Expression of Normal Myeloid-Associated Antigens by Acute Leukemia Cells

By Patricia A. Dinndorf, Robert G. Andrews, Denis Benjamin, Derry Ridgway, Lawrence Wolff, and Irwin D. Bernstein

Monoclonal antibodies that react with hematopoietic cells and their precursors in a stage and lineage restricted fashion were used in indirect immunofluorescence assays to examine leukemic cells from 105 pediatric age patients. The differentiative states of blasts from 42 patients with acute nonlymphocytic leukemia (ANLL) were defined by these antibodies. When these were compared to their morphologic and histochemical levels of differentiation as defined by the French-American-British (FAB) classification, no direct relationship was found. The reactivity of these antibodies with leukemic cells from 63 patients with acute lymphocytic leukemia (ALL) was also investigated, and the usefulness of these antibodies in distinguishing leukemias of myeloid from those of lymphoid origin was demonstrated.

A CUTE nonlymphocytic leukemia (ANLL) is a heterogeneous group of disorders that originates at the level of hematopoietic progenitor cells. ANLL subtypes have classically been categorized according to morphologic and histochemical criteria, which have been more precisely defined in recent years by the French-American-British (FAB) classification. An alternate approach to the heterogeneity of ANLL is to use monoclonal antibodies that react with antigens expressed by normal hematopoietic cells and their precursors in a lineage and stage-restricted fashion.

We have used such monoclonal antibodies to examine the differentiative state of ANLL in children. Importantly, a comparison of the differentiative state of ANLL cells based on morphologic and histochemical criteria (defined by the FAB classification), with their antigenic phenotype failed to reveal a direct relationship. In addition, we have used these antibodies to examine acute lymphocytic leukemia (ALL) cells, and demonstrated their usefulness in distinguishing leukemias of myeloid versus lymphoid origin.

MATERIALS AND METHODS

Cells. Heparinized marrow or peripheral blood obtained at the time of the initial diagnosis or relapse of leukemia was obtained from pediatric patients at Children's Orthopedic Hospital and Medical Center, Seattle, Washington, and at the Doernbecher Children's Hospital, Oregon Health Sciences University, Portland. Samples from children with ANLL were collected beginning in 1973 at the Children's Orthopedic Hospital and Medical Center, and at the University of Oregon beginning in September 1979 when the Children's Cancer Study Group (CCSG) 251 study for the treatment of ANLL was opened. ALL samples were collected beginning in January 1979 with the inception of the CCSG study 191 for the treatment of average and high risk ALL, and from Oregon beginning in May 1983 with the inception of CCSG protocols 104, 105, 106, 107, and 123 for the treatment of children with various risk factors.

Cells were separated by ficoll-hypaque density centrifugation (density 1.077), suspended in newborn calf serum supplemented with 10% dimethyl sulfoxide (DMSO), and frozen in liquid nitrogen until studied. All samples had greater than 75% malignant blast cells, as determined by examination of Wright stained cytospin preparations of thawed cells at the time of phenotyping, and viability was greater than 75% as assessed by trypan blue dye exclusion.

Monoclonal antibodies. Unfractionated ascites fluids containing monoclonal antibodies were used at dilutions from 1:250 to 1:10,000 in all studies. Antibody T11D7, an isotype identical (IgM) murine monoclonal antibody of irrelevant specificity (antimouse Thy 1.1; kindly provided by Dr E. Clark, University of Washington, Seattle) served as a negative control. Production and characterization of monoclonal antibodies 1G10, 5F1, L4F3, and T5A7 has been described elsewhere and their reactivities are summarized in Table 1. Monoclonal antibodies to lymphoid-associated antigens used to characterize ALL included CALLA (J-5 provided by Dr John Pesando or BA-3 provided by Dr John Kersey) and T-cell antibodies (9.6, 10.2, 12.1, and 38.1 provided by Dr John Hansen and 3A1 provided by Dr Barton Haynes).

