

BOSSY'S REVENGE: RISK MANAGEMENT IMPLICATIONS OF TWO FOOD SAFETY CRISES

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ABSTRACT

The worldwide emergence of pathogenic strains of *Escherichia coli* and the appearance of Bovine Spongiform Encephalopathy, primarily in Great Britain, are considered as case studies in the application of risk assessment, risk communication, and risk management. We conclude that the risk from *E. coli* can best be achieved by a defense-in-depth. Remaining scientific uncertainties prevent clear specification of a strategy for managing the risk associated with BSE and the new variant Creutzfeldt-Jakob Disease, but history of the crisis to date highlights the paramount importance of maintaining credibility by honestly reporting those uncertainties and their impact.

I. INTRODUCTION

In the last 15 years, we have seen two unrelated food safety crises, both centered, however, on the production and consumption of beef and dairy products. The first was the emergence of pathogenic strains of *Escherichia coli*, a common, and not normally pathogenic, resident of the mammalian intestinal tract. The second was the "mad cow" syndrome affecting the United Kingdom and, to a lesser extent, Europe. These crises exemplify distinct challenges of risk assessment, risk communication, and risk management.

Both crises involve infectious agents. As such, the issues of infectivity and virulence affect the analysis of the risk and its management. Infectivity is a measure of the ease with which a disease is passed from one host to another. Virulence is a measure of the severity of its consequences. Some diseases, like the common cold, are

highly infectious but not particularly virulent. Other diseases, like rabies, are highly virulent but require rather specific types of exposure for passage from one host to another. Ecological factors will also be important; what is the prevalence of the infectious agent and how well does it survive in the environment.

This paper will discuss these crises by first providing some discussion of the biological and medical background, followed by discussion of the risk implications of the crisis. We will consider assessment of the risk, followed by communication of the risk to decision makers and the public and the role of that in perception of the risk. Finally, the impact of analysis, communication, and perception on the implementation of a program for management of the risk will be described.

II. BIOLOGICAL BACKGROUND OF THE *E. COLI* CRISIS

The first crisis was the emergence of *Escherichia coli* O157:H7, a highly pathogenic strain of a common bacterial resident of human and animal intestinal tracts. This was quite a surprise, although in 1973, the Commissioner of the Food and Drug Administration had noted the theoretical hazard that antibiotic resistant strains of *E. coli* with the ability to produce enterotoxins might someday jump from livestock populations to be pathogenic in humans.¹

The first recognized outbreaks of human disease caused by *E. coli* occurred in 1982 in restaurants of the same fast food chain in Oregon and Michigan. A combination of field epidemiology and lab tests seemed to indict a rare

strain of *E. coli*, generally considered to be a harmless resident of the human gut. When researchers checked their database of 3000 stored *E. coli* samples for earlier appearance of the suspect strain, only one was O157:H7. It was collected in 1975 from a 50 year-old navy officer; while researchers were unable to locate her, they were able to obtain her medical records, which showed a disease course identical to the patients in Oregon and Michigan. At the same time, a doctor studying a third outbreak in Ontario, Canada, was able to make the connection between severe diarrhea caused by the new *E. coli* strain and "progression" to hemolytic uremic syndrome (HUS), a life-threatening complication involving kidney damage.²

Most variants of *E. coli* O157:H7 are resistant to one or more antibiotics. They are acid-tolerant and can survive for a long time at low temperatures. They are killed by high temperatures; cooking hamburger to an interior temperature of 160 deg F will kill O157:H7 and denature its Shiga-like toxin. Although apparently rare at the time of its initial identification as a pathogen, O157:H7 is now found worldwide and in significant fractions (3% to 20%) of randomly sampled meat animals on the farm and meat samples in the market.^{3,4} Cattle and other livestock and many wild animals are carriers of O157:H7 and it appears that some adult humans may be asymptomatic carriers also.

