I. E. COLI O157:H7 AND LEAFY GREENS – A BRIEF HISTORY

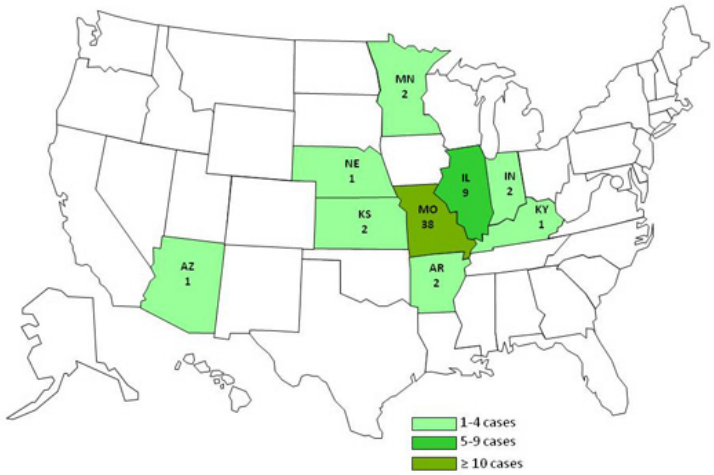
E. coli outbreaks associated with lettuce or spinach, specifically the "pre-washed" and "ready-to-eat" varieties, are by no means a new phenomenon. In fact, the frequency with which this country's fresh produce consuming public has been hit by outbreaks of pathogenic bacteria is astonishing. By way of illustration, in October 2003, thirteen (13) residents of a California retirement home were sickened, and two (2) people died, after eating *E. coli*-contaminated, pre-washed spinach; in September 2003, nearly forty (40) patrons of a California restaurant chain fell ill after eating salads prepared with bagged, pre-washed lettuce; and in July 2002, over fifty (50) young women fell ill with *E. coli* O157:H7 at a dance camp after eating "pre-washed" lettuce, leaving several hospitalized and one (1) with life-long kidney damage. And this is just a small sampling of the twenty (20) or more *E. coli* outbreaks since 1995 in which spinach or lettuce was the source. Several more, including the September 2005 Dole lettuce outbreak, and the infamous September 2006 Dole baby spinach outbreak, appear in the chart below, which is based on information gathered by the Center for Science in the Public Interest:

Date	Vehicle	Etiology	Confirmed Cases	States/Provinces
Aug. 1993	Salad Bar	*E. coli* O157:H7	53	1:WA
July 1995	Lettuce (leafy green; red; romaine)	*E. coli* O157:H7	70	1:MT
Sept. 1995	Lettuce (romaine)	*E. coli* O157:H7	20	1:ID
Sept. 1995	Lettuce (iceberg)	*E. coli* O157:H7	30	1:ME
Oct. 1995	Lettuce (iceberg; unconfirmed)	*E. coli* O157:H7	11	1:OH
May-June 1996	Lettuce (mesclun; red leaf)	*E. coli* O157:H7	61	3:CT, IL, NY
May 1998	Salad	*E. coli* O157:H7	2	1:CA
Feb.-Mar. 1999	Lettuce (iceberg)	*E. coli* O157:H7	72	1:NE
July-Aug. 2002	Lettuce (romaine)	*E. coli* O157:H7	29	2:WA, ID
Oct. 2003-May 2004	Lettuce (mixed salad)	*E. coli* O157:H7	57	1:CA
Apr. 2004	Spinach	*E. coli* O157:H7	16	1:CA
Sept. 2005	Lettuce (romaine)	*E. coli* O157:H7	32	3:MN, WI, OR
Sept. 2006	Spinach (baby)	*E. coli* O157:H7 and other serotypes	204	Many States
Nov/Dec 2006	Lettuce	*E. coli* O157:H7	71	NY, NJ, PA, DE
Nov/Dec	Lettuce	*E. coli* O157:H7	81	IA, MN, WI

Date	Vehicle	Etiology	Confirmed Cases	States/Provinces
2006				
May 2008	Romaine	*E. coli* O157:H7	9	WA
April	Romaine	*E. coli* O145	33	MI, NY, OH, PA, TN

II. AN OUTBREAK OF *E. COLI* O157:H7 LINKED TO ROMAINE LETTUCE, 2011

On March 23, 2012 the Centers for Disease Control and Prevention (CDC) issued its final update on the multistate outbreak of *E. coli* O157:H7 infections linked to romaine lettuce.[1] The outbreak investigation was assigned outbreak code 1110MOEXH-2. Genetically, the outbreak strain was identified by Pulsenet pattern designations EXHX01.0047/EXHA26.0015. At the completion of the investigation on March 21, 2012, 58 persons residing in 9 states were infected with the outbreak strain of *E. coli* O157:H7. The number of ill persons identified in each state is as follows: Arizona (1), Arkansas (2), Illinois (9), Indiana (2), Kansas (3), Kentucky (1), Minnesota (3), Missouri (38), and Nebraska (1).



Persons infected with the outbreak strain of *E. coli* O157:H7, by state.

Among persons for whom information was available, illnesses began from October 10, 2011 to November 4, 2011. Ill persons ranged in age from one to 94 years, with a median age of 29 years old. Sixty-three percent (63%) were female. Among persons for whom information was available, illnesses began from October 9, 2011 to November 7, 2011. Ill persons ranged in age from 1 to 94 years, with a median age of 28 years. Among the 49 ill persons with available information, 33 (67%) were hospitalized, and 3 developed hemolytic uremic syndrome (HUS). No deaths were reported.

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[1] *See* http://www.cdc.gov/ecoli/2011/ecoliO157/romainelettuce/032312/index.html.

Collaborative investigative efforts of state, local, and federal public health agencies indicated that romaine lettuce sold primarily at several locations of a single grocery store chain, Schnucks, was the likely source of illnesses in this outbreak. Contamination occurred before the product reached Schnucks stores.

During October 10 to November 4, 2011, public health officials in several states and CDC conducted an epidemiologic study by comparing foods eaten by 22 ill and 82 well persons, including 45 well persons who shopped at a Schnucks grocery store during the week of October 17, 2011. Analysis of this study indicated that eating romaine lettuce was associated with illness. Ill persons (85%) were significantly more likely than well persons (46%) to report eating romaine lettuce in the week before illness. Ill persons (85%) were also significantly more likely than well persons (46%) to report shopping at a Schnucks store. Among ill and well persons who shopped at Schnucks, ill persons (89%) were significantly more likely than well persons (9%) to report eating a salad from the salad bar at Schnucks. Several different types of lettuce were offered on the Schnucks salad bars. Of 18 ill persons who reported the type of lettuce eaten, 94% reported eating romaine lettuce. No other type of lettuce or other item offered on the salad bar was reported to be eaten by more than 55% of ill persons.

Ill persons reported purchasing salads from salad bars at Schnucks between October 5 and October 24, 2011. A total of nine (9) store locations were identified where more than one (1) ill person reported purchasing a salad from the salad bar in the week before becoming ill. This included two (2) separate locations where four (4) ill persons reported purchasing a salad at each location. For locations where more than one (1) ill person reported purchasing a salad from the salad bar and the date of purchase was known, dates of purchase were all within four (4) days of other ill persons purchasing a salad at that same location. Schnucks voluntarily removed suspected food items from the salad bar on October 26, 2011. Romaine lettuce served on salad bars at all locations of Schnucks had come from a single lettuce processing facility owned and operated by Vaughan Foods, Inc., located in Moore, Oklahoma. Vaughan Foods was also the sole distributor of processed romaine lettuce to Schnucks stores.

The FDA and several state agencies conducted traceback investigations for romaine lettuce to try to identify the source of contamination. Traceback investigations focused on ill persons who had eaten at salad bars at several locations of Schnucks, and on ill persons at two college campuses, in Minnesota (1 ill person) and Missouri (2 ill persons). Traceback analysis determined that a single common lot of romaine lettuce harvested from "Farm A" was used to supply Schnucks locations as well as the Centennial Dining Hall at the University of Minnesota during the time of the illnesses. This lot was also provided to a distributor that supplied lettuce to the university campus in Missouri, but records were not sufficient to determine if this lot was sent to this university campus. Preliminary findings of investigation at Farm A did not identify the source of the contamination. Farm A was no longer in production during the time of the investigation.

The Minnesota case in this outbreak was critical to both the epidemiological and traceback analysis. The Minnesota case's stool isolate was indistinguishable from the outbreak

strain on PFGE analysis. Minnesota Department of Agriculture documents establish that C & E Farms (more specifically Gubser Ranch Lot 21R23, harvested 10/05/2011 and 10/06/2011) was a supplier of romaine lettuce via FoodSource to GO Fresh, which is the Minnesota processor that prepared and sold the romaine lettuce product that ultimately sickened the University of Minnesota student.

