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Bovine Spongiform Encephalopathy

Bovine spongiform encephalopathy (BSE) is a relatively new disease found primarily in cattle. This disease of the bovine breed was first seen in the United Kingdom in November 1986 by histopathological examination of affected brains (Kimberlin, 1993) . From the first discovery in 1986 to 1990 this disease developed into a large-scale epidemic in most of the United Kingdom, with very serious economic consequences (Moore, 1996).

BSE primarily occurs in adult cattle of both male and female genders. The most common age at which cows may be affected is between the ages of four and five (Blowey, 1991). Due to the fact that BSE is a neurological disease, it is characterized by many distinct symptoms: changes in mental state 'mad-cow', abnormalities of posture, movement, and sensation (Hunter, 1993). The duration of the clinical disease varies with each case, but most commonly lasts for several weeks. BSE continues to progress and is usually considered fatal (Blowey, 1991).

After extensive research, the pathology of BSE was finally determined. Microscopic lesions in the central nervous system that consist of a bilaterally symmetrical, non-inflammatory vacuolation of neuronal perikarya and grey-matter neuropil was the scientists' overall conclusion (Stadthalle, 1993). These lesions are consistent with the diseases of the more common scrapie family. Without further investigation, the conclusion was made that BSE was a new member of the scrapie family (Westgarth, 1994).

Transmission of BSE is rather common throughout the cattle industry. After the incubation period of one to two years, experimental transmission was found possible by the injection of brain homogenates from clinical cases (Swanson, 1990). This only confirmed that BSE is caused by a scrapie-like infectious agent.

How does the transmission become so readily available among the entire United Kingdom feedlot population? Studies showed that the mode of infection was meat and bone meal that had been incorporated into concentrated feedstuffs as a protein-rich supplement (Glausiusz, 1996). It
is thought that the outbreak was started by a scrapie infection of cattle, but the subsequent course of the epidemic was driven by the recycling of infected cattle material within the cattle population (Lyall, 1996). Although the average rate of infection is very low, the reason why this led to such a large number of BSE cases is that much of the United Kingdom dairy cattle population was exposed for many, continuous years (Kimberlin, 1993).

To help control the outbreak, the British government in 1988 introduced a ban on the feeding of ruminant protein to other ruminant animals (Lacey, 1995). Such knowledge for the pathogenesis of the BSE disease shows precisely the actions that must be taken in order to control and minimize the risk of infection in healthy cattle around the world (Darnton, 1996).

The appearance of BSE has made a sizable impact throughout much of the world even though few countries, other than the United Kingdom, have experienced positive cases (Burton, 1996). The scare of an outbreak in other countries has led to a great disruption in the trade economy, as well as other factors concerning each of the country's general welfare. However, a rapid increase in the understanding of the disease over the last four years leaves few unanswered questions of major importance (Masood, 1996). BSE has been prevented, controlled and eradicated.

As mentioned, BSE was first recognized in the United Kingdom and it is only there that a large-scale epidemic has occurred (Burton, 1996). By the end of 1990 well over 20,000 cases of BSE had been has been confirmed in England, Scotland, and Wales (Filders, 1990). The deadly epidemic started simultaneously in several parts of the country and cases have been distributed over a wide area ever since (Cowell, 1996).

Besides the United Kingdom, cases of BSE have occurred in the Republic of Ireland. Some of these cases were associated with the importation of live animals,

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meat, and bone meal from the United Kingdom (Cherfas, 1990).

Two cases of BSE have also occurred in cattle from the country of Oman. These animals were thought to be part of a consignment of fourteen pregnant heifers imported from England in 1985. Various cases have also been confirmed in Europe, Switzerland, and France (Patel, 1996).

