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Effect of Sodiummonoiodoacetate upon Metabolism of Streptobacterium casei.

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From the Laboratories of Physiology, Stanford University.

Present evidence indicates that sodiummonoiodoacetate is a fairly specific inhibitor of the lactic acid cycle in frog's muscle and in many other tissues.[1] However, the electrical, mechanical, and thermal aspects of the first few contractions of poisoned muscle differ little if at all from those of unpoisoned muscle, indicating that in this instance the Meyerhof cycle can be temporarily replaced by other mechanisms.

The authors studied the effect of this substance upon an organism in which lactic acid formation is practically the whole metabolic story. *Streptobacterium casei* was chosen,[2, 3] a pure culture of which was kindly furnished by Dr. C. B. van Niel of the Hopkins Marine Station.

The bacteria were grown on agar plates, 0.2% glucose and containing $CaCO_3$. Mass cultures so prepared were scraped from the agar and suspended in sterile solutions of 0.2% glucose, 0.3% sodium bicarbonate. Lactic acid production was measured in the usual way, using Warburg respirometers in a thermostat at 21.5°C.[4] The sodiummonoiodoacetate was placed in the bulb or "pear" of the manometer vessel, and added to the suspension about an hour after the beginning of the experiment, so that each preparation furnished its own control. Blank determinations were also run, and proper corrections made.

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[1] Meyerhof, O., "Die Chemischen Vorgänge im Muskel". Berlin, 1930.

[2] Orla-Jensen, S., "The Lactic Acid Bacteria". Copenhagen, 1919.

[3] Kluyver, A. J., and Donker, H. J. L., *Proc. Roy. Acad. Amsterdam*, 1925, **28**, 605.

[4] Warburg, O., "Uber den Stoffwechsel der Tumoren". Berlin, 1926.

Briefly, it was found that concentrations of sodiummonoiodoacetate in the respirometer vessels of up to 0.008% had little effect upon the rate or extent of lactic acid production, while at a concentration of 0.08% a change in slope was noted 65 minutes after the addition of the reagent, and at a concentration of 0.16% such change in slope developed 35 minutes after addition. Both the rate and final amount of lactic acid formed decreased as the concentration of sodiummonoiodoacetate was raised. That this was not merely an osmotic effect is indicated by the fact that no such changes were observed in the presence of isomotic concentrations of sodium acetate.

The action of sodiummonoiodoacetate on the metabolism of the organisms with a substrate of methyl glyoxal, hexose phosphate esters, and other carbohydrate precursors of lactic acid is now being determined.

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Action of Sodiummonoiodoacetate on Aerobic and Anaerobic Glycolysis in Blood.

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The rates of the glycolytic processes in blood vary from animal to animal and class to class, being fairly rapid in man, dog and sheep, but slow in ox and pig.[1] The important enzymes are largely present in the formed elements of the blood.[2] Lundsgaard[3] has shown that glycolysis is inhibited by monoiodoacetate in concentrations which do not interfere with many other enzymic processes. It is well known that glycolysis is more rapid in nitrogen than in oxygen (Pasteur reaction), probably because the oxidative and fermentative enzyme systems are competing for a compound such as methyl glyoxal, important in each type of catabolism. Accordingly it seemed possible that the Lundsgaard reagent would be more effective in inhibiting aerobic than anaerobic glycolysis, since in the latter case there is but one anti-glycolytic mechanism, in the former in a sense there are two.

To test this assumption tubes were made up as follows, using

[1] Macleod, J. J. R., *J. Biol. Chem.*, 1913, **15**, 497.

[2] Katayama, L., *J. Lab. and Clin. Med.*, 1926, **12**, 239.

[3] Lundsgaard, E., *Biochem. Z.*, 1930, **217**, 162.