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NEWS & ANALYSIS

Mad Science and Mad Cows: The Case for EPA Regulation of Prionicidal Substances

by Christopher Busch and Jan Paul Mincarelli

I. Introduction: Confirmation of the Inevitable

On December 23, 2003, the U.S. Department of Agriculture (USDA) announced that it had diagnosed a single Holstein dairy cow near Yakima, Washington, with bovine spongiform encephalopathy (BSE), commonly known as mad cow disease. The cow was sent to the BSE international reference laboratory in Weybridge, England, where the diagnosis was confirmed on Christmas day, finally confirming the cattle industry's worst fears that mad cow disease would one day be detected in American herds. News of this discovery sent an immediate shockwave through the U.S. economy, resulting in declining restaurant stock prices, minimal beef futures trading, and public fear of beef consumption. On December 23, the Washington meat company that had slaughtered the BSE-positive cow voluntarily recalled over 10,000 pounds of beef that they believed might have been exposed to BSE-infected tissues.[1] On December 24, the USDA Food Safety and Inspection Service (FSIS), the same administrative division that had twice inspected the BSE-positive Holstein before releasing it for use as food for human consumption, mandated the recall of beef from cattle that had been slaughtered in the same plant on the same day as the BSE-infected cow.[2] The infected specimen was shown to be 1 of 81 cows that had been shipped to the United States from Canada on September 4, 2001.[3] The Secretary of Agriculture, Ann Veneman, immediately appointed an international panel of BSE experts to assess the response to the identification of the infected cow, to identify areas for improvement of current BSE safeguards, and to trace the whereabouts of the remaining 80 cattle that entered the United States with the infected cow.[4] Despite the earlier contention of the USDA's chief veterinary officer, W. Ron DeHaven, that they would "[b]e able to determine the whereabouts of most, if not all, of [the infected] animals within [s]everal days," he confessed seven weeks later that many of the cows' ear tags had been lost and that the likelihood of finding the other cattle from the herd was "pretty slim at this point."[5] Of the original 80 imported Canadian cattle, 52 have proven to elude the USDA's tracing methods. Eleven of those are believed to be at a particularly high risk for BSE due to their potential exposure to the contaminated feed that infected the BSE-positive Holstein.[6] When Dr. DeHaven admitted that "some of [the untraceable cows] very likely have gone to slaughter,"[7] it became immediately apparent that a number of cattle that had a high likelihood of being positive for BSE had been cleared for human consumption and assimilated into the channels of domestic beef distribution, thereby producing a pathogenic threat to the United States that could remain undetected for decades.

In the weeks that followed the discovery of the infected cow, scientific experts engaged in a series of heated debates regarding the necessity of further safeguards against mad cow disease, whether additional cattle were likely to be found with BSE, and to what extent the public was at risk for infection. While some asserted that "America is highly unlikely to suffer even one human case of 'mad cow' disease,"[8] Secretary Veneman's international panel reported that there was a "high probability" of additional cases of BSE being found in American cattle and that the United States needed to immediately establish additional safeguards for livestock feed and pet food.[9]

Despite these well-founded concerns and responsive measures, the press is overlooking a very serious issue that specifically pertains to the prospective prophylaxis and sanitation of these unique pathogens, as well as the administrative regulation of agents that may be developed and marketed for such purposes. Consider a hypothetical in which a group of cattle are tested and determined to be positive for BSE after having been slaughtered and processed for human

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1. Press Release, U.S. Department of Agriculture (USDA), Washington Firm Recalls Beef Products Following Presumptive BSE Determination (Dec. 23, 2003).
2. Centers for Disease Control and Prevention, *Bovine Spongiform Encephalopathy in a Dairy Cow—Washington State, 2003*, MMWR Wkly., Jan. 9, 2004.
3. *See id.*
4. *See id.*; Shankar Vedantam, *U.S. Ends Investigation of Mad Cow Case*, Wash. Post, Feb. 10, 2004, at A1.
5. Vedantam, *supra* note 4.
6. *See id.*
7. *See id.*
8. Dennis Avery & Alex Avery, *People Are "Fully Protected,"* USA Today, Jan. 29, 2004, at A14.
9. *See, e.g.*, Marc Kaufman, *More U.S. Cattle Likely to Have Mad Cow Disease*, Wash. Post, Feb. 4, 2004, at A2.

consumption.[10] In an attempt to protect themselves from liability and to allay the fears of the USDA and the general populace, the meat processing plant attempts to seek out a "pesticide" that would sterilize their facilities and allow them to resume their business activities without being administratively closed. Because it is unlikely that the managerial staff of the meat processing plant possesses any knowledge of biochemistry or pathogenic microbiology, they are certain to be at a loss in terms of what pesticidal agents could be employed to simultaneously maximize safety and efficacy.

In the event that a scenario of this sort were to materialize, the shortcomings of USDA and Food and Drug Administration (FDA) contingency plans for BSE outbreaks[11] would undoubtedly be problematic in terms of disease containment and public safety. However, an equally significant issue arises from the government's role in the regulation of any chemical agent potentially used by the meat processing plant in its attempt to eradicate the BSE contamination. Specifically, the U.S. Environmental Protection Agency (EPA) is confronted with a possible regulatory obstacle due to a technicality in their Federal Insecticide, Fungicide, and Rodenticide Act's (FIFRA's)[12] definition of "pest."[13] What EPA has potentially failed to address is that BSE is not caused by a traditional virus, bacterium, or other microorganism, but rather by an unprecedented infectious pathogen known as a prion, which causes a number of invariably fatal neurodegenerative diseases and utilizes an infectious mechanism that has forced contemporary biologists to rethink their naive faith in traditionally accepted notions of pathology.[14]

The purpose of this Article is threefold: first, to provide a scientific analysis of prions in order to adequately distinguish them from traditional pathogens; second, to analyze the nuances of FIFRA and discuss what other federal agencies have done to respond to the threat of BSE; and third, to discuss what regulatory courses of action are available to EPA and what level of deference they would receive from a reviewing court.

II. Scientific Background and Development of the Prion Model

Although it is historically unclear when prions first emerged, the British House of Commons recorded a discussion in 1755 describing the economic effects of a fatal and transmissible disease in sheep and the need for government to do something about it.[15] The disease that the British parliament was probably describing is now known as scrapie, a malady caused by prions that specifically infect sheep. Although they did not know of the existence of prions at the time, British, French, and German veterinarians began studying scrapie in the middle of the 19th century.[16] The term "slow virus" was coined by a Swedish scientist named Bjorn Sigurdsson, who was studying the prevalence of scrapie in sheep in Iceland in 1954. In 1957, two physicians, Daniel C. Gajdusek and Vincent Zigas, described a rare and invariably fatal neurological disorder that they had observed in the Fore Tribe in the highlands of Papua, New Guinea.[17] They concluded that the disease, which they called "kuru," was caused by the practice of ritualistic cannibalism, in which the Fore tribesmen prepared and consumed the tissues of deceased family members, including brain and spinal cord.[18] Two years later, American veterinarian William Hadlow proposed that kuru was very similar to scrapie and concluded that kuru was also caused by a "slow virus."[19] Igor Klatzo went on to describe what he believed to be an analogous relationship between kuru and another rare neurodegenerative disease, which had been reported extensively by 1930, known as Creutzfeldt-Jakob disease (CJD).[20] In both cases, Hadlow and Klatzo formulated their scientific conclusions after observing striking similarities of central nervous system (CNS) damage between kuru and scrapie or CJD-infected tissue.[21] In 1972, Dr. Stanley B. Prusiner, who would later win the Nobel Prize for his groundbreaking work with these emerging pathogens, developed an interest in the subject during his neurology residency when one of his patients was stricken with a disease that killed her within two months by destroying her brain and leaving her body unaffected by the process.[22] Although this patient displayed none of the classical symptoms afflicting individuals with viral infections (such as fever or immune response), Dr. Prusiner was told by his attending physician that his patient was suffering from a "slow virus."[23]

The term prion (derived from "proteinaceous" and "infectious") was coined by Dr. Prusiner during the course of his research (which was first published in the spring of 1982)[24] and is most appropriately defined as a proteinaceous infectious particle that lacks nucleic acid (deoxyribonucleic acid (DNA) or ribonucleic acid (RNA)).[25] We now know that prions are the cause of a family of diseases (found in animals and humans) called transmissible spongiform encephalopathies (TSEs).[26] Specifically, prions are known to be the cause of kuru, CJD, and variant Creutzfeldt-Jakob

10. The potential reality of this scenario becomes glaringly apparent when one considers the fact that the aforementioned BSE-positive cow in Washington passed through three different meat processing plants before a definitive diagnosis was made.
11. *See generally* FDA, *BSE Contingency Plan*, *at* http://www.fda.gov/oc/bse/contingency.html; *see also* USDA, *Bovine Spongiform Encephalopathy (BSE) Response Plan Summary*, *at* http://foia.aphis.usda.gov/lpa/issues/bse/bsesum.pdf.
12. 7 U.S.C. §§136-136y, ELR STAT. FIFRA §§2-34.
13. *See id.* §136(t).
14. Stanley Prusiner, *Prions*, 95 PROC. NAT'L ACAD. SCI. 13363, 13366 (1998).
15. Paul Brown & Raymond Bradley, *1755 and All That: Historical Primer of Transmissible Spongiform Encephalopathy*, 317 BRIT. MED. J. 1688 (1998).
16. *Id.* at 1689.
17. Daniel C. Gajdusek & Vincent Zigas, *Degenerative Disease of the Central Nervous System in New Guinea: Epidemic Occurrence of "Kuru" in the Native Population*, 257 NEW ENG. J. MED. 974 (1957).
18. *Id.*
19. W.J. Hadlow, *Scrapie and Kuru*, 2 LANCET 289 (1959).
20. Igor Klatzo et al., *Pathology of Kuru*, 8 LAB. INVEST. 799 (1959).
21. *Id.*; Hadlow, *supra* note 19.
22. Prusiner, *supra* note 14, at 13363.
23. *Id.*
24. *See* Stanley Prusiner, *Novel Proteinaceous Infectious Particles Cause Scrapie*, 216 SCIENCE 136 (1982).
25. Stanley Prusiner et al., *Review Prion Protein Biology*, 93 CELL 337 (1998).
26. TSEs are a group of rare degenerative brain disorders characterized by tiny holes that give the brain a "spongy" appearance.

