of the plaintiffs’ case and, in doing so, took on the task of attacking the health department’s conclusions, which were supportive of the plaintiffs’ case. For instance, the defendant disputed the health department’s conclusions that the taco meal was the source of the *E. coli* O157:H7 outbreak amongst the students, and it disputed that F.M. had actually suffered an *E. coli* O157:H7 infection (F.M.’s stool did not culture positive for the bacteria, but she had been diagnosed as having been infected, and the health department deemed her a “probable” secondary case). The defendant also took the position that, even if F.M. had an *E. coli* O157:H7 infection, there was insufficient evidence to demonstrate that the taco meal was the source of her illness.
The case was bifurcated for trial. The liability portion of the trial lasted four weeks, and it resulted in a plaintiffs’ verdict. Testimony from many of the health department officials involved in the investigation was, in the jury’s collective mind, highly important.

2. Can the Plaintiff Make a Case Without Health Department Support in Either an Outbreak or Isolated Case?

Health departments will not report a “confirmed” outbreak, or pinpoint a restaurant as the “confirmed” source of a food illness without near certainty. Without 95% confidence in a particular conclusion, health departments are likely to define individuals or outbreaks as “possible.” This is the case even where the confidence in a particular conclusion is well above the legal standard of more likely than not.

If the health department has investigated and found a claimant’s illness did not come from a particular source, the plaintiff will face the same uphill battle taken on by so many defendants.

Although this scenario occurs infrequently, it is possible for a plaintiff to make a claim for damages. In these cases, reliable expert opinion or examination of the health department investigators themselves can establish the source of a plaintiff’s illness with sufficient certainty to meet the legal burden of proof.

IV. Prior Health Inspections/Violations

One extraordinarily effective tool in establishing the defectiveness of a product that no longer exists is uncovering documentation of the food service establishment’s sordid past. This may include information regarding prior incidents or accusations of food contamination and prior inspections of the facility and the establishment’s food production and service procedures. Supportive documents can be acquired through the discovery process or through the applicable freedom of information act.

The uncovered documents will help the plaintiff make his case in a variety of ways. Sometimes, there may be documentation of improper food handling procedures that can circumstantially prove the manner of contamination. In other situations, a list of improper techniques and code violations can serve as a tool for limiting a defendant’s trial options, or it can position a case for early and favorable settlement. Finally, particularly egregious or repetitive examples of improper food handling techniques can build a punitive damages case, where such damages are available.

A. Identifying the Improper Procedure that Led to the Contamination of the Food
It is a rare case, at least with respect to restaurant-based food poisoning claims, where contaminated leftovers will be located by the time investigative agencies or lawyers are on the scene. This missing piece of the puzzle can be supplied, however, by identifying specific errors in the preparation of the suspected food or foods.

1. Improper Cooking Procedures

In 2001, a young girl suffered a particularly severe *E. coli* O157:H7 infection that left her with permanent kidney damage. The little girl had eaten a hamburger purchased from a midsized southern California fast-food chain. Hamburgers have been commonly viewed as the source of *E. coli* O157:H7 infections in humans and nothing else in the girl’s food history was a likely source of the infection. By the time health department officials investigated, however, the case of hamburgers out of which the girl’s had been chosen was long gone. The health department did not find any food on site that tested positive for *E. coli* O157:H7. A thorough review of the restaurant’s current and prior inspections though, revealed a serious flaw in the firm’s cooking method that provided an explanation for the client’s exposure. According to the inspection report:

> Hamburger buns are toasted on the grill immediately adjacent to the cooking patties, and it is conceivable that, early in the cooking process, prior to pasteurization, meat juices and blood containing active pathogens might possibly splash onto a nearby bun.

In fact, on six separate occasions spanning three years, the management of the restaurant had been advised of the dangers of cross contamination of the hamburger buns by hamburger juices. The plaintiff’s expert also reviewed the prior inspection reports and concluded that the chain’s cooking methods presented a high risk of cross contamination. The matter settled shortly after the presentation of this information.

2. Improper Refrigeration

In another case, a Chinese restaurant in Ohio was the suspected source of an *E. coli* O157:H7 outbreak in the fall of 2002. Again, no contaminated leftover food was found. In addition, the restaurant was buffet-style, which complicated the identification of a single contaminated food item. A disproportionate number of ill patrons were children, and it began to appear that the culprit food might in fact be Jell-O. Obtaining the health department investigation report provided the answer to the obvious question: how might Jell-O have become the source of an *E. coli* O157:H7 outbreak?
The report noted a host of food handling errors in the restaurant, none more important than this one: “raw meat stored above the Jell-O in the refrigerator.” Officials concluded that “the likely source of \textit{E. coli} O157:H7 in the Jell-O was from raw meat juices dripping on the Jell-O while it was solidifying in the refrigerator.” The defendant never seriously contested liability once the report was obtained.

3. Improper Storage and Cooking Procedures

In 2003, a group of people who had attended a banquet hosted by a restaurant in Washington State fell ill in the days following the banquet. Many of the banquet goers tested positive for \textit{Salmonella}, but leftover food items had either been discarded or had tested negative. The health department’s subsequent investigation of the event provided the information necessary to establish liability. The food service establishment had violated state food regulations by “pooling” dozens, if not hundreds, of raw eggs in a single bucket for storage overnight. This process allows bacterial contamination from a single egg to taint exponentially larger amounts of food, thereby placing many more consumers at risk. The establishment subsequently used the raw eggs as a “wash” on a specialty dessert. Then, once again in violation of food code, the food workers failed to cook the egg thoroughly. When these actions were taken together with the fact that raw eggs are a particularly notorious source of \textit{Salmonella}, the smoking gun was back in the defendant’s hands.

B. Patterns of Poor Food Handling Practices

In some circumstances, damaging inspection documents can also dissuade a defendant from contesting liability in front of the jury. In a case where defending the case from a liability standpoint is a less than certain undertaking, defense counsel may be wary of admission of evidence that will make the defendant less sympathetic in the eyes of the jury.

