UNITED STATES DISTRICT COURT
DISTRICT OF MINNESOTA

SUSAN PORTER & SAMUEL PORTER
Individually, and A.P., a minor, by and
through her parents and natural guardians,
SUSAN PORTER & SAMUEL PORTER,

Plaintiffs,

vs.

CARGILL MEAT SOLUTIONS CORP.,
a foreign corporation,

Defendant.

Case No. _____

COMPLAINT FOR DAMAGES

JURY DEMAND

COME NOW the Plaintiffs, SAMUEL & SUSAN PORTER individually, and A.P., a minor, by and through her parents and natural guardians, SUSAN PORTER & SAMUEL PORTER, by and through their attorneys of record, asserting claims against the Defendant, CARGILL MEAT SOLUTIONS CORP., and state and allege as follows:

I. PARTIES

1.1 Plaintiffs SUSAN & SAMUEL PORTER are the parents and natural guardians of the minor child A.P. The Porter family resides in Signal Mountain, Tennessee.

1.2 Defendant CARGILL MEAT SOLUTIONS CORPORATION aka Cargill Protein (hereinafter "Cargill") is a corporation organized under the laws of the State of Delaware, registered as a corporation in the State of Kansas and with a registered office address of 1010 Dale St. N., St. Paul, MN 55117-5603. Cargill is a subsidiary of parent corporation Cargill, Incorporated, which maintains its principal place of business in Minnesota. Further, at all times relevant hereto, Cargill was authorized to and did business in the State of Minnesota.

II. JURISDICTION AND VENUE

2.1 This Court has jurisdiction over the subject matter of this action pursuant to 28 USC § 1332(a) because the matter in controversy exceeds $75,000.00, exclusive of costs; it is between citizens of different states; and because the Defendant has certain minimum contacts with the State of Minnesota such that maintenance of the suit in this district does not offend traditional notions of fair play and substantial justice.

2.2 Venue in the United States District Court for the District of Minnesota is proper pursuant to 28 USC § 1391(b)(1) and (c)(2) because the Defendant Cargill was subject to personal jurisdiction in this judicial district at the time of the commencement of the action.

III. GENERAL ALLEGATIONS

CARGILL GROUND BEEF 2018 *E. COLI* O26 – 17 ILL, 1 DEATH.

3.1 As of September 19, 2018, 18 people infected with the outbreak strain of *E. coli* O26 were reported from 4 States – Colorado (1), Florida (15), Massachusetts (1) and Tennessee (1). Illnesses started on dates ranging from July 5, 2018, to July 25, 2018. Ill

people ranged in age from one year to 75, with a median age of 16. Sixty-seven percent of ill people were male. Of 18 people with information available, 6 (33%) were hospitalized, including one person who died in Florida.

3.2 Epidemiologic, laboratory, and traceback evidence indicates that ground beef from Cargill Meat Solutions was a likely source of this outbreak.

3.3 In interviews, ill people answered questions about the foods they ate and other exposures in the week before they became ill. Fourteen (100%) out of fourteen people interviewed reported eating ground beef. Ill people purchased ground beef from several different grocery stores, including Publix Super Markets, Inc.

3.4 USDA-FSIS conducted traceback investigations from stores where ill people reported buying ground beef. Initial information collected from ill people in Florida indicated that the ground beef was purchased from various Publix grocery stores. On August 30, 2018, Publix Super Markets, Inc. recalled ground chuck products sold in several Florida counties.

3.5 Further traceback investigation by USDA-FSIS identified Cargill Meat Solutions in Fort Morgan, Colorado as the source of the contaminated ground beef linked to illness, including the recalled ground beef sold at Publix stores in Florida. On September 19, 2018, Cargill Meat Solutions recalled ground beef products that were produced and packaged on June 21, 2018. Products are labeled with the establishment number “EST. 86R” inside the USDA mark of inspection. The products were shipped to retailers nationwide.

3.6 Laboratory testing identified the outbreak strain of *E. coli* O26 in leftover ground beef collected from the home of one ill person in Florida. WGS analysis showed that the *E. coli* O26 strain identified in the leftover ground beef was highly related genetically to the *E. coli* O26 strain isolated from ill people.

A.P.'S CONSUMPTION OF AND *E. COLI* O26 INFECTION.

3.7 On July 4, 2018, A.P. consumed ground beef that her parents purchased from the Publix Super Market Located at Plantation Grove Shopping Center, 2600 Maguire Road, Ocoee, Florida 34761.

