Heating human milk

Human milk is valuable in the feeding of preterm infants not only because of its nutritional suitability but also because it contains substances which help to protect against infection. But, no matter how carefully the milk is collected and stored, pools of expressed breast milk may contain bacteria harmful to the infant, notably *Escherichia coli* and *Staphylococcus aureus*.

The possibility of the presence of these bacteria has made it usual to heat pools of breast milk before use. Ideally the milk should be heated enough to kill the bacteria but not so much as to seriously reduce the protection it gives against infection. This property depends both on substances such as IgA and lactoferrin, which are relatively sensitive to heat, and on others such as lysozyme, which are more resistant. One example is the heat-stable macromolecule inhibitory to viruses recently reported by Matthews et al.,1 which lost its antiviral activity after treatment at about 100°C for 30 minutes but which was unaffected by heating at 56°C for 30 minutes.

The question to be answered, therefore, is how little heating human milk needs to decontaminate it without seriously reducing its anti-infective properties. Raptopoulou-Gigi and her colleagues,2 working in Edinburgh, subdivided samples of human milk, which they then subjected to different heat treatments, to immunological studies by electrophoresis and antibody titrations, to bacteriological culture, and to gamma-irradiation (2-5 Mrads from a cobalt-60 source). The samples were examined before and after heat treatment or irradiation and the results were compared with those given by an aliquot of the processed sample stored at 4°C. Standard treatment (heating the milk to 105°C, then freezing and thawing it, and repeating this cycle) decontaminated the milk, including some samples which were heavily, artificially contaminated with *Staph aureus* and *E coli*. So did gamma-irradiation and pasteurisation (62-5°C for 30 minutes). Only pasteurisation, however, did not change the mean levels of IgA or lactoferrin or the haemagglutinin titres against pooled *E coli* antigen. After the standard treatment to 105°C neither IgA nor lactoferrin could be detected in any sample. Gamma-irradiation produced a slight fall in IgA levels and a substantial drop in lactoferrin levels.

In a separate study, Ford et al.3 reported from Reading that holder pasteurisation (62-5°C for 30 minutes) reduced the IgA levels by 20% and destroyed the small amount of IgM and most of the lactoferrin. While these observations appear to contradict those from Edinburgh the cause of the apparent discrepancy may lie in two important technical differences between the heat-testing procedures used. At Edinburgh the samples consisted of whole milk; and no mention was made in the report of the size of the sample or of the type of container in which it was heated. It would be of interest to know the full details of the procedure and to know what temperatures were actually reached at the centre of the sample and for how long. At the National Institute for Research in Dairying at Reading Ford et al used 1-5 ml samples sealed into small glass ampoules and heated in a bath of detergent maintained at the required temperature ±0-1°C. Furthermore, the milk to be sampled had been centrifuged at 2°C for one hour at 75 000 g and the aqueous phase carefully decanted and filtered through Kleenex tissue. The fat and sediment had been discarded. Inevitably these two important differences would have ensured much more rapid and complete heat penetration than in the Edinburgh investigations.

The conclusions from the two studies were, however, essentially the same. If treatment is necessary, holder pasteurisation is better than "standard" heat treatment. Both groups emphasised that we need to know more about which of the various substances are important as anti-infective agents; and both ask whether decontamination of human milk is really necessary. The case for a combined operation by obstetricians, paediatricians, nurses, microbiologists, immunologists, and dairy scientists seems to be as clear as can be. The work need not be unduly difficult or tedious, and the results should be both of interest and of practical value.

Motilin: Actor in search of a play

Years ago, when acronym-construction was in its infancy, a group of viruses was labelled ECHO, meaning Enteric Cytopathogenic Human Orphan. No one knew what damage, if any, they caused or what other function they had; they were viruses—easily recognisable by standard biological methods—in search of a disease. Recent rapid advances in knowledge of gastrointestinal hormones have produced similar problems. Not that gut hormones are new. This year is the 75th anniversary of Bayliss and Starling’s discovery of the original gut hormone secretin, they having first deduced its existence from studies of pancreatic secretion; while gastrin had been presumed to exist by Edkins over 50 years before it was isolated by Gregory and Tracy. These and the other gastrointestinal hormones have powerful effects on motor, secretary, and absorptive processes throughout the alimentary tract. These actions may readily be shown in animals and in various isolated preparations. What relationship the effects have to physiological control mechanisms or to human disease, however, is far less certain.

Gut hormones now number 26 or more.4,5 Tiny concentrations in the serum may readily be measured by radioimmunoassay, their origins in the intestine can be shown by histological immunofluorescence, and chemists can elucidate their structure. They can be purified and used to...