The following charts make the route of distribution of C&E Farms romaine lettuce clear. The first gives the relevant data on the Gubser ranch romaine lettuce that ultimately ended up at Schnucks salad bars, accounting for a vast majority of outbreak cases:

Farm	**Grower/ Shipper**	**Broker/ Consignee**	**Processor**	**Wholesaler**	**Retailer**
Tierra Sol Farms Gubser Ranch Lot 21R23 Harvested 10/05/2011 and 10/06/2011	C&E Farms Salinas, CA	FoodSource Monterey, CA Shipped 648 mini bins romaine lettuce 10/06/2011 PO FM27795	Vaughan Foods Moore, OK Received product 10/08/2011 Processed (chopped) and packaged into 2 pound packages and shipped 180 6X2lb. cases to Schnucks 10/11/2011	Schnucks Fresh Foods Warehouse Bridgeton, MO Received product from Vaughan Foods on 10/12/2011	Schnucks Fresh Foods Salad bars in store delicatessens Onset date of illnesses: 10/20/2011 10/21/2011 10/22/2011 Dates of likely consumption: 10/16 through 10/20/2011

The next chart gives the relevant data on the Gubser ranch romaine lettuce that ultimately ended up at Centennial Hall at the University of Minnesota:

Farm	Grower/ Shipper	Broker/ Consignee	Processor	Wholesaler	Retailer
Tierra Sol Farms Gubser Ranch Lot 21R23 Harvested 10/05/2011 and 10/06/2011	C&E Farms Salinas, CA	FoodSource Monterey, CA Shipped 980 romaine 24 count 10/07/2011 PO 117235 Shipped 490 romaine 24 count 10/11/2011 PO 117296 Shipped 336 mini bins 10/11/2011 PO 117296	GO Fresh Minneapolis,, MN Received product 10/11/2011 Processed (chopped) romaine and packaged 10/12/2011 Received product 10/14/2011 Processed (chopped) romaine and packaged 10/14, 17 & 18/2011	Sysco Foods Blaine, MN Received product 10/13/2011 180-4/2.5 lb. cases 70-6/2 lb. cases Sysco Foods Blaine MN Received product 10/14/2011 240-4/2.5 lb. cases 100- 6/2 lb. cases 10/17/2011 90-4/2.5 lb. cases 40- 6/2 lb. cases 10/18/2011 30-4/2.5 lb. cases 40- 6/2 lb. cases 10/19/2011 150-4/2.5 lb. cases 20- 6/2 lb. cases	ARA Mark University of Minnesota dining Centennial Hall, Minneapolis, MN campus, received product from Sysco Foods on: 10/17/2011 7 cases 4/2.5 lb. 10/18/2011 5 cases 4/2.5 lb. 10/20/2011 2 cases 4/2.5 lb. 10/21/2011 4 cases 6/2 lb. Onset date of illness: 10/24/2011 Meal dates: 10/19/2011 10/20/2011 10/21/2011 10/22/2011

The conclusion from this traceback investigation is that C&E Farms was the only common supplier of romaine lettuce to both Vaughan's and Go Fresh, and both of these entities utilized the raw romaine from Gubser Ranch Lot 21R23 in the production of lettuce that wound up in Schnucks grocery stores during the outbreak exposure period, as well as at University of Minnesota's Centennial Hall. Additionally, counsel for Vaughans has confirmed that Vaughan's was not buying or receiving any romaine product from Bogiatto, Dole, D'arrigo, Epic Veg, or Green Giant during September or October 2011. The reason that this is relevant is because Go Fresh received romaine lettuce product from these suppliers during the relevant time frame.

III. THE *E. COLI* O157:H7 BACTERIA

A. Sources, Characteristics, and Identification

E. coli is an archetypal commensal bacterial species that lives in mammalian intestines. *E. coli* O157:H7 is one of thousands of serotypes *Escherichia coli*.[2] The combination of letters and numbers in the name of the *E. coli* O157:H7 refers to the specific antigens (proteins which provoke an antibody response) found on the body and tail or flagellum[3] respectively and distinguish it from other types of *E. coli.*[4] Most serotypes of *E. coli* are harmless and live as normal flora in the intestines of healthy humans and animals.[5] The *E. coli* bacterium is among the most extensively studied microorganism.[6] The testing done to distinguish *E. coli* O157:H7 from its other *E. coli* counterparts is called serotyping.[7] Pulsed-field gel electrophoresis (PFGE),[8] sometimes also referred to as genetic fingerprinting, is used to compare *E. coli* O157:H7 isolates to determine if the strains are distinguishable.[9] A technique called multilocus

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[2] *E. coli* bacteria were discovered in the human colon in 1885 by German bacteriologist Theodor Escherich. Feng, Peter, Stephen D. Weagant, Michael A. Grant, *Enumeration of* Escherichia coli *and the Coliform Bacteria*, in BACTERIOLOGICAL ANALYTICAL MANUAL (8th Ed. 2002), http://www.cfsan.fda.gov/~ebam/bam-4.html. Dr. Escherich also showed that certain strains of the bacteria were responsible for infant diarrhea and gastroenteritis, an important public health discovery. *Id*. Although the bacteria were initially called Bacterium *coli*, the name was later changed to *Escherichia coli* to honor its discoverer. *Id*.

[3] Not all *E. coli* are motile. For example, *E. coli* O157:H7 which lack flagella are thus *E. coli* O157:NM for non-motile.

[4] CDC, Escherichia coli *O157:H7, General Information, Frequently Asked Questions: What is* Escherichia coli *O157:H7?*, http://www.cdc.gov/ncidod/dbmd/diseaseinfo/escherichiacoli_g.htm.

[5] Marion Nestle, *Safe Food: Bacteria, Biotechnology, and Bioterrorism*, 40-41 (1st Pub. Ed. 2004).

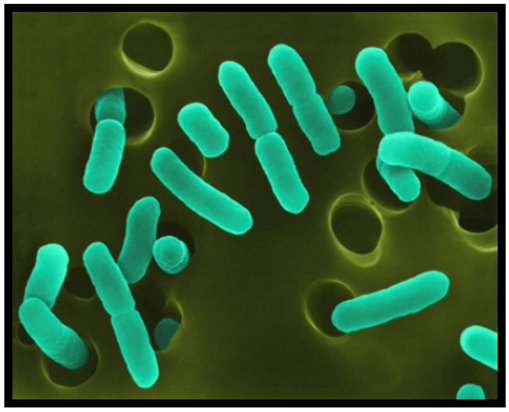
[6] James M. Jay, MODERN FOOD MICROBIOLOGY at 21 (6th ed. 2000). ("This is clearly the most widely studied genus of all bacteria.")

[7] Beth B. Bell, MD, MPH, *et al. A Multistate Outbreak of* Escherichia coli *O157:H7-Associated Bloody Diarrhea and Hemolytic Uremic Syndrome from Hamburgers: The Washington Experience*, 272 JAMA (No. 17) 1349, 1350 (Nov. 2, 1994) (describing the multiple step testing process used to confirm, during a 1993 outbreak, that the implicated bacteria were *E. coli* O157:H7).

[8] Jay, *supra* note 6, at 220-21 (describing in brief the PFGE testing process).

[9] *Id*. Through PFGE testing, isolates obtained from the stool cultures of probable outbreak cases can be compared to the genetic fingerprint of the outbreak strain, confirming that the person was in fact part of the outbreak. Bell, *supra* note 7, at 1351-52. Because PFGE testing soon proved to be such a powerful outbreak investigation tool, PulseNet, a national database of PFGE test results was created. Bala Swaminathan, *et al*. *PulseNet: The Molecular Subtyping Network for Foodborne Bacterial Disease Surveillance, United States*, 7

variable number of tandem repeats analysis (MLVA) is used to determine precise classification when it is difficult to differentiate between isolates with indistinguishable or very similar PFGE patterns.[10]



The* E. coli *O157:H7 Bacteria

E. coli O157:H7 was first recognized as a pathogen in 1982 during an investigation into an outbreak of hemorrhagic colitis[11] associated with consumption of hamburgers from a fast food chain restaurant.[12] Retrospective examination of more than three thousand *E. coli* cultures obtained between 1973 and 1982 found only one (1) isolation with serotype O157:H7, and that was a case in 1975.[13] In the ten (10) years that followed there were approximately thirty (30) outbreaks recorded in the United States.[14] This number is likely misleading, however, because *E. coli* O157:H7 infections did not become a reportable disease in any state until 1987 when Washington became the first state to mandate its reporting to public health authorities.[15] As a

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Emerging Infect. Dis. (No. 3) 382, 382-89 (May-June 2001) (recounting the history of PulseNet and its effectiveness in outbreak investigation).

[10] Konno T. *et al. Application of a multilocus variable number of tandem repeats analysis to regional outbreak surveillance of Enterohemorrhagic* Escherichia coli *O157:H7 infections*. Jpn J Infect Dis. 2011 Jan; 64(1): 63-5.

[11] "[A] type of gastroenteritis in which certain strains of the bacterium *Escherichia coli* (*E. coli*) infect the large intestine and produce a toxin that causes bloody diarrhea and other serious complications." The Merck Manual of Medical Information, 2nd Home Ed. Online, http://www.merck.com/mmhe/sec09/ch122/ch122b.html.

[12] L. Riley, *et al*. *Hemorrhagic Colitis Associated with a Rare Escherichia coli Serotype*, 308 New. Eng. J. Med. 681, 684-85 (1983) (describing investigation of two outbreaks affecting at least 47 people in Oregon and Michigan both linked to apparently undercooked ground beef). Chinyu Su, MD & Lawrence J. Brandt, MD, *Escherichia coli O157:H7 Infection in Humans*, 123 Annals Intern. Med. (Issue 9), 698-707 (describing the epidemiology of the bacteria, including an account of its initial discovery).

[13] Riley, *supra* note 12 at 684. *See also* Patricia M. Griffin & Robert V. Tauxe, *The Epidemiology of Infections Caused by* Escherichia coli *O157:H7, Other Enterohemorrhagic* E. coli*, and the Associated Hemolytic Uremic Syndrome*, 13 Epidemiologic Reviews 60, 73 (1991).

[14] Peter Feng, Escherichia coli *Serotype* O157*:H7: Novel Vehicles of Infection and Emergence of Phenotypic Variants*, 1 Emerging Infect. Dis. (No. 2), 47, 47 (April-June 1995) (noting that, despite these earlier outbreaks, the bacteria did not receive any considerable attention until ten years later when an outbreak occurred 1993 that involved four deaths and over 700 persons infected).

