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.

blood from a single animal (dog) for each series of experiments: Defibrinated blood, 10.0 ml., 1% glucose 5.0 ml., Sorensen's phosphate buffer (pH 7.2) 10.0 ml., distilled water or various concentrations of sodium monoiodoacetate to volume of 40.0 ml.

These tubes were placed in a thermostat at 37°C. for 4 to 5 hours. Oxygen-free nitrogen was passed through some of these, air through others, and the decrease in glucose then determined. With a concentration of Lundsgaard's reagent of 1.88 mg./ml. in the experimental tubes, the following data were obtained. A pair of experiments, a and b, were performed in each case.

	Loss of glucose in mg./ml. in 5 hours at 37°C.	
(1) Dog 14	a	b
Aerobic control	0.13	0.13
Anaerobic control	0.23	0.24
Aerobic-iodo acetate	0.00	0.00
Anaerobic-iodo acetate	0.17	0.17
(2) Dog 15		
Aerobic control	0.20	0.20
Anaerobic control	0.33	0.31
Aerobic-iodo acetate	0.00	0.00
Anaerobic-iodo acetate	0.23	0.24

With 1.4 mg. of iodo acetate per ml. in the experimental tubes.

(1) Dog 12		
Aerobic control	0.51	0.53
Anaerobic control	0.71	0.71
Aerobic-iodo acetate	0.18	0.18
Anaerobic-iodo acetate	0.27	0.26
	Loss of glucose in mg./ml.	
	a	b
(2) Dog 13		
Aerobic control	0.50	—
Anaerobic control	1.05	1.03
Aerobic-iodoacetate	0.12	0.10
Anaerobic-iodoacetate	0.20	0.22

The data indicate that anaerobiosis diminishes the inhibitory action of sodiummonoiodoacetate on glycolysis in dog's blood under the conditions of these experiments. The fate of the glucose disappearing, the rates of reaction, and the effect of arsenates and other agents modifying fermentative activity will be the subject of a subsequent communication.