UNITED STATES DEPARTMENT OF AGRICULTURE
FOOD SAFETY AND INSPECTION SERVICE

Petition for an Interpretive Rule Declaring all enterohemorrhagic Shiga Toxin-producing Serotypes of *Escherichia coli* (*E. coli*), Including Non-O157 Serotypes, to be Adulterants Within the Meaning of 21 U.S.C. § 601(m)(1)	)	Docket No. _____

CITIZEN PETITION

Submitted by:

Marler Clark LLP, PS

Outbreak, Inc.

The Family of June Dunning

Megan Richards

Shiloh Johnson

October 5, 2009

FSIS Docket Clerk
Department of Agriculture
Food Safety and Inspection Service
Room 2534 South Building
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I. REQUESTED ACTIONS

A. Issuance of an Interpretive Rule

Pursuant to 5 U.S.C. § 553(e), 9 C.F.R. § 392, and 7 C.F.R. § 1.28, we submit this petition requesting the administrator of the Food Safety and Inspection Service (FSIS) to issue an interpretive rule declaring all enterohemorrhagic (EHEC) Shiga toxin-producing serotypes of *Escherichia coli* (*E. coli*), including non-O157 serotypes, to be adulterants within the meaning of the Federal Meat Inspection Act (FMIA).[1] The relevant FMIA provision, 21 U.S.C. § 601(m)(1), states in pertinent part that a carcass, part thereof, meat, or meat food product is adulterated "if it bears or contains any poisonous or deleterious substance which may render it injurious to health." FSIS interpreted this provision in 1994 to declare *E. coli* O157:H7 as an adulterant. It is respectfully submitted, however, that the 1994 interpretive rule, and its subsequent application and enforcement, ignores the grave dangers that current scientific and medical research demonstrates are not limited to *E. coli* O157:H7, but instead extend to all Shiga toxin-producing *E. coli* (STEC). As a result of the narrow scope of the 1994 interpretive rule, the safety of American consumers is at risk. Issuing a new interpretive rule that declares that all STEC are

[1] For ease of reference and to avoid an implicit redundancy, EHEC Shiga toxin-producing serotypes of *E. coli*, which are by definition pathogenic, will be referred to as non-O157 STEC or STEC.

adulterants within the meaning of the FMIA will encourage increased monitoring efforts and better ensure the safety of the general public, as is required by the FMIA.[2]

B. A Grant of Expedited Review

Because this petition requests action intended to enhance the public health by reducing food safety hazards, the petitioners ask for expedited review. As stated in the recently amended FSIS petition procedures, 9 CFR § 392.8(a):

> A petition will receive expedited review by FSIS if the requested action is intended to enhance the public health by removing or reducing foodborne pathogens or other potential food safety hazards that might be present in or on meat, poultry, or egg products.

This petition requests an interpretive rule that will prompt better monitoring of all enterohemorrhagic *E. coli*, thus decreasing foodborne contamination. In accordance with 9 CFR § 392.8(b), the requested action is supported by scientific information that demonstrates that such an interpretive rule will reduce foodborne pathogens that are likely to be present in meat products. For these reasons, the petitioners request FSIS to grant this petition expedited review.

II. ABOUT THE PETITIONERS

Marler Clark LLP, PS, located in Seattle, Washington, is the nation's foremost law firm representing victims of foodborne illness. The Marler Clark attorneys spend the majority of their time working on food-related cases, representing victims of *Campylobacter*, *E. coli* O157:H7, non-O157 STEC, Hepatitis A, *Listeria*, *Norovirus*, *Salmonella*, and *Shigella* outbreaks across the country.

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[2] As stated in the FMIA, "It is essential in the public interest that the health and welfare of consumers be protected by assuring that meat and meat food products distributed to them are wholesome, not adulterated, and properly marked, labeled, and packaged." 21 U.S.C. § 602 (2004).

Outbreak, Inc. was formed in 1998 by Marler Clark's founding partners as the nonprofit consulting arm of the firm. Each lawyer travels several days a month on behalf of Outbreak, Inc., giving speeches to food-industry groups and health agencies, focusing on preventing foodborne illness.

June Dunning, represented here by her family, was a Hagerstown, Maryland woman whose *E. coli* O146:H21 infection led to her unfortunate and untimely death in 2006.

Megan Richards is a Millville, Utah woman who, due to an *E. coli* O121:H19 infection in 2006, suffered a protracted illness punctuated by a lengthy hospitalization with severe complications due to hemolytic uremic syndrome.

Shiloh Johnson is a young girl from Pryor, Oklahoma who developed hemolytic uremic syndrome after becoming infected with *E. coli* O111 in 2008. She endured a lengthy hospitalization and required numerous dialysis treatments.

III. SOME BACKGROUND

Although first isolated in 1975, and subsequently associated with foodborne illness in 1982, FSIS did not interpret *E. coli* O157:H7 to be an adulterant under the FMIA until 1994. The classification of *E. coli* O157:H7 as an adulterant came in the wake of a 1993 large-scale foodborne outbreak that left over six-hundred persons ill and four children dead.[3] In a FSIS policy statement, dated January 19, 1999, the agency emphasized the continuing risk of *E. coli* contamination:

> Exposure to *E. coli* O157:H7 has been linked with serious, life-threatening human illnesses (hemorrhagic colitis and hemolytic uremic syndrome). Raw ground beef

[3] This outbreak is commonly referred to as the "Jack in the Box outbreak." *See* Company News; Jack in the Box's Worst Nightmare, N.Y. Times, Feb. 6, 1993, *available at* http://query.nytimes.com/gst/fullpage.html?res=9F0CE7DB153CF935A35751C0A965958260&sec=&spon=. At the time, the outbreak, originating from tainted hamburger patties, was the largest *E. coli* O157:H7 outbreak to date.

