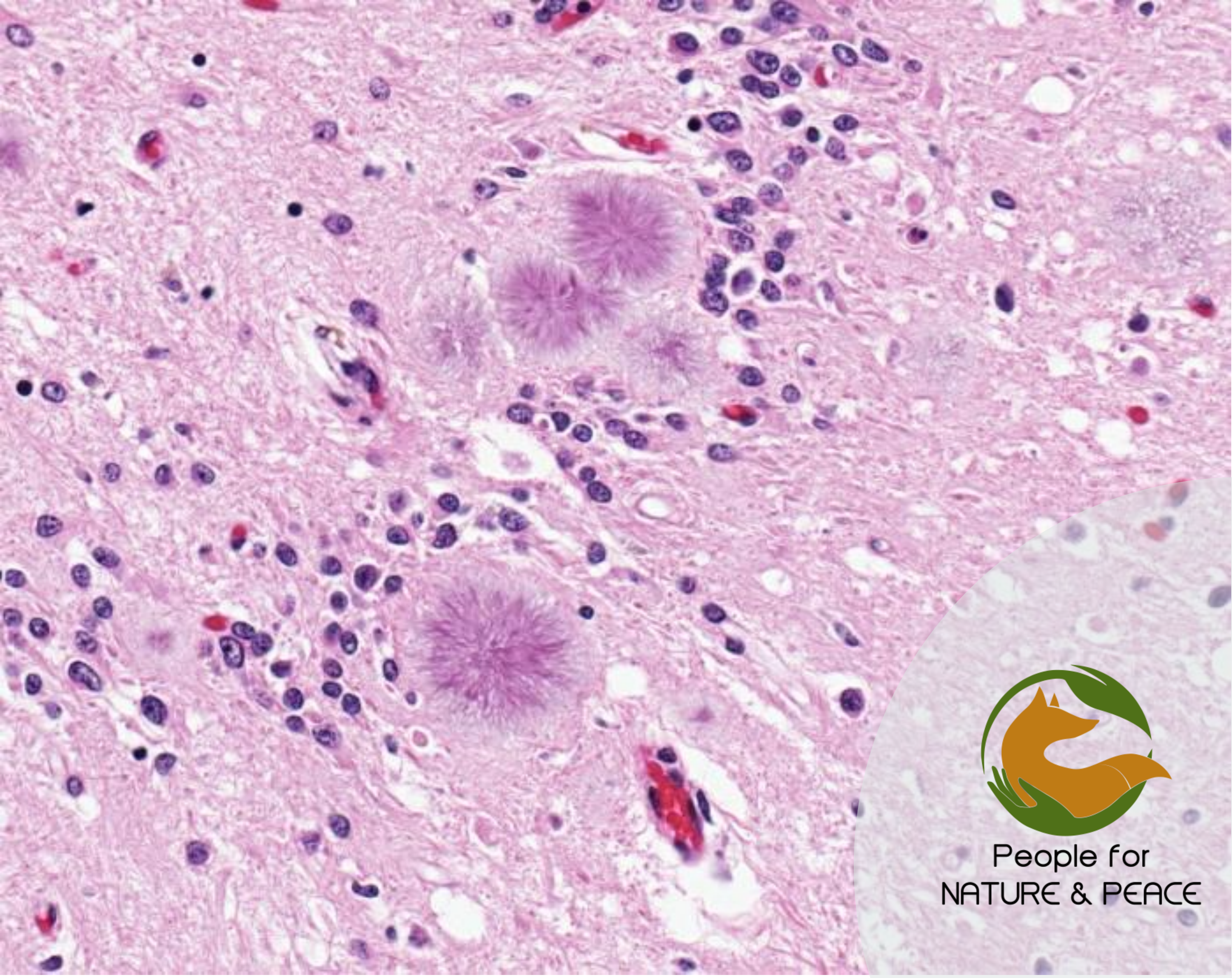

People for NATURE & PEACE

USING CARNIVORES FROM FUR-FARMS AS LIVESTOCK FEED

A One Health Risk Profile

AUTHOR

Dr Barbara Maas
Founder and Chief Executive
People for Nature and Peace
2025

COVER PHOTO

Brain tissue with presence of amyloid plaques found in a case of variant Creutzfeldt-Jakob disease (vCJD)
Centers for Disease Control and Prevention

1. OVERVIEW

A proposal to use carcasses from fur-farm animals—such as mink, foxes, and raccoon dogs—as ingredients in feed for livestock (e.g., cattle, poultry, pigs) or to other fur-farm animals raises serious scientific and public concerns with a very real potential to substantially affect human health, animal health, and the broader environment. A host of zoonotic viral and bacterial pathogens, including Transmissible Spongiform Encephalopathies (TSEs) – are relevant in this context a class of incurable prion diseases

1.1 WHAT IS ONE HEALTH?

"One Health" is an integrated approach that recognizes the inescapable interconnection between the health of people, animals, and the environment. When human activities threaten one of these components, the ripple effects can cause wide-ranging harm to each.

