IN THE UNITED STATES DISTRICT COURT
WESTERN DISTRICT OF VIRGINIA
Harrisonburg Division

DONNA JOY GARBER **551 Elgin Dr.,** **Luray, VA 22835**	**:**	
Plaintiff,	**:**	
v.	**:**	**Civil Action No. _____**
GRIMMWAY ENTERPRISES, INC. **12064 Buena Vista Blvd,** **Arvin, CA 93203**	**:** **:**	
SERVE: Corporation Service Company **Registered Agent** **330 N Brand Blvd,** **Glendale, CA 91203**	**:** **:**	
Defendant.	**:**	

COMPLAINT

COMES NOW, Plaintiff Donna Garber, by and through undersigned counsel, and respectfully moves for judgment against Defendant on the grounds and in the amount set forth below:

JURISDICTION AND VENUE

1. The jurisdiction of this Court is proper pursuant to 28 U.S.C. § 1332(a), since the matter in controversy far exceeds, exclusive of interests and costs, the sum of Seventy-Five Thousand Dollars ($75,000.00) and there is diversity of citizenship between Plaintiff and Defendant.

2. Venue is proper in this judicial district as the facts giving rise to Plaintiff's Complaint arose in this judicial district.

PARTIES

3. Plaintiff is an adult resident of the Commonwealth of Virginia.

4. Defendant Grimmway Enterprises, Inc. (Grimmway Farms) is a Delaware corporation with its principal place of business in Arvin, CA. As such, Defendant is a citizen of both Delaware and California. Notwithstanding, Defendant regularly conducts business in the Commonwealth of Virginia.

5. At all times relevant to this action, Defendant was engaged in the manufacture, distribution, and sale of carrots to customers nationwide, including within the Commonwealth of Virginia.

