PRION DISEASES IN ANIMALS

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Prion diseases are transmissible neurodegenerative conditions affecting human and a wide range of animal species. The pathogenesis of prion diseases is associated with the accumulation of aggregates of misfolded conformers of host-encoded cellular prion protein (PrP^C). Animal prion diseases include scrapie of sheep and goats, bovine spongiform encephalopathy (BSE) or mad cow disease, transmissible mink encephalopathy, feline spongiform encephalopathy, exotic ungulate spongiform encephalopathy, chronic wasting disease of cervids and spongiform encephalopathy of primates.

Prion diseases occur in many animal species, most notably in ruminants. While scrapie in sheep has been recognised for three centuries and goat scrapie has been recognised for decades, BSE in cattle is a relatively novel disease which was first diagnosed in the UK in the mid 1980s. BSE was most likely caused through dietary exposure to animal feed contaminated with prions and disease was subsequently transmitted to people. The BSE epidemic is almost at an end, but the recent identification of so called atypical forms of BSE and scrapie pose many questions about the possible spectrum of prion diseases in animals and their transmissibility to other species, including humans.The pathogenesis of animal prion diseases has been studied both in natural infections and in experimental animal models. Detection of infectivity is greatly helped by suitable rodent models, in particular transgenic mice. Clinically infected animals show characteristic neuropathology in the brain and spinal cord which is accompanied by the accumulation of a conformationally altered, protease-resistant host protein.

The main diagnostic histology in the spongiform encephalopathies consists of a degenerative and usually symmetrical vacuolation of neurons and a spongiform lesion in the neuropil. Sometimes there can be asymmetry. This pathology is usually confined to grey matter, but an additional white matter vacuolation is sometimes typical. The degeneration can progress to neuronal necrosis, with reactive glial changes. Photoreceptor loss in the retina occurs in some experimental models. Cerebral amyloidosis is conspicuous in many types of scrapie-like pathology in animals, but is sometimes not recognised or may be absent

Transmission experiments

In the 1980s, Williams and Young demonstrated that CWD was transmissible by intracerebral(IC) inoculation of CWD brain homogenate into deer with an incubation period of 17–21 months[19]. Recently, experiments demonstrated that oral exposure of mule deer fawns to CWD using brain homogenate results in detection of PrP in lymphoid tissues (retropharyngeal lymph node, tonsil, Peyer's patches, ileocaecal lymph node) within 6 weeks' postexposure[27] and clinical disease with an incubation period of 15–25 months (ES Williams and MW Miller, unpublished findings). PrP^{CWD}accumulates within the lymphoid germinal centres in a manner similar to vCJD and scrapie. Phenotyping studies have revealed that within germinal centres, PrP^{CWD}accumulates on cell membranes of follicular dendritic cells and/or B cells and within the cytoplasm of tangible body macrophages[28]. In advanced cases of CWD in naturally infected deer, PrP^{CWD} accumulates in tonsil, spleen, Peyer's patches, and lymph nodes throughout the body, as well as nerves and ganglia, pancreatic islets, and adrenal medulla.

Prion diseases occur in many animal species, most notably in ruminants, also deasese are a common feture in the organism our aim is to decrease the numbers of ill animals.