disease (vCJD), among others in humans, BSE in cattle, and scrapie in sheep.[27] Unlike bacteria or viruses, prions can manifest themselves as hereditary, infectious, or spontaneous disorders, all of which surprisingly involve the biochemical modification of the prion protein (PrP), a naturally occurring endogenous[28] protein found in mammalian cells.[29] Indeed, prions are biological agents that are distinguishable from viruses or bacteria in a number of ways. Bacteria are primitive microorganisms that differ from traditional animal cells in that they do not have a membrane-bound nucleus, but rather have a dense "nucleoid" region where the genetic material (DNA) is localized, and lack the functioning cellular components known as organelles.[30] Viruses, on the other hand, are obligate intracellular parasites[31] that consist of a nucleic acid (either DNA or RNA) core surrounded by a protein coat and sometimes a lipoprotein envelope. They invade cells and incorporate their own genetic code into the host cell, causing the cell to produce viral DNA or RNA and protein coats. New viruses leave the host cell to infect other cells by one of two mechanisms. The first possibility involves a process known as budding, in which each virus particle (virion) exits the cell and, depending on the type of virus, takes a piece of the host cell's cytoplasmic membrane, cell surface membrane, or nuclear membrane with it to form a protective envelope,[32] ultimately resulting in the destruction of the host cell. The second possibility employs a simpler process in which the infected cell merely ruptures (known as lysis) and releases the new virions into circulation. A prion, however, is neither a microorganism, e.g., bacterium, nor a virus and may be distinguished from these traditional pathogens because it: (1) lacks DNA or RNA; (2) is incapable of reproducing or performing metabolic activities; and (3) is merely a constituent (protein) of living organisms. As implied above, a prion is nothing more than a modified protein isoform[33] that is converted into the pathologic form (denoted PrP^{Sc}) from the normal cellular PrP (denoted PrP^{C}) through a process in which its secondary alpha-helical structure is refolded into beta-pleated sheets.[34]

It has been estimated that nearly one million cattle were infected with prions in England during the BSE epidemic and that more than 160,000 (primarily dairy cows) have died of BSE over the past decade.[35] Studies of the British epidemic revealed that BSE was spread by the feeding of meat and bone meal (prepared from the butchering byproducts of sheep, cattle, pigs, and chickens) to cattle.[36] The incubation period[37] of BSE is approximately five years, which explains why most of the aforementioned cattle did not show physical symptoms of the disease, as they were slaughtered between two and three years of age.[38] BSE is known to be able to cross the species barrier and manifest itself in humans in the form called vCJD. Recent studies have statistically determined the incubation period of vCJD to be approximately 16.7 years,[39] raising a startling epidemiological conclusion of critical significance to public health: namely, if American citizens were exposed to infected tissue from any of the elusive cattle that were imported with the BSE-positive cow, we could find ourselves confronted with a "hidden epidemic" of sorts that would not manifest itself for nearly two decades.

III. The Emerging BSE Crisis

In response to escalating concerns surrounding the impending threat of BSE in America, the USDA entered into a cooperative agreement with the Harvard University School of Public Health's Center for Risk Analysis, which generated a comprehensive BSE risk assessment report (based on a three-year study) on November 26, 2001. This report was publicized as being the most comprehensive study of BSE and its potential risk factors ever performed in the United States.[40] The risk analysis was structured to accomplish three primary objectives: (1) to conduct a comprehensive review of current scientific information on BSE; (2) to determine the ways in which BSE could ever potentially enter the United States; and (3) to evaluate existing USDA policies and regulations in place to prevent the spread of BSE within the United States if it were to occur.[41] The authors of the assessment conducted a series of elaborate computer-based simulations and generated statistical distributions in order to empirically arrive at the conclusion that the risk of BSE occurring in the United States was extremely low.[42] In

27. Prusiner, *supra* note 14, at 13363.

28. "Endogenous" refers to being produced or synthesized within the organism.

29. Prusiner, *supra* note 14, at 13364.

30. Organelles are a group of small membrane-bound structures (located within the cytoplasm of eukaryotic cells) that are involved in a number of specialized cellular functions. Examples include mitochondria, golgi bodies, lysosomes, and endoplasmic reticula.

31. Unlike bacteria, viruses are incapable of existing independently of a living host and therefore do not technically constitute living organisms. However, viruses have a genetic code and, like living organisms, are capable of multiplying.

32. This envelope serves to protect the viral genome from physical, chemical, or enzymatic damage.

33. A protein may be thought of as a chain of subunits called amino acids. Isoforms are simply proteins that share the same primary sequence of amino acids in their chain, although they may fold differently, thus comprising a different three dimensional structure.

34. Prusiner, *supra* note 14, at 13364; *see also* K.M. Pan et al., *Conversion of Alpha Helices Into Beta Sheets Features in the Formation of the Scrapie Prion Proteins*, 90 Proc. Nat'l Acad. Sci. 10962 (1993).

35. R.M. Anderson et al., *Transmission Dynamics and Epidemiology of BSE in British Cattle*, 382 Nature 779 (1996); N. Nathanson, *Bovine Spongiform Encephalopathy (BSE): Cause and Consequences of a Common Source Epidemic*, 145 Am. J. Epidemiology 959 (1997).

36. Nathanson, *supra* note 35; J.W. Wilesmith, *Bovine Spongiform Encephalopathy—Epidemiologic Studies on the Origin*, 128 Veterinary Rec. 199 (1991).

37. Incubation period is defined as the time between pathogen entry and presentation of physical symptoms. The pathogen is spreading inside the host organism during this time but has not yet reached a level that is sufficient to cause illness. The period's length varies with disease. Lansing M. Prescott et al., Microbiology 724 (4th ed. 1999).

38. *Id*; *see also* Nathanson, *supra* note 35.

39. A.J. Valleron et al., *Estimation of Epidemic Size and Incubation Time Based on Age Characteristics of vCJD in the United Kingdom*, 23 Science 1663 (2001).

40. Press Release, USDA, Administration Continues to Strengthen BSE Protection Systems—Announces Status Report of Actions, Including Doubling of Testing for BSE in Cattle, Following the Harvard BSE Risk Assessment (Feb. 26, 2002), *available at* http://www.usda.gov/news/releases/2002/02/0070.htm.

41. *See* Joshua T. Cohen et al., *Evaluation of the Potential for Bovine Spongiform Encephalopathy in the United States* (Nov. 26, 2001), *at* http://www.aphis.usda.gov/lpa/issues/bse/madcow.pdf.

42. *See id*. at vii, 47-48, 111-13.

terms of risk factors associated with potential human exposure to BSE (possibly resulting in vCJD), Joshua Cohen and his colleagues reported that consumption of cattle brain, spinal cord, intestine, and tissue that had previously come into contact with infected CNS tissue posed the greatest risk.[43] Additionally, the study asserted that blood or skeletal muscle meat that had not come into contact with the CNS would not constitute a risk to humans.[44] Four months later, Dr. Prusiner and his colleagues would publish an article on the prevalence of prions in skeletal muscle that would refute these conclusions and render a portion of the Harvard risk analysis obsolete.[45] In a startling disclosure, this publication concluded:

> [Notwithstanding] previous studies [that] have generally reported low prion titers in muscle tissue . . . our studies demonstrate that mouse skeletal muscle is intrinsically capable of propagating prions . . . [and that] high prion titers may be found in skeletal muscle *even if central nervous system and lymphatic tissues are carefully excluded from the muscle*, *rais*[*ing*] *the concern that humans consuming meat from prion-infected animals are at risk for acquiring infection*.[46]

The study went on to hypothesize:

> If prions accumulate in certain muscles of humans with prion disease to levels near those that we[re] found in mice with prion disease, *it should be possible to definitively diagnose all forms of CJD and related disorders by using muscle tissue for biopsy*. This approach would offer significant advantages over the relatively difficult and morbid brain biopsy procedure, which is currently the only way to definitively diagnose prion disease in humans.[47]

The notion that prions could be present in human skeletal muscle at levels high enough to facilitate a diagnosis from a simple muscle biopsy contravenes every biological and epidemiological assumption held to date, while corroborating the possibility of a hidden human epidemic and presenting an emergent threat to the American cattle industry. Despite the landmark biological findings articulated in this study, the USDA has surprisingly continued to maintain (even weeks after the discovery of the BSE-positive cow) that BSE is found only in the CNS, small intestine, and eyes of cattle,[48] producing an inaccurate source of information that could inevitably mislead consumers and public health officials alike.

Another apparent shortcoming of the Harvard study pertains to its claim that blood does not constitute a threat of prional transmission or infectivity.[49] Although previously thought to be extremely unlikely, two studies appeared in early February 2004 in *The Lancet*, a prominent British medical journal, reporting the likelihood of vCJD infection via blood transfusions. The first article documented the clinical occurrence of vCJD transmission (resulting in eventual death) in an older British patient who had received blood from someone who was later diagnosed with the disease.[50] The second, published by a group of scientists from the French Atomic Energy Commission, substantiated the claims made in the clinical report by experimentally demonstrating that macaque monkeys could be infected with BSE orally or intravenously.[51] They concluded that blood transfusions should be regarded as a "likely route of contamination for vCJD patients with a medical history involving a transfusion during the period of risk [1980 to 1996]."[52] A leading expert on vCJD, Prof. Adriano Aguzzi of Zurich University, presented a commentary in which he said that "[s]hocking as it may be, the finding that vCJD can be transmitted via blood transfusion is not surprising," as similar studies have found that the same paths of infection can be reproduced in sheep.[53] This conclusion undoubtedly presents a number of staggering implications for the Department of Health and Human Services (HHS), the FDA, and the Centers for Disease Control (CDC) monitoring of blood and blood products and further demonstrates the overwhelming unpreparedness of the United States in its reliance on incomplete and inaccurate administrative reports.

IV. Prional Sterilization and Decontamination Methods

Perhaps the most startling feature of prions is their extraordinary resistance to inactivation by traditional methods of sterilization and their ability to bind to metal and plastic surfaces without losing their infectivity.[54] Conversely, due to the inherent parasitic nature of viruses, viruses are incapable of surviving independent of a living host, rarely remain viable for more than 24 hours on an inanimate surface, and are readily inactivated by traditional disinfectants and heat (in excess of 56 degrees centigrade). Pathogenic microorganisms such as bacteria or fungi are capable of remaining viable (and will replicate under ideal conditions) on inanimate surfaces but will typically respond to disinfectants (or to antibiotics in living organisms). It has been shown that most disinfectants are inadequate for eliminating prional infectivity[55] and that prions are specifically resistant to heat, formaldehyde, glutaraldehyde, radiation, freezing, drying, autoclaving, and organic detergents.[56] In fact, some studies

43. *See id*. at viii.
44. *See id*. at viii, 35.
45. *See* Patrick J. Bosque et al., *Prions in Skeletal Muscle*, 99 Proc. Nat'l Acad. Sci. 3812 (2002).
46. *Id.* at 3817 (emphasis added).
47. *Id* (emphasis added).
48. *See* USDA Food Safety and Inspection Service, *Bovine Spongiform Encephalopathy "Mad Cow Disease" Frequently Asked Questions* (updated Jan. 14, 2004), *at* http://www.fsis.usda.gov/Oa/FAQ/bse_general.htm.
49. *See* Cohen et al., *supra* note 41, at 35.
50. *See* C.A. Llewelyn et al., *Possible Transmission of Variant Creutzfeldt-Jakob Disease by Blood Transfusion*, 363 Lancet 417 (2004).
51. C. Herzog et al., *Tissue Distribution of Bovine Spongiform Encephalopathy Agent in Primates After Intravenous or Oral Infection*, 363 Lancet 422 (2004).
52. *See id.*
53. Patricia Reaney, *Blood Transfusions Can Spread vCJD* (Feb. 7, 2004), *at* http://smh.com.au/articles/2004/02/06/1075854059470.html.
54. C. Weissmann et al., *Transmission of Prions*, 99 Proc. Nat'l Acad. Sci. 16378 (2002).
55. *See*, *e.g.*, William Rutala & David Weber, *Creutzfeldt-Jakob Disease: Recommendations for Disinfection and Sterilization*, 32 Clinical Infectious Disease 1348 (2001).
56. P. Brown et al., *Newer Data on the Inactivation of Scrapie Virus or Creutzfeldt-Jakob Disease Virus in Brain Tissue*, 153 J. Infectious Disease 1145 (1986); P. Brown et al., *Chemical Disinfection of Creutzfeldt-Jakob Disease Virus*, 306 New Eng. J. Med. 1279 (1982); D. Taylor, *Inactivation of the Unconventional Agents of Scrapie, Bovine Spongiform Encephalopathy and Creutzfeldt-Jakob Disease*, 18 J. Hosp. Infections 141 (1991).

