TABLE III. Influence of Composition of Media upon Protein Metabolism of 48 Hour Incubated Trichinella Larvae.

Amino acid	Glycine-2-C-14		Dl-alanine-2-C-14	
Media	Serum and Krebs-Ringer solution	Krebs-Ringer solution	Serum and Krebs-Ringer solution	Krebs-Ringer solution
cpm/mg of lyophilized larvae	19,634	23,362	670	959
cpm in protein from 1 mg lyophilized larvae	13,660	19,712	212	545
% C^{14} incorporated into larval protein	69.6	84.4	31.7	56.8

serum media, indicating that they utilized metabolites present in normal mouse serum.

The C^{14} activity from glycine-2-C-14 was incorporated into larval protein material in higher quantity than alanine-2-C-14 regardless of the media used. Glycine, in addition to its incorporation into protein peptides as glycine, may be converted into other amino acids which are eventually incorporated into protein. Furthermore, there is the possibility of incorporation of glycine into purines and pyrimidines of the nucleo-proteins. The higher values of C^{14} activity from alanine-2-C-14 in nonprotein larval material suggests that the larvae are able to convert alanine into nonprotein components, such a glycogen, glucose-1-PO_4 and others *via* the pyruvate pathway.

Summary. 1. *Trichinella spiralis* larvae incorporated C^{14} when cultured *in vitro* in a serum-Krebs-Ringer medium or Krebs-Ringer medium containing either dl-alanine-2-C-14 or glycine-2-C-14. 2. Carbon-14 analysis of the larvae revealed a progressive uptake of the C^{14}-labeled amino acids in both types of media through 48 hours of incubation. 3. The larvae incorporated more C^{14} activity (cpm) from glycine-2-C-14-labeled media than from dl-alanine-2-C-14-labeled media. 4. Of the total C^{14} incorporated by larvae cultured 48 hours in glycine-2-C-14-labeled media about 70 to 84% was precipitable by tungstic acid, and was presumably chiefly in the proteins. Larvae cultured in the dl-alanine-2-C-14-labeled media incorporated about 32-57% of their total C^{14} content into material precipitable by tungstic acid.

We wish to acknowledge the technical assistance of Rhoda Palter and Alina Jakubowski.

1. Stoner, R. D., and Hankes, L. V., *Exp. Parasitol.*, 1955, v4, 435.

2. Cohen, P. P., *Manometric Techniques,* Burgess Pub. Co., 1945, 193.

3. Van Slyke, D. D., Steele, R., and Plazin, J., *J. Biol. Chem.*, 1951, v192, 769.

4. Sinex, F. M., Plazin, J., Clareus, D., Bernstein, W., Van Slyke, D. D., and Chase, R., *ibid.*, 1955, v213, 673.

5. Albritton, E. C., *Standard Values in Blood,* Air Force Technical Report, No. 6039, 1951, 99.

Received February 15, 1956. P.S.E.B.M., 1956, v91.

Effect of Intravenous Administration of Lactate on Blood Pyruvate Level in Man. (22289)

MARK D. ALTSCHULE, GEORGE M. PERRIN, AND PHYLLIS D. HOLLIDAY.

Laboratory of Clinical Physiology McLean Hospital, Waverley, and the Department of Medicine, Harvard Medical School, Boston, Mass.

The role of circulating lactate in body metabolism is not entirely clear. On the one hand, the concept of the Cori cycle implies that most or all circulating lactate becomes glycogen in the liver; on the other hand, there is ample evidence that lactate can serve as a source of energy in some organs(1). Knowledge of whether the blood pyruvate

level changes after intravenous infusion of lactate should afford some evidence concerning whether or not lactate enters the general metabolic pool. Accordingly, the former was studied in normal human subjects.

Material and methods. Eight subjects, ranging in age from 18 to 30 years, were used; 3 were women. All were well nourished and had taken, in addition to their usual diets, added amounts of carbohydrate for at least three days before receiving the lactate infusion. The studies were made with subjects recumbent after a fast of 14 hours and a rest of at least half an hour. Each subject was given an intravenous infusion of 17.4 g of sodium d-lactate, dissolved in 500 ml of isotonic sodium chloride solution, in 30 minutes. Blood samples were taken before, and at 15-minute intervals for one hour after, the start of infusion. The method of Barker and Summerson(2) was used to measure blood lactic-acid concentration; the method of Seligson and Shapiro(3) was used to measure blood pyruvic and α-ketoglutaric acid concentrations. In addition, 4 subjects, including one who had received a lactate infusion, were given 50 g of glucose intravenously under similar experimental conditions; blood pyruvate concentrations were measured before and immediately after infusion.

Observations. The blood pyruvate level rose during and after infusion of sodium d-lactate (Fig. 1); the average rise at the end of infusion was 0.55 mg per 100 ml of blood, or 89% of average control value of 0.62 mg per 100 ml. After infusion blood pyruvate level usually fell more slowly than did blood lactate concentration (Fig. 1).

Blood α-ketoglutaric-acid concentration rose in 6 experiments (including 2 in which a slight initial fall occurred). It was unchanged in 1 and fell in 1 during and after lactate infusion (Fig. 2).

Intravenous administration of 50 g of glucose in 30 minutes caused no rise in blood pyruvate level in any of four experiments.