3.8 A.P.'s symptom onset began on July 7, 2018. She experienced vomiting, nausea, stomach cramps, fatigue, and headaches. Two days later, she developed bloody diarrhea which prompted her parents to seek out professional medical treatment.

3.9 A.P. was eventually admitted to emergency care at the Children's Hospital at Erlanger Emergency Room, where physicians conducted numerous tests and treated A.P.'s severe gastrointestinal symptoms, including her development of hemolytic uremic syndrome ("HUS")

3.10 A stool specimen obtained from A.P. eventually tested positive for Shiga toxin producing *E. coli*. Samuel and Susan Porter were contacted by the Hamilton Health Department on multiple occasions to discuss A.P.'s possible food exposures, her HUS diagnosis, and to confirm the address of the Publix where the ground beef was purchased.

3.11 A.P. continues to recover from her *E. coli* illness, and currently receives ongoing treatment from her primary care physician.

A HISTORY OF PRIOR OUTBREAKS

3.12 **Cargill Ground Beef 2012 – 40 Ill – *Salmonella***. On July 22, 2012, Cargill Meat Solutions announced a recall of 29,339 pounds of fresh ground beef products due to possible contamination with *Salmonella* Enteritidis. Using epidemiologic and traceback data public health investigators in 8 states (MA, ME, NH, NY, RI, VA, VT, and WV) and the CDC linked 40 patients diagnosed with S. Enteritidis to consumption of Cargill ground beef sold at Hannaford grocery stores in Massachusetts, Maine, New Hampshire, New York and Vermont. Among 40 persons for whom information is available, illness onset dates ranged from June 6, 2012 to July 9, 2012. Eleven patients were hospitalized. The Vermont Department of Health isolated the outbreak strain in leftover product.

3.13 **Cargill Meat Solutions Ground Turkey 2011 – 181 Ill – *Salmonella***. The U.S. Department of Agriculture's Food Safety and Inspection Service (FSIS) issued a public health alert, on July 29, 2011, due to concerns about illnesses caused by *Salmonella* Heidelberg that associated with the use and the consumption of ground turkey. The alert was initiated after continuous medical reports, ongoing investigations and testing conducted by various departments of health across the nation determined an association between consumption of ground turkey products and illness. On August 3, 2011, Cargill Meat Solutions issued a recall of ground turkey products. The products subject to recall bear the establishment number "P-963" inside the USDA mark of inspection. On August 4, 2011, the Centers for Disease Control published their first outbreak summary. The *Salmonella* Heidelberg was multi-drug resistant, resistant to

ampicillin, streptomycin, tetracycline, and gentamycin. The CDC began their investigation on May 23, 2011, after recognizing an "unusual clustering" of *Salmonella* Heidelberg cases. About the same time, routine surveillance by a federal food monitoring system found the same strain of *Salmonella* Heidelberg in ground turkey in stores. On July 29, 2011, the initial outbreak strain and a second, closely related, strain of *Salmonella* Heidelberg was isolated from a sample of leftover unlabeled frozen ground turkey from the home of an outbreak case in Ohio. Since February 27, 2011, a total of 23 ill persons were reported to Pulse Net with this second, closely related, strain. Eighty-four ill persons were infected with the initial strain. The consumer product sample originated from the Cargill Meat Solutions Corporation establishment in Springdale, Arkansas. On September 11, 2011, Cargill Meat Solutions recalled an additional, approximately 185,000 pounds, of ground turkey contaminated with an identical strain of *Salmonella* Heidelberg that had led to the earlier recall on August 3, 2011. As of September 27, 2018, no illnesses had been linked to the additionally recalled, ground turkey products.

3.14 **Cargill Meat Solutions/BJ's Wholesale Club Ground Beef 2010 – 3 Ill – *E. coli* O26**. A recall of ground beef was issued on August 28, 2010 when three people developed illnesses caused by rare strain of *E. coli* O26 after they had eaten the product. The ground beef produced by Cargill Meat Solutions of Pennsylvania and was distributed to BJ's Wholesale Clubs in New York, Maine, Connecticut, Virginia, New Jersey, New Hampshire, Massachusetts, and Maryland.