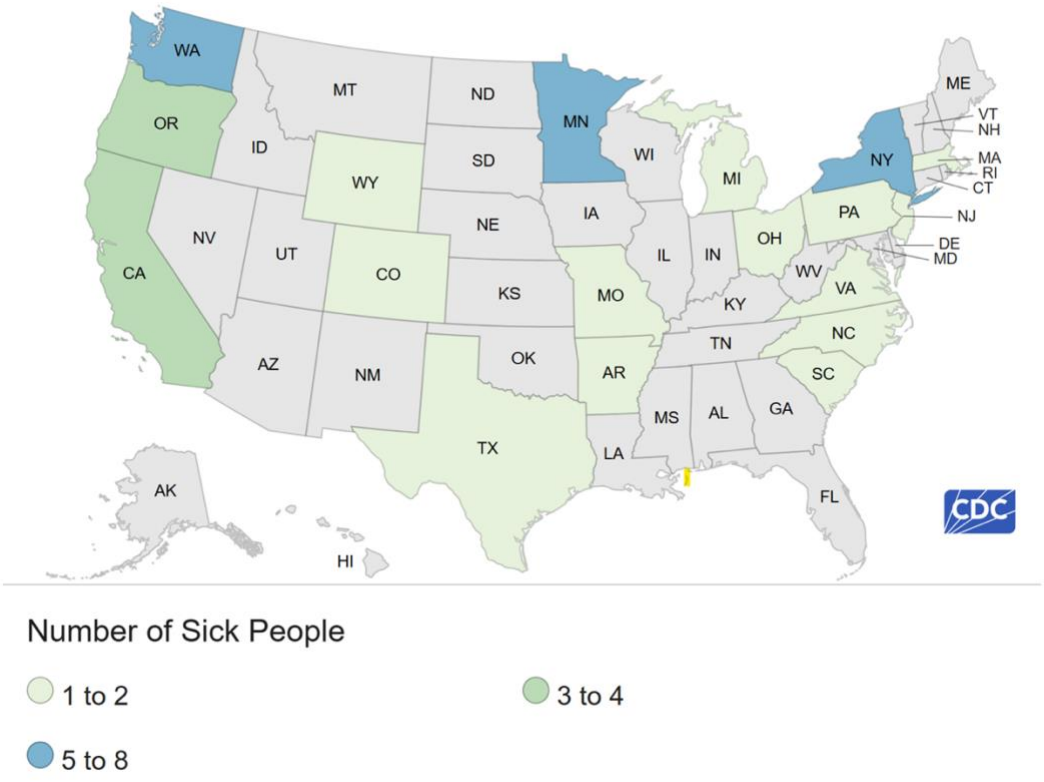
FACTS

6. Plaintiff repeats and realleges the prior allegations as if set forth herein.

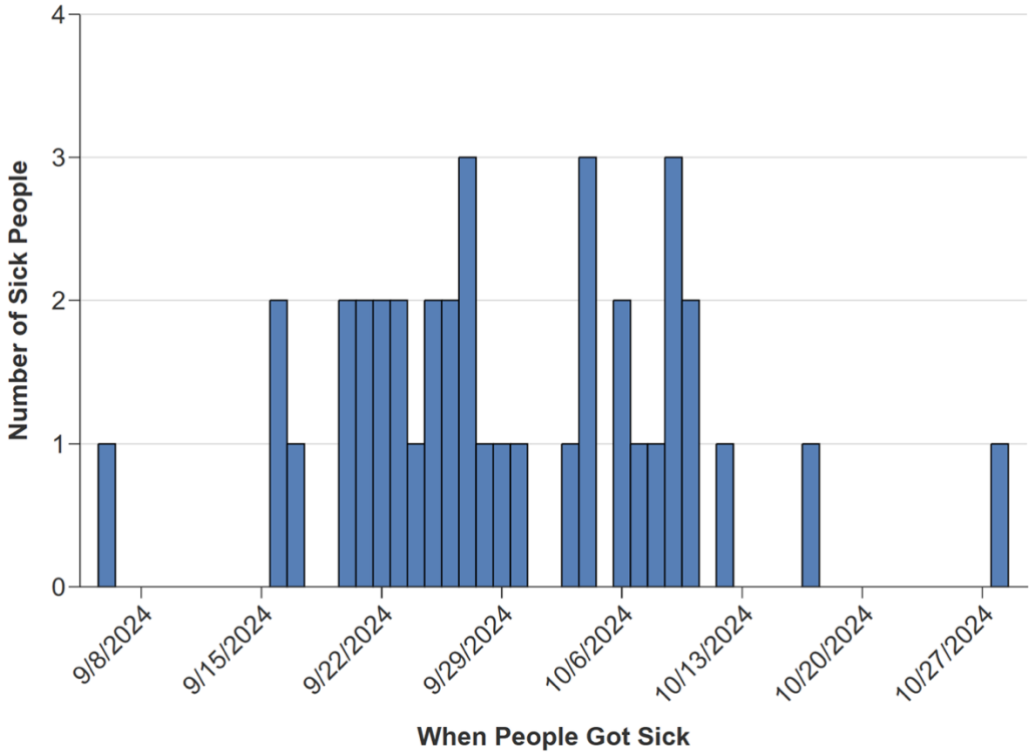
7. This is an action against Defendant for injuries arising from the manufacture, distribution, and sale of contaminated organic carrots which were consumed by Plaintiff Donna Garber and caused her serious injuries.

The 2024 *E. coli* O121 Outbreak

8. 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: 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.



9. 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.



10. 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. If you have any recalled carrots in your home, throw them out or return them to the store.

11. 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 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

12. *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

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,

combination of letters and numbers in the name of the *E. coli* O121 refers to the specific antigens (proteins which provoke an antibody response) found on the body and tail or flagellum[2] respectively and distinguish it from other types of *E. coli.*[3] Most serotypes of *E. coli* are harmless and live as normal flora in the intestines of healthy humans and animals.[4] The *E. coli* bacterium is among the most 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]

13. *E. coli* O157:H7 was first recognized as a pathogen in 1982 during an investigation

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an important public health discovery. *Id.* Although the bacteria were initially called Bacterium coli, the name was later changed to *Escherichia coli* to honor its discoverer. *Id.*

