



## POSITION STATEMENT MATERNAL TRANSMISSION OF vCJD

### Issue

1. The Chief Medical Officer for England asked SEAC to consider current evidence and comment on the potential transmission of vCJD from mother to child via human breast milk. *In utero* transmission was also considered. The committee also commented on the scientific basis of a risk reduction measure for possible transmission of vCJD via banked breast milk.

### Background

2. No diagnostic test is currently available for the detection of abnormal PrP in milk. Research is under way to develop tests to screen for the possible presence of abnormal prion protein (PrP) in milk samples from cattle experimentally infected with BSE<sup>1</sup>. These modified tests may also be applicable to human milk. However, it is not yet clear when/if a reliable test will be available.
3. A small number of breast milk banks in the UK supply highly vulnerable premature babies for whom no milk may be available from the mother. A model developed by the Department of Health to assess the effect of pooling breast milk from multiple donors on the possible risks of transmission of vCJD via breast milk banks was considered.
4. There is some, albeit limited, published epidemiological and experimental research on maternal transmission of prion diseases. There are also unpublished surveillance data of children born to vCJD cases from the National CJD Surveillance Unit and UK surveillance of neurological illness in children which might inform on potential risks of maternal transmission.

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<sup>1</sup> A joint FSA/SEAC milk working group is monitoring and providing advice on this research carried out at the Veterinary Laboratories Agency.

## **Breast milk banks**

5. There is no evidence that vCJD infectivity has ever been transmitted through breast milk. However, a theoretical risk exists. Modelling studies clearly show that the practice of pooling breast milk increases the number of donors to which a recipient is exposed and thereby increases the potential risk of an infant receiving milk contaminated with vCJD infectivity. The theoretical risk of infection can be minimised by not pooling the milk, by the use of individual hand operated breast milk pumps for single donors, and by the use of single-use sterilised bottles for collection. In addition, available evidence suggests that infection/inflammation of the breast results in increased lymphocytes in milk and therefore increased risk of infectivity. This risk would be minimised if milk from donors showing signs of infection is not used.
6. The committee suggested that, if practicable, milk could be stored for an appropriate period of time to allow the health status of donors to be monitored, before it is released. However, information was not available to the committee on whether long-term storage of human milk is detrimental to its nutritional quality.

## **Maternal transmission**

7. There is evidence from animal studies for low level maternal transmission of prions in cattle and sheep. This transmission may occur *in utero*, via milk and/or perinatally. However, the possibility that this putative maternal transmission might have been due to another mode of transmission, for example through a contaminated environment or feed, cannot be ruled out.
8. In contrast, in humans there is no evidence for maternal transmission in cases of familial prion disease, other than the transfer of a mutant form of the PrP gene, and there is no evidence of maternal transmission of Kuru. However, compared with other human prion diseases vCJD may pose a greater risk because of the greater involvement of the lymphoreticular system in vCJD pathogenesis. Although, breast tissue (and placenta) from a single vCJD case tested negative for PrP<sup>vCJD</sup>, transfer of infectivity to breast milk may depend on the physiological status of the mammary gland. Similar tests or infectivity bioassays have not been conducted on breast tissue from lactating patients with vCJD.

9. A published study suggesting transmission of sCJD in colostrum<sup>2</sup> was considered unreliable because tissues not normally associated with high levels of infectivity (blood and placenta) showed equivalent infectivity to that of the brain in this study.
10. Analysis of prospective surveillance data of UK children born to mothers with, or that had subsequently developed clinical vCJD, provide no evidence for maternal transmission of vCJD. However, the number of cases is very small and the incubation period of vCJD, if transmitted from mother to child, is unknown and so the children may yet be too young to have developed symptoms.
11. The phenotype of BSE infection in humans expressing PrP genotypes other than M/M at codon 129 is not known. Given recently published studies in mice expressing the human PrP gene<sup>3</sup>, which suggest that the human PrP genotype may affect disease phenotype, the committee considered it very important that undiagnosed neurological diseases be carefully monitored. In this respect, amongst others, it is recommended that the careful monitoring of neurological illnesses through the PIND surveillance of children<sup>4</sup> continue.

## Conclusions

12. In summary, there is currently no epidemiological evidence for maternal transmission of vCJD, including transmission via breast milk. However, there is a hypothetical risk. Although available evidence is limited and mostly indirect rather than direct, this risk, if any, appears to be low. As a risk cannot be excluded, a watching brief should be maintained.

SEAC  
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<sup>2</sup> Tamai Y *et al.* Demonstration of the transmissible agent in tissue from a pregnant woman with CJD. *New Eng J Med* 1992 327, 649.

<sup>3</sup> Wadsworth *et al.* Human prion protein with valine 129 prevents expression of variant CJD phenotype. *Science*. 2004 306, 1793-1796.

<sup>4</sup> Devereux G *et al.* Variations in neurodegenerative disease across the UK: findings from the national study of Progressive Intellectual and Neurological Deterioration (PIND). *Arch Dis Child*. 2004 89, 8-12.