IN THE CIRCUIT COURT OF ST. LOUIS COUNTY, MISSOURI

Jennifer Cumbus, as Mother and Next Friend of A.M., a minor, Plaintiff, vs. Banquet Enterprises, Inc., Defendant.	Cause No.:_____ Division No.:_____ JURY TRIAL DEMANDED

PETITION FOR DAMAGES

COMES NOW the plaintiff, Jennifer Cumbus, as Mother and Next Friend of A.M., a minor, by and through their attorneys, Aleshire Robb & Rapp and Marler Clark, Inc., PS, and allege upon information and belief as follows:

PARTIES

1.1 The plaintiff, Jennifer Cumbus, as Mother and Next Friend of A.M ("Plaintiff") is a resident of Fenton, St. Louis County, Missouri.

1.2 The defendant, Banquet Enterprises, Inc. ("Defendant"), is a for-profit corporation organized and existing under the laws of the State of Missouri, with its principal place of business located at 4254 Telegraph Rd., St. Louis, Missouri 63129. Defendant was the manufacturer, supplier, packager, distributor and/or seller of the adulterated food product that is the subject of this action. Defendant's registered agent is John Armengol, Jr., who can be served at the same address as its principal place of business.

JURISDICTION AND VENUE

2.1 Plaintiff's cause of action arose and accrued in St. Louis County, Missouri and Defendant Banquet Enterprises, Inc.'s principal place of business is in St. Louis County, Missouri. Therefore, jurisdiction and venue are proper in this Court.

GENERAL ALLEGATIONS

An Outbreak of *E. coli* O157:H7 linked to Andre's Banquets and Catering (Defendant)

3.1 The Saint Louis County Department of Public Health (DPH) is continuing to actively investigate an outbreak of *Escherichia coli* O157 (E. coli) affecting students, parents and guests of Rockwood Summit High School. As of Friday, November 15, 2024, 69 cases have been identified, with some laboratory-confirmed and others presenting compatible symptoms and epidemiological links to confirmed cases.

3.2 Further investigation has revealed that individuals who tested positive for *E. coli* attended two separate events hosted at or catered through Andre's Banquets and Catering in association with Rockwood Summit High School. Although current details are suggestive of salad being the source of illness, the DPH has not identified a specific ingredient, nor do they have information on the nature or timing of the contamination (i.e., farm vs. later).

3.3 DPH Communicable Disease Investigators and Environmental Health Inspectors are conducting interviews to gather detailed food history, symptom onset, and exposure details from affected individuals. In collaboration with the Rockwood School District, an electronic survey was distributed to event attendees, which has significantly aided in collecting data on food consumption and symptoms. Environmental inspectors have also been evaluating associated locations and will be collecting environmental and food samples for further testing.

3.4 It is important to note that current findings do not implicate any single source or party. Additional testing and data analysis are ongoing to determine the exact source of the contamination.

3.5 The Rockwood School District has been exceptionally cooperative, working closely with DPH to ensure thoroughness in identifying the source of this outbreak. Their proactive measures, including the distribution of the electronic survey, have provided invaluable information to the investigation.

3.6 DPH urges anyone experiencing symptoms of *E. coli* infection – including severe stomach cramps, diarrhea (often bloody), vomiting, and fever – to seek medical attention immediately. Preventative measures such as proper handwashing and food safety practices remain critical during this time.

The *E. coli* O157:H7 Bacteria

3.7 *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*.[1] 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[2] respectively and distinguish it from other types of *E. coli*.[3] Most serotypes of *E. coli* are harmless and live as normal flora in the intestines of healthy humans and animals.[4] The *E. coli* bacterium is among the most

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[1] *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.*

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

[3] 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.

