evidence that local automatism ever develops in this part of the esophagus, at least as long as the vagi are intact. We have, therefore, in this case an illustration of antiperistalsis coordinated reflexly through the central nervous system.

3163

In vitro studies on ammonia and urea formation by tissues.

HAROLD C. GOLDTHORPE. (Introduced by F. C. Koch).

[*From the Department of Physiological Chemistry, University of Chicago, Chicago, Ill.*]

This investigation was undertaken in the hope that light would be thrown on the subject of desaminase action by the various tissues of the body. The subject is in a more or less unsettled state, some workers[1] even disclaiming a true deaminizing action in tissues, believing that the ammonia production is due to deamidase action. Still others[2] take the view that the amino acids instead of yielding ammonia, are attacked in the carbon chain itself, thus being broken down and oxidized, the products formed producing cyanic acid which can be converted into urea by the addition of ammonia formed by deamidase action.

In this work an attempt was made to study the action of ammonium salts, or of mixtures of amino acids and peptides, or of amino acids alone upon ammonia and urea formation or utilization by tissues *in vitro*.

The tissues used were obtained from recently killed dogs or from the abattoir, in this case using hog tissue. These were minced as soon as possible, mixed with a buffer phosphate solution and after an hour the juice was filtered and pressed out. Of this well mixed fluid, 25 cc. portions were taken and incubated with the additions referred to. All the necessary control estimations were made and the methods used were critically studied before using them on the problem. The tissues studied were liver and kidney.

[1] Luck, J. M., *Biochem. J.*, 1924, viii, 814.

[2] Weiner, E. A., "Chemistry of Urea," 1923, London.

The main observations made are as follows:

1. Ammonium salts added to the tissue extracts inhibit ammonia production. The chloride is most inhibiting. The lowered ammonia production is usually accompanied by increased urea production. Thus, in four experiments ammonium as acetate, phosphate or lactate stimulated urea production, while in two experiments all the ammonium salts retarded urea formation, the phosphate being the least inhibiting.

2. Half saturation of liver tissue extract with carbon dioxide stimulates urea production.

3. Addition of trypsin hydrolyzed casein to the tissue extract caused a marked increase in both ammonia and urea after incubation. The ammonia formed may come from either amide or amino groups in this case.

4. Acid hydrolyzed casein was also employed as a substrate free from amide nitrogen. When this was added to the tissue extract distinct increase in both ammonia and urea formation was observed with liver tissue. The ammonia formed suggests deaminase action but may possibly be due to a stimulating or activating action of the amino acids in the deamidase tissue extract. With kidney the ammonia is increased but urea is lost or destroyed.

5. Addition of large amounts of ammonium salts to liver extracts followed by immediate heat coagulation is accompanied by a loss of 82 to 87 per cent of the ammonia added. The ammonia is in part converted into urea and the remainder changed otherwise.

6. Addition of either small or large amounts of ammonium salts to kidney extract is accompanied, either in heat coagulation or during incubation, by a loss in urea and an increase in ammonia.