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EPIDEMIOLOGY OF BOVINE SPONGIFORM ENCEPHALOPATHY

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Epidemiological studies of BSE commenced shortly after its identification in November 1986. These indicated that the first cases occurred in April 1985 and that meat and bone meal, used as a protein supplement in feedstuffs, was the most likely vehicle of a scrapie-like agent. A case control study of calf feeding practices subsequently provided supporting evidence for this hypothesis.

A survey of rendering practices in Great Britain revealed that a reduction in the use of hydrocarbon solvents to maximise the extraction of fat in the early 1980's coincided with the estimated time that cattle were exposed to infection as derived from simulation studies. The feeding of ruminant derived protein to ruminants was prohibited in July 1988, and in September 1990 certain specified bovine offals, which from previous research on sheep scrapie were considered likely to contain high titres of the BSE agent, were banned from all animal feed. The first effects of the ruminant to ruminant feed ban were evident in 1991 and 1992 when the incidence in 2 and 3 year old animals was reduced. Subsequently the national incidence began to decline during 1993. This has been sustained such that at the present time the number of suspect cases reported each week is approximately 100 of which 15% will prove not to be cases of BSE.

An important aspect of the BSE epidemic is that, whatever the origin, the majority of cases have been due to the recycling of infected cattle tissues via meat and bone meal. However, an important aspect of the epidemiology in recent years has been to determine if there are non-feedborne means of transmission of the BSE agent. Investigating the reason for the occurrence of cases of BSE in animals born after the ban has been an important aspect of this.

A within-herd case-control study of animals born after the feed ban was conducted during 1995. The results indicated that neither maternal nor horizontal transmission could explain the majority of cases of BSE in animals born after the ban. Further analyses of the main epidemiological database revealed a change in the geographical incidence of BSE with the northern and eastern regions of England experiencing a relatively greater incidence of BSE in animals born after July 1988. Subsequent studies and investigations have revealed that there have been problems of accidental cross-contamination of cattle feedstuffs with meat and bone meal in feed mills producing feed for both ruminants and mono-gastric animals. The relatively high incidence in the northern and eastern regions of England could therefore be explained because the pig and poultry populations are concentrated in these regions and the risks of accidental cross-contamination were therefore greatest in these areas.! In addition there has been an in complete compliance of the specified bovine offals ban which should have removed the risks from accidental cross-contamination. As a result all mammalian derived protein has been prohibited from all farm animal feedstuffs since March 1996.

Although the case-control study indicated that maternal transmission could not explain the cases of BSE in animals born after the ban, as for all such studies it could never rule out the possibility of maternal transmission. The results indicated that if maternal transmission occurred it would be at a rate between 0 and 13%. Recently a large, long-term cohort study, which was initiated in July 1989, to examine specifically for the occurrence of maternal transmission has been completed. This involved a comparison of the incidence between two cohorts of animals. The members of one cohort were the offspring of confirmed cases of BSE and the members of the other cohort were the offspring of cows which had reached at least six years of age and had not developed clinical signs of BSE. All offspring were allowed to live until their seventh birthday unless death or the need to slaughter them intervened.

The results of this cohort study revealed an excess risk for offspring of BSE-affected dams in developing BSE themselves. The absolute risk difference was 9.6%. However, there was evidence of a period effect in that this difference was much reduced for animals born after the feed ban. This suggested that there may be a genetic influence, either affecting susceptibility to infection with the BSE agent or a more general effect of a proportion of animals having an increased risk of infection because of their food intake.

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The other observation which complicated the interpretation of the findings was that the risk was apparently greater for animals born within 5 months of the onset of clinical signs in their dam. This apparent effect is more consistent with true maternal transmission, but was not fully interpretable from the cohort study as there were very few animals born more than 6 months before the onset of clinical signs in their dams.

As a result of these findings further analyses of both the results of the cohort study and the relevant data contained on the main BSE epidemiological database have been carried out. The results of these have not been conclusive in disentangling the two possible explanations of the basic results of the cohort study, in that they could be a result of a genetic effect or of true maternal transmission, or a combination of these two. However, whatever the underlying explanation is this maternally associated risk factor will neither prolong the BSE epidemic significantly nor affect the future incidence. Therefore the evidence is that the incidence of BSE will continue to decline and will be very low at the beginning of the next millennium.