
Two effects of administration of lipopolysaccharide (LPS) to C57BL mice were studied: (1) changes in levels of plasma hemopexin (hpx) and (2) phagocytic uptake in liver and spleen of \( ^{51} \text{Cr} \)-labeled sheep red cells (SRC). Levels of hpx were markedly elevated 24 hr after injection of LPS and returned to normal values 2 wk later. Hepatic phagocytosis of SRC was significantly impaired between 2 to 7 days after LPS treatment, while after 2 wk normal function was recovered. Splenic phagocytosis was less markedly affected. There was a significant increase of splenic weight from two to seven days after LPS injection, whereas hepatic weights were unchanged. The possible relationship between elevation of hpx and interference with phagocytic function resulting from administration of LPS is considered and the findings are discussed within the framework of previous investigations of the interactions of LPS with the reticuloendothelial system.—B.R.

CORRESPONDENCE

To the Editor:

In a recent article, Tisman and Herbert\(^1\) reported that bone marrow cells from patients with untreated pernicious anemia show defective uptake of 5-methyltetrahydrofolate (methylfolate) and that this can be corrected by the in vitro addition of vitamin B\(_{12}\). In this paper, a previous study by Das and Hoffbrand\(^2\) was mentioned in which uptake of methylfolate by vitamin B\(_{12}\)-deficient PHA-stimulated lymphocytes was found to be defective. Tisman and Herbert considered these previous studies to be invalid, however, because of an apparent discrepancy of 250 times between the uptake of folic acid and of methylfolate by normal PHA-transformed lymphocytes. After correspondence between us, however, it has become clear that this discrepancy did not exist but appeared to be so because of the far greater quantity of methylfolate than of folic acid put into the lymphocyte cultures by Das and Hoffbrand (because of the low specific activity of the methylfolate available to them). Thus, the results of Das and Hoffbrand suggesting defective uptake of methylfolate by vitamin B\(_{12}\)-deficient cells remain valid. The present experiments of Tisman and Herbert\(^1\) confirm these results using physiological rather than supra-physiological amounts of methylfolate, using bone marrow cells instead of lymphocytes, and also extend the observations by showing that defective uptake of methylfolate by vitamin B\(_{12}\)-deficient cells can be reversed in vitro by addition of vitamin B\(_{12}\).

Department of Haematology, Royal Postgraduate Medical School, Du Cane Road, London W12 OHS.

V. HERBERT, M.D.
Department of Pathology, Columbia University, and Veterans Administration Hospital, 130 West Kingsbridge Road, Bronx, New York, 10468

REFERENCES


To the Editor:

We wish to answer Dr. Popesco's letter to the editor (Blood 41, No 4, 1973). We were aware of the work of Dr. Popesco and have mentioned it, as well as those of many others after the pioneer’s work of Diguglielmo and of Undritz. However, we have called the attention to the help of electron microscopy study in identifying cells that are often confused with lymphocytes. The identification of the specific peroxidase activity in the perinuclear space and in short segments of endoplasmic reticulum demonstrates that these cells are micromegacaryocytes when the cytoplasm is limited to a narrow ring around the nucleus.

J. BRETON GORIUS
B. DREYFUS
C. SULTAN
Unité INSERM U. 91
Hôpital Henri Mondor
94010—Creteil—France

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The Blood Resource Branch of the National Heart and Lung Institute has a limited supply of urokinase which is available for free distribution to the Scientific community for use in well-designed, controlled research or clinical protocols. The supply includes urinary urokinase which is suitable for in vivo experiments on an I.N.D. basis and tissue culture urokinase suitable for in vitro or animal work. Enquiries or requests including well-designed protocols, quantities required, and justifications should be forwarded to: Blood Resource Branch, National Heart and Lung Institute, Building 31, Room 4A-03, National Institutes of Health, Bethesda, Md. 20014; Telephone 301-496-5911.

KARNOFSKY FELLOWSHIP

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