[15] William E. Keene, *et al*. *A Swimming-Associated Outbreak of Hemorrhagic Colitis Caused by* Escherichia coli *O157:H7 and Shigella Sonnei,* 331 New Eng. J. Med. 579 (Sept. 1, 1994). *See also* Stephen M. Ostroff, MD,

result, only the most geographically concentrated outbreak would have garnered enough notice to prompt further investigation.[16]

E. coli O157:H7's ability to induce injury in humans is a result of its ability to produce numerous virulence factors, most notably Shiga-like toxins.[17] Shiga toxin (Stx) has multiple variants (e.g. Stx1, Stx2, Stx2c), and acts like the plant toxin ricin by inhibiting protein synthesis in endothelial and other cells.[18] Shiga toxin is one of the most potent toxins known.[19] In addition to Shiga toxins, *E. coli* O157:H7 produces numerous other putative virulence factors including proteins, which aid in the attachment and colonization of the bacteria in the intestinal wall and which can lyse red blood cells and liberate iron to help support *E. coli* metabolism.[20]

E. coli O157:H7 evolved from enteropathogenic *E. coli* serotype O55:H7, a cause of non-bloody diarrhea, through the sequential acquisition of phage-encoded Stx2, a large virulence plasmid, and additional chromosomal mutations.[21] The rate of genetic mutation of *E. coli* O157:H7 indicates that the common ancestor of current *E. coli* O157:H7 clades[22] likely existed some 20,000 years ago.[23] *E. coli* O157:H7 is a relentlessly evolving organism[24], constantly mutating and acquiring new characteristics, including virulence factors that make the emergence

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John M. Kobayashi, MD, MPH, and Jay H. Lewis*, Infections with* Escherichia coli *O157:H7 in Washington State: The First Year of Statewide Disease Surveillance*, 262 JAMA (No. 3) 355, 355 (July 21, 1989). ("It was anticipated the reporting requirement would stimulate practitioners and laboratories to screen for the organism.")

[16] *See* Keene, *supra* note 15 at 583. ("With cases scattered over four counties, the outbreak would probably have gone unnoticed had the cases not been routinely reported to public health agencies and investigated by them.") With improved surveillance, mandatory reporting in 48 states, and the broad recognition by public health officials that *E. coli* O157:H7 was an important and threatening pathogen, there were a total of 350 reported outbreaks from 1982-2002. Josef M. Rangel, *et al*. *Epidemiology of* Escherichia coli *O157:H7 Outbreaks, United States, 1982-2002*, 11 Emerging Infect. Dis. (No. 4) 603, 604 (April 2005).

[17] Griffin & Tauxe, *supra* note 13, at 61-62 (noting that the nomenclature came about because of the resemblance to toxins produced by *Shigella dysenteries*).

[18] Sanding K, *Pathways followed by ricin and Shiga toxin into cells,* Histochemistry and Cell Biology, vol. 117, no. 2:131-141 (2002). Endothelial cells line the interior surface of blood vessels. They are known to be extremely sensitive to *E. coli* O157:H7, which is cytotoxigenic to these cells making them a primary target during STEC infections.

[19] Johannes L, *Shiga toxins—from cell biology to biomedical applications*. Nat Rev Microbiol 8, 105-116 (February 2010). Suh JK, *et al. Shiga Toxin Attacks Bacterial Ribosomes as Effectively as Eucaryotic Ribosomes, Biochemistry, 37* (26); 9394–9398 (1998).

[20] Welinder-Olsson C, Kaijser B. *Enterohemorrhagic Escherichia coli (EHEC).* Scand J. Infect Dis. 37(6-7): 405-16 (2005). *See also* USDA Food Safety Research Information Office *E. coli* O157:H7 Technical Fact Sheet: *Role of 60-Megadalton Plasmid (p0157) and Potential Virulence Factors*, http://fsrio.nal.usda.gov/document_fsheet.php?product_id=225.

[21] Kaper JB and Karmali MA. *The Continuing Evolution of a Bacterial Pathogen*. PNAS vol. 105 no. 12 4535-4536 (March 2008). Wick LM, *et al. Evolution of genomic content in the stepwise emergence of Escherichia coli O157:H7. J Bacteriol* 187:1783–1791(2005).

[22] A group of biological taxa (as species) that includes all descendants of one common ancestor.

[23] Zhang W, *et al. Prob*ing *genomic diversity and evolution* of Escherichia coli *O157 by s*ing*le nucleotide polymorphisms. Genome Res* 16:757–767 (2006).

[24] Robins-Browne RM. *The relentless evolution* of *pathogenic* Escherichia coli. *Clin Infec Dis*. 41:793–794 (2005).

of more dangerous variants a constant threat.[25] The CDC has emphasized the prospect of emerging pathogens as a significant public health threat for some time.[26]

Although foods of a bovine origin are the most common cause of both outbreaks and sporadic cases of *E. coli* O157:H7 infections[27], outbreak of illnesses have been linked to a wide variety of food items. For example, produce has, since at least 1991, been the source of substantial numbers of outbreak-related *E. coli* O157:H7 infections.[28] Other unusual vehicles for *E. coli* O157:H7 outbreaks have included unpasteurized juices, yogurt, dried salami, mayonnaise, raw milk, game meats, sprouts, and raw cookie dough.[29]

According to a recent study, an estimated 93,094 illnesses are due to domestically acquired *E. coli* O157:H7 each year in the United States.[30] Estimates of foodborne acquired O157:H7 cases result in 2,138 hospitalizations and 20 deaths annually.[31] The colitis caused by *E. coli* O157:H7 is characterized by severe abdominal cramps, diarrhea that typically turns bloody within twenty-four (24) hours, and sometimes fevers.[32] The incubation period—which is to say the time from exposure to the onset of symptoms—in outbreaks is usually reported as three(3) to four (4) days, but may be as short as one (1) day or as long as ten (10) days.[33] Infection can occur in people of all ages but is most common in children.[34] The duration of an uncomplicated illness can range from one (1) to twelve (12) days.[35] In reported outbreaks, the rate of death is 0-2%, with rates running as high as 16-35% in outbreaks involving the elderly, like those have occurred at nursing homes.[36]

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[25] Manning SD, *et al.* Variation in virulence among clades of Escherichia coli O157:H7 associated with disease outbreaks. PNAS vol. 105 no. 12 4868-4873 (2008). ("These results support the hypothesis that the clade 8 lineage has recently acquired novel factors that contribute to enhanced virulence. Evolutionary changes in the clade 8 subpopulation could explain its emergence in several recent foodborne outbreaks; however, it is not clear why this virulent subpopulation is increasing in prevalence.")

[26] Robert A. Tauxe, *Emerging Foodborne Diseases: An Evolving Public Health Challenge*, 3 Emerging Infect. Dis. (No. 4) 425, 427 (Oct.-Dec. 1997). ("After 15 years of research, we know a great deal about infections with *E. coli* O157:H7, but we still do not know how best to treat the infection, nor how the cattle (the principal source of infection for humans) themselves become infected.")

[27] CDC, *Multistate Outbreak of Escherichia coli O157:H7 Infections Associated With Eating Ground Beef—United States, June-July 2002*, 51 MMWR 637, 638 (2002) *reprinted in* 288 JAMA (No. 6) 690 (Aug. 14, 2002).

[28] Rangel, *supra* note 16, at 605.

[29] Feng, *supra* note 14, at 49. *See also* USDA Bad Bug Book, *Escherichia coli* O157:H7, http://www.fda.gov/food/foodsafety/foodborneillness/foodborneillnessfoodbornepathogensnaturaltoxins/badbugbook/ucm071284.htm.

[30] Scallan E, *et al. Foodborne illness acquired in the United States –major pathogens*, Emerging Infect. Dis. Jan. (2011), http://www.cdc.gov/EID/content/17/1/7.htm.

[31] *Id.*, Table 3.

[32] Griffin & Tauxe, *supra* note 13, at 63.

[33] Centers for Disease Control, Division of Foodborne, Bacterial and Mycotic Diseases, *Escherichia coli* general information, http://www.cdc.gov/nczved/dfbmd/disease_listing/stec_gi.html. *See also* PROCEDURES TO INVESTIGATE FOODBORNE ILLNESS, 107 (IAFP 5th Ed. 1999) (identifying incubation period for *E. coli* O157:H7 as "1 to 10 days, typically 2 to 5").

[34] Su & Brandt, *supra* note 12 ("the young are most often affected").

[35] Tauxe, *supra* note 26, at 1152.

[36] *Id*.

What makes *E. coli* O157:H7 remarkably dangerous is its very low infectious dose,[37] and how relatively difficult it is to kill these bacteria.[38] Unlike *Salmonella*, for example, which usually requires something approximating an "egregious food handling error, *E. coli* O157:H7 in ground beef that is only slightly undercooked can result in infection,"[39] as few as twenty (20) organisms may be sufficient to infect a person and, as a result, possibly kill them.[40] And unlike generic *E. coli*, the O157:H7 serotype multiplies at temperatures up to 44°F, survives freezing and thawing, is heat resistant, grows at temperatures up to 111°F, resists drying, and can survive exposure to acidic environments.[41]

And, finally, to make it even more of a threat, *E. coli* O157:H7 bacteria are easily transmitted by person-to-person contact.[42] There is also the serious risk of cross-contamination between raw meat and other food items intended to be eaten without cooking. Indeed, a principle and consistent criticism of the USDA *E. coli* O157:H7 policy is the fact that it has failed to focus on the risks of cross-contamination versus that posed by so-called improper cooking.[43] With this pathogen, there is ultimately no margin of error. It is for this precise reason that the USDA has repeatedly rejected calls from the meat industry to hold consumers primarily responsible for *E. coli* O157:H7 infections caused, in part, by mistakes in food handling or cooking.[44]

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[37] Griffin & Tauxe, *supra* note 13, at 72. ("The general patterns of transmission in these outbreaks suggest that the infectious dose is low.")