have demonstrated that aldehydes, such as formaldehyde, actually *enhance* the resistance of prions and that treatment of scrapie-infected brain tissue with formaldehyde *eliminated* the inactivating effect of autoclaving.[57] The idea that prions are capable of binding to an inanimate (such as stainless steel) surface has raised legitimate medical concerns for iatrogenic[58] transmission of CJD.[59] Iatrogenic CJD has been described in humans in three situations: (1) after use of contaminated medical equipment; (2) after use of extracted pituitary hormones or gonadotropin; and (3) after implantation of contaminated grafts from humans (including corneal and brain dura mater grafts).[60] One study in particular reported an instance in which medical wire electrodes had been implanted in a patient known to have CJD and were then cleaned with benzene and "sterilized" with 70% alcohol and formaldehyde vapor.[61] Two years after the procedure, the electrodes were retrieved and implanted into a chimpanzee in which the disease developed.[62] In response to these concerns, a number of studies have been conducted to determine what substances, if any, could be effective in inactivating prions and eliminating their infectivity.[63] The results of these analyses concluded that the most consistent inactivation results were seen in those chemical compounds containing chlorine[64] (however, the corrosive nature of chlorine would make it unsuitable for certain medical devices such as endoscopes),[65] guanidine thiocyanate,[66] formic acid,[67] or phenol.[68]

When one considers the combination of facts that BSE continues to escalate as a public health threat and that the only substances empirically proven thus far to be effective against prions are those considered too toxic for human exposure, an administrative policy dilemma requiring legislators and agency administrators to choose between two mutually exclusive outcomes seems to arise. Specifically, is it in our best interest to allow chemical manufacturers to produce compounds with toxic prionicidal components that could potentially poison the public, or to allow a safer but ineffective chemical to be sold and used, thus exposing the public to continued risk of transmission of vCJD and economic exploitation (particularly to those in the beef processing industry) with false claims of prionicidal efficacy? One way out of this dilemma would be to encourage the development of a product that is sufficiently safe and effective; however, there would be a need to adequately substantiate these claims. It is clear that the need for such an evaluation of these competing policies has arisen due to the fact that corporations have publicly announced the development of chemical products designed to eradicate prions on inanimate surfaces.[69] Genencor, for example, in response to the fact that traditional decontamination procedures have proven unsuccessful at destroying prions on contaminated equipment, has developed a disinfectant with protease enzymes that they believe can completely eliminate prions.[70] U.S. Global Nanospace, on the other hand, has commercially extended themselves even further by announcing the development of an agent that they refer to as "All-Clear."[71] In February of 2004, U.S. Global Nanospace issued a public statement to their shareholders highlighting their confidence in All-Clear's ability to wipe out mad cow disease.[72] They explained:

> We would like to reiterate that we do believe that "All-Clear" can be expanded to include remediation of agro-terrorism or pesticide threats and to mitigate other livestock or agricultural risk from diseases such as Mad Cow (BSE). . . . If a viral agent is indeed part of the not-yet-fully-understood reproduction of prions, which are recognized as the key causal agent of TSEs, it is possible that All-Clear can be of use in the mitigation of threats from Mad Cow disease.[73]

While it is entirely possible that All-Clear is a potent virucide, U.S. Global Nanospace has made an overt pesticidal claim that appears to be unsubstantiated, unproven, and conclusively based on false biological assumptions. As established earlier, prions are not viruses (but rather a unique pathogen comprised entirely of infectious protein) and do not utilize any viral agents in their physiology. While the likes of Dr. Prusiner have paved the way for us to have a more definitive understanding of prion biology than that de-

57. Rutala & Weber, *supra* note 55, at 1353; *see also* P. Brown et al., *Resistance of Scrapie Infectivity to Steam Autoclaving After Formaldehyde Fixation and Limited Survival After Ashing at 360° C: Practical and Theoretical Implications*, 161 J. Infectious Disease 467 (1990).

58. Iatrogenic is defined as any adverse condition in a patient occurring as the result of treatment by a physician or surgeon. Dorland's Illustrated Medical Dictionary 758 (25th ed. 1974).

59. *See* Eva Zobeley et al., *Infectivity of Scrapie Prions Bound to a Stainless Steel Surface*, 5 Molecular Med. 240 (1999).

60. Rutala & Weber, *supra* note 55, at 1350; P. Brown et al., *Iatrogenic Creutzfeldt-Jakob Disease at the Millennium*, 55 Neurology 1075 (2000).

61. Rutala & Weber, *supra* note 55, at 1350; C. Bernoulli et al., *Danger of Accidental Person-to-Person Transmission of Creutzfeldt-Jakob Disease by Surgery*, 55 Lancet 478 (1977).

62. *Id.*

63. *See* Victoria Steelman, *Creutzfeldt-Jakob Disease: Recommendations for Infection Control*, 22 Am. J. Infection Control 312 (1994); Darwin Ernst & Richard Race, *Comparative Analysis of Scrapie Agent Inactivation Methods*, 41 J. Virological Methods 193 (1993); Jun Tateishi et al., *Practical Methods for Chemical Inactivation of Creutzfeldt-Jakob Disease Pathogen*, 35 Microbiological Immunology 163 (1991); Laura Manuelidis, *Decontamination of Creutzfeldt-Jakob Disease and Other Transmissible Agents*, 3 J. NeuroVirology 62 (1997); D.M. Taylor et al., *Decontamination Studies With the Agents of Bovine Spongiform Encephalopathy and Scrapie*, 139 Archives Virology 313 (1994); Rutala & Weber, *supra* note 55.

64. *See* Rutala & Weber, *supra* note 55; Taylor et al., *supra* note 63; Tateishi et al., *supra* note 63.

65. *See* Rutala & Weber, *supra* note 55.

66. *See* Manuelidis, *supra* note 63; *see also* Tateishi et al., *supra* note 63. Guanidine thiocyanate is a strong protein denaturant that is considered to be extremely toxic and hazardous when it comes into contact with the skin, eyes, clothing, or mucous membranes of the respiratory tract (via inhalation). *See* Sigma Chemical Co., *Material Safety Data Sheet*, *at* http://www.aben.cornell.edu/bmb_lab/safety/guanidine.html.

67. Tateishi et al., *supra* note 63; *see also* P. Brown et al., *A Simple and Effective Method for Inactivating Virus Infectivity in Formalin-Fixed Tissue Samples From Patients With Creutzfeldt-Jakob Disease*, 40 Neurology 887 (1990).

68. Tateishi et al., *supra* note 63.

69. *See* Gaia Vince, *Volcanic Pool Enzyme Kills Prions*, New Scientist, July 29, 1999, at 50, *available at* http://www.newscientist.com/news/news.jsp?id=ns99993999; Press Release, U.S. Global Nanospace, U.S. Global Nanospace, Inc. Investor Update (Feb. 3, 2004), *available at* http://biz.yahoo.com/iw/040203/062559.html.

70. *See* Vince, *supra* note 69.

71. *See* Press Release, *supra* note 69.

72. *Id.*

73. *Id.*

scribed in U.S. Global Nanospace's press release, the idea that chemical manufacturers are developing prionicidal agents while simultaneously struggling to keep up with contemporary pathogenic microbiology, raises a number of serious concerns for the ultimate health of the environment and the public. Resultantly, it would be in the consumers' best interest to have established administrative procedures in place to evaluate whether proposed pesticidal agents are concurrently safe and effective. As it turns out, this authority resides with EPA and is articulated in one of its principle organic statutes, FIFRA.

V. Overview of FIFRA

The administrative regulation of pesticides dates back to the early 20th century when the U.S. Congress enacted the Federal Insecticide Act of 1910,[74] a statute intended to protect consumers from fraudulent marketing tactics of chemical product companies by "making it unlawful to manufacture and sell insecticides that were adulterated or misbranded."[75] Congress replaced the Act in 1947 with the enactment of FIFRA,[76] a considerably broader regulation that was initially regarded as "a licensing and labeling statute"[77] designed to ensure the "safe use and labeling of pesticides [in order] to protect those who came in immediate contact with them"[78] by mandating the inclusion of "directions for use; warnings to prevent harm to people, animals, and plants; and claims made about the efficacy of the product."[79] Although FIFRA originally fell under the jurisdiction of the USDA, the authority was transferred to EPA shortly after its formation by President Richard M. Nixon in 1970.[80] In response to the rapidly expanding pesticide industry, FIFRA was amended in 1972 to address the "mounting public concern about the safety of pesticides and their effect on the environment and because of a growing perception that the existing legislation was not equal to the task of safeguarding the public interest."[81] This revision was termed the Federal Environmental Pesticide Control Act of 1972[82] and essentially "transformed FIFRA from a labeling law into a comprehensive regulatory statute."[83] In accordance with the Act, EPA instituted "comprehensive labeling requirements governing the scope, content, wording, and format" of pesticide and herbicide labeling.[84] The regulation requires that "the manufacturer itself design and formulate the content of the label," and that it "file with the EPA a statement which included 'the name of the pesticide,' 'a complete copy of the labeling of the pesticide, a statement of all claims to be made for it and any directions for its use,' and 'a full description of the tests made and the results thereof upon which the claims are based.'"[85] The regulation grants the Administrator the authority to withhold a pesticidal registration until the manufacturer provides additional information, such as chemical composition, physical and chemical characteristics, and chemical metabolism.[86] A pesticidal registrant is approved when the Administrator has determined that the chemical's composition is such as to warrant the proposed claims for it and that the chemical will perform its intended function without unreasonable adverse effects on the environment (including injury to the applicator).[87] In short, these particular goals of FIFRA and its predecessors have been consistent for nearly a century: Congress wants to protect consumers from misrepresentations as to pesticides' efficacy, safety, or other qualities, and thus manufacturers must prove that the "claims" they make for their products are true.[88] In addition to the registration requirements, FIFRA confers considerable enforcement authority to EPA, including the issuance of a "[s]top sale, use, removal, and seizure" order,[89] civil penalties of up to $5,000 per violation,[90] and criminal penalties of up to $50,000 and/or up to one year of imprisonment.[91]

