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Dissociation Constants of Hexosephosphoric Acids.

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Hexosediphosphoric acid from yeast and hexosemonophosphoric acid from muscle (lactacidogen) were kindly made available from preparations by Professor Embden. Electrometric titration showed both stronger than pure phosphoric acid in the region of the first and, more conspicuously, in that of the second dissociation constant. In each case hexosemonophosphoric acid was the stronger.

From titration data with pure phosphoric acid a second constant at 18 degrees of pK_2 equals 6.82 was calculated, agreeing with the usual values given.

For hexosemonophosphoric acid the value similarly determined gave $pK_2 = 6.12$.

For hexosediphosphoric acid only an apparent value of the constant corresponding to the actual third and fourth together was determined at $pK_2 = 6.25$, corresponding with the value of Meyenhof and Suranyi of pK_2, 6.2.

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Influence of Thyroid and Insulin Treatment on Lactacidogen Synthesis in Muscle.

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Rabbits fed with 0.9 grams of dried thyroid tablets daily, with an abundant diet, showed rapid emaciation, followed by death in an exhausted state after 1 to 4 weeks. The muscle brei from such animals shows a great reduction, or almost complete failure of the ability to cause the disappearance of inorganic phosphoric acid under the

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influence of sodium fluoride. This phosphoric acid disappearance has been shown by Embden and his coworkers to result from its synthesis with carbohydrate into a hexosephosphoric acid ester, termed by Embden lactacidogen. It has been shown in the same Institute that impairment of the synthesis accompanies exhaustion, and certain pathological conditions involving muscular weakness. In the experiments here reported, the greatly reduced synthesizing ability appears associated with the diminution of muscle lactic acid, lactacidogen, and glycogen to almost negligible amounts.

Muscle brei of animals dying after insulin convulsions also suffered a great loss in synthesizing ability. The same result followed insulin treatment of animals already subjected to thyroid feeding. Under all these conditions the carbohydrate reserve is reduced to minimum values.

Hoet and Marks[1] observed the connection between rapidity of appearance of postmortal rigor and depleted glycogen, lactacidogen and lactic acid content of the muscles of such animals. Normally the postmortal decline in synthesizing ability is relatively slow. In our experiments the residual synthesizing ability immediately after death diminished to hardly detectable values within a half hour or an hour after the rapid onset of rigor.

Soluble starch was used for the necessary source of carbohydrate to effect the synthesis. Experiments with normal animals and the fact of the observed small synthesis usually requiring much more carbohydrate than the minimal muscle glycogen could supply indicate that the wheat starch is utilizable.

In these experiments it is impossible to distinguish whether carbohydrate lack or impairment of synthesizing ability may be regarded as cause of the condition. It is likely that each would contribute to the other, and so initiate the decline in vitality which finally leads to complete exhaustion of the animals from failure of the proper carbohydrate necessary for maintenance of a living muscle system.

This is a preliminary report.

[1] Hoet, J. P., and Marks, H. P., *Proc. Roy. Soc.* (London) B, 1926, C, 72-86.