Following development, only one pigmented red-orange band was present. This band was removed from the column and transferred as above to pure petroleum ether. Identification of the pigment was made by measuring the light absorption at 2-mμ intervals in wavelength from 375–700 mμ with a Beckman DU spectrophotometer. Fig. 1 shows the absorption curve obtained for the pigment isolated from *C. replicatum*. The absorption maxima are at 443, 468, and 498 mμ, and the curve is a characteristic one for the carotene, lycopene. TURIAN AND HAXO[4] published a similar curve with absorption peaks at 445, 470, and 500 mμ for lycopene which they extracted from the gametophytic plants of *Allomyces javanicus*. The authors are indebted to Dr. L. J. WILLOUGHBY for making this isolate of *C. replicatum* available for their study.

Department of Botany, Brown University, Providence 12, R.I. (U.S.A.)

MELVIN S. FULLER
JAMES E. TAVARES

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The effect of heart mitochondria on glycolytic systems from brain and heart

In recent years a number of investigators[1,2] have utilized the Pasteur effect in attempts to elucidate regulatory mechanisms involved in carbohydrate metabolism. Among these have been the studies of AISENBERG, REINAFARJE AND POTTER[3], AISENBERG[4] and more recently, CREMER[5]. AISENBERG *et al.* found that liver mitochondria inhibited glycolysis of a brain supernatant system, while CREMER described an inhibitory or stimulatory effect of rat-liver and -brain mitochondria, respectively, on the same system.

CREMER indicated that addition of mitochondria to a glycolytic system prepared from the same tissue would not result in an inhibitory effect in contrast to the studies by AISENBERG *et al.* in which mitochondria were added to a glycolytic system obtained from different tissues. CREMER further suggested that the stimulatory, rather than inhibitory, action of brain mitochondria on glycolysis of brain supernatant was probably due to the presence of a high hexokinase activity in brain mitochondria.

During our studies of oxidative phosphorylation of normal and failed guinea pigs[6], it became apparent that heart mitochondria from both the normal and failed animals produced a stimulation instead of a depression of a glycolytic system obtained from brain tissue.

The present report concerns studies the data of which appear to be pertinent to the above work. It describes experiments in which both normal and "failed" guinea-pig-heart mitochondria were added to either brain-or heart-supernatant glycolytic systems under aerobic conditions. The mitochondria from failed guinea pigs were

Abbreviations: ATP, adenosine triphosphate; DPN, diphosphopyridine nucleotide; EDTA, ethylenediaminetetraacetate.

obtained from animals in which experimental congestive heart failure was produced. These mitochondria were severely uncoupled with respect to oxidative phosphorylation. The normal heart and liver mitochondria were obtained from healthy guinea pigs and showed normal P:O ratios. In all cases a marked stimulation of glycolytic rate by the heart mitochondria was observed.

TABLE I

THE EFFECT OF MITOCHONDRIA ISOLATED FROM NORMAL GUINEA-PIG LIVER AND HEART AND FROM FAILED HEART ON AEROBIC GLYCOLYSIS OF BRAIN-SUPERNATANT SYSTEM

Glycolytic activity of the guinea-pig-brain supernatant (S_3) was measured by the disappearance of glucose[7] as well as the production of lactic acid[8]. The glycolytic medium contained 0.006 *M* glucose, 0.6 m*M* ATP, 0.009 m*M* DPN, 0.025 *M* $KHCO_3$; and 0.004 *M* $MgCl_2$ in a total volume of 3.0 ml. The pH was adjusted to 7.4. Incubation was carried out in a Dubnoff Metabolic Shaker at 38° for 30 min in an atmosphere of O_2–CO_2 (95:5). Each flask contained 1 ml of mitochondrial suspension (1:1 dilution with 0.16 *M* KCl or 0.25 *M* sucrose with 0.001 *M* EDTA and containing 3–5 mg protein/ml) and 0.6 ml supernatant (S_3) representing approx. 240 mg of original tissue. All values (average of 4 expts.) are changes during 30-min incubation.