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

extensively studied microorganism.[5] The testing done to distinguish *E. coli* O157:H7 from its other *E. coli* counterparts is called serotyping.[6] Pulsed-field gel electrophoresis (PFGE),[7] sometimes also referred to as genetic fingerprinting, is used to compare *E. coli* O157:H7 isolates to determine if the strains are distinguishable.[8] A technique called multilocus 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.[9]

3.8 *E. coli* O157:H7 was first recognized as a pathogen in 1982 during an investigation into an outbreak of hemorrhagic colitis[10] associated with consumption of hamburgers from a fast food chain restaurant.[11] 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.[12] In the ten (10) years that followed there were approximately thirty (30)

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

[6] 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).

[7] Jay, *supra* note **Error! Bookmark not defined.**, at 220-21 (describing in brief the PFGE testing process).

[8] *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 **Error! Bookmark not defined.**, at 1351-52. Because PFGE testing soon proved to be s uch 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 Emerging Infect. Dis. (No. 3) 382, 382-89 (May-June 2001) (recounting the history of PulseNet and its effectiveness in outbreak investigation).

[9] 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.

[10] "[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.

[11] 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).

[12] Riley, *supra* note **Error! Bookmark not defined.** at 684. See also Patricia M. Griffin & Robert V. Tauxe, T he 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).

outbreaks recorded in the United States.[13] 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.[14] As a result, only the most geographically concentrated outbreak would have garnered enough notice to prompt further investigation.[15]

3.9 *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.[16] 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.[17] Shiga toxin is one of the most potent toxins known.[18] 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.[19]

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[13] 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).

[14] 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, 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.")

[15] *See* Keene, *supra* note **Error! Bookmark not defined.** at 583. ("With cases scattered over four counties, t he 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).

[16] Griffin & Tauxe, *supra* note **Error! Bookmark not defined.**, at 61-62 (noting that the nomenclature came a bout because of the resemblance to toxins produced by Shigella dysenteries).

[17] 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.

[18] 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).

[19] 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:

3.10 *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.[20] The rate of genetic mutation of *E. coli* O157:H7 indicates that the common ancestor of current *E. coli* O157:H7 clades[21] likely existed some 20,000 years ago.[22] *E. coli* O157:H7 is a relentlessly evolving organism,[23] constantly mutating and acquiring new characteristics, including virulence factors that make the emergence of more dangerous variants a constant threat.[24] The CDC has emphasized the prospect of emerging pathogens as a significant public health threat for some time.[25]

3.11 Although foods of a bovine origin are the most common cause of both outbreaks and sporadic cases of *E. coli* O157:H7 infections[26], 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.[27] Other unusual vehicles for

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Role of 60-Megadalton Plasmid (p0157) and Potential Virulence Factors, http://fsrio.nal.usda.gov/document_fsheet.php?product_id=225.

[20] 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).

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

[22] Zhang W, *et al.* Probing genomic diversity and evolution of *Escherichia coli* O157 by single nucleotide polymorphisms. *Genome Res* 16*:*757–767 (2006).

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

[24] 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.")

[25] 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.")

[26] 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).

[27] Rangel, *supra* note **Error! Bookmark not defined.**, at 605.

E. coli O157:H7 outbreaks have included unpasteurized juices, yogurt, dried salami, mayonnaise, raw milk, game meats, sprouts, and raw cookie dough.[28]

3.12 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.[29] Estimates of foodborne acquired O157:H7 cases result in 2,138 hospitalizations and 20 deaths annually.[30] 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.[31] 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.[32] Infection can occur in people of all ages but is most common in children.[33] The duration of an uncomplicated illness can range from one (1) to twelve (12) days.[34] 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.[35]

3.13 What makes *E. coli* O157:H7 remarkably dangerous is its very low infectious dose,[36] and how relatively difficult it is to kill these bacteria.[37] Unlike *Salmonella*, for example,

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[28] Feng, *supra* note **Error! Bookmark not defined.**, at 49. *See also* USDA Bad Bug Book, *Escherichia coli* O 157:H7, http://www.fda.gov/food/foodsafety/foodborneillness/foodborneillnessfoodbornepathogensnaturaltoxins/badbugbook/ucm071284.htm.