1.2 BROAD ZOONOTIC DISEASE RISKS

High-Risk Species: Mink, foxes, and raccoon dogs can harbour a range of pathogens, including viruses (e.g., coronaviruses, influenza), bacteria (e.g., Salmonella, Campylobacter), and parasites (e.g., Echinococcus, Toxoplasma) that either already do or have the potential to infect human beings.

Intensive Farming Conditions: Animals in fur farms live in high-density caging systems that exert biological stress and favour the rapid spread of infectious agents. Healthcare and veterinary oversight is mostly minimal. Once carcasses from these operations enter the feed chain, contaminated material can expose livestock or other animals such as birds, rodents to pathogens they would normally never encounter.

Potential for Reassortment and New Strains: Viruses like influenza e.g., can recombine when multiple strains co-infect a single host species. Cross-infection of other livestock can generate novel variants capable of infecting humans. This happened for instance in the case of swine-flu, bird flu.

Hidden Transmission: Many pathogens have incubation periods during which animals show no signs of illness yet harbour and shed infectious agents. If feed is contaminated, entire herds or flocks are at risk of being infected before the problem is even recognized.

The following text provides an overview of the risks concerning corona- and influenza viruses, as well as Transmissible Spongiform Encephalopathies (TSEs or prion diseases). A selection of key references is provided at the end to allow for further reading and validation.

2. OVERVIEW OF THE SUPPLY CHAIN

The following list provides an overview of the major nodes in the supply chain - from on-farm activities to transport, processing, and use of the final product. In doing so, it aims to highlight the key biological hazards and potential pathways for pathogen emergence or transmission.

2.1 FUR FARMS

Housing: Large farms with high-density caging systems. Poor hygiene is common in these close confinement systems.

Healthcare: Often minimal veterinary oversight or inconsistent vaccination protocols.

Animal welfare is consistently poor with animals exhibiting physical and behavioural indicators of biological stress, which raises zoonotic risks.

These conditions amplify pathogen transmission (e.g., viruses, bacteria, parasites), causing a zoonotic risk to farm workers exposed to infected animals, faeces, or bodily fluids. Co-infection with multiple viruses (e.g., influenza subtypes) can facilitate viral reassortment in animals.

2.2 PREPARATION AND TRANSPORT

Animal slaughter (often seasonal).

Carcass storage: Temporary holding (cold storage or freezing) prior to transport.

Transport: Bulk movement of animal bodies from farm sites to rendering or feed-processing facilities.

These stages in the supply chain present a high potential for inadequate cold-chain maintenance or spillage and contamination during handling etc. If carcasses are not adequately contained or if leachate (fluids) escapes, pathogens can contaminate soil, vehicles, or water sources. Personnel who handle carcasses face risk from direct contact. Furthermore, it is impossible to render small, medium, and large-scale fur farm operations bio-secure with regard to the prevention of cross contamination with external wildlife such as birds or rodents.

2.3 PROCESSING INTO FEED

Rendering (e.g., high-heat treatment) or partial processing (e.g., grinding, mincing).

Feed production: Production of meat-and-bone meal or similar by-products.

Suboptimal rendering temperatures or poor regulation can allow survival of certain pathogens (e.g., spore-forming bacteria, viruses resilient to moderate heat, prions). Cross-contamination between raw and finished product poses further risks. Aerosol or dust generation during grinding or pulverizing can spread pathogens within facilities.

2.4 USE OF ANIMAL FEED

Fed to livestock (poultry, swine, ruminants) and/or to young animals on fur farms.

On-farm feed storage, mixing, feeding equipment, and leftover feed disposal.

These activities have the potential for the introduction of novel viruses or bacteria into poultry, swine, or ruminant populations. There is also a risk of species-jump to humans (e.g., farm workers, veterinarians) if livestock become infected. If the derived feed is fed to animals on fur farms, this perpetuates a cycle of exposure within the same populations and risks in-breeding of pathogens over multiple generations, potentially selecting more virulent or adapted strains.