Under FIFRA, the word "pesticide" is arguably the most significant term defined within the text of the statute, which reads: "(1) any substance or mixture of substances intended for preventing, destroying, repelling, or mitigating any pest, (2) any substance or mixture of substances intended for use as a plant regulator, defoliant, or desiccant. . . ."[92] The determination of what constitutes a "pesticide" (at least for the purposes of part (1)) is therefore closely hinged to the word "pest," which is defined by FIFRA as: "(1) any insect, rodent, nematode, fungus, weed, or (2) any other form of terrestrial or aquatic plant or animal life or virus, bacteria, or other microorganism . . . which the Administrator declares to be a pest under [§]136w(c)(1)."[93] At first glance, prions do not appear to conveniently fit a verbatim reading of FIFRA's definition of pest[94] and would, therefore, potentially exempt any chemical agent that made a pesticidal claim against prions from enforcement action under the aforementioned registration requirements. In 2002, Congress enacted the Animal Health and Protection Act (AHPA)[95]

74. Pub. L. No. 61-152, 36 Stat. 331 (1910).
75. Ruckelshaus v. Monsanto, 467 U.S. 986, 991, 14 ELR 20539 (1984); *see also* H.R. REP. No. 838, 92d Cong. (1972), *reprinted in* 1972 U.S.C.C.A.N. 3933, 3999.
76. Pub. L. No. 80-104, 61 Stat. 163 (1947) (codified at 7 U.S.C. §§136-136y, ELR STAT. FIFRA §2-34).
77. *Monsanto*, 467 U.S. at 991 (citing Pub. L. No. 80-104, §§2(u)(2), 3(a)(3), 61 Stat. 163).
78. William T. Smith & Kathryn M. Coonrod, Cipollone*'s Effect on FIFRA Preemption*, 61 UMKC L. REV. 489, 490 (1993).
79. *Monsanto*, 467 U.S. at 991.
80. *Id*. (citing Reorganization Plan No. 3 of 1970, 35 Fed. Reg. 15623 (Oct. 6, 1970)).
81. *Id*. (citing S. REP. No. 838, 92d Cong. 3-9 (1972); S. REP. No. 970, 92d Cong. 9 (1972); H.R. REP. No. 511, 92d Cong. 5-13 (1972)).
82. Pub. L. No. 92-516, 86 Stat. 973 (1972).
83. *Monsanto*, 467 U.S. at 991 (citing H.R. REP. No. 511, *supra* note 81, at 1).
84. King v. E.I. DuPont de Nemours & Co., 996 F.2d 1346, 1347, 23 ELR 21282 (1st Cir. 1993) (citing 40 C.F.R. §156 (1996)), *cert. dismissed*, 114 S. Ct. 490 (1993).
85. *Id*. at 1347 (citing 7 U.S.C. §136a(c)(1)(B)-(D)).
86. 7 U.S.C. §136a(c)(2).
87. *Id*. §§136a(c)(1)(5), 136(d)(1)(C).
88. In re Roger Antkiewicz & Pest Elimination Prods. of Am., Inc., 8 E.A.D. 218, 242 (EAB 1999).
89. 7 U.S.C. §136k. A "stop sale" order can be thought of as an injunction against future violations.
90. *Id*. §136*l*(a).
91. *Id*. §136*l*(b).
92. *Id*. §136(u).
93. *Id*. §136(t).
94. This is true due to the previously established fact that prions are unique biological entities that are unlike viruses, bacteria, or other microorganisms. *See supra* notes 24-34 and accompanying text.
95. The AHPA was passed as Subtitle E of the Farm Security and Rural Investment Act of 2002, Pub. L. No. 107-171, 116 Stat. 134 (codified at 7 U.S.C. §§8302 et seq.).

and adopted a more expansive definition of "pest" (albeit for a different regulatory purpose), stating that the term includes, in pertinent part, "any of the following that can directly or indirectly injure, cause damage to, or cause disease in livestock . . . (E) A virus or viroid[,] (F) An infectious agent or other pathogen[,] . . . (I) A *prion*[,] . . . (K) Any organism similar to or allied with any of the organisms described in this paragraph."[96] While the authors of the definition of "pest" in the AHPA undoubtedly had the benefit of contemporary literature in the areas of molecular biology and physiology, it is clear that the drafters of FIFRA were at a scientific and chronological disadvantage. The definition of "pest" in FIFRA was last amended in 1972,[97] when TSEs were still classified as "slow viruses"; therefore, the addition of the word "virus" to the definition would have provided sufficient authority to regulate claims against the assumed causative agent of TSEs. FIFRA itself was last amended in 1996, one full year prior to Dr. Prusiner being awarded the Nobel Prize for his characterization of prions as nonviral, proteinaceous infectious particles. Until Dr. Prusiner's famed Nobel Lecture in 1997, there was still considerable debate in the scientific community regarding the pathogenic premise for TSEs.

Despite the apparent problem with the definition of "pest" in FIFRA, the statute does provide two possible solutions. First, the definition states that a "pest" may encompass any form of terrestrial or aquatic plant or animal life or virus, bacteria, or other microorganism that the Administrator declares to be a pest under §25(c)(1),[98] which states that "the Administrator, after notice and opportunity for hearing, is authorized to declare a pest any form of plant or animal life . . . which is injurious to health or the environment."[99] The regulatory provision promulgated as the exercise of that authority is codified at 40 C.F.R. §152.5. This section states:

> An organism is declared to be a pest under circumstances that make it deleterious to man or the environment, if it is:
>
> (a) Any vertebrate animal other than man;
>
> (b) Any invertebrate animal, including but not limited to, any insect, other arthropod, nematode, or mollusk such as a slug and snail, but excluding any internal parasite of living man or other living animals;
>
> (c) Any plant growing where not wanted, including any moss, alga, liverwort, or other plant of any higher order, and any plant part such as a root; or
>
> (d) Any fungus, bacterium, virus, or other microorganisms, except for those on or in living man or other living animals and those on or in processed food or processed animal feed, beverages, drugs (as defined in FFDCA [§]201(g)(1)) and cosmetics (as defined in FFDCA [§]201(i)).

This language appears to indicate that EPA sought to regulate the entire scope of the statutory term and declare all microorganisms as pests.

Second, in addition to this authority to promulgate a rule, §28(a) of FIFRA[100] specifies that "the Administrator, in coordination with the Secretary of Agriculture, *shall* identify those pests that must be brought under control."[101] It is clear that this could be readily accomplished at this point in time due to the AHPA's inclusion of prions in its definition of "pest" (which falls under the authority of the USDA) and that a BSE-positive cow has been detected in the United States, marking a pest that requires agency attention. Third, §28(d) of FIFRA stipulates that

> the Administrator, in coordination with the Secretary of Agriculture and the Secretary of Health and Human Services, *shall* identify pests of significant public health importance and, in coordination with the Public Health Service, develop and implement programs to improve and facilitate the safe and necessary use of chemical, biological, and other methods to combat and control such pests of public health importance.[102]

These remedies might ultimately be considered limited by FIFRA's definition of "pest"; and, therefore, they might be read in a constrained manner so as to limit EPA's ability to act in the wake of the BSE crisis. However, the Environmental Appeals Board has stated that FIFRA should be construed liberally, so as to effectuate its purposes, one of which is "alleviating health risks attributable to unsubstantiated and possibly misleading claims for the effectiveness of disinfectants."[103] In addition to an overall liberal construction of FIFRA, EPA should have the ability to interpret the definition of pest to include prions under basic notions of administrative flexibility (in order to adapt to emerging needs). Section VI will discuss how other agencies have faced the issue of whether prions can be called "organisms" for the purpose of regulations, and Section VII will discuss how a reviewing court should address an EPA interpretation of pest to include prions.

VI. How Other Agencies Have Regulated Prions

Although federal agencies obviously represent the same branch of the same government, prions have been defined in two general ways in regulations. The first approach is represented by the USDA's proposed definition of "organism" in 1986 for the purposes of regulating plant pests.[104] In a notice of proposed rulemaking, the USDA sought to define an "organism" very broadly, to mean

> any active, infective, or dormant stage or life form of an entity characterized as living, including vertebrate and invertebrate animals, plants, bacteria, fungi, mycoplasmas, mycoplasma like organisms, as well as viroids, viruses, and prions, or any entity related to the foregoing and any part, copy, or analog thereof . . . which is infectious.[105]

96. *Id*. §8302 (13) (emphasis added). The AHPA and FIFRA are the only two statutes that specifically define "pest."

97. *See* Pub. L. No. 92-516, 86 Stat. 973.

98. *See supra* note 93.

99. 7 U.S.C. §136w(c)(1).

100. *Id*. §136w-3.

101. *Id*. §136w-3(a) (emphasis added).

102. *Id*. §136w-3(d) (emphasis added).

103. In re Sporicidin Int'l, 3 E.A.D. 589, 604 (EAB 1991).

104. Specifically, the USDA was concerned with "articles [that] would be regulated in order to prevent the introduction, spread, or establishment of plant pests that are new to or not known to be widely prevalent or distributed within and throughout the United States." The definition of "plant pest" included the word organism, and thus the definition of organism was very important to the overall regulatory scheme. Introduction of Organisms and Products Altered or Produced Through Genetic Engineering Which Are Plant Pests or Which There Is Reason to Believe Are Plant Pests, 51 Fed. Reg. 23352, 23353 (proposed June 26, 1986) (to be codified at 7 C.F.R. pts. 330, 340).

105. *Id*. at 23354.

The USDA further explained that their proposed definition encompassed not just "whole organisms" but also "portions of organisms."[106] Additionally, the USDA stated that "[s]ome organisms, such as viruses, viroids, and prions, are considered by some people to be living (as in 'active'), and by other people not to be alive."[107] Even if the USDA did not consider prions to be "alive" (a position it did not take), this would not preclude prions from being regulated as organisms: "[Even] inactivated or dead organisms, or portions of these organisms, are covered under this definition because they may be active or infectious in that they are capable of functioning or affecting the functioning of another organism."[108]

After receiving comments, the USDA decided to remove prions from the definition of organism:

> Prions have been removed from the list of organisms which are or contain plant pests in §340.2. There is no evidence at the present time that any prion is associated with a plant pest. All of the prions identified to date have been associated with diseases in animals. If in the future a prion should be found to be associated with a plant pest or suspected of causing a plant disease that organism could be added to the list.[109]

In other words, the USDA redacted prions from its proposed definition not because they felt that it would stretch the definition of organism too far but that it was just not necessary to include prions since there was no evidence at that point (or now) that prions adversely affected plant life. The USDA specifically left the door open to add prions to their definition of organism.

The U.S. Department of Transportation (DOT) currently takes a different approach with respect to the classification of prions. Instead of trying to make the case that prions can fit within the term "microorganism" or "virus," the agency decided, when defining "infectious substance" for the purpose of hazardous material transport, to specifically include prions as a separate item in addition to viruses or microorganisms: "[I]nfectious substance[ ] means a material known to contain or suspected of containing a pathogen. A pathogen is a virus or microorganism (including its viruses, plasmids, or other genetic elements, if any) or a proteinaceous infectious particle (prion) that has the potential to cause disease in humans or animals."[110] The DOT specifically added prions to the definition after receiving comments. They stated that while "prions are not microorganisms . . . we have modified the definition to specifically include them."[111]

One could make the argument that Congress ratified the USDA approach (that "organism" for regulatory or statutory purposes can mean much more than the technical definition) when it passed the AHPA.[112] However, it is important to note that the two regulatory approaches employed by the USDA and the DOT do agree that, no matter where prions fit in a classification scheme, because of their status as an infectious agent, they need to be addressed by regulations in order to protect human health and the environment.