> products present a significant public health risk because they are frequently consumed after preparation (*e.g.*, cooking hamburger to a rare or medium rare state) that does not destroy *E. coli* O157:H7 organisms that have been introduced below the product's surface by chopping or grinding (*e.g.*, ground beef, veal patties, and beef pattie mix).
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> The public health risk presented by beef products contaminated with *E. coli* O157:H7 is not limited, however, to raw ground beef products. Given the low infectious dose of *E. coli* O157:H7 associated with foodborne disease outbreaks and the very severe consequences of an *E. coli* O157:H7 infection, the Agency believes that the status under the FMIA of beef products contaminated with *E. coli* O157:H7 must depend on whether there is adequate assurance that subsequent handling of the product will result in food that is not contaminated when consumed.[4]

Despite strong scientific evidence that many strains of non-O157 STEC are as pathogenic as *E. coli* O157:H7, FSIS has thus far failed to include all STEC as adulterants under the FMIA. Recent studies have repeatedly shown that non-O157 STEC is a serious food safety hazard. According to one study, non-O157 STEC are prevalent in beef production systems at rates as high as 70.1%.[5] A United States Department of Agriculture (USDA) study states that non-O157 STEC have been found in ground beef and on cattle hides and feces at levels comparable to *E. coli* O157:H7.[6] Furthermore, European studies indicate that non-O157 STEC infections occur more frequently than *E. coli* O157:H7 infections.[7] With such a ubiquitous presence, the potential risk for harm caused by non-O157 STEC may be on par with, or even greater than, the risk created by *E. coli* O157:H7. Indeed, another study concluded that "non-O157 STEC can cause severe illness that is comparable to the illness caused by STEC O157."[8]

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[4] Federal Register. January 19, 1999. [Docket No. 97-068N].

[5] Hussein, H. S. 2006. Prevalence and pathogenicity of shiga toxin-producing *Escherichia coli* in beef cattle and their products. J Anim Sci. 85:E65.

[6] Eblen, Denise. Public Health Importance of Non-O157 Shiga Toxin-Producing *Escherichia coli* (non-O157 STEC) in the US Food Supply. 2007. FSIS.

[7] Bareta, J. K. Edge, S. Lathrop. 2009. Shiga Toxin-producing *Escherichia coli*, New Mexico, USA, 2004-2007. 15 Emerging Infect Dis. (No. 8) (Aug. 2009).

[8] Brooks, J. T., E. G. Sowers, J. G. Wells, K. D. Greene, P. M. Griffin, R. M. Hoekstra, and N. A.

On October 17, 2007, FSIS, along with the Food and Drug Administration's Center for Food Safety and Applied Nutrition (FDA CFSAN) and the National Centers for Disease Control and Prevention (CDC), co-sponsored a public meeting to consider the public health significance of non-O157 STEC. In the Notice of the meeting, published on October 9, 2007, FSIS stated:

> In the United States, there is growing awareness that STECs other than *E. coli* O157:H7 (non-O157:H7 STECs) cause sporadic and outbreak-associated illnesses. This awareness is attributable in part to the increasing availability of laboratory reagents that can be used to diagnose illnesses and to detect strains of STECs in food and other environmental samples. The number of non-O157:H7 STEC infections reported to the CDC from 2000 to 2005 increased from 171 to 501 cases, suggesting a higher burden of illness than previously thought.
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> Outbreaks associated with non-O157:H7 STECs have been reported worldwide, including thirteen in the United States from 1990 to 2006. The 2006 data is still preliminary. Many outbreaks were attributed to consumption of fresh produce; none were attributed to ground beef consumption. However, in 2006, non-O157:H7 STEC illness was diagnosed in a patient in New York who had consumed ground beef shortly before illness onset. The same STEC strain, indistinguishable by pulsed field gel electrophoresis, was detected in the patient's stool and in leftover ground beef that the patient had consumed. In this case, FSIS was unable to take further action because the product could not be definitively traced to a production lot.[9]

The interpretive rule proposed in this Petition is consistent with FSIS' current policies and objectives. As stated in the FSIS 2008-2013 Strategic Plan, one of FSIS' current primary goals is to "enhance the development of science and risk-based policies and systems."[10] To that end, FSIS has created an objective seeking "reduced *E. coli* O157:H7 and *other Shiga toxin-producing E. coli (STEC)* consistent with Healthy People 2010 and Healthy People 2020 goals through development and implementation of policy."[11] The goal of this petition, and the

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Strockbine. 2005. Non-O157 shiga toxin-producing *Escherichia coli* infections in the United States, 1983-2002. J Infect Dis. 192:1422-9.

[9] Federal Register. October 9, 2007. [Docket No. FSIS-2007-0041].

[10] FSIS. 2008. 2008-2013 Strategic Plan. 27.

[11] *Id.* (emphasis added).

interpretive rule it proposes, is to accomplish precisely what the FSIS Strategic Plan objective seeks: reduced *E. coli* O157:H7 and other STEC through better monitoring and prevention standards, which will be precipitated by the declaration that all STEC are adulterants.

What follows is divided into three sections. The first states the grounds—both legal and scientific—for issuing the proposed interpretive rule. The second describes the stories of three victims affected by non-O157 STEC. The third section concludes with a request for action to resolve the clear danger that *both E. coli* O157:H7 and non-O157 STEC represent to the United States food supply.

