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**Bovine Spongiform Encephalopathy and Variant Creutzfeldt-Jakob Disease**

**To the Editor:** The article by Brown et al. (1) contains the statement “it appears likely that changes in the rendering process that had taken place around 1980 allowed the etiologic agent in infected carcasses to survive.” If that is the case, why not revert to the rendering methods used before 1980? That measure would seem more cost-effective than trade embargoes and mass killing of cattle. Meal made from meat and bone was used as a livestock feed additive in many countries without the apparent disastrous effect seen in the United Kingdom. Did the rendering methods remain unchanged in these other countries? Historically, rendering was viewed somewhat differently in continental Europe; the primary purpose was not to make animal feed but to destroy infectious agents, as indicated by the very name of the facility: destruction plant (Destruktionsanstalt). The plants were under governmental inspection and there were mandatory time-temperature requirements for processing meat, bones, and other offal. Temperature requirements varied from 120°C to 140°C, which presumably would reduce if not eliminate prions. It would be of interest to see a description and objective analysis of rendering methods and regulations in various countries before and after 1980. In these times when the concept of hazard analysis and critical control points (HACCP) is gaining popularity, it would seem natural to extend the principle of process control to rendering. One might easily get the impression that policies to control bovine spongiform encephalitis are dominated by some of the stakeholders: the researchers writing their next research grant proposals, the public agencies eager to show prompt response, and those who would like to see their competitors’ beef kept off the market.

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**Mad Cow Disease**

**To the Editor:** In regards to “Bovine Spongiform Encephalopathy and Variant Creutzfeldt-Jakob Disease: Background, Evolution, and Current Concerns” (1), use of the name “variant Creutzfeldt-Jacob disease” (vCJD) for human cases of bovine spongiform encephalopathy (BSE) is regrettable. The disease that occurs in humans exposed to BSE is zoonotic and CJD is not; in addition, the human form of BSE has important clinical, pathologic, and epidemiologic differences from CJD. Continued use of this terminology perpetuates the error.

The fact that 12 years after the feed ban bovine cases continue to occur in the United Kingdom at a much higher rate than in any other country could have two possible causes: inefficient controls or additional routes of transmission. Data on alternative routes of transmission must be evaluated, and important gaps in our understanding of BSE in cattle must be addressed.

Extensive epidemiologic data on BSE in the United Kingdom seemed to clearly implicate the practice of feeding cattle bovine offal as the primary, if not the sole, cause of the spread of BSE. Alternative theories for the origin and spread of BSE, e.g., use of insecticides on bovines or the practice of artificial insemination, appear to have been ruled out quickly on the basis of early epidemiologic data. Confidence in the reliability of these data seems to have been so great as to unduly delay transmission experiments to assess the role of alternative pathways (e.g., artificial insemination) in the propagation of BSE. Is prion protein present in semen or is it not? What if it were present in semen? Would this route lead to shorter incubation than the alimentary route? There seems to be no experimental information on the effect of freezing mutated prion protein to -196°C and placing it, after thawing, directly on the stimulated uterine mucosa.

Although the United Kingdom has acknowledged that compliance with the feed ban improved after 1996, we should not be too eager to accept lack of compliance as the only possible reason for the persistence of BSE. Fifteen years after the BSE epidemic began, it cannot be disputed that the ban on offal feed has interrupted spread of the disease. However, if continued spread by the alimentary route can be excluded as the cause of more recent cases, each of these cases should be carefully evaluated to uncover heretofore unknown or underappreciated routes of transmission.

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