[29] 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.

[30] *Id.*, Table 3.

[31] Griffin & Tauxe, *supra* note **Error! Bookmark not defined.**, at 63.

[32] 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").

[33] Su & Brandt, *supra* note **Error! Bookmark not defined.** ("the young are most often affected").

[34] Tauxe, *supra* note **Error! Bookmark not defined.**, at 1152.

[35] *Id.*

[36] Griffin & Tauxe, *supra* note **Error! Bookmark not defined.**, at 72. ("The general patterns of transmission i n these outbreaks suggest that the infectious dose is low.")

[37] 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

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,"[38] as few as twenty (20) organisms may be sufficient to infect a person and, as a result, possibly kill them.[39] 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.[40]

3.14 And, finally, to make it even more of a threat, *E. coli* O157:H7 bacteria are easily transmitted by person-to-person contact.[41] 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.[42] With this pathogen, there is ultimately no margin of error. It is for this precise reason that the USDA has

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to 130°F, there is no bacterial destruction, and at 140°F, there is only a 2-log reduction of *E. coli* present).

[38] Griffin & Tauxe, *supra* note **Error! Bookmark not defined.**, at 72 (noting that, as a result, "fewer bacteria a re needed to cause illness that for outbreaks of salmonellosis"). Nestle, *supra* note **Error! Bookmark not defined.**, at 41. ("Foods containing *E. coli* O17:H7 must be at temperatures high enough to kill all of them.") (italics in original)

[39] 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 **Error! Bookmark not defined.**, at 41 ("less than 50"). Regardless of these inconsistencies, everyone a grees that the infectious dose is, as Dr. Nestle has put it, "a miniscule number in bacterial terms." *Id.*

[40] Nestle, *supra* note **Error! Bookmark not defined.**, at 41.

[41] Griffin & Tauxe, *supra* note **Error! Bookmark not defined.**, at 72. The apparent "ease of person-to-p erson 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 **Error! Bookmark not defined.**, at 605-06 (finding that 80% of the 50 reported person-to-person outbreak f rom 1982-2002 occurred in daycare centers).

[42] *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/.

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.[43]

3.15 *E. coli* O157:H7 infections can lead to a severe, life-threatening complication called hemolytic uremic syndrome (HUS).[44] HUS accounts for the majority of the acute deaths and chronic injuries caused by the bacteria.[45] HUS occurs in 2-7% of victims, primarily children, with onset five to ten days after diarrhea begins.[46] It is the most common cause of renal failure in children.[47] Approximately half of the children who suffer HUS require dialysis, and at least 5% of those who survive have long term renal impairment.[48] The same number suffers severe brain damage.[49] While somewhat rare, serious injury to the pancreas, resulting in death or the development of diabetes, can also occur.[50] There is no cure or effective treatment for HUS.[51]

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[43] 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.") (citing Pathogen Reduction, 61 Fed. Reg. at 38966).

[44] Griffin & Tauxe, *supra* note **Error! Bookmark not defined.**, 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).

[45] 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), at http://www.pediatrics.org/cgi/content/full/100/1/e12.

[46] Tauxe, *supra* note **Error! Bookmark not defined.**, 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*.")

[47] Su & Brandt, *supra* note **Error! Bookmark not defined.**.

[48] Safdar, *supra* note **Error! Bookmark not defined.**, 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).

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

[50] 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").

[51] Safdar, *supra* note **Error! Bookmark not defined.**, at 996. *See also* Siegler, *supra* note **Error! Bookmark not defined.**, at 1379. ("There are no treatments of proven value, and care during the acute phase of the illness, which

3.16 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.[52] 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.[53] Some organs seem more susceptible, perhaps due to the presence of increased numbers of receptors, and include the kidney, pancreas, and brain.[54] 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).[55]

3.17 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.[56] Among survivors, at least five percent will suffer end stage renal disease (ESRD) with the resultant need for dialysis or transplantation.[57] But, "[b]ecause renal failure can progress slowly over decades, the eventual incidence of ESRD cannot yet be determined."[58] 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.[59] Since the longest available follow-up studies of HUS victims are 25 years, an accurate lifetime prognosis is not really available

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is merely supportive, has not changed substantially during the past 30 years.")