2.5 ENVIRONMENTAL PATHWAYS

Runoff of waste by-products or landfills from processing facilities can contaminate water sources. Wildlife and scavengers accessing carcasses, waste or feed increase the risk of pathogen dissemination into local ecosystems as well a cross-species exposure.

3. CORONAVIRUSES

SARS-CoV-1 (the virus responsible for the 2002–2003 SARS outbreak) is relevant to the current discussion about coronaviruses in fur-farm animals. Raccoon dogs and civets were implicated as potential intermediate hosts or reservoirs for SARS-CoV-1. This means that they can harbour and potentially transmit coronaviruses with human pandemic potential. While SARS-CoV-1 is no longer circulating widely in humans, its history highlights that raccoon dogs—and likely other fur-farm species—have the biological capacity to sustain, amplify, and transmit coronaviruses capable of infecting humans.

SARS-CoV-2 (the virus causing COVID-19) and other coronaviruses: Mink are highly susceptibility to SARS-CoV-2, with documented outbreaks on mink farms including instances of mink-to-human and human-to-mink transmission in several countries. Raccoon dogs have been infected experimentally and can shed the virus via respiratory and oral secretions, raising concerns as potential intermediate hosts. Coronaviruses can be environmentally resilient, especially in cold-chain processes. Other coronaviruses (e.g., canine or feline coronaviruses) could also circulate in carnivorous species, though less extensively documented in fur-farm settings.

3.1 KEY RISK FACTORS

High-density husbandry: Fur-farm animals often live in crowded conditions, facilitating rapid coronavirus spread. Even mild or subclinical infections can go unnoticed.

Virus shedding & environmental contamination: Infected animals can shed coronaviruses in respiratory droplets, saliva, and faeces. Contamination of cages, surfaces, or feed-handling areas raises the risk of onward spread.

Carcass handling, transport, and processing: If coronaviruses remain active on carcasses (particularly if cold-chain conditions preserve viral viability), there is a chance of cross-contamination during transport and processing. While high-heat rendering can inactivate coronaviruses, pre-processing steps (collection, storage, grinding) create multiple points of potential exposure for workers and cross-contamination of equipment on sites and during transport.

Use as Feed for other animals: If rendering or disinfection is inadequate or incomplete, residual coronavirus could enter feed for other fur-farm animals or livestock (especially if stored in suboptimal conditions). Immunologically naïve animal populations may amplify any coronavirus that remains infectious, risking spillover to people.

3.2 POTENTIAL OUTCOMES

Genetic adaptation/mutation: With repeated cross-species transmission (e.g., from humans to mink, then back to other animals), coronaviruses can accumulate mutations. Some variants may gain enhanced transmissibility or altered virulence.

Zoonotic spillover: Individuals handling contaminated carcasses or feed could contract new or adapted coronavirus strains.

Economic & public health impact: Outbreaks on fur farms have led to large-scale killings and sector internal and external commercial repercussions. New variants could trigger significant broader public health challenges.

4. INFLUENZA VIRUSES

Mink and other mustelids are susceptible to various subtypes of Influenza A viruses, including avian influenza (e.g., H5N1, H5N8), swine influenza, and even human influenza strains. Foxes and raccoon dogs can also be infected with certain avian influenza strains, sometimes via feeding on contaminated poultry by-products. Co-circulation of multiple influenza strains in carnivores can lead to reassortment events - where gene segments from different viral strains mix to form novel variants.

4.1 KEY RISK FACTORS

Susceptibility to multiple strains: Mink, in particular, can act as “mixing vessels” for influenza if they become infected with avian, swine, or human strains concurrently.

Possible high viral load: Experimental and field data suggest that infected mink can produce substantial amounts of virus, raising the potential for farm-wide or broader animal/human outbreaks.

Carcass-to-feed pathways: Influenza viruses can be inactivated by high temperatures; however, if any step in the processing chain (from carcass storage to rendering) is suboptimal, viable viruses would be able to survive. Frozen carcasses (common in fur-farm operations) can preserve the virus for extended periods before it is eventually processed.

Amplification in livestock: Once present in poultry or swine populations, influenza viruses are notorious for evolving further. New strains can emerge with altered host ranges, transmissibility, or pathogenicity - with pandemic potential if they adapt to human hosts.

