UNITED STATES DISTRICT COURT
WESTERN DISTRICT OF WASHINGTON

JACQUELINE SHARON RAE MINTY AND SEAN THOMAS MINTY, as parents of S.R.M., Plaintiffs, v. GRIMMWAY ENTERPRISES, INC.; Defendant.	CASE NO. **COMPLAINT FOR DAMAGES AND JURY DEMAND**

NOW come Plaintiffs, JACQUELINE SHARON RAE MINTY AND SEAN THOMAS MINTY, as parents of S.R.M., by and through their attorney of record, William D. Marler, Esq. of Marler Clark, Inc., PS, and allege upon information and belief as follows:

PARTIES

1. The Plaintiffs, Jacqueline Sharon Rae Minty and Sean Thomas Minty, parents of S.R.M. ("Plaintiffs"), reside in Snoqualmie, King County, Washington, and are therefore citizens of the State of Washington.

2. Defendant GRIMMWAY ENTERPRISES, INC., ("Grimmway" or "Defendant") is a corporation organized and existing under the laws of Delaware with its principal place of business

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located at 12064 Buena Vista Blvd, Arvin, CA 93203. Grimmway is therefore a citizen of both Delaware and California. Upon information and belief, at all times material to this matter, Grimmway manufactured, distributed, and sold the adulterated food product at issue in this matter, carrots, to grocery stores across the United States, including in the State of Washington, and specifically to the store Plaintiffs purchase the carrots from.

JURISDICTION AND VENUE

3. This Court has jurisdiction over the subject matter of this action pursuant to 28 U.S.C. section 1332(a) because the matter in controversy far exceeds $75,000.00, exclusive of costs, and it is between citizens of different states.

4. Venue in the United States District Court of the Western District of Washington is proper pursuant to 28 U.S.C. section 1391(b)(2) as a substantial part of the events giving rise to Plaintiffs' claims occurred in the Western District when Plaintiffs purchased, and S.R.M. consumed and was sickened by, Defendant's product there.

5. Defendant is subject to personal jurisdiction in the District Court for the Western District of Washington as, at all times relevant to this matter, Defendant contracted to do business and supplied the adulterated food product to stores in the State of Washington, including the specific food product and grocery store at issue in this complaint. As such, Defendant maintains minimum contacts with the Western District of Washington such that maintenance of this suit in this Court is appropriate, fair, and just.

GENERAL ALLEGATIONS

The 2024 *E. coli* O121 Outbreak

6. As of November 17, 2024, 39 people infected with the outbreak strain of *E. coli* have been reported from 18 states according to the CDC:

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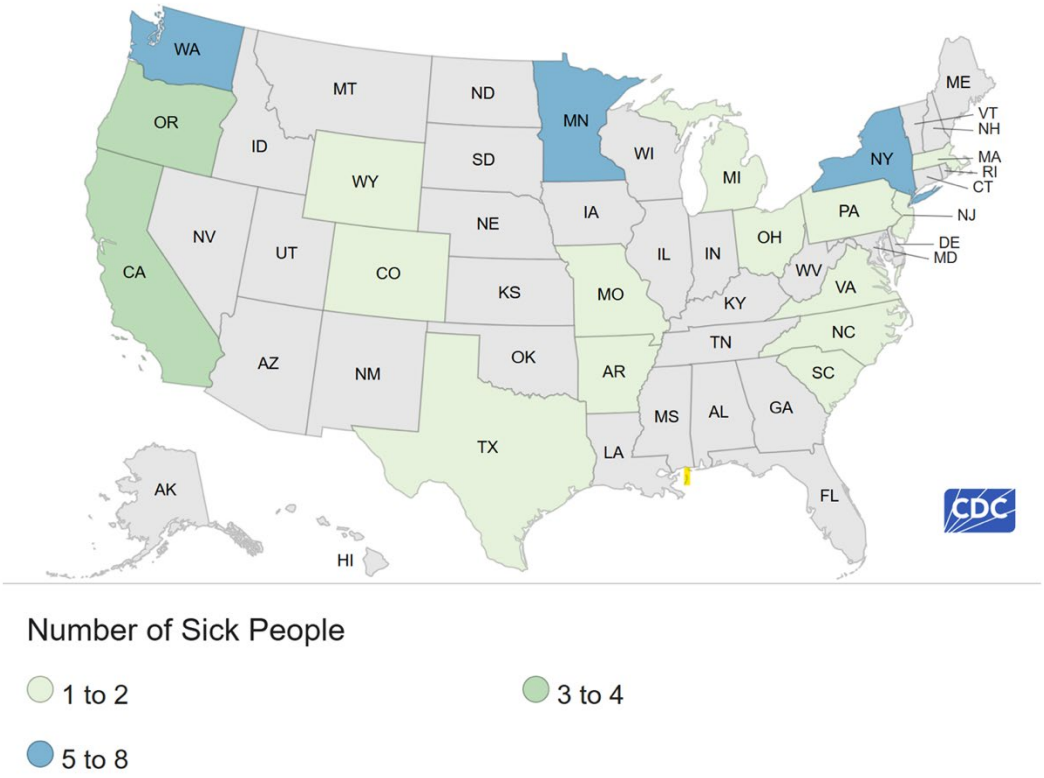
Wyoming 1, Washington 8, Virginia 1, Texas 1, South Carolina 2, Pennsylvania 1, Oregon 3, Ohio 1, North Carolina 1, New York 5, New Jersey 2, Missouri 1, Minnesota 5, Michigan 1, Massachusetts 1, Colorado 1, California 3, Arkansas 1



7. Illnesses started on dates ranging from September 6, 2024 to October 28, 2024. Of 38 people with information available, 15 have been hospitalized and none developed hemolytic uremic syndrome, a serious condition that can cause kidney failure. One death has been reported from California.

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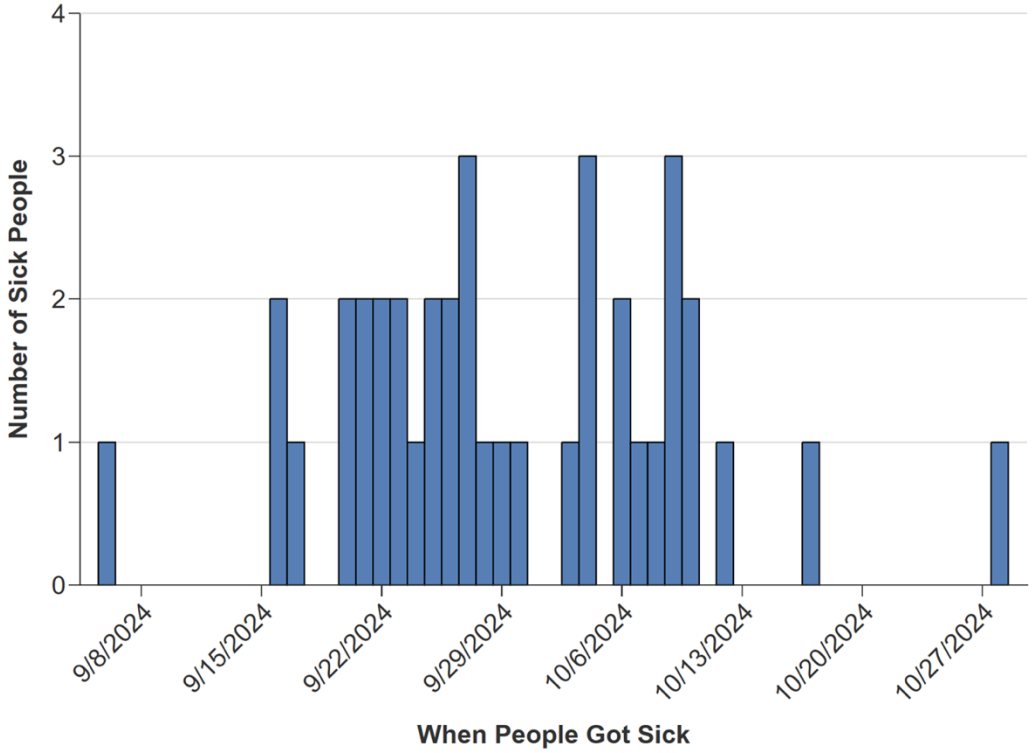

8. CDC and public health officials in several states are investigating a multistate outbreak of *E. coli* O121 infections linked to multiple brands of recalled organic whole bagged carrots and baby carrots sold by Grimmway Farms. Carrots on store shelves right now are likely not affected but may be in people's homes.

9. The true number of sick people in this outbreak is likely much higher than the number reported, and the outbreak may not be limited to the states with known illnesses. This is because many people recover without medical care and are not tested for *E. coli*. In addition, recent illnesses may not yet be reported as it usually takes 3 to 4 weeks to determine if a sick person is part of an outbreak.