[52] Garg, *supra* note **Error! Bookmark not defined.**, at 1360.

[53] *Id*. Siegler, *supra* note **Error! Bookmark not defined.**, at 1509-11 (describing what Dr. Siegler refers to as t he "pathogenic cascade" that results in the progression from colitis to HUS).

[54] Garg, *supra* note **Error! Bookmark not defined.**, at 1360. *See also* Su & Brandt, *supra* note **Error! Bo okmark not defined.**, at 700.

[55] Garg, *supra* note**Error! Bookmark not defined.**, at 1360. *See also* Su & Brandt, *supra* note **Error! Bo okmark not defined.**, at 700.

[56] Siegler, *supra* note **Error! Bookmark not defined.**, at 1519 (noting that in a "20-year Utah-based p opulation study, 5% dies, and an equal number of survivors were left with end-stage renal disease (ESRD) or chronic brain damage.")

[57] Garg, *supra* note **Error! Bookmark not defined.**, at 1366-67.

[58] Siegler, *supra* note **Error! Bookmark not defined.**, at 1519.

[59] *Id*. at 1519-20. *See also* Garg, *supra* note **Error! Bookmark not defined.**, at 1366-67.

and remains controversial.[60] 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.

3.18 The term reactive arthritis refers to an inflammation of one or more joints, following an infection localized at another site distant from the affected joints. The predominant site of the infection is the gastrointestinal tract. Several bacteria, including *E. coli*, induce septic arthritis.[61]The resulting joint pain and inflammation can resolve completely over time or permanent joint damage can occur.[62]

3.19 The reactive arthritis associated with Reiter Syndrome may develop after a person eats food that has been tainted with bacteria. In a small number of persons, the joint inflammation is accompanied by conjunctivitis (inflammation of the eyes), and urethritis (painful urination). *Id*. This triad of symptoms is called Reiter syndrome.[63] Reiter syndrome, a form of reactive arthritis, is an uncommon but debilitating syndrome caused by gastrointestinal or genitourinary infections. The most common gastrointestinal bacteria involved are *Salmonella*, *Campylobacter*, *Yersinia*, and *Shigella*. Reiter syndrome is characterized by a triad of arthritis, conjunctivitis, and urethritis, although not all three symptoms occur in all affected individuals.[64]

3.20 Although the initial infection may not be recognized, reactive arthritis can still occur. Reactive arthritis typically involves inflammation of one joint (monoarthritis) or four or fewer joints (oligoarthritis), preferentially affecting those of the lower extremities; the pattern of

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60 Garg, *supra* note **Error! Bookmark not defined.**, at 1368.

61 *See* J. Lindsey, "Chronic Sequellae of Foodborne Disease," *Emerging Infectious Diseases*, Vol. 3, No. 4, Oct-Dec, 1997.

62 *Id*.

63 *Id*. *See also* Dworkin, *et al*., "Reactive Arthritis and Reiter's Syndrome following an outbreak of gastroenteritis caused by Salmonella enteritidis," *Clin. Infect. Dis*., 2001 Oct. 1;33(7): 1010-14; Barth, W. and Segal, K., "Reactive Arthritis (Reiter's Syndrome)," *American Family Physician*, Aug. 1999, online at www.aafp.org/afp/990800ap/ 499.html.

64 Hill Gaston JS, Lillicrap MS. (2003). Arthritis associated with enteric infection. *Best Practices & Research Clinical Rheumatology*. 17(2):219-39.

joint involvement is usually asymmetric. Inflammation is common at entheses – *i.e.*, the places where ligaments and tendons attach to bone, especially the knee and the ankle.

