Imported food risk advice

Prions in human milk and human milk products

Context of this risk advice

- Human milk means expressed milk collected from lactating women to be fed to infants that are not the biological infants of the women supplying the milk.
- Human milk products means products derived from human milk that have been specially formulated to meet the specific nutritional needs of infants such as fortifiers and formula.
- The level of risk for this hazard in human milk and human milk products was determined assuming that the most vulnerable category of infants (preterm infants in hospital neonatal intensive care units) would be receiving the products.

Nature of the hazard

Prion-related disease is caused by the abnormal mis-folded form of the prion protein, PrP^{Sc}, which is often referred to as a 'prion'. The normal form of the prion protein, PrP^{C} (cellular form), is a normal constituent of cell membranes in vertebrates. Prions are highly resistant to inactivation with conventional sterilization procedures, such as autoclaving at 121^{o}C for 20 min (FSANZ 2013; Trevitt and Singh 2003). Prions cause transmissible spongiform encephalopathy (TSE), a life threatening disease.

Transmission

There is no evidence that prions can be transmitted through mother-to-infant transmission or sexual contact. There is evidence of transmission between humans iatrongenically[1] and historically via consumption of diseased brain tissue (Brown and Mastrianni 2010; FSANZ 2013; Trevitt and Singh 2003; WHO 2006). Foodborne transmission of bovine spongiform encephalopathy (BSE), a prion disease of cattle, can cause variant Creutzfeldt-Jakob disease (vCJD) in humans and has been associated with consumption of contaminated bovine material. To protect human health, particular lymphoid and central nervous tissues known to harbour infectivity, termed specific risk materials (SRM), have now been removed from the food supply (FSANZ 2013; Gough and Maddison 2010; Trevitt and Singh 2003). To date there is no evidence of abnormal prion protein in bovine milk (Everest et al. 2006; Gough and Maddison 2010). A search of the scientific literature via EBSCO did not identify any evidence of abnormal prion protein in human milk. TSEs such as CJD that show limited transmissibility between individuals would be highly unlikely to be naturally secreted, i.e. in milk (Gough and Maddison 2010).

Disease severity

Prions cause a range of neurological diseases collectively referred to as TSEs. Prions are a severe hazard as they cause life threatening disease. TSEs have a long incubation period but then cause incurable fatal neurodegenerative diseases. There are several TSEs that can occur in humans: Creutzfeldt-Jakob disease (CJD), Gerstmann-Straussler-Scheinker syndrome (GSS), fatal familial insomnia (FFI), kuru and vCJD. The majority of human TSEs occur sporadically, possibly due to a somatic mutation or spontaneous conversion, or are inherited as a mutation. Less than 1% of cases of human TSE is acquired either iatrogenically[1] or via foodborne transmission (kuru due to cannibalism or vCJD due to BSE-contaminated bovine products) (Dormont 2002; Gough and Maddison 2010; Trevitt and Singh 2003).

[1] Relating to medical procedures, e.g. contaminated instruments, contaminated tissue implants or products and potentially blood transfusion

Infectivity
Human milk has been classified in the low-infectivity category (which incorporates low-infectivity or no detectable infectivity) for CJD and other TSEs, such as GSS, FFI and kuru, by the Australian Department of Health (vCJD was not classified as vCJD has not yet been reported in Australia) (DOH 2013).

Risk mitigation
In the UK and North America potential donors are excluded if they are considered to be at risk of CJD. This includes symptomatic individuals, those who may be at risk from iatrogenic exposure or have a family history of CJD (at risk of CJD linked to genetic mutation). An additional exclusion criteria for North America is for individuals that have spent long periods of time in particular European countries where BSE cases have been reported (FDA 2016; HMBANA 2015; OiE 2016; UKAMB 2003). In Australia there are no CJD-related exclusions for human milk donors (Hartmann et al. 2007).

Evaluation of uncertainty
There is no evidence that prions are transmitted via human milk. There is uncertainty if transmission could potentially occur through blood if the mother had cracked or bleeding nipples.

Pooling of human milk from multiple donors is common practice amongst many human milk banks, however some milk banks only pool milk from individual donors (Haiden and Ziegler 2016). The Australian Red Cross milk bank pasteurises human milk in single donor batches (Australian Red Cross 2018).

Risk characterisation
Although prions may cause severe fatal disease, there is no evidence that prions are present in human milk and human milk has been classed as low infectivity for CJD and other TSEs (DOH 2013). There is a very low likelihood of exposure as there is a lack of evidence for mother-to-infant transmission of prions via human milk. Therefore, prions in imported human milk and human milk products do not present a potential medium or high risk to public health and safety.

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