IV. STATEMENT OF GROUNDS

A. Pathogenesis of Shiga Toxin-producing *E. coli*

The virulence of *E. coli* is a result of the ability of certain strains to produce Shiga-like toxins.[12] It has been theorized that generic *E. coli* picked up this deadly ability through horizontal transfer of virulence genes from the *Shigella* bacteria.[13] STEC strains are known to cause diarrhea and hemolytic uremic syndrome (HUS).[14] The most common STEC that causes illness in the United States is *E. coli* O157:H7. As the CDC notes, however, non-O157 STEC strains are emerging pathogens that pose a significant health threat, with more strains reported every year.[15] Non-O157 STEC have caused multiple outbreaks in the United States. Furthermore, as documented by several studies, non-O157 STEC have been isolated from

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[12] 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, 61-62 (1991) (noting that the nomenclature came about because of the resemblance to toxins produced by *Shigella dysenteriae*).

[13] *Id.* at 62 (using the more technical term "phage-mediated transfer").

[14] CDC. 2005. Bacterial Foodborne and Diarrheal Disease National Case Surveillance Annual Report, 2005. 16.

[15] *Id.*

diarrheal stools as frequently as *E. coli* O157:H7.[16]

After a susceptible individual ingests a sufficient quantity of *E. coli*, the bacteria attach to the inside surface of the large intestine and initiate an inflammatory reaction. The result is bloody diarrhea and intense abdominal cramps, both symptoms of severe infectious gastroenteritis.

HUS accounts for the majority of the chronic illness and death caused by *E. coli* bacteria.[17] It is the most common cause of renal failure in children.[18] Approximately half of the children who suffer HUS require dialysis, and at least 5% of those who survive have long term renal impairment.[19] The same number suffers severe brain damage.[20] While somewhat rare, serious injury to the pancreas, resulting in death or the development of diabetes, can also occur.[21] There is no cure or effective treatment for HUS.[22] And, tragically, as too many parents can attest, children with HUS often die.[23]

HUS develops when the Shiga toxins from the bacteria enter the body's circulation

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[16] *Id.*

[17] Richard L. Siegler, MD, The Hemolytic Uremic Syndrome, 42 Ped. Nephrology, 1505 (Dec. 1995). ("[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.

[18] Chinyu Su, MD & Lawrence J. Brandt, MD, *Escherichia coli* O157:H7 Infection in Humans, 123 Annals Intern. Med. (Issue 9), 698-707.

[19] 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) (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).

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

[21] 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").

[22] Safdar, *supra* note 19, at 996; *see also* Siegler, *supra* note 20, 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.")

[23] Su & Brandt, *supra* note 18 ("the mortality rate is 5-10%"). *See also Kriefall v. Excel*, 265 Wis.2d 476, 483, 665 N.W.2d 417 (2003). ("three-year old Brianna Kriefall died from food that everyone party to this appeal...recognize was cross-contaminated by *E. coli* O157:H7 bacteria from meat sold by Excel.")

through the inflamed bowel wall.[24] Shiga toxins, and most likely other chemical mediators, attach to receptors on the inside surface of blood vessel cells (endothelial cells) and initiate a cascading chemical reaction that results in the formation of tiny thrombi (blood clots) within these vessels.[25] Some organs seem more susceptible, perhaps due to the presence of increased numbers of receptors; these include the kidneys, pancreas, and brain.[26] By definition, when fully expressed, HUS presents with a triad of conditions or diagnoses: hemolytic anemia (destruction of red blood cells), thrombocytopenia (low platelet count), and acute renal failure (loss of the filter function of the kidney).[27]

As already noted, there is no known therapy to halt the progression of infectious gastroenteritis to HUS. HUS is a frightening complication that even in the best American medical centers has a notable mortality rate.[28] Among survivors, at least five percent will suffer end stage renal disease ("ESRD") with the resultant need for dialysis or transplantation.[29] But, "[b]ecause renal failure can progress slowly over decades, the eventual incidence of ESRD cannot yet be determined."[30] 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.[31] Because the longest available follow-up studies of HUS victims cover only 25 years, an accurate lifetime prognosis is not available and remains

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[24] 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).

[25] *Id.* Siegler, *supra* note 20, at 1509-11 (describing what Dr. Siegler refers to as the "pathogenic cascade" that results in the progression from colitis to HUS).

[26] Garg, *supra* note 24, at 1360; *see also* Su & Brandt, *supra* note 18, at 700.

[27] Garg, *supra* note 24, at 1360; Su & Brandt, *supra* note 18, at 700.

[28] Siegler, *supra* note 20, 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.")

[29] Garg, *supra* note 24, at 1366-67.

[30] Siegler, *supra* note 20, at 1519.

[31] *Id.* at 1519-20; Garg, *supra* note 24, at 1366-67.

controversial.[32] 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.

B. Legal Basis for Declaring All STEC Adulterants Under the FMIA

The FMIA does not require the USDA to engage in substantive rulemaking as a predicate to interpreting the Act to deem a particular substance an adulterant.[33] Pursuant to the Administrative Procedures Act (APA), 5 U.S.C. § 553(b)(3)(A), agencies may issue "interpretive rules, general statements of policy, or rules of agency organization, procedure, or practice" without the notice and comment procedures required for proposed rule making. In 1994, several supermarket and meat industry organizations sought an injunction against the USDA, attempting to prevent the agency from declaring *E. coli* O157:H7 an adulterant, and barring it from implementing an *E. coli* sampling program.[34] Addressing the petitioners' claims, the court was careful to distinguish interpretive rules from substantive rules by stating that interpretive rules do not create new law, instead they are "statements as to what the administrative officer thinks the regulation means."[35]

To determine whether the 1994 declaration of *E. coli* O157:H7 as an adulterant was an interpretive rule, the *Espy* court relied on criteria established in *American Mining Congress v. Mine Safety & Health Administration*[36], which stated:

> Accordingly, insofar as our cases can be reconciled at all, we think it almost exclusively on the bases of whether the purported interpretive rule has "legal effect," which in turn is best ascertained by asking (1) whether in the absence of the rule there would not be an adequate legislative basis for enforcement action or

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[32] Garg, *supra* note 24, at 1368.
[33] *Texas Food Industry Ass'n, et al. v. Espy* 870 F. Supp. 143, 147 (1994).
[34] *See Id.*
[35] *Id.* at 147.
[36] *American Mining Congress v. Mine Safety & Health Administration* 302 U.S. App. D.C. 38, 995 F.2d 1106 (D.C. Cir. 1993).

