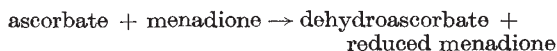


The reduction of vitamin K₃ by vitamin C appears to occur to a considerable extent, indicating that the following reaction is displaced to the right in the presence of neotetrazolium chloride:



This reaction between vitamin C and menadione is stimulated by tissue suspensions¹, but whether this stimulation is enzymic in character must await further investigation. The stimulatory power of homogenates is relatively insensitive to boiling. The stimulatory power of both rat liver and rat mammary gland homogenates is largely concentrated in the soluble fraction after removing the mitochondria and microsomes by differential centrifugation in 0.25 molar sucrose.

Reduced diphosphopyridine nucleotide reacted only slightly, if at all, with menadione to yield reduced menadione; reduced diphosphopyridine nucleotide had no direct effect on neotetrazolium chloride, as has been reported by Zöllner and Rothmund⁴. Typical results are shown in Table 1.

It seems possible, therefore, that in certain cases the fact that vitamin K₃ is involved in redox reactions can be demonstrated by the ready interaction of reduced menadione with neotetrazolium chloride.

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Effect of β -Hydroxybutyrate Injections on the Pancreatic Activity of Guinea Pigs

β -HYDROXYBUTYRATE has been found to cause hyperglycaemia, glycosuria and lowered glucose tolerance in guinea pigs and rabbits when injected in gradually increasing doses for some time^{1,2}. On injection in rabbits, this compound produces an initial state of hypoglycaemia followed in the long run by permanent hyperglycaemia. That this may be due to an initial state of hyper-insulinism, followed by hypo-insulinism due to an overwork atrophy of the *B*-cells, caused by continued stimulation has been suggested³⁻⁶.

Since β -hydroxybutyrate is an important intermediary metabolite, its effect on the normal pancreas is evidently very interesting, and the present series of investigations have therefore been started to elucidate its mechanism of action. The present report indicates the effect produced by continued injections of β -hydroxybutyrate (sodium salt) on the amylolytic and insulin activity of the pancreatic tissue of guinea pigs.

The preliminary experiments reported here were carried out on two groups of guinea pigs. One group was kept as control, and the animals of the other

Table 1. AVERAGE VALUES OF PANCREATIC AMYLASE AND INSULIN OF GUINEA PIGS SUBJECTED TO INJECTIONS OF β -HYDROXYBUTYRATE (SODIUM SALT)

No.	Days of injections	Amylase activity (mgm. of maltose/gm. of dry pancreas)	Insulin potency (percentage reduction/gm. pancreas)
1	0 (control)	654.1	14.72
2	12	596.7	86.10
3	25	457.2	—
4	36	100.8	30.6

group were given injections of sodium β -hydroxybutyrate (L. Light and Co., Ltd.). The initial dose was 50 mgm. per animal, and this was gradually increased to 100 mgm. per animal. At regular intervals the pancreas was removed from two animals of each group and a portion of pancreatic tissue assayed for amylase activity by Willstätter's method⁷. Another portion of the tissue was extracted for insulin⁸ and the extracted insulin assayed on rabbits by the method of Burn⁹. The results are given in Table 1.

Table 1 reveals that the pancreatic amylase activity decreases with gradual increase in the dosage of β -hydroxybutyrate injections, whereas the insulin potency shows an initial increase and is then followed by a decrease. The decrease in pancreatic amylolytic activity may be due to a possible lowering of the rate in the proteosynthetic activity of the acinar cells as a result of a possible pharmacological response produced by β -hydroxybutyrate, or it might be due to a physiological response of the organ to maintain the overall enzyme-hormone balance or both. The changes in insulin potency as observed are in conformity with the order of earlier observations (*loc. cit.*), namely, initial stimulation of *B*-cells followed by a possible overwork atrophy as a response to β -hydroxybutyrate injections.

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Leucine Aminopeptidase Activity in Mast Cells

DURING investigations of the behaviour of leucine aminopeptidase activity of blood-serum in patients with dermatoses and tumours¹⁻³, in a case of diffuse skin mastocytosis of a new-born, we detected heightened activity of leucine aminopeptidase. After mechanical friction of the affected skin, greater activity of leucine aminopeptidase occurred. From this we supposed a release of leucine aminopeptidase from the mast cell infiltrates of the skin. As is well known, in urticaria pigmentosa mechanical irritation of the skin lesions give rise to a degranulation of mast cells^{4,5} with liberation of histamine and serotonin. Indeed, there is no doubt that mast cells contain the enzyme leucine aminopeptidase. Using Burstone and Folk's histochemical method⁶, we were able to demonstrate a strong positive cytoplasmic