4.2. POTENTIAL OUTCOMES

Genesis of Novel Reassortants: If carnivores on fur-farm harbour multiple influenza strains that then pass to other animals, there is a risk of viral gene swapping, which gives rise to new variants with unknown infectiousness or virulence.

Zoonotic Transmission: People who handle infected carcasses during any stage of the supply chain or come into contact with newly infected livestock may be exposed to emerging influenza viruses without the benefit of any existing immunity.

Epidemic or Pandemic Threat: Historically, influenza has demonstrated the capacity to jump from animals to humans, with devastating global effects (e.g., 1918 H1N1 pandemic, more recent H1N1pdm09).

5. SUMMARY OF RISKS: CORONAVIRUSES & INFLUENZA VIRUSES

On-farm risks: High potential for viral maintenance and spread among fur-bearing carnivores, leading to large viral loads in tissues.

Transport & processing: Inadequate temperature control or poor biosecurity can allow pathogen survival and cross-contamination in feed-production facilities.

External biosecurity risk elimination: It is impossible to render small, medium, and large-scale fur farm operations bio-secure with regard to external wildlife species such as birds or rodents.

Feed use: If contaminated products are fed to livestock or other carnivores on fur farms, novel outbreaks can arise, facilitating viral adaptation and possible spillover to humans.

Magnitude of impact: Depending on the virus and its mutations, the impact may range from localized farm outbreaks to regional or global public health threats—especially if an influenza strain or coronavirus evolves heightened transmissibility or severity in humans.

These factors highlight the substantial inherent risks in the production chain if carnivore-derived materials are repurposed into feed for herbivores or other carnivores. Historical precedents (e.g., mink-associated SARS-CoV-2 outbreaks, influenza reassortment in farm animals) demonstrate the potential scale and severity of underestimating these hazards.

6. TRANSMISSIBLE SPONGIFORM ENCEPHALOPATHIES (TSE)

TSEs are inevitably fatal neurodegenerative diseases caused by prions. Prions are misfolded proteins, which propagate their abnormal shape and can spread between animals and in some cases, to humans. Known as prion diseases, they include Bovine Spongiform Encephalopathy (BSE) in cattle, scrapie in sheep and goats, Transmissible Mink Encephalopathy (TME) in farmed mink, and variant Creutzfeldt-Jakob Disease (vCJD) in humans.

Prions have extended incubation periods, are unusually resistant to heat, chemical disinfectants, and standard food-processing methods. Rendering processes that kill most bacteria and viruses do not reliably eliminate prion infectivity.

Host species	TSE Context
Mink	Transmissible Mink Encephalopathy (TME) has been documented in commercial mink farms.
Foxes, raccoon dogs	While not classically associated with TSEs, limited surveillance means subclinical infections could go unnoticed.
Ruminants (e.g., cattle, sheep, goats)	BSE and scrapie can infect other species under certain conditions, demonstrating the potential for cross-species transmission of prions.

Humans	Creutzfeldt-Jakob Disease (CJD). Variant Creutzfeldt-Jakob Disease (vCJD) is of particular relevance as it is linked to BSE exposure via the consumption of infected beef. Kuru disease is historically associated with ritualistic cannibalism in certain regions.

6.1 HISTORICAL LESSONS

6.1.1 Bovine Spongiform Encephalopathy (BSE)

Bovine Spongiform Encephalopathy (BSE), or "Mad Cow Disease" arose when infected meat and bone meal derived from ruminants such as cows or sheep was used in cattle feed. BSE appeared in cattle in the 1980s after they were fed meat-and-bone meal that contained infected ruminant tissues. The disease subsequently spread to other European countries throughout the late 1980s and early 1990s. Later on, cases were reported in parts of Asia, North America, and the Middle East. To prevent the spread of BSE the UK implemented a ban on feeding ruminants (cows, goats, sheep) any material (incl. meat and bone meal) that contains animal protein in 1988. It also prohibits feeding processed animal protein to all farmed animals, including fish and horses. The European Union followed suit in 2001. These measures led to a decline in BSE cases.

BSE caused significant international concern due to the potential for human infection through the consumption of contaminated beef. BSE prions eventually crossed the human species barrier, leading to variant Creutzfeldt-Jakob Disease (vCJD) in humans. Although cases are relatively rare, the disease is invariably fatal and caused widespread alarm amongst experts and the public. The global response involved banning most mammalian proteins in ruminant feed to prevent further prion amplification. Even so, the outbreak claimed tens of thousands of cattle and caused profound economic and societal disruption. BSE provided a hard lesson and demonstrated that feeding animal protein back to the same or other species can lead to devastating outbreaks.