> other agency action to confer benefits or ensure the performance of duties, (2) whether the agency has published the rule in the Code of Federal Regulations, (3) whether the agency has explicitly invoked its general legislative authority, or (4) whether the rule effectively amends a prior legislative rule. If the answer to any of these questions is affirmative, we have a [substantive], not an interpretive rule.[37]

Applying these criteria, the court held that the declaration of *E. coli* O157:H7 as an adulterant was within the USDA's interpretive rulemaking powers, and thus did not require notice and comment procedures.

The legal process to issue an interpretive rule declaring all STEC to be adulterants under the FMIA is identical to the process utilized by the USDA in the 1994 *E. coli* O157:H7 declaration. As with the rule upheld in *Espy*, the interpretive rule proposed in this Petition fits well within the *American Mining Congress* criteria. First, as reaffirmed in *Espy*, because the FMIA does not require the USDA to engage in substantive rulemaking to determine whether a particular substance is an adulterant, the agency has "the discretion to proceed through case-by-case adjudication and interpretive orders, rather than through the rulemaking process."[38] Second, the request in this Petition does not require FSIS to publish the rule in the Code of Federal Regulations, or invoke its general legislative authority. Finally, the proposed interpretive rule does not amend a prior legislative rule. Thus, all of the *American Mining Congress* criteria are sufficiently met.

Other legal concerns raised by opponents in *Espy*, namely that the requested action would be arbitrary and capricious, and that the FMIA does not grant the USDA authority to declare non-O157 STEC adulterants, would also be unfounded. First, as stated in *Espy*, the USDA may properly declare substances to be adulterants with the intended purpose of spurring industry to

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[37] *Id.* at 1112.
[38] *Texas Food Industry Ass'n, et al. v. Espy* 870 F. Supp. 143, 147 (1994).

create and implement preventative measures.[39] Similarly, the purpose here is to encourage the meat industry to engage in more effective oversight measures in order to prevent STEC outbreaks. Second, despite a court ruling over thirty years ago that *Salmonella* is not an adulterant *per se*[40] (as was conceded at the time by FSIS), certain Shiga toxin-producing *E. coli* strains are properly declared to be adulterants on account of the unique health risk they present. This is due to the fact that, as stated in FSIS policy documents,[41] low infectious doses of such *E. coli* often cause severe health consequences. Furthermore, products contaminated with such *E. coli* are often consumed after preparation that does not fully destroy the pathogens. Indeed, as is the case with *E. coli* O157:H7, "proper" cooking of meat will not necessarily protect consumers from infection from all STECs. As stated in *Espy*:

> [U]nlike other pathogens, it is not "proper" cooking but "thorough" cooking that is necessary to protect consumers from *E. coli.* The evidence submitted by Defendants indicates that many Americans consider ground beef to be properly cooked rare, medium rare, or medium. The evidence also indicated that *E. coli* contaminated ground beef cooked in such a manner may cause serious physical problems, including death. Therefore, *E. coli* is a substance that renders "injurious to health" what many Americans believe to be properly cooked ground beef. Based on this evidence, the Court finds that *E. coli* fits the definition of an adulterant under the FMIA.[42]

In sum, as established by both the USDA and prior judicial decisions, the interpretive rule proposed in this Petition has clear legal precedent and does not violate APA procedures.

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[39] *Id.* at 148.

[40] A ruling that, given the wealth of scientific data detailing the prevalence and toxicity of *Salmonella* (especially of the antibiotic resistant variety), is now controversial, to say the least.

[41] Federal Register. January 19, 1999. [Docket No. 97-068N].

[42] *Texas Food Industry Ass'n, et al. v. Espy* 870 F. Supp. 143, 149 (1994).

C. Scientific Basis for the Regulation of Shiga Toxin-Producing *E. coli*

1. Prevalence of Shiga Toxin Producing *E. coli*

Non-O157 STEC are the causative agents of zoonotic emerging infectious diseases, often of bovine origin. Below is a general review of non-O157 STEC prevalence studies in humans, cattle, and beef products.

a. Humans

Non-O157 STEC infections are under-recognized and under-reported due to inadequate epidemiological and laboratory surveillance. In the United States, *E. coli* O157:H7 became nationally notifiable in 1994, whereas non-O157 STEC infections were not reportable until 2000, following adoption of a position statement (2000 ID#1) by the Council for State and Territorial Epidemiologists (CSTE). At that time, the CSTE recognized that the threat to public health from STEC infections extended beyond just the *E. coli* O157:H7 serogroup.