VII. Can EPA Interpret "Pest" to Include Prions?: Administrative Mechanisms and Their Ability to Stand Up in Court

A. EPA Could Attempt to Rely on the Statute and Regulations as They Are Currently Worded

Because of the current ignorance of mad cow disease and fear of health and economic consequences that consumers, and in particular, meat processing plant owners face in light of the recent outbreak in the United States, EPA should act quickly in order to protect consumers from unsafe and/or ineffective products advertised to "kill" or "remove" prions. As this section will demonstrate, there are many procedural means that EPA may employ in an attempt to assert regulatory authority under FIFRA over products that make claims against prions. This section will describe each of these means and will assess its effectiveness to survive judicial review, in order from least action required from EPA to most action required.

The first tactic the Agency could take is to rely on FIFRA and the regulations promulgated pursuant to that statute as they currently stand. In other words, without any formal regulation or notice to the regulated community, EPA could internally take the stance that prions are pests and thus register prionicidal substances that meet safety and efficacy requirements, approve protocols for use, and take enforcement actions against manufacturers that advance prionicidal claims in the labeling of a product that violate the requirements of FIFRA. If any EPA action was challenged in court, EPA could take the position that prions fit within its statutory (FIFRA §2(t))[113] and regulatory authority (40 C.F.R. §152.5).

EPA could argue that Congress intended the Agency to regulate prions based on the specific wording of the definition of pest. Of key importance is a change in language from the 1947 FIFRA definition of "economic poison"[114] (since renamed "pesticide") to the current definition of "pest" that has not been amended since it was first passed in 1972.[115] In 1947, FIFRA defined economic poison as "any substance or mixture of substances intended for preventing, destroying, repelling, or mitigating any insects, rodents, fungi, weeds, and any other forms of plant or animal life *or viruses* . . . which the Administrator shall declare to be a pest."[116] This

106. *Id*.

107. *Id*.

108. *Id*.

109. Introduction of Organisms and Products Altered or Produced Through Genetic Engineering Which Are Plant Pests or Which There Is Reason to Believe Are Plant Pests, 52 Fed. Reg. 22892, 22899 (June 16, 1987) (to be codified at 7 C.F.R. pts. 330, 340).

110. 49 C.F.R. §173.134(a)(1).

111. Hazardous Materials: Revision to Standards for Infectious Substances, 67 Fed. Reg. 53118, 53120 (Aug. 14, 2002) (to be codified at 49 C.F.R. pts. 171, 172, 173, 177, and 178). Prions were added because the proposed definition of infectious substance read "a material known to contain or suspected of containing a pathogen that has the potential to cause disease when exposure to it occurs. Pathogens are microorganisms (including bacteria, viruses, rickettsia, parasites, and fungi) or recombinant microorganisms (hybrid or mutant) that cause infectious disease in humans or animals." Hazardous Materials: Revision to Standards for Infectious Substances and Genetically Modified Microorganisms, 66 Fed. Reg. 6942, 6956 (Jan. 22, 2001) (to be codified at 49 C.F.R. pts. 171, 172, 173, 177, and 178).

112. *See supra* notes 95-97 and accompanying text.

113. 7 U.S.C. §136(t).

114. FIFRA, ch. 125, §2, 61 Stat. 163 (1947).

115. Federal Environmental Pesticide Control Act of 1972, Pub. L. No. 92-516, §2(n), 86 Stat. 973.

116. 61 Stat. 163 (emphasis added).

implies that Congress, at that time, did not consider viruses to be a "form of life." However, a reasonable interpretation of the current definition (specifically, "virus, bacteria, or other microorganism . . .")[117] would be that the congressional listing of "virus" before "bacteria" and "other microorganism" implies that Congress considers viruses to be a microorganism similar to bacteria, because to preclude this interpretation they could have retained similar language from the 1947 version. Similarly, the definition of "pest" in the AHPA indicates that Congress takes a more expansive view of the term "organism" for regulatory purposes than microbiologists might, placing an emphasis on infectivity and not on whether the entity reproduces or metabolizes on its own.[118] Also, Congress expressly includes viruses as pests, by definition; additionally, under §25(c)(1) a pest needs to be a "form of life." Therefore, it is reasonable to infer that viruses and prions could reasonably be considered to be within the scope of "organisms," "microorganisms," or "form[s] of life" in FIFRA.

In addition, Congress has not had the option to address the statutory ambiguity since wide acceptance of the prion hypothesis has occurred only recently. The definition of pest has not been amended since 1972,[119] when TSEs were considered to be caused by slow viruses.[120] Accordingly, at the time of defining pest, the addition of "virus" to the definition would have provided sufficient authority to regulate claims against the causative agent of TSEs.[121] Furthermore, FIFRA was last amended in 1996, one year before Dr. Prusiner was awarded the Nobel Prize. In 1996 there was still considerable debate in the scientific community as to whether TSEs were caused by prions or whether a virus was still necessary for infectivity.[122] Thus, at that time Congress did not have sufficient evidence that the definition of pest needed to be changed, and an amendment to change the definition of pest was not yet ripe. Since 1996, however, the conferral of the Nobel Prize to Dr. Prusiner and subsequent studies have resulted in broad acceptance of the prion hypothesis; by 2002, Congress was concerned enough to enact the AHPA. EPA could argue that since FIFRA was a remedial statute designed to effectuate consumer protection, it should be given a liberal construction in order to implement congressional intent.[123]

A federal court would most likely not afford this informal construction of EPA's own organic statute and regulation much formal deference. Although the U.S. Supreme Court has said that "at the frontiers of science . . . a reviewing court must be at its most deferential,"[124] the Court has declined to give deference to "agency litigating positions that are wholly unsupported by regulations, rulings, or administrative practice."[125] Consequently, EPA's argument would most likely be reviewed de novo like any other statutory construction case. Although it is conceivable that these arguments could prevail based on ideas of congressional intent and other canons of statutory interpretation, it would be preferable for the Agency to cite to something that would be afforded formal deference by a reviewing court.

B. PR Notice 2002-1 Is an Agency Interpretation of "Pest" That Should Receive Judicial Deference

1. Section 28(d) of FIFRA Charges EPA With Compiling a List of Public Health Pests

There is one document that EPA could argue confers upon them sufficient legal authority to regulate prionicidal products. Section 28(d) of FIFRA states that "[t]he Administrator, in coordination with the Secretary of Agriculture and the Secretary of Health and Human Services, shall identify pests of significant public health importance. . . ."[126] On September 11, 2002, EPA implemented the congressional mandate via a Pesticide Registration Notice, specifically PR Notice 2002-1 (PR Notice),[127] which specifically identifies prions as one of many "public health pests." According to EPA, the list was "a cooperative effort by HHS, [the] USDA, and EPA" and that "[i]ssuance of this list fulfills the requirement of FIFRA [§]28(d) to identify pests of significant public health importance as a part of this process."[128] In addition to its cooperation with HHS and the USDA, EPA also sought public comments[129] and provided notice to the regulated community in the *Federal Register*.[130] EPA has thus asserted that prions are a "public health pest" and therefore that they are a "pest."[131]

117. 7 U.S.C. §136(t)(2).

118. *Id*. §8302(13).

119. *See* Pub. L. No. 92-516, §2(n), 86 Stat. 973.

120. *See supra* notes 16-23 and accompanying text.

121. It would be an absurd result if an advance in the characterization of pathogens could remove something that was once part of EPA's jurisdiction under FIFRA merely because the agent lacked nucleic acid. Since the scientific community was split for over a decade on the question of whether the prion hypothesis was valid or whether a virus was still involved in causing TSEs, *see*, *e.g.*, K.Y. Kreeger, *Heretical Ideas*, SCIENTIST, June 10, 1996, *available at* http://www.thescientist.com/yr1996/june/research_960610.html, this would pose an interesting question of at what point of acceptance in the scientific community the former "pest" would be extracted from EPA's authority.

122. *See*, *e.g.*, *id*.

123. *See supra* note 103 and accompanying text.

124. Baltimore Gas & Elec. Co. v. Natural Resources Defense Council, 462 U.S. 87, 103, 13 ELR 20544 (1983).

125. Bowen v. Georgetown Univ. Hosp., 408 U.S. 204, 212 (1983).

126. 7 U.S.C. §136w-3(d). The rest of the section states: "[A]nd, in coordination with the Public Health Service, develop and implement programs to improve and facilitate the safe and necessary use of chemical, biological, and other methods to combat and control such pests of public health importance." *Id*.

127. *PR Notice 2002-1*, *at* http://www.epa.gov/oppmsd1/PR_Notices/pr2002-1.pdf.

128. *Id*.

129. *See* Lists of Pests of Significant Public Health Importance; Notice of Availability, 65 Fed. Reg. 16615-701 (Mar. 29, 2000) (providing notice of a draft PR Notice and requesting comments); Lists of Pests of Significant Public Health Importance; Extension of Comment Period, 65 Fed. Reg. 36442-502 (June 8, 2000) (extending the comment period).

130. *See* Lists of Pests of Significant Public Health Importance; Notice of Availability, 67 Fed. Reg. 57597-601 (Sept. 11, 2002) (providing notice of the PR Notice and the final list).

131. "Public health pest" is not defined in FIFRA, although "public health pesticide" is. *See* 7 U.S.C. §136(nn), defining it as

> any minor use pesticide product registered for use and used predominantly in public health programs for vector control or for other recognized health protection uses, including the prevention or mitigation of viruses, bacteria or other microorganisms (other than viruses, bacteria, or other microorganisms on or in living man or other living animal) that pose a threat to public health.

2. How Much Agency Deference Should the PR Notice Be Afforded?

If EPA's interpretation of "pest" that would include prions (based on the listing of prions as a public health pest) were challenged in federal court, it would pose an interesting question of how much deference should be afforded the PR Notice under the Court's holding in *United States v. Mead Corp.*[132] In *Mead*, the Court held that

> administrative implementation of a particular statutory provision qualifies for *Chevron* deference when it appears that Congress delegated authority to the agency generally to make rules carrying the force of law, and that the agency interpretation claiming deference was promulgated in exercise of that authority. Delegation of such authority may be shown in a variety of ways, as by an agency's power to engage in adjudication or notice-and-comment rulemaking, or by some other indication of a comparable congressional intent.[133]

For this reason, the test for whether the PR Notice is afforded *Chevron, U.S.A., Inc. v. Natural Resources Defense Council, Inc.*[134] deference (as opposed to the lesser *Skidmore v. Swift & Co.*[135] deference) is whether Congress intended EPA to compile a list of public health pests that carries the force of law, and whether the PR Notice was promulgated in exercise of that authority.

Although the PR Notice was not issued as a legislative rule[136] or as the result of adjudication, EPA could make the argument that the PR Notice meets the ill-defined category of "some other indication of a comparable congressional intent."[137] First, the statutory charge in §28(d) of FIFRA does appear to meet the first requirement under *Mead*, i.e., that Congress delegated authority to the Agency, because the statute states that EPA's "Administrator, in coordination with the Secretary of Agriculture and the Secretary of Health and Human Services, *shall* identify pests of significant public health importance. . . ."[138] Obviously, the imperative language explicitly demonstrates that Congress intended EPA to compile the list.

