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6 IN THE SUPERIOR COURT OF THE STATE OF WASHINGTON

7 IN AND FOR THE COUNTY OF KING

8 JENNIFER ABRAMSKI and JUPITER
9 BARTON, parents of M.B., a minor child

Case No.:

10 Plaintiffs

COMPLAINT

11 vs.

12 PURE EIRE, LLC, d/b/a Pure Eire Dairy,
13 a Washington Limited Liability company

14 Defendants

15 **COMPLAINT**

16 COME NOW the Plaintiffs, Jennifer Abramski and Jupiter Barton, individually and as
17 the parents of M.B. (“the Plaintiffs”), by and through their attorney of record, William D. Marler
18 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**

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

23 2. At all times relevant to this action, the Defendant is a Washington limited liability
24 company residing in Adams County, Washington. Therefore, the defendant is a citizen of the state
25 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 9. *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 10. *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.
6

7 11. *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 12. 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 13. 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.

11 14. 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.

21 15. 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.

3
4 **Hemolytic Uremic Syndrome (HUS)**

5 16. *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 17. 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 18. 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.

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. Other long-term problems include
6 the risk for hypertension, proteinuria (abnormal amounts of protein in the urine that can portend a
7 decline in renal function), and reduced kidney filtration rate. Since the longest available follow-
8 up studies of HUS victims are 25 years, an accurate lifetime prognosis is not really available and
9 remains controversial. All that can be said for certain is that HUS causes permanent injury,
10 including loss of kidney function, and it requires a lifetime of close medical monitoring.
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13 **M.B.’s *E. coli* O157:H7 Infection**

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15 19. Jennifer Abramski and Jupiter Barton purchased yogurt from the PCC-brand yogurt
16 from the PCC Community Market in View Ridge, Washington, on multiple occasions from April
17 15 through April 20th. Their daughter, M.B., consumed the product multiple times throughout that
18 period.

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20 20. On or about April 21, 2021, M.B. began experiencing symptoms including fever,
21 headaches, and diarrhea, which turned bloody.

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23 21. M.B.’s symptoms began to increase in severity quickly, prompting her parents to
24 take her to Seattle Children’s hospital on May 3, 2020. Shortly after her arrival M.B. was admitted
25 for intensive treatment.
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1 31. The Defendant is strictly liable to the Plaintiffs for the harm proximately caused by
2 their distribution and sale of an unsafe and defective food product.
3

4 **COUNT II**
5 **(Negligence)**

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

8 33. The Defendant had a duty to comply with all statutory and regulatory provisions
9 that pertained or applied to the manufactured, distribution, storage, labeling, and sale of the food
10 products that injured the minor Plaintiff, including the applicable provisions of the Federal Food,
11 Drug and Cosmetic Act, and similar Washington food and public health statutes, including without
12 limitation the provisions of the Washington Product Liability Act, RCW 7.72 et seq., and the
13 Washington State Retail Food Code, chapter 246-215 WAC, all of which prohibit the sale of any
14 food that is adulterated or otherwise injurious to health.
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16 34. The subject product was adulterated within the meaning of the Federal Food, Drug
17 and Cosmetic Act, and similar Washington statutes, because it contained a deleterious substance
18 that rendered it injurious to health, i.e., *E. coli* O157:H7 bacteria.
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20 35. The Defendant violated federal, state, and local food safety regulations by their sale
21 of adulterated food. These federal, state, and local food safety regulations are applicable here, and
22 establish a positive and definite standard of care in the sale of food. The violation of these
23 regulations constitutes negligence as a matter of law.
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25 36. The minor Plaintiff is in the class of persons intended to be protected by these
26 statutes and regulations, and the minor Plaintiff was injured as the direct and proximate result of
27 the Defendant's violation of applicable federal, state, and local food safety regulations.
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1 incurred, or to be incurred, by the Plaintiffs as the direct and proximate result of the acts
2 and omissions of the Defendants;

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

6 (4) That the Court award the Plaintiffs the opportunity to amend or modify the
7 provisions of this Complaint as necessary or appropriate after additional or further discovery is
8 completed in this matter, and after all appropriate parties have been served; and
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10 (5) That the Court award such other and further relief as it deems necessary and proper
11 in the circumstances.

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13 Dated this 17th day of May, 2021

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15 MARLER CLARK LLP, PS

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18 _____
19 William D. Marler WSBA #17233
20 Attorney for Plaintiffs