[38] V.K. Juneja, O.P. Snyder, A.C. Williams, and B.S. Marmer, *Thermal Destruction of* Escherichia coli *O157:H7 in Hamburger*, 60 J. Food Prot. (vol. 10). 1163-1166 (1997) (demonstrating that, if hamburger does not get to 130°F, there is no bacterial destruction, and at 140°F, there is only a 2-log reduction of *E. coli* present).

[39] Griffin & Tauxe, *supra* note 13, at 72 (noting that, as a result, "fewer bacteria are needed to cause illness that for outbreaks of salmonellosis"). Nestle, *supra* note 5, at 41. ("Foods containing *E. coli* O17:H7 must be at temperatures high enough to kill *all* of them.") (italics in original)

[40] Patricia M. Griffin, *et al*. *Large Outbreak of* Escherichia coli *O157:H7 Infections in the Western United States: The Big Picture*, in RECENT ADVANCES IN VEROCYTOTOXIN-PRODUCING ESCHERICHIA COLI INFECTIONS, at 7 (M.A. Karmali & A. G. Goglio eds. 1994). ("The most probable number of *E. coli* O157:H7 was less than 20 organisms per gram.") There is some inconsistency with regard to the reported infectious dose. *Compare* Chryssa V. Deliganis, *Death by Apple Juice: The Problem of Foodborne Illness, the Regulatory Response, and Further Suggestions for Reform*, 53 Food Drug L.J. 681, 683 (1998) ("as few as ten") *with* Nestle, *supra* note 5, at 41 ("less than 50"). Regardless of these inconsistencies, everyone agrees that the infectious dose is, as Dr. Nestle has put it, "a miniscule number in bacterial terms." *Id*.

[41] Nestle, *supra* note 5, at 41.

[42] Griffin & Tauxe, *supra* note 13, at 72. The apparent "ease of person-to-person transmission…is reminiscent of *Shigella*, an organism that can be transmitted by exposure to extremely few organisms." *Id*. As a result, outbreaks in places like daycare centers have proven relatively common. Rangel, *supra* note 16, at 605-06 (finding that 80% of the 50 reported person-to-person outbreak from 1982-2002 occurred in daycare centers).

[43] *See, e.g.* National Academy of Science, Escherichia coli *O157:H7 in Ground Beef: Review of a Draft Risk Assessment*, Executive Summary, at 7 (noting that the lack of data concerning the impact of cross-contamination of *E. coli* O157:H7 during food preparation was a flaw in the Agency's risk-assessment), http://www.nap.edu/books/0309086272/html/.

[44] *Kriefall v. Excel*, 265 Wis.2d 476, 506, 665 N.W.2d 417, 433 (2003). ("Given the realities of what it saw as consumers' food-handling patterns, the [USDA] bored in on the only effective way to reduce or eliminate food-borne illness"—*i.e.*, making sure that "the pathogen had not been present on the raw product in the first place.")

E. coli O157:H7 infection may lead to severe complications, both acute and chronic. Hemolytic uremic syndrome (HUS) is a potentially fatal complication of the infection, discussed in detail below. *E. coli* O157:H7 infection is also linked to the development of post-infectious irritable bowel syndrome (IBS). The Walkerton Health Study notes that, "Between 5% and 30% of patients who suffer an acute episode of infectious gastroenteritis develop chronic gastrointestinal symptoms despite clearance of the inciting pathogens."[45] There is a strong and significant relationship between acute enteric infection and subsequent IBS symptoms.[46] Irritable bowel syndrome (IBS) is a chronic disorder characterized by alternating bouts of constipation and diarrhea, both of which are generally accompanied by abdominal cramping and pain.[47] In one recent study, over one-third of IBS sufferers had had IBS for more than ten years, with their symptoms remaining fairly constant over time.[48] IBS sufferers typically experienced symptoms for an average of 8.1 days per month.[49]

Not surprisingly, *E. coli* O157:H7 infection is associated with long-term emotional disruption as well, not just for the victim, but also for entire families. A recent study reported that "parents experienced long-term emotional distress and substantive disruption to family and daily life" following an *E. coli* O157:H7 infection in the family.[50]

B. Hemolytic Uremic Syndrome (HUS)

E. coli O157:H7 infections can lead to a severe, life-threatening complication called hemolytic uremic syndrome (HUS).[51] HUS accounts for the majority of the acute deaths and chronic injuries caused by the bacteria.[52] HUS occurs in 2-7% of victims, primarily children,

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(citing Pathogen Reduction, 61 Fed. Reg. at 38966)

[45] J. Marshall, *et al. Incidence and Epidemiology of Irritable Bowel Syndrome After a Large Waterborne Outbreak of Bacterial Dysentery*, Gastro., 2006; 131; 445-50 (hereinafter "Walkerton Health Study" or "WHS"). The WHS followed one of the largest *E. coli* O157:H7 outbreaks in the history of North America. Contaminated drinking water caused over 2,300 people to be infected with *E. coli* O157:H7, resulting in 27 recognized cases of HUS, and 7 deaths. *Id*. at 445. The WHS followed 2,069 eligible study participants. *Id*.

[46] WHS, *supra* note 45, at 449.

[47] A.P.S. Hungin, *et al. Irritable Bowel Syndrome in the United States: Prevalence, Symptom Patterns and Impact*, Aliment Pharmacol. Ther. 2005:21 (11); 1365-75.

[48] *Id*. at 1367.

[49] *Id*.

[50] Pollock, KG, *et al.* Pyschosomatics (2009), May-Jun; 50(3):263-9.

[51] Griffin & Tauxe, *supra* note 13, at 65-68. *See also* Josefa M. Rangel, *et al*. *Epidemiology of* Escherichia coli *O157:H7 Outbreaks, United States, 1982-2002*, 11 Emerging Infect. Dis. (No. 4) 603 (April 2005) (noting that HUS is characterized by the diagnostic triad of hemolytic anemia—destruction of red blood cells, thrombocytopenia—low platelet count, and renal injury—destruction of nephrons often leading to kidney failure).

[52] Richard L. Siegler, MD, *The Hemolytic Uremic Syndrome*, 42 Ped. Nephrology, 1505 (Dec. 1995) (noting that the diagnostic triad of hemolytic anemia, thrombocytopenia, and acute renal failure was first described in 1955). ("[HUS] is now recognized as the most frequent cause of acute renal failure in infants and young children.") *See also* Beth P. Bell, MD, MPH, *et al*. *Predictors of Hemolytic Uremic Syndrome in Children During a Large Outbreak of Escherichia coli O157:H7 Infections*, 100 Pediatrics 1, 1 (July 1, 1997), http://www.pediatrics.org/cgi/content/full/100/1/e12.

with onset five (5) to ten (10) days after diarrhea begins.[53] It is the most common cause of renal failure in children.[54] Approximately half of the children who suffer HUS require dialysis, and at least 5% of those who survive have long-term renal impairment.[55] The same number suffers severe brain damage.[56] While somewhat rare, serious injury to the pancreas, resulting in death or the development of diabetes can also occur.[57] There is no cure or effective treatment for HUS.[58] And, tragically, as too many parents can attest, children with HUS too often die.[59]

HUS is believed to develop when the toxin from the bacteria, known as Shiga-like toxin (SLT), enters the circulation through the inflamed bowel wall.[60] SLT, and most likely other chemical mediators, attach to receptors on the inside surface of blood vessel cells (endothelial cells) and initiate a chemical cascade that results in the formation of tiny thrombi (blood clots) within these vessels.[61] Some organs seem more susceptible, perhaps due to the presence of increased numbers of receptors, and include the kidney, pancreas, and brain.[62] By definition, when fully expressed, HUS presents with the triad of hemolytic anemia (destruction of red blood cells), thrombocytopenia (low platelet count), and renal failure (loss of kidney function).[63]

As already noted, there is no known therapy to halt the progression of HUS. HUS is a frightening complication that even in the best American centers has a notable mortality rate.[64] Among survivors, at least five percent (5%) will suffer end stage renal disease (ESRD) with the resultant need for dialysis or transplantation.[65] But, "[b]ecause renal failure can progress slowly

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[53] Tauxe, *supra* note 26, at 1152. *See also* Nasia Safdar, MD, *et al*. *Risk of Hemolytic Uremic Syndrome After Treatment of* Escherichia coli *O157:H7 Enteritis: A Meta-analysis*, 288 JAMA (No. 8) 996, 996 (Aug. 28, 2002). ("*E. coli* serotype O157:H7 infection has been recognized as the most common cause of HUS in the United States, with 6% of patients developing HUS within 2 to 14 days of onset of diarrhea."). Amit X. Garg, MD, MA, *et al*. *Long-term Renal Prognosis of Diarrhea-Associated Hemolytic Uremic Syndrome: A Systematic Review, Meta-Analysis, and Meta-regression*, 290 JAMA (No. 10) 1360, 1360 (Sept. 10, 2003). ("Ninety percent of childhood cases of HUS are…due to Shiga-toxin producing *Escherichia coli*.")

[54] Su & Brandt, *supra* note 12.

[55] Safdar, *supra* note 53, at 996 (going on to conclude that administration of antibiotics to children with *E. coli* O157:H7 appeared to put them at higher risk for developing HUS).

[56] Richard L. Siegler, MD, *Postdiarrheal Shiga Toxin-Mediated Hemolytic Uremic Syndrome*, 290 JAMA (No. 10) 1379, 1379 (Sept. 10, 2003).

[57] Pierre Robitaille, *et al*., *Pancreatic Injury in the Hemolytic Uremic Syndrome*, 11 Pediatric Nephrology 631, 632 (1997) ("although mild pancreas involvement in the acute phase of HUS can be frequent").

[58] Safdar, *supra* note 53, at 996. *See also* Siegler, *supra* note 56, at 1379. ("There are no treatments of proven value, and care during the acute phase of the illness, which is merely supportive, has not changed substantially during the past 30 years.")