Part of the explanation of the emergence of virulence in *E. coli* is the general promiscuity with which bacteria share genetic material. There are three different mechanisms by which bacterial DNA can be passed from one organism to others. Perhaps the most important is the packaging of virulence and/or antibiotic resistance on plasmids, little loops of DNA that can be passed intact between bacteria, even to bacteria of different species. In fact, one of the names for the O157:H7 and other strains producing the same toxin is Shiga-like Toxin producing *E. coli* (STEC or SLTEC). The toxin is essentially indistinguishable from the toxin produced by *Shigella dysenteriae* type 1, the major causative agent of human dysentery, and is very similar to the toxin responsible for the diarrhea and dehydration of cholera.⁵ Shiga toxin and Shiga-like toxin, along with the closely related bacterial protein toxins, botulinum toxin and tetanus toxin, are the most lethal poisons known, with a human LD50 of 1 nanogram per kg of body weight (the LD50 is the dose that kills 50% of whatever test animal is being challenged).⁶

The importance of cattle in our diet and the ubiquity of *E. coli* in the bovine gut and in bovine feces, gave strain O157:H7 an importance quite beyond the actual risk it

represented when it first emerged in 1982. Another facet of the risk management problem presented by O157:H7 was the severity of the diarrhea in the clinical cases of the disease and the very serious and very distinctive clinical syndrome, hemolytic uremic syndrome, that affected 2-5% of the clinical cases. Something on the order of 10% of HUS victims die and many of the survivors have permanent kidney damage. *E. coli* has become the most frequent cause of bloody diarrhea in the U.S. and HUS has become the most frequent cause of fatal kidney failure in children in the U.S. In addition, highly-specific diagnostic tests have been developed that allow the pinpointing of the foods responsible for many outbreaks of O157:H7 disease. Thus, unlike many food-borne illnesses, O157:H7 HUS was potentially fatal, tended to announce itself, and there were techniques available to pinpoint the source of the infection.

III. RISK IMPLICATIONS OF *E. coli*

Significant scientific uncertainties were not a problem in this crisis. *E. coli* had been for many years the "lab animal" of choice of many research geneticists; many aspects of its behavior were well understood. There was the "ecological" puzzle of why this strain of a generally non-pathogenic bacterial had developed the ability to produce a toxin that could threaten the life of previously healthy children and adults. Early on, scientists and doctors had a significant problem in figuring out which strains were pathogenic and in making a realistic assessment of their prevalence, because most laboratories were not set up to identify the pathogenic strains and the mechanisms of infectivity and virulence were not understood.

One of the striking aspects of *E. coli* O157:H7 outbreaks is the high infectivity. Most estimates of an infective dose for O157:H7 are on the order of 10 organisms or less. This high infectivity means that cross-contamination of other foods during food preparation is a significant threat. In addition, secondary transmission to family members, acquaintances, and health care providers is a feature of many outbreaks. Because of this, O157:H7 outbreaks are particularly hazardous in day care centers, senior centers, and other populations with less-competent immune systems. Most cases of HUS occur in the young and most cases of a related neurological complication, thrombotic thrombocytopenia purpura, occur in the elderly.

The combination of virulence and the ability to trace outbreaks to the specific strain of *E. coli* and frequently even to the food processing plant or slaughterhouse that

produced the food creates enormous psychological and political pressure to "do something". This pressure has overwhelmed the previous laissez faire regime of "poke and sniff" inspection by the USDA and industry responsibility for more detailed control of the safety of meat. An example of the political pressure is the government response to a relatively minor outbreak of foodborne disease (only 15 people sickened, none seriously) traced to *E. coli* O157:H7 in ground beef produced by Hudson Foods. Government pressure led Hudson Foods to agree to the recall of 25 million pounds of hamburger; eventually Hudson sold the facilities involved to another food processor. The political pressures created by the Hudson outbreak and recall was probably responsible for the recent FDA decision to allow the irradiation of beef. The beef irradiation application had languished at FDA for almost three years prior to the Hudson Foods *E. coli* incident.

The Hudson outbreak provided an interesting window into some little known aspects of *E. coli* risk management. Hudson was primarily a poultry producer; they set up the beef processing plant that was the source of the outbreak at the request of one of their major customers, Burger King. Burger King, however, mindful of the substantial impact that a foodborne disease outbreak can have on a large chain of restaurants (that impact made clear in the 1992-3 Jack-in-the-Box outbreak in the Pacific Northwest), required Hudson to hold all shipments of ground beef until negative results of testing for *E. coli* were available.

Even though other food-borne pathogens, such as salmonella on chicken and eggs and campylobacter on chicken are responsible for greater morbidity and mortality than *E. coli*, it is frequently easier to point to specific sets of patients suffering and dying from *E. coli* and to identify the specific contaminated food. A related issue is that the focus of the public and the medical community on *E. coli* O157:H7 may be an example of the "drunk and the lamppost" phenomenon -- the drunk looked for his lost wallet under the lamppost rather than 50 meters away where he lost it because "there's no light over there". There are 60 strains of *E. coli* that produce the same toxin as O157:H7 but most of them are not tested for, even as infrequently as O157:H7. Some outbreaks attributed to O157:H7 may actually be caused by other strains; some outbreaks caused by other strains may not be attributed to any agent because the appropriate tests were not performed.

