Table *Difference in composition of Babymilk Plus and day 6 breast milk related to plasma aldosterone levels*

	*Cow & Gate Babymilk plus**	*Breast milk (day 6)*†
Na^+ (mEq/l)	13	17±5·02
K^+ (mEq/l)	22	16·8±2·54
*p*H	6·53	7·10±0·16
Plasma aldosterone‡	2040 pmol/l (73·44 mg/100 ml)	1012 pmol/l (36·43 mg/100 ml)

*Barrie *et al.* (1975).
†Ansell *et al.* (1976).
‡Dillon *et al.* (1976).

than infants fed cows' milk (Hatemi and McCance, 1961) because of the greater net acid content of cows' milk. Sulyok (1971), investigating the relationship between electrolyte and acid-base balance in term infants found a close association between the renal loss of sodium in the first 2 weeks of life and the development of metabolic acidosis, and suggested that this might be due to transient adrenal mineralocorticoid deficiency; and Pitts (1950) showed that adrenalectomized rats failed to show an increase of ammonium excretion in response to an acid load and that this could be corrected by adrenal cortical extract.

The infant's ability to cope with an acid load seems to be dependent at least in part on adequate renal sodium-hydrogen ion exchange (Sulyok *et al.*, 1972). As aldosterone plays an important part in this, it is not surprising that it is present in higher concentrations in those infants receiving a greater acid intake. Infants fed on Cow & Gate Babymilk Plus with its lower sodium and higher acid content might be expected to have higher plasma aldosterone levels than breast-fed infants as found by Dillon *et al.* (1976) in order to conserve sodium and excrete hydrogen ions.

ANGELA MOORE
Department of Paediatrics,
Charing Cross Hospital, London W6 8RF

Drs. Dillon and de Swiet comment:

We are grateful to Dr. Moore for emphasizing the electrolyte differences between colostrum, transitional milk, and Cow & Gate Baby Milk Plus. We would agree that on the basis of the higher concentration of sodium in early breast milk compared with that in Cow & Gate Babymilk Plus, breast-fed infants may receive more sodium in the first week of life than those artificially fed with this particular milk formula. Though this might have contributed to the lower values of plasma aldosterone (PAldo) in the breast-fed group of infants in our study (Dillon *et al.*, 1976), we were surprised that there were no parallel changes in the values of plasma renin activity (PRA). In our experience PRA is a more sensitive index of sodium status than PAldo, and we would have expected similarly low values in the breast-fed babies if they had been significantly sodium-loaded compared with the artificially-fed group. Furthermore, we failed to show differences in urine sodium excretion on day 6 of life between those babies fed breast milk and those fed Babymilk Plus, suggesting that on day 6 the sodium intake of both groups was very similar.

Aldosterone may well play a part in the newborn infant's acid-base homoeostasis. We are, however, unaware of any clear evidence that this is an important role or that aldosterone secretion is specifically increased in response to an acid load. None the less we accept that in situations where aldosterone secretion is clearly deficient the administration of mineralocorticoids can increase the bicarbonate threshold by improving sodium reabsorption (Oetliker and Zurbrügg, 1970).

It is intriguing to speculate on the reasons for increased activity of the renin aldosterone system in the infant and young child. We would agree with Dr. Moore that this is likely to be related to sodium conservation, as we stated in our recent *Archives* paper. However, it seems that the levels of PRA and PAldo in infancy and childhood are almost entirely dependent on age and only marginally attributable to differences in sodium intake (Dillon and Ryness, 1975). We would therefore not wish to put too great an emphasis on the findings of higher PAldo levels in the bottle-fed babies in our neonatal study. The numbers were comparatively small and the differences only just statistically significant. Further study seems required to clarify the situation.

M. J. DILLON
Institute of Child Health,
30 Guilford Street London WC1N 1EH
M. DE SWIET
Queen Charlotte's Maternity Hospital
London W6.

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