by the traditional '-in' suffix (as for insulin and renin). At that time we avoided naming the hormone by its action as we were uncertain whether the neuromuscular effect was physiological; indeed, it now does not seem to be so.

Unfortunately, there is confusion between the polypeptide hormone thymin and the base thymine, which was also named for its original isolation from thymus. Would the name thymopoietin be more acceptable? This would indicate the action of the hormone in inducing stem cell differentiation to, or 'producing', thymocytes (as in erythropoietin and granulopoietin).

I agree that the numbers I and II are provisional and that this will be resolved by structural work, which is in progress.

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(Na + K)-ATPase activity in cattle red cells

SIR,—Schatzmann[1] has described a positive correlation between ouabain-sensitive ATPase activity and red cell K levels in cattle, cells with higher K levels giving a greater enzyme activity. These determinations were made with Na=150 mM, K=10mM. If ouabain-sensitive ATPase is determined as a function of K concentration (at K levels >5 mM to saturate the outside pump site) in fragmented red cell ghosts from individual cattle with different erythrocyte K levels, it can be seen that, as in other ruminant red cells[2], the enzyme shows a variable degree of K inhibition (Fig. 1). Thus, membranes derived from cells having an original K level of 26 mmol per l cells are 50% inhibited at K=20 mM, whereas membranes from cells originally having 77 mmol K per l cells are not 50% inhibited even at K=100 mM. From the 26 cows so far investigated a continuous spectrum of K sensitivities has been found, with the degree of K inhibition correlating well with the resting K level of the original red cells. It therefore seems likely that the internal affinity for K of the ouabain-sensitive ATPase in cattle red cells may be the dominant determinant of resting cell K levels, and not the amount of enzyme activity measured at relatively low K concentrations.

Yours faithfully,

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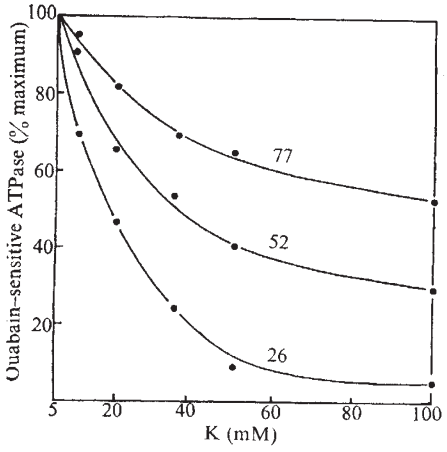

Fig. 1 Ouabain-sensitive ATPase activity in cattle red cell ghosts as a function of K concentration. Ghosts prepared and assayed as previously described[3]. Incubation conditions: NaCl 150 mM, $MgCl_2$ 1.25 mM, Na_4–ATP 1.25 mM, 10 mM Tris *p*H 7.7 at 37° C, (KCl + choline Cl) 100 mM, ouabain, when present, 0.1 mM. Ghost concentration equivalent to 25% haematocrit, original packed cells. All conditions measured in quadruplicate. Curves shown for cells from three individual cattle with red cell K 77, 56, 26 mmol per l cells. Data were normalised as % maximum ouabain-sensitive ATPase activity (at 5 mM K). Actual maximum ouabain-sensitive ATPase activities at 5 mM K; upper curve, (77 mmol K per l original cells), ATPase 375 nmol P_i per mg protein per h; middle curve, (52 mmol K per l original cells), ATPase 660 nmol P_i per mg protein per h; lower curve, (26 mmol K per l original cells), ATPase 260 nmol P_i per mg protein per h.

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DR SCHATZMANN REPLIES: The marked variability of the K affinity of the internal Na site found by Dr Ellory renders untenable my tacit assumption that the ATPase activity measured at 10 mM K and 100 mM Na reflects the number of pump sites per cell. But unless our conditions of Na concentration, *p*H and ionic strength considerably enhance the effect due to different affinities it does not seem to account completely for the variability of the Na+K−ATPase which we reported. From the regression in our experiments cells with $[K]_{cells} = 77$ mmol l^{-1} might be expected to have 4.5 times the ATPase activity of those with 26 mmol l^{-1}, whereas the curves shown above only yield a factor of about 1.4 at 10 mM K. Yet I fully agree with the conclusion that the internal K affinity seems more important than the number of pump sites per cell in determining the cellular K concentration.

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No chromosome 9q+ in Ph¹-negative CML

SIR,—To demonstrate that in Ph¹-positive cells the additional band at the end of the long arms of chromosome 9 (9q+) stems from chromosome 22, Rowley[1] suggested the examination of metaphases derived from bone marrow from patients with Ph¹-negative chronic myelocytic leukaemia (CML). We studied the chromosomes of three such patients, two of whom had symptoms considered to be characteristic for Ph¹-negative CML[2], that is, mild leukocytosis, anaemia, severe thrombocytopenia and discrete splenomegaly.

Case 1 died of a cerebral haemorrhage 3 months after first presentation. Case 2 entered blastic phase and died 20 months after CML had been diagnosed; chromosomes were studied during blastic phase. Case 3 has typical CML and is well 3 yr after the diagnosis has been established.

The chromosome banding pattern was obtained by the trypsin-saline-Giemsa method. The karyotype of case 1 was normal, 46,XY. Case 2 had two clones; the major clone (60%) had the karyotype 47,XY, 20q-,+21 and the minor clone (40%) had 46,XY,20q-. In case 3 the marrow cells revealed a missing Y on several occasions; the karyotype was 45,XO while the PHA-stimulated lymphocytes were chromosomally normal.

Chromosome 9q+ could not be detected in any metaphase of the Ph¹-negative cases. Thus, these findings corroborate Rowley's assumption[1] that the additional material on chromosome 9 comes from chromosome 22. Furthermore, an abnormal banding pattern which would characterise Ph¹-negative CML could not be seen. This may be related to the finding that patients with Ph¹-negative CML represent a rather heterogenous group[2].

Yours faithfully,

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