3.21 *Salmonella* has been the most frequently studied bacteria associated with reactive arthritis. Overall, studies have found rates of *Salmonella*-associated reactive arthritis to vary between 6 and 30%.[65] The frequency of postinfectious Reiter syndrome, however, has not been well described. In a Washington State study, while 29% developed arthritis, only 3% developed the triad of symptoms associated with Reiter syndrome.[66] In addition, individuals of Caucasian descent may be more likely those of Asian descent to develop reactive arthritis,[67] and children may be less susceptible than adults to reactive arthritis following infection with *Salmonella*.[68]

3.22 A clear association has been made between reactive arthritis and a genetic factor called the human leukocyte antigen (HLA) B27 genotype. HLA is the major histocompatibility complex in humans; these are proteins present on the surface of all body cells that contain a nucleus and are in especially high concentrations in white blood cells (leukocytes). It is thought that HLA-B27 may affect the elimination of the infecting bacteria or an individual's immune response.[69] HLA-B27 has been shown to be a predisposing factor in one-half to over two-thirds of individuals with reactive arthritis.[70] While HLA-B27 does not appear to predispose to the initial infection itself, it increases the risk of developing arthritis that is more likely to be severe and prolonged.

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65 *Id*.

66 Dworkin MS, Shoemaker PC, Goldoft MJ, Kobayashi JM, "Reactive arthritis and Reiter's syndrome following an outbreak of gastroenteritis caused by *Salmonella* enteritidis. *Clin. Infect. Dis.* 33(7):1010-14.

67 McColl GJ, Diviney MB, Holdsworth RF, McNair PD, Carnie J, Hart W, McCluskey J, "HLA-B27 expression and reactive arthritis susceptibility in two patient cohorts infected with *Salmonella* Typhimurium," Australian *and New Zealand Journal of Medicine* 30(1):28-32 (2001).

68 Rudwaleit M, Richter S, Braun J, Sieper J, "Low incidence of reactive arthritis in children following a *Salmonella* outbreak," *Annals of the Rheumatic Diseases.* 60(11):1055-57 (2001).

69 Hill Gaston and Lillicrap, *supra* Note 7.

70 *Id*.; Barth WF, Segal K., "Reactive arthritis (Reiter's syndrome)," *American Family Physician,* 60(2):499-503, 507 (1999).

This risk may be slightly greater for *Salmonella* and *Yersinia*-associated arthritis than with *Campylobacter*, but more research is required to clarify this.[71]

3.23 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 decades ago.[72] The Walkerton Health Study 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.[73]

3.24 In terms of its own data, the "study confirm[ed] a strong and significant relationship between acute enteric infection and subsequent IBS symptoms."[74] The WHS also identified risk-factors for subsequent IBS, including younger age; female sex; and four features of the acute enteric illness – diarrhea for > 7days, presence of blood in stools, abdominal cramps, and weight loss of at least ten pounds.[75]

3.25 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.[76] In one recent study, over one-third of IBS sufferers had had IBS for more

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71 Hill Gaston and Lillicrap, *supra* Note 7.

72 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*. For *Salmonella* specific references, *see* Smith, J.L., Bayles, D.O., *Post-Infectious Irritable Bowel Syndrome: A Long-Term Consequence of Bacterial Gastroenteritis*, Journal of Food Protection. 2007:70(7);1762-69.

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

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

75 *Id*. at 447.

76 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.

than ten years, with their symptoms remaining fairly constant over time.[77] IBS sufferers typically experienced symptoms for an average of 8.1 days per month.[78]

3.26 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.[79] And even when able to work, a significant majority (67%), felt less productive at work because of their symptoms.[80] IBS symptoms also have a significantly deleterious impact on social well-being and daily social activities, such as undertaking a long drive, going to a restaurant, or taking a vacation.[81] Finally, although 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.[82]

PLAINTIFF'S *E. COLI* ILLNESS

4.1 Plaintiff A.M. consumed food prepared and catered by the Defendant on November, 8, 2024.

4.2 Plaintiff A.M. began to experience symptoms consistent with an *E. coli* infection on November 10, 2024.

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77 *Id*.at 1367.
78 *Id*.
79 *Id*. at 1368.
80 *Id*.
81 *Id*.
82 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* 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).

