tional roles of these cells and the reasons for their increase in lymphoproliferative disorders remain to be determined.

C.E. GROSSI
A. VELARDI
A. LANDAY
E.F. PRASTHOFER
L. T. CLEMENT
Department of Pathology and the Cellular Immunobiology Unit of the Tumor Institute, University of Alabama in Birmingham
Birmingham, AL 35294

REFERENCES

1. Gatenby PA, Kansas GS, Xian CY, Evans RL, Engleman EG: Dissection of immunoregulatory subpopulations of T lymphocytes within the helper and suppressor sublineages in man. J Immunol 129:1977, 1982

2. Armitage RJ, Linch DC, Worman CP, Cawley JC: The morphology and cytochemistry of human T-cell subpopulations defined by monoclonal antibodies and Fc receptors. Br J Haematol 52:605, 1982

3. Bernard J, Dufer J: Cytochemical analysis of human peripheral blood lymphocyte subsets defined by monoclonal antibodies. Scand J Immunol 17:89, 1983

4. Clement LT, Grossi CE, Gartland GL: Morphological and phenotypic features of the subpopulation of Leu2$^+$ cells that suppresses B cell differentiation. J Immunol (in press)

5. Velardi A, Grossi CE, Cooper MD: A large subpopulation of lymphocytes with T helper phenotype (Leu3/T4$^+$) exhibits the property of binding to NK cell targets and granular lymphocyte morphology. J Immunol (in press)

6. Velardi A, Prchal JT, Prasthofer EF, Grossi CE: Expression of NK-lineage markers on peripheral blood lymphocytes with T-helper (Leu3$^+$/T4$^+$) phenotype in B-cell chronic lymphocytic leukemia. Blood (this issue)

7. Poppema S, Visser L, DeLeij L: Reactivity of presumed natural killer cells antibody Leu7 with intrafollicular T lymphocytes. Clin Exp Immunol 54:834, 1983

8. Porwit-Ksiazek A, Ksiazek T, Biberfeld P: Leu7$^+$ (HNK-1$^+$) cells. I. Selective compartmentalization of Leu7$^+$ cells with different immunophenotypes in lymphatic tissues and blood. Scand J Immunol 18:485, 1983

B CELL DIFFERENTIATION

To the Editor:

In the June 1984 issue of *Blood*, Dr Anderson et al,[1] in an attempt to define a model for normal B cell differentiation, are using data derived from their studies on B cell malignancies. They proposed that pre-B cells evolve from an Ia+ CALLA+ B1− to an Ia+ CALLA+ B1+ cell expression, followed by an Ia+ CALLA+ B1+ cytoplasmic IgM (cIgM)+ stage. As pre-B cells enter the "isotype diversity" compartment and become more mature, they lose the expression of CALLA and cIgM and they acquire the B2 antigen as well as surface immunoglobulins (sIgs).

Recently we observed an acute lymphoblastic leukemia with 69% Ia+ and 10% B1+ cells in the bone marrow (80% blasts) population, but the cells were negative for CALLA, B2, sIgs, cIgM and TDT. The WBC count was 2.1 × 10^9/L with 81% lymphocytoid microblasts and 59% cells Ia+. As in bone marrow, the circulating cells were negative for B1, B2, CALLA, and sIgs. There are two possible explanations for the Ia+ B1+ phenotype in the bone marrow: (1) these cells represent a different pathway than the one mentioned by Dr Anderson et al, which proceeds or is independent of the CALLA+ pathway, or (2) these cells are in a transitory stage from the pre-B to immature B cell compartment. In that stage they have lost the CALLA and cIgM expression prior to the acquisition of the B2 and sIgM while they remain Ia+ B1+.

We therefore consider that a transitory stage may exist between the pre-B and immature B compartment proposed by Dr Anderson. The phenotype at that stage is Ia+ B1+ CALLA− cIgM− B2−, unless a different pathway of maturation exists and which is independent of the one described.

ANNA P. EFREMIDIS
J. GEORGE BEKESI
Mount Sinai School of Medicine
New York, NY 10029

MARY S. VESSONIARAKI
Bayley Seton Hospital
Staten Island, NY

REFERENCE

1. Anderson KC, Bates MP, Slaughenhoupt BL, Pincus GS, Schlossman SP, Nadler LM: Expression of human B cell-associated antigens on leukemias and lymphomas: Model of human B cell differentiation. Blood 63:1424, 1984

To the Editor:

Dr Efremidis correctly points out that our model of B cell differentiation based upon studies of B cell malignancies postulates that pre-B cells evolve through the following sequence: Ia+B4+CALLA−B1−, Ia+B4+CALLA+B1−, Ia+B4+CALLA+B1+, and Ia+B4+CALLA+B1+cytoplasmic u+. As cells enter the post-pre-B cell stages of B cell differentiation, CALLA and cytoplasmic u (cu) are lost and cell surface immunoglobulin (sIg) and B2 antigen are acquired. They observed a patient with acute lymphoblastic leukemia (ALL) who had 69% Ia+ cells, 10% B1+ cells, and 80% blasts in the bone marrow. In addition, this patient was circulating Ia+B1−B2−CALLA−sIg− tumor cells. On the basis of the Ia+B1+CALLA−B2− cu+ phenotype which may identify marrow tumor cells, they suggest this patient may reflect an alternative pathway of B cell differentiation or an additional differentiative stage within our model of B cell differentiation. An alternative interpretation is that those bone marrow cells which were Ia+ were in fact largely blasts (69% Ia+, 80% blasts), but that the 10% anti-B1 reactive cells were either normal B cells or else a minority of tumor cells.

Dual fluorescence experiments could be done to examine whether Ia and B1 are coexpressed on these tumor cells. If, in fact, the B1+ cells were normal B cells, then the phenotype of the tumor cells within both bone marrow and blood would be Ia+CALLA−B1−B2− cu−. Twenty-five of 138 patients with non-T ALL recently studied (18%) expressed this phenotype.[1] Moreover, the B4