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Neonatal thymus placed in a Millipore diffusion chamber for 1 week loses its cortical cells while the epithelial reticular cells remain viable. Grafts of these remnants in neonatally thymectomized mice are completely reconstituted from cells of the thymectomized host. Evidence is presented which suggests that the bone marrow may be a source of cells which reform the thymus cortex.

These remnant grafts result in lymphoid reconstitution of neonatally thymectomized mice. The grafted animals also reject allografts of skin and lymphoma cells in a normal manner. However, these C3H mice, neonatally thymectomized and reconstituted with allografts of AKR thymic epithelial reticular cells, are tolerant to grafts of AKR lymphoma cells.


Negative staining of whole mounts of platelets preserves several structures not previously described. External to the plasma membrane there is a coat which contains electron-dense bodies of varying shape. External to the coat is a region which stains intensely with phosphotungstate. Platelet granules co-exist with tubules in the granulomere as well as with a large number of other membraneous structures.

A speculative interpretation of the function served by these structures in platelet physiology is proposed. The PTA halo denotes a cloud of adsorbed plasma proteins, including fibrinogen maintained in the platelet environment by the coat. During viscous metamorphosis, coagulation is catalyzed by discharge of the contents of the granules and shedding of the coat.


Hodgkin’s disease, lymphosarcoma, and acute leukemia have been studied after treatment with the *Vinca rosea* alkaloids (vinblastine and vincristine), in order to demonstrate and evaluate the mitosis-arresting effects of the two drugs on the malignant cells. Histologic sections and lymph node and bone marrow aspirations were performed immediately before and 24 hours after the intravenous administration of the drugs in 15 cases of Hodgkin’s disease, 12 of lymphoblastic lymphosarcoma, and 12 of acute leukemia, besides other miscellaneous cases.

In Hodgkin’s disease aspirates and sections showed a clear-cut metaphase arrest in the post-VLB specimens, chiefly affecting the pre-Sternberg or Hodg-
kin cells. This effect, besides corroborating the fundamentally stathmokinetic mechanism of VLB in Hodgkin's disease, was considered an additional factor in confirming the widely proposed conception that these cells represent the fundamentally proliferating and malignant tissue of this disease.

In the acute leukemias and lymphoblastosarcomas, cytomorphologic and quantitative studies demonstrated that the oncolytic effects correlated well with the magnitude of metaphasic blockade. It is postulated that only actively proliferating cells—the so-called "growth fraction"—are the target for these alkaloids.


The fibrinolytic activity of human leukocytes was studied by the fibrin plate method and by the histochemical fibrin slide method, using plasminogen-rich and plasminogen-free fibrin substrates. Lysis is caused by a protease. Plasminogen activator is absent. The slide method showed the effect of leukocytes to be weak in comparison to that produced by the plasminogen activator in capillary endothelial cells invading fibrin deposits in the body.


Five LDH and two MDH isozyme bands were obtained with acrylamide gel electrophoresis of leukocyte extracts. Normal lymphocytes showed a high total H-LDH (heart type) activity (67 per cent) with 25 per cent in LDH-1 and only 3.5 per cent in LDH-5. Lymphocytes from chronic lymphatic leukemia (CLL) and lymphosarcoma leukemia (LSA-LL) had less LDH-1 and more LDH-3 and LDH-4 than normal lymphocytes. The H-LDH fell to 60.5 per cent in CLL and 56 per cent in LSA-LL. PMN leukocytes had low H-LDH activity (38.8 per cent) with 3.3 per cent in LDH-1 and 25.8 per cent in LDH-5. In myelogenous leukemia, myeloblasts had the most LDH-1 and H-LDH, while mature PMN had the least. PMN leukocytes isolated from CLL, LSA-LL, and myelogenous leukemia had LDH patterns like the normal. Monocytes from acute monocytic leukemia were low in LDH-1 and LDH-5, but had a high total enzyme content. They evidently were rich in LDH-2, 3, and 4.

Lymphocytes had less MDH-1 (60 per cent) than PMN leukocytes (78 per cent). In CLL, lymphocyte MDH-2 increased. In myelogenous leukemia, myeloblasts had the most MDH-2 and mature PMN the least. Monocytes from monocytic leukemia contained a little more MDH-2 than PMN leukocytes.

In general, white cell immaturity and/or ability to divide was associated with high levels of LDH-1, total H-LDH, and MDH-2.


These radioautographic studies using parabiotic rats and partial marrow shielding showed that cells responsible for recovery of irradiated bone marrow
had their origin in the shielded marrow. Three morphologically distinct cell types appeared in the blood of these parabionts: mature granulocytes, small lymphocytes, and monocytoid cells. The monocytoid was the major cell type which crossed from the shielded to nonshielded marrow, and the observations suggested that it is this cell which served as a stem cell for both the erythrocytic and granulocytic cell lines.

Labeled erythroblasts and myeloblasts were observed in the recovering marrow and the labeling intensity of these cells indicated that they were the second or third division products of labeled immigrant cells.

The effect of marrow shielding upon the recovery of lymphopoiesis in spleen, thymus, lymph nodes, and bone marrow is also discussed.


The hypothesis of undue susceptibility to leukemia or lymphoma in individuals with autoimmune disease is evaluated by an analysis of case reports and an analysis of the mortality experience of series of patients with systemic lupus erythematosus or rheumatoid arthritis. No direct support for the hypothesis is found. The difficulty in accumulating a sufficient number of person years at risk in series of individuals with autoimmune disease is noted and alternative approaches to validating this hypothesis are discussed.