1. Improper Sanitation I

In 2000, a producer and distributor of high-end fresh food items were identified by various health agencies as the source of a large \textit{Shigella} outbreak on the west coast. The firm, a relative new-comer to the food industry, operated with a marketing stance and inward belief in the high quality of its products. Health department inspections, however, revealed serious problems at the firm’s production facilities, including the lack of fully operational bathrooms for employees, insects near food production sites, and evidence of rodents in the facility. Through discovery, it was also uncovered that a major commercial purchaser of the firm’s product had conducted its own inspection of the facilities, and had refused to purchase any more products until a number of significant upgrades were made to the facility.
2. Improper Sanitation II

In 2002, a Seattle-area restaurant was suspected by health officials as the source of a medium sized outbreak of food poisoning. Even though one of the patrons experienced an unusually severe acute illness, medical practitioners and health officials were unable to pinpoint the particular pathogen that had sickened the various individuals. The defendant and its insurer were initially unwilling to concede liability in part based on the unidentified causative agent in the outbreak. Acquisition of the prior inspection reports, however, revealed a consistent pattern of poor food handling practices. The repeat occurrences of numerous health code violations led the health department to close the restaurant and temporarily revoke its license. In the end, the proposition of contesting liability proved too risky for the defendant.

C. Punitive Damages

Much in the same manner as other products liability cases, evidence of knowledge of prior incidents of improper behavior can be the cornerstone of a punitive damages claim. Because a food establishment must sign off on its inspection reports, these documents are useful in establishing both prior violations and the defendant’s prior knowledge.

In 1996, fresh juice manufacturer Odwalla was identified as the source of a major outbreak of \textit{E. coli} O157:H7 on the west coast. Through discovery requests, the plaintiffs sought documentation of inspections by governmental agencies, Odwalla itself, and private parties. After considerable motion practice, the plaintiffs uncovered previously undisclosed inspection reports, including a report from the United States Department of the Army. This report revealed that the U.S. Army had inspected Odwalla’s production methods prior to the outbreak and determined not to buy its products. In a letter to Odwalla, it stated:

\begin{quote}
We reviewed deficiencies noted in the report, which our inspector discussed with you at the time of the inspection. As a result, we determined that your plant sanitation program does not adequately assure product wholesomeness for military consumers. This lack of assurance prevents approval of your establishment as a source of supply for the Armed Forces at this time.
\end{quote}

Through further discovery, the plaintiffs recovered internal company emails reacting to the U.S. Army’s inspection and subsequent refusal to purchase products from the company. One employee suggested implementing a microbiological testing program to address some of the problems uncovered in the inspection. The following is a portion of an email responding to the suggestion:

\begin{verbatim}
9
\end{verbatim}
...why are we doing it, why now, what do we WANT TO PROVE...IF THE DATA is bad, what do we do about it. Once you create a body of data, it is subpoenaable...you should look at this as though the Fresno Bee has looked into the results and asked a lot of questions...

At the time of the E. coli O157:H7 outbreak, the company had not adopted the suggested testing regimen. The plaintiffs filed a motion to apply California law regarding punitive damages due to Odwalla’s prior knowledge that its product was unsafe. With the punitive damages motion pending, the cases were resolved.

V. Medical Records

A. What medical evidence can make a case?

1. Laboratory Testing

Both stool cultures, and less commonly blood cultures, can identify the particular pathogen causing a claimant’s illness. These tests and their impact on subsequent legal claims are discussed at length below. In reviewing a claim, it is important to recognize that laboratory testing is not always ordered by health care providers.

2. Matching symptoms and food with an expected incubation period

Each foodborne pathogen carries with it an expected incubation period – the amount of time expected to transpire between exposure to the pathogen and the onset of symptoms, as discussed above. The incubation period can encompass a significant period of time, and can thus lessen the effectiveness in a given situation. Nevertheless, it can still be useful. For example, people often assume that the last meal they consumed before falling ill was the culprit. With many pathogens, however, this is very unlikely. The typical incubation for E. coli O157:H7, for example, is 2-7 days, with a reported range of 24 hours to 20 days.

3. Matching symptoms with typical profiles of a given pathogen, or given outbreak

Most common bacterial and viral pathogens found in food share reasonably similar symptoms- nausea, vomiting, diarrhea, fever, aches, chills, and the like. Various pathogens can have more typical courses. While these cannot be used alone to determine the pathogen affecting a claimant, it can be part of the puzzle. For example, Hepatitis A infections are often characterized by yellow skin and eyes, or jaundice. E. coli O157:H7 infections are most often characterized by excessively painful, bloody diarrhea.
VI. Lab Tests

A. Stool/Blood Cultures and PFGE

1. Mandatory Reporting

Health care providers may in some instances order testing of an ill person’s blood or stool to help determine the cause of illness. In many circumstances a positive result in such a test must be reported to the health authorities pursuant to statute or regulation. Many states require reporting of positive tests for a number of pathogens, including *E. coli* O157:H7, *Salmonella*, *Shigella*, *Listeria*, Hepatitis A, *Campylobacter*, and others. It is the report of such positive results that often triggers health department investigations and creates awareness of outbreaks.

2. Pulse Field Gel Electrophoresis, or PFGE

When a sample of a form of bacteria, such as *E. coli* O157: H7, *Salmonella*, or *Shigella*, is taken from a stool culture or a piece of meat or poultry, it can be cultured to obtain and identify the bacterial isolate. Bacterial isolates can be further broken down into their various component parts, creating a DNA “fingerprint.”

The process of obtaining the DNA fingerprint is called Pulse Field Gel Electrophoresis, or PFGE. It operates by causing alternating electric fields to run the DNA through a flat gel matrix of an agarose, a polysaccharide obtained from agar. The pattern of bands of the DNA fragments – or “fingerprints”- in the gel after the exposure to the electrical current is unique for each strain and sub-type of bacteria. By performing this procedure, scientists can identify hundreds of strains of *E. coli* O157:H7 as well as strains of *Listeria*, *campylobacter*, and other pathogenic bacteria.

The PFGE pattern of bacteria isolated from contaminated food can be compared and matched to the PFGE pattern of the strain isolated from the stool of infected persons who consumed the contaminated product. When PFGE patterns match, they, along with solid epidemiological work, are proof that the contaminated product was the likely source of the person’s illness.

Combined with some epidemiological evidence, PFGE is an extremely potent causation argument. This is particularly true where the PFGE pattern has not been reported elsewhere. For example, suppose two unrelated persons both test positive for a genetically identical, unique, strain of *E. coli* O157:H7 in a given town within a matter of days. If the subsequent, mandatory health department inquiry into these two illnesses reveals no other common exposures between the two people other than a hamburger from the same restaurant on the same day, finding a credible, alternate explanation for their illness can prove nearly impossible.
For more information on PFGE, visit http://www.fsis-pfge.org.