EPA could also argue that the PR Notice issued under §28(d) was promulgated in exercise of that authority, in order for that interpretation to be afforded *Chevron* deference. The statute requires only one procedural requirement to be followed: that EPA coordinate the compilation of the list with HHS and the USDA. The statute requires neither notice-and-comment rulemaking nor adjudication; in fact, in §25(c)(1), the other section in FIFRA that confers upon the Administrator of EPA authority to declare pests, Congress explicitly required notice-and-comment rulemaking and thus arguably could have mirrored that language in §28(d).[139] Interestingly, EPA did allow for public notice-and-comment rulemaking in compiling the list (although the final list was not in the form of a legislative rule codified in the *Code of Federal Regulations*), and hence the Agency went above and beyond the procedural requirements as explicitly intended by Congress.[140]

One challenging the PR Notice declaration of prions as a "public health pest" would argue, among other things, that the notice should be reviewed under the less deferential *Skidmore* standard. The best argument that the PR Notice should be afforded *Skidmore* deference (rather than *Chevron* deference) is that the notice does not carry "the force of law" as required in *Mead*.[141] Specifically, EPA states in the *Federal Register*:

> While the requirements in the statutes and Agency regulations are binding on EPA and the applicants, the PR Notice is not binding on either EPA or pesticide registrants, and EPA may depart from the guidance where circumstances warrant and without prior notice. Likewise, pesticide registrants may assert that the guidance is not appropriate generally or not applicable to a specific pesticide or situation.[142]

The fact that EPA itself has reserved the right to deviate from its stance without notice is a factor demonstrating that the PR Notice is meant merely as guidance and therefore under *Mead* should not receive *Chevron* deference. Additionally, although *Mead* stated that "we have sometimes found reasons for *Chevron* deference even when no such administrative formality [such as rulemaking or adjudication] was required and none was afforded,"[143] the only case the Court cited to support *Chevron* deference absent such procedures was one in which "long-standing precedent" supported deference to the Comptroller General.[144] Here,

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It is questionable whether prions could be considered a public health pest but not a "pest" for the purposes of §2(t); rather, "public health pest" was probably intended to be a subset of "pest." However, the emphasis in the definition of public health pesticide on vector control and public health programs confirms that Congress intended EPA to regulate pesticides that stem the spread of communicable disease. The definition of both pest and public health pest should thus be broad enough to include prions, since a product that would disinfect or sterilize a surface from prions would be "used predominantly in public health programs." *Id*. Ultimately, if it is true that all public health pests must also be pests, then by implication EPA's declaration that a prion is a "public health pest" is also an official agency construction of "pest" to include prions, i.e., for the purposes of §2(t).

132. 533 U.S. 218 (2001).

133. *Id*. at 226-27.

134. 467 U.S. 837, 14 ELR 20507 (1984).

135. 323 U.S. 134 (1944).

136. Although EPA provided notice of the list in the *Federal Register* and sought public comments, the PR Notice does not appear in the *Code of Federal Regulations*.

137. *Mead*, 533 U.S. at 226-27.

138. 7 U.S.C. §136w-3(d) (emphasis added).

139. In 7 U.S.C. §136w(c)(1), Congress authorized the Administrator to declare a pest "after notice and opportunity for hearing." *Id*. There is no such requirement in §28(d). *See id.* §136w-3(d).

140. One major reason that courts defer greatly to agency interpretations that are the result of notice-and-comment rulemaking is that the procedural requirements allow notice to and participation by the regulated community, and thus it seems fair to bind the regulated community because it could have earlier voiced its concerns to the agency. *See*, *e.g.*, Stephanie Stern, *Cognitive Consistency: Theory Maintenance and Administrative Rulemaking*, 63 U. PITT. L. REV. 589 (2002) (according to proponents, notice-and-comment rulemaking enhances fairness, quality, and democratic legitimacy through public participation and rational deliberation. Therefore, if EPA did provide notice and comment, even if the end result does not appear in the *Code of Federal Regulations*, it could be argued that the PR Notice should be afforded *Chevron* deference.

141. 533 U.S. at 227.

142. Lists of Pests of Significant Public Health Importance; Notice of Availability, 67 Fed. Reg. 57597 (Sept. 11, 2002).

143. 533 U.S. at 231.

144. *Id*. at 231 n.13 (citing NationsBank of N.C., N.A. v. Variable Annuity Life Ins. Co., 513 U.S. 251, 256-57 (1995)).

there is no long-standing precedent to support deference to a PR Notice.[145]

The lack of clear standards in *Mead* makes it difficult to predict whether a court would afford *Chevron* or *Skidmore* deference to the PR Notice.[146] Although there appears to be a clear delegation of authority from Congress to compile the list of public health pests, and that the PR Notice was promulgated in the exercise of that authority, it seems by EPA's own admission that the PR Notice is not a binding "rule of law" and therefore should be afforded *Skidmore* deference. Thus, although Congress in §28(d) "explicitly left a gap for [EPA] to fill"[147] and "that Congress would expect the agency to be able to speak with the force of law when it . . . fills a space in the enacted law,"[148] it appears that EPA may have taken its interpretation outside the realm of *Chevron* deference by declaring that it is not binding.[149] However, the fact that EPA has declared prions to be pests under §28(d) in the PR Notice is still an interpretation of the Agency, and as such it should be afforded deference under *Skidmore*.[150] The following section will apply the *Skidmore* factors to the PR Notice position, and the section after that will apply *Chevron* standard if it were found, under the nebulous standards of *Mead*, that the PR Notice was worthy of greater deference.

145. No opinions have resolved the issue of what deference any of the many PR Notices should be afforded.

146. *See Mead*, 533 U.S. at 241, 245 (Scalia, J., dissenting) (stating: "The Court has largely replaced *Chevron*, in other words, with the test most beloved by a court unwilling to be held to rules (and most feared by litigants who want to know what to expect): th'ol' 'totality of the circumstances' test. . . . The principal effect [of *Mead*] will be protracted confusion."). Recent cases have not shed any further light on how the *Mead* test is to be applied in difficult cases. *See*, *e.g.*, General Dynamics Land Sys., Inc. v. Cline, 124 S. Ct. 1236 (2004) (stating that although the parties contested how much deference an Equal Employment Opportunity Commission (EEOC) regulation was due, "the recent cases are not on point here. In Edelman v. Lynchburg College we found no need to choose between *Skidmore* and *Chevron*, or even to defer, because the EEOC was clearly right; today we neither defer nor settle any degree of deference because the Commission is clearly wrong."). *Id.* at 1248. The Court's "clearly right/clearly wrong" test obviously would not help in a situation like the PR Notice where there are reasonable arguments to be made on both sides of the issue of whether FIFRA's definition of pests could include prions. However, a recent U.S. Court of Appeals for the District of Columbia Circuit case ruled that *Chevron* deference applied to an Agency interpretation that was neither the result of notice-and-comment rulemaking nor adjudication, *see* Pharmaceutical Research & Mfrs. of Am. v. Thompson, 2004 WL 690497, at *3 (D.C. Cir. 2004) (rejecting an argument that an interpretation did not carry the force of law because authority was explicitly conferred on the agency).

147. *Chevron*, 467 U.S. at 843-44.

148. *Mead*, 533 U.S. at 229.

149. It is unclear why EPA felt that it was required to do this. Just because the congressional mandate to compile a list did not require EPA to go through notice-and-comment rulemaking (and thus there was no requirement that the list be published in the *Code of Federal Regulations*) did not mean that Congress intended the list to be merely for purposes of guidance. In fact, at the very least the PR Notice should bind the agency, under the clear terms of the statute, to coordinate with the Public Health Service in the development and implementation of programs to improve and facilitate safe and necessary methods "to combat and control such pests of public health importance." *See* 7 U.S.C. §136w-3(d).

150. *See*, *e.g.*, Bragdon v. Abbott, 524 U.S. 624, 642 (1998) ("[T]he well-reasoned views of the agencies implementing a statute 'constitute a body of experience and informed judgment to which courts and litigants may properly resort for guidance.'" (quoting *Skidmore*, 323 U.S. at 139-40)).

3. Application of the *Skidmore* Factors to EPA's Interpretation of "Pest" in the PR Notice

In *Skidmore*, Justice Robert Jackson enunciated the following factors to apply to an agency determination that is not "controlling" on the Court: "[T]he thoroughness evident in its consideration, the validity of its reasoning, its consistency with earlier and later announcements, and all those factors which give it the power to persuade, if not the power to control."[151] Thus the first factor to which a reviewing court would look is the thoroughness of the agency consideration. This factor should weigh heavily in EPA's favor: the PR Notice represents (even if it does not bind) the position of the entire Agency and is not merely a response of a low-level staff member. In addition, the list of public health pests was compiled in coordination with two other agencies (the USDA and HHS) and with public participation, thus supplying two levels of extraagency consideration.

Next, under "the validity of its reasoning" factor, the Court may look at whether the determination of prions is in accordance with the overall intent of FIFRA. This factor could weigh in EPA's favor if the reviewing court accepted the argument that the essential factor that ties "virus, bacteria, or other microorganism" together, and the reason for Congress to further address the issue of pests of public health importance, is that Congress intended EPA to regulate products that make claims against any agent that causes disease.[152] Since prions cause disease, and since EPA is charged under FIFRA to regulate products claiming to be effective against such infectious agents, it is valid for EPA to extend its regulatory authority within the sphere Congress envisioned. The fact that Congress has defined pest in the AHPA to explicitly include prions and any other infectious agent also demonstrates the validity of EPA's reasoning.[153]

The third *Skidmore* factor, consistency, does not necessarily weigh in favor of EPA or a potential challenger. Since EPA did not take a position one way or the other on their regulatory authority with respect to prions before the PR Notice, there is no previous position against which to compare the list of public health pests to determine consistency.

"Other factors" courts have utilized in *Skidmore* analysis include contemporaneousness and agency expertise. Contemporaneousness may weigh in EPA's favor since prions appeared on the first (and only) list compiled in execution of the statutory mandate. Agency expertise should also weigh in favor of EPA, since the issue of the classification of organisms and "novel pathogens" is at the "frontiers of science" where agency deference "should be at its greatest."[154] The regulatory nature of FIFRA, which requires EPA to evaluate registrants' studies in the areas of toxicology, organic chemistry, and microbiology, mandates technical ex-

151. 323 U.S. at 140.

152. *See supra* note 118 and accompanying text.

153. One could argue that Congress may have intended different regulatory scopes for the USDA and EPA and that if Congress could broadly define pest for the USDA then it could for EPA as well. While this is a possibility, it is also true that Congress defined pest for EPA in 1972 when prions had yet to be discovered and no amendment to pest under FIFRA has yet to be proposed, raising the possibility that Congress would prefer such a small gap as whether prions could be considered "other microorganism[s]" to be filled by the Agency.