4.3 Plaintiff A.M. symptoms have consisted of severe and persistent cramping, malaise, dizziness, and continuous diarrhea, which later escalated to bloody diarrhea. As a result, Plaintiff sought emergency medical treatment for dehydration and pain management.

4.4 Plaintiff AM. has endured extreme pain, has been unable to eat, and has lost over seven pounds in the last week alone. She has been unable to keep food our water down due to nausea.

4.5 Plaintiff A.M. remains severely fatigued, continues to experience severe pain, and her diarrhea continues to be bloody.

CAUSES OF ACTION

Strict Liability – Count I

5.1 Plaintiff incorporates by reference paragraphs 1.1 – 4.5 herein by reference.

5.2 At all times relevant hereto, the Defendant is the manufacturer, supplier, packager, distributor and/or seller of the adulterated food products that are the subject of this action.

5.3 The adulterated food product that the Defendant manufactured, supplied, packaged, distributed, and/or sold was, at the time it left the Defendant's control, defective and unreasonably dangerous for its ordinary and expected use because it contained *E. coli*, a deadly pathogen.

5.4 The adulterated food product that the Defendant manufactured, supplied, packaged, distributed, and/or sold was delivered to the Plaintiff without any change in its defective condition. The adulterated food product that the Defendant manufactured, supplied, packaged, distributed, and/or sold was used in the manner expected and intended, and was consumed by the Plaintiff.

5.5 The Defendant owed a duty of care to the Plaintiff to manufacture, supply, package, distribute and/or sell food that was not adulterated, that was fit for human consumption, that was

reasonably safe in construction, and that was free of pathogenic bacteria or other substances injurious to human health. The Defendant breached this duty.

5.6 The Defendant owed a duty of care to the Plaintiff to manufacture, supply, package, distribute and/or sell food that was fit for human consumption, and that was safe to consume to the extent contemplated by a reasonable consumer. The Defendant breached this duty.

5.7 Plaintiff suffered injury and damages as a direct and proximate result of the defective and unreasonably dangerous condition of the adulterated food product that the Defendant manufactured, supplied, packaged, distributed and/or sold.

WHEREFORE, the Plaintiff respectfully requests judgment against the Defendant on Count I of this Petition in an amount that is fair and reasonable, for her costs incurred and for any other relief to which she may be entitled.

Breach of Warranty – Count II

5.8 Plaintiff incorporates by reference paragraphs 1.1 – 5.7 herein by reference.

5.9 The Defendant is liable to the Plaintiff for breaching express and implied warranties that it made regarding the adulterated product that Plaintiff purchased. These express and implied warranties include the implied warranties of merchantability and/or fitness for a particular use. Specifically, the Defendant expressly warranted, through its sale of food to the public and by the statements and conduct of its employees and agents, that the food it prepared and sold was fit for human consumption and not otherwise adulterated or injurious to health.

5.10 The contaminated food that the Defendant sold to Plaintiff would not pass without exception in the trade and was therefore in breach of the implied warranty of merchantability.

5.11 The contaminated food sold to Plaintiff was not fit for the uses and purposes intended, *i.e.,* human consumption; this product was therefore in breach of the implied warranty of fitness for its intended use.

5.12 As a direct and proximate cause of the Defendant's breach of warranties, as set forth above, the Plaintiff sustained injuries, death and damages in an amount to be determined at trial.

WHEREFORE, the Plaintiff respectfully requests judgment against the Defendant on Count II of this Petition in an amount that is fair and reasonable, for her costs incurred and for any other relief to which she may be entitled.

