
**Abstract**

Studies to test the transmissibility of the bovine spongiform encephalopathy (BSE) agent to pigs began in 1989. Parenteral inoculation of the agent by three routes simultaneously (intracranially, intravenously and intraperitoneally) produced disease with an incubation period range of 69–150 weeks. Pre-clinical pathological changes were detected in two pigs killed electively at 105 and 106 weeks post-inoculation. Infectivity was detected by bioassay in inbred mice in the CNS of those pigs that developed spongiform encephalopathy. Infectivity was also found in the stomach, jejunum, distal ileum and pancreas of terminally affected pigs. These findings show that pigs are susceptible to BSE. In contrast, disease failed to occur in pigs retained for 7 years after exposure by feeding BSE-affected brain on three separate days, at 1–2 week intervals. The amounts fed each day were equivalent to the maximum daily intake of meat and bone meal in rations for pigs aged 8 weeks. No infectivity was found in tissues assayed from the pigs exposed orally. This included tissues of the alimentary tract. It is suggested that these pigs did not become infected. The relatively high oral exposure used in these experiments compared with feed-borne exposure in the field may explain the absence of an epidemic of spongiform encephalopathy in domestic pigs concurrent with the BSE epidemic in the UK.

**Conclusion**

The present studies show that, although pigs are susceptible to BSE when injected by combined i.c., i.v. and i.p. routes, there was no evidence of transmission after exposure by feeding three doses of BSE-infected brain in amounts equivalent to the maximum daily intake of MBM formerly used in commercial pig rations. The simplest explanation of this finding is that the effective exposure of pigs by the oral route was insufficient to establish infection. This explanation provides an understanding of why repeated primary exposures of commercial pigs to BSE, together with the considerable potential for pig-to-pig recycling of infection (until April 1996), has not resulted in natural cases of TSE in pigs. These observations are in contrast to the susceptibility of cattle to oral infection with gram quantities of BSE-affected brain and to the major feedborne epidemic in the UK.