Outbreak sub-cluster: 35 Isolates

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Distance between selected isolates: minimum = 0 SNPs, maximum = 17 SNPs, average = 2 SNPs (34 isolates, without the bottom one that is on its own branch, minimum = 0 SNPs, maximum = 6 SNPs, average = 2 SNPs)

WGS date range: 2024-10-04 to 2024-11-12.

The *E. coli* Bacteria

10. *E. coli* is an archetypal commensal bacterial species that lives in mammalian intestines. *E. coli* O121, like O157:H7, is one of thousands of serotypes *Escherichia coli*.[1] The combination of letters and numbers in the name of the *E. coli* O121 refers to the specific antigens (proteins which

[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.*

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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 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* O121 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]

11. *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

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[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).

[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 5, 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 6, 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).

[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.

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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) 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]

12. *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

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[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 11 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).

[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 14 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).

[16] Griffin & Tauxe, *supra* note 12, at 61-62 (noting that the nomenclature came about because of the resemblance to toxins produced by Shigella dysenteries).

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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]

13. *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

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[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: 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.")

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pathogens as a significant public health threat for some time.[25]

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

15. 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

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[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 15, at 605.

[28] Feng, *supra* note 13, at 49. *See also* USDA Bad Bug Book, *Escherichia coli* O157: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 12, 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").

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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]

16. 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, 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]

17. And, finally, to make it even more of a threat, *E. coli* O157:H7 bacteria are easily

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[33] Su & Brandt, *supra* note 11 ("the young are most often affected").

[34] Tauxe, *supra* note 25, at 1152.

[35] *Id.*

[36] Griffin & Tauxe, *supra* note 12, at 72. ("The general patterns of transmission in 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 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 12, at 72 (noting that, as a result, "fewer bacteria are needed to cause illness that for outbreaks of salmonellosis"). Nestle, *supra* note 4, 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 4, 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.*

[40] Nestle, *supra* note 4, at 41.

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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 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]

18. *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

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[41] Griffin & Tauxe, *supra* note 12, 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 15, at 605-06 (finding that 80% of the 50 reported person-to-person outbreak from 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/.

[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 12, 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 25, 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-*

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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]

19. 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]

20. As already noted, there is no known therapy to halt the progression of HUS. HUS is a

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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 11.

[48] Safdar, *supra* note 46, 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 46, at 996. *See also* Siegler, *supra* note 49, 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.")

[52] Garg, *supra* note 46, at 1360.

[53] *Id*. Siegler, *supra* note 45, at 1509-11 (describing what Dr. Siegler refers to as the "pathogenic cascade" that results in the progression from colitis to HUS).

[54] Garg, *supra* note 46, at 1360. *See also* Su & Brandt, *supra* note 11, at 700.

[55] Garg, *supra* note46, at 1360. *See also* Su & Brandt, *supra* note 11, at 700.

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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 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.

21. 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]

22. 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.*

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[56] Siegler, *supra* note 45, 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.")

[57] Garg, *supra* note 46, at 1366-67.

[58] Siegler, *supra* note 45, at 1519.

[59] *Id*. at 1519-20. *See also* Garg, *supra* note 46, at 1366-67.

[60] Garg, *supra* note 46, at 1368.

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

[62] *Id*.

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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]

23. 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 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.

24. *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

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[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.

[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).

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susceptible than adults to reactive arthritis following infection with *Salmonella*.[68]

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

26. 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]

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[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).

[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).

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27. 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]

28. 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 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]

29. 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

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[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.
[77] *Id*.at 1367.
[78] *Id*.
[79] *Id*. at 1368.
[80] *Id*.
[81] *Id*.

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cause IBS or its symptoms.[82]

S.R.M.'s *E. coli* O121 Infection

30. On or about September 23, 2024, Plaintiff Jacqueline Sharon Rae Minty purchased O Organic Peel Baby carrots at Safeway in Snoqualmie, Washington.

31. S.R.M. consumed the carrots as part of her lunch at school during the week of September 24 to September 27, 2024, as well as with some dinners that week.

32. S.R.M. first develop symptoms on or about Sunday, September 29, 2024, when she began to have diarrhea and slight stomach cramping.

33. S.R.M.'s condition became significantly worse on the evening of September 30 and the early hours of October 1; S.R.M. had to go to the bathroom with diarrhea every hour and was sobbing with severe cramping pain the entire night and could not fall asleep.

