SUMMARY

(1) Different microconstituents present in the milk samples from Bengalee mothers were quantitatively estimated. The milk samples from 253 donors (volunteers or inmates of local hospitals) were analysed. The mean age of the mothers was 26.9 ± 6.5 years.

(2) Average activity of alkaline phosphatase in transitional milk from normal Bengalee mothers was found to be 4.70 with a S.D. of 1.38 K.A.U. Out of 48 samples tested, 70 per cent showed activity range of 3.0-6.5 K.A.U.

(3) Alkaline phosphatase activity in the human colostrum (13.38 ± S.D. 1.81 K.A.U.) is significantly higher than that in human transitional milk.

(4) Optimum pH for alkaline phosphatase activity in both human and cow milk was found to lie between 9.8 and 10.0 with phenylphosphate as substrate.

(5) Human milk alkaline phosphatase was found more thermostable than alkaline phosphatase of bovine milk and possible occurrence of an isoenzyme, heat-stable phosphatase (normally found in human placenta and serum of pregnant women) in human milk was indicated.

(6) The major lipase activity of the human milk is stimulated by detergent. This enzyme shows a low activity when emulsified
triglyceride was used as substrate.

(7) The range of lipolytic activity ($X \pm 2 SD$) in the transitional milk of normal Bengalee mothers is 22.48 - 45.28 units/ml. The activity of this enzyme in colostrum or mature milk is not significantly different from that in the transitional milk.

(8) Bovine milk showed 4-5 times less lipolytic activity (with or without detergent) than the human milk.

(9) Heating at 65°C for 15 min inactivates the lipolytic activity of human milk. About 73-75% of the activity of this enzyme is lost when the milk is heated at 55°C for 15 min.

(10) The milk from Bengalee mothers also capable of hydrolysing low molecular weight organic esters. This esterase activity is probably different from the lipolytic activity present in the milk. The average esterase activities in colostrum, transitional and mature milk are 8.13, 5.87 and 6.39 units/ml respectively.

(11) The mature milk of bovine origin could not hydrolyse low molecular weight organic ester.

(12) Fairly strong amylolytic activity is present in the milk of Bengalee mothers. Amylase activities in the transitional milk and mature milk are marginally higher than that in colostrum, these differences however are not significant.

(13) The amylase activity in human milk is 30-40 times higher than that in bovine milk, assayed under similar conditions.
(14) The normal range ($\bar{x} \pm 2$ SD) of amylase activity in transitional milk is 2.55-5.99 units/ml (1 unit of enzyme is capable liberating 1 mg of maltose in 5 min).

(15) The human milk amylase is very much heat labile as this enzyme is almost completely inactivated within 15 min at 55°C. More than half of the original activity is lost when milk is heated to 45°C for 15 min.

(16) The lactoperoxidase activity in the colostrum (5.66 ± 0.84 units/ml) is significantly higher than that in the transitional milk (3.05 ± 0.61 units/ml) of Bengalee mothers. The bovine milk contain higher activity of this enzyme than the human milk.

(17) The normal range of lactoperoxidase activity ($\bar{x} \pm 2$ SD) in transition milk of Bengalee mothers is 1.83-4.27 units/ml.

(18) The lactoperoxidase of human milk is activated by heating at 25-55°C. The maximum activation of the enzyme could be achieved by heating the milk 45-55°C for 15 min. However, when heated above 55°C, the activity of the enzyme declines sharply and heating at 80°C for 15 min completely inactivates human lactoperoxidase.

(19) Oligosaccharides containing fucosyl residue are important microconstituent of human milk as some of these oligosaccharides stimulate the growth of Lactobacillus bifidus in large intestine of breast fed babies.
(20) Total fucose contents in transitional milk and colostrum of Bengalee mothers vary between 0.93 and 2.20 mg/ml. Out of this, a substantial amount (0.55-1.55 mg/ml) is free fucose (i.e. not glycosidically bound).

(21) The fucose content of human milk is dependent on ABO blood groups of the mothers. Blood group AB and B holders exhibit high fucose content in their breast milk.

(22) Ampicillin, a broad spectrum oral penicillin is excreted in the milk when this antibiotic is received by the mother even at a small dose.

(23) The concentration of ampicillin excreted in the milk is dependent on its oral dose to the mothers. The antibiotic takes 2 hours for its transportation from the stomach of the mothers to their milk. This time lag for the appearance of antibiotic in the milk following its oral intake is independent of its oral dose.

(24) The maximum concentration of ampicillin in the milk was detected in between 5th and 6th hours after the oral dose of this antibiotic.

(25) Ampicillin residues were detected persistently in milk samples collected from the mothers who were on ampicillin therapy. The antibiotic excreted continuously in milk so long mothers received this antibiotics.

(26) When ampicillin was allowed to react with human milk in vitro, about 40-60% of its antibiotic activity was lost presumably
by immobilization of this antibiotic by binding with milk constituents.

(27) The ratio of milk : blood plasma concentration of ampicillin (M : P ratio) following the oral consumption of this antibiotic was found to be fairly low, i.e. 0.11 when rate of its excretion in milk was stabilized.

(28) The total amount of the antibiotic excreted in the milk of mothers on ampicillin therapy is too small to produce any adverse microbiological effects on breast fed babies. The effective blood concentration of ampicillin in babies receiving milk from the mothers on ampicillin therapy will be very low and insignificant for possible stimulation of the selection of ampicillin resistant pathogens or for inhibiting the growth of helpful microflora in their system.

(29) The ampicillin residue in the milk may be harmful to the babies who are allergic to this antibiotic.

(30) Sulphamethoxazole residues were detected in milk when this popular sulfa drug was given to the mothers orally. A small time lag (less than 60 min) was found between the oral administration and first appearance of this drug in the milk.

(31) The maximum concentration of sulphamethoxazole in milk was detected on 4th hour after its oral intake by the mothers.

(32) When the mothers were previously on continuous sulphamethoxazole dosages, the drug residue was detected in milk even 12 hours after the last dose.
(33) The sulphamethoxazole concentrations of the excreted milk were fairly high (3.31 - 6.25 μg/ml) when the mothers were on continuous sulphamethoxazole therapy.

(34) M : P ratio (milk : blood plasma concentration ratio) for sulphamethoxazole remains fairly constant (0.33-0.36) at different plasma concentrations of this drug.

(35) In vitro binding of sulphamethoxazole by milk constituents was found higher than that by blood plasma.

(36) As absorption of sulphamethoxazole from human G.I. tract is very slow, the G.I. tract of the breast fed babies will be flushed continuously with residues of this drug excreted by the mothers in their milk. The amount of the drug excreted in the milk may be very small, but the continuous presence of the drug, even at small concentration in the G.I. tract may alter the microfloral composition in the infants' intestine.

(37) The expressed milks from Bengalee mothers are contaminated with bacteria mostly from the skin surface of the mothers.

(38) Total viable bacterial counts in the milk were highly variable and presumably dependent on the living conditions and hygienic sense of the mothers.

(39) Most common organism detected in the milk was Staphylococcus albus. This organism accounted for about 80% of all viable bacteria found in lightly contaminated milk. Other bacteria found in the milk were S. aureus, Streptococcus faecalis.
and *Streptococcus viridans*.

(40) Enterbacteria of faecal origin were also detected in the milk of Bengalee mothers. *Escherichia coli*, *Klebsilla aerogens*, *Alcagenes faecalis* and *Acinetobacter* sp. were detected in the milk which collectively accounted for about 15% of total viable counts in several milk samples tested. The enterobacteria were most conspicuous in the milk samples laden with high bacterial counts.