

Transmissible Mink Encephalopathy

*Mink Spongiform Encephalopathy,
Mink Scrapie*

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Importance

Transmissible mink encephalopathy (TME) is a progressive and fatal neurodegenerative disease that affects ranched mink (*Neovison (Mustela vison)*). Most or all of the adult animals on a ranch may be affected, and once an animal becomes symptomatic, death is inevitable. This disease is still poorly understood. It is very rare, with only a few outbreaks reported in the U.S. and other countries. Outbreaks seem to result from feeding contaminated food containing prions to mink; however, the origin of these prions is unknown. Recent evidence suggests they might be an atypical variant of the bovine spongiform encephalopathy (BSE) agent.

Etiology

TME is a member of the transmissible spongiform encephalopathies (TSEs), a group of neurodegenerative disorders caused by prions, infectious proteins that appear to replicate by converting a normal cellular protein into copies of the prion. The cellular protein, which is called PrP^c, is found on the surface of neurons. Pathogenic isoforms of PrP^c are designated PrP^{res}. (The 'res' refers to the proteinase K-resistant nature of prions, compared to normal PrP^c). PrP^{TME} or PrP^{TSE} are other names for this protein. Prions that cause different diseases (e.g. BSE or scrapie) are considered to be different strains of PrP^{res}.

Mink seem to acquire the TME prion when they eat contaminated feed, but the origin of this agent is still uncertain. TME could be caused by the scrapie prion, an agent found in sheep and goats, although this currently seems unlikely. Intracerebral inoculation of mink with U.S. (but not U.K) strains of scrapie can cause neurological signs, but sheep tissues were not fed to mink in all TME outbreaks, and mink fed scrapie prions in experiments did not become ill. Likewise, mink were susceptible to the cervid-associated chronic wasting disease (CWD) prion by intracerebral but not oral inoculation. Bovine spongiform encephalopathy (BSE), which occurs in cattle and some other species, may be a more likely source of TME. Epidemiological investigations suggest that some TME outbreaks were linked to feeding tissues from non-ambulatory ("downer") or dead cattle, and mink infected orally with BSE prions develop neurological disease. A study in transgenic mice suggests that the TME agent most closely resembles L-type BSE, an atypical BSE prion that has been reported rarely in cattle and has a lower molecular mass than the classical BSE prion. L-BSE is thought to arise spontaneously in cattle, similarly to some prion diseases in other species (e.g., spontaneous Creutzfeldt-Jakob disease in humans).

Species Affected

TME has been reported only in ranched mink; however, experimental infections can be established in other species. Raccoons (*Procyon lotor*) are readily infected by oral as well as parenteral inoculation. Species that have been infected by intracerebral inoculation include striped skunks (*Mephitis mephitis*), ferrets (*Mustela putorius*), American pine marten (*Martes americana*), beech marten (*Martes foina*), cattle, sheep, goats, hamsters and various nonhuman primates, such as rhesus macaques (*Macaca mulatta*), cynomolgus macaques (*Macaca fascicularis*), stump-tailed macaques (*Macaca arctoides*) and squirrel monkeys (*Saimiri* sp.). The natural susceptibility of these species is unknown, as intracerebral inoculation bypasses normal species barriers to prions. Cattle, sheep, goats, hamsters, raccoons, striped skunks and squirrel monkeys are relatively easy to infect by the intracerebral route, but the long incubation period in ferrets suggests that a species barrier exists. Wild type mice are not susceptible to TME.

Zoonotic potential

Although nonhuman primates and humanized mice develop neurological disease after intracerebral inoculation of TME prions, there is no evidence that this agent can normally be transmitted to humans. However, caution is warranted with any poorly understood prion disease, particularly one that may be linked to BSE; ingestion of classical BSE prions can cause variant Creutzfeldt–Jakob disease (vCJD) in humans.

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Geographic Distribution

Several outbreaks of TME were reported in the United States between 1947 and 1985; no cases have been documented in the U.S. since that time. Many of the incidents occurred in Wisconsin, but ranches in Minnesota and Idaho were also affected during some years. TME has also been seen in ranch-raised mink in Canada, Finland, Germany and the former U.S.S.R.

Transmission

TME is thought to be transmitted orally. Outbreaks seem to occur when mink ingest prions in their feed. Studies in hamsters suggest that wounds on the tongue may facilitate transmission. TME prions can also be transmitted experimentally between mink by intramuscular injection, and some authors have suggested that breaks in other mucous membranes or skin might provide additional entry points. During an outbreak, TME might be able to spread between animals in the same cage by cannibalism: TME prions have been reported in some lymphoid tissues including the spleen and mesenteric and retropharyngeal lymph nodes, the thymus, kidney, liver, intestine and salivary gland of experimentally infected mink that had prions in the CNS. Nevertheless, mink-to-mink transmission is thought to be rare; in at least one outbreak, kits sharing a cage with their dam did not become infected. In addition, adult mink are usually housed individually in cages, making mink-to-mink transmission unlikely. TME is not known to be transmitted vertically, and mink born during one outbreak had no signs of disease the following year.

