Detection of Both T-Cell and Ia-like Antigens on Cells from Patients with Acute Myelomonocytic Leukemia and Chronic Myelogenous Leukemia in Blast Crisis

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ABSTRACT

Appropriately absorbed antisera to the lymphoid cell lines HSB and SB detect a human T-lymphocyte-associated antigen (TLAA) and the human Ia-like antigens, respectively. Cells from some patients with acute myelomonocytic leukemia (AMML) and chronic myelogenous leukemia in blast crisis expressed both TLAA and Ia antigens when tested in a complement-dependent microcytotoxicity assay (>90% lysis with both antisera). When patients were in remission, expression of TLAA and Ia antigens returned to normal values. Quantitative absorption of anti-TLAA sera with increasing numbers of AMML cells showed that these cells could remove reactivity of the serum for both HSB and human thymocytes. Similarly, absorption of anti-Ia serum with AMML cells removed all serological reactivity when this serum was tested on chronic lymphocytic leukemia cells or normal B-cells. These serological findings were confirmed by immunoprecipitation and sodium dodecyl sulfate-polyacrylamide gel electrophoresis studies using radiolabeled antigens. Cells from an AMML patient were labeled with $^{35}$S using lactoperoxidase; both the TLAA and Ia antigens were precipitated from the resulting solubilized membrane preparation. Leukemic cells from one AMML patient and one patient with chronic myelogenous leukemia in blast crisis were studied for Ia and TLAA antigens with a double fluorescence technique. Over 80% of the cells showed dual fluorescence.

INTRODUCTION

Previously, we have reported on the serological specificity and partial molecular characterization of the antigens detected by appropriately absorbed antisera to HSB and SB cell lines (3–5). The absorbed anti-HSB sera detect a TLAA present on T-lymphoblastoid cell lines, normal peripheral blood lymphocytes, thymocytes, and cells from some patients with ALL. Radioimmunoprecipitation and SDS-polyacrylamide gel electrophoresis revealed that anti-TLAA sera precipitated a high-molecular-weight glycoprotein (M.W. ~170,000) from radiolabeled, detergent-solubilized preparations of HSB, thymus, and peripheral blood lymphocytes, and TLAA-positive ALL cells. Several lines of evidence have indicated that the TLAA is not the E-rosette receptor. Appropriately absorbed antisera to the SB cell line were shown to detect a B-cell-associated antigen by cytotoxicity assay and to precipitate the M.W. 35,000 and M.W. 28,000 human Ia-like antigens by radioimmunoprecipitation and SDS-polyacrylamide gel electrophoresis studies.

Some acute leukemia patients who did not have typical ALL or acute myeloid leukemia appeared to exhibit both T- and B-cell markers when tested by cytotoxicity with these antisera (3). That is, 80 to 100% of the cells were lysed by both the anti-TLAA and anti-Ia antisera. In order to investigate this dual reactivity and to determine if the antigens being detected were the same as those previously described, we undertook a study of cells from patients with AMML and CML-BC.

MATERIALS AND METHODS

Antisera. Antiserum were prepared in monkeys and rabbits by immunization with HSB and SB tissue culture cell lines and were appropriately absorbed to detect the T-cell and Ia antigen specificities previously described (3, 5). The rabbit and monkey antisera detected the same antigen specificities and immunoprecipitated the same molecules. In some instances, rabbit antiserum prepared to affinity purified Ia antigens from the SB cell line was used in place of absorbed antisera to SB.

For quantitative absorption studies, aliquots of serum were twice absorbed with increasing numbers of cells for 30 min at room temperature. The sera were centrifuged at 25,000 x g for 30 min before being used in cytotoxicity and precipitation studies.

Cells. The leukemic patients studied were in the untreated or relapse stage of their disease and the peripheral blood or bone marrow samples from AMML patients had 80% or more leukemic blasts. Samples from CML-BC patients had 40% or more leukemic blasts. Samples for study were provided by the Hematology Division of the Department of Medicine at Duke University Medical Center.