3. **“PulseNet”**

In 1993, a large outbreak of foodborne illness caused by *E. coli* O157:H7 occurred in the western United States. In this outbreak, scientists at the CDC performed DNA "fingerprinting" by PFGE and determined that the strain of *E. coli* O157:H7 found in patients had the same PFGE pattern as the strain isolated from hamburger patties served at a large chain of regional fast food restaurants. A more prompt recognition of this outbreak and its cause may have prevented more than the estimated 750 illnesses. As a result, the CDC developed standardized PFGE methods and, in collaboration with the Association of Public Health Laboratories, created PulseNet so that scientists at public health laboratories throughout the country can rapidly compare the PFGE patterns of bacteria isolated from ill persons and determine whether they are similar; thus indicating an outbreak linked to exposure to a common source of bacteria.

PulseNet is an early warning system for outbreaks of foodborne disease. It is a national network of public health laboratories that performs DNA “fingerprinting” on bacteria that may be foodborne. The network identifies and labels each “fingerprint” pattern and permits rapid comparison of these patterns through an electronic database at the CDC to identify related strains.

At present, PulseNet tracks four foodborne disease-causing bacteria: *E. coli* O157:H7, nontyphoidal *Salmonella*, *Shigella* and *Listeria monocytogenes*.

B. **The Impact of a Negative Test**

While the lack of a laboratory test or a negative result may detract from the strength of a claimant’s case, it is unwise to assume invulnerability where a lack of a positive test can be easily explained by other factors.

1. **Antibiotics**

The consumption of antibiotics, whether or not related to the illness at issue, essentially renders a stool culture worthless. A negative result after commencement of antibiotics is common.

2. **Untimely Testing**

For different pathogens and different people, the speed with which the pathogen exits the body varies widely. The symptoms can continue well after the pathogen has been expelled from the body. Testing that occurs more than a few days after the onset of symptoms is unreliable, and a negative result at that time is not necessarily indicative that the pathogen had not been previously present.
3. No Test Given

Health care providers do not order blood and stool cultures for all, or even most, cases of gastroenteritis. In many cases, there simply will not be testing to include in the determination of the source of illness.

4. Look at the circumstances as a whole

With an isolated illness, the lack of a positive stool culture may be problematic for a claimant. In the context of most outbreaks however, it is not a significant problem. Circumstantial evidence may easily compensate. One such example is where one member of a dining party does not get tested, and others do. Three of four persons who all ate together fall ill with the same, documented, pathogen. The fourth demonstrates the same symptoms in the same time frame, but his or her doctor does not order stool cultures. Liability can be easily established without the positive stool culture.

C. Testing the Food

In food poisoning cases there is generally no food to test because, not surprisingly, it was eaten. But left-over food, or uncooked portions of the food that was eaten, that tests positive for the given bacteria or virus is powerful evidence that the food is the likely cause of the illness. This is convincingly so if the bacteria or virus in the food is a PFGE match to the ill person’s stool culture isolate.

If there is food to be tested (whether the request is by the State investigators or a party to a suit), one must be aware of chain of custody issues that may arise to question the results.

VII. The Legitimate Case: What does a legitimate foodborne illness claim look like?

A. The Majority of Foodborne Illnesses are Avoidable Errors

1. Ammonia Nuggets?

The plaintiffs were a group of school children and teachers who consumed chicken contaminated with ammonia on November 24, 2002 at the Laraway Elementary School in Joliet, Illinois. The poisoning resulted from the acts and omissions of three corporate entities.

In 2001, the State of Illinois, through the Illinois State Board of Education (ISBE), contracted with Tyson to have United States Department of Agriculture (USDA) commodity bulk chicken processed into chicken tenders for the National
School Lunch Program (NLSP). The processing was completed at the Tyson plant located in New Holland, Pennsylvania. The state of Illinois also contracted with Lanter Refrigeration to warehouse the chicken tenders. For an as yet unknown reason, Tyson’s delivery of the chicken greatly exceeded Lanter’s shipping and storage capacity. Lanter then contracted with Gateway Cold Storage to house the overflow chicken products at its facility in St. Louis, Missouri. The tenders were stored at the facility along with large amounts of other food intended for consumption at Illinois schools.

On November 18, 2001, there was a large anhydrous ammonia leak on the 6th floor of the Gateway St. Louis facility. Massive amounts of food destined for the school lunch program, including the chicken tenders, were exposed to ammonia.

Inexplicably, Gateway and Lanter notified neither the local health authorities, nor the Illinois State Board of Education. Even more remarkably, Gateway and Lanter continued shipping food from the facility, without any concern for the leak or the obvious risks it presented to consumers – particularly school-aged children. According to officials at the St. Louis City Health Department (SLCHD), Lanter and Gateway shipped approximately 800,000 pounds of product from the facility after the leak without any notice to consumers.

In fact, it was a shipment of potato wedges to Illinois schools that first alerted authorities in Illinois to the leak. On November 27, nine days after the leak, schools began complaining to the ISBE that they had received potato wedges that stunk of ammonia. Once complaints about the potato wedges had prompted an inquiry about the storage/condition of the food, Gateway and Lanter admitted over the phone to the ISBE that a leak had in fact occurred.

On November 27, 2001 ISBE sent a letter to Lanter. The letter acknowledged the leak and stated, “[A]t the advice of the IDPH, any food coming in direct contact with the ammonia leak should be destroyed.” The letter advised Lanter to place all food connected with the ISBE on hold, pending further evaluation.

The FDA at this time “determined to place all product stored at Gateway at time of ammonia leak on hold until procedures are established for clean up and treatment of products to dissipate ammonia odor.”

Gateway and Lanter immediately began devising a plan that would allow them to release product that had been exposed to the ammonia leak. Eventually, a plan was devised by Gateway and representatives from Lanter that was submitted to the FDA on or about December 3, 2001. The original plan submitted to the FDA was rejected. The Missouri Department of Health complained that “the firm did not commit to any testing for ammonia residue in the product.”
On or about December 7, 2001, the FDA approved a plan for the testing and release of FDA-regulated products, i.e. fruits and vegetables. It is not clear when, if ever, any agency approved any plan for release of the chicken tenders.

At this point, Gateway began shipping food to the schools again. The chicken tenders, however, were apparently not ready for release. There were 361 boxes of chicken tenders, each with 20-22 pounds of product in 5 individual plastic bags. The boxes were cardboard, and were labeled as Tyson product. The tenders had been so thoroughly exposed to the ammonia leak that the boxes and labels were either destroyed, saturated with ammonia and the ammonia smell, or both. Gateway, Lanter, and Tyson made the decision that rather than destroy the food, and swallow the small loss associated, that they would re-box, re-label, and “re-condition” the boxes, and then send them on to the schools. The original plan was to re-box the tenders some time in February, 2002.

