Authors' Summaries of Articles Accepted September, 1965

Schmid, J. B., Kiely, J. M., Tauxe, W. N., and Owen, C. A.: Synthesis in human bone marrow cells: a study of 12 normal subjects and 12 patients with lymphoplasmocytic disorders. Submitted March 26, 1965; accepted for publication Sept. 11, 1965.

In 12 normal subjects, DNA and RNA synthesis by individual marrow cells was studied in vitro by autoradiography after short-term incubation with tritiated nucleosides. The DNA labeling index and RNA synthesis rate were highest in normal immature cells. The DNA index was zero in all mature cells, including small lymphocytes and plasma cells. In the most actively proliferating normal immature cells, the RNA synthesis rate was about five times higher than in normal plasma cells. Since both normal immature cells and plasma cells are characterized by a high content of RNA and active protein synthesis, the results of the present study suggest that a high RNA turnover may not be necessary for protein synthesis to proceed in nonproliferating, protein-producing marrow cells.

In ten patients with myeloma and in two patients with primary macroglobulinemia, the abnormal plasma cells were found to be labeled with H^3-thymidine in each instance. The RNA synthesis rate was about three times greater in plasmocytoid myeloma cells than in mature plasma cells of normal marrows, and also about three times greater in lymphocytoid myeloma cells than in mature lymphocytes of normal marrows. The abnormal cells in the lymphoplasmocytic disorders apparently differ from their counterparts in normal marrows not only by a higher proliferative potential, but also by a much greater rate of RNA synthesis.

In actively dividing normal bone marrow cells, high values for both DNA and RNA synthesis were found. By contrast, in the neoplastic lymphoplasma cells studied, a discrepancy was noted between relatively low values of DNA synthesis and high values of RNA synthesis. Production of abnormal proteins within long-lived neoplastic plasmocytic cells may account for this finding.

Sandborn, E. B., LeBuis, J-J., and Bois, P.: Cytoplasmic microtubules in blood platelets. Submitted July 8, 1965; accepted for publication Sept. 11, 1965.

Regularly arranged cytoplasmic microtubules are present in blood platelet fixed in liver sinusoids during aldehyde perfusion of the animal. This finding, in addition to other recent reports on the cytoplasmic microtubule, suggests that these organelles might be found in all cytoplasm if the ultrastructure is preserved adequately.

Note: Subsequent to the submission of this article a description of filaments and microtubules in spread platelets by Bessis and Breton-Gorius[18] appeared. The greater number of filaments in their illustrations suggests a breakdown of some of the microtubules to the smaller filaments.[11] These authors speculate on the possibility of a contractile function for microtubules.

Rosse, W. F., and Waldman, T. A.: Factors controlling erythropoiesis in birds. Submitted June 7, 1965; accepted for publication Sept. 11, 1965.

1. Erythropoiesis in birds is stimulated by bleeding and environmental hypoxia and is suppressed by induced polycythemia, indicating that the physiologic response is similar to that in mammals.

2. This response is mediated through a humoral factor which stimulates erythropoiesis of birds but not of mammals. Similarly, the erythropoietin of mammals does not stimulate the erythropoiesis of birds.

3. Although both the erythropoiesis stimulating factor of birds and of mammals appear to require an intact protein structure for biological activity, the erythropoietin of birds differs from that of mammals in that it is not destroyed by sialidase and does not lose its activity when reacted with antibody to human urinary erythropoietin.

Gabuzda, T. G.: Hemoglobin H and the red cell. Submitted June 9, 1965; accepted for publication Sept. 11, 1965.