One of the most frequent responses in the food industry and elsewhere in the wake of the Hudson Foods outbreak

was that if consumers only had the sense to fully cook their hamburgers, occasional contamination with *E. coli* wouldn't be a problem. This is an inadequate approach to management of the risk of *E. coli* O157:H7 and related strains and other foodborne illnesses. Because of the very low infective dose, cross-contamination of other foods during food preparation is a serious problem. Moreover, many of the *E. coli* outbreaks have been associated with foods that are not normally cooked. Several have been traced back to unpasteurized milk or apple juice. Others are associated with fruits or vegetables apparently contaminated by livestock manure prior to picking. A few outbreaks appear to have been caused by contamination of swimming lakes or wading pools by human feces or livestock manure. There have been outbreaks traced to acidic foods like yogurt, mayonnaise, or apple cider that are generally considered safe because of their acidity.

E. coli is part of the normal intestinal flora for most mammals and some other animals. It would be impossible to eliminate *E. coli* and probably not even desirable; *E. coli* synthesizes vitamins B and K. A more interesting question is whether it is possible to bias the mixture of *E. coli* strains away from those that are pathogenic in man, that is, to somehow manipulate the environment of *E. coli* to favor the benign strains.

Another possibility being pursued is the development of a vaccine specifically targeted against *E. coli* O157:H7, the strain most frequently involved in serious human outbreaks. Initial trials on one such vaccine have been promising. If it proves effective, beef and dairy herds could be vaccinated. A difficulty with this approach is that more than 60 strains of *E. coli* are capable of producing the same toxin as *E. coli* O157:H7. Some of them have been responsible for significant human disease outbreaks. Other *E. coli* strains produce less dangerous toxins, although some of these are sufficiently resistant to heat that the usual guidelines for cooking hamburgers are not enough to denature the toxin.

IV. THE BOTTOM LINE FOR *E. COLI* RISK MANAGEMENT

Because of the ubiquity of *E. coli* O157:H7 and other STEC strains, the essential role of meat protein worldwide, the high infectivity and virulence of the STEC, and the demonstrated wide variety of ways humans have been infected, this is a risk that ought to be managed by defense in depth.

On the farms, there should be a move back to lower-density, more hygienic farming practices and away from

the use of sub-therapeutic feeding of antibiotics. Farmers raising fruits and vegetables must take actions to avoid contaminating them with livestock manure. In the slaughterhouses, improved methods of removing surface fecal contamination from carcasses should be implemented. Processing plants should institute online testing for generic *E. coli* and other organisms that can serve as sentinels for fecal contamination of meat. When fecal contamination is detected, more detailed testing for pathogens can be used to identify sources of contamination. There is a role for irradiation of meat and other foods most at risk of contamination. Home, restaurant, and institutional cooks need to be more careful that cooked foods are thoroughly cooked and that cross-contamination of uncooked foods from previous handling of raw meats and other potentially contaminated foods is avoided. Disease outbreaks, particularly in institutional settings must be carefully managed to avoid secondary, person-to-person transmission. Municipal water systems and recreational waters require constant vigilance against fecal contamination.

It could be argued that the infectivity and virulence of O157:H7 and the other STEC justifies considering untreated livestock manure as a biohazard. Clearly, we cannot use the same control measures used in research laboratories, but we ought to determine what control measures are cost-effective.

V. BIOLOGICAL BACKGROUND OF BOVINE SPONGIFORM ENCEPHALOPATHY

Bovine spongiform encephalopathy (BSE, or more commonly call mad cow disease) burst upon Britain in 1986. The affected cattle appeared nervous, lost weight, and had difficulty walking; milk yields declined (for the cows) and the cattle died within six months or so of the appearance of symptoms. Reliable diagnosis could only be made by post-mortem examination of the brain, which exhibited tissue shot through with cavities and individual cells filled with plaques. The cavities and the plaques are both characteristic of other transmissible spongiform encephalopathies (TSEs), such as the human diseases kuru and Creutzfeldt-Jakob Disease (CJD) and the animal diseases scrapie (a disease of sheep known for 250 years) and chronic wasting disease (which is enzootic in deer and elk in parts of Colorado and Wyoming).⁷

It is now generally accepted that the causative agent of mad cow disease (and new variant Creutzfeldt-Jakob Disease) is a prion, which is simply a cellular protein of unknown function. Stanley Prusiner proposed that prions were responsible for TSEs 20 years ago; initially, he was

ridiculed for proposing a disease agent containing no genetic material. Prusiner won the Nobel Prize for Medicine this year, but there are still respected scientists that do not believe prions are the sole cause of prion diseases. Their reluctance is related to the novelty of the disease process that Prusiner was proposing. He argued that if a distorted form of a normal cellular protein were introduced into a cell, it could serve as a template and cause normal protein to "snap" into the same dysfunctional three-dimensional shape. The previously-normal, newly-distorted prion protein would have the same ability to change the shape of normal protein thus resulting in a chain reaction that would eventually fill the cell with dysfunctional prion, killing it. The process has been likened to Kurt Vonnegut's fictional ice-nine, a form of water whose crystalline structure was such that if a molecule was accidentally introduced into a natural body of water, it would rapidly recruit all of the water to the ice-nine crystalline structure -- effectively irreversibly "freezing" the natural body of water.

When mad cow disease first came to the attention of the British government, it commissioned an epidemiological study to try to ascertain the cause. This nicely done study of the first 200 cases established that the only common factor was the feeding of meat and bone meal (MBM) containing beef and sheep protein to the affected cattle. This suggested that the agent responsible for scrapie in sheep might have jumped the "species barrier" to cattle. The species barrier is one of the puzzles of biology and medicine. Most disease organisms cause disease in a fairly small number of related target species. In addition, the disease may have a host to which it is so well adapted that it doesn't even cause disease -- essentially, the host is a refuge from which the disease organism ventures forth to cause disease in its targets. In some cases, like smallpox, the disease appears to have only one target species. Why a disease can jump from a host to a target species, but not jump to other species is not well understood. The fact that a strain of influenza jumped directly from birds to humans in Hong Kong earlier this year aroused an enormous amount of attention and concern that it might have the same combination of high infectivity and virulence as the strain that killed 20 million people in the influenza pandemic in 1918. One of the things that mislead British scientists and policy makers in their response to BSE was the preponderance of evidence that scrapie had never jumped the species barrier to man, in spite of hundreds of years of close association between man and scrapie-infected sheep.

Even accepting the theory that scrapie jumped from sheep to cattle (which not all scientists do), there is still

the question, "Why Great Britain and why 1985-86?" The most popular theory is that the Arab Oil Embargo of 1973 made petrochemicals and energy so expensive that British rendering plants changed the process they used to produce MBM. The new, continuous process used lower temperatures and eliminated a solvent extraction step used in the previous batch process. The distorted prion is quite resistant to heat, common sterilants, other chemical agents, extremes of pH, ultraviolet and ionizing radiation, and to the proteases that normally break down proteins in the gut. The assumption is that the new rendering process allowed the infective prion to survive to infect the cattle eating the MBM.⁷

Between April 1985 and July 1997, there were 170,000 confirmed cases of BSE in the United Kingdom (UK). It is generally accepted that there were a substantial number of additional, unreported cases. An epidemiological study estimated a total of 900,000 British cattle with BSE, 730,000 of which are estimated to have entered the human food chain between 1984 and 1995.⁷ For much of that time, both British MBM and British cattle were exported, particularly to the rest of Europe (60,000 cattle exported to Europe from 1985 to 1990). The British BSE epizootic peaked in 1992 with 37,500 cases and has been dropping steadily since then (to 7400 cases in 1996), apparently in response to measures taken to remove the infectious agent from cattle feed.

Finally, as a consensus developed on the cause of the epidemic (well, strictly speaking, the epizootic) in cattle, isolated cases of a fatal neuro-degenerative disease began to appear in humans. This disease was similar to CJD, but with some unsettling differences that suggested that it might be caused by the same agent that causes mad cow disease. By stages during 1995 and 1996, scientific evidence accumulated that "new-variant Creutzfeldt-Jakob disease" (nvCJD) was probably caused by the prion that causes mad cow disease in cattle. The British government announced in March 1996 that BSE was "probably" the cause of nvCJD. Subsequent medical and molecular biological research culminated in the publication in the 2 October 1997 issue of *Nature* of two papers that established more or less conclusively that BSE was the cause of nvCJD.^{8,9}