In the meantime, it was becoming evident that other product shipped from the Gateway facility still stunk of ammonia, and was being rejected by schools and other public programs that received it. In January of 2002, a cafeteria worker in Champaign, Illinois complained about the powerful smell in some beef patties, and refused to serve them to school children.

In March of 2002, ISBE reported that a number of schools were rejecting products from the Gateway facility due to the odor of the boxes. ISBE noted that “some commodities are not in tightly sealed vapor proof packaging and the food itself has an odor.” ISBE eventually asked and received permission from the USDA to use the rejected food as animal feed. Officials at Lanter and Gateway were informed of the schools’ complaints.

The re-packaging of the chicken tenders did not occur in February, 2002. Piecing together the exact manner in which the chicken tenders were eventually re-boxed is complicated by the discrepancies in reports given by Lanter to health agencies after the students’ illnesses.

Nonetheless, it is clear that in late June 2002, the chicken tenders were shipped from the Gateway facility to Lanter. At the same time, a request was made that Tyson send new labels for each box. The labels were designed to look exactly like the original labels on the chicken tender boxes. When the boxes were shipped, Lanter noted that “some of the cases were in ‘bad’ shape and just did not look good, probably due the handling during the reconditioning.” Apparently the product had already been through a reconditioning process designed to remove the ammonia smell from the boxes and product. While this process was designed to remove the smell, which might alert the recipient to the presence of ammonia, nothing was done to actually remove any ammonia from the product.

In the same time frame, a Tyson Representative visually inspected the product. He suggested that samples of the product be sent to Tyson for ammonia testing at
the Tyson lab in Springfield, Arkansas. While it is not clear if the testing was ever actually completed, Tyson nonetheless contacted the ISBE and informed them the chicken was safe. Tyson called ISBE on July 3, 2002 and “notified [ISBE] the product is fine and [they] will change the casing.” Either the testing was never done despite the representations to ISBE, or was done so poorly as to miss what would later prove to be ammonia levels more than 100 times the legal limit. Either way, Tyson’s blessing on the chicken helped move it closer to the Laraway Elementary School.

The chicken tenders were re-boxed and re-labeled in early July 2002. When questioned by the IDPH, Lanter originally stated that it had no knowledge of the re-packing, and that no one at Lanter had participated. Shortly thereafter, Lanter told IDPH investigators that it had been “determined” that Lanter did in fact re-box roughly 320 cases of Tyson chicken tenders that were received from Gateway in three loads on June 24 and 25, 2002. Lanter said that the re-boxing was done at a Lanter warehouse on July 5, 2002. New labels from Tyson were affixed to the boxes of chicken tenders. Although the original plan had called for the presence of a USDA employee at the re-boxing, the process went forward without supervision. Following the re-boxing, Lanter began shipping the chicken tenders to schools in Illinois. The evidence of their ammonia exposure, i.e. the smell and the damage to the packaging, had been removed, but the threat to the students remained.

When school began in August 2002, so did complaints to ISBE regarding food that smelled of ammonia. These complaints were, as a matter of course, passed on to Lanter. At this time, the complaints did not involve the chicken tenders, which had apparently not yet arrived.

In October of 2002, the Chicago Heights school rejected chicken tenders due to an ammonia smell. Lanter was notified but none of the defendants took any action to notify schools that had received the chicken tenders of the re-boxing, or of the complaint from Chicago Heights. Other schools joined in with complaints shortly thereafter. Still, the defendants did nothing to warn the other recipients of the chicken, despite having a list of those schools in their possession.

On November 25, 2002, the chicken tenders were served to students at Laraway elementary school. Within minutes of consuming the chicken 157 students, roughly half the school, fell ill. The scene verged on total chaos. Students and teachers were running into the halls vomiting, with their throats and noses burning. Students panicked. School administrators called in ambulances, and children were taken to five local hospitals.

The IDPH would later verify the obvious--that the illnesses were caused by high concentrations of ammonia in the chicken. The chicken was the Tyson product that had been shipped by Lanter, and stored at the Gateway facility during the 2001 leak. Remarkably, even without considering the leak, the food should never
have reached the students, as it had been in holding well past the 270 day legal limit. According to the USDA, Lanter had a consistent problem with rotating stock to avoid such problems.

On December 2, 2002, the Food Safety and Inspection Service (FSIS), a branch of the USDA, verified very high ammonia levels in the chicken tenders through its own laboratory analysis. This verified testing was conducted by an independent laboratory at the behest of IDPH. The tests revealed contamination of the tenders with ammonia at 500-2,000 ppm. According to the IDPH the legal limit for such concentrations is 15 ppm.

2. Who Needs Hot Water?

On June 30, 2003, the Lake County Health Department (LCHD) received a report from Lake Forest Hospital indicating that a patient was ill with a *Salmonella* infection. The LCHD immediately contacted the patient and interviewed him, using a questionnaire that is standard for the epidemiological investigation of foodborne illness outbreaks. One of the first things learned by the interviewer was that the patient had recently eaten at the Chili’s Grill & Bar in Vernon Hills, Illinois.

About an hour after receiving this first report, a second person contacted LCHD to report that a family member had become ill after eating at Chili’s in Vernon Hills. This prompted the LCHD to send investigators to the restaurant to inspect it. What they found was disturbing. The restaurant’s dishwashing machine was broken and corroded; the tube that fed chlorine into the machine was plugged, preventing proper sanitization of dishes. Employees told the investigators that the machine had not worked properly for at least a week. In fact, according to the LCHD Final Report, “[e]mployees had wrapped plastic bags around the line to stop the chlorine from spraying into the air.” Despite the obvious broken condition of the dishwasher, the restaurant management still had done nothing to get the machine repaired—that is, until caught by the health department.

During their inspection, the investigators also found food not stored at proper temperatures in the cooler. And following questioning of the on-duty manager, investigators learned that three employees, plus another manager, had called in sick that day with flu symptoms.

The next day, LCHD received two new reports of individuals with *Salmonella* infections who had eaten at Chili’s on June 26, while Chili’s management reported six more ill employees. With evidence of the outbreak-source growing increasingly clear, investigators returned to the restaurant to instruct employees on hand-washing procedures, to require the use of nailbrushes, and to issue a glove-use order. This meant that no further bare-hand contact of food was to be allowed at the restaurant. The investigators also collected stool samples from the employees there in addition to interviewing each one of them regarding
gastrointestinal symptoms. As a result of these interviews, investigators discovered thirteen employees who had been allowed to work despite suffering from diarrhea and other symptoms.

Because of the large number of infected employees identified, the LCHD ordered the restaurant to close. A statement issued by LCHD Executive Director Dale Gallassie announced that:

Due to the large number of ill employees, and the high potential for spread of this illness, Chili’s was required to cease all operation or face suspension or revocation of its food service permit, at which time Chili’s management made the decision to voluntarily close the establishment.

