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Zusammenfassung

Der Umbau von 4,6-C^{14}-Adenin in Ribo- und Desoxyribonukleinsäure wurde im Gewebe von jungen Mäusen vom Stamm Webster 17 h nach intraperitonealer Injektion von markiertem Purin analysiert. Das Verhältnis der spezifischen Aktivität von RNA zu DNA variiert von 1,1 bis 1,6 für sämtliche Gewebe, ausgenommen Leber, wo ein Verhältnis von 6,4 gefunden wurde.

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Do Any Morphological Pictures of Separated Excretion of Histamine and Heparine in Tissue Mast Cells Exist?

Recently a notice was published in this journal by HILL[1] on the morphological effects of histamine and mucopolysaccharide secretion in the tissue mast cells. The author assumed that the releasing of both substances proceeds in two stages: first, histamine is released and thereafter heparine. With the first stage, a change in the stainability of the granulations is involved. As this statement is of great importance in the histophysiological and histopathological interpretation of the morphological pictures, I decided to examine in a model whether or not the existence of histamine has an influence on the staining properties of heparine, and whether a destruction of the mucopolysaccharide has a releasing effect on the histamine.

We have prepared mixtures of hyaluronic acid, heparine and histamine each substance alone or two together, and have stained them with toluidine blue. The binding of histamine to any of the mucopolysaccharides examined has no influence on the staining properties of the latter.

In a second series of experiments, we have observed the process of histamine-heparine molecule destruction using hyaluronidase (Hyalase-Benger Lab. Ltd.) or heparinase prepared by us. We have stated that, despite the destruction of the heparine molecule, a significant amount of histamine is still bound to the latter. The process of releasing histamine in the mast cells may proceed another way, of course, but we suppose that a separate secretion of histamine and heparine is not probable.

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Zusammenfassung

An Modellpräparaten wurde festgestellt, dass die Färbbarkeit der konjugierten Heparin-Histamin-Moleküle gleich ist wie die des Heparins selbst. Der fermentative Abbau des Heparins führt nicht zur vermehrten Freisetzung von Histamin.

[1] J. HILL, Exper. *13*, 395 (1957).

Phloridzin and Red Cell Phosphate Turnover

It is well known that phloridzin and its aglycone, phloretin, inhibit the movement of sugars into erythrocytes and other cells. The action has been attributed to interference with phosphorylation but this explanation meets the difficulty that sugar phosphates do not themselves penetrate red cells easily, if at all (LEFEVRE[1], WILBRANDT[2]). If, however, the mechanism of sugar movement involves a series of temporary combinations with a succession of phosphate groups forming part of the cell structure then the slow movement of ready-made sugar phosphate would not be inconsistent with the requirement for particular phosphorylations to take place. If the phosphate groups of the cell are concerned with sugar movement then substances which slow sugar movement might act by reducing the rate of turnover of the phosphate. This was tested by using P 32 labelled phosphate incorporation as an indicator of the phosphate turnover.

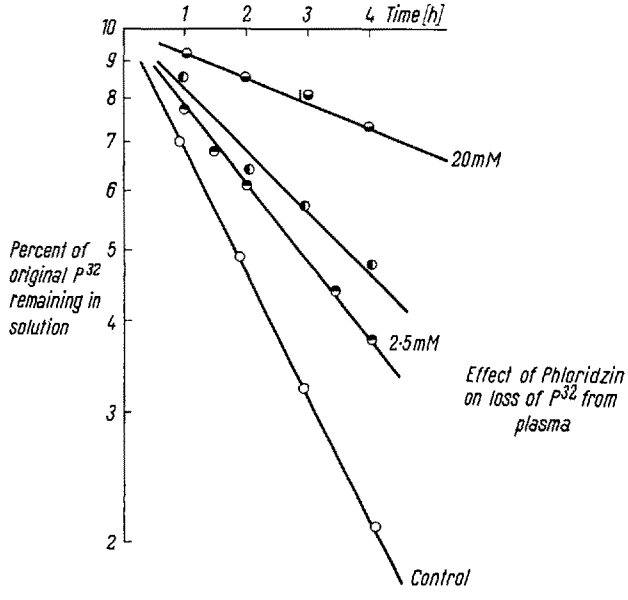

Fig. 1.—The fall of the P 32 phosphate content of plasma during incubation of the plasma with red cells, compared with and without phloridzin. In these experiments the P 32 phosphate was added to whole heparinised blood. The P 32 uptake by the cells is limited by the falling P 32 concentration in the medium.

Experiments were made either (a) by adding a trace of P 32 phosphate to whole heparinised blood and measuring the P 32 remaining in the plasma at intervals according to the method of PRANKERD and ALTMAN[3] or (b) by adding P 32 and carrier phosphate (1·2 *mM*) to a suspension medium in which the haematocrit was only 5% so that the P 32 level in the medium remained nearly constant, the activity associated with the cells being measured at intervals. Both methods showed that incorporation of the P 32 into the cells was reduced in the presence of phloridzin (Fig. 1 and 2). Using method (b) an inhibition by 0·03% w/v phloretin was found (Fig. 2). The phosphate esters and 'inorganic' phosphate of the cells after the method (a) were separated chromatographically and their radioactivities were measured (Table). Although the con-

[1] P. G. LEFEVRE, Symp. Soc. exp. Biol. *8*, 118 (1954).
[2] W. WILBRANDT, Symp. Soc. exp. Biol. *8*, 137 (1954).
[3] T. A. J. PRANKERD and T. ALTMAN, Biochem. J. *58*, 622 (1954).