154. Baltimore Gas & Elec. Co. v. Natural Resources Defense Council, 462 U.S. 87, 103, 13 ELR 20544 (1983).

pertise in order to adequately implement. Therefore, EPA has expertise in an area that a reviewing court generally does not: pathogenic microbiology. Thus, EPA can advance a position that may seem to be inherently contradictory to those without the expertise: although a prion may not fit the definition of "virus," "bacteria," or "microorganism" (as they are usually defined by these experts), nevertheless a prion is sufficiently similar to all three since it causes transmissible disease, and therefore, for regulatory purposes, a decision to include prions under the term "other microorganism" would not be arbitrary or capricious.[155]

Although it is far from clear, most *Skidmore* factors, therefore, would tend to uphold EPA's position that prions should be considered pests since they have been identified as a public health pest under the PR Notice. Although it is supposedly more deferential, since the two-part *Chevron* test frames the issue differently than *Skidmore*, however, it is important to understand how a court would assess the validity of the PR Notice under *Chevron*.

4. Application of the *Chevron* Test to EPA's Interpretation of Pest

If *Chevron* deference were warranted EPA's interpretation of the definition of pest, the first issue facing the reviewing court was "whether Congress has directly spoken to the precise question at issue," i.e., whether congressional intent was clear.[156] One challenging EPA's jurisdiction of prions under FIFRA would most likely focus their argument here, stating that Congress has directly spoken to the issue of whether prions are pests. Such an argument would best be bolstered by a so-called textual approach whereby the challenger would cite dictionary definitions that support the view that none of the three terms (virus, bacteria, and microorganism) include prions.[157] If these three terms included in the definition of pest were considered separately in this textual manner, it is possible that a court could rule that Congress clearly foreclosed prions from EPA's regulatory reach.

EPA could advance two arguments if its PR Notice were afforded *Chevron* deference. First, it could attempt to argue that under *Chevron* step one, Congress has clearly spoken to the issue in their favor, i.e., that prions fit clearly within the definition of pest. This argument, based on ideas of the overall intent of FIFRA,[158] would most likely be unpersuasive. A better tactic would be for EPA to argue that Congress has not spoken to the issue of whether prions are pests, i.e., that the definition of pest is ambiguous. EPA could argue that the definition of pest, specifically the language "virus, bacteria, or other microorganism," is ambiguous because the placing of virus as one of two enumerated examples of a microorganism demonstrates that Congress considered viruses to be microorganisms.[159] Since Congress therefore defined "microorganism" differently than the scientific community, there is an inherent ambiguity as to what exactly Congress intended the term "other microorganism" to mean.[160]

If a court found that the term "other microorganism" was inherently ambiguous, the next question would be whether a construction to include prion was a "permissible" one.[161] This issue should be resolved in EPA's favor. EPA could cite many factors that demonstrate the reasonableness of their interpretation. First, EPA could argue that prions are sufficiently similar to bacteria and viruses in that they have the potential to cause disease in humans and therefore should be treated similarly for the purposes of the regulatory regime in order to protect consumers, public health, and the environment.[162] Second, the fact that Congress has defined pest to include prions, albeit in another statute (the AHPA), demonstrates that EPA's interpretation is reasonable. Third, the fact that the definition has not been amended since 1972 when TSEs were considered to be caused by "slow viruses" (and the existence of prions was unknown) and that therefore EPA is merely updating the statute to accord with the development of a "novel pathogen" is another argument that the interpretation is reasonable.

One challenging EPA's interpretation of pest under a *Chevron* step-two analysis would most likely have to make an argument that allowing EPA to broadly interpret "other microorganism" is irrational because there would be no sufficient bound on EPA's regulatory authority. This slippery slope argument should fail because the ability of EPA to add entities into the definition of pest would be limited to whether that entity could cause transmissible disease (the unifying factor between "virus, bacteria, or other microorganism").[163]

155. *Skidmore* and *Chevron* could be considered different shorthand means of assessing whether Agency action is arbitrary or capricious under the Administrative Procedure Act (*see* 5 U.S.C. §706(2)(A)). *See*, *e.g.*, Einer Elhauge, *Preference–Estimating Statutory Default Rules*, 102 COLUM. L. REV. 2027, 2132 (calling *Chevron* an "implicit interpretation of the Administrative Procedure Act").

156. 467 U.S. at 842-43.

157. For an example of one Court case in which there was extensive discussion of the role of dictionaries in *Chevron* step-one analysis, see MCI Telecommunications Corp. v. AT&T, 512 U.S. 218 (1994). The approach employed by the majority can be considered "textual" because it places much, if not all, of the emphasis of *Chevron* review on the supposedly objective meaning of words (rather than legislative history or the general intent of the statute as a whole). *See*, *e.g.*, Thomas W. Merrill, *Textualism and the Future of the* Chevron *Doctrine*, 72 WASH. U. L.Q. 351 (1994) ("The critical assumption is that interpretation should be objective rather than subjective; that is, the judge should ask what the ordinary reader of a statute would have understood the words to mean at the time of enactment, not what the intentions of the enacting legislators were."). For an argument that this textualist approach is inconsistent with *Chevron*, see generally *id.* Justice John Paul Stevens best summarized the opposing approach to textualism in *AT&T* when he stated that "[d]ictionaries can be useful aids in statutory interpretation, but they are no substitute for close analysis of what words mean as used in a particular statutory context." 512 U.S. at 240 (Stevens, J., dissenting).

158. *See supra* notes 113-25 and accompanying text.

159. *See supra* notes 114-18 and accompanying text.

160. It is true that there are microorganisms other than bacteria to which the term could apply, e.g., protozoans such as *Giardia*. However, if viruses are considered microorganisms as well, there is an inherent ambiguity as to the scope of the term. In addition, it could be argued that "other microorganism" is a catchall, and therefore should be treated as an explicit delegation by Congress to EPA to interpret and to adapt to emerging concerns such as prions.

161. *Chevron*, 467 U.S. at 843.

162. Also, the declaration in §28(d) of FIFRA that EPA compile a list of pests of public health importance and the definition of "public health pesticide" in §2(nn) (which emphasizes vector control and combating threats to public health) demonstrates a congressional intent that EPA play an active role in public health programs. Since prions and BSE are a major threat to public health, EPA can fulfill this mandate by registering safe and effective products, approving protocols for use, and enjoining sale of unregistered products, all of which would aid in the development of programs that would adequately prevent a potential "hidden epidemic."

163. Arguably, the agents that cause vCJD fit within FIFRA's definition of pest when they were considered to be caused by "slow viruses." A simple change in the terminology describing the agents should not remove them from the definition if there has not been a change in the

It is important to note that EPA's stance would not confer authority to bring carcinogens or some other broad class of environmental agents into the definition of pest, because there a few critical distinctions between carcinogens and prions.[164] Cancer is a disease that, unlike vCJD, cannot be transferred from person to person or animal to person. In addition, while prions can constantly increase their number in the body,[165] carcinogens cannot replicate and thus generally require higher concentrations and multiple or chronic exposures in order to cause deleterious effects.[166] Thus, carcinogens are sufficiently distinct from prions, viruses, or bacteria and would not be considered part of the definition of pest if prions were included in that definition.

C. An Interpretation of 40 C.F.R. §152.5

Since FIFRA states that pest "means . . . any . . . virus, bacteria, or other microorganism . . . *which the Administrator declares to be a pest under [§]25(c)(1) of this title*,"[167] the statute required EPA to name which microbes it considered to be pests. Section 25(c)(1) required EPA to declare pests "after notice and opportunity for comment" if they are "injurious to health or the environment."[168] Implementing this authority, EPA promulgated a legislative rule codified at 40 C.F.R. §152.5 stating that an "organism is declared to be a pest under circumstances that make it deleterious to man or the environment, if it is: (d) any fungus, bacterium, virus, or other microorganism [sic]. . . ."[169] Thus, EPA's rule, since it incorporates almost identical language as the statute,[170] is just as ambiguous as to what "other microorganism" means as the statute. Therefore, one mechanism EPA could employ in order to incorporate prions under the definition of pest would be to issue an interpretation of §152.5 that the rule includes prions (or any other infectious agent).

The benefits of such an interpretation would be that one could be issued quickly and that, if it were challenged, a court would likely grant it great deference. The test for an agency interpretation of its own ambiguous rule was enunciated in *Thomas Jefferson University v. Shalala*.[171] There, the Court stated that the agency interpretation must be given "controlling weight unless it is plainly erroneous or inconsistent with the regulation."[172] A reviewing court "must defer to the [agency's] interpretation unless an alternative reading is compelled by the regulation's plain meaning or by other indications of the agency's intent at the time of the regulation's promulgation."[173] Thus, if EPA issued an interpretation of its regulation that prions were pests, that interpretation would be controlling unless the court ruled that the plain language foreclosed the interpretation or that the agency had a contrary intent at the time of issuance of the rule.

One challenging an interpretation of the rule would most likely have to make the argument that the interpretation runs contrary to the plain language of the rule. Such an argument mirrors the textual argument discussed above in the context of a *Chevron* step-one analysis.[174] It is true that if one looks at each word of the regulation separately and defines them according to accepted notions of microbiology, the plain meaning may foreclose an interpretation that prions could fit within the regulatory definition. However, this approach does not take into account that the regulation, when read as a whole, is ambiguous because the presence of "virus" in a list of types of organisms before the catchall "other microorganisms" demonstrates that EPA defines "organism" differently than microbiologists. Additionally, Congress defined "form of life" differently than microbiologists when, in §25(c)(1) of FIFRA, it authorized the Administrator to "declare [as] a pest any form of plant or animal life" but went on to except "bacteria, virus, and other microorganisms on or in living man" (which would be subject to FDA jurisdiction) thus implying that Congress considered a virus to be a "form of life."[175] Therefore, the statutory charge and the interpretation as a whole are ambiguous as to what "other microorganism" (and even "form of life") means, and an "alternative reading" of the regulation is not "compelled."[176]

understanding of the net effect of what the agent does in the human body. *See supra* note 121. Additionally, the novelty of the characterization of prions as a nonorganismal, nonviral infectious agent demonstrates that allowing prions to fit within the definition of pest would not render the definition without reasonable bounds. The discovery of prions as "an entirely new genre of disease causing agents," Press Release, The 1997 Nobel Prize in Physiology or Medicine, *available at* http://www.nobel.se/medicine/laureates/1997/press.html, is matched most recently with the discovery that "by the beginning of [the 20th] century, it had been established that filterable viruses were different from bacteria and could cause diseases in plants, livestock and humans." *See* PRESCOTT ET AL., *supra* note 37, at 337. Thus, the discoveries of prions and viruses as infectious agents distinct from organisms are separated by almost a century. Despite how many categories of infectious agents there may be, it is natural to consider them as one large category, *see*, *e.g.*, *id.* (stating that "[u]ntil well into the [19]th century, harmful agents were often grouped together and sometimes called viruses"), and therefore it is reasonable to define pest in this matter and it is defined in a way that will not expand.

164. Although EPA does have the authority to regulate chemicals designed for mitigating an undesirable plant, animal, virus, or other microorganism, a chemical designed to remove carcinogens would most likely be a "cleaning" solution and thus specifically excluded from EPA's regulatory authority. *See* 40 C.F.R. §152.10(a).