On July 2, investigators returned to Chili’s and collected 50 more employee stool samples, then issued a press release advising the public of the outbreak. People who had eaten at the restaurant between June 23 and July 1 were instructed to seek medical help if ill, and to report their illness to the health department. Just a few hours later, LCHD was flooded with telephone complaints of illness from people who had eaten at the restaurant. LCHD had to enlist the aid of two additional communicable disease nurses to help interview all of the people calling in about the outbreak.

The next day, on July 3, LCHD received a call from a customer that had dined at Chili’s on June 27. She informed LCHD that the establishment had no running water while she had been there for lunch. The customer estimated that Chili’s had no water for at least an hour or two. This was information that Chili’s management had not thought necessary to share with investigators at the time of their initial interviews.

On July 7, LCHD received notice from the lab that the stool samples of seventeen employees had cultured positive for Salmonella. One of the employees had also worked at the Chili’s restaurant located in Gurnee, which was immediately inspected. This was the fourth restaurant potentially implicated in the Salmonella outbreak as a result of infected Chili’s employees working at more than one restaurant.

Returning to the Vernon Hills Chili’s restaurant, LCHD investigators interviewed restaurant managers again and confirmed that there had been no water during the lunch rush on June 27, and no hot water the entire day before. No one could explain why the decision was made to keep the restaurant open in violation of food-safety regulations requiring that hot water be available at all times during a restaurant’s operation.
On the afternoon of July 8, LCHD issued a statement announcing that 31 cases of Salmonellosis had by that time been confirmed, and well over 100 cases were suspected to be related to the Chili’s outbreak. Of the confirmed cases, 14 had eaten at the restaurant, and 17 others were employees.

Investigators inspected the restaurant on July 10, and then again on July 11 right before its reopening. LCHD staff provided a hand-washing demonstration for Chili’s employees, and then formally gave approval to operate. Chili’s reopened at 11:00 a.m. for lunch. The restaurant had been closed for over two weeks as a result of the outbreak.

At the time of the restaurant’s reopening, a total of 19 employees and 67 patrons had been confirmed positive for Salmonella, with an additional 128 cases suspected to be linked to the outbreak. Of the total cases so far, nine had been serious enough to require hospitalization.

On July 16, the results of microbiological testing performed on food samples from the restaurant, and from leftovers provided by customers, came back from the lab. Only two food samples had tested positive for Salmonella, both from customer leftovers: one from the Vernon Hills restaurant, and one from the Gurnee restaurant.

By July 18, LCHD concluded its investigation and determined the outbreak was under control. No secondary cases had been reported, but over 300 individuals had been sickened as a result of consuming contaminated food at Chili’s. Of those, 141 customers and 28 employees had tested positive for the Salmonella bacteria, while 105 other infected individuals met the LCHD’s definition of a probable case. LCHD issued a preliminary report that concluded the outbreak was caused by infected employees who contaminated food with Salmonella as a result of poor sanitary practices and improper food-handling. It was by this time also determined that the Salmonella associated with the outbreak was Salmonella serotype javiana, a relatively rare and virulent strain often associated with foodborne transmission.

Once the LCHD believed the outbreak was controlled, the department sent a letter by certified mail informing the restaurant’s management of a hearing scheduled for July 31 to discuss their failure to cease operations during periods where no hot water, or no water at all, was available, failure to adequately monitor their employees’ health, and the steps management had implemented to prevent future outbreaks.

Following the hearing, LCHD stated that Chili’s had violated local ordinances by remaining open and serving customers while without available water.
VIII. Real People, Really Sick

A. Hepatitis A - Liver transplant, Brain Damage – Richard and Linda

Richard and Linda’s Hepatitis A infections arose out of an extremely large outbreak in Western Pennsylvania in late 2003. The infections were linked by federal, state, and local health officials to green onions served in salsa prepared at a Chi Chi’s restaurant.

Richard and Linda were health department-confirmed members of the outbreak. Richard and Linda dined at the implicated Chi Chi’s between September 14 and October 17, 2003, the period of identified exposure. The onset of their illnesses matched the expected incubation period for Hepatitis A. Finally, both Richard and Linda had laboratory confirmations of acute Hepatitis A virus (HAV) infection, i.e. positive IgM anti-HAV.

On Sunday, October 12, 2003, Richard and Linda ate lunch at Chi Chi’s in the Beaver Valley Mall. On Tuesday, October 28, just days after visiting their son and daughter-in-law, Linda and Richard developed flu-like symptoms. Over the following days, their symptoms grew worse, particularly Richard’s.

By early November, Richard had been identified as one of hundreds of victims of the Chi-Chi’s Hepatitis A outbreak. He was treated in the ER and released. Several days later, Richard became non-responsive. Linda dialed 911 and he was taken by ambulance to the hospital for the beginning of a lengthy and near fatal stay.

Laboratory studies conducted in the hospital showed that Richard’s liver was failing due to the Hepatitis A. Richard showed signs of coagulopathy—tiny blood clots forming in his liver—consistent with worsening synthetic liver dysfunction. The admission diagnoses included metabolic encephalopathy—a brain disorder that can follow liver failure—and fulminant hepatic (liver) failure.

Richard continued to show signs of a persisting, severe coagulopathy. Doctors made the decision to list him for liver transplant. Accordingly, Richard was soon admitted to the hospital’s Liver Transplant Intensive Care Unit, where his continuing liver dysfunction could be more closely monitored and observed. Doctors also ordered that Richard be watched closely for respiratory failure. Richard’s family was stunned by the news that it was a matter of mere hours before Richard’s liver would shut down completely. He needed a transplant and fast.

Richard’s son David was on his way home, driving west on Interstate 76 from Philadelphia to Pittsburgh, when he learned that his father had been put on the transplant list. “I remember being about halfway home . . . driving at an average
speed of 80 miles an hour when I received the next call from my brother for an update. My father needed a transplant,” David recalls, or “in the next 15 hours he could or most likely would die. Now I was driving at 90 miles an hour to get home.”

The family gathered that night to pray for Richard’s life. Because his condition continued to deteriorate, Richard had been moved to the top of the transplant list. “I remember the nurse telling us that he was moved to the top of the list,” David recalls. Conflicted feelings ran through his mind. “I was praying that a miracle would come for my father at the loss of another. It is hard to believe that you are praying that another man dies within the immediate future.”