[59] Su & Brandt, *supra* note 12("the mortality rate is 5-10%"). *See also Kriefall*, 265 N.W.2d at 483 ("three-year old Brianna Kriefall died from food that everyone party to this appeal…recognize was cross-contaminated by *E. coli* O157:H7 bacteria from meat sold by Excel.")

[60] Garg, *supra* note 53, at 1360.

[61] *Id*. Siegler, *supra* note 52, at 1509-11 (describing what Dr. Siegler refers to as the "pathogenic cascade" that results in the progression from colitis to HUS).

[62] Garg, *supra* note 53, at 1360. *See also* Su & Brandt, *supra* note 12, at 700.

[63] Garg, *supra* note53, at 1360. *See also* Su & Brandt, *supra* note 12, at 700.

[64] Siegler, *supra* note 52, at 1519 (noting that in a "20-year Utah-based population study, 5% dies, and an equal number of survivors were left with end-stage renal disease (ESRD) or chronic brain damage.")

[65] Garg, *supra* note 53, at 1366-67.

over decades, the eventual incidence of ESRD cannot yet be determined."[66] Other long-term problems include the risk for hypertension, proteinuria (abnormal amounts of protein in the urine that can portend a decline in renal function), and reduced kidney filtration rate.[67] Since the longest available follow-up studies of HUS victims are 25 years, an accurate lifetime prognosis is not really available and remains controversial.[68] All that can be said for certain is that HUS causes permanent injury, including loss of kidney function, and it requires a lifetime of close medical monitoring.

C. Post-infectious Irritable Bowel Syndrome

A recently-published study surveyed the extant scientific literature and noted that post-infectious irritable bowel syndrome (PI-IBS) is a common clinical phenomenon first-described over five (5) decades ago.[69] The Walkerton Health Study (WHS) further notes that:

> Between 5% and 30% of patients who suffer an acute episode of infectious gastroenteritis develop chronic gastrointestinal symptoms despite clearance of the inciting pathogens.[70]

In terms of its own data, the "study confirm[ed] a strong and significant relationship between acute enteric infection and subsequent IBS symptoms."[71] The WHS also identified risk-factors for subsequent IBS, including: younger age; female sex; and four features of the acute enteric illness—diarrhea for > 7 days, presence of blood in stools, abdominal cramps, and weight loss of at least ten (10) pounds.[72]

Irritable bowel syndrome (IBS) is a chronic disorder characterized by alternating bouts of constipation and diarrhea, both of which are generally accompanied by abdominal cramping and pain.[73] In one recent study, over one-third of IBS sufferers had had IBS for more than ten (10) years, with their symptoms remaining fairly constant over time.[74] IBS sufferers typically experienced symptoms for an average of 8.1 days per month.[75]

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66 Siegler, *supra* note 52, at 1519.

67 *Id*. at 1519-20. *See also* Garg, *supra* note 53, at 1366-67.

68 Garg, *supra* note 53, at 1368.

69 J. Marshall, *et al. Incidence and Epidemiology of Irritable Bowel Syndrome After a Large Waterborne Outbreak of Bacterial Dysentery*, Gastro., 2006; 131;445-50 (hereinafter "Walkerton Health Study" or "WHS"). The WHS followed one of the largest *E. coli* O157:H7 outbreaks in the history of North America. Contaminated drinking water caused over 2,300 people to be infected with *E. coli* O157:H7, resulting in 27 recognized cases of HUS, and 7 deaths. *Id*. at 445. The WHS followed 2,069 eligible study participants. *Id*.

70 *Id*. at 445 (citing multiple sources).

71 WHS, *supra* note 34, at 449.

72 *Id*. at 447.

73 A.P.S. Hungin, *et al. Irritable Bowel Syndrome in the United States: Prevalence, Symptom Patterns and Impact*, Aliment Pharmacol. Ther. 2005:21 (11); 1365-75.

74 *Id*.at 1367.

75 *Id*.

As would be expected from a chronic disorder with symptoms of such persistence, IBS sufferers required more time off work, spent more days in bed, and more often cut down on usual activities, when compared with non-IBS sufferers.[76] And even when able to work, a significant majority (67%), felt less productive at work because of their symptoms.[77] IBS symptoms also have a significantly deleterious impact on social well-being and daily social activities[78], such as undertaking a long drive, going to a restaurant, or taking a vacation.[79] Finally, while a patient's psychological state may influence the way in which he or she copes with illness, and responds to treatment, there is no evidence that supports the theory that psychological disturbances in fact cause IBS or its symptoms.[80]

IV. LISA BRYANT'S *E. COLI* O157:H7 ILLNESS

Lisa K. Bryant is a 50 year old bookkeeper who lives with her husband James in Wentzville, Missouri. They have two grown daughters in their 30's. Lisa is employed by Industrial Aid in St. Louis. She has been a generally healthy woman without any significant medical history of serious illnesses or major surgeries, other than cesarean section delivery of her children. She saw her family doctor on October 6, 2011 in follow-up of exercise induced asthma and reactive hypertension, which were stable. Other than some knee pain from bursitis, she was well.

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[76] *Id*. at 1368.

[77] *Id*.

[78] Post-infective Irritable Bowel Syndrome is a well-known sequelae of *Salmonella* infection, particularly among women whose acute infectious were protracted and severe. *See* K.R. Neal, L. Barker, R.C. Spiller, "Prognosis in post-infective irritable bowel syndrome: a six year follow up study," *Gut*, Vol. 51, at 410-413 (January 28, 2002), *available at* http://gut.bmjjournals.com/cgi/content/full/51/3/410; Luis A. Garcia Rodriguez & Ana Ruigomez, "Increased risk of irritable bowel syndrome after bacterial gastroenteritis: cohort study," *BMJ*, Vol. 318 at 565-66 (Feb. 27, 1999), http://bmj.bmjjournals.com/cgi/content/full/318/7183/565; and Sally Parry & Ian Forgacs, "Intestinal infection and irritable bowel syndrome," *European Journal of Gastroenterology & Hepatology*, Vol. 17(1), at 5-9 (Jan. 2005), http://www.eurojgh.com/pt/re/ejgh/abstract/00042737-200501000-00002.htm.

[79] *Id*.

[80] Amy Foxx-Orenstein, DO, FACG, FACP, *IBS—Review and What's New*, General Medicine 2006:8(3) (Medscape 2006) (collecting and citing studies). Indeed, PI-IBS has been found to be characterized by more diarrhea but less psychiatric illness with regard to its pathogenesis. *See also* Nicholas J. Talley, MD, PhD, *Irritable Bowel Syndrome: From Epidemiology to Treatment*, from American College of Gastroenterology 68th Annual Scientific Meeting and Postgraduate Course (Medscape 2003).

Lisa and her family

Thursday, October 20, 2011 was a workday for Lisa. A co-worker had recently had a salad from the Schnuck's salad bar on Arsenal Street that looked fresh, so Lisa decided to go to Schnuck's for lunch on October 20. She selected a variety of fruits and vegetables for her salad, including romaine lettuce, spinach, broccoli, peas, tomatoes, cantaloupe, and honeydew melon, hardboiled eggs, bacon pieces, and cheese. She paid cash for the salad, and then returned to work to eat lunch.

Onset of Lisa's symptoms occurred the afternoon of Tuesday, October 25, 2011. Symptoms initially included abdominal cramps and diarrhea, which soon became painful enough for her to call the family doctor, Scott Groesch, MD, who called in a prescription for Lomotil. The medication was ineffective, however, and that night, the diarrhea became worse. By early Wednesday morning it had become so severe that she was forced to call in sick to work. By late Wednesday, Lisa began vomiting and also began to notice blood in her stool that continued to get worse throughout the night. Around 3:00 AM on Thursday morning, she asked her husband to take her to the ER.

On October 27 at 3:20 AM, Lisa presented to Progress West Medical Center, where she was evaluated for nausea, vomiting, and diarrhea. Nancy Niemann-Royer, MD was on duty in the ER that morning and she reported the sequence of events leading up to her arrival at the ER.

Dr. Niemann-Royer started Lisa on IV fluids with Zofran for her nausea. A couple of hours later, she gave Lisa one dose of oral Ciprofloxacin 500 mg. Lisa's abdomen was nondistended but demonstrated diffuse tenderness to palpation; the exam was otherwise unremarkable. She was afebrile with normal vital signs. Dr. Royer sent a stool culture to the lab, and also requested *C. difficile* testing and fecal leukocytes. She ordered a CBC and a Basic Metabolic Profile. The fecal leukocyte test was negative, as was the *C. difficile* toxins. Lisa's WBC was elevated at 16.1, and her sodium was marginally low. Dr. Royer diagnosed Lisa with "infectious diarrhea" and discharged her home from the ER after infusing 2 liters of normal saline over the course of two and a half hours of observation. She was also given IV Dilaudid for pain. Dr. Royer prescribed a continued course of Ciprofloxacin to be taken twice a day for

ten days and told Lisa to eat a bland diet. She cautioned her on the appropriate hand washing and other hygiene for infectious diarrhea, and advised her the culture results would take some time to come back.

On October 28 at 9:07 AM, the hospital lab called an alarm value to Dr. Hsu in the ER, reporting that Lisa's stool had tested positive for Shiga toxin. The isolate was sent to the State Lab for confirmation and typing. The stool culture was still too early to call. On October 31, the Missouri State Public Health Laboratory reported confirmation of Shiga toxin-producing organisms, which were identified to be *Escherichia coli* O157:H7.