4.1.2 Transmissible Mink Encephalopathy (TME)

Mink on fur farms in Canada, Finland, Germany, the former U.S.S.R., and most recently the United States have developed prion disease from ingesting contaminated material, such as tissues from sheep with scrapie or possibly cattle with subclinical BSE-like agents. Cooking or rendering does not destroy the infectious particles.

TME is a reminder of the unexpected risks of feeding carcass-derived feed to farm animals. And that prions can establish themselves in carnivorous species. If affected animals are rendered into feed, the prions could be passed on to other animals.

Infected animals may appear healthy for months or even years, creating a "silent spread" risk. Prion diseases mostly have long incubation periods which gives the infection abundant time to proliferate undetected. By the time symptoms appear, many animals may already be infected, and prion-contaminated feed or by-products may have been widely distributed.

6.2 KEY RISK FACTORS

Inherent resilience: Prions survive many rendering processes that effectively kill bacteria and viruses. Standard cooking temperatures or basic chemical treatments generally fail to inactivate prion infectivity.

Use of infected tissues as feed: TME emerged on mink farms where animals were fed scraps from sheep with scrapie or cattle harbouring unrecognized prion disease. If carnivores on fur farms carry either diagnosed or undetected prions and their carcasses are rendered, these prions can re-enter the food animal chain.

Silent spread: TSEs typically have long incubation periods, during which infected animals appear healthy. Prion diseases often incubate for years, which means infected animals could enter the food chain or breeding programs with no obvious warning signs. Prion contamination can occur early on in the rendering chain (e.g., carcass collection, transport, grinding) before final inactivation steps, allowing cross-contamination of surfaces or other materials. Due to the resistance of prions to conventional inactivation measures, which may therefore not be effective.

Potential Cross-Species Adaptation: Pathogens can adapt or mutate when transferred between different animal hosts. Even if a pathogen is initially not dangerous to humans, repeated passage through multiple species can produce more virulent or transmissible strains. When prions pass through new hosts, they can adapt or change their “strain” characteristics. Feeding carnivore-derived proteins to herbivores or to other carnivores, risks generating novel prion strains or amplifying existing ones.

Human Exposure: While direct human infections from carnivores are not well-documented, feed contamination in large-scale production scenarios elevates the chance of a novel prion crossing over to humans.

6.3 POTENTIAL OUTCOMES

Emergence of new Prion strains: Similarly to the emergence of BSE, which subsequently crossed over into humans as variant CJD, any prions present in carnivores on fur farms could mutate or adapt when passing into new species. This could mean a TSE outbreak in livestock or a new form of prion disease in humans. Past crises (e.g., BSE in cattle) demonstrate the catastrophic implications of feeding any mammalian proteins back to livestock. Emerging evidence of transmissible diseases in farmed carnivores (including mink and raccoon dogs susceptible to coronaviruses) magnifies the associated risks.

Under-reported and under-surveilled: Prion diseases in particular may go undetected for extended periods, manifesting only when widespread infection has already occurred.

Human Health Risks: Although direct human infections from mink TME are not confirmed, BSE demonstrated that zoonotic transmission to humans is possible once a prion strain adapts to a novel host. The economic and societal impacts from any prion-related outbreak are profound, as witnessed during the BSE crisis (see below).

6.4 BROADER ONE-HEALTH RISKS OF TSE DISEASES

The proposed use of carnivore carcasses as feed raises significant One Health concerns with regard to TESs.

Animal Health	TSEs are invariably fatal. Introducing new or mutated pathogens can trigger outbreaks among livestock that are costly, difficult to control, and may result in the need for mass killings as a counter measure.
Human Health	Infected livestock can become a reservoir for emerging infectious diseases. As seen with BSE, certain prions can cross the species barrier to humans. Novel influenza strains could also evolve and spread among people.
Environmental Impact	Prions persist in the environment (e.g., soil) for extended periods and can potentially infect other species, including wildlife. If pathogens spread via manure, runoff, or unintended spills during transport, the local environment and wildlife populations can become additional reservoirs for contagions.
Economic and Societal Costs	Beyond the potential health toll. large-scale outbreaks can trigger severe trade restrictions, loss of consumer confidence, and severe economic harm. They can also erode consumer trust in the safety of food supply chains.

