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6 IN THE SUPERIOR COURT OF THE STATE OF WASHINGTON

7 IN AND FOR THE COUNTY OF KING

8 ASHLEY JOHNSON and JARET JOHNSON,  
9 parents of R. J. AND R. J., minor children,

10 Plaintiffs,

11 v.

12 PURE EIRE, LLC, d/b/a Pure Eire Dairy,  
13 a Washington limited liability company,

14 Defendant.

Case No.:

**COMPLAINT**

15 **COMPLAINT**

16 COME NOW the Plaintiffs, Ashley Johnson and Jaret Johnson, individually and as the  
17 parents of R. J. and R. J. (“the Plaintiffs”), by and through their attorney of record, William D.  
18 Marler of Marler Clark, LLP, PS, complaining of the Pure Eire, LLC, d/b/a Pure Eire Dairy (“the  
19 Defendant”), a Washington limited liability company, and allege and state as follows:

20 **PARTIES AND JURISDICTION**

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22 1. At all times relevant to this action, the Plaintiffs resided in King County,  
23 Washington. The Plaintiffs are therefore citizens of the State of Washington.

24 2. At all times relevant to this action, the Defendant is a Washington limited liability  
25 company based in Adams County, Washington. Therefore, the Defendant is a citizen of the state  
26 of Washington.  
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1 distinguish it from other types of *E. coli*. Most serotypes of *E. coli* are harmless and live as normal  
2 flora in the intestines of healthy humans and animals. The *E. coli* bacterium is among the most  
3 extensively studied microorganism. The testing done to distinguish *E. coli* O157:H7 from its other  
4 *E. coli* counterparts is called serotyping. Pulsed-field gel electrophoresis (PFGE), sometimes also  
5 referred to as genetic fingerprinting, is used to compare *E. coli* O157:H7 isolates to determine if  
6 the strains are distinguishable. A technique called multilocus variable-number tandem repeat  
7 analysis (MLVA) is used to determine precise classification when it is difficult to differentiate  
8 between isolates with indistinguishable or very similar PFGE patterns. An even newer technique  
9 called Whole Genome Sequencing (WGS) is now used to determine relatedness between *E. coli*  
10 O157:H7 isolates that is even more conclusive.

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

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25 9. *E. coli* O157:H7's ability to induce injury in humans is a result of its ability to  
26 produce numerous virulence factors, most notably Shiga-like toxins. Shiga toxin (Stx) has multiple  
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1 variants (e.g., Stx1, Stx2, Stx2c), and acts like the plant toxin ricin by inhibiting protein synthesis  
2 in endothelial and other cells. Shiga toxin is one of the most potent toxins known. In addition to  
3 Shiga toxins, *E. coli* O157:H7 produces numerous other putative virulence factors including  
4 proteins, which aid in the attachment and colonization of the bacteria in the intestinal wall, and  
5 which can lyse red blood cells and liberate iron to help support *E. coli* metabolism.  
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7 10. *E. coli* O157:H7 evolved from enteropathogenic *E. coli* serotype O55:H7, a cause  
8 of non-bloody diarrhea, through the sequential acquisition of phage encoded Stx2, a large virulence  
9 plasmid, and additional chromosomal mutations. The rate of genetic mutation of *E. coli* O157:H7  
10 indicates that the common ancestor of current *E. coli* O157:H7 clades likely existed some 20,000  
11 years ago. *E. coli* O157:H7 is a relentlessly evolving organism, constantly mutating and acquiring  
12 new characteristics, including virulence factors that make the emergence of more dangerous  
13 variants a constant threat. The CDC has emphasized the prospect of emerging pathogens as a  
14 significant public health threat for some time.  
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16 11. Although foods of a bovine origin are the most common cause of both outbreaks  
17 and sporadic cases of *E. coli* O157:H7 infections, outbreak of illnesses have been linked to a wide  
18 variety of food items. For example, produce has, since at least 1991, been the source of substantial  
19 numbers of outbreak-related *E. coli* O157:H7 infections. Other unusual vehicles for *E. coli*  
20 O157:H7 outbreaks have included unpasteurized juices, yogurt, dried salami, mayonnaise, raw  
21 milk, game meats, sprouts, and raw cookie dough.  
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23 12. According to a recent study, an estimated 93,094 illnesses are due to domestically  
24 acquired *E. coli* O157:H7 each year in the United States. Estimates of foodborne acquired  
25 O157:H7 cases result in 2,138 hospitalizations and 20 deaths annually. The colitis caused by *E.*  
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1 *coli* O157:H7 is characterized by severe abdominal cramps, diarrhea that typically turns bloody  
2 within twenty-four (24) hours, and sometimes fevers. The incubation period—which is to say the  
3 time from exposure to the onset of symptoms—in outbreaks is usually reported as three (3) to four  
4 (4) days but may be as short as one (1) day or as long as ten (10) days. Infection can occur in  
5 people of all ages but is most common in children. The duration of an uncomplicated illness can  
6 range from one (1) to twelve (12) days. In reported outbreaks, the rate of death is 0-2%, with rates  
7 running as high as 16-35% in outbreaks involving the elderly, like those that have occurred at  
8 nursing homes.