Cell suspensions were prepared for testing by mixing the heparinized blood or bone marrow with Plasmagel and allowing the erythrocytes to sediment for 20 min at 37°C. The supernatant was centrifuged over Ficoll-Hypaque, and the cells at the interface were removed, washed in 0.9% NaCl solution and in Hanks’ balanced salt solution, and adjusted to 4 x 10^6 cells/ml.

Cytotoxicity Testing. A modification of the micromethod described by Mittal et al. (16) was used. Antiserum (1 μl) diluted in Hanks’ balanced salt solution was mixed with 0.5 μl cells (4 x 10^6/ml) in a Falcon microtest plate and incubated for 35 min at room temperature. Rabbit complement (5 μl) was added,
and the mixture was incubated for 35 minutes at 37°. Five μl
eosin and 2 μl formalin were sequentially added to each well.
The plates were read on an inverted phase microscope. The
reaction was considered positive if the immune sera gave a
30% increase in lysis over the normal serum controls.

Rosette Studies. Details of the methods used in our labora-
tory to detect cells rosetting with sheep erythrocytes have been
reported previously (14). Sheep erythrocytes were washed and
adjusted to 0.5% (v/v) concentration in Hanks' balanced salt
solution. Equal volumes of this suspension and leukemic cells
or normal peripheral blood lymphocytes (4 × 10⁶/ml in Hanks'
balanced salt solution with 20% fetal calf serum) were mixed
and spun at 100 × g for 5 min. After 1 hr incubation at room
temperature or overnight incubation at 4°, the cell button was
gently resuspended, toluidine blue was added, and the per-
centage of rosetting mononuclear cells was determined. At
least 200 mononuclear leukocytes were counted. A cell was
considered to be E-rosetting if 3 or more sheep erythrocytes
were attached to it.

Fluorescence Studies. For fluorescence studies, 10⁶ cells
were incubated with rabbit anti-TLAA serum for 30 min at 4°.
They were washed 3 times with PBS containing 2% bovine
serum albumin and incubated with fluorescein conjugate swine
anti-rabbit immunoglobulins (Bio-Rad Labs, Richmond, Calif.)
for 30 min at 4°. After washing, the cells were incubated with
monkey anti-la serum, washed, and then incubated with rho-
damine-conjugated goat anti-monkey IgG (Cappel Labs, Coch-
ranville, Pa.). Control experiments showed that the antiglobulin
reagents alone did not react with the cells. The antisera were
used at dilutions previously determined to result in specific
reactivity for HSB, SB, peripheral blood lymphocytes, and cells
from a patient with chronic lymphocytic leukemia.

Cells were examined with a Zeiss Standard RA phase con-
trast microscope equipped with IV/F epifluorescence condens-
ator, narrow band fluorescence and rhodamine filters, and ×40
Ph 2 objective.

Radiolabeling of Cell Surface Antigens. Cells were labeled
with 125I using lactoperoxidase (19). The reaction mixture (1 ml
volume) contained 10⁶ cells, 10⁻⁵ m sodium iodide, 40 μg
lactoperoxidase (Calbiochem, La Jolla, Calif.), and 1 mCi 125I
(Amersham, Arlington Heights, Ill.). Iodination was initiated by
adding 10 μl freshly prepared 10⁻² m hydrogen peroxide. At 5-
min intervals, additional 5-μl aliquots of hydrogen peroxide
were added until a total reaction time of 30 min had elapsed.
The cells were then diluted in 15 ml PBS and pelleted. The
cells were washed twice in PBS containing Nal (20 g/liter)
instead of NaCl and solubilized in 1 ml 10 mM Tris:150 mM
NaCl buffer, pH 8.0, containing 0.5% Triton X-100. The sus-
pension was centrifuged at 100,000 × g for 60 min in a Beckman L-2 ultracentrifuge to remove unsolubilized material.

The supernatant was dialyzed overnight at 4° against 10 mM
Tris:0.205 m glacial acetic acid:0.1% (w/v) SDS. Electrophore-
resis was carried out at 60 V until the samples entered the gel
and then at 125 V until the tracking dye had reached the end
of the gel. The gels were sliced into 1-mm fractions using a
Gilson automatic gel fractionator and counted with a Beckman
Biogamma Counter.

