and at necropsy hepatocellular carcinoma was found.⁶ This may indicate a serious prognosis for these patients.

Finally, I should like to point out that the statement: 'Galactosaemia was excluded by finding normal activity of red cell galactose-1-phosphate uridyl transferase'¹ does not exclude the possibility of galactokinase deficiency.⁷

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Role of aldosterone in late hyponatraemia

Sir,

I read with interest the report by Al-Dahhan *et al* on the beneficial effect of salt supplementation on the clinical course of low birthweight preterm infants.¹ On the basis of the lower urinary potassium:sodium ratio seen in supplemented preterm infants, the authors concluded that '... salt supplements cause some suppression of aldosterone secretion' and 'the tubule is capable of responding to the hormone'.

This statement may give the false impression that the role of aldosterone in the development of late hyponatraemia has not yet been studied. In fact, in recent years

several clinical studies have already been carried out in preterm infants showing that:

(1) In response to the renal salt wasting, negative sodium balance, and fall in the plasma sodium value, excessive increase occurs in the activity of the renin-angiotensin-aldosterone system.²⁻⁴

(2) The increased aldosterone secretion rate results in a rapid improvement of distal tubular sodium reabsorption and contributes to the re-establishment of positive sodium balance.^{5, 6}

(3) By giving supplemental sodium the activity of the renin-angiotensin-aldosterone system may be suppressed^{7, 8} even to the degree seen in neonates of the same postnatal age.⁸

The results presented by Al-Dahhan *et al*, therefore, provide only indirect evidence to confirm these previous observations.

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