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

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

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

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.

into an outbreak of hemorrhagic colitis[10] associated with consumption of hamburgers from a fast food chain restaurant.[11] Retrospective examination of more than three thousand *E. coli* cultures obtained between 1973 and 1982 found only one (1) isolation with serotype O157:H7, and that was a case in 1975.[12] In the ten (10) years that followed there were approximately thirty (30) 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]

14. *E. coli* O157:H7's ability to induce injury in humans is a result of its ability to

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

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

12 Riley, *supra* note 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).

produce numerous virulence factors, most notably Shiga-like toxins.[16] Shiga toxin (Stx) has multiple variants (e.g. Stx1, Stx2, Stx2c), and acts like the plant toxin ricin by inhibiting protein synthesis in endothelial and other cells.[17] Shiga toxin is one of the most potent toxins known.[18] In addition to Shiga toxins, *E. coli* O157:H7 produces numerous other putative virulence factors including proteins, which aid in the attachment and colonization of the bacteria in the intestinal wall and which can lyse red blood cells and liberate iron to help support *E. coli* metabolism.[19]

15. *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

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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).

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.* Prob**in**g genomic diversity and evolution **of *Escherichia coli*** O157 by s**in**gle 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).

a constant threat.[24] The CDC has emphasized the prospect of emerging pathogens as a significant public health threat for some time.[25]

16. 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]

17. 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

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

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

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

27 Rangel, *supra* note 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.

(4) days, but may be as short as one (1) day or as long as ten (10) days.[32] Infection can occur in people of all ages but is most common in children.[33] The duration of an uncomplicated illness can range from one (1) to twelve (12) days.[34] In reported outbreaks, the rate of death is 0-2%, with rates running as high as 16-35% in outbreaks involving the elderly, like those have occurred at nursing homes.[35]

18. 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

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

33 Su & Brandt, *supra* note 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.*

exposure to acidic environments.[40]

19. And, finally, to make it even more of a threat, *E. coli* O157:H7 bacteria are easily transmitted by person-to-person contact.[41] There is also the serious risk of cross-contamination between raw meat and other food items intended to be eaten without cooking. Indeed, a principle and consistent criticism of the USDA *E. coli* O157:H7 policy is the fact that it has failed to focus on the risks of cross-contamination versus that posed by so-called improper cooking.[42] With this pathogen, there is ultimately no margin of error. It is for this precise reason that the USDA has 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]

20. *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

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

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.

to ten days after diarrhea begins.[46] It is the most common cause of renal failure in children.[47] Approximately half of the children who suffer HUS require dialysis, and at least 5% of those who survive have long term renal impairment.[48] The same number suffers severe brain damage.[49] While somewhat rare, serious injury to the pancreas, resulting in death or the development of diabetes, can also occur.[50] There is no cure or effective treatment for HUS.[51]

21. 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

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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-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.

platelet count), and renal failure (loss of kidney function).[55]

22. As already noted, there is no known therapy to halt the progression of HUS. HUS is a frightening complication that even in the best American centers has a notable mortality rate.[56] Among survivors, at least five percent will suffer end stage renal disease (ESRD) with the resultant need for dialysis or transplantation.[57] But, "[b]ecause renal failure can progress slowly over decades, the eventual incidence of ESRD cannot yet be determined."[58] Other long-term problems include the risk for hypertension, proteinuria (abnormal amounts of protein in the urine that can portend a decline in renal function), and reduced kidney filtration rate.[59] Since the longest available follow-up studies of HUS victims are 25 years, an accurate lifetime prognosis is not really available 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.

23. 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]

24. 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

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

accompanied by conjunctivitis (inflammation of the eyes), and urethritis (painful urination). *Id.* This triad of symptoms is called Reiter syndrome.[63] Reiter syndrome, a form of reactive arthritis, is an uncommon but debilitating syndrome caused by gastrointestinal or genitourinary infections. The most common gastrointestinal bacteria involved are *Salmonella*, *Campylobacter*, *Yersinia*, and *Shigella*. Reiter syndrome is characterized by a triad of arthritis, conjunctivitis, and urethritis, although not all three symptoms occur in all affected individuals.[64]

25. 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.