Negligence – Count III

5.13 Plaintiff incorporates by reference paragraphs 1.1 – 5.12 herein by reference.

5.14 The Defendant owed to the Plaintiff a duty to use reasonable care in the manufacture, supply, packaging, distribution and sale of their food product, which duty would have prevented or eliminated the risk that the Defendant's food products would become contaminated with *E. coli* or any other dangerous pathogen. The Defendant breached this duty.

5.15 The Defendant had a duty to comply with all federal, state and local statutes, laws, regulations, safety codes and provisions pertaining to the manufacture, distribution, storage, and sale of its food product, but failed to do so, and was therefore negligent. The Plaintiff was among the class of persons designed to be protected by these statutes, laws, regulations, safety codes and provisions pertaining to the manufacture, distribution, storage, and sale of similar food products.

5.16 The Defendant had a duty to properly supervise, train and monitor its respective employees, and to ensure that its respective employees complied with all applicable statutes, laws,

regulations, safety codes and provisions pertaining to the manufacture, distribution, storage, and sale of similar food products. The Defendant, however, failed to do so and was therefore negligent.

5.17 The Defendant had a duty to use ingredients, supplies, and other constituent materials that were reasonably safe, wholesome and free of defects, and that otherwise complied with applicable federal, state, and local laws, ordinances, regulations, codes and provisions and that were clean, free from adulteration, and safe for human consumption. The Defendant, however, failed to do so and was therefore negligent.

5.18 As a direct and proximate result of the Defendant's negligence, the Plaintiff sustained injuries and damages in an amount to be determined at trial.

WHEREFORE, the Plaintiff respectfully requests judgment against the Defendant on Count III of this Petition in an amount that is fair and reasonable, for her costs incurred and for any other relief to which she may be entitled.

Negligence *Per Se* – Count IV

5.19 Plaintiff incorporates by reference paragraphs 1.1 – 5.18 herein by reference.

5.20 The Defendant had a duty to comply with all applicable state and federal regulations intended to ensure the purity and safety of their food product, including the requirements of the Federal Food, Drug and Cosmetics Act (21 U.S.C. § 301, *et seq.*) and similar Missouri state regulations.

5.21 The Defendant failed to comply with the provisions of the health and safety acts identified above, and, as a result, was negligent *per se* in its manufacture, distribution, and sale of food adulterated with *E. coli*, a deadly pathogen.

5.22 As a direct and proximate result of conduct by the Defendant that was negligent *per se*, the Plaintiff sustained injury and damages in an amount to be determined at trial.

WHEREFORE, the Plaintiff respectfully requests judgment against the Defendant on Count IV of this Petition in an amount that is fair and reasonable, for her costs incurred and for any other relief to which she may be entitled.

DAMAGES

6.1 The Plaintiff has suffered general, special, incidental, and consequential damages as a direct and proximate result of the acts and omissions of the Defendant, which damages shall be fully proven at the time of trial, including, but not limited to: damages for loss of enjoyment of life, both past and future; medical and medical-related expenses, both past and future; travel and travel-related expenses, past and future; emotional distress and future emotional distress; pharmaceutical expenses, past and future; wage and other economic damages; loss of consortium; and other ordinary, incidental, and consequential damages as would be anticipated to arise under the circumstances.

PRAYER FOR RELIEF

WHEREFORE, Plaintiff prays as follows:

(1) That the Court award Plaintiff judgment against Defendant for damages.

(2) That the Court assess all such other sums as shall be determined to fully and fairly compensate Plaintiff for all general, special, incidental, and consequential damages incurred, or to be incurred, by Plaintiff as the direct and proximate result of the acts and omissions of the Defendant;

(3) That the Court award Plaintiff her costs, disbursements, and reasonable attorneys' fees incurred;

(4) That the Court grant Plaintiff the opportunity to amend or modify the provisions of this Complaint as necessary or appropriate after additional or further discovery is completed in this matter, and after all appropriate parties have been served; and

(5) That the Court grant such other and further relief as it deems necessary and proper in the circumstances.

DATED: November 18, 2024

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