Two commercial protein mixtures (M.W. 53,000 to 265,000,
Gallard-Schlesinger, Carle Place, N. Y.; low-molecular-weight
calibration kit, Pharmacia Fine Chemicals, Piscataway, N. J.)
were treated as described for the immunoprecipitates, loaded
onto separate gels, and electrophoresed simultaneously with
the labeled antigens. The gels were fixed for 4 hr in isopropyl
alcohol:acetic acid:water (4:1:5) and stained overnight
with 0.5% Coomassie Blue in 7% acetic acid. Gels were
destained at 37° in 7% acetic acid. The molecular weights of
the labeled antigens were estimated from log molecular weight
versus mobility curves of the standards.

RESULTS

The serological reactivity and diagnosis of the 20 AMML
and CML-BC patients studied are given in Table 1. The patients
were all adults being seen in the hematology clinic of the
Department of Medicine at Duke University Medical Center. All
patients classified as AMML had typical myeloblasts as well
as cells with monoblastic features. With one exception (Patient
19), all of the CML-BC patients were positive for Ph¹ chromo-

\[
\begin{array}{cccc}
\text{Patient} & \text{Anti-TLAA} & \text{Anti-la} & \text{Normal serum} & \text{Diagnosis} \\
\hline
1 & 100 & 90 & 30 & NT^a AMML \\
2 & 85 & 80 & 15 & NT AMML \\
3 & 80 & 90 & 10 & NT AMML \\
4 & 90 & 90 & 15 & 5 AMML \\
5 & 70 & 90 & 10 & 27 AMML \\
6 & 90 & 90 & 30 & 5 AMML \\
7 & 100 & 90 & 15 & NT AMML \\
8 & 80 & 90 & 10 & 5 AMML \\
9 & 70 & 90 & 15 & 15 AMML \\
10 & 95 & 95 & 15 & 1 AMML \\
11 & 100 & 100 & 20 & 1 AMML \\
12 & 90 & 90 & 30 & NT AMML \\
13 & 100 & 90 & 15 & NT AMML \\
14 & 100 & 100 & 10 & 1 AMML \\
15 & 100 & 100 & 10 & NT AMML \\
16 & 80 & 80 & 10 & 1 AMML \\
17 & 55 & 75 & 10 & 8 AMML \\
18 & 70 & 90 & 10 & 6.5 CML-BC \\
19 & 85 & 75 & 15 & 0 CML-BC \\
20 & 75 & 95 & 15 & 5 CML-BC \\
\end{array}
\]

^a NT, not tested.
The patients were studied in the untreated or relapse phase of their disease. Peripheral blood samples were tested from all patients except Patients 9 and 16, which were bone marrow specimens. All patients showed a high degree of reactivity with xenonantisera defining the human TLAA and Ia-like antigens. In general, more than 75% of the cells were lysed by the anti-TLAA serum, and 90% or more of the cells were lysed by the anti-la serum. The titer of both antisera was generally greater than 1:40 when tested against these cells. In several cases, the cells were completely lysed by both antisera. When E-rosettes were also done on 13 of these patients, the values were low, with only 2 patients having more than 10% E-rosette-forming cells. A heterogeneous group of 8 acute nonlymphocytic leukemia patients showed reactivity only with the anti-la serum (data not shown). Cells from 26 ALL patients reacted with either the TLAA or la antisera but not with both reagents (5).

Cells from one AMML (Patient 4) and one CML-BC patient (Patient 19) were studied using both cytotoxicity assay and immunofluorescence. By cytotoxicity, the cells were strongly reactive with antisera detecting both T-cell and Ia antigens (Table 1). Cells from both patients formed 5% or less E-rosettes. When the same cells were tested by immunofluorescence using the rabbit anti-T-cell serum plus a fluoresceinated antiglobulin reagent and the monkey anti-B-cell serum plus a rhodamine-labeled antiglobulin reagent, 80% or more of the cells showed dual fluorescence.