26. *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

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62 *Id.*

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

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

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.

be more likely those of Asian descent to develop reactive arthritis,[67] and children may be less susceptible than adults to reactive arthritis following infection with *Salmonella*.[68]

27. 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]

28. 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:

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

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

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

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

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.

29. 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]

30. 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]

31. 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]

32. 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

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73 *Id*. at 445 (citing multiple sources).
74 WHS, *supra* note 34, at 449.
75 *Id*. at 447.
76 A.P.S. Hungin, *et al., Irritable Bowel Syndrome in the United States: Prevalence, Symptom Patterns and Impact,* Aliment Pharmacol. Ther. 2005:21 (11); 1365-75.
77 *Id*.at 1367.
78 *Id*.
79 *Id*. at 1368.
80 *Id*.

long drive, going to a restaurant, or taking a vacation.[81] Finally, although a patient's psychological state may influence the way in which he or she copes with illness and responds to treatment, there is no evidence that supports the theory that psychological disturbances in fact cause IBS or its symptoms.[82]

Donna Garber's *E. coli* O121 Infection

33. At all relevant times, Plaintiff Donna Garber regularly purchased and consumed Defendant's carrots from Kroger's in Harrisburg, Virginia, and Walmart in Luray, Virginia.

34. On or about November 12, 2024, Plaintiff began experiencing symptoms of illness, including abdominal cramps and diarrhea, which quickly progressed to bloody diarrhea. Plaintiff also developed malaise and lack of appetite.

35. When her symptoms persisted and worsened, Plaintiff sought medical attention on November 14, 2024, at the Page Memorial Hospital Emergency Room.

36. At the Emergency Room, Plaintiff underwent blood tests and a CT scan. The CT scan revealed acute colitis, and Plaintiff was subsequently admitted to the hospital for treatment.

37. During her hospitalization, Plaintiff provided a stool sample, which was later confirmed to be positive for *Escherichia coli* O121.

38. Plaintiff remained at the hospital until November 17, 2024.

39. Plaintiff continues to suffer from weakness, fatigue, loss of appetite, and persistent stomach cramping.

40. As a direct result of being sickened by Defendant's defective food product, Plaintiff

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81 *Id.*

82 Amy Foxx-Orenstein, DO, FACG, FACP, *IBS—Review and What's New*, General Medicine 2006:8(3) (Medscape 2006) (collecting and citing studies). Indeed, PI-IBS has been found to be characterized by more diarrhea but less psychiatric illness with regard to its pathogenesis. *See* Nicholas J. Talley, MD, PhD, *Irritable Bowel*

has incurred, and will continue to incur, substantial medical bills and expenses associated with the treatment of her injuries; has suffered, and will continue to suffer, lost wages and wage-earning capacity; and has suffered, and will continue to suffer, significant pain, emotional anguish, and other damages.

COUNT I
(Negligence)

41. Plaintiff repeats and realleges the prior allegations as fully set forth herein.

42. At all relevant times, Defendant was engaged in the business of manufacturing, distributing, supplying and introducing into the stream of commerce food products intended for human consumption.

43. Defendant had a duty to Plaintiff and others avoid manufacturing, distributing, supplying, and introducing into the stream of commerce contaminated food, including carrots. Defendant breached this duty.

44. Defendant owed a duty to the Plaintiff and others to use supplies and raw materials that complied with federal, state, and local food laws, ordinances, and regulations, including without limitation the statutes in Code of Virginia Title 3.2, Subtitle IV, Chapter 51, Articles 1-3; that were safe and reliable sources; that were clean, wholesome, and free from adulteration; and that were safe for human consumption and for their intended purposes. Defendant breached this duty.

45. Defendant owed a duty to Plaintiff and others to use reasonable care in the handling, manufacture, storage, and distribution of its food products, to keep them free of contamination with *E. coli* O121. Defendant breached this duty.