In recent years, improved diagnostic assays for non-O157 STEC have contributed to an increased appreciation of the severity of disease caused by these strains including hemolytic uremic syndrome (HUS). Notably, the number of non-O157 STEC cases reported to CDC's FoodNet has risen steadily each year; from 2000-2006, there was an overall four-fold increase in incidence (0.12 cases per 100,000 to 0.42 cases per 100,000 population) at FoodNet sites. The most common serogroups reported to cause foodborne illness in the United States are O26, O111, O103, O121, O45, and O145.[43]

Johnson *et al* evaluated the emerging clinical importance of non-O157 STEC and concluded that these strains may account for up to 20 to 50% of all STEC infections in the

[43] Brooks, J. T., E. G. Sowers, J. G. Wells, K. D. Greene, P. M. Griffin, R. M. Hoekstra, and N. A. Strockbine. 2005. Non-O157 shiga toxin-producing *Escherichia coli* infections in the United States, 1983-2002. J Infect Dis. 192:1422-9.

United States.[44] Clearly, the prevalence of non-O157 STEC infections is placing an enormous burden on society and the health care system in the United States.

b. Cattle as Reservoirs

Beef and dairy cattle are known reservoirs of *E. coli* O157:H7 and non-O157 STEC strains.[45] In reviews of STEC occurrence in cattle worldwide, the prevalence of non-O157 STECs ranged from 4.6 to 55.9% in feedlot cattle, 4.7 to 44.8% in grazing cattle, and 0.4 to 74% in dairy cattle feces. The prevalence in beef cattle going to slaughter ranged from 2.1 to 70.1%.[46] While most dairy cattle-associated foodborne disease outbreaks are linked to milk products, dairy cattle still represent a potential source of contamination of beef products when they are sent to slaughter at the end of their useful production life (termed "cull" or "spent" dairy cows); this "dairy beef" is often ground and sold as hamburger.

The high prevalence of non-O157 STEC in some cattle populations, combined with the lack of effective on-farm control strategies to reduce carriage, represents a significant risk of contamination of the food supply and the environment.

c. Beef Products

Numerous non-O157 STEC serotypes known to cause human illness are from bovine origin, thus putting the beef supply at-risk. Both *E. coli* O157:H7 and non-O157 STEC may colonize the gastrointestinal tract of cattle, and potentially contaminate beef carcasses during processing. Although not as well studied, the risk factors for contamination of beef products

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[44] Johnson, K. E., C. M. Thorpe, and C. L. Sears. 2006. The emerging clinical importance of non-O157 shiga toxin-producing *Escherichia coli*. Clin Infect Dis. 43:1587-95.

[45] Hussein, H. S. 2006. Prevalence and pathogenicity of shiga toxin-producing *Escherichia coli* in beef cattle and their products. J Anim Sci. 85:E63-72; Hussein, H. S. and T. Sakuma. 2005. Prevalence of shiga toxin-producing *Escherichia coli* in dairy cattle and their products. J Dairy Sci. 88:450-65.

[46] *Id.* at 465.

from cattle colonized with non-O157 STECs are likely the same or very similar to *E. coli* O157:H7. For example, cattle hides contaminated with *E. coli* O157:H7 during slaughter and processing are a known risk factor for subsequent *E. coli* O157:H7 contamination of beef products. One study showed that the prevalence of non-O157 STEC (56.6%) on hides is nearly as high as that found for *E. coli* O157:H7 (60.6%).[47]

Hussein and Bollinger evaluated published reports from over three decades and found that non-O157 STEC were more prevalent in beef products compared with *E. coli* O157. In their study, the prevalence of non-O157 STEC ranged from 1.7 to 58% in packing plants, from 3 to 62.5% in supermarkets, and an average of 3% in fast food restaurants. In a recent survey of retail ground beef products in the United States, 23 (1.9%) of 1,216 samples were contaminated with non-O157 STEC.[48] In another study, researchers found a 10 to 30% prevalence of non-O157 STEC in imported and domestic boneless beef trim used for ground beef.[49]

2. Non-*E. coli* O157:H7 Outbreaks

Worldwide, non-O157 STEC outbreaks emerged in the 1980s, and the first reported outbreaks in the United States occurred in the 1990s.[50] Although the number of reported outbreaks due to non-O157 STECs remains relatively low in the United States, most experts

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[47] Barkocy-Gallagher, G. A., T. M. Arthur, M. Rivera-Betancourt, X. Nou, S. D. Shackelford, T. L. Wheeler, and M. Koohmaraie. 2003. Seasonal prevalence of Shiga toxin-producing *Escherichia coli*, including O157:H7 and non-O157:H7 serotypes, and Salmonella in commercial beef processing plants. J Food Prot. 66:1978-86.

[48] Samadpour, M., V. Beskhlebnaya, and W. Marler. 2009. Prevalence of non-O157 enterohaemmorrhagic *Escherichia coli* in retail ground beef in the United States. 7th International Symposium on Shiga Toxin (Verocytoxin)-producing Escherichia coli Infections. Buenos Aires, Argentina.

[49] Bosilevac J. M., M. N. Guerini, D. M. Brichta-Harhay, T. M. Arthur, and M. Koohmaraie. 2007. Microbiological characterization of imported and domestic boneless beef trim used for ground beef. J Food Prot. 70:440-9.

[50] Hussein, H. S. 2006. Prevalence and pathogenicity of shiga toxin-producing *Escherichia coli* in beef cattle and their products. J Anim Sci. 85:E63-72; Brooks, J. T., E. G. Sowers, J. G. Wells, K. D. Greene, P. M. Griffin, R. M. Hoekstra, and N. A. Strockbine. 2005. Non-O157 shiga toxin-producing *Escherichia coli* infections in the United States, 1983-2002. J Infect Dis. 192:1422-9.

agree that documented outbreaks only represent the "tip of the iceberg." From 1990 to 2007, twenty-two non-O157 STEC outbreaks were reported in the United States.[51] This number, however, pales in comparison to the estimated 36,700 illnesses, 1,100 hospitalizations, and 30 deaths the CDC annually attributes to non-O157 STEC.[52] If the past is any indication, the number of reported outbreaks will only increase as more laboratories test for non-O157 STEC.