6.5 SUMMARY OF RISK FOR TSES

On-farm risks: Mink are known hosts for TME; foxes and raccoon dogs have not been thoroughly evaluated for subclinical prions. High-density housing may facilitate transmission of prions among animals if contaminated feed or tissue contact occurs.

Transport and processing: Prions can contaminate machinery, transport containers, and surfaces; once present, they are difficult to eliminate. Even under rigorous heat processing or disinfection the inactivation of TSE agents is not guaranteed.

Use as feed: Feeding prion-contaminated material to ruminants or other carnivores risks cross-species adaptation and disease amplification. Historical parallels (e.g., BSE in cattle, TME in mink) demonstrate how dangerous and devastating recycling infected tissues back into the feed supply can be.

Magnitude of Impact: TSEs carry a high severity risk: although they may appear rare, the impact of an emerging outbreak on public confidence, trade, and animal health is likely to be massive.

This assessment underscores that while TSE outbreaks may be less frequent than viral or bacterial incidents, their potential consequences for animal welfare, human health, and the economy are exceptionally high. The recycling of any animal-derived proteins - especially from carnivores - into feed for other species is a well-known risk pathway for prion amplification and species-barrier jumps.

7. CONCLUDING REMARKS

Using animal carcasses from fur-farms in feed poses a substantial and very real risk of introducing or amplifying dangerous diseases. Prions e.g., are infamous for their capacity to evade conventional inactivation methods and jump species barriers—developments that can trigger widespread outbreaks and potentially also place humans in severe jeopardy. With global awareness heightened by past feed-borne epidemics, the proposed course of action is extremely ill advised to avoid what could become an unprecedented public and animal health crisis.

8. KEY REFERENCES AND FURTHER READING

Baron, T., & Biacabe, A. G. (2016). Origin of Prion Strains. In: Prion Diseases (pp. 131–149). Research in Veterinary Science, 105.

Berhane, Y., et al. (2014). Highly pathogenic avian influenza virus A (H5N1) in mink. Emerging Infectious Diseases, 20(12).

Berhane, Y., et al. (2014). *Highly Pathogenic Avian Influenza Virus A (H5N1) in Mink*. Emerging Infectious Diseases, 20(12).
4. Sun, H., et al. (2022). *Reassortment of H1N1 and H3N2 influenza viruses in experimentally infected mink*. Veterinary Microbiology, 265.

Deem, S. L., et al. (2000). Canine distemper in terrestrial carnivores: A review. Journal of Zoo and Wildlife Medicine, 31(4).

Dubey, J. P. (2010). Toxoplasmosis of animals and humans (2nd ed.). CRC Press.

Eckert, J., et al. (2001). WHO/OIE Manual on Echinococcus in Humans and Animals: A Public Health Problem of Global Concern. WHO.

European Commission. (2002). Regulation (EC) No 1774/2002 laying down health rules concerning animal by-products not intended for human consumption. Official Journal of the European Communities.

Fisher, D., et al. (2020). Potential role of cold environments in the transmission of SARS-CoV-2. Applied and Environmental Microbiology, 86(19).

Freuling, C. M., et al. (2020). Experimental infection of raccoon dogs with SARS-CoV-2. Nature, 586(7831).

Hammer, A. S., et al. (2021). SARS-CoV-2 transmission between mink (*Neovison vison*) and humans. Emerging Infectious Diseases, 27(2).

Marsh, R. F., & Hanson, R. P. (1969). Physical, Chemical, and Immunological Properties of the Transmissible Mink Encephalopathy Agent. Journal of Virology, 3(2).

McEwen, S. A., & Collignon, P. J. (2018). Antimicrobial resistance: a One Health perspective. Microbiology Spectrum, 6(2).

Miller, M. W., et al. (2000). Epizootiology of Chronic Wasting Disease in Free-Ranging Cervids in Colorado and Wyoming. Journal of Wildlife Diseases, 36(4). (For background on TSEs in wildlife.)

Oreshkova, N., et al. (2020). SARS-CoV-2 infection in farmed minks, the Netherlands, April–June 2020. Eurosurveillance, 25(23).

Stack, M. J., et al. (2013). Monitoring for the presence of the bovine spongiform encephalopathy agent in non-ruminant animal feed in Great Britain. Preventive Veterinary Medicine, 112(3–4).

Wells, G. A., et al. (1987). A Novel Progressive Spongiform Encephalopathy in Cattle (Bovine Spongiform Encephalopathy). Veterinary Record, 121(18).

WHO. (2018). WHO Expert Consultation on Rabies: Third report. WHO Technical Report Series.