Immunofluorescent studies. Cells were incubated with monoclonal antibody diluted in Minimal Essential Medium (MEM) supplemented with 5% newborn calf serum and 2% human AB serum (Irvine Scientific, Santa Ana, Calif) for 30 minutes, washed, and then incubated with a 1:40 dilution of an affinity-purified fluorescein-conjugated goat antimouse IgM antiseraum (List Bio- netics, Kensington, Md) as previously described. As a negative control, cells also were stained with the isotype identical antibody, T11D7 (antimouse Thy 1.1). Stained cells were analyzed by flow microfluorimetry using a FACS 440 (Becton Dickinson, Oxnard, Calif). A total of 4,000 cells was analyzed for each antibody. Antibody staining of cells was considered positive if more than 25% of cells exhibited fluorescence intensity greater than 95% of the cells stained with the negative control antibody. Twenty five percent was used as the cutoff because all samples analyzed contained no more than 25% normal contaminating cells and, therefore, at this level at least some of the reactivity was with leukemic cells. In the case of L4F3, if there was a homogeneous shift of the median fluorescence intensity (peak) to greater than 1.5 times that of cells stained with T11D7, cells were also considered positive. In cases where there was a homogeneous shift, 100% of the cells were considered to express the antigen. This conclusion was confirmed by demonstrating that for 23 leukemic samples that had been determined to express L4F3 by these criteria, all cells were susceptible to complement-mediated lysis by L4F3 in a 51Cr release assay. The complement-mediated cytotoxicity assay has been previously described.

Cytochemical studies and FAB classification. The diagnosis of ALL or ANLL was made at the institution of origin based on standard morphological and cytochemical criteria. Leukemia classified as ALL was not peroxidase, sudan black, or chloracetate esterase positive; most were PAS positive. Marrows of patients...
Table 1. Monoclonal Antibodies to Myeloid-Associated Antigens

<table>
<thead>
<tr>
<th>Antibody</th>
<th>Biochemic Specificity</th>
<th>Reactivity With Hematopoietic Cells and Precursors</th>
<th>Equivalent Antibodies</th>
</tr>
</thead>
<tbody>
<tr>
<td>1G1O</td>
<td>X-hapten Gαβ1-4GlcNAcR 3 Fucα1</td>
<td>PB: granulocytes (bright) 10-15%, monocytes (barely detectable staining) BM: myelocytic cells past myeloblast stage; portion of myeloblasts Progenitor cells: portion CFU-GM&lt;sup&gt;3&lt;/sup&gt;</td>
<td>Multiple [CD 15]&lt;sup&gt;*&lt;/sup&gt; antibodies&lt;sup&gt;5&lt;/sup&gt;</td>
</tr>
<tr>
<td>5F1</td>
<td>85 Kd&lt;sup&gt;†&lt;/sup&gt; glycoprotein</td>
<td>PB: monocytes, platelets BM: monocytes, nucleated erythroid cells, megakaryocytes Progenitor cells: CFU-E&lt;sup&gt;3&lt;/sup&gt;</td>
<td>20.3&lt;sup&gt;a&lt;/sup&gt; [CDw14]&lt;sup&gt;*&lt;/sup&gt;</td>
</tr>
<tr>
<td>L4F3</td>
<td>62 Kd protein</td>
<td>PB: monocytes and granulocytes (weak) BM: immature myelocytes, monocytes Progenitor cells: CFU-GM, portion of BFU-E, CFU-Meg, CFU-Mix&lt;sup&gt;3.2&lt;/sup&gt;</td>
<td>MY-9&lt;sup&gt;a&lt;/sup&gt; L1B2&lt;sup&gt;x&lt;/sup&gt;</td>
</tr>
<tr>
<td>T5A7</td>
<td>lactosylceramides</td>
<td>PB: granulocytes, monocytes BM: mature granulocytic cells and some band forms (bright), band forms and metamyelocytes (moderately bright) Progenitor cells: portion Day 7 CFU-GM&lt;sup&gt;3&lt;/sup&gt; proportion PHA-stimulated lymphocytes</td>
<td>G035&lt;sup&gt;a&lt;/sup&gt; [CDw17]&lt;sup&gt;*&lt;/sup&gt;</td>
</tr>
</tbody>
</table>

Abbrev: PB, peripheral blood; BM, bone marrow.

*Designation as defined by 2nd International Workshop on Human Leucocyte Differentiation Antigens.
†Bernstein I, Andrews AG: unpublished observations.

