To determine the quantity of oxalacetate present in the reaction mixtures, samples were acidified with trichloracetic acid, a carbon dioxide–nitrogen mixture was bubbled through to remove trace amounts of $^{14}CO_2$, carrier oxalacetate was added and the samples were decarboxylated with either aluminium ions[5] or with aniline citrate[6]. The liberated $^{14}CO_2$ was collected in a solution of sodium hydroxide, converted to carbonate, and counted on planchets. Results of such experiments showed that the radioactivity originally present and due to aspartate was not affected by the decarboxylation, and that oxalacetate was formed in the reaction mixtures in concentrations about 1·3 times that of aspartate. Thus, the aspartate which was counted on heat-dried planchets represented only about four-tenths of the total carbon dioxide fixed.

When either glycerate-3-phosphate or glycerate-2-phosphate was added to the reaction mixture described earlier, oxalacetate and aspartate were formed but at a slower rate than when phospho-enol-pyruvate was used. The glycerate phosphates were probably first converted to phospho-enol-pyruvate by the action of glyceric acid mutase and enolase in the chloroplast extract. When either ribulose-1-5-diphosphate or ribose-5-phosphate plus adenosine triphosphate was added, and the reaction mixture chromatographed, labelled carbon appeared in both glycerate-phosphate and aspartate. In view of the fact that these substances are among the compounds formed when whole isolated chloroplasts fix carbon dioxide in the light[7] and since Fuller and Anderson[8] suggest the importance of aspartic acid formation as an early photosynthetic event in photosynthetic purple sulphur bacteria, we find it interesting that carboxylations in chloroplasts can lead to the rapid formation of an amino-acid.

We are indebted to Dr. Clinton Ballou for the sodium salts of phospho-enol-pyruvate and glycerate-2-phosphate.

LAWSON L. ROSENBERG
JOHN B. CAPINDALE
F. R. WHATLEY
Laboratory of Plant Physiology,
Department of Soils and Plant Nutrition,
University of California, Berkeley.
Nov. 12.

[1] Whatley, F. R., Allen, M. B., Rosenberg, L. L., Capindale, J. B., and Arnon, D. I., *Biochim. Biophys. Acta*, 20, 462 (1956).
[2] Bandurski, R. S., and Greiner, C. M., *J. Biol. Chem.*, 204, 781 (1953).
[3] Tchen, T. T., and Vennesland, B., *J. Biol. Chem.*, 21, 533 (1953).
[4] Utter, M. F., and Kurahashi, K., *J. Biol. Chem.*, 207, 787 (1954).
[5] Krebs, H. A., *Biochem. J.*, 36, 303 (1942).
[6] Edson, N. L., *Biochem. J.*, 29, 2082 (1935).
[7] Allen, M. B., Arnon, D. I., Capindale, J. B., Whatley, F. R., and Durham, L. J., *J. Amer. Chem. Soc.*, 77, 4149 (1955).
[8] Fuller, R. C., and Anderson, I. C., *Proc. Plant Physiol.*, 32, 16 (1957).

Histogenesis of Osteoclastomata

Two of the main problems concerning the histogenesis of giant cell tumours of bones have been: (1) the nature and function of the giant cell—this has been variously classified as an endothelial cell, a foreign body giant cell, a megakaryocyte, an osteoclast or as a giant cell formed by agglomeration or fusion of stromal cells; (2) the histogenesis of the giant cell—the overwhelming consensus of opinion among the supporters and opposers of the osteoclast nature of the giant cell[1–5] suggests that the giant cells are formed from the spindle-shaped stromal cells.

In two cases of osteoclastomata recently investigated I have observed a strong acid phosphatase in the cytoplasm of the giant cells. The stromal cells gave a negative reaction. The presence of this enzyme was demonstrated both with the lead phosphate and azo-dye coupling techniques. Inhibition by 0·01 *M* sodium fluoride excluded the possibility of non-enzymatic impregnation of the giant cells[7]. Using *o*-acetyl-5-bromoindoxyl as substrate for esterases, both the stromal and the giant cells were negative for these enzymes.

These findings suggest that the giant cells are not in any way related to the stromal cells. It might be argued that the stromal cells are precursors of the giant cells with different enzyme reactions, but all the histochemical and biochemical evidence points to the fact that the enzyme systems of parent cells and their descendants are the same.

It has also been stated that the giant cells, because of their apparent age, show signs of early degeneration[2]. Histochemically, however, it would appear as if the giant cells are very active cells (probably osteoclasts) and the stromal cells inactive with regard to phosphatase and esterase activities.

Details concerning these findings and further experimental work will be published elsewhere.

W. J. PEPLER
Institute for Pathology, Beatrix Street,
Pretoria. Oct. 10.

[1] Evans, R. W., "Histological Appearances of Tumours", 141 (1st edit. Livingstone, Edinburgh and London, 1956).
[2] Geschickter, C. F., and Copeland, M. M., "Tumours of Bone", 295 (3rd edit., Lippincott, London, 1949).
[3] Willis, R. A., "Pathology of Tumours", 680 (1st edit., Butterworth and Co., London, 1948).
[4] Lichtenstein, L., "Bone Tumours", 140 (1st edit., Mosby Co., St. Louis, 1952).
[5] Jaffé, H. L., Lichtenstein, L., and Portis, R. B., *Arch. Path.*, 30, 993 (1940).
[6] Pearse, A. G. E., "Histochemistry: Theoretical and Applied", 460, 461, 463 (1st edit., Churchill, London, 1953).
[7] Gomori, G., "Microscopic Histochemistry—Principles and Practice", 192 (1st edit., University of Chicago Press, Chicago, 1952).

Inhomogeneity of α-Casein from Goat Milk

IT is now known that α-casein from cow milk, which appears electrophoretically homogeneous under many different conditions, consists of at least two compounds which have been designated α- and *k*-casein[1]. α-Casein from goat milk, which has similar properties to that from cow milk[2] and the same phosphorus content[3], showed signs of inhomogeneity during electrophoresis by the moving-boundary method[3]. Using zone electrophoresis on filter paper it has now been found that the α-component of freshly prepared goat casein does consist of at least two electrophoretically distinct fractions which, until further characterization, will be called α_1- and α_2-casein in order of decreasing electrophoretic mobility.

The experiments were made with unfractionated casein, which was obtained in the usual way by acid precipitation, and with α-casein, which was prepared from it by Warner's method[4], and had the same phosphorus and nitrogen content as α-casein prepared from cow milk.

Electrophoretic runs were made on Whatman No. 3 paper with veronal buffer, *p*H 7·6, $\mu = 0{\cdot}1$, at 3 V./cm. for 15 hr. After staining with naphthalene black 12*B* 200 the electropherograms were scanned. The scanning patterns obtained