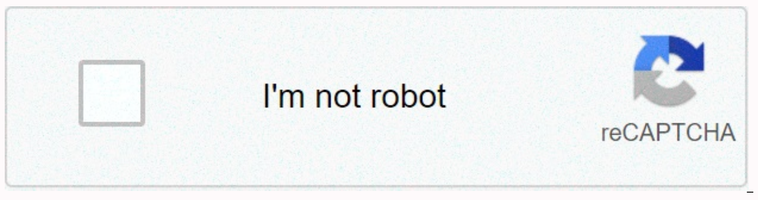

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Bovine spongiform encephalopathy history

Cases of bovine spongiform encephalopathy (BSE) born after the total feed ban. EFSA Biological Hazard Panel (BIOHAZ), Ricci A, Allende A, Bolton D, Chemaly M, Davies R, Fernández Escámez PS, Gironés R, Herman L, Koutsoumanis K, Lindqvist R, Nørrung B, Robertson L, Sanaa M, Simmons M, Skandamis P, Snary E, Speybroeck N, Kuile BT, Threlfall J, Wahlström H, Adkin A, De Koeijer A, Ducrot C, Griffin J, Ortiz Pelaez A, Latronico F, Ru G. EFSA Panel on Biological Hazards (BIOHAZ), et al. EFSA J. 2017 Jul 13;15(7):e04885. doi: 10.2903/j.efsa.2017.4885. eCollection 2017 Jul. EFSA J. 2017. PMID: 32625550 Free PMC article. Alan Colchester and Nancy Colchester (September 3, p.s. 856) suggest a provocative hypothesis according to which bovine spongiform encephalopathy (BSE) in cattle may have occurred in the UK after incorporation into animal food products of human remains imported from India. They argue that this new theory is needed given the lack of supporting evidence for previously proposed theories. These theories assumed primarily an animal origin for BSE, either from scrapie in small ruminants, or from a previously unaccounted form of transmissible spongiform encephalopathy (TSE) in cattle, similar to sporadic Creutzfeldt-Jakob disease (CJD) in humans. In the case of scrapie, currently available experimental data can hardly be considered sufficient to discard this hypothesis. Transmission studies of scrapie to cattle have involved a pool of some uncharacterized U.S. scrapie isolates, and results suggesting that they may not be transferable in cattle per year have been limited to an 8-year follow-up of some (five out of 18) of experimentally challenged cattle. Although oral transmission may require much longer incubation periods, it should be stressed that strengthening and recovering a TSE agent would not necessarily require the development of clinical disease in the infected animals.2Hope J Wood SCER Birkett CR et al. Molecular analysis of sheep prion protein identifies similarities between BSE and an experimental isolate of natural scrapie, CH1641. J Gen Virol. 1999; 80: 1-4 Transmission studies in cattle from British scrapie isolates, especially after oral challenge, have not been reported yet. The diversity of TSE agents in small ruminants is high, and the experiments mentioned above are far too limited to represent the full spectrum of scrapie. Some scrapie isolates showed some, but not all, molecular similarities to BSE in sheep.2Hope J Wood SCER Birkett CR et al. Molecular analysis of sheep prion protein identifies similarities between BSE and an experimental isolate of natural scrapie, CH1641. J Gen Virol. 1999; 80: 1-4, 3Lezmi S Martin S Simon S et al. Comparative molecular analysis of the abnormal prion protein in field scrapie cases and experimental BSE in sheep using westernplot and immunohistochemical method. J Virol. 2004; 78: 3654-3662Crossref PubMed Scopus (69) Google Scholar first appeared with the British CH1641 CH1641 scrapie isolate.2Hope J Wood SCER Birkett CR et al. Molecular analysis of sheep prion protein identifies similarities between BSE and an experimental isolate of natural scrapie, CH1641. J Gen Virol. 1999; 80: 1-4 The experimental isolate CH1641 could not be transmitted in wild-type mice,2Hope J Wood SCER Birkett CR et al. Molecular analysis of sheep prion protein identifies similarities between BSE and an experimental isolate of natural scrapie, CH1641. J Gen Virol. 1999; 80: 1-4 which revealed some distinct differences with the BSE agent isolated from cattle. But as also underlined by Colchesters, features of a TSE agent can be strongly modified by passages in a new host species or serial passages of the same species. It should also be remembered that a case of TSE recently described in a goat showed features indistinguishable from the BSE agent, including after transmission in a panel of mouse lines. Whether such a case arose from contamination of the BSE agent or whether it is due to a small IDISSLARE TSE agent such as the one that could have been at the origin of BSE is unknown. Crucial experiments regarding a potential scrapie origin of BSE are thus lacking and could focus on some specific isolates that may be better candidates, given their close similarities to BSE. Colchester and Colchester also comment on the finding of some atypical cases of TSE in cattle, which revealed some differences with the typical phenotype of BSE. The frequency of such isolates is also probably low. As an illustration, a retrospective study of TSE cases diagnosed in French cattle in 2003 and 2004 identified five atypical cases (three similar to the cases first described in France, and two similar to the cases first described in Italy). These cases were identified through exhaustive tests of around 6 million cattle (healthy slaughtered, self-fallen animals or TSE clinical suspects) and among a total of 196 TSE cases identified during those 2 years. The origin of such atypical phenotypes remains unexplained, but their frequency may well be consistent with the presence of sporadic disease. Interestingly, the situation in cattle, with at least two molecular types different from typical BSE, is reminiscent of the situation in humans, in which there are distinct molecular types of sporadic CJD in addition to the variant CJD (vCJD) linked to BSE. Assuming that such cases could have been the origin of BSE, this would also assume that some changes after recovery at the origin of the BSE epidemic, even in the absence of any species barrier that could benefit such changes. Compared to other theories, the transmission of a bovine TSE agent in cattle would certainly be much easier than that of an agent from other species, especially given the species barrier expected between humans and cattle. We declare that we have no conflict of interest. Published: 28 January 2006DOI: Elsevier Ltd. (2006) Alla Alla Reserved. Gaining access to this article on ScienceDirect Origin of bovine spongiform encephalopathy – The authors' response of Claire Ainsworth and Damian Carrington A few days before Christmas 1984, know David Bee was called to Stent Farm in Sussex to examine a sick cow with strange symptoms – his back was arched and it had lost weight. Within six weeks, cow was 133 dead, after developing head shaking and a loss of coordination. Seven months later, the British Central Veterinary Laboratory diagnosed spongiform encephalopathy. At this time, other cows were also showing symptoms. The epidemic had begun. Four animal species had been affected when the Central Veterinary Office alerted the Ministry of Agriculture, Fisheries and Food (MAFF) in June 1987. Almost a year later, the government set up a task force led by Oxford University professor of zoology, Richard Southwood, to investigate bovine spongiform encephalopathy (BSE) and possible consequences for human health. Ad Cattle cannibalism Shortly after this, the government banned the feeding of cattle with protein derived from other cattle and sheep. Epidemiological studies done by MAFF researchers had identified this cannibalism as the only plausible cause of BSE. At this time it was known that BSE was a prion disease but whether the infectious prion came from scrapie infected sheep or another source is still not known. The Southwood Committee reported in February 1989, recommending a ban on the use of offal in baby food. The British Government went further, and in November 1989 the use of specified bovine offal (SBO) was banned in all human food. In October of the previous year, BSE was shown to be transferable to mice, by injecting contaminated material from brain to brain. But making assumptions based on past experience with scrapie, the Southwood Committee decided that it was unlikely that BSE could be transferred to humans. However, the report added: 'If our assessments of these probabilities are incorrect, the consequences would be very serious. Photo: Nigel