3. Products Implicated in Previous Outbreaks

There is some lack of information identifying specific vehicles of transmission for human non-O157 STEC infections, nonetheless, contaminated raw dairy products, produce, and water have been implicated in the United States.[53] A review of non-O157 STEC in Connecticut showed that exposures, including ground beef, were similar in both non-O157 STEC and *E. coli* O157:H7 cases, suggesting that the routes of transmission are similar.[54] Considering the relatively high prevalence of both *E. coli* O157:H7 and non-O157 STEC in cattle populations, it is not surprising that ground beef and other beef products could be a common food vehicle.

Outbreaks of non-O157 STEC infection and illness attributed to ground beef and its sausage products have been documented outside the United States including Argentina, Australia, Germany, and Italy. These beef-related outbreaks involved eight STEC serogroups (O1, O2, O15, O25, O75, O86, O111, and O160). HUS cases were reported in five of the six outbreaks, predictably most often striking children and the elderly.

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[51] Gould, L. Hannah. September 14, 2009. Update on the Epidemiology of Shiga toxin-producing *E. coli* in the United States. Capital Area Food Protection Association Meeting.

[52] *Id.*

[53] Brooks, J. T., E. G. Sowers, J. G. Wells, K. D. Greene, P. M. Griffin, R. M. Hoekstra, and N. A. Strockbine. 2005. Non-O157 shiga toxin-producing *Escherichia coli* infections in the United States, 1983-2002. J Infect Dis. 192:1422-9.

[54] CDC. 2007. Laboratory-confirmed non-O157 shiga toxin-producing *Escherichia coli* – Connecticut, 2000-2005. MMWR. 56:29-31.

More rigorous investigation into the cause of non-O157 STEC outbreaks is needed to better understand the role of beef products and other foods in the contamination of the human food supply with these strains. Bettelheim described non-O157 STECs as "under-rated pathogens."[55] Indeed, the surveillance trends suggest that if left unchecked, it is only a matter of time before the United States experiences large non-O157-related outbreaks. Amending FMIA regulations to include pathogenic non-O157 STEC strains under the definition of "adulterated" is an urgently needed step in the prevention and control of these potentially deadly pathogens.

V. THE SUFFERING CAUSED BY NON-O157 *E. COLI* INFECTIONS

What follows are just a few of the personal stories associated with non-O157 STEC outbreaks. These stories are presented on behalf of the Petitioners to give a small insight into the significant harm that results when the STEC already present in the national food supply causes illness.

A. June Dunning, *E. coli* O146:H21, 2006, Death

Right up until the time of her death, June Dunning remained an active, self-aware, and outgoing woman. Her health had always been good. For the last seven years of her life, she lived in Hagerstown, Maryland with her daughter and son-in-law. On August 28, 2006, June consumed a small amount of Dole baby spinach from a bag her daughter had purchased at the local grocery store seven days earlier. The bag later tested positive for *E. coli* O146:H21.

June fell ill on September 2, 2006. Her illness quickly progressed and she was taken to the hospital the following day. She was first seen by a triage nurse, who noted that June had experienced a sudden onset of diarrhea the night before, which had progressed to bloody stools

[55] Bettelheim, K. A. 2007. The non-O157 shiga-toxigenic (verocytotoxigenic) *Escherichia coli*: under-rated pathogens. Crit Rev Microbiol. 33:67-97.

and severe abdominal pain in the morning. June rated her pain at "9" on a 10 point scale. Further examination and blood tests revealed a number of disturbing problems. A CT scan showed diffuse thickening and swelling of the colon, with severe, acute inflammatory colitis of the ascending and transverse colon. Her blood pressure was elevated and she was beginning to show signs of renal insufficiency. Concerned about her worsening condition, her physician admitted her to the hospital and started her on intravenous fluids.

Admission to the hospital did not slow the deterioration of June's condition. She began to lose her mental faculties. She spoke, but her words did not make sense. She often spoke of going to see her husband, who has passed away ten years prior. All the while, she continued to suffer from frequent, painful bloody diarrhea. Her renal failure worsened. Her doctors were concerned that the colitis would soon lead to systemic toxemia, and thus determined that she needed surgical removal of a portion of her colon.

June survived the surgery, but her overall health continued to deteriorate. She became anemic and was placed in the intensive care unit. She soon stopped producing urine, and progressed to a coma-like state. In the early morning hours of September 7, she suffered a grand mal seizure. On September 9, she suffered another seizure, followed by a drop in her oxygen levels. In reaction to her failing bodily functions, she was placed on mechanical ventilation. By this point in the hospitalization, her medical bills totaled nearly $50,000.

From this point forward, it was painfully clear what the unfortunate outcome of June's condition would be. An EEG on September 11 showed slowing brain activity. Her daughter and son-in-law stayed with her for the final hours. Late in the evening on September 11, the ventilator and all medical support except for morphine were disconnected. The doctors said they

expected June to pass within the hour. Instead, she persevered without life-support. For the majority of the next 36 hours, she appeared to be resting comfortably. In one frightening episode during the early hours of September 12, however, she experienced one final seizure. She gripped her daughter's hand, eyes wide open, moaning and sighing. Thankfully, the seizure passed. June clung to life until just after dawn on September 13, passing away at 6:45 AM.

B. Megan Richards, *E. coli* O121:H19, 2006

In 2006, Megan Richards, of Millville, Utah, was a young wife, mother, and educational conference coordinator with a bright future. On June 30 of that year, she consumed a seemingly safe take-out lunch from a Wendy's restaurant in Ogden, Utah. Three days later, Megan fell ill with significant painful diarrhea. Despite treatment by her regular physician, her condition did not improve and, on July 10, she developed persistent vomiting. That afternoon, she was rushed to an emergency room in Logan, Utah.