VI. RISK IMPLICATIONS OF BOVINE SPONGIFORM ENCEPHALOPATHY

In many ways, mad cow disease is the risk management problem from hell and the British government gave us an object lesson in the importance of maintaining credibility. The disease involved an activity at the center of human

life, eating, and impacted the livelihood of a significant fraction of British families. At the beginning, there was no real understanding of the cause in cattle and whether it represented any risk to people. The government claimed to know more than it really knew, responded with half measures, and continued to assure the British public they were safe. When the facts caught up with this policy there was a stunning loss of credibility, both internally and externally. A week after the British government's 20 March 1996 announcement that "in the absence of any credible alternative, the most likely explanation at present is that these cases are linked to exposure to BSE before the introduction of the SBO ban in 1989", the European Union (EU) banned exports of British beef to the rest of the European community (a number of countries, including the U.S. had expressed their lack of confidence in British responses to the crisis by banning imports of British beef in 1989). The British public was shocked, confused, and outraged. Beef sales within Britain dropped precipitously.

Issues of infectivity, virulence, and diagnostic testing complicated the risk analysis task for BSE. What parts of the animal were infective? Were humans at risk? Determining answers to these questions proceeded in slow motion because there was no quick diagnostic test. Questions of infectivity were answered by injecting homogenized brain, meat, milk, or whatever into mouse brains and then waiting a year or two to see if the mouse developed a disease similar to BSE. One major experiment involved following 315 calves of BSE-infected cows and 315 control calves for seven years to see if they developed BSE. In addition, a four- to eight-year incubation period after exposure for cattle and apparently a similar or longer period for humans further slowed the research needed to determine the risk and raised the stakes on any delay in instituting effective risk management. One of the most telling indicators of the infectivity of prions is that research laboratories studying them use containment and handling rules at the Biosafety Level 2 or 3. Biosafety Level 4 is reserved for agents such as the Ebola and Lassa fever viruses.¹⁰

Both risk analysis and risk communication were intrinsically difficult because the questions of whether there WAS a human hazard and, if there were, how serious it was, involved extremely complicated research in molecular biology, biochemistry, and medicine. Indeed, without the techniques of molecular biology, we wouldn't have a substantive basis for understanding BSE and designing risk management strategies.

From the first, even though it didn't understand the

cause of the disease, the British government assured the public that it was at no risk from eating beef or dairy products. Later, as some understanding of the likely cause of the disease developed, the government put in place some administrative controls on the movement into the human food chain of the parts of the beef carcass judged to be dangerous. Additional controls made illegal the feeding practices that appeared to be responsible for the spread of the disease among dairy and beef herds.

Farmers were forbidden to send cows that had begun to show symptoms of the disease to the slaughterhouse; the government paid them 50% of the animal's market value for animals that had BSE and 100% of the value for animals that tested negative. The combination of partial compensation, assurances that there was no real risk to people, and weak enforcement of the administrative controls that had been put in place encouraged farmers to evade the controls. The British media reported the failures of enforcement and flouting of the regulations, increasing public disquiet and distrust.

This administrative control approach was an application of the "stoplight" principle -- that is, that red lights stop trucks; the government again assured the public that now the problem was solved, even though it was still not understood whether people were at risk from beef already consumed or whether the measures taken would succeed in removing the infectious agent from the human food chain.

Even though many animal diseases are kept under control by draconian extermination of the affected herds or flocks, that option was considered but rejected by the British government. There is a rationale for that rejection. If contamination of MBM feed was indeed the cause of the BSE epizootic, then slaughtering of whole herds would not help. In addition, it can be argued the destruction of herds would lead farmers to conceal isolated cases of BSE among their animals. The French and Belgian governments, however, have been sacrificing whole herds in an attempt to control BSE.

The British government had ignored the old adage that "the absence of evidence is not evidence of absence" and argued that since there wasn't any credible evidence that mad cow disease could be passed to humans, it didn't happen. The government tried to control the scientific and public discourse by ridiculing dissident scientists, firing them, or starving them for funding. It withheld the raw data produced by government-sponsored research and/or limited review to "friendly" scientists.¹¹ The uncertainty resulting from the newness of the disease and

the complexity of the research methods provided years of cover for the British government's "no problem" policy. The strength and duration of the government's reassurances led to the massive loss of credibility when the facts finally caught up.