Dickinson/Still Pictures After the Southwood report, the government introduced a mass slaughter programme for all cows suspected of having BSE. However, farmers received only 50% in compensation for each confirmed case. Critics have argued that this policy led to infected animals being sold illegally for human consumption until 1990, when 100% compensation was offered. The first hint that humans might be affected came in 1990, when a Siamese cat named Max fell ill with the cat version of BSE. In the same year, researchers showed that the disease could be transmitted orically to mice. The assumption that BSE could not cross the species barrier proved increasingly uncertain. Despite this, the government assured the public that British beef was safe to eat, with Agriculture Minister John Gummer famously feeding his daughter a beefburger. BSE cases peaked between 1992 and with 3 out of 1000 cows affected. The Incidence Incidence to decline in 1993, and in March, the government's Chief Medical Officer reiterated the 1990 assurance that beef was safe. Research in 1994 showed that cattle could be infected with BSE or less. but it was in 1995 that the human tragedy began. Premature death In May, 19-year-old Stephen Churchill died from a disease similar to Creutzfeldt-Jakob disease. CJD is a rare and sporadic brain disease. It affects up to 60 people a year in the UK, but they are usually over 55 years old. Two other deaths occurred that year, showing similar symptoms to Churchill. James Ironside, a pathologist at the National CJD Surveillance Unit, discovered that the brains of these patients showed unusual spongiform symptoms. He named the condition variant CJD (vCJD). In November 1995, the MAFF researchers informed the Spongiform Encephalopathy Advisory Committee (SEAC), established in May 1990, that random checks on slaughterhouses had revealed that the SBO ban was not strictly enforced. Clothed carcasses in some slaughterhouses were found to contain small pieces of the spinal cord. This meant that potentially highly contagious material could have entered the human food chain for six years after the ban to prevent its introduction. Robert Will, vice-chairman of SEAC, told the BSE inquiry: 'I was appalled. In 1996, there had been a total of eight cases of vCJD, mainly in young people. SEAC advised the government in March that the most likely cause of this new disease was to eat beef products contaminated with BSE agents. Health Minister Stephen Dorrell, related the information to Parliament on the same day. Almost immediately, the European Union banned all exports of British beef. The British Government introduced new measures in an attempt to limit BSE, such as selective slaughter of cattle reared alongside bse between 1989 and 1993. So far there have been 84 confirmed and probable vCJD cases in the UK. But scientists are still waiting to see if this will snowball in a major epidemic, killing hundreds of thousands of people, or whether it will be limited to a few hundred cases. More on these topics: topics: