centages of only 1.4, 11, and 14%, whereas the remaining 4 children had no lymphocyte transformation at all. Again, the lymphocytes obtained from the spinal fluid in 3 evaluable patients of 7 examined, all affected by meningeal leukemia, and from the pleural effusion in 1 patient did not undergo any blastic transformation. It appears that a quite different behavior concerning PHA blastogenesis of lymphocytes in ALL can be observed according to the source the lymphocytes come from.

66. *In vitro* sensitivity to vincristine of acute lymphocytic leukemia cells—a tentative to correlate *in vivo* and *in vitro* results. L. Picent Sereni, G. Masera, V. Carnelli, and F. Sereni. *Univ. of Milan, Italy.*

Data were obtained on the influence of vincristine (7×10^{-5}M) on the *in vitro* incorporation of both 6-^{14}C-orotate into RNA and of 1-^{14}C-leucine into proteins of leukemic cells from acute lymphocytic leukemia. Orotate and leucine incorporations were measured after 2 and 12 hr of incubation. A total of 17 patients were examined. Most of the *in vitro* studies were conducted on peripheral blast cells. In 4 instances also bone marrow leukemic cells were tested. In any case in which a good clinical response by vincristine administration was recorded, also a strong *in vitro* inhibition of both RNA and protein synthesis was observed. On the contrary, in the only 2 cases in which a definitive *in vivo* resistance to vincristine was observed, also an *in vitro* lack of response was found. Practical implications of these studies will be discussed.

67. Gluconeogenesis in human platelets. K. A. Zuppinger, N. Nakamura, J. P. Colombo, and P. Walter. *Universitäts-Kinderklinik and Univ. of Bern, Switzerland.*

Quantitative evaluation of gluconeogenesis in humans is difficult since liver and kidney are not suitable for repeated investigation. Therefore, gluconeogenesis in easily available tissue such as platelets was studied during incubation in buffered solution. Upon addition of 20 mM pyruvate glucose formation in the incubation medium amounted to 6.99 μmoles/hr/10^{11} platelets and glycogen utilization was inhibited to 28.8%. Incorporation of label from pyruvate-2-^{14}C into carbohydrates revealed 2.22 μmoles pyruvate incorporated/hr/10^{11} platelets. These results are interpreted as evidence for the existence of gluconeogenesis in human platelets. In two patients with glycogen storage disease type I incorporation of label from pyruvate-2-^{14}C into carbohydrates was decreased to 0.95 μmole/hr/10^{11} platelets. Only 7% of the total radioactivity was found to be in glucose in these patients as compared to 81% in normals, indicating that most radioactivity was incorporated into nonglucose carbohydrates, probably glycogen. Incorporation of label from pyruvate-2-^{14}C into carbohydrates by platelets of two patients with glycogen storage disease type III showed no increase in comparison to normals; however, in these patients 95% of the label was found in glucose. This finding indicates that in type III glycogenosis a greater relative part of pyruvate is incorporated into glucose than in normals. The conclusion is drawn from these studies that human platelets might prove to be a valuable material for studying gluconeogenesis under various conditions.

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