3.15 **Beef Packers, Inc., Cargill, Ground Beef 2009 – 2 Ill – *Salmonella***. In December 2009, Beef Packers, Inc., owned by Cargill, recalled over 20,000 pounds of

ground beef contaminated with a drug-resistant strain of *Salmonella* Newport. The company issued an earlier recall in August 2009, due to contamination of ground beef with the same strain of *Salmonella* Newport. This contaminated ground beef was produced in September and was distributed to Safeway grocery stores in Arizona and New Mexico. The Arizona Department of Health linked two illnesses to the ground beef.

3.16 **Beef Packers, Inc., Cargill, Ground Beef 2009 – 68 Ill – *Salmonella***. A Beef Packers, Inc. plant in California—owned by Cargill—distributed approximately 830,000 pounds of ground beef that was likely contaminated with *Salmonella* Newport. The beef was shipped to distribution centers in Arizona, California, Colorado, and Utah where it was repackaged into consumer-sized packages and sold under different retail brand names. The contaminated beef contained a strain of *Salmonella* resistant to several commonly used antibiotics (called MDR-AmpC resistance). Sixty-eight outbreak associated cases were reported by 15 states. Most of the ill in Colorado had purchased the ground beef at Safeway grocery stores.

3.17 **Cargill Ground Beef Sold at Sam's Club Stores 2007 – 46 Ill – *E. coli* O157:H7**. A multistate outbreak of *E. coli* O157:H7 began in August 2007 and led to the eventual recall of 845,000 pounds of Cargill ground beef. Forty-six cases were reported by 15 states. Interviews with the case-patients found a common exposure of Cargill hamburger.

3.18 **Emmpak/Cargill Ground Beef 2002 – 57 Ill – *E. coli* O157:H7**. Wisconsin epidemiologists noted a cluster of *E. coli* O157:H7 cases. The health department interviewed case-patients and found a common exposure. All victims had

eaten ground beef from Emmpak, a meat producer, purchased by Cargill. The same strain of *E.coli* O157:H7 was isolated from the ground beef. The case investigation resulted in a 2.8-million-pound recall of Emmpak meat and resulted in related illnesses in at least six states. The responsible Emmpak plant was closed for inadequate sampling and testing procedures.

3.19 **Cargill Deli Turkey 2000 - 29 Ill – *Listeria***. A case-control study implicated sliced, processed, turkey deli meat in a multistate (11 state) outbreak. A traceback investigation identified a single processing plant in Texas as the likely source of the outbreak. The company recalled 16 million pounds of processed meat. The same plant had been implicated in a *Listeria* contamination involving the same strain of Listeria more than a decade previously.

WHAT IS *E. COLI*?

3.20 *E. coli* is an archetypal commensal bacterial species that lives in mammalian intestines. *E. coli* O26 is one of thousands of serotypes *Escherichia coli*.[1] The combination of letters and numbers in the name of the *E. coli* O26 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*. Most serotypes of *E. coli* are harmless and live as normal flora in the intestines of healthy

[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). 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.

humans and animals.[3] The *E. coli* bacterium is among the most extensively studied microorganism.[4] The testing done to distinguish *E. coli* O26 from its other *E. coli* counterparts is called serotyping.[5] Pulsed-field gel electrophoresis (PFGE),[6] sometimes also referred to as genetic fingerprinting, is used to compare *E. coli* O26 isolates to determine if the strains are distinguishable.[7] 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.[8]

> **NOTE**: The remainder of this section concerns a close relative of *E. coli* O26. The literature on *E. coli* O157:H7 is much more comprehensive than on *E. coli* O26 because the O157:H7 strain is more prevalent, and has caused more recognized outbreaks. Nonetheless, the characteristics described below are equally applicable to the O26 strain.

3.21 *E. coli* O157:H7 was first recognized as a pathogen in 1982 during an investigation into an outbreak of hemorrhagic colitis[9] associated with consumption of

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

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

[5] 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).

[6] Jay, *supra* note 4, at 220-21 (describing in brief the PFGE testing process).

[7] *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 5, 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 Emerging Infect. Dis. (No. 3) 382, 382-89 (May-June 2001) (recounting the history of PulseNet and its effectiveness in outbreak investigation).

[8] 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.