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

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

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4 **Hemolytic Uremic Syndrome (HUS)**

5 15. *E. coli* O157:H7 infections can lead to a severe, life-threatening complication called  
6 hemolytic uremic syndrome (“HUS”). HUS accounts for the majority of the acute and chronic  
7 illness and death caused by the bacteria. HUS occurs in 2-7% of victims, primarily children, with  
8 onset five to ten days after diarrhea begins. It is the most common cause of renal failure in children.  
9 Approximately half of the children who suffer HUS require dialysis, and at least 5% of those who  
10 survive have long-term renal impairment. The same number suffers severe brain damage. While  
11 somewhat rare, serious injury to the pancreas, resulting in death or the development of diabetes,  
12 can also occur. There is no cure or effective treatment for HUS. And, tragically, as too many  
13 parents can attest, children with HUS too often die.

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

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25 17. As already noted, there is no known therapy to halt the progression of HUS. HUS  
26 is a frightening complication that, even in the best American centers, has a notable mortality rate.  
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1 Among survivors, at least five percent will suffer end stage renal disease (ESRD) with the resultant  
2 need for dialysis or transplantation. But “[b]ecause renal failure can progress slowly over decades,  
3 the eventual incidence of ESRD cannot yet be determined.” Other long-term problems include the  
4 risk for hypertension, proteinuria (abnormal amounts of protein in the urine that can portend a  
5 decline in renal function), and reduced kidney filtration rate. Since the longest available follow-  
6 up studies of HUS victims are 25 years, an accurate lifetime prognosis is not really available and  
7 remains controversial. All that can be said for certain is that HUS causes permanent injury,  
8 including loss of kidney function, and it requires a lifetime of close medical monitoring.  
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11 **R. J.’s and R. J.’s *E. coli* O157:H7 Infections**

12 18. Ashley Johnson and Jaret Johnson purchased PCC-brand yogurt from the PCC  
13 Community Market in Issaquah, Washington on April 17 and 22, 2021. Their sons, R. J., three  
14 years old, and R. J., one year old, both consumed the product soon thereafter.  
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16 19. On or about April 25, 2021, three-year-old R. J. began experiencing symptoms  
17 including fever, severe stomach pain, cramping, and diarrhea, which turned bloody.  
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19 20. R. J.’s, symptoms increased in severity quickly, prompting his parents to seek  
20 multiple medical visits until admission Swedish Hospital on April 29, 2021. He was transferred to  
21 Seattle Children’s Hospital on May 1, 2021, where he was diagnosed with Hemolytic Uremic  
22 Syndrome, and discharged on May 5, 2021 after receiving treatment including a transfusion.  
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24 21. Their second child, one-year-old R. J., also began to exhibit symptoms on April 29,  
25 2021. He, too, was admitted to Seattle Children’s Hospital on May 1, 2021, with symptoms  
26 including fever, severe stomach pain, cramping, and diarrhea, which turned bloody.  
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**COUNT II**  
**(Negligence)**

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

31. The 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 the minor Plaintiffs, including the applicable provisions of the Federal Food, Drug and Cosmetic Act, and similar Washington food and public health statutes, including without limitation the provisions of the Washington Product Liability Act, RCW 7.72 et seq., and the Washington State Retail Food Code, chapter 246-215 WAC, all of which prohibit the sale of any food that is adulterated or otherwise injurious to health.

32. The subject product was adulterated within the meaning of the Federal Food, Drug and Cosmetic Act, and similar Washington statutes, because it contained a deleterious substance that rendered it injurious to health, i.e., *E. coli* O157:H7 bacteria.

33. The Defendant violated federal, state, and local food safety regulations by its sale of adulterated food. These federal, state, and local food safety regulations are applicable here, and establish a positive and definite standard of care in the sale of food. The violation of these regulations constitutes negligence as a matter of law.

34. The minor Plaintiffs are in the class of persons intended to be protected by these statutes and regulations, and the minor Plaintiffs were injured as the direct and proximate result of the Defendant's violation of applicable federal, state, and local food safety regulations.





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

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

7 (3) That the Court award the Plaintiffs their costs, disbursements, and reasonable  
8 attorneys' fees incurred;

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

13 (5) That the Court award such other and further relief as it deems necessary and proper  
14 in the circumstances.  
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17 Dated this 17<sup>th</sup> day of May, 2021

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20 MARLER CLARK LLP, PS

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22 \_\_\_\_\_  
23 William D. Marler WSBA #17233  
24 Attorney for Plaintiffs