Blood tests in the emergency room indicated that Megan's kidneys were failing, and so she was admitted to the hospital. Her illness was later determined to be one of many illnesses in an outbreak of *E. coli* O121:H19 linked by public health officials to food served at Wendy's. Over the next day, her kidney function continued to slow, eventually halting altogether. She was transferred to McKay-Dee hospital in Ogden, Utah, to receive more specialized care. There, a diagnosis of hemolytic uremic syndrome (HUS) was confirmed. On July 14, Megan endured a kidney biopsy. The results were frightening: "necrosis of nearly the entire specimen [noted to be kidney cortex]." The renal cortex is where the kidney's filtering units are located and cortical necrosis indicates permanent loss of those filters—a finding typically found only in the most severe cases of HUS. The finding carried dire prognostic significance.

That same day, the nurses found Megan unresponsive and exhibiting seizure-like activity. A code was called. Dr. Pittman responded and arrived to find Megan with a heart rate of 160 beats per minute and tonic clonic seizures. Her oxygen saturation level was shockingly low at 71%. Fortunately, the physicians were able to get her seizures under control and her oxygen levels back up; it was clear, however, at this point that she was fighting for her life. On July 15, she began hemodialysis and plasmaphoresis to compensate for loss of kidney function. She remained hospitalized through July 28. Upon discharge, her kidneys were still not functioning normally, thus she continued treatment in an out-patient hemodialysis program.

Megan returned to the hospital three days a week for hemodialysis through September 7, at which point she was reduced to two sessions a week. Throughout this time, her kidney function remained abnormally low. She finally was able to discontinue regular dialysis in early October 2006. Her medical bills were over $350,000. Despite the extensive medical treatment, the damage to her kidneys was permanent and irreversible. Her prognosis as of 2008 was reported as follows:

> Based on the severity of her HUS, the evident extensive damage to her renal cortex, her markedly reduced estimated filtration rate of currently only 35 mls/min and the fact that Megan also now has evidence of significant proteinuria, it is my opinion, based on reasonable medical probability, that Megan will develop end stage renal disease (ESRD) and require renal replacement therapy in the form of chronic dialysis or kidney transplantation in the future.

It is estimated that Megan will require renal replacement therapy or a transplant by age 40 to 45. And after that, her future is still uncertain.

Megan will face many challenges once she undergoes a kidney transplant operation. She will need to take immunosuppressive medications for the rest of her life. Such medications are not only very costly, they also have significant side effects including high blood pressure,

diabetes, osteoporosis, altered appearance (such as moon faces due to steroids, and either hair loss or excessive hair growth with calcineurin inhibitors), and memory impairment. Immunosuppressive medications also significantly increase the risk for life-threatening infection or cancer.

C. Shiloh Johnson, *E. coli* O111, 2008

Shiloh Johnson was one of hundreds of persons sickened in the August 2008 *E. coli* O111 outbreak at the Country Cottage restaurant in Locust Grove, Oklahoma. Shiloh developed bloody diarrhea, and was hospitalized on August 22, 2008.

Once admitted, Shiloh's stool sample was tested and subsequently cultured positive for *E. coli* O111. Immediately after the start of the hospitalization, she began to suffer from hemolytic uremic syndrome (HUS). Her kidneys failed and her red blood cell and platelet counts plummeted. With a complete loss of kidney function, she required dialysis to survive. She was placed on continuous renal replacement therapy.

Forty-eight hours into the dialysis treatment, disaster struck. Shiloh developed a significant pericardial effusion (fluid around the heart) with tamponade (stoppage of blood flow caused by fluid). She went into cardiorespiratory arrest. She was endotrachoeally intubated and the pericardial fluid was drained. She was given a round of epinephrine, and the arrest was reversed. Shiloh remained on a ventilator through September 12. Soon, the area around her lungs also became inundated with fluid, necessitating the placement of chest tubes.

Throughout this time, Shiloh experienced full renal failure. She received dialysis treatment around the clock. On September 10, her doctors placed a periotoneal catheter and

switched her to peritoneal dialysis. The dialysis continued through September 27. She was finally discharged on October 3. By this point, her medical bills amounted to $450,000.

The severity of Shiloh Johnson's HUS, and in particular the length of her renal failure, puts her at serious risk of future complications including end stage renal disease. The extent of her long-term injury is still being assessed.

VI. CONCLUSION

In light of current scientific and medical research, the health hazards posed by STEC are undeniable. The CDC recognized these hazards in 2000 when the agency made all STEC nationally notifiable. Since reporting was implemented in 2001, instances of non-O157 STEC have steadily increased year by year. In 2005 alone, 501 cases of non-O157 STEC were reported through the National Notifiable Diseases Surveillance System.[56] This has become an issue that is too big to ignore any longer. Indeed, in a presentation given on September 14, 2009, L. Hannah Gould, MS, PhD from the CDC stated that non-O157 STEC causes an estimated 36,700 illnesses, 1,100 hospitalizations, and 30 deaths annually.[57]

Accordingly, the petitioners urge the administrator of FSIS to issue an interpretive rule declaring all STEC adulterants within the meaning of the FMIA in order to avoid the same kind of large-scale disaster that precipitated the 1994 declaration of *E. coli* O157:H7 as an adulterant. With this action, FSIS will take a significant leap forward in ensuring the safety of American consumers.

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[56] CDC. 2005. Bacterial Foodborne and Diarrheal Disease National Case Surveillance Annual Report, 2005. 16.