Mitochondria	+ Δ *Lactate (μmoles)*		— Δ *glucose (μmoles)*	
	S_3 alone	*Mitochondria + S_3*	*S_3 alone*	*Mitochondria + S_3*
Liver	4.53	0.04	—	—
	5.96	2.78	5.50	4.00
Normal heart	6.60	15.75	6.10	12.80
	3.42	5.08	4.00	5.70
	2.80	6.30	3.40	7.20
	2.10	3.00	2.70	3.70
Failed heart	1.41	4.70	3.00	5.90
	3.75	8.80	3.70	6.20
	3.60	6.15	3.40	3.80
	3.29	7.26	3.40	5.60

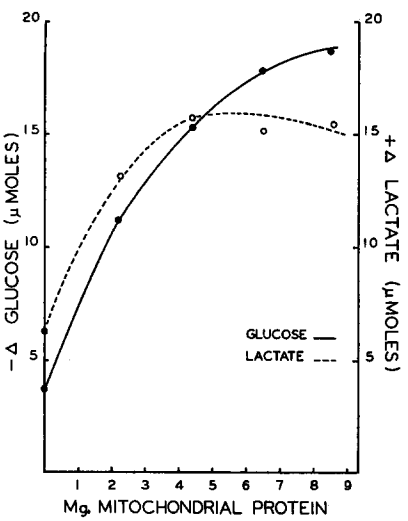

Fig. 1. The effect of graded amounts of guinea-pig-heart mitochondria on glycolysis of guinea-pig-heart supernatant. The glycolytic medium was the same as that in Table I, except for the addition of 40 μmoles potassium phosphate buffer, pH 7.4, to each flask.

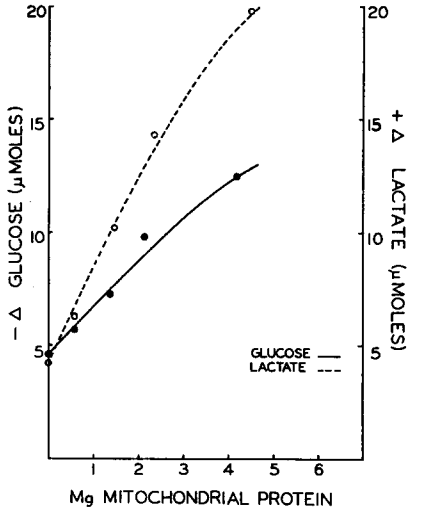

Fig. 2. The effect of graded amounts of guinea-pig-heart mitochondria on glycolysis of guinea-pig-brain supernatant. Glycolytic rate is expressed in μmoles glucose disappeared and in μmoles lactic acid produced. The glycolytic medium and incubation conditions were the same as in Table I.

The results presented in Table I show that guinea-pig-liver mitochondria, like the rat-liver preparations of AISENBERG *et al.*, effected a marked inhibition of glycolysis of brain supernatant. On the other hand, both normal and uncoupled heart mitochondria, doubled the rate of glycolysis. The relative amounts of mitochondria, glycolytic medium and the supernatant were similar to those employed by AISENBERG and by CREMER. The latter author, however, included inorganic phosphate while the former and the present authors did not. Inorganic phosphate increases the absolute rate of glycolysis but does not alter the stimulating effect of the heart mitochondria, as is indicated in Fig. 1.

The results plotted in Figs. 1 and 2 show that the stimulating effect of heart mitochondria on brain or heart supernatant is graded, *i.e.* with increasing amounts of mitochondrial protein, there is an increasing stimulation on glycolysis.

The addition of heart mitochondria to a glycolytic system prepared from the same tissues does not affect the results, as indicated in Figs. 1 and 2, where the stimulating effect of heart mitochondria is seen with either brain or heart supernatant (the glycolytic supernatant from heart was prepared in the same way as from brain[3]). Mitochondria isolated from severely failed guinea-pig hearts whose P:O ratio was reduced to less than 50 % of the control values still bring about the same type of stimulating action on glycolysis (Table I). This is in agreement with the data of AISENBERG *et al.* who found that uncoupling of liver mitochondria did not alter their results, and is in keeping with their suggestion that the Pasteur effect observed in this type of system is not explainable on the basis of a mitochondrial competition with glycolytic system for available inorganic phosphate or phosphate acceptor.

The stimulating effect of heart mitochondria on glycolysis is not explained by the presence of hexokinase as in brain, since heart mitochondria appear to contain little or no hexokinase activity (unpublished data).

The fact that mitochondria from brain, heart and certain types of tumor[3] bring about a stimulation of glycolysis of tissue supernatants (so called "negative Pasteur effect") rather than an inhibition as is observed with mitochondria from liver, kidney, and spleen, suggests the interesting possibility of the presence of biochemical differences among mitochondria from different tissues.

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Department of Pharmacology, Downstate Medical Center, State University of New York, Brooklyn 3, N.Y. (U.S.A.)

ARNOLD SCHWARTZ
KWANG SOO LEE

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