Discussion. Increasing circulating blood lactate concentration in normal man caused a striking increase in blood pyruvate level. There was no good correlation between rise

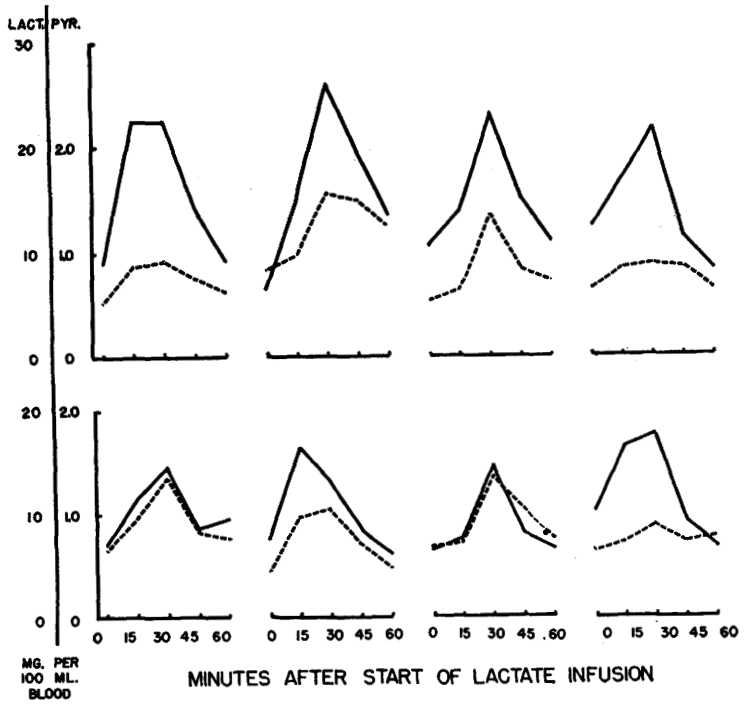

FIG. 1. Changes in blood lactic (solid line) and pyruvic (broken line) acid concentrations during and after infusion of sodium d-lactate.

in blood lactic acid level and that of blood pyruvic acid concentration. Of 3 subjects who showed smallest increases in blood pyruvate concentration, 1 excreted large amounts of lactate in the urine. The other 2, on the other hand, showed large increases in blood lactate; hence urinary excretion of that substance could not account for the small rises in blood pyruvate concentration.

The present data allow no conclusions about quantitative relations between lactate administration and pyruvate formation, for several reasons: 1) Varying amounts of lactate were excreted in the urine; 2) there is no evidence that rate of removal of pyruvate from circulation was the same in all cases; and 3) it is probable that rate of transformation of lactate to pyruvate was not uniform among the subjects. No information is available concerning the site where lactate was changed to pyruvate under conditions of the experiment.

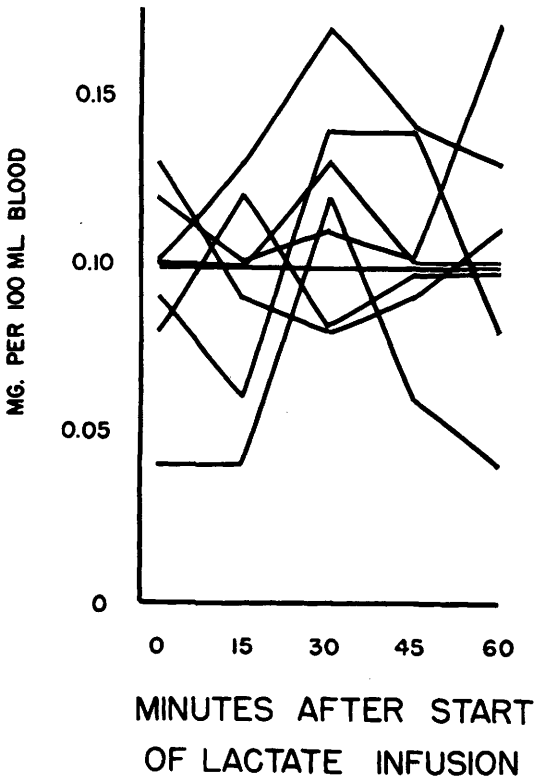

FIG. 2. Changes in blood α-ketoglutaric acid concentration during and after infusion of sodium d-lactate.

It is evident that circulating lactate can contribute rapidly and in significant degree to the general metabolic pool in resting, fasting, well-nourished man. The rise in blood α-ketoglutaric-acid level that usually accompanied the rise in pyruvic-acid concentration supports this conclusion. Actually, the rise in blood pyruvate concentration that occurred after a half-hour infusion of 17.4 g of lactate was far greater than that which occurred after a half-hour infusion of 50 g of glucose. This fact suggests that lactate enters the metabolic pool far more rapidly than does glucose.

Conclusion. The striking rise in pyruvate concentration that accompanies the infusion of lactate in fasting, resting human subjects indicates that circulating lactate contributes to the general metabolic pool.

1. White, A., Handler, P., Smith, E. L., and Stetten, De W., Jr., *Principles of Biochemistry,* McGraw-Hill Book Co., Inc., 1954.

2. Barker, S. B., and Summerson, W. H., *J. Biol. Chem.,* 1941, v138, 535.

3. Seligson, D., and Shapiro, B., *Analyt. Chem.,* 1952, v24, 754.

Received February 15, 1956. P.S.E.B.M., 1956, v91.