[57] Gould, L. Hannah. September 14, 2009. Update on the Epidemiology of Shiga toxin-producing *E. coli* in the United States. Capital Area Food Protection Association Meeting.

As the numbers of reported illnesses from non-O157 STEC steadily increase, immediate action on this issue is critical.

Very truly yours,

William Marler, Esq., on behalf of:

Marler Clark LLP, PS
Outbreak, Inc.
The Family of June Dunning
Megan Richards
Shiloh Johnson

Enclosures

ATTACHMENTS

Attachment No. 1. *American Mining Congress v. Mine Safety & Health Administration* 302 U.S. App. D.C. 38, 995 F.2d 1106 (D.C. Cir. 1993).

Attachment No. 2. 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 (Sept. 10, 2003).

Attachment No. 3. Bareta, J. K. Edge, S. Lathrop. 2009. Shiga Toxin-producing *Escherichia coli*, New Mexico, USA, 2004-2007. 15 Emerging Infect Dis. (No. 8) (Aug. 2009).

Attachment No. 4. Barkocy-Gallagher, G. A., T. M. Arthur, M. Rivera-Betancourt, X. Nou, S. D. Shackelford, T. L. Wheeler, and M. Koohmaraie. 2003. Seasonal prevalence of Shiga toxin-producing *Escherichia coli*, including O157:H7 and non-O157:H7 serotypes, and Salmonella in commercial beef processing plants. J Food Prot. 66:1978-86.

Attachment No. 5. 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.

Attachment No. 6. Bettelheim, K. A. 2007. The non-O157 shiga-toxigenic (verocytotoxigenic) *Escherichia coli*: under-rated pathogens. Crit Rev Microbiol. 33:67-97.

Attachment No. 7. Bosilevac J. M., M. N. Guerini, D. M. Brichta-Harhay, T. M. Arthur, and M. Koohmaraie. 2007. Microbiological characterization of imported and domestic boneless beef trim used for ground beef. J Food Prot. 70:440-9.

Attachment No. 8. Brooks, J. T., E. G. Sowers, J. G. Wells, K. D. Greene, P. M. Griffin, R. M. Hoekstra, and N. A. Strockbine. 2005. Non-O157 shiga toxin-producing *Escherichia coli* infections in the United States, 1983-2002. J Infect Dis. 192:1422-9.

Attachment No. 9. CDC. 2005. Bacterial Foodborne and Diarrheal Disease National Case Surveillance Annual Report, 2005. 16.

Attachment No. 10. CDC. 2007. Laboratory-confirmed non-O157 shiga toxin-producing *Escherichia coli* – Connecticut, 2000-2005. MMWR. 56:29-31.

Attachment No. 11. Chinyu Su, MD & Lawrence J. Brandt, MD, *Escherichia coli* O157:H7 Infection in Humans, 123 Annals Intern. Med. (Issue 9), 698-707.

Attachment No. 12. Company News; Jack in the Box's Worst Nightmare, N.Y. Times, Feb. 6, 1993, *available at* http://query.nytimes.com/gst/fullpage.html?res=9F0CE7DB153CF935A35751C0A965958260&sec=&spon=.

Attachment No. 13. Eblen, Denise. Public Health Importance of Non-O157 Shiga Toxin-Producing *Escherichia coli* (non-O157 STEC) in the US Food Supply. 2007. FSIS.

Attachment No. 14. Federal Register. January 19, 1999. [Docket No. 97-068N].

Attachment No. 15. Federal Register. October 9, 2007. [Docket No. FSIS-2007-0041].

Attachment No. 16. FSIS. 2008. 2008-2013 Strategic Plan.

Attachment No. 17. Gould, L. Hannah. September 14, 2009. Update on the Epidemiology of Shiga toxin-producing *E. coli* in the United States. Capital Area Food Protection Association Meeting.

Attachment No. 18. Hussein, H. S. 2006. Prevalence and pathogenicity of shiga toxin-producing *Escherichia coli* in beef cattle and their products. J Anim Sci. 85.

Attachment No. 19. Hussein, H. S. and T. Sakuma. 2005. Prevalence of shiga toxin-producing *Escherichia coli* in dairy cattle and their products. J Dairy Sci. 88:450-65.

Attachment No. 20. Johnson, K. E., C. M. Thorpe, and C. L. Sears. 2006. The emerging clinical importance of non-O157 shiga toxin-producing *Escherichia coli*. Clin Infect Dis. 43:1587-95.

Attachment No. 21. *Kriefall v. Excel*, 265 Wis.2d 476, 665 N.W.2d 417 (2003).

Attachment No. 22. 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).

Attachment No. 23. 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 (1991).

Attachment No. 24. Pierre Robitaille, *et al.*, Pancreatic Injury in the Hemolytic Uremic Syndrome, 11 Pediatric Nephrology 631, 632 (1997).

Attachment No. 25. Richard L. Siegler, MD, The Hemolytic Uremic Syndrome, 42 Ped. Nephrology, 1505 (Dec. 1995).

Attachment No. 26. Richard L. Siegler, MD, Postdiarrheal Shiga Toxin-Mediated Hemolytic Uremic Syndrome, 290 JAMA (No. 10) 1379, 1379 (Sept. 10, 2003).

Attachment No. 27. Samadpour, M., V. Beskhlebnaya, and W. Marler. 2009. Prevalence of non-O157 enterohaemmorrhagic *Escherichia coli* in retail ground beef in the United States. 7th International Symposium on Shiga Toxin (Verocytoxin)-producing Escherichia coli Infections. Buenos Aires, Argentina.

Attachment No. 28. *Texas Food Industry Ass'n, et al. v. Espy* 870 F. Supp. 143 (1994).