34. At about 6:00 AM on October 1, when S.R.M. went to the bathroom, there was the first trace of blood in her stool. Concerned, Plaintiff Jacqueline Sharon Rae Minty took her to the Overlake Urgent Care in Issaquah at 7:00 AM. There, the Urgent Care Doctor recommended she take S.R.M. to the Emergency Room at Children's Hospital.

35. Plaintiff Jacqueline Sharon Rae Minty and S.R.M. arrived at Children's Hospital shortly after 8:00 AM on October 1, 2024. S.R.M. continued to be in severe pain, which brought her to tears multiple times. By this time, S.R.M.'s every bathroom visit included a large amount of blood in her diarrhea, and S.R.M. felt like she needed to go every 20 minutes.

36. At Children's Hospital, S.R.M. received an ultrasound to rule out appendicitis, and she

[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).

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gave a stool sample for the team later that morning. By 1:00 PM, the Emergency Room Doctor was able to confirm the stool culture results: positive for O121 Shiga Toxin *E. coli*. S.R.M. was admitted.

37. Upon admission to Children's Hospital, S.R.M. could not eat and drinking was difficult, so she received IV for fluid and nutrition. The only medicine S.R.M. could receive for pain management was Tylenol but she mostly had to just endure the pain during her stay at Children's Hospital.

38. S.R.M. had severe cramping all day, every day and had to use the restroom every 30 minutes all day. She had large amounts of blood in her stool, which persisted until about October 7, 2024. S.R.M. was not able to get more than 1-2 hours of sleep at a time during her hospital stay. She was eventually discharged to home care on October 3, 2024.

39. S.R.M.'s digestive system has been very sensitive since she returned home, and it has taken her weeks to get back to her normal appetite and sleep schedule

40. S.R.M. has been able to recover physically, but the experience has created a lot of stress and anxiety surrounding food and going to the bathroom, which is not something she ever worried about or had prior to falling sick.

CAUSES OF ACTION

Count I – Strict Products Liability

41. Plaintiffs incorporate by reference and make a part of this Count each foregoing paragraph of this Complaint.

42. At all times relevant hereto, Defendant was the manufacturer and distributor of the adulterated and/or harmful food product that was consumed by S.R.M.

43. The adulterated and/or harmful food product that Defendant manufactured, and

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distributed was, at the time it left Defendant's control, defective and unreasonably dangerous for its ordinary and expected use by the intended public, including Plaintiffs, because Defendant's product was adulterated and/or harmful to human health by virtue of being contaminated with *E. coli O121*.

44. The adulterated and/or harmful food product that Defendant manufactured and distributed was delivered to Plaintiffs without any change in its defective condition. The adulterated and/or harmful food product that Defendant manufactured and distributed was consumed by S.R.M. in a manner to be expected.

45. Defendant owed a duty of care to the public, including Plaintiffs, to manufacture and distribute food that was not adulterated and/or harmful and that was free of pathogenic bacteria or other substances injurious to human health. Defendant breached this duty.

46. Defendant owed a duty of care to the public, including Plaintiffs, to manufacture and distribute food that was fit for human consumption and that was safe to consume to the extent contemplated by a reasonable consumer. Defendant breached this duty.

47. As a direct and proximate result of the defective and unreasonably dangerous condition of the adulterated and/or harmful food product that Defendant manufactured and distributed, as set forth above, S.R.M. developed an *E. coli* O121 infection and related illness and injury. S.R.M. suffered physical injury and pain and loss of enjoyment of life, all of which amount of economic injury in an amount to be proved at trial. Plaintiffs also incurred, and will incur, medical bills, amounting to economic damages in an amount to be proved at trial.

Count II - Negligence

48. Plaintiffs incorporate by reference and make a part of this Count each foregoing paragraph of this Complaint.

49. Defendant owed to Plaintiffs a duty to use reasonable care in the manufacture and distribution of its food product, the observance of which duty would have prevented or eliminated the risk that Defendant's food product would become adulterated and/or harmful with any

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dangerous pathogen. Defendant, however, breached this duty and was therefore negligent.

50. 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.

51. Plaintiffs were among the class of persons designed to be protected by these statutes, laws, regulations, safety codes, and provisions pertaining to the manufacture, packaging, distribution, and sale of similar food products. Defendant however, breached this duty and was therefore negligent.

52. Defendant had a duty to properly supervise, train, and monitor its employees and to ensure that its employees complied with all applicable statutes, laws, regulations, safety codes, and provisions pertaining to the manufacture, distribution, packaging, and sale of similar food products. Defendant, however, breached this duty and was therefore negligent.