Results of monoclonal antibodies with ANLL cells. The reactivity of monoclonal antibodies 1G10, 5F1, T5A7, and L4F3 with leukemic samples from 42 pediatric patients with ANLL was examined by indirect immunofluorescence. The specificities of these antibodies and antibodies that appear to recognize the same determinants are summarized in Table 1. In brief, two of these antibodies discriminate between granulocytic and monocytic cells. 1G10, an X-hapten reactive antibody (CD15),* reacts with cells at all stages of granulocytic differentiation to the promyelocyte level, but only with a portion of myeloblasts and granulocyte-macrophage progenitors (CFU-GM). In contrast, antibody 5F1 reacts with monocytes, platelets, megakaryocytes, nucleated erythrocytes, and erythroid colony-forming cells (CFU-E), but not with granulocytic cells.

The other two antibodies, L4F3 and T5A7, react with antigens expressed at maximal concentrations on granulocytic cells at different stages of maturation. Thus, L4F3 reacts most intensely with metamyelocytes, myelocytes, pro-myelocytes, and a portion of myeloblasts, and virtually all CFU-GM, while the T5A7 antigen (lactosylceramide) is expressed maximally on mature granulocytes, and a minor proportion of Day 7 CFU-GM. Both of these antibodies also react with monocytes. In addition, T5A7 reacts with 20% of PHA-stimulated lymphocytes.

*Designation of the 2nd International Workshop on Human Leucocyte Differentiation Antigens.
The results show that 40 of 42 leukemic samples examined reacted with at least one of the antibodies (see Fig 1). 1G10, which predominantly reacts with granulocytes, bound to 55% of the samples tested, while cells from 69% of the samples bound 5F1, the monococyte reactive antibody. Thirty-five specimens (83%) reacted with either one or both of these antibodies. Ninety percent of the samples tested reacted with L4F3, the antibody that binds maximally to immature myeloid cells. Cells from 61% of the samples reacted with T5A7, the antilactosylceramide antibody that binds specimens (83%) somal translocation), 16 as M4 (myelomonocytic), 4 classification for ANLL. Cells from 15 patients were classi-

FAB classification of ANLL cells. For each patient studied, bone marrow aspirates obtained at initial diagnosis were stained with Wright Giemsa, and for peroxidase, sudan black and NSE, and were classified according to the FAB classification for ANLL. Cells from 15 patients were classified as M1 (myeloblast), 4 as M2 (myeloid with maturation), 1 as M3 (promyelocytic) (confirmed by the 15:17 chromosomal translocation), 16 as M4 (myelomonocytic), 4 as M5 (monocytic), and 2 as M6 (erythroblastic). The MS category was subdivided into 2 cases of M5A (immature) and 2 cases of M5B (differentiated).

Comparison between antigenic phenotype and FAB classification of ANLL cells. The differentiative state of leu-
kemic cells defined according to FAB classification or antigenic phenotype were generally discordant (Table 2). First, most specimens classified as FAB M1 did not react with 1G10 which reacts with most granulocytic elements. This may reflect the immaturity of these cells with respect to granulocytic differentiation. However, leukemic cells which appeared by conventional criteria to have differentiated further along the granulocytic pathway, ie, specimens classified as M2 and M3, also failed to react with 1G10 in three of five instances. In contrast, the majority of these cells (M1, M2, and M3) reacted unexpectedly with 5F1, a reaction not seen with normal granulocytes. Also unexpected was the reaction of all leukemias classified as FAB M5 with 1G10. One pattern that did emerge was that specimens classified as FAB M4 tended to react with all the antibodies tested.

The relationship between maturation according to anti-
genic and morphologic criteria was also evaluated. Since the surface expression of the antigen identified by L4F3 is greatest on the least as compared to the most mature myeloid cells, we determined whether this relationship was also found between leukemic cells classified as less or more differentiated. A comparison of the relative fluorescence intensity of samples stained with L4F3 among the FAB subgroups revealed no obvious relationship between fluores-
cence intensity and maturation (data not shown). This however may have been a consequence of the small number of more mature (M2, M3, M5b) specimens. A similar lack of a relationship between morphologic and antigenic differen-
tiation was not found for TSA7 which reacts maximally with mature myeloid cells. Seven of 15 leukemias classified as M1 (myeloblast) reacted with T5A7, while only one of five samples that were morphologically more mature and classified as M2 or M3 reacted with T5A7.