[9] "[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.

hamburgers from a fast food chain restaurant.[10] Retrospective examination of more than three thousand *E. coli* cultures obtained between 1973 and 1982 found only one isolation with serotype O157:H7, and that was a case in 1975.[11] In the ten years that followed there were approximately thirty outbreaks recorded in the United States.[12] 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.[13] As a result, only the most geographically concentrated outbreak would have garnered enough notice to prompt further investigation.[14]

3.22 *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.[15] Shiga toxin (Stx) has multiple variants (e.g. Stx1, Stx2, Stx2c), and acts like the plant toxin ricin by

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[10] 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).

[11] Riley, *supra* note 10 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).

[12] 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).

[13] 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.")

[14] *See* Keene, *supra* note 13 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).

[15] Griffin & Tauxe, *supra* note 11, at 61-62 (noting that the nomenclature came about because of the resemblance to toxins produced by *Shigella dysenteries*).

inhibiting protein synthesis in endothelial and other cells.[16] Shiga toxin is one of the most potent toxins known.[17] 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.[18]

3.23 *E. coli* O157:H7 evolved from enteropathogenic *E. coli* serotype O55:H7, a cause of nonbloody diarrhea, through the sequential acquisition of phage-encoded Stx2, a large virulence plasmid, and additional chromosomal mutations.[19] The rate of genetic mutation of *E. coli* O157:H7 indicates that the common ancestor of current *E. coli* O157:H7 clades[20] likely existed some 20,000 years ago.[21] *E. coli* O157:H7 is a relentlessly evolving organism,[22] constantly mutating and acquiring new characteristics, including virulence factors that make the emergence of more dangerous variants a

[16] 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.

[17] 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).

[18] 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*.

[19] 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).

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

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

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

constant threat.[23] The CDC has emphasized the prospect of emerging pathogens as a significant public health threat for some time.[24]

3.24 Although foods of a bovine origin are the most common cause of both outbreaks and sporadic cases of *E. coli* O157:H7 infections,[25] 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.[26] 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.[27]

3.25 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.[28] Estimates of foodborne acquired O157:H7 cases result in 2,138 hospitalizations and 20 deaths annually.[29] The colitis caused by *E. coli* O157:H7 is characterized by severe abdominal cramps, diarrhea that typically turns bloody within twenty-four hours, and sometimes

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[23] 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.")

[24] 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.")

[25] 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).

[26] Rangel, *supra* note 14, at 605.

[27] Feng, *supra* note 12, at 49; *see also* USDA Bad Bug Book, *Escherichia coli* O157:H7.

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

[29] *Id.*, Table 3.

fevers.[30] The incubation period—which is to say the time from exposure to the onset of symptoms—in outbreaks is usually reported as three to four days, but may be as short as one day or as long as ten days.[31] Infection can occur in people of all ages but is most common in children.[32] The duration of an uncomplicated illness can range from one to twelve days.[33] 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.[34]

3.26 What makes *E. coli* O157:H7 remarkably dangerous is its very low infectious dose,[35] and how relatively difficult it is to kill these bacteria.[36] 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,"[37] as few as twenty organisms may be sufficient to infect a person and,

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30 Griffin & Tauxe, *supra* note 11, at 63.

31 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").

32 Su & Brandt, *supra* note 10 ("the young are most often affected").

33 Tauxe, *supra* note 24, at 1152.

34 *Id*.

35 Griffin & Tauxe, *supra* note 11, at 72. ("The general patterns of transmission in these outbreaks suggest that the infectious dose is low.")

36 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).

37 Griffin & Tauxe, *supra* note 11, at 72 (noting that, as a result, "fewer bacteria are needed to cause illness that for outbreaks of salmonellosis"). Nestle, *supra* note 3, at 41. ("Foods containing *E. coli* O17:H7 must be at temperatures high enough to kill *all* of them.") (italics in original).

as a result, possibly kill them.[38] And unlike generic *E. coli*, the O157:H7 serotype multiplies at temperatures up to 44° Fahrenheit, survives freezing and thawing, is heat resistant, grows at temperatures up to 111° Fahrenheit, resists drying, and can survive exposure to acidic environments.[39]

3.27 And, finally, to make it even more of a threat, *E. coli* O157:H7 bacteria are easily transmitted by person-to-person contact.[40] 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.[41] 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.[42]

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[38] 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 3, 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*.

[39] Nestle, *supra* note 3, at 41.

[40] Griffin & Tauxe, *supra* note 11, 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 14, at 605-06 (finding that 80% of the 50 reported person-to-person outbreak from 1982-2002 occurred in daycare centers).

[41] *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), available at http://www.nap.edu/books/0309086272/html/.