Hb H is found in red cells as a result of an imbalance in the synthesis of the polypeptide units of globin. A report of trace amounts present in normal umbilical cord blood suggests the possibility of a slight physiologic imbalance during embryonic life, either due to a slight deficiency of alpha chains or an excess of beta chains.[13] Larger amounts of Hb H presumably stem from a primary genetic deficiency of alpha chain synthesis—i.e., alpha thalassemia, although the precise genetic definition of this disorder is far from clear. Hb H may thus be viewed as a surplus product, but it is of no value in oxygen transport, and, because of its labile nature, its presence in the erythrocyte is detrimental to the cell life span. An understanding of the pathogenetic mechanisms involved in H-thalassemia may give insight into those of hemolytic anemia associated with other unstable hemoglobins, as well as into those of homozygous beta thalassemia, a disorder in which it has been suggested that labile globin fractions are also of pathogenetic significance.[18,64]

Keighley, G., and Lowy, P. H.: Actinomycin and erythropoiesis and the production of erythropoietin in mice. Submitted July 19, 1965; accepted for publication Sept. 11, 1965.

In $B6D2F_1$ female mice a single dose of 10 μg. of actinomycin will suppress normal erythropoiesis. In polycythemic mice 2 μg. is enough to prevent the stimulatory effect of 0.25 A of erythropoietin. The curves of suppression and recovery versus time support the hypothesis that erythropoietin acts for a short time in an early stage of erythropoiesis; after this early stage developing cells are no longer so sensitive to actinomycin. Recovery, even from repeated doses, is rapid and adequate. Amounts of actinomycin which are adequate to abolish erythropoiesis do not prevent the new appearance of erythropoietin in the plasma of hypoxic mice.

Winkelstein, A., Sparkes, R. S., and Craddock, C. G.: Trisomy of Group C in a myeloproliferative disorder. Submitted June 29, 1965; accepted for publication Sept. 11, 1965.

A patient with an atypical myeloproliferative syndrome characterized by

leukocytosis with myeloid immaturity, ineffective extramedullary erythropoiesis, overt hemolysis, and low leukocyte alkaline phosphatase activity demonstrated a trisomy in the C group of bone marrow metaphase chromosomes.

Kotani, M., Seiki, K., Yamashita, A., and Horii, I.: Lymphatic drainage of thymocytes to the circulation in the guinea pig. Submitted May 19, 1965; accepted Sept. 11, 1965.

1. An extensive network of lymphatic vessels coming from the thymus of the guinea pig and carrying thymocytes to the general circulation has been demonstrated.

2. Local accumulations or infiltrations of cells at the surface of the lobule or in the interlobular septa have been observed through which thymocytes appear to leave the thymus.

3. Irregular, tortuous canaliculi, resembling a lymph sinus in a lymph node, seem to play an important role in transporting thymocytes from these accumulations to the typical lymphatic vessels.

4. The output of thymocytes via lymphatic vessels to the general circulation is about 12.1×10^6 per day.

Abernathy, M. R.: Döhle bodies associated with uncomplicated pregnancy. Submitted April 13, 1965; accepted for publication Sept. 8, 1965.

Döhle bodies have been described in the neutrophilic leukocytes of uncomplicated gravid women. Of 500 blood films screened from uncomplicated pregnancies, all but nine exhibited Döhle bodies. Serial studies of 15 uncomplicated pregnancies followed throughout all three trimesters, and for 6 to 8 weeks postpartum, repeatedly showed Döhle bodies. No Döhle bodies were found in the leukocytes of the 500 normal nonpregnant women used as a control.

The appearance of Döhle bodies in uncomplicated pregnancy and their relationship with the inclusions found in various states is unexplained.

Jepson, J. H., and Lowenstein, L.: Inhibition of erythropoiesis by a factor present in the plasma of patients with erythroblastopenia. Submitted June 14, 1965; accepted for publication Sept. 8. 1965.

An erythropoietic inhibitor was found in the plasma of each of two patients with erythroblastopenia. Because these plasmas exhibited similar biological behavior to that of antisera to ESF, it is postulated that the inhibitory effect of these plasmas may be due to the formation of antibody directed against ESF.