Table 2. Reactivity of Monoclonal Antibodies With Subgroups of ANLL

<table>
<thead>
<tr>
<th>FAB/Cytochemistry</th>
<th>1G10</th>
<th>5F1</th>
<th>L4F3</th>
<th>TSA7</th>
</tr>
</thead>
<tbody>
<tr>
<td>M1/peroxidase</td>
<td>3/15</td>
<td>10/15</td>
<td>14/15</td>
<td>7/15</td>
</tr>
<tr>
<td>(Immature myeloid)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>M2/peroxidase</td>
<td>2/4</td>
<td>2/4</td>
<td>2/4</td>
<td>1/4</td>
</tr>
<tr>
<td>(Myeloid with maturity)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>M3/peroxidase</td>
<td>0/1</td>
<td>0/1</td>
<td>ND</td>
<td>0/1</td>
</tr>
<tr>
<td>(Promyelocytic)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>M4/peroxidase, NSE</td>
<td>14/16</td>
<td>12/16</td>
<td>16/16</td>
<td>13/16</td>
</tr>
<tr>
<td>(Myelomonocytic)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>M5A/NSE</td>
<td>2/2</td>
<td>1/2</td>
<td>1/2</td>
<td>2/2</td>
</tr>
<tr>
<td>(Monocytic, immature)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>M5B</td>
<td>2/2</td>
<td>2/2</td>
<td>2/2</td>
<td>1/2</td>
</tr>
<tr>
<td>(Monocytic, mature)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>M6</td>
<td>0/2</td>
<td>2/2</td>
<td>2/2</td>
<td>1/1</td>
</tr>
<tr>
<td>(Erythroblastic)</td>
<td></td>
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Number of samples reacting with antibody over number of samples tested of each FAB subclassification.
Comparison between peripheral blood and bone marrow phenotype of ANLL cells. Because peripheral blood rather than marrow cells was often evaluated, we compared antigenic phenotypes of leukemic cells obtained from the peripheral blood and marrow in eight patients in whom both types of specimens were available. These paired samples were obtained at the same time in the patient's disease, that is, at initial diagnosis or at the time of a subsequent relapse. Importantly, the proportion of leukemic cells reactive with the antibodies 1G10 and 5F1 as determined in peripheral blood or bone marrow was similar (within 15%). In all instances, except for one patient each in the case of antibody 5F1, 1G10, and TSA7, the positive or negative nature of the bone marrow or peripheral blood sample was concordant.

In studies with TSA7, a slightly greater percent (<15% except in one instance where it was 25%) of leukemia cells were positive in the peripheral blood as compared to the marrow in almost every case. Again these differences altered the interpretation of TSA7 reactivity in only one case. This may have been the consequence of a greater number of normal mature cells contaminating the peripheral blood specimens or a higher proportion of the circulating malignant cells as compared to those in the marrow which express lactosylceramide in the periphery. The former, however, is unlikely in the absence of a consistently greater activity of 1G10 or 5F1 with peripheral blood granulocytes or monocytes, respectively, that might have been present in the peripheral blood specimens.

Expression of myeloid-associated antigens by ALL cells. Leukemic samples from 63 pediatric patients with ALL were also examined for reactivity with the anti myeloid monoclonal antibodies (see Fig 1). These patients were diagnosed as having ALL based on morphological and cytochemical criteria. The reactivity of these cells with lymphoid-associated monoclonal antibodies was reported in a previous study and is summarized in Table 3. None of the specimens reacted with either antibody 1G10 or 5F1. Lymphoblasts from 5 of 62 samples tested reacted with L4F3. Of the five, three expressed CALLA and the other two lacked both CALLA and T-cell antigens. Clinically, these five patients all underwent remission induction successfully and remain in complete continual remission 38, 53, 54, 59, and 68 months from initial diagnosis.

<table>
<thead>
<tr>
<th>Table 3. Reactivity of Monoclonal Antibodies With Subgroups of ALL</th>
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</thead>
<tbody>
<tr>
<td><strong>Myeloid Antibodies</strong></td>
</tr>
<tr>
<td><strong>Serologically Defined Subgroups</strong></td>
</tr>
<tr>
<td>T+ CALLA + or -</td>
</tr>
<tr>
<td>T- CALLA +</td>
</tr>
<tr>
<td>T- CALLA -</td>
</tr>
</tbody>
</table>

Indirect immunofluorescent reactivity of monoclonal antibodies with malignant cells by FACS analysis. For each antibody a specimen was considered positive if ≥25% of cells were reactive. Results are reported as (number of samples positive)/(number of samples tested).