46. Defendant's actions as described herein were negligent. As a result of Defendant's

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Syndrome: From Epidemiology to Treatment, from American College of Gastroenterology 68th Annual Scientific

negligence and noncompliance with applicable law and safety regulations, it manufactured, distributed, and sold food products that were not reasonably safe, and, as a proximate and direct result, caused Plaintiff to suffer severe personal injuries, as well as economic loss; caused her to suffer bodily pain and mental anguish; caused her to suffer past and future pain of body and mind; caused her to incur past and future medical and related expenses; and caused her to suffer other past and future damages.

COUNT II
(Breach of Express Warranty)

47. Plaintiff incorporates by reference the preceding paragraphs as if fully set forth herein.

48. Defendant is a manufacturer, distributor, supplier, and seller of carrots. Defendant, through its manufacture, distribution, supply, and sale of carrots, expressly warranted that its products were reasonably safe for their ordinary and foreseeable purpose (*i.e.*, consumption).

49. Defendant was the manufacturer, distributor, supplier, and seller of the carrots consumed by Plaintiff that caused Plaintiff's exposure to *E. coli* O121 infection.

50. Defendant did not disclaim the warranties.

51. To the contrary, Defendant marketed its carrots, expressly promising that they were healthy and safe for consumption.

52. Plaintiff is a consumer.

53. The carrots manufactured, supplied, and sold by Defendant were contaminated with *E. coli* O121, a potentially fatal pathogen. As such, the carrots were unreasonably dangerous for their ordinary and foreseeable use.

Meeting and Postgraduate Course (Medscape 2003).

54. The carrots were contaminated with *E. coli* O121 when they left the possession and control of Defendant and were subsequently consumed by Plaintiff.

55. Defendant breached the warranty of the safety of its goods for their expected and foreseeable purpose. This breach was the direct and proximate cause of Plaintiff's personal, economic, and other injuries, and Defendant is therefore liable to Plaintiff for the injuries Defendant caused.

COUNT III
(Breach of Implied Warranty of Merchantability, Fitness For a Particular Purpose, and Wholesomeness)

56. Plaintiff repeats and realleges the prior allegations as fully set forth herein.

57. Under Virginia Code § 8.2-315, where a seller at the time of contracting has reason to know any particular purpose for which the goods are required and that the buyer is relying on the seller's skill or judgment to furnish suitable goods, there is an implied warranty that the foods shall be merchantable, fit for such purpose, and wholesome.

58. At the time of sale, a merchant of food for human consumption impliedly warrants that the food is merchantable, fit for a particular purpose (consumption), and wholesome.

59. Defendant was a merchant of food products.

60. The food that Defendant manufactured, distributed, supplied, and sold was not merchantable, fit for Plaintiff's consumption, or wholesome because it was contaminated by *E. coli* O121.

61. In manufacturing, distributing, supplying, and selling the contaminated food, Defendant breached the implied warranties as described above.

62. As a direct and proximate result of the breach of implied warranties by Defendant,

Plaintiff was caused to suffer serious injuries, has suffered bodily pain and mental distress, has suffered and will suffer in the future pain of body and mind, has incurred medical and related expenses, and has suffered and will suffer in the future other damages.

COUNT IV
(Breach of Implied Warranty of Merchantability)

63. Plaintiff repeats and realleges the prior allegations as fully set forth herein.

64. Under Virginia Code § 8.2-314, a merchant that sells goods impliedly warrants that the goods are merchantable; *i.e.*, that the goods will pass without objection in the trade under the contract description; that the goods are fit for the ordinary purposes for which such goods are used; and, that the goods are adequately contained, packaged, and labeled as the agreement requires.

65. The food that Defendant manufactured, distributed, supplied, and sold was objectionable because it contained *E. coli* O121.

66. In manufacturing, distributing, supplying, and selling the contaminated food, Defendant breached the implied warranties as described above.