[42] *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).

3.28 *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."[43] There is a strong and significant relationship between acute enteric infection and subsequent IBS symptoms.[44] 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.[45] 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.[46] IBS sufferers typically experienced symptoms for an average of 8.1 days per month.[47]

3.29 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

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[43] 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*.

[44] WHS, *supra* note 43, at 449.

[45] 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.

[46] *Id*. at 1367.

[47] *Id*.

disruption to family and daily life" following an *E. coli* O157:H7 infection in the family.[48]

HEMOLYTIC UREMIC SYNDROME (HUS)

3.30 *E. coli* O157:H7 infections can lead to a severe, life-threatening complication called hemolytic uremic syndrome (HUS).[49] HUS accounts for the majority of the acute deaths and chronic injuries caused by the bacteria.[50] HUS occurs in 2-7% of victims, primarily children, with onset occurring five to ten days after diarrhea begins.[51] It is the most common cause of renal failure in children.[52] Approximately half of the children who suffer HUS require dialysis, and at least 5% of those who survive have long term renal impairment.[53] And the same number suffers severe brain damage.[54] While somewhat rare, serious injury to the pancreas, resulting in death or the development of

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

[49] Griffin & Tauxe, *supra* note 11, 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).

[50] 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.

[51] Tauxe, *supra* note 24, 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*.")

[52] Su & Brandt, *supra* note 10.

[53] Safdar, *supra* note 51, 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).

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

diabetes, can also occur.[55] There is no cure or effective treatment for HUS.[56]

3.31 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.[57] 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.[58] Some organs seem more susceptible, perhaps due to the presence of increased numbers of receptors, and include the kidney, pancreas, and brain.[59] 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).[60]

3.32 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.[61] Among survivors, at least five percent will suffer end stage renal disease (ESRD) with the resultant need for dialysis or transplantation.[62] But "[b]ecause renal

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[55] 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").

[56] Safdar, *supra* note 51, at 996. *See also* Siegler, *supra* note 54, 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.")

[57] Garg, *supra* note 51, at 1360.

[58] *Id*. Siegler, *supra* note 50, at 1509-11 (describing what Dr. Siegler refers to as the "pathogenic cascade" that results in the progression from colitis to HUS).

[59] Garg, *supra* note 51, at 1360. *See also* Su & Brandt, *supra* note 10, at 700.

[60] Garg, *supra* note51, at 1360. *See also* Su & Brandt, *supra* note 10, at 700.

[61] Siegler, *supra* note 50, 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.")

[62] Garg, *supra* note 51, at 1366-67.

failure can progress slowly over decades, the eventual incidence of ESRD cannot yet be determined."[63] 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.[64] Since the longest available follow-up studies of HUS victims are 25 years, an accurate lifetime prognosis is not really available and remains controversial.[65] 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.

IV. CAUSES OF ACTION

Strict Liability—Count I

4.1 At all times relevant hereto, the Defendant was a manufacturer and seller of the adulterated food product that is the subject of this action, and the Defendant was in the business of manufacturing and selling like products.

4.2 The adulterated food product that the Defendant manufactured and 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* O26, a harmful and potentially lethal foodborne pathogen.

4.3 The adulterated food product that the Defendant manufactured and sold was delivered to the Plaintiffs without change in its defective condition. The Plaintiffs thereafter used the product in a reasonably foreseeable manner by consuming it.

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63 Siegler, *supra* note 50, at 1519.

64 *Id*. at 1519-20. *See also* Garg, *supra* note 51, at 1366-67.

65 Garg, *supra* note 51, at 1368.

4.4 The Defendant owed a duty of care to the Plaintiffs to design, manufacture, 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. The Defendant owed a duty of care to the Plaintiffs to design, prepare, serve, and sell food that was fit for human consumption, and that was safe to the extent contemplated by a reasonable consumer. The Defendant breached this duty.

4.5 The Plaintiff's illness and associated legal injuries occurred as a direct and proximate result of the defective and unreasonably dangerous condition of the adulterated food product that the Defendant manufactured and sold.

Breach of Warranty—Count II

4.6 The Defendant is liable to the Plaintiffs for breaching express and implied warranties that it made regarding the adulterated product that caused the Plaintiffs' injuries. These express and implied warranties included, among others, the implied warranties of merchantability and fitness for a particular use. Specifically, though not exclusively, the Defendant expressly warranted, through its sale of food to the public and by the statements and conduct of its employees and agents, that its food was fit for human consumption and not otherwise adulterated or injurious to health.