Lisa did not do well after going home, and on November 2 she returned to the ER at Progress West at 3:56 PM. She reported that nothing was working for her nausea and she continued to have dry heaves and could not stay hydrated. She began having diarrhea again for over 24 hours before coming back in to the ER. David Fouts, MD was on duty in the emergency department that afternoon and he started Lisa on more IV fluids and Zofran, sending in blood work to the lab. Lisa's white count was significantly elevated at 39.6 (RR 4.5-11), and she was anemic with a hemoglobin of 11.1 and hematocrit 31.4%. Her platelets had dropped to 105K, and her BUN and creatinine were elevated to 94 and 3.0. Her liver profile was also marginally abnormal with an ALT 70 and AST 73, and her total bilirubin was elevated to 3.0 (RR 0.1-1.0). Dr. Fauts consulted with the Hospitalist Service, and Drs. Zia and Lotsoff agreed that Lisa should be admitted to the hospital with a continued diagnosis of infectious diarrhea.

On November 3 at 8:10 AM, Nadia Zia, MD came in to do Lisa's admission history and physical, at which time Lisa was reporting extreme lethargy, weakness, and dizziness. Dr. Zia reviewed the onset of Lisa's illness a few days before and the treatment she received in the ER the first time around. While her blood work looked "fine" on the 27th of October, her stool had subsequently grown out E. coli O157:H7 with shiga toxin before she came back in to the ER on November 2. According to Lisa, when she went back home on October 27, she still had bloody diarrhea, had bloody diarrhea over the weekend and Monday her diarrhea had completely stopped. However, on November 2, her diarrhea started again and she felt weaker and came to the ER. Dr. Zia found it notable that Lisa's white count was so elevated, and she was particularly concerned about the low platelets.

At the time of Dr. Zia's exam, Lisa's vital signs were stable and she was afebrile. Her abdomen was generally tender to palpation, but her exam was otherwise unremarkable. Dr. Zia's admit diagnosis was *E. coli* O157:H7 with positive shiga toxin induced diarrhea. She advised supportive care with IV fluids, pain and nausea medications. Dr. Zia felt that Lisa's lab results demonstrated hemolytic uremic syndrome with low hemoglobin and hematocrit and renal insufficiency. Without any neurological signs or fever at that time, she felt they could monitor her hemoglobin and platelets closely. Lisa was placed under observation and a request was put in for both a Gastroenterology and a Nephrology Consultation. Dr. Zia thought they could consider steroids if the counts were decreasing and also consider a Hematology/Oncology Consultation for possible plasmapheresis.

At 9:00 AM, Sarah E. Hagan, PA-C came in for a Gastroenterology Consultation under the supervision of Michael Zerega, MD. She reviewed Lisa's history and did an exam. Ms. Hagan noted that Lisa had already been placed on Ciprofloxacin and Lomotil a few days earlier, but noted that with infectious diarrhea caused by E. coli O157:H7, there was no role for antibiotics or antidiarrheals in such a case. Ms. Hagan was quite concerned about Lisa's worsening anemia and thrombocytopenia and advised a Renal and Hematology Consultation for possible plasma exchange, although she noted that Lisa had shown some improvement in her renal function labs overnight. Until those consults could be obtained, she recommended continued supportive care and IV fluid hydration, as well as advancing Lisa's diet as tolerated. Ms. Hagan discussed Lisa's case with Dr. Zerega, who agreed with that plan.

Henrikas Juknis, MD came in about an hour later for a Renal Consultation, noting Lisa's lab results from the day before, which had included the presence of schistocytes in the peripheral smear. By the time of his exam, Lisa's overnight lab work was back that showed slightly improved BUN and creatinine levels of 86 and 2.2, but her platelets had fallen further to 66K. Dr. Juknis diagnosed Lisa with acute renal failure and possibly hemolytic uremic syndrome. He ordered continued IV fluid hydration with normal saline, and continued lab studies to include urinalysis, LDH, and haptoglobin, and he wanted a renal ultrasound as soon as possible. He felt that Lisa should be transferred to a facility capable of performing plasmapheresis.

On November 3 at 10:50 AM, Jamie Colonnello, MD performed a retroperitoneal sonogram, which showed no evidence of hydronephrosis, although there was a trace amount of free abdominal fluid near the gallbladder of uncertain significance.

Dr. Zerega discussed Lisa's case with the consulting physicians, and she ordered a transfer to Barnes Jewish Hospital as soon as a bed was available.

Lisa recalls the events of her visits to Progress West Medical Center, and then Barnes Jewish Hospital:

> The ER doctor treated me for dehydration, collected a stool sample and sent me home. My husband was scheduled for cervical fusion surgery on Friday morning and I was unable to go with him because the diarrhea and vomiting had not ceased. When my husband came home I tried to care for him but it was extremely difficult due to the unrelenting symptoms that continued to weaken and dehydrate me. I began to have severe abdominal pain and the anti-diarrhea, anti-nausea, and pain medicine I had been prescribed did little to relieve my symptoms. Water was the only thing I could tolerate and by the following Tuesday, I was so weak that I was unable to care for my husband. I finally had to call my parents, who live almost two hours away, to ask for their help. They came the next day and took care of both of us but had to return home. On Thursday I barely moved from the couch and though my husband encouraged me to drink, he noticed that I had taken in very little fluid throughout the day. He called my daughter and she came

> over and insisted that I go back to the ER. I was immediately admitted for supportive care.

After arriving at Barnes Jewish Hospital on November 3, 2011, Amber L. Tierney, MD admitted Lisa into the Medicine Service. Dr. Tierney noted that Lisa had no significant prior medical history before presenting with symptoms of gastroenteritis. Lisa told Dr. Tierney that she had eaten at a Schnuck's salad bar a couple of weeks before, and a few days after that she developed nausea, vomiting and diarrhea. The diarrhea was severe, with multiple episodes per day. The day after the diarrhea started, her stools became bloody and were unresponsive to Imodium.

Lisa explained to Dr. Tierney that she continued to have nausea, vomiting, and diarrhea after she returned home. The cramping and diffuse abdominal pain only became worse, turning to dizziness, lightheadedness, and general weakness. Feeling even worse on November 1, Lisa returned to the ER at Progress West Medical Center. Her white blood cell count had risen to 39.6, up from 16.1, her platelets fell from 324 to 105, and her hemoglobin dropped from 14.3 to 11.1. The ER physician determined that Lisa was in acute renal failure with a creatinine of 3.0, which had risen from 0.7. Lisa reportedly was admitted from the ER to the Medicine floor and started on aggressive IV fluid resuscitation as well as pain and nausea medications, out of concern for hemolytic uremic syndrome.

Continuing her review of Lisa's treatment at Progress West, Dr. Tierney observed that Lisa's bloody stools resolved, although she continued to have diarrhea. At first unable to tolerate solid food, she began to tolerate liquids. A Gastroenterology Consultant recommended continuing with symptoms-only management. However, on the morning of November 3, Lisa's LDH was found to be >1,000 and her platelets dropped even further to 66, and her hemoglobin went down to 8.2. The physicians at Progress West Medical Center recognized that Lisa needed a higher level of care than their facility could offer, and so they transferred her to Barnes Jewish Hospital for further evaluation and possible plasmapheresis for thrombotic thrombocytopenia purpura (TTP).

Lisa recalls the fear that came over her upon learning from the doctors at Progress West that she had a potentially deadly illness:

> The next morning a doctor came into my room and explained to me that my kidneys were failing and that my best hope was a procedure called Plasmapheresis. He told me that they would need to transfer me to Barnes-Jewish in St. Louis for that procedure. I had no idea that my condition was that serious and his sense of urgency and concern really scared me. I called my daughter to let her know what was happening and I ended up breaking down on the phone. She was so upset that her boss told her to leave so she could come to the hospital to be with me. I was transferred to Barnes later that afternoon.

Settled into her hospital room at Barnes Jewish Hospital, Lisa reported to Dr. Tierney that

she had some nausea and had an episode of diarrhea that morning but had not had any since. She was no longer having any abdominal pain and no emesis since transfer. She had noticed no fevers or confusion, and her family reported that they had not noticed any neurological deficits or changes in her personality. She had noticed no further hematochezia, hematemesis, hematuria, easy bruising, rash or gingival bleeding.

To Dr. Tierney's exam, Lisa's vital signs were normal and stable, and she was oxygenating well on room air. Her family was at her bedside. Her abdominal exam was significant for mild tenderness to palpation in the umbilical region, and her bowel sounds were hyperactive, but her exam was otherwise unremarkable. Her significant lab results were WBC 34, hemoglobin 8.9, LDH 1209, bilirubin 2.8, AST 99, ALT 84, BUN 64, serum creatinine 1.67. Dr. Tierney concurred with the Progress West physicians that Lisa's presentation was consistent with hemolytic uremic syndrome (HUS) with TTP, which were secondary to Shiga toxin *E. coli* O157:H7 that she acquired from food poisoning.

Addressing Lisa's diagnosis of TTP, Dr. Tierney consulted with Eric Knoche, MD of Hematology about Lisa's abnormal lab values and did a peripheral smear, which revealed multiple schistocytes. They agreed that Lisa should undergo plasmapheresis (PLEX) that night. She currently had thrombocytopenia, anemia, and renal failure, but did not have any neurologic changes or fever. Her anemia and thrombocytopenia were improved from earlier that morning and her LDH was trending down. Lisa did show signs of end organ damage with increasing liver enzymes and elevated creatinine, although her creatinine was improving. Dr. Tierney ordered an ADAMTS13[81] quantitative level and inhibitor to evaluate for deficiency. She also ordered daily CBC with peripheral smears, daily LDH, bilirubin and creatinine. They would continue symptomatic management with IV fluids, Zofran and Compazine as needed for nausea, and IV Dilaudid for pain. Lisa was already on a clear liquid diet, but that would be advanced as tolerated.

Dr. Tierney ordered further lab tests to follow-up on Lisa's anemia, which was microcytic, angiopathic hemolytic anemia (MAHA) secondary to HUS. She planned to check a direct Coombs level and haptoglobin. Lisa's fibrinogen was elevated at 656 and her reticulocytes were high at 3.6, and Dr. Tierney planned to recheck a CBC after plasmapheresis.

