

The haploid number for both *E. Johnstoni* and *E. globulus* is eleven. There appears to be an interesting tendency to secondary pairing in *E. globulus* which makes the determination of the number in metaphase I and anaphase I less certain than with *E. Johnstoni*; but the diakinesis figures seem conclusive.

Work is proceeding with the view of tracing the course of the whole meiotic series of changes. These show interesting variations from the normal.

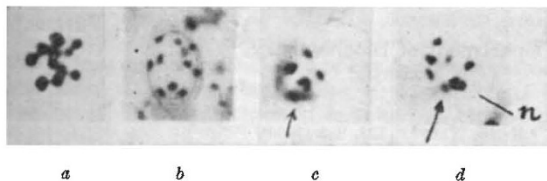


FIG. 1. (a) Metaphase I of *E. Johnstoni*. $\times 2000$. (b) Late diakinesis of *E. Johnstoni*. Two photographs of the same cell, focused on different planes, are superimposed. The nucleolus has disappeared. $\times 1000$.

(c) and (d) Two photographs of the same cell in early diakinesis (*E. globulus*). The nucleolus is seen in (d) in close conjunction with a chromosome (n). The chromosome marked by the arrow appears in both photographs. $\times 1000$.

Figs. 1 a and b show a typical metaphase I, and a composite photograph of diakinesis in *E. Johnstoni*; figs. 1 c and d are two photographs focused on different planes in a nucleus in diakinesis in *E. globulus*.

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Carbohydrate Metabolism

EXPERIMENTS which have been in progress in this laboratory during the past six months have had results very similar to those described by Krebs¹ in a recent letter. The formation of α -ketoglutaric acid in the course of pyruvic acid oxidation was made very probable by my earlier results². In recent experiments the formation of succinic acid from pyruvic acid, acetic acid and α -ketoglutaric acid could be demonstrated both anaerobically in minced brain and aerobically in the minced brain poisoned with malonic acid. Two explanations of these facts are possible: (1) there are two alternative paths of succinic acid formation from pyruvic acid, one leading to α -ketoglutaric acid by the condensation of two molecules of pyruvic acid and subsequent decarboxylation², the other starting with decarboxylation of pyruvic acid to acetic acid and subsequent condensation of two molecules of acetic acid³; (2) α -ketoglutaric acid is formed by condensation of one molecule of pyruvic acid and one molecule of acetic acid. It has not yet been possible to decide which is correct.

The decarboxylation of α -ketoglutaric acid by brain slices under anaerobic conditions depends on the presence of suitable hydrogen acceptors. The carbon dioxide evolution is increased on addition of a neutralized dye (Brilliant cresyl blue) and reduced to the initial value as soon as the dye is decolorized (Fig. 1). The carbon dioxide evolution due to the reduction of the dye⁴ is very small in the case of

Brilliant cresyl blue and can easily be allowed for. The amount of carbon dioxide given off by decarboxylation of α -ketoglutaric acid is almost exactly equivalent to half the amount of the added dye. Since two molecules of dye correspond to one molecule of oxygen, the R.Q. under these conditions is 1, the same as in aerobic experiments. Since the amount of hydrogen acceptor is the limiting factor of the

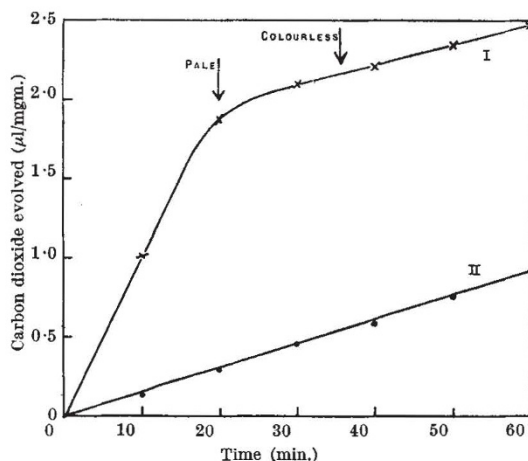
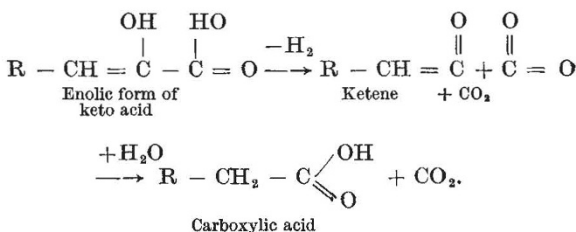


FIG. 1. Carbon dioxide evolution by slices of guinea pig brain in nitrogen in presence of *m/50* α -ketoglutaric acid; I, after addition of Brilliant cresyl blue (0.15 ml. of *m/50* sol.; carbon dioxide corrected for the amount due to reduction of the dye); II, without Brilliant cresyl blue.

decarboxylation, it is concluded that a dehydrogenation precedes the decarboxylation, thus excluding the possibility of an aldehyde as intermediate. In analogy to the oxidative deamination, the following mechanism of decarboxylation may be suggested:



Glucose, which provides suitable hydrogen acceptors, can replace the dye, as shown by anaerobic succinic acid formation from α -ketoglutaric acid. Pyruvic acid liberates carbon dioxide and forms succinic acid anaerobically even in absence of glucose or other hydrogen acceptors, no doubt because it acts itself as hydrogen acceptor, being partly reduced to lactic acid. The system β -hydroxybutyric acid \rightleftharpoons acetoacetic acid, quoted by Krebs, is only one of many reversible oxidation-reduction systems in the cell, which act as reserve hydrogen acceptors guaranteeing the progress of vital oxidations independently of the varying oxygen supply.

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¹ Krebs, NATURE, 133, 288 (1936).
² Weil-Malherbe, Biochem. J., 30, 665 (1936).
³ Thunberg, Skand. Arch. Physiol., 40, 1 (1920).
⁴ Reid, Biochem. Z., 242, 159 (1931).