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Evidence That Transmissible Mink Encephalopathy Results from Feeding Infected Cattle

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ABSTRACT

Epidemiologic investigation of a new incidence of transmissible mink encephalopathy (TME) in Stetsonville, Wisconsin suggests that the disease may have resulted from feeding infected cattle to mink. This observation is supported by the transmission of a TME-like disease to experimentally inoculated cattle, and by the recent report of a new bovine spongiform encephalopathy in England.

INTRODUCTION

Transmissible mink encephalopathy (TME) was first reported in 1965 by Hartsough and Burger who demonstrated that the disease was transmissible with a long incubation period, and that affected mink had a spongiform encephalopathy similar to that found in scrapie-affected sheep (Hartsough and Burger, 1965; Burger and Hartsough, 1965). Because of the similarity between TME and scrapie, and the subsequent finding that the two transmissible agents were indistinguishable (Marsh and Hanson, 1969), it was concluded that TME most likely resulted from feeding mink scrapie-infected sheep.

The experimental transmission of sheep scrapie to mink (Hanson et al., 1971) confirmed the close association of TME and scrapie, but at the same time provided evidence that they may be different. Epidemiologic studies on previous incidences of TME indicated that the incubation periods in field cases were between six months and one year in length (Hartsough and Burger, 1965). Experimentally, scrapie could not be transmitted to mink in less than one year.

To investigate the possibility that TME may be caused by a particular strain of scrapie which might be highly pathogenic for mink, 21 different strains of the scrapie agent, including their sheep or goat sources, were inoculated into a total of 61 mink. Only one mink developed a progressive neurologic disease after an incubation period of 22 months (Marsh and Hanson, 1979). These results indicated that TME was either caused by a strain of sheep scrapie not yet tested, or was due to exposure to a scrapie-like agent from an unidentified source.

OBSERVATIONS AND RESULTS

A New Incidence of TME. In April of 1985, a mink rancher in Stetsonville, Wisconsin reported that many of his mink were "acting funny", and some had died. At this time, we visited the farm and found that approximately 10% of all adult mink were showing typical signs of TME: insidious onset characterized by subtle behavioral changes, loss of normal habits of cleanliness, deposition of droppings throughout the pen rather than in a single area, hyperexcitability, difficulty in chewing and swallowing, and tails arched over their backs like squirrels. These signs were followed by progressive deterioration of neurologic function beginning with locomotor incoordination, long periods of somnolence in which the affected mink would stand motionless with its head in the corner of the cage, complete debilitation, and death. Over the next 8-10 weeks, approximately 40% of all the adult mink on the farm died from TME.

Since previous incidences of TME were associated with common or shared feeding practices, we obtained a careful history of feed ingredients used over the past 12-18 months. The rancher was a "dead stock" feeder using mostly (>95%) downer or dead dairy cattle and a few horses. Sheep had never been fed.

Experimental Transmission. The clinical diagnosis of TME was confirmed by histopathologic examination and by experimental transmission to mink after incubation periods of four months. To investigate the possible involvement of cattle in this disease cycle, two six-week old castrated Holstein bull calves were inoculated intracerebrally with a brain suspension from affected mink. Each developed a fatal spongiform encephalopathy after incubation periods of 18 and 19 months.

DISCUSSION

These findings suggest that TME may result from feeding mink infected cattle and we have alerted bovine practitioners that there may exist an as yet unrecognized scrapie-like disease of cattle in the United States (Marsh and Hartsough, 1986). A new bovine spongiform encephalopathy has recently been reported in England (Wells et al., 1987), and investigators are presently studying its transmissibility and possible relationship to scrapie. Because this new bovine disease in England is characterized by behavioral changes, hyperexcitability, and aggressiveness, it is very likely it would be confused with rabies in the United States and not be diagnosed. Presently, brains from cattle in the United States which are suspected of rabies infection are only tested with anti-rabies virus antibody and are not examined histopathologically for lesions of spongiform encephalopathy.

We are presently pursuing additional studies to further examine the possible involvement of cattle in the epidemiology of TME. One of these is the backpassage of our experimental bovine encephalopathy to mink. Because there are as yet no agent-specific proteins or nucleic acids identified for these transmissible neuropathogens, one means of distinguishing them is by animal passage and selection of the biotype which grows best in a particular host. This procedure has been used to separate hamster-adapted and mink-adapted TME agents (Marsh and Hanson, 1979). The intracerebral backpassage of the experimental bovine agent resulted in incubations of only four months indicating no de-adaptation of the Stetsonville agent for mink after bovine passage. Mink fed infected bovine brain remain normal after six months. It will be essential to demonstrate oral transmission from bovine to mink if this proposed epidemiologic association is to be confirmed.

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