Ardeman, S., Chanarin, I., Jacobs, A., and Griffiths, L.: Family study in Addisonian pernicious anemia. Submitted June 24, 1965; accepted for publication Sept. 8, 1965.

Addisonian pernicious anemia was present in six of nine siblings (Generation II). All nine siblings had evidence of gastric pathology as judged by the presence of antibodies against parietal cells, abnormalities in the gastric biopsy, and diminution of secretion of acid and intrinsic factor.

Parietal cell antibodies were present in 10 of 17 offspring of these nine siblings who were available for study. Gastric biopsy in the older members of this generation (III) showed atrophic gastritis; some of the younger members showed superficial gastritis.

Loss of intrinsic factor was always accompanied by loss of gastric secreting cells and there was no evidence that decline of intrinsic factor secretion occurred independently of hydrochloric acid.

Aho, K., and Christian, C. L.: Studies of incomplete antibodies. I. Effect of papain on red cells. Submitted June 15, 1965; accepted for publication Sept. 8, 1965.

A variety of typical agglutinating γG antibodies agglutinated papain-treated cells in a uniform manner in about sixteen-fold higher dilutions than non-treated cells. With γM antibodies the corresponding titer increase was on the average four-fold. The papain-treatment of cells also increased the titers of cold agglutinins, phytohemagglutinins, and preparations of PVP. Anti-globulin consumption experiments revealed that no more antibody was fixed from the same volume of antiserum onto papain-treated than onto nontreated cells. Possible mechanisms underlying enhanced agglutination of enzyme-treated cells were discussed. It is suggested that the same type of antibody may react with papain-treated and nontreated cells, but that a smaller number of antibody molecules are needed for agglutination of papain-treated cells because of altered surface properties of treated erythrocytes.

Nachman, R. L., Horowitz, H. I., and Silver, R. T.: Platelet amino acid levels in essential thrombocytosis. Submitted July 23, 1965; accepted for publication Sept. 20, 1065.

Studies on the platelets from three patients with essential thrombocytosis were presented. The intraplatelet free amino acid levels were elevated and the pseudohyperkalemic phenomenon was present. In one subject the pseudo-hyperkalemic phenomenon was reversed after a therapeutic remission, but the elevation of amino acids persisted. The data suggest that a qualitative abnormality may exist in the platelets of patients with essential thrombocytosis.

Carper, H. A., and Hoffman, A. A.: The intravascular survival of transfused canine Pelger-Huët neutrophils and eosinophils. Submitted Aug. 6, 1965; accepted for publication Sept. 25, 1965.

1. A case history of the Pelger-Huët leukocyte anomaly in the dog is presented. The trait appears similar to that observed in humans and rabbits.

2. Disappearance from the circulation of transfused Pelger-Huët neutrophils was exponential with a $+\frac{1}{2}$ value of 4.8 hours.

3. Transfused Pelger-Huët eosinophils rapidly disappeared from the circulation with a $+\frac{1}{2}$ value approaching 30 minutes.

Aksoy, M., Camli, N., and Erdem, S.: Roentgenographic bone changes in chronic iron deficiency anemia. A study in 12 patients. Submitted Aug. 17, 1965; accepted for publication Sept. 15, 1965.

Radiologic bone changes of the skull, long and short bones similar to those of thalassemia, are described in 12 patients with chronic iron deficiency

anemia. Certain differences between the roentgen findings of the present study and those of the cases reported by other investigators are discussed.

Brooks, R. E., and Siegel, B. V.: Normal human lymph node cells: an electron microscopic study. Submitted May 4, 1965; accepted for publication Sept. 25, 1965.

Lymph nodes, from 15 patients undergoing surgery for conditions not related to lymphoid tissue disease, have been examined with the electron microscope. The human lymph node cell types, including lymphocytic, reticular, and plasma cells, have been described at low and medium electron microscopic magnifications, and the criteria for their identification are discussed. The characteristic features outlined for identification of these cell types provide a basis for comparison with pathologically altered lymph node cells.