67. As a direct and proximate result of the breach of implied warranties by Defendant, Plaintiff was caused to suffer serious injuries; has suffered bodily pain and mental anguish; has suffered, and will suffer, in the future pain of body and mind; has incurred, and will continue to incur, medical and related expenses; and has suffered, and will suffer, other damages.

COUNT V
(Negligence *Per Se*)

68. Plaintiff repeats and realleges the prior allegations as fully set forth herein.

69. At all relevant times, the food that Defendant manufactured, distributed, supplied, and sold was adulterated with *E. coli* O121 and was poisonous to Plaintiff.

70. Virginia Code § 3.2-5126 prohibits the manufacture, sale, delivery, and offering or sale of adulterated food.

71. Virginia Code § 3.2-5126 prohibits the dissemination of any false advertisement in connection with food.

72. Virginia Code § 3.2-5126 prohibits the giving of a guaranty or undertaking concerning a food, which guaranty or undertaking is false.

73. Plaintiff is a member of the class of people for whose protection Virginia Code § 3.2-5126 and Title 3.2, Chapter 31, Article 3 of the Virginia Code were enacted.

74. The aforesaid violations of Virginia Code § 3.2-5126 constitute negligence *per se.*

75. As a direct and proximate result of the aforesaid violations, acts of and/or omissions, Plaintiff was caused to be grievously injured.

76. As a direct and proximate result of Defendant's negligence *per se* as described herein, Plaintiff was caused to suffer serious injuries; has suffered bodily pain and mental anguish; has suffered, and will suffer in the future, pain of body and mind; has incurred, and will continue to incur, medical and related expenses; and has suffered, and will suffer in the future, other damages.

COUNT VI
(Virginia Consumer Protection Act)

77. Plaintiff repeats and realleges the prior allegations as fully set forth herein.

78. At all relevant times, Defendant was a supplier of goods under the Virginia Consumer Protection Act, Virginia Code § 59.1-196, *et seq*.

79. Defendant marketed its carrots, which were consumed by Plaintiff, as "fresh, healthy, and safe produce" and "fresh, premium carrots" on their website.

80. At all relevant times, Defendant was engaged in a consumer transaction with Plaintiff,

and Defendant intended Plaintiff to rely on its representations.

81. The Virginia Consumer Protection Act prohibits a supplier that is engaged in a consumer transaction from misrepresenting that its goods and services have certain characteristics, ingredients, uses, or benefits; mispresenting that its goods and services are of a particular standard or quality; and, using any other deception, fraud, false pretense, false promise, or misrepresentation in connection with a consumer transaction.

82. The carrots consumed by Plaintiff were not safe, nor were they "fresh, healthy, and safe" as they were contaminated with *E. coli* O121.

83. The Defendant's false and misleading representations to the public concerning its *E. coli*-contaminated products, along with its other breaches as described herein, breached and constituted prohibited practices under the Virginia Consumer Protection Act (VCPA), Virginia Code § 59.1-200.

84. Plaintiff relied on the defendant's false and misleading representations to the public concerning their *E. coli*-contaminated products.

85. The defendant's breaches of the VCPA were willful and caused Plaintiff's injuries.

86. Virginia Code § 59.1-204 permits consumers who are injured by a defendant supplier's willful violation of the Virginia Consumer Protection Act to recover actual damages, as well as attorneys' fees and court costs. Accordingly, Plaintiff seeks damages, as well as costs and attorneys' fees under the VCPA against the Defendant.

87. As a direct and proximate result of Defendant's violations of the VCPA, Plaintiff was caused to suffer serious injuries; has suffered bodily pain and mental anguish; has suffered, and will suffer in the future, pain of body and mind; has incurred, and will continue to incur, medical and related

expenses; and has suffered, and will suffer in the future, other damages.

WHEREFORE, Plaintiff demands judgment against Defendant in the sum of ONE MILLION DOLLARS ($1,000,000.00) for compensatory damages, plus interest from the date of injury and costs; and an award of attorneys' fees as may be permitted by law.

TRIAL BY JURY IS DEMANDED BY PLAINTIFF.

DONNA GARBER

By Counsel

Respectfully submitted,

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