Whether TME prions can survive long-term in the environment is unknown. Other prions are reported to remain infectious for 2 to 3 years and possibly longer; however, TME does not seem to recur during subsequent years on the same farm, and hamster-adapted TME was reported to degrade more quickly than some other prions (e.g., CWD) in some laboratory experiments. Prions can remain infectious after passage through the digestive systems of birds (crows) and mammals (coyotes).

The distribution of prions in tissues that may be fed to mink

Currently, ruminants and ranched cervids are the only livestock known to be natural hosts for prions. The highest concentrations of classical and atypical BSE, scrapie, CWD and other prions normally occur in the central nervous system (CNS); however, these agents may also be found in various lymphoid tissues (especially those associated with the intestinal tract), peripheral nerves, adrenal gland and some other tissues, depending on the host species, specific prion and stage of the disease. L-BSE is still incompletely understood, but in addition to the CNS, small amounts of prion protein or infectivity have been detected in peripheral nerves, nerve ganglia, sensory receptors (muscle spindles) and the adrenal gland. One study found L-BSE in the muscles of infected cattle by immunostaining, and in

muscle homogenates with a highly sensitive mouse bioassay. Even very small amounts of prions can infect an animal, and meat can become contaminated with prions from other tissues during slaughter or processing. Cooking or rendering does not destroy these agents.

Disinfection

Complete decontamination of prion-contaminated tissues, surfaces and environments can be difficult. These agents are very resistant to most disinfectants, including formalin and alcohol. They are also resistant to heat, ultraviolet radiation, microwave irradiation and ionizing radiation, particularly when they are protected in organic material or preserved with aldehyde fixatives, or when the prion titer is high. Prions can bind tightly to some surfaces, including stainless steel and plastic, without losing infectivity. Prions bound to metal seem to be highly resistant to decontamination.

Few prion decontamination techniques have been published and confirmed to be effective for routine use. A 1-2 N sodium hydroxide solution, or a sodium hypochlorite solution containing 2% available chlorine (20,000 ppm), has traditionally been recommended for equipment and surfaces. Surfaces should be treated for more than one hour at 20°C (68°F). Overnight disinfection is recommended for equipment. Cleaning before disinfection removes organic material that may protect prions. Experimentally, some milder treatments have also been effective against certain prions, under some conditions. They include a specific phenolic disinfectant, various alkaline and enzymatic detergents (although the efficacy of specific agents within these classes varies), hydrogen peroxide gas plasma, radiofrequency gas plasma, sodium dodecyl sulfate plus acetic acid, copper plus hydrogen peroxide, and others. New commercial decontaminants have been developed for prions, though published tests of their efficacy vary. Some laboratories pre-treat tissues with formic acid (98%) to decrease infectivity before sectioning tissue blocks.

Physical inactivation of prions (e.g., on surgical instruments) can be carried out by porous load autoclaving at 134°C (273°F) for 18 minutes at 30 lb/in². Tissue films containing prions are more difficult to decontaminate by steam after they have dried, and human guidelines for surgical instruments recommend that, after use, they be kept moist or wet until decontamination is performed. The cleaning agent used before autoclaving should also be chosen with care, as certain agents (e.g., some enzymatic treatments) can increase the resistance of prions to steam sterilization. Some types of samples cannot be decontaminated effectively even at the recommended temperatures. For example, tissue macerates containing BSE were reported to require wet heat sterilization at ≥ 155°C (311°F) for 20 minutes, and resisted even these temperatures if the sample was dehydrated. Dry heat is less effective than moist heat; hamster-adapted scrapie prions can survive dry heat at temperatures as high as 360°C

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(680°F) for an hour, and one group even reported that infectivity survived incineration at 600°C (1112°F). A combination of chemical and physical decontamination can be more effective than either procedure alone, and effective combinations of chemical agents (e.g., NaOH) and autoclaving have been published. Even the harshest combination of chemical and physical disinfection is not guaranteed to destroy all prions in all types of samples.

Anecdotal evidence and a recent study on scrapie suggest that decontaminating contaminated facilities, especially sites such as animal pens, can be very difficult. Incineration is commonly used for carcasses, but two studies found that composting may reduce or eliminate prions in tissues, while another suggested that soil microorganisms might degrade prions in buried carcasses. Repeated cycles of wetting and drying were also found to reduce the infectivity of a soil sample containing hamster-adapted TME.

Incubation Period

The estimated incubation period is 6 to 12 months in ranchered mink. Experimental infections can become apparent as early as 5 months. Adult raccoons infected experimentally by the oral route became ill in approximately 10 months.

Clinical Signs

Transmissible mink encephalopathy causes neurological signs including behavioral changes. The early clinical signs can be subtle, and may include difficulty eating and swallowing, and changes in normal grooming behavior. Affected mink often soil the nest or scatter feces in the cage. Later, animals may become hyperexcitable and bite compulsively. Affected mink often carry their tails arched over their backs like squirrels. Incoordination, circling, clenching of the jaw and self-mutilation (particularly of the tail) may also be seen. When death is imminent, mink tend to become somnolent and unresponsive; convulsions can occur but are not common. Once the clinical signs appear, TME is always progressive and fatal. Death usually occurs within 2-8 weeks.