*Specimens were tested with either antibodies 9.6 and 10.2 separately or with a cocktail consisting of 9.6, 10.2, 12.1, 3A1, and 38.1.

Cells from six of the patients with ALL did not express the T-cell-associated antigens that were evaluated or CALLA. Cells from two of these patients reacted with L4F3 as mentioned above, and one reacted with both L4F3 and TSA7. Of the three cases in this group that reacted only with TSA7, two were infants who died within one year of diagnosis, an outcome typical of this disease in infants. The third remains in continuous remission at 31 months. The patient whose cells failed to react with any of the antibodies tested also remains in initial remission at 23 months.

The antigen defined by TSA7, lactosylceramide, was expressed on a significant proportion of lymphoblasts in 19 of 59 specimens tested. Eight of 15 T-cell leukemias tested expressed this antigen. This is not surprising, since TSA7 is known to react with a proportion of PHA-stimulated peripheral blood T cells. Four of eight patients with T-cell ALL whose blasts reacted with TSA7 remain in complete continual remission from 25 to 54 months from initial diagnosis while three of seven patients whose cells did not react with TSA7 are alive 26 to 61 months from initial diagnosis. Cells of seven of 38 CALLA positive cases were reactive with TSA7. However, there appeared to be no difference in this small series in the survival of patients whose cells did and did not react with TSA7 (data not shown).

Terminal deoxynucleotidyl transferase (TdT) determinations were done on all but two of the ALL specimens which stained with one or more antimyeloid antibodies. Of the five that were L4F3 positive, four were tested for TdT expression, and all were positive; and of the 19 specimens that reacted with TSA7, 18 were tested and all were positive.

DISCUSSION

ANLL is generally categorized according to morphological and histochemical criteria set forth by the FAB group and is based on expression of characteristics normally associated with granulocytic, mononuclear, or erythroid cells. This classification has largely failed to identify groups of prognostic importance. This system relies on subjective criteria and concordance between hematopathologists is not high. We have directly compared the assignment of cell lineage defined by the FAB system to that defined by reactivity with antimyeloid monoclonal antibodies. In the present study, a comparison of differentiative states of leukemic cells defined by the presence of histochemical markers versus that defined by the expression of myeloid-associated antigens failed to demonstrate a direct relationship between these two. Specifically, an association between the presence of peroxidase and the expression of a granulocyte-associated antigen (1G10) was not found. Nor did we observe a direct correlation between the presence of NSE and the expression of a monocyte-associated antigen (5F1).

This failure to define a relationship between lineage assignments defined by these two methods is not surprising since each of these approaches examines the expression of only a few of the gene products normally displayed by myeloid cells of specific lineages or at specific stages of maturation. Thus, in the FAB system only cytoplasmic peroxidase and NSE, which are selectively expressed during
normal granulocytic or monocytic differentiation, respectively, are evaluated. Examination of the expression of cell surface differentiation antigens thus provides evidence of other maturation-linked characteristics that can be evaluated to assess the differentiative state of ANLL cells.

Evaluation of the morphologic appearance of cells also did not strictly correlate with the antigenic phenotype. An immature appearance (FAB-M1 cell) was not necessarily associated with expression of an antigen predominantly found on immature granulocytes (L4F3) nor was a more mature appearance (FAB-M3) associated with expression of an antigen found on mature granulocytes (TSA7). There were, however, two FAB subclasses that demonstrated distinct patterns of antigenic expression. Specifically, most of the specimens of FAB M1 ANLL did not express the antigen detected by 1G10 (12 of 15). This antigen is expressed by virtually all granulocytic cells except the least mature myeloblasts and CFU-GM (where only a portion are 1G10 positive). In the second instance FAB M4 specimens frequently reacted with all of the antibodies used in the present study, consistent with the multiple expression of cytoplasmic enzymes detected by histochemic methods. Nevertheless, analysis of cell surface antigens provides an independent assessment and can provide a novel basis for the classification of patients with ANLL.

A number of other investigators have examined the expression of myeloid antigens on ANLL cells.15–22 Hermann et al17 demonstrated quantitative differences in the number of ANLL cells expressing antigens in the various subgroups defined by the FAB classification. One of the monoclonal antibodies used was VIMD5, which also reacts with the X-hapten.21 Analogous to our results with 1G10, they found the X hapten only 15% to 30% of the FAB M1 leukemias tested, which was a lower proportion than that found for the other FAB subclasses, except for M6.