Meanwhile, in the intensive care unit, catheters were placed for Richard’s comfort and a peripheral IV site was established. Nursing assessments indicate that Richard was jaundiced, his blood pressure was high, and he was agitated, disoriented, and unable to follow simple commands. And over the next few hours, Richard’s condition continued to deteriorate. He became even more agitated and confused, repeatedly pulling on his tubes to disconnect his IV’s. Restraints had to be placed on his upper extremities, enabling the aggressive hydration treatment to continue intravenously.

While he awaited a life-saving transplant, Richard’s brain function grew worse and worse. Doctors eventually decided that immobilization (temporary paralyzation) was the best way to control Richard’s erratic actions. He was sedated, intubated through the mouth, and placed on mechanical ventilation so that he could breathe. Doctors were then able to monitor Richard’s brain waves. Studies found that his encephalopathy was worsening: Richard’s head and brain would require extensive post-transplant monitoring, if, that is, a transplant even occurred.

Later that night, Linda learned that a compatible liver had been located. She consented to the operation immediately. In the hours before transplantation, Richard became highly fevered. Cooling blankets and ice packs were applied to his body. Additionally, a chest x-ray revealed another complication: an accumulation of fluid in Richard’s lungs, possibly pneumonia.

At 7:00 a.m. on Saturday, November 8, 2003, Richard was transported to the operating room of UPMC Presbyterian, where he underwent orthotropic liver transplantation. The operation lasted over seven hours and was not without complication. Elevated intracranial pressure readings indicated continuing brain dysfunction, and Richard suffered cardiac arrest. Doctors acted quickly to reverse the arrest with CPR, atropine, and epinephrine. A post-operative, pathological review of Richard’s liver revealed submissive to massive hepatocellular necrosis, secondary to a hepatitis A infection.
On Monday, November 10, Richard remained critically ill with encephalopathy, intracranial pressure, and respiratory failure. He was still unresponsive to verbal and tactile stimuli, and he remained intubated and paralyzed, breathing only with the assistance of a mechanical ventilator. His oxygen requirements had increased, too, due to the onset of pneumonia. The new liver, however, seemed to be functioning.

After twenty-seven days of hospitalization, Richard was discharged on December 2, 2003. The final diagnoses were fulminant hepatitis A, left vocal cord paralysis, cardiac arrest, sepsis secondary to nosocomial pneumonia, metabolic encephalopathy, and orthotopic liver transplant. Discharge medications included Lopressor, Nystatin, Acyclovir, Bactrim, Aspirin, Carafate, Magnesium oxide, Protonix, Prednisone, and FK-506. Richard must take the Bactrim and FK-506, or their equivalents, as long as he lives; these are his anti-rejection medications, which help prevent Richard’s immune system from destroying his new, though “foreign,” liver.

Since his discharge, Richard has struggled to cope with the ramifications of his liver transplant and brain damage. Richard’s first day home was strange to say the least. The surroundings were familiar, but he had been away so long and had returned such a different person, quite literally, that he felt deeply insecure. Several times, uneasy about the distance from medical help, Richard caught himself almost longing for the familiarity of a hospital room. Nonetheless, with Linda’s help, Richard stayed home and managed to last that first, difficult day.

Several difficult days later, Richard began to have well-grounded fears that the incision across his abdomen would burst. Though it was secured by so many staples that it looked like a giant, three-pronged zipper, every movement seemed to stress the wound, leaving Richard largely helpless and immobile. When he did feel secure enough to move, he often asked Linda for help.

Linda, Richard’s constant companion for the previous thirty-six years, now stayed closer than ever. Richard recalls that she was never more than ten feet away because, with his injuries, “anything can happen.” Consequently, Linda’s days were long and tiring, the task of caring for such a badly injured person requiring constant and close attention. Among the more complex of her duties, Linda had to organize and dispense Richard’s medications, and the list was growing longer.

Nighttime was just as taxing for both. Linda woke up for every movement or sound, ready to help however she could, and Richard rarely slept; the business of avoiding further injury and pain required too much care. His left arm ached terribly, and his legs went numb if he did not move them frequently. Moreover, for fear of aggravating or, worse, bursting his incision, Richard was unable to roll onto his side or stomach to sleep. So he kept his torso still and, most of the time, laid in bed awake.
Neuropsychological tests conducted months after the hospitalization revealed evidence of mild cognitive slowing and attention difficulties. Specifically, Richard exhibited mild auditory verbal processing difficulties and some impulsivity, mild attention difficulty with non-verbal tests, mild perseveration, and mildly reduced manual dexterity in both hands. Dr. Saxton concluded that Richard was suffering from Anoxic Brain Injury.

Many of Richard’s injuries, and his significant deconditioning, will hinder him until he dies. More probably than not, Richard’s liver transplantation in November 2003 was not his last. Richard’s transplant surgeon has projected that Richard should reasonably expect to undergo at least one re-transplantation in his lifetime. Moreover, his neurological function, though improving, will never be what it once was due to the anoxic brain damage done during his encephalopathic period.

As mentioned above, Richard’s new liver will likely fail at some point, requiring that he endure the transplantation process, and all of its painful trappings, yet again. He will also remain on an aggressive anti-rejection regime, which, though medically necessary, suppresses his immune system, thus hindering his body’s capacity to fight infection. Richard will, therefore, forever be at an increased risk of developing serious infections, including pneumocystis carinii pneumonia, fungal infections, herpes zoster, and cytomegalovirus. Also, Richard will always face the prospects of a viral infection called Post-transplant Lymphoproliferative Disorder, which occurs in about 2% of transplant recipients and can be fatal if not treated urgently at a Transplant Center.

Aside from these infections, an increased incidence of malignancy is a complication of immunosuppression in transplant recipients. Other complications following liver transplant, and of which Richard will constantly be on guard, include hypertension, kidney failure, diabetes, and anemia. Dr. Fontes has indicated that the most likely complications in Richard’s case are infection, cancer, and hypertension.

At this point, though, no matter how likely, the above-listed complications are mere possibilities, and they therefore cause no more than a constant sense of foreboding in both Linda and Richard. More definite is the supportive, monitoring, and diagnostic treatment that Richard must endure for the rest of his life. The list of tests and required studies is long and complex.

Among the more notable of his care requirements, Richard must submit to weekly blood-testing; he will be evaluated at the transplant clinic twice yearly to ensure that his new liver, and all future livers, function properly; and he must return to the neurology and otolaryngology clinics at least once a year to monitor the effects of his brain and vocal cord injuries. And, as can reasonably be expected of any person who has endured what Richard has, he will require extensive psychological counseling, likely for the rest of his life.
It is forecasted, conservatively, that Richard will require a full week’s hospitalization each year for the rest of his life to address the various complications and treatments that necessarily follow liver transplantation. This estimate speaks volumes about the quality of life that Richard, or any liver transplant recipient, can expect. It is one in which the driving focus is staying alive, not actually living.