The economic consequences of BSE in the United Kingdom have been considerable. At the beginning, the only losses due to BSE were those directly associated with the death or slaughter of BSE infected animals (Cowell, 1996). In August 1988, a slaughter policy with part compensation was introduced to help lessen the burden on individual farmers. As the number of BSE cases increased , and more farmers were experiencing a second case, full compensation was introduced in February 1990 (Moore, 1996). In 1989 alone over 8,000 suspected and confirmed cases of BSE were slaughtered. The compensation costs for the year were well over 2.8 million pounds and the slaughter costs amounted to 1.6 million pounds (Cockburn, 1996).

Once studies had identified meat and bone meal as the vehicle of infection, the United Kingdom Government banned the feeding of all ruminant-derived protein to ruminants (Glausiusz, 1996). This had an immediate impact on the cattle industry in terms of reduced exports and domestic sales of meat and bone meal (Hager, 1996). In 1990, the Commission of the European Communities banned the importation, from the United Kingdom, of all live cattle born before July 1988. Panic throughout the world caused many countries to entirely ban the importation of all live cattle from the United Kingdom. Some even went as far as to ban the importation of milk and milk products (Hunter, 1993).

BSE has also had economic consequences in the human food industries. In the winter of 1989/1990, the United Kingdom Government banned the use for human food of certain specified bovine meats which contained suspicious amounts of BSE (Cockburn, 1996). This ban was introduced as a precautionary measure to help ensure the risks to public health from BSE were kept to a minimum.

Most of the information concerning BSE has come from extensive studies of the scrapie agent. The agent is small enough to pass through bacteriological filters, thus demonstrating that it is virus-like or subviral in size (Kimberlin, 1993). Unfortunately, the agent has other properties which are atypical of viruses. The first contradiction is that infectivity is highly resistant to many physicochemical treatments, such as heat, and exposure to ionizing or ultra violet radiation (Swanson, 1990). Second, the disease does not induce an immune response from the host (Stadthalle, 1993). These two controversies along with the long incubation period explain why the scrapie group of agents have long been known as the "unconventional slow virus" (Westgarth, 1994).

BSE is clearly not a disease of genetic origin. It has occurred in the majority of United Kingdom dairy breeds and their crosses, in the proportion expected from their representation in the national herd (Kimberlin, 1993). Analysis of available pedigrees excludes a simple Mendelian pattern of inheritance as the sole cause of the disease. Studies further showed that the occurrence of BSE was not associated with the importation of cattle, the use of semen, or the movement of breeding animals between herds (Hunter, 1993).

By examining the epidemic curve, one can deduce that the disease is characteristic to that of an extended-source epidemic. By a simple process of elimination, the only common factor to be identified was the feeding of proprietary feedstuffs (Darnton, 1996). Commercial calf pellets, cow cakes, or protein supplements to home mixed rations have been fed to all cases where the infectious BSE disease is subsequently present. The balance of evidence shows that meat and bone meal is the primary vehicle of infection (Lacey, 1995).

As previously explained, it is now assumed that scrapie was the original cause of the BSE epidemic. It is very likely that the epidemic was started by one scrapie strain that is common in different breeds of sheep, or possibly, a few strains that behave in a similar manner when crossing the sheep-to-cattle species barrier (Hunter, 1993). However, the continued exposure of cattle to sheep scrapie was not the ultimate driving force of the epidemic. On the contrary, the epidemic

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would inevitably have been amplified into a severe outbreak by the subsequent recycling, through meat and bone meal, of infected cattle material within the cattle population (Westgarth, 1994). Because of the length of BSE incubation periods, recycling would have already been established as the pattern for the epidemic long before BSE was even recognized (Cherfas, 1990).

After a comprehensive study of nearly 200 cases of BSE, scientists were able to conclude that three significant clinical signs were present.

Changes in mental state were observed, most commonly seen as apprehension, frenzy and nervousness when confronted by doorways and other entrances.

Abnormalities of posture and movement occurred in 93 percent of the cases. The most common manifestations were hind-limb ataxia, tremors, and falling.

