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## **The intestinal absorption of different sugars by adrenalectomized rats as tested in successive experiments**

by  
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As shown in previous papers, all the methods employed to investigate intestinal absorption, provoke in adrenalectomized rats a shocklike state, sufficient to account for the low results of absorption generally obtained in these animals with glucose and other selectively absorbed substances. Wilbrandt and Lengyel and later on other investigators showed, that in adrenalectomized rats intestinal absorption of xylose and of other non-selective sugars appeared to be undiminished, the animals always being sacrificed after one sole test.

Intestinal absorption of glucose, fructose, xylose and sorbose has been tested in four groups of adrenalectomized rats and in four groups of normal rats, three or more successive experiments of thirty minutes each being practiced in each animal by Sols and Ponz' method. In the first half-hour, average absorption of xylose and sorbose by adrenalectomized rats was not significantly different from that by normal rats; but in subsequent periods adrenalectomized rats showed a progressive diminution of absorption. The diminution of glucose absorption in adrenalectomized rats, initially already fifty per cent inferior to that in normal rats, showed in successive experiments a faster progressive fall. The decrease of fructose absorption in adrenalectomized rats, at first twenty-five per cent inferior, was more progressive than that of xylose and sorbose, but not so intense as that of glucose. In normal animals, absorption in successive experiments always remained constant.

It is concluded that when shock provoked in adrenalectomized rats by investigation techniques is fully developed, intestinal absorption of non-selective sugars diminishes in proportion to circulatory failure which delays removal and diffusion of substances absorbed. Absorption of selective sugars is more precociously and intensively disturbed, as soon as relative anoxia reduces the energetic metabolism and the activity of the epithelial cells.

I should now like to present some results which we have obtained on the effect of sodium Mono-iodo-acetate and other inhibitors upon absorption, as they are in connexion with this communication.

The inhibition of glucose absorption by Mono-iodo-acetate was considered initially as a proof that selective absorption occurred by a phosphorylation reaction. The said effect has been explained later on as an inhibition of phosphorylizing reactions in the energetic metabolism of the intestinal cells. We have recently obtained results which show that other factors must be taken into account to explain the effects of MIA and of other inhibitors.

Successive experiments were practiced by Sols and Ponz' method simultaneously in two intestinal loops of each animal, isotonic solutions of the same sugar being employed, whilst the inhibitor was applied only in one of the loops, generally the inferior one.

The absorption of glucose diminished when MIA was applied in the second and also in the third period, and the diminution accentuated progressively even when the inhibitor was no longer applied. It was equally observed in the loop where the inhibitor was never applied. The animals developed progressive symptoms of circulatory collapse and spontaneous death occurred generally within three hours. On account of this the diminution of absorption may be attributed to the general shock-like state provoked by MIA acting on the central nervous system.

On the contrary, Phlorrhizin provoked only a transitory diminution of glucose absorption, the normal value being restored in further successive experiments. In the control loop of the same animals, absorption remained always normal, shock or spontaneous death being never observed although the experiments were repeated for more than three hours.

The absorption of non-selective sugars like sorbose was not significantly modified either in the loop in which Phlorrhizin was applied or in the control loop.

By testing non-selective sugars like sorbose, MIA provoked

a small and progressive diminution of absorptior in the control loop in relation to circulatory failure; but in the loop where the inhibitor was applied an increase was observed immediately afterwards. The latter effect was transitory and may be attributed to a local and direct vasodilatory action of MIA, which we have confirmed macro-and microscopically. When explaining the effect of MIA and other inhibitors upon absorption results, the direct action of same on intestinal motility must also be borne in mind.

My intention in presenting these additional experiments is the point out that MIA may provoke a shock-like state, as observed in adrenalectomized animals, and that the low absorption results obtained in either case are due to the same cause and must not be attributed to the inhibition of a specific reaction of phosphorylation.