Over the eight years between 1988 and 1995 there were at least 71 separate legislative actions related to BSE. This is double-edged; it shows a UK government interest in improving the management of BSE risk, but it also reflects an agonizing step-by-step tightening of controls that, with accumulating evidence or political pressure are judged to be inadequate. The end result is a bit like the clock that struck 13. Not only do we not believe it, but all of its earlier utterances are called into question.

VII. THE BOTTOM LINE FOR RISK MANAGEMENT OF BSE

To date only 25 or so cases of nvCJD have appeared in Britain and Europe. Estimates of the eventual number of cases range from a few hundred to tens of thousands. The EU ban on British beef remains in place two years after the British government announcement that BSE was the likely cause of nvCJD. The government's committed cost of dealing with the BSE epidemic in cattle and nvCJD in humans is on the order of 3.3 billion pounds Sterling (US \$5 billion) over the 1996-2000 period.¹² The cattle sector of the British economy has borne substantial additional costs. Handling of the BSE crisis by the Conservative governments under Margaret Thatcher and John Major probably played a part in the stunning defeat of the Conservatives in 1997 by Labor under Tony Blair. Under pressure from families of nvCJD and the press, the government has launched a non-judicial inquiry under Lord Justice Phillips, into the handling of the crisis up to the 26 March 1996 announcement. The protocols for the inquiry seem quite attractive to the author, who has witnessed a number of similar inquiries in the U.S., in the form of congressional committee investigations or blue ribbon commissions. The BSE inquiry will be posting all of its testimony to a dedicated WWW site and those challenged by the testimony or evidence collected by the inquiry may have a right to legal representation funded by the inquiry.

Although the consequences and costs of earlier actions and inaction are becoming clearer, the appropriate risk management strategy for the future remains unclear because of the remaining scientific uncertainties. The current government appears to have learned the lesson of not overstating their degree of understanding of the disease and the residual risks to humans. They are

moving more cautiously in the face of uncertainty about whether bone marrow, lymphatic tissue, and white blood cells are infective, imposing some interim bans on any uses of these tissues that might expose humans to the prions while the scientific questions are being answered. Presence of BSE prions in lymph and white blood cells also calls into question the safety of the British blood supply. Another concern is that BSE might jump back to sheep, causing a scrapie-like disease that, unlike scrapie, can be transmitted to humans. Some countries within the EU, at substantial risk due to earlier imports of British cattle and MBM, appear to be proceeding less cautiously.

Another issue is the integrity of the risk assessment and risk management process itself. The British government was slow to set up scientific advisory committees, waiting until two years after the initial appearance of the disease (the Southwood committee in 1988). It compounded this delay by being resistant to recommendations and by attempting to shape the advice it got from advisory committees. It accepted the Southwood committee's recommendation to ban the use of scrapie-contaminated feed and to slaughter diseased cattle, but made it clear it would not accept a ban on human consumption of beef brains. Southwood persevered and a ban on brain and other nervous tissue was finally put in place a year later. That ban was probably the beginning of the end of significant exposure of humans to BSE.¹³

VIII. CONCLUSIONS

With mad cow disease, because of the many uncertainties surrounding the disease and the complexity of what was known, risk assessment and risk communication have been intrinsically very difficult. In addition, because of the economic importance of the beef and dairy industries, the British government pretended to know more than it did and offered assurances to the British public and Britain's trading partners that it could not really support scientifically. When scientific knowledge finally caught up, the British government suffered a massive loss of credibility that resulted in serious economic losses due to loss of internal and export markets and the direct cost of risk management programs. On an ironic note, several EU governments seem bound and determined to repeat the British government's risk management mistakes, even with the potential consequences so prominently displayed.

With *E. coli* O157:H7, scientific uncertainties have been much less of a factor, but the economic importance of the beef and dairy industries still make the choice of a risk management strategy difficult. The industry has to a

certain extent damaged its credibility by resisting improvements in hygiene and diagnostics with the argument that consumers would be unwilling to pay the extra cost. The O157:H7 problem has arisen at a time of increased resistance to regulatory initiatives. It is unclear why O157:H7 and related strains of *E. coli* have emerged as serious human pathogens and what is the optimal strategy to control or eliminate the pathogenic strains.

Government and industry credibility and public skepticism and outrage have been much less of a factor in the management of the risk of *E. coli* and other foodborne pathogens in the U.S. and abroad than with mad cow disease in Britain, probably because governments and industry have been more candid about their state of knowledge and the difficulties of risk management.

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