B. Salmonella and Death – Henry

Henry’s claim arose out of an outbreak of Salmonellosis in Washington, Oregon, and a number of other Western states during June of 1999. During June of 1999, both the Washington State Health Department and the Oregon Health Division independently investigated clusters of diarrheal illness attributed to *Salmonella* serotype Muenchen infections in each state. As of July 13, 1999, 207 confirmed cases associated with this outbreak had been reported by 15 states and two Canadian provinces; an additional 91 cases of *S*. Muenchen infection were reported, and were still under investigation. By early July, 1999, 85 persons with this illness were identified in Washington State alone.

Epidemiological investigations by the health departments linked the outbreak of this relatively rare strain of *Salmonella* to unpasteurized orange juice products produced by Sun Orchard, Inc., an Arizona based company. Similar strains of *Salmonella* were eventually detected in unopened containers of Sun Orchard juice products and in blenders where smoothies were made. The Sun Orchard product was distributed in a variety of forms to retail stores, restaurants and other food service institutions. The unpasteurized orange juice was identified as freshly squeezed or fresh orange juice, and was also sold in a frozen form to restaurants and other food retailers. Genetic matches were quickly established between the lab results of the stool cultures from victims and the Sun Orchard product. Henry was one of the persons who purchased and consumed the juice. He later tested positive for a strain of *Salmonella* genetically identical to the strain associated with the outbreak.

Henry was a jovial, energetic and vital man, prior to his *Salmonella* infection. Despite his age of 88 years, he lived independently, worked full time, and was in excellent health. He hated doctors and hospitals, and his records indicate he rarely required medical treatment during the decade prior to his death, and then only for routine colds and coughs. His long term family doctor indicates he was an extremely healthy man for his age, with a “physiologic age” of approximately 70, in contrast to his chronologic age of almost 89.

Henry was working full time as a security guard for American Commercial Security Services, on the Microsoft Redmond campus, prior to his illness. He had commenced working there in 1991, at the age of ‘81, to supplement his income. His employment records reflect his outstanding record of attendance and
performance at work, with only a one week illness-related absence in 1995. His working hours were remarkably consistent during the last six quarters prior to his illness.

Henry drank the contaminated orange juice at the Empress Hotel in Victoria, B.C., on or about June 19, 1999.

Henry then became sick, and had the first onset of explosive diarrhea on Tuesday, June 22, 1999. He then spent most of the next few days in bed, did not eat or drink anything, and became extremely fatigued and dehydrated. His daughter took him to the emergency room on June 24, 1999, despite his wishes, where he was found to be experiencing atrial fibrillation (excessively rapid contractions of the heart). The medical records at the time indicate that he was still jovial, making jokes and in good spirits, and expressed his desire to continue working. The records also confirm he had no prior history of heart disease, hypertension, diabetes, or stroke. He was then released, and returned home.

At Evergreen Hospital Henry submitted a stool sample for testing. A positive culture resulted, confirming Henry had been stricken with the relatively rare Salmonella Muenchen bacteria associated with the Sun Orchard outbreak.

On June 26, 1999, his daughter found him at home, slumped down between a wall and his bed. She did not know how long he had been there, and he was unable to get up on his own. He had apparently been trying to move and get help for hours. He was returned to the ER by ambulance. He was confused, and was unable to provide an accurate history. It was eventually determined that he had suffered an acute cerebral vascular accident (a stroke), and he was admitted to Evergreen Hospital Medical Center in serious condition.

Henry was a patient at Evergreen Hospital for ten days, from June 26, 1999 to July 6, 1999. He was confused, had severe left side weakness and paralysis, could not feed himself, and had difficulty swallowing. At first, the records indicate he was still occasionally very funny and jovial. Over a period of days, however, the nurses’ notes record Henry’s decline into a state of extreme depression, eventually requiring related medication. He started to intentionally withdraw, became weepy, and started asking about dying. Visitors were shocked to see him lying there, paralyzed on one side and barely able to open his eyes. He had often told others that he never wanted to be helpless or to be a burden on anyone, and he had always said he would rather blow his brains out.

Henry was eventually transferred to a nursing home in early July of 1999, for further care. At the time he still had left side weakness, impaired mobility, and reported increasing confusion and loss of cognitive skills.
Henry died on July 23, 1999, approximately one month after his *Salmonella* infection and the immediately subsequent stroke. His family doctor, Dr. Blume, believes it is very likely that he simply “gave up”, and died.

C. *E. coli* O157:H7 - Acute Kidney Failure, Diabetes and Brain Damage – Katelyn

Young Katelyn’s *E. coli* O157:H7 infection was linked to a relatively small outbreak of the illness linked to ground beef sold at BJ’s Wholesale Club. Public health officials first learned of a possible outbreak when, on May 28, 2002, the Rockland County Health Department (“RCHD”) became aware of a sudden increase of *E. coli* O157:H7 infections through physician reports and laboratory test-results.

Suspecting the source to be ground beef, by far the most common cause of *E. coli* O157:H7 outbreaks, RCHD went to Katelyn’s home and took what was left of the frozen meatballs and hamburger patties made from the ground beef purchased at the West Nyack BJ’s store. Samples were then submitted to the New York State Department of Health’s Wadsworth Center for microbiological testing.

The microbiological test results came back positive for the presence of *E. coli* O157:H7. Subsequent pulse-field gel electrophoresis (“PFGE”) tests of the bacterial isolates then revealed an exact match between the *E. coli* O157:H7 in the leftover ground beef and the strain identified as cause of the outbreak cluster, also known as the “outbreak strain.”

Approximately two weeks later, a consumer with an unopened package of BJ’s ground beef came forward to offer it for testing. The results of this testing proved conclusive: it tested positive for the outbreak strain. Consequently, on July 16, 2002, over two months from the date the adulterated ground beef was originally purchased, BJ’s initiated a recall, mailing a letter to those people who had bought the ground beef at the West Nyack store between May 8 and 13, 2002.

What began for Katelyn, then age six, on May 20, 2002, as a seemingly minor gastroenteritis, progressed over the course of a week to painful, bloody diarrhea that completely debilitated her, and then to the vastly more serious complication of HUS that nearly killed her and left her hospitalized for three and a half weeks. From her first symptoms to her initial discharge from Westchester Medical Center on June 22, Katelyn endured a roller-coaster of sudden complications that brought her near to death.