Griffin et al20 defined patterns of antigen expression with a series of monoclonal antibodies that correspond to discrete levels of differentiation. They observed patterns that corresponded to some FAB groups, that is, M3 and M5, but these patterns were not exclusively expressed by leukemias of these subtypes, and the number of patients in these groups was small. Similar to our results with 5F1, MY-4, an antibody that reacts with monocyttes, was expressed by some samples that were NSE negative. This is in contrast to a study in which monoclonal antibodies were described that reacted with nearly every case of FAB M4 and M5 leukemia tested.21

Of potential interest is that antigenic phenotyping of ANLL with these antibodies may identify groups of prognostic importance. The FAB classification has been only minimally successful in identifying groups of clinical relevance. In a preliminary report by Vaughan et al24 a correlation between successful remission induction and reactivity with MY-1, another antibody to the X-hapten, was reported.25 In contrast, Griffin et al20 failed to detect a relationship between antigenic phenotype and probability of remission induction. Unfortunately, the patients in the present study with ANLL were collected over a 10-year period and were not treated in a uniform fashion and therefore a clinical analysis is not possible. We are, however, presently using these antibodies to study a larger number of pediatric patients in a prospective manner who are enrolled in clinical protocols of the Children's Cancer Study Group.

A subclassification of ANLL that is not accounted for in the FAB schema is acute megakaryoblastic leukemia. One patient who was not included in the present series experienced a classical course of acute megakaryoblastic leukemia.26 Leukemic cells from this patient failed to react with any of the antibodies used in the present study although they did react with C7E10, an antibody prepared in our laboratory to glycoprotein Ib.27 Nineteen specimens of ANLL included in the present study were not found to react with C7E10 (data not shown).

In the present study we also examined leukemic cells of lymphoid origin and observed that antibodies 1G10, 5F1, and L4F3 are potentially useful in differentiating ALL from ANLL. Ninety-five percent of the cases of ANLL tested reacted with at least one of these three antibodies. Eighty-three percent of the ANLL samples tested reacted with either 1G10 or 5F1 or both. None of the 63 cases of ALL tested reacted with either of these antibodies. Thus, these antibodies are potentially useful in cases where the differentiation between these diseases is not straightforward. In a previous report, we identified two such patients who were diagnosed as having ALL although their leukemic cells reacted with 1G10 and/or 5F1. These patients were subsequently reclassified as ANLL, based on a change in morphology in one patient and in the clinical course in the other. Cells from these two patients failed to express CALLA, B-cell, or T-cell antigens.12

Reactions of antimyeloid antibodies with cells that were unequivocally of lymphoid origin were also observed in a number of instances. Antibody L4F3, which reacted with 90% of the ANLL specimens tested also reacted with 8% of the ALL samples tested. Similarly, Griffin et al have reported that MY-9 reacted with 90% of ANLL specimens, and with 2% of ALL specimens obtained from adults.9 Studies in this laboratory have shown that L4F3 and MY-9 may detect the same antigen as determined by competitive binding-inhibition studies (unpublished observation, R. Andrews). Furthermore, antibody T5A7 reacted with 32% of the ALL samples tested, especially with T cell leukemias. This reactivity with T-cell leukemias is not surprising since T5A7 is known to react with PHA-stimulated lymphocytes. The evaluation of additional patients with ALL who express these antigens is required to assess the clinical significance of these unexpected expressions of myeloid-associated antigens on leukemic cells of lymphoid origin.

REFERENCES


25. Huang LC, Civin CI, Magnani JL, Shaper JH, Ginsburg V: My-l, the human myeloid specific antigen detected by mouse monoclonal antibodies, is a sugar sequence found in Lacto-N-Fucopentaose III. Blood 61:1020, 1983


27. Nugent D, Kuniciki T, Montgomery R, Bernstein ID: Monoclonal antiplatelet antibody specific for GPIb which blocks ristocetin induced agglutination but does not bind to glycopcin on crossed immunoelectrophoresis. (Submitted for publication)

28. Andrews RG, Takahashi M, Segal G, Powell J, Bernstein ID, Singer JW: The L4F3 antigen is expressed by uni- and multi-potent colony forming cells but not by their precursors. (Submitted for publication)
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