165. Prions cause endogenous host isoform protein to conform to their disease-causing shape, a process akin to "replication" because the number of disease causing agents can continually increase without additional exposure to disease causing prions; *see supra* notes 28-34 and accompanying text.

166. It is important to distinguish prion "replication" from chemical bioaccumulation. Since prions are infectious agents that can increase their numbers (*see supra* note 165), one low-level exposure could, given time, cause disease because the number of prions continually increases. Bioaccumulation, however, refers to the uptake of chemicals through the food chain that the body can not eliminate, and thus the concentration of that chemical in the body could increase over time as there are multiple exposures to that chemical, e.g., high mercury concentrations from repeatedly eating fish. However, the concentration of any chemical cannot, at any time, be higher than that which the body has ingested or otherwise been exposed to since the chemicals can not replicate or otherwise increase its number.

167. 7 U.S.C. §136(t) (emphasis added).

168. *Id.* §136w(c)(1).

169. 40 C.F.R. §152.5. The rest of §152.5(d) excepts those microorganisms "on or in living man or other living animals and those on or in processed food or processed animal feed, beverages, drugs . . . and cosmetics . . . ," which would fall into the FDA's jurisdiction. *Id.*

170. *See*, *e.g.*, *PR Notice 2002-1*, *supra* note 127 ("EPA in its regulations in 40 C.F.R. [§]152.5 has broadly defined the term to cover each of the organisms mentioned except with respect to the organisms specifically excluded by the definition.").

171. 512 U.S. 504 (1994).

172. *Id.* at 521.

173. *Id.*

174. *See supra* note 157 and accompanying text.

175. 7 U.S.C. §136w(c)(1).

176. *Thomas Jefferson Univ.*, 512 U.S. at 521.

EPA would also likely prevail on the issue of whether the interpretation is "inconsistent with the regulation."[177] The regulation was last modified in 1988,[178] which was at a time when the prion hypothesis was regarded with great skepticism, and nothing in the discussion of the regulation in the *Federal Register* mentions prions.[179] Therefore, it cannot be said that there was clear agency intent at the time of promulgation to not include prions within the definition of pest.[180]

In addition, it cannot be argued that the addition of prions would otherwise be inconsistent with the regulatory definition of pest. The statutory charge in FIFRA states that EPA can declare a pest if it is "injurious to health or the environment,"[181] which prions clearly are. Thus, the interpretation would have to be inconsistent with the definition of "organism,"[182] "other microorganism[ ],"[183] or "form of life,"[184] which is an argument about plain meaning (see above). Thus, an EPA interpretation of §152.5 would seem to be able to survive a challenge.[185]

D. A Legislative Rule

One last approach that EPA could employ in order to include prions in the definition of pest would be to utilize its authority under §25(c)(1) of FIFRA to amend its rule (codified at 40 C.F.R. §152.5) via notice-and-comment rulemaking. Such an amended rule could track the language of the AHPA, could merely include prions along with "fungus, bacterium, [and] virus,"[186] or could make it clear that the definition includes any agent that can cause infectious disease. The benefit of this rulemaking approach would be that a challenge to the final rule would be afforded *Chevron* deference.[187] However, the procedural requirements of notice-and-comment rulemaking would require more time before EPA could regulate prionicidal products than would other administrative mechanisms mentioned above.[188] Whether the rule would survive review under *Chevron* analysis would most likely depend upon how textual of an approach the court would employ; however, the rule should stand because there is sufficient ambiguity in the definition as a whole and an interpretation to include prions is a reasonable one.[189]

E. A Congressional Amendment

The last means to remedy this statutory problem would be a congressional amendment to FIFRA's definition of pest. Such action would obviously confer upon EPA sufficient authority to regulate prionicidal products and would provide a chance to update the statute. Additionally, it would provide EPA a safe harbor from judicial review. However, it is unlikely that Congress would entertain such a technical amendment. Although Congress did enact a broad definition of pest in the AHPA, the lack of any legislative history regarding the definition and the sheer volume of the bill that was passed indicate that Congress may not have even been aware of any possible controversy regarding the definition.[190] Considering that the broad statutory language employed mirrors that of the USDA's proposed regulation of plant pests,[191] it is entirely possible that the definition in the AHPA was the result of lobbying on the part of the USDA and was not even scrutinized by Congress. Ultimately, the relatively small change required in FIFRA's definition of pest and the significant expertise required to administer the statute are two factors that would indicate that this is an appropriate situation for agency gap filling.

VIII. Conclusion

Mad cow disease is present in the United States. Despite certain government assurances, specifically that the "downer cow" came from Canada and that only one cow has tested positive for BSE, it is very likely that mad cow has been present in America for some time, will continue to be present in American livestock, and will manifest itself in the form of human vCJD cases at some time in the future.[192] In

177. *Id*.

178. *See* Pesticide Registration Procedures; Pesticide Data Requirements, 53 Fed. Reg. 15952, 15976 (May 4, 1988) (to be codified at 40 C.F.R. pts. 152, 153, 156, 158, and 162).

179. *See generally id*.

180. If there is any agency intent regarding prions and the definition of pest, the skepticism prevalent at the time toward the prion hypothesis would argue that prions should be included in the definition, since the contrary hypothesis was that TSEs were cause by viruses. *See supra* note 163.

181. 7 U.S.C. §136w(c)(1). The regulatory definition phrases the requirement slightly differently: "An organism is declared to be a pest under circumstances that make it deleterious to man or the environment. . . ." 40 C.F.R. §152.5.

182. 40 C.F.R. §152.5.

183. *Id*. §152.5(d).

184. 7 U.S.C. §136w(c)(1).

185. It seems somewhat perplexing that an informal interpretation could receive more deference than the PR Notice, which was subject to notice and comment (assuming the PR Notice was entitled to *Skidmore* as opposed to *Chevron* deference). Nevertheless, this is the result of *Mead* and *Thomas Jefferson Univ.* (which has been reaffirmed since *Mead*; *see*, *e.g.*, Wisconsin Dep't of Health & Family Servs. v. Blumer, 534 U.S. 473, 497 (2002)). *See also* United States v. Mead Corp., 533 U.S. 218, 246 (2001) (Scalia, J., dissenting) ("Agencies will now have high incentive to rush out barebones, ambiguous rules construing statutory ambiguities, which they can then in turn further clarify through informal rulings entitled to judicial respect.").

186. 40 C.F.R. §152.5(d).

187. *See*, *e.g.*, *Mead*, 533 U.S. at 230 ("[T]he overwhelming number of our cases applying *Chevron* deference have reviewed the fruits of notice-and-comment rulemaking or formal adjudication.").

188. *See*, *e.g.*, Lars Noah, *Doubts About Direct Final Rulemaking*, 51 Admin. L. Rev. 401, 403 (1999):

> Agencies face significant obstacles when they promulgate regulations. In the last few decades, the courts, Congress, and the executive branch have placed a number of analytical hurdles in the way of informal rulemaking. As a result, notice-and-comment rulemaking has become more formal and cumbersome, and agencies increasingly shy away from using this formerly efficient method for formulating and announcing their rules and enforcement policies.

189. *See supra* notes 156-66 and accompanying text.

190. The AHPA was passed as Subtitle E of the Farm Security and Rural Investment Act of 2002, Pub. L. No. 107-171, 116 Stat. 134; for the legislative history of the AHPA, see H.R. Conf. Rep. No. 107-424, at 388 (2002).

191. *See supra* notes 104-08 and accompanying text.

192. *See*, *e.g.*, John Stauber, *U.S. Needs to Do Right Thing to Stop Mad Cow Disease*, Capitol Times, Jan. 5, 2004, at A9 (criticizing the USDA for taking inadequate steps to deal with the crisis and for a public relations "spin" that is comprised of a "litany of falsehoods" and stating that "mad cows can also seem completely healthy at the time of slaughter, which is why testing all animals must be the goal"); *see also supra* notes 37-39 and accompanying text (stating that the length of the incubation period of the disease in cattle is such that a cow could have a high concentration of prions without outwardly manifesting clinical signs of BSE and that the even longer incubation period in humans could result in a "hidden epidemic").

addition, the USDA's assertion that avoiding blood and central nervous tissue can prevent the disease has been cast into significant doubt by recent scientific studies.[193]

Public fear and ignorance, coupled with the gravity of the symptoms and lethality of the disease, have rendered the typical consumer extremely vulnerable to claims of manufacturers whose products are effective at "killing," "removing," or "inactivating" prions. Additionally, owners of meat processing plants or slaughterhouses face such potentially severe economic consequences that their livelihood may be at stake. As a result, an oversight mechanism needs to be in place to assess the veracity of these claims in order to ensure that the public has access to products that are truly safe and effective.

EPA, in its regulatory authority under FIFRA, is the most natural repository for such an administrative safeguard. Currently, EPA regulates products that make claims to disinfect or sterilize inanimate surfaces from microbes and viruses. Although the causative agent of BSE and vCJD is neither a microorganism nor a virus, as those terms are commonly used by microbiologists, prions act sufficiently like microorganisms and viruses (in that they cause infectious disease), and therefore they should be treated similarly for regulatory purposes.

EPA should have jurisdiction over prionicidal products because these agents will be marketed as disinfectants or sterilants, and the assessment of the safety and efficacy of the product, as well as the truthfulness of the claims made by the manufacturers, will require great scientific expertise. A reading of FIFRA's definition of pest (which does not specifically mention prions) should not foreclose an interpretation including prions. This would hinder attempts to stem the spread of the disease from cattle to humans and would set a regrettable precedent for agency flexibility in the face of cutting-edge advances in science. The term "other microorganism" (in the definition of pest), in addition to specific authority granted to EPA to engage in rulemaking to declare pests, should provide sufficient support for delegation by Congress to EPA to adapt to unforeseen advances in science (such as the characterization of the causative agent of TSEs as prions, something distinct from viruses).

Although the USDA and HHS have had and will continue to have a large regulatory role in responding to the threat of mad cow disease, EPA does occupy a crucial niche in the regulatory sphere and must begin to coordinate with these agencies in order to protect public health. EPA, with its experience in the area of antimicrobial pesticides, is best equipped to assess many policy objectives facing the American government in the wake of the presence of BSE: to prevent the spread of BSE to humans, to prevent chemical poisonings or any other adverse effect from using an unsafe product to eradicate prions, and to prevent meat processing plants or slaughterhouses from shutting down (or any other substantial economic harm resulting from prional contamination). In addition, EPA must implement its statutory charges to coordinate with the USDA to identify pests that must be brought under control, and to coordinate with the Public Health Service to "develop and implement programs to improve and facilitate the safe and necessary use of chemical, biological, and other methods to combat and control" prions.[194]

Regulation of prionicidal products by EPA will result in registered products and prosecution of products that are unregistered, make unapproved claims, or are otherwise misbranded or adulterated. This in turn could result in a greater feeling of trust in the government, which could be critical if more cows test positive or if a potential hidden epidemic does indeed manifest itself. Therefore, the idea of an overly technical reading of FIFRA's definition of pest must yield to Agency flexibility because the protection of public health and the consumer is at stake.

193. *See supra* notes 45-53 and accompanying text.

194. FIFRA §28(d), 7 U.S.C. §136w-3.