For example, how can one feed 'raw' milk of 'low bacterial count' when an analysis of bacterial count may take at least a day and the milk must be stored (and is therefore no longer 'raw') during that time? Is there evidence that milk of 'low bacterial count' is always low in number of potential pathogens? How does one decide upon a 'minimum pasteurising temperature for bacterial killing', when this may vary depending on the species of bacteria present? Are milk cells of any importance in milks that are collected from donors past the colostrum period and, if so, how can they be protected when milk is routinely frozen?

Finally, contrary to the statement of Evans *et al.*, a commercial small-scale human milk pasteuriser does exist in the UK (Vickers).

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Drs Evans and Dodge comment:

We should like to answer some of Dr Gibbs's questions. We used the word 'raw' in the first sense of the word as described in the *Shorter Oxford Dictionary*—i.e. uncooked or unheated. Williamson *et al.* (1978) described a method of bacteriological screening of human milk and, although several of their guidelines for safety are empirical, we agree with their aims and methods.

Dr Gibbs correctly states that there is a 'species' variability in bacterial killing by heat and we would also add that the survival is related to the initial concentration of bacteria in the unheated milk.

There is still insufficient information to answer Dr Gibbs's question on the importance of the cellular component of human milk, although *in vitro* 'milk cells' can synthesise complement and immunoglobulins and they can exhibit phagocytosis of bacteria and yeasts.

Although subsequently unconfirmed, it was reported that a graft versus host reaction was induced by feeding milk lymphocytes from a genetically unrelated donor in suckling rats (Beer *et al.*, 1974). Until more knowledge is gained, we are content to impair or destroy milk cells by deep freezing when milk is given to an infant from a woman other than his mother.

When we prepared our paper in 1977, we were not aware of the Vickers' pasteuriser but have since seen, although not evaluated, the apparatus. We thank Dr Gibbs for drawing our attention to it.

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Congenital heart block and hypothyroidism

Sir,

We read with interest the case report by Syed (*Archives*, 1978, **53**, 256). In the family history of the case, the mother suffered from rheumatoid arthritis, treated with aspirin, before her pregnancy. In our paper (McCue *et al.*, 1977), we found that connective tissue diseases (including rheumatoid arthritis) of the mother, may produce congenital complete heart block in the baby.

The mechanism is that antinuclear antibodies of the IgG class cross the placental barrier. The transmission of such antibodies may affect the fetal cardiac conduction system and myocardium, as well as other organ systems (skin, blood, etc.). The cardiac pathology of the case described by Syed also supports this possibility.

For these reasons, we believe the congenital complete heart block to be connected with the maternal rheumatoid arthritis. We do not know if the hypothyroidism is an incidental finding or whether the same mechanism is responsible for both lesions.

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Reference

- McCue, C. M., Mantakas, M. E., Tingelstad, J. B., and Ruddy, S. (1977). Congenital heart block in newborns of mothers with connective tissue disease. *Circulation*, **56**, 82–90.

Dr Syed comments:

I thank Drs McCue and Mantakas for their comments. The hypothesis is interesting and may be the mechanism in this case. Please note an error. X-ray report should read—'No ossification of distal femoral epiphysis or at wrist'.

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