Changes in sensation were a feature of 95 percent of all cases. The most striking evidence was continuous hyperaesthesia, to both touch and sound.

These three most common clinical signs are consistent with a diffuse central nervous system disorder (Stadthalle, 1993). Other common signs were loss of body condition (78 percent), live weight loss (73 percent), and a reduced milk yield (70 percent). At some stage in the clinical course, about 79 percent of all cases showed one of the above general signs along with signs in each of the three neurological categories previously listed (Swanson, 1990).

Unfortunately, the slaughter of the great majority of affected animals becomes necessary at an early stage because of unmanageable behavior and injury from repeated falling and uncontrollable behavior (Cowell, 1996). The duration of the clinical disease, from the earliest signs to death or slaughter, can range from under two weeks to as long as a year. The average period is about one to two months (Lyall, 1996).

BSE resembles other members of the scrapie family in not having any gross pathological lesions associated with disease. Characteristic histo-pathological changes are found in the nervous system (Kimberlin, 1993). In common with the other diseases in the scrapie family, BSE has a distinctive non-inflammatory pathology with three main features.

The most important diagnostic lesion is the presence of bilaterally symmetrical neuronal vacuolation, in processes and in soma.

Hypertrophy of astrocytes often accompanies vacuolation.

Cerebral amyloidosis is an inconstant histopathological feature of the scrapie family of diseases.

At times, only one of the above will occur in an infected animal, while more often a combination of the three will occur (Swanson, 1990).

Unfortunately, there are no routine laboratory diagnostic tests to identify infected cattle before the onset of clinical disease. The diagnosis of BSE therefore depends on the recognition of clinical signs and confirmation by histological examination of the central nervous system (Westgarth, 1994). A clinical diagnosis can also be confirmed by simple electron microscope observations, biochemical detection of SAF, or the constituent protein PrP (Hunter, 1996).

At present, vaccination is not an appropriate way of preventing any of the diseases in the scrapie family. There is no known protective immune response to infection for a vaccine to enhance (Blowey, 1991). However, BSE is obviously not a highly contagious disease and it can be prevented by other simple means because the epidemiology is also relatively simple.

Restrictions on trade in live cattle
Restrictions on trade in meat and bone meal
Sterilization of meat and bone meal
Restricted use of meat and bone meal
Minimizing exposure of the human population
Minimizing the exposure of other species (Moore, 1996)

A great deal of concern, much of it avoidable, has been expressed over the

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possible public health consequences of BSE. This is understanding given that the scrapie family of diseases include some that affect human beings (Patel, 1996). As a result of research, the circumstances in which BSE might pose a risk to public health can be defined quite precisely, and simple measures have been devised to prevent this risk (Kimberlin, 1993). It is important to emphasize that any primary human exposure would still be across a species barrier and there would be no recycling of food-born infection in the human population, as happened with kuru and with BSE in cattle (Patel, 1996). The logical way to address this risk is to make sure that exposure to BSE is kept to a bare minimum.

There are two scenarios for the future course of BSE. The first is that BSE, like TME and kuru, is a dead-end disease. If this is true and meat and bone meal was the sole source of the infection, then removing this source would be sufficient for the eventual eradification of BSE from the United Kingdom (Hager, 1996). The alternative scenario is that there are natural routes of transmission of BSE and that the outbreak could turn into an endemic infection of cattle the way scrapie is in sheep (Burton, 1996). To sustain BSE infection in the cattle population requires that each breeding cow is replaced by at least one infected female calf, which then transmits infection to at least one of her offspring. For BSE to become an endemic, the number of infected cattle would need to increase by horizontal spread as seen in scrapie (Masood, 1996). The essential prerequisite for controlling such a deadly disease is through good breeding and movement records which are currently being compiled in the United Kingdom following recent legislation (Stadthalle, 1993). Meanwhile the precautionary measures to safeguard other species, including human beings, are already in place and refined to meet today's needs.