In one experiment, mink inoculated orally with the classical BSE agent developed a fatal neurological disease that resembled TME; however, the animals tended to become unusually docile rather than aggressive. There are no reports of the clinical signs after oral inoculation of mink with L-BSE.

Raccoons experimentally inoculated with TME prions developed neurological signs including lethargy, abnormal responses to external stimuli, altered behavior and incoordination.

Post Mortem Lesions [Click to view images](#)

No characteristic gross lesions are found in animals with TME; however, the carcass may be dehydrated and the fat deposits can be depleted. The typical histopathologic

lesions are confined to the CNS. Neuronal vacuolation and non-inflammatory spongiform changes in the gray matter are pathognomonic. Astrocytosis can be seen, but amyloid plaques are not found.

Diagnostic Tests

Laboratory tests

Transmissible spongiform encephalopathies are usually diagnosed by detecting prions in the CNS, at necropsy, with immunoblotting (Western blotting) or immunohistochemistry. There is no live animal test. Prions have sometimes been detected in other tissues of experimentally infected mink, but only after prions are found in the brain. One study examined two ELISA-based rapid tests used for TSE surveillance, and found that one assay appeared to detect TME; however, another test missed some infected tissue samples (see Jenelle et al., in the reference list). Histological examination of the brain can be very helpful, although it is not generally used as the sole confirmatory test, but some mink (e.g., aged mink of the Chediak-Higashi genotype) may have few or no spongiform changes.

Highly sensitive assays, including protein misfolding cyclic amplification (PMCA) and quaking-induced conversion (QuIC), can be used to detect small quantities of prions, but are not widely available and have not been validated for TME. These techniques detect tiny amounts of prions by their ability to convert PrP^c (the normal cellular protein) into prions *in vitro*. TME can also be detected by inoculation into mice (rodent bioassays); however, an incubation period of several months makes this technique impractical for routine diagnosis. Serology is not useful, as antibodies are not made against prions.

Treatment

There is currently no effective treatment for TME or any other prion disease.

Control

Disease reporting

Veterinarians who encounter or suspect TME should follow their national and/or local guidelines for disease reporting. In the U.S., state or federal veterinary authorities should be informed immediately.

Prevention

There is no vaccine or treatment for TME. Feed that may contain prions, particularly BSE or scrapie, should not be given to mink. Tissues from non-ambulatory cattle should be avoided in mink feed, unless the carcass was tested for BSE. Complete avoidance is generally necessary, as cooking or rendering cannot completely inactivate prions. Mink may be able to acquire TME by cannibalizing infected animals; however, because mink are typically housed in individual cages, this is unlikely to be a concern

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in most circumstances. Kits housed with their dam do not appear to become infected.

Because the TME agent may be related to BSE, scrapie or other ruminant prions, mink tissues are not allowed in ruminant feed in the U.S.

Although there is no evidence that the TME agent is transmitted to mink from the environment, contamination should be avoided whenever possible.

Morbidity and Mortality

TME is very rare, and has only been seen in ranched mink. Several outbreaks have been reported in the U.S. The first occurred in 1947, on a ranch in Wisconsin. Another facility in Minnesota that received animals from this ranch was also affected. Outbreaks were reported on five ranches in Wisconsin in 1961; in Idaho, Minnesota and Wisconsin in 1963; and on a single ranch in Wisconsin in 1985. In at least one case, multiple facilities were linked by a common food source. TME has not been reported in wild mink populations.

Only adult mink are usually affected; in at least one outbreak, kits housed with their dams and eating the same diet were completely spared. TME is always fatal once the clinical signs appear. The mortality rate on a ranch is variable, but can be as high as 60-90%; in one outbreak, all of the adult mink died. An outbreak may last several months, but the disease does not seem to recur.

Public Health

Caution is warranted with any poorly understood prion disease, particularly one that may be linked to BSE; ingestion of classical BSE prions can cause variant Creutzfeldt–Jakob disease (vCJD) in humans. Standard precautions when working with prions include the use of protective clothing and the avoidance of penetrating injuries, contamination of abraded skin, and ingestion. A negative pressure laminar flow hood should be considered for tissue manipulations. Because prions may be able to survive in the environment for years and are difficult to disinfect, precautions should be taken to avoid contamination of surfaces and equipment. Disposable plastic-coated paper sheets can be used to protect tables and other surfaces. Disposable instruments and work clothing can also be used. No vaccine is available.

Internet Resources

The Merck Veterinary Manual

http://www.merckvetmanual.com/mvm/index.jsp?cfile=htm/bi/mvm_bkindex.htm

European Commission. TSE/BSE

http://ec.europa.eu/food/food/biosafety/tse_bse/index_en.htm

World Organization for Animal Health (OIE)

<http://www.oie.int>

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