Acetylphosphate is not oxidized by a dialysed brain dispersion[3] (pigeon) in the presence of phosphate, fumarate, and adenylic acid. A typical experiment is given in Table 1. Each sample contained 1·5 ml. enzyme (dialysed 4 hours), phosphate buffer (*p*H 7·3), 1·2 mgm. sodium fumarate, and 1 mgm. adenylic acid (total volume of 2 ml.).

Table 1.

	μl oxygen uptake in 30 minutes (air, 38°).		
Blank	Acetyl-P (1·12 mgm. P)	2 mgm. sodium pyruvate	Acetyl-P + sodium pyruvate
89	66	496	450

Experiments with muscle extract, in which a rapid transfer of phosphate to adenylic acid from phosphate donators such as phosphopyruvic acid takes place, have been negative with acetylphosphate. The experiments of Table 2 were carried out with an acetone powder extract from rabbit's muscle dialysed 6 hours. Each sample contained (in 1·5 ml.) 1 ml. extract plus phosphate buffer (*p*H 7·3), adenylic a id (with 0·35 mgm. P), and either phosphoglyceric a id (0·8 mgm. P) or acetylphosphate (1·95 mgm. P) as phosphate donators. Phosphate was determined colorimetrically by the method of Lohmann and Jendrassik.

Table 2.

P donator	Incubation at 30°	mgm. direct P	mgm. pyrophosphate P
Phosphoglyceric acid	5 min.	0·37	0·49*
,, ,,	30 ,,	0·38	0·56*
Acetyl-P	30 ,,	2·16	0·00
,,	60 ,,	2·30	0·04

*Theoretical value for complete phosphorylation of added adenylic acid to adenosine triphosphate, 0·70 mgm. P.

These results indicate that acetylphosphate is neither an intermediate in the oxidation of pyruvic acid by brain nor does it act significantly as a donator of phosphate to adenylic acid in muscle extract. They do not exclude the possibility that acetylphosphate might be an intermediate in the dehydrogenation of pyruvic to acetic acid by preparations of *Bacterium Delbrückii*, but the fact that some acetic acid is produced when pyruvate is oxidized by brain[4], and the indirect nature of Lipmann's evidence, make it more probable that there is some other, as yet unknown, phosphorylated intermediate common to both bacteria and animal tissues. The existence of such an intermediate is strongly suggested by the observation of Banga *et al.*[3] that adenylic acid is involved in a stage of pyruvate oxidation by brain beyond that of oxidative decarboxylation.

We are indebted to the Nuffield Trustees and the Rockefeller Foundation for grants in aid of this work.

S. OCHOA.
Department of Biochemistry, R. A. PETERS.
Oxford. Sept. 22. L. A. STOCKEN.

[1] NATURE, 144, 381 (1939).
[2] *Ann.*, 131, 165 (1864).
[3] Banga, Ochoa and Peters, NATURE, 144, 74 (1939).
[4] Long, *Biochem. J.*, 32, 1711 (1938).

Oxidation of Methyl Esters of Monocarboxylic Fatty Acids by Normal and Neoplastic Tissue

THE metabolism of the fats in tumours has been little studied. Haven[1], feeding elaidin to rats, the method used by Sinclair[2] in classifying phospholipids into the metabolic and the non-metabolic type, found that the phospholipids of tumours are mainly of the non-metabolic type, having to do with cellular structure rather than with the burning of fatty acids for energy. According to Kisch[3], the Jensen sarcoma does not oxidize the low fatty acids. I have observed[4] that the oxygen uptake of slices of various experimental and human tumours is not increased when saturated and unsaturated monocarboxylic fatty acids in the form of sodium salts are added to the medium, and that in no instance were acetone (ketone) bodies formed. The β-hydroxybutyric acid, on the other hand, is oxidized into aceto-acetic acid.

The latter data exclude the fact that the neoplastic cell can β oxidize fatty acids. My aim was to find out whether the tumour could oxidize fatty acids by means of ω-oxidation.

I have studied manometrically the oxidation of methyl esters of monocarboxylic fatty acids from C_1 to C_8 in comparison with the corresponding free acids, in the presence of tissue slices of liver, kidney, spleen, brain cortex, striated muscle, Rous I sarcoma, round cell chicken sarcoma, and Ehrlich adenocarcinoma. It has been found that, without exception, the oxygen consumption of normal tissue slices is increased by fatty acids esters much more than by the free acids. For example, some esters increased the oxygen uptake in the liver eight times, in the brain cortex eleven times, and in the spleen seventeen times more than the corresponding acids. As a rule, the oxygen uptake increases as the fatty chain increases up to a maximum at about C_6–C_7, and decreases at about C_8, owing to the low solubility in water of this ester. With methyl formiate—in connexion with which a true ω-oxidation is obviously impossible—it is the ester which is mostly burnt in certain tissues (spleen, brain, striated muscle). Only the fatty acid component of the esters is burnt, whereas the alcohol component is not.

Neoplastic tissues do not oxidize the free fatty acids but actively oxidize the corresponding esters. The neoplastic cell in which enzymes of β-oxidation are lacking—with the exception of the enzyme which transforms the secondary alcoholic group of the β-hydroxybutyric acid into a ketonic group—can, by means of the ω-oxidation enzymes, use the fats as a source of energy.

Institute of General Pathology, E. CIARANFI.
University of Naples,
Italy. Sept. 14.

[1] Haven, F. L., *J. Biol. Chem.*, 118, 111 (1937).
[2] Sinclair, R. G., *J. Biol. Chem.*, 111, 515 (1935).
[3] Kisch, B., *Biochem. Z.*, 253, 379 (1932).
[4] Ciaranfi, E., *Amer. J. Cancer*, 32, 561 (1938).

A Cell-free Enzymatic Model of *l*-Amino-acid Dehydrogenase ('*l*-Deaminase')

ATTEMPTS to obtain cell-free enzyme preparations of animal origin effecting deamination of the *monocarboxylic l*-amino acids have hitherto been practically unsuccessful. (Specific enzymes deaminizing *d*-amino-acids and *l*-amino *dicarboxylic* acids have been obtained in solution and purified by several authors.) H. A. Krebs[1] attributes the deamination of *l*-amino-acids to an enzyme, termed '*l-deaminase*', depending for its activity upon the integrity of cell structure and rapidly deteriorating when the cells are damaged.

It appears from the data of Krebs that '*l*-deaminase' is even more sensitive to the dilution of cell-contents in ground tissue suspensions than to cell destruction as such, and further, that the *di*carboxylic amino-acids are oxidized at a very much higher rate than the *mono*carboxylic ones (in fact, *l*-aspartic acid was the substrate used in most experiments).

© 1939 Nature Publishing Group