Katelyn’s symptoms of diarrhea and vomiting had persisted, and worsened, over the course of a week when she was admitted to Westchester Medical Center on May 27, 2002 with abdominal pain, bloody diarrhea, and volume depletion. By the 29th, she had cultured positive for *E. coli* O157:H7 and was developing the
first laboratory signs of HUS.

Over the next week, Katelyn’s labs would reveal developing HUS, with progressive destruction of red blood cells, platelets, impaired kidney function, and pancreatitis. In addition to the abdominal pain associated with a severely inflamed large intestine that is one of the hallmarks of an *E. coli* O157:H7 infection, Katelyn suffered the exquisite abdominal pain of pancreatitis.

On June 3, Katelyn began to exhibit the signs of central nervous system involvement, a condition a pediatric neurologist described as “toxic metabolic encephalopathy.” Overt brain involvement became apparent on June 4 as Katelyn became increasingly irritable and confused, and then unresponsive. She then suffered a seizure with left-sided body twitching, a fixed upward, lateral gaze, and decreased oxygen saturation levels. An EEG was positive for focal seizure activity and she was sedated and treated with anti-seizure medications.

On June 9, Katelyn was transferred out of the Pediatric Intensive Care Unit though she was sometimes incontinent, had twitching around her left eye, and could only speak in a “whispered” voice. She remained insulin-dependent due to her ongoing pancreatitis. While Katelyn’s renal, pancreatic, and blood problems dominated her hospital course, she also had to suffer through pleural effusions, liver involvement, and high blood pressure, a problem that remains today.

By June 18, Katelyn was slowly improving, though she was so weak she could barely walk. Her labs were still abnormal, her speech was abnormal, and it was becoming apparent that she would remain in need of insulin injections secondary to pancreatic damage. On June 22, Katelyn was able to go home—to her bed. She was hardly recovered. Three days after discharge she saw a pediatric gastroenterologist for persistent abdominal pain with bowel movements. She was also undergoing home physical therapy.

On June 29th, Katelyn was back at the Westchester Medical Center ER with vomiting, diarrhea, and abdominal pain. She was readmitted. An abdominal CT scan revealed a pancreatic pseudocyst. After discharge from this second hospitalization, Katelyn began what would be an endless course of outpatient visits to a variety of medical specialists to deal with her complex of ongoing medical problems.

The vast majority of type 1 diabetics become so because the body’s autoimmune system has attacked and destroyed the insulin-producing beta cells within the islets of Langerhans. This seems to occur through a combination of genetic predisposition and environmental triggers. It is also possible to become a type 1 diabetic, like Katelyn, through the destruction of the pancreas by trauma or an acute disease process. However, almost all type 1 diabetics, unlike Katelyn, retain the exocrine function of the pancreas and are able to produce digestive enzymes.
The list of serious complications arising from Insulin Dependent Diabetes Mellitus (IDDM) is depressingly long. Virtually every part of the body is at risk for damage as a result of long-term diabetes which accounts for IDDM’s huge impact on early death and health care cost statistics. Even a cursory review of major diabetes complications is sobering:

- **Heart disease.** This is the leading cause of diabetes-related deaths. Adults with diabetes have heart disease-related death rates 2 to 4 times greater than nondiabetics;
- **Stroke.** The risk for stroke is 2 to 4 times higher among diabetics;
- **High blood pressure.** Almost 75 percent of adults with diabetes have elevated blood pressures and/or use prescription antihypertensives;
- **Blindness.** Diabetes is the leading cause of new cases of blindness among adults 20 to 74 years old. Diabetic retinopathy causes 12,000 to 14,000 new cases of blindness every year;
- **Kidney disease.** Diabetes is the leading cause of end-stage renal disease (“ESRD”), accounting for 43 percent of new cases;
- **Nerve damage.** About 60 to 70 percent of diabetics have some form of nerve damage. The result of such damage commonly results in impaired sensation or pain in the hands and feet, slowed digestion of food, and carpal tunnel syndrome. Severe diabetic nerve damage is a major contributing factor to lower extremity amputation;
- **Amputations.** More than 60 percent of nontraumatic lower-limb amputations in the US occur among diabetics;
- **Dental disease.** Diabetics have an increased incidence of periodontal or gum diseases. About one-third of diabetics have severe periodontal disease;
- **Complications of pregnancy.** Poorly controlled diabetes before conception and in the first trimester of pregnancy can result in severe birth defects and spontaneous abortions. Poorly controlled diabetes in the second and third trimesters can lead to excessively large babies. Diabetics are at increased risk for preeclampsia which can cause dangerously high blood pressure late in pregnancy.
- **Poor healing/immune system compromise.** Diabetics suffer poor wound healing and are more susceptible to illnesses generally. Once sick, they have a poorer prognosis than nondiabetics and are more likely to die
from common illnesses such as influenza and pneumonia.

Katelyn had “severe” HUS which included renal failure, severe anemia, thrombocytopenia, hypertension, profound pancreatitis, and central nervous system complications.

One of the several cruel realities of this case is that Katelyn’s *E. coli* infection will cost her a good portion of her life. Insulin-dependent diabetics do not live normal life expectancies, mostly due to cardiovascular disease. There is a vast amount of statistical data about the life expectancies of those with IDDM. Broadly speaking, Katelyn can expect to lose ten to twenty years of her life. This estimate, if anything, is probably conservative. There are relatively few IDDM sufferers who develop their type 1 diabetes as young as age six. The damage from diabetes is cumulative. And certainly there are extraordinarily few type 1 diabetics who have also suffered the additional renal insult that comes with HUS.

Two leading pediatric nephrologists have opined on Katelyn’s prognosis and predict that more likely than not she will suffer kidney failure as a result of the combined insult of HUS and IDDM. Her diabetes doctor stated:

> The quality of [Katelyn’s] life has been severely reduced. Even with excellent care and attention from a number of different medical specialists, and great determination from herself and her family, it is likely that her overall life expectancy will also be reduced by as much as ten to twenty years.

Katelyn has been robbed of her childhood, and her retirement years. Everything between will be a daily struggle with multiple injuries – all resulting from a seven year old eating a single hamburger.

**IX. Resources on the Web**

- http://www.about-campylobacter.com
- http://www.about-ecoli.com
- http://www.about-hepatitis.com
- http://www.about-hus.com
- http://www.about-listeria.com
- http://www.about-norwalk.com
- http://www.about-reiters-syndrome.com
- http://www.about-salmonella.com
- http://www.about-shigella.com
- http://www.about-ttp.com
- http://www.campylobacterblog.com
- http://www.ecoliblog.com
- http://www.ecolilitigation.com