Dr. Tierney also concluded that Lisa's diagnoses included acute kidney injury (AKI), which she felt could be related to the TTP, or perhaps to dehydration. They would keep an eye on her BMP along with her input and output (I/O).

Lisa's transaminitis was also felt to be secondary to TTP and elevated schistocytes. Her elevated bilirubin was likely secondary to the microcytic angiopathic anemia. Dr. Tierney planned to follow-up her liver function panel following plasmapheresis. DVT prophylaxis was currently addressed with sequential compression devices (SCD's).

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[81] ADAMTS13 ADAM metallopeptidase with thrombospondin type 1 motif, 13 [*Homo sapiens*(human)]. Defects in this gene are associated with thrombotic thrombocytopenic purpura. http://www.ncbi.nlm.nih.gov/gene/11093.

At 10:12 PM on November 3, Lisa underwent a central line placement of a triple lumen pheresis catheter, after which David Gierada, MD performed a chest x-ray to verify the correct position. Dr. Knoche ordered daily plasma exchange (PLEX) until remission. On November 4 at 12:45 AM, Lisa's first pheresis procedure was underway. It was over at 2:25 AM and was without complications, and Lisa remained afebrile with stable vital signs.

Lisa recalls the insertion of the pheresis catheter and everything leading up to it as a traumatic event:

> When I arrived at Barnes-Jewish, two doctors came in to talk with me about the seriousness of my condition and the urgency of beginning treatment right away. They explained that they were going to do an emergency procedure to place a catheter into the vein in my groin. They did the procedure in my room, covering me from head to toe with a surgical sheet and preceding [sic] without administering even a local anesthetic. It was extremely painful and frightening. After the catheter was placed they gave me Dilaudid for pain and I finally felt some relief from the abdominal pain, but my memories of the next few days are a little less clear. I believe it was later that night that they began my first plasmapheresis procedure and I received several of those over the next several days.

On November 4, John Cras, MD came in for the Medicine Service to check on Lisa, noting that she was lying comfortably in bed. Her skin was slightly jaundiced on exam, and he noted the lab had found schistocytes on her peripheral smear. He deferred her HUS/TTP management to Hematology. He increased her IV fluids in response to her acute renal failure.

Eric Marshall Knoche, MD came in for a follow-up Hematology Consultation that afternoon for Lisa's hemolytic anemia and thrombocytopenia. He reviewed the onset of Lisa's current illness that began with a hospitalization at Progress West Hospital for nausea, vomiting, and diarrhea. He asked Lisa how this had all started, and she reported that on October 25,[82] she had Schnuck's Salad Bar and later developed abdominal pain and diarrhea. She noted on the following day that the diarrhea had progressed to bloody stools and increased frequency of her diarrhea. He reviewed her first ER visit on October 27, where she received IV fluids, Imodium and Ciprofloxacin. Dr. Knoche found it notable that her hemoglobin dropped to 8.0 and her platelets had decreased to 88, with an LDH of greater than 1200 or beyond the lab's ability for quantification.

At the time of Dr. Knoche's evaluation, Lisa reported having persistent abdominal pain with loose stools approximately twice daily. She felt feverish subjectively but without nausea and vomiting. She was alert and oriented, and her family continued to deny mental status

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[82] As Lisa's earlier statements and the health department records both show, the date of her meal at Schnucks on Arsenal Street was, in fact, October 20, 2011.

changes or confusion. In addition to her other lab results, Dr. Knoche noted that her most recent peripheral smear revealed 12 to 15 schistocytes per high-powered field, and reactive leukocytosis "consistent with leukemoid reaction and giant platelets."

Dr. Knoche agreed with the other physicians' diagnosis of HUS/TTP. In addition to the provision of supportive therapy in addition to placing a plasmapheresis catheter for the initiation of plasma exchange (PLEX), he wanted to watch Lisa's levels for both the ADAMTS inhibitor and ADAMTS absolute quantification, as described above. Given that this was HUS-associated TTP, he did not think there was a clear indication for immunosuppression with steroids. They would offer nutritional supplementation for hematopoiesis with daily folate 5 mg doses. He made no changes to the lab studies already ordered by Dr. Tierney.

On November 5, Dr. Cras came in to see Lisa in the morning. Another plasmapheresis was scheduled for that day. She required a blood transfusion for severe anemia, but her thrombocytopenia had resolved to a platelet count of 185. Dr. Knoche came in, noting that Lisa had tolerated the plasmapheresis well the day before. He advised continued plasmapheresis over the weekend, given the high number of schistocytes on her peripheral smear and increased LDH of 466-591. He also ordered additional blood transfusions to keep her hematocrit greater than 20. Lisa's next plasmapheresis began at 10:25 AM on November 5, and ended at 12:58 PM. It was uncomplicated and her vitals remained normal and stable.

Dr. Cras came in to see Lisa on November 6, noting a further increase in her transaminase levels, which he though was likely secondary to the high number of schistocytes. He changed her DVT prophylaxis to subcutaneous Lovenox.

Lisa's next plasmapheresis began at 3:18 PM on November 6, and ended at 6 PM. It was uncomplicated and her vitals remained normal and stable.

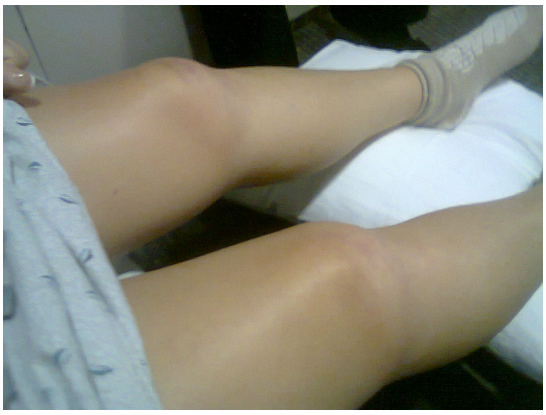
On November 7, Dr. Cras came in to see Lisa, who reported that she felt lightheaded when up walking. Her diarrhea had improved, however, and she was otherwise feeling slightly better. On exam, he noted diffuse mild non-pitting edema, or anasarca. Dr. Knoche came in and held her plasmapheresis for the day because her creatinine had improved and was now down to 1.08. However, her transaminases were further elevated and so Michael Yu, MD performed an x-ray of Lisa's abdomen, which was suspicious for colitis.

Lisa remembers that she was still not feeling well at this point during her hospitalization:

> I still don't know why, but by Monday I was having trouble breathing and found it extremely difficult to talk. I struggled to say even a few words without gasping for air. My hands also began to shake uncontrollably and I began retaining fluid. At one point (I believe I have this right because I was so shocked) I weighed over 160 pounds. I typically weigh around 127 pounds but weighed only 111 pounds when I arrived at the hospital. The nurses began to have difficulty taking blood because I was so swollen. I remember one night, after trying several times, they

had to call in a more experienced nurse and she was finally able to draw blood from my knuckle. I was so weak and carrying so much fluid that it was difficult to walk, or even stand long enough to brush my teeth, and I had to lift my legs with my arms or ask for assistance to lift them onto the bed. The first time I tried to walk, I nearly collapsed before I made it to the nurse's station and my husband had to get me a wheelchair.

On November 8, Dr. Knoche came back in to see Lisa, noting she had required another blood transfusion the day before to keep her blood count up. Her kidney function had improved on IV fluids and he decided to change that over to oral fluids only, as she was hydrating well. An x-ray a day or so before had noted a lung nodule, which was followed by a CT that afternoon. This no longer showed the nodule but did show small moderate bilateral pleural effusion, with trace pulmonary edema and subcutaneous edema. Dr. Cras had already noted the development of anasarca; Lisa was noted to have gained about 30 pounds since admission and felt bloated. Dr. Knoche ordered Lasix for fluid overload.



Lisa's marked swelling of the lower extremities

Later that night, Lisa complained of diplopia ("seeing double"), and so Neurology was called to consult. Necta Lal, MD came in for the consultation and a head CT was done that looked normal. He diagnosed Lisa with isolated Cranial Nerve VI palsy, but did not think it was related to elevated intracranial pressure.

This turn of events was very frightening for Lisa, as she recalls:

The most frightening moment of all came in the middle of the night. My days had begun to run together so I have no idea what night it was, but the nurse woke me to draw blood and I saw two of her. I rubbed my eyes and tried to focus but I continued to see two of everything so clearly that I could not discern which one was real and which was the double. I mentioned it to the nurse and she immediately called the doctor. After examining me, he called for a neurology consult and then sent me for a CAT scan. I was so afraid that I had had a stroke. I called my husband and began sobbing. He was still unable to drive because of

his surgery and so he had to call my parents and ask them to drive him to the hospital. The double vision subsided by morning and as far as I'm aware, they were never able to determine the cause.

By the following morning of November 9, Lisa reported that her double vision was gone and her vision had returned to normal. However, she now reported an "intention tremor" (her words) over the past 24 hours that was causing her to drop food and was affecting her tongue the most. Dr. Lal did not think she had any neurological illness and could find nothing abnormal on exam. Later that night, Lisa's hemoglobin and hematocrit dropped so low, she required a transfusion of 2 units platelets.

On November 10, Dr. Knoche noted that Lisa's platelets were holding stable and continued to hold further plasmapheresis. He discontinued her pheresis catheter. Her liver enzymes had also now returned to normal range, but she continued to be severely anemic and he ordered the transfusion of two more units.

On November 11, Eric Yanamara, MD came in for the Medicine Service and noted that Lisa had responded favorably to the blood transfusion. Her AKI had now resolved with IV fluids, although she now suffered from volume overload and demonstrated continued anasarca. She was being diuresed with Lasix. The doctors all agreed that Lisa had recovered sufficiently to where she could to continue to convalesce at home. Dr. Tierney came in to examine Lisa and write up a discharge summary. She listed Lisa's principal and secondary diagnoses as:

Preliminary diagnosis:
1. Hemolytic uremic syndrome/thrombotic thrombocytopenic purpura.