53. 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. Defendant, however, breached this duty and was therefore negligent.

54. As a direct and proximate result of Defendant's negligence as described above, Plaintiffs and S.R.M. sustained injuries and damages in an amount to be determined at trial.

Count III – Negligence *Per Se*

55. Plaintiffs incorporate by reference and make a part of this Count each foregoing paragraph of this Complaint.

56. Defendant had a duty to comply with all statutory and regulatory provisions that pertained or applied to the manufacture, distribution, storage, labeling, and sale of the food products that injured S.R.M., including the applicable provisions of the Federal Food, Drug and Cosmetic Act, and similar state and local regulations relating to the manufacture, distribution, and

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sale of food, which prohibit the sale of any food that is adulterated or otherwise injurious to health.

57. In breach of this duty, 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, packaging, and/or sale of adulterated food.

58. As a direct and proximate result of conduct by Defendant that was negligent *per se*, Plaintiffs and S.R.M. sustained injuries and damages in an amount to be determined at trial.

Count IV - Breach of Express and Implied Warranties

59. Plaintiffs incorporate by reference and make a part of this Count each foregoing paragraph of this Complaint.

60. Defendant manufactured, produced, distributed, and sold the contaminated food product that injured S.R.M. and caused her *E. coli* O121 infection. Defendant is, therefore, a manufacturer, distributor, and/or seller of an adulterated food product, and the adulterated food product reached Plaintiffs and was ultimately consumed by S.R.M. without substantial change from the condition in which it was sold by Defendant.

61. Defendant is subject to liability to Plaintiffs and S.R.M. for its breaches of express and implied warranties made to Plaintiffs with respect to the food product sold to them, including the implied warranties of merchantability and of fitness for a particular use. Further, Defendant expressly warranted, through the sale of food to the public, and by the statements and conduct of its employees and agents, that the food product ultimately sold to Plaintiffs and consumed by S.R.M. was fit for human consumption, and not otherwise adulterated or injurious to health.

62. The food product sold by Defendant and ultimately consumed by S.R.M., which product was contaminated with *E. coli* O121 and related filth and adulteration, would not pass without exception in the trade, and was thus in breach of the implied warranty of merchantability.

63. Plaintiffs further allege that the contaminated food sold by Defendant and consumed by S.R.M. was not fit for the uses and purposes intended by either Plaintiffs, S.R.M., or Defendant, i.e., human consumption, and that this product was therefore in breach of the implied warranty of

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fitness for its intended use by virtue of its contamination with *E. coli* O121.

64. As a further direct and proximate result of the conduct of Defendant and its agents, servants, and/or employees as aforesaid, S.R.M. suffered an *E. coli* O121 infection and the adverse effects associated with the same, as described in previous paragraphs of this complaint.

65. As a further direct and proximate result of the conduct of Defendant and its agents, servants, and/or employees, S.R.M. was forced to endure great pain, suffering, and inconvenience and may endure the same in the future. She was forced to submit to medical care and may be forced to submit to the same in the future.

66. As a further direct and proximate result of the conduct of Defendant and its agents, servants, and/or employees, S.R.M. suffered an inability to perform the activities of daily living or some of them, and Plaintiffs and S.R.M. sustained injuries and damages in an amount to be determined at trial.

DAMAGES

67. Plaintiffs suffered general, special, incidental, and consequential damages as the direct and proximate result of the acts and omissions of Defendant, in an amount that shall be fully proven at the time of trial. These damages include but are not limited to past and future pain and suffering, past and future damages for loss of enjoyment of life, past and future emotional distress, past and future medical and related expenses, including pharmaceutical expenses, travel, and travel-related expenses, and all other ordinary, incidental, or consequential damages that would or could be reasonably anticipated to arise under the circumstances.

JURY DEMAND

68. Plaintiffs hereby demands a jury trial.

PRAYER FOR RELIEF

WHEREFORE, Plaintiffs pray for judgment against Defendant as follows:

a. Ordering compensation for all general, special, incidental, and consequential damages

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suffered by Plaintiffs because of Defendant's conduct.

b. Plaintiffs demand $1,000,000 dollars in damages.

c. Awarding Plaintiffs costs and expenses, including reasonable attorneys' fees to the fullest extent allowed by law; and

d. Granting all such additional and/or further relief as this Court deems just and equitable.

Dated: November 20, 2024

MARLER CLARK, INC., PS.

By: /s/ *William D. Marler*

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