4.7 The adulterated food that the Defendant manufactured and sold to the Plaintiffs would not pass without exception in the trade and was therefore in breach of the implied warranty of merchantability.

4.8 The adulterated food that the Defendant manufactured and sold to the Plaintiffs was not fit for the uses and purposes intended, *i.e.* human consumption, and the product was therefore in breach of the implied warranty of fitness for its intended use. The Plaintiff's *E. coli* O26 illness and the Plaintiffs' associated legal injuries occurred as a direct and proximate result of Defendant's several breaches of the express and implied warranties discussed above.

Negligence—Count III

4.9 The Defendant owed to the Plaintiffs a duty to use reasonable care in the manufacture, distribution, handling, and sale of its food products, the observance of which duty would have prevented or eliminated the risk that the Defendant's food products would become contaminated with *E. coli* O26 or any other dangerous pathogen, and that the Plaintiffs, or any other consumer, would be infected with *E. coli* O26 by consuming the Defendant's food products. The Defendant breached this duty.

4.10 The Defendant owed to the Plaintiffs a duty to comply with all statutes, laws, regulations, or safety codes pertaining to the manufacture, distribution, handling, and sale of its food products. The Defendant failed to observe this duty and was therefore negligent.

4.11 The Plaintiffs were among the class of persons intended to be protected by the statutes, laws, regulations, and safety codes referenced above that pertain to the manufacture, distribution, handling, and sale of similar food products.

4.12 The Defendant owed a duty to the Plaintiffs to properly supervise, train, and monitor its employees, and to ensure its employees' compliance with all applicable

statutes, laws, regulations, or safety codes pertaining to the manufacture, distribution, storage, and sale of similar food products. The Defendant failed to observe this duty and was therefore negligent.

4.13 The Defendant owed a duty to the Plaintiffs 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, and regulations, and that were clean, free from adulteration, and safe for human consumption. The Defendant failed to observe this duty and was therefore negligent.

4.14 The Plaintiff's *E. coli* O26 illness and the Plaintiffs' associated legal injuries occurred as a direct and proximate result of Defendant's several breaches of duty discussed above.

Negligence Per Se—Count IV

4.15 The Defendant had a duty to comply with all applicable state and federal regulations intended to ensure the purity and safety of its food products, including the requirements of the Federal Food, Drug and Cosmetics Act (21 U.S.C. § 301 et seq.), and the Minnesota Food Code, Minn. Stat. § 31.01, et seq.

4.16 The Defendant failed to comply with the provisions of the health and safety laws identified above, and, as a result, was negligent per se in its manufacture, distribution, and sale of food adulterated with *E. coli* O26, a harmful and potentially lethal foodborne pathogen.

4.17 Plaintiff's *E. coli* O26 illness and the Plaintiffs' associated legal injuries occurred as a direct and proximate result of conduct by the Defendant that constituted negligence per se.

V. DAMAGES

Plaintiffs are entitled to receive just compensation for the following:

(a) the pain and suffering that Plaintiffs incurred by reason of Defendant's fault and several breaches of duty, including its negligent acts and omissions, past present and future;

(b) all related medical expenses and costs incurred, past, present, and future;

(c) the physical harm that Plaintiffs suffered by reason of Defendant's fault and several breaches of duty, including its negligent acts and omissions, past, present, and future;

(d) the emotional suffering and distress that Plaintiffs suffered by reason of Defendant's fault and several breaches of duty, including its negligent acts and omissions, past, present, and future;

(e) the loss of enjoyment of life that the Plaintiffs sustained, past present, and future; and all other ordinary, incidental or consequential damages that could be reasonably anticipated to arise under the circumstances, including, but not limited to, loss of earning capacity.

PRAYER FOR RELIEF

WHEREFORE, the Plaintiffs pray for judgment against the Defendant as follows:

A. Judgment against the Defendant for just compensation in a fair and reasonable amount for all general, special, incidental and consequential damages suffered by Plaintiffs as a result of Defendant's conduct; and

B. Judgment in a fair and reasonable amount for expenses and legal injuries incurred, in a fair and reasonable amount for the damages suffered; and

C. Such additional and/or further relief, including interest, costs, and reasonable attorney fees, as this Court deems just and equitable.

DATED: October 2, 2018.

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