Secondary diagnoses:
1. Microcytic angiopathic hemolytic anemia.
2. Thrombocytopenia.
3. Acute kidney injury secondary to dehydration.
4. Transaminitis.

At the time of her discharge, Dr. Tierney noted that Lisa was ambulating and tolerating a regular diet, had resolution of her diarrhea, and was stable enough for discharge home. Her hemoglobin was now stable for over 24 hours without a transfusion. Lisa was discharged with a prescription for folic acid and hydrochlorothiazide to continue diuresing her fluid overload. She was advised to follow up with her primary care provider on Monday, November 14 for a post-hospitalization visit and a repeat CBC to evaluate for anemia. Because Lisa's hemoglobin had dropped down to 6.0 during the course of her hospitalization, she was educated on symptoms of anemia and told to come to the ER if she felt lightheaded, dizzy, or had syncope. She was additionally advised to follow-up at the Hematology clinic, who would contact her to make an appointment.

Lisa recalls finally going home from the hospital, only to discover the physical

repercussions of having been so ill:

> I was released from the hospital on November 11, 2011. I was still retaining a large amount of fluid and had to wear my husband's pajamas for nearly a week because I had no other clothes that fit. I worked from home the following week, and the week after that I worked part-time at the office. I returned to work full time the week following. I missed a total of 17 days of work and worked a reduced schedule for another 10 days, which had a considerable impact on my employer.

On November 15, 2011, Lisa presented to see her family doctor, Scott D. Groesch, MD, in follow-up of her hospitalization from HUS related to *E. coli* poisoning. He commented on how severely ill Lisa was while hospitalized. He felt she needed blood work to include a CBC and basic metabolic profile, and he felt she should be referred to a hematologist. Lisa reported feeling generally fatigued, but she was not having any fevers, chills, or night sweats. She had no respiratory symptoms or current GI complaints. Her weight that day was 132#. Dr. Groesch diagnosed Lisa with clinically resolved HUS and planned to follow-up as needed after the lab results were back. Lisa's lab work came back showing anemia with a hematocrit of 30.6%, and an elevated LDH of 399 (RR 100-200), but normal platelets and normal kidney function.

On November 29, 2011, Lisa's lab work came back showing mild anemia with a hematocrit of 33.5%, but normal platelets and normal kidney function.

On June 11, 2012, Lisa went back to see Dr. Groesch in follow-up of her hospitalization for HUS. She was still having problems with perceptible edema, which had continued to be bothersome since she left the hospital the previous Fall. Since that time, Lisa continued to have a propensity for fluid retention. She had stopped her HCTZ a month earlier for unexplained reasons, after which she definitely noticed further edema. She was not having any shortness of breath, cough, wheezing, or other respiratory complaints. Her weight was 130# that day. Dr. Groesch continued to diagnose Lisa with HUS, resolved clinically. Lisa reported normal home blood pressures, which she would continue to monitor. Dr. Groesch checked a CMP, TSH, CBC and BNP.

On July 2, 2012, Lisa's lab work came back normal, with normal blood count, thyroid, and BNP values.

On October 30, 2013, Lisa returned to see Dr. Groesch, who performed a complete physical exam. She was having some heel pain but had no other major complaints. Dr. Groesch listed "hemolytic uremic syndrome" among Lisa's "Active Problems," which also included palpitations and reactive hypertension. She had resumed her diuretic (HCTZ) and was still taking 25 mg/day, and she had a Ventolin inhaler to use as needed for exercise induced asthma. She weighed 132 pounds that day, about the same as her visit in October 2012.

On November 22, 2013, Lisa presented to Mason Ridge Surgery Center, where she

underwent a colonoscopy by Robert Shuman, MD. Shuman identified a normal appearing colon with no polyps or other abnormalities.

Lisa reflects on her health since her *E. coli* O157:H7 HUS illness:

> I experienced the loss of a considerable amount of muscle due to the extreme circumstances of my illness and it took nearly a full year to regain my strength and ability to perform most physical activities at the level I was performing before the *E. coli*/HUS. There are some things that are still not the same. I have continued to struggle with high blood pressure and have to take medication to control it; and I'm honestly not sure if it's related, but since my illness I have noticed a few other differences such as difficulty urinating and requiring the use of an inhaler when performing strenuous physical exercise such as running. Additionally, three months after my illness my hair began falling out at an alarming rate, which I understand is common after a traumatic illness.

V. PROGNOSIS

We have retained William Clark, MD, nephrologist from the University of Western Ontario, for his opinion on Lisa Bryant's prognosis.

> It is always difficult to provide an accurate prognosis for someone who has suffered from Haemolytic Uremic Syndrome secondary to shigatoxin E. coli O157:H7 as Ms. Bryant has. However, the prognostic features of concern in this patient are:
>
> 1) Her marked leukocytosis at the time of admissions and for the first few days following her admission with the diagnosis of HUS, which has been shown to bear some prognostic significance in the long-term in most, but not all, subjects studied (1).
> 2) The presence of significant hypertension, which she had when she left hospital on anti-hypertensive therapy, also bears an increased risk for late renal and cardiovascular sequelae (1, 2,3).
> 3) The other interesting feature is that she had a transient epidsode of sixth-nerve palsy, which was attributed to muscle compression, which I am unaware of as an explanation for 6th nerve palsy, however transient 6th nerve palsy has been reported in a patient with HUS presumed secondary to transient thrombotic microangiopathy, which is a prognostic feature that is in keeping with an increased risk of a poor long-term outcome (1, 4). However she did not require dialysis and she was not anuric. The features that are of concern are her elevated white count, her persisting hypertension, her transient neurological abnormality and her proteinuria at the time of her illness (1, 2, 3). On the basis of a meta-analysis of the world literature she does have features of concern being leukocytosis, neurologic abnormality, proteinuria and

hypertension during the illness (1). The good features that she has are that she was neither anuric nor required hemodialysis therapy. With these features it is very difficult to make a precise prognostication in this situation and certainly at the end of five years if her isotope GFR and her 24-hour urine protein excretion was normal and she did not have an abnormal microalbumin to creatinine ratio (evidence of hyperfiltration) then it would be less likely that she would have a significantly negative outcome. However there are still case reports of patients with E coli HUS who have had normal renal function paramets at 10 years and end up on dialysis at 20-25 years follow-up (5, 6). So, the idea of accurately prognosticating outcome is very difficult. I think overall the patient has certain risk factors which suggest she may experience either persistence or later onset of hypertension, cardiovascular complications and structural renal impairment which may lead to further loss of kidney function (1, 5, 6). The other feature this patient has which often bears on long-term prognosis is her age. It is not possible to accurately adjust for her vascular age which may make her more susceptible to ongoing damage in the process of HUS than a person with a much younger vasculature (1). The patient also did suffer hemorrhagic colitis and in view of our assessment of long-term prognosis from the Walkerton Health Study, just having the hemorrhagic colitis places her at a 33% increased risk of hypertension, a three-fold increased risk of vascular complications (heart attack, stroke or heart failure) and a three-fold increase in structural renal impairment in a 7-year follow up study of over 2000 patients (7).

I would conclude that this patient is at risk of long-term sequellae due to her episode of HUS and the persistence of hypertension warrants careful follow-up and treatment with a view to renal and cardio-protection.

VI. LIABILITY AND CAUSATION

The causal link between Lisa Bryant's *E. coli* O157:H7 infection and the consumption of romaine lettuce made from salad bar ingredients at Schnuck's Markets is clear. Ms. Bryant consumed salad purchased at the Schnuck's store located at 5055 Arsenal Street in St. Louis on October 20, 2011. She experienced symptom onset on October 25, 2011. Her stool specimen collected on October 27, 2011 was positive for *E. coli* O157:H7 at the Missouri Department of Health and Senior Services (MO DHSS) Public Health Laboratory (PHL). Molecular testing of the isolate cultured from his stool specimen (PHL #2011139028) was conducted using pulsed field gel electrophoresis (PFGE) analysis. PFGE results showed that Lisa Bryant was infected with a strain identified by Pulsenet as EXHX01.0047/EXHA26.0015. Additional testing by MLVA confirmed that her *E. coli* O157:H7 strain was a genetic match to the strain found in other patients identified as cases in Outbreak #1110MOEXH-2, the outbreak associated with romaine lettuce sold at Schnuck's.

VII. DAMAGES

A. Medical Expenses

Barnes-Jewish Hospital	$74,393.75
Progress West Health Care	$6,115.50
Scott Groesch, MD	$110.00
Mason Ridge Surgery Center	$4,679.00
TOTAL	**$85,298.25**

B. Wage Loss

Industrial Aid, Inc.
Financial Manager
She missed 145 hours at $27.90 p/h

TOTAL **$4,045.50**

VIII. CONCLUSION

Lisa reflects on having lived through a life-threatening illness due to food poisoning with *E. coli*, through no fault of her own:

> Relative to the many possible outcomes of *E. Coli*/HUS, I accepted the lingering effects I was experiencing as minor in comparison. Extremely grateful for my recovery, and unaware that there was a potential for long-term effects, I was ready to put the experience behind me. Recently my doctor scheduled me for a routine colonoscopy and made the comment that he hoped the procedure wouldn't re-trigger the HUS symptoms. In that moment, I didn't ask him to expand on his comment but as I thought about it I became increasingly concerned and began to research the subject. To my relief, I discovered that HUS can't be re-triggered, however I also found information regarding the potential long-term effects that I had not been aware of. There is evidence that a percentage of HUS victims experience mild to severe kidney problems, related to the damage done during the initial HUS attack, that take more than ten years to become symptomatic. This revelation has caused me to become greatly concerned about the future of my health due to the possible long-term effects of E. Coli/HUS.