Serial studies done on one AMML patient (Patient 10) are shown in Table 2. When first tested, the patient presented an essentially normal serological profile, with 70% E-rosette-forming cells and 90% lysis by the anti-T-cell serum. When the patient relapsed, approximately 2 weeks later, both peripheral blood and bone marrow samples showed a strong dual reactivity. The serological pattern again returned to normal when the patient was in remission several months later. With a subsequent relapse in March 1978, the cells were again reactive with antiserum defining both the T-cell and Ia antigens. At this time, only 1% of the cells formed E-rosettes. Several other AMML patients have shown patterns similar to this. That is, when the patient is in remission, the serological pattern is essentially normal. Approximately 80% of the remission lymphocytes are lysed by the anti-TLAA sera. Bone marrow samples from remission patients or from normal donors are negative when tested with anti-TLAA antisera. When the patient relapses, the cells from peripheral blood or bone marrow are reactive with both the anti-TLAA and anti-B-cell sera.

Aliquots of both the anti-TLAA and anti-la sera were quantitatively absorbed with cells from AMML Patient 14. The results are given in Chart 1. Absorption of the anti-TLAA serum with increasing numbers of AMML cells removed reactivity when the absorbed serum was tested against HSB cells and thymocytes. Similarly, absorption of the anti-la serum with AMML cells removed the reactivity of this antisera for chronic lymphocytic leukemia cells and peripheral blood B-cells from normal donors. Absorption of aliquots of the monkey anti-TLAA antisera with peripheral blood lymphocytes or thymocytes showed that 8 x 10⁵ cells/ml and 5 x 10⁶ cells/ml, respectively, were required to reduce the TLAA to background levels when the antisera were tested by immunoprecipitation and gel electrophoresis against 125I-lactoperoxidase-labeled HSB cells. Additional absorption of the monkey anti-TLAA antisera with 10⁶ cells/ml from pooled B-lymphoblastoid cell lines did not reduce the titer when the antisera was tested by cytotoxicity on normal peripheral blood lymphocytes or T-cell lines. Absorption of the monkey anti-la with 2 x 10⁵ pooled cells from B-lymphoblastoid cell lines per ml was sufficient to remove cytotoxic reactivity of the antisera for B-cell lines (data not shown). Conversely, additional absorption of the anti-la sera with 10⁵ cells from pooled T-lymphoblastoid cell lines did not reduce the cytotoxic titer when the antisera was tested on B-cell lines. Thus, while the quantitative amounts of TLAA expressed on AMML cells may nearly equal that of lymphocytes and thymocytes, the quantity of la-like antigens is at least 25 times less than that expressed by B-lymphoblastoid cell lines.

To confirm the quantitative absorption studies, radioimmunoprecipitation and SDS-polyacrylamide gel electrophoresis were performed with the absorbed sera and various 125I-lactoperoxidase-labeled membrane antigen preparations. A high-molecular-weight antigen (M.W. ~170,000) was precipitated from 125I-labeled, detergent-solubilized peripheral blood lymphocyte membranes by the anti-TLAA antisera (Chart 2). When this antisera was additionally absorbed with 5 x 10⁶ AMML cells/ml and then used for radioimmunoprecipitation and SDS-polyacrylamide gel studies, this high-molecular-weight peak was reduced to background levels.

Similar studies were performed using 125I-lactoperoxidase-labeled, detergent-solubilized SB membranes and the monkey anti-la antisera (Chart 3). The M.W. 35,000 and M.W. 28,000 la antigens which were precipitated from SB membranes by the HSB-absorbed antisera were reduced to background levels when the antisera was further absorbed with AMML cells.

To further establish the presence of TLAA and la antigens on AMML cells, cells from one patient (Patient 8) were labeled.

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**Table 2**

<table>
<thead>
<tr>
<th>Date</th>
<th>Sample tested</th>
<th>Clinical status</th>
<th>% of lysis at 1:10 antisera dilution</th>
</tr>
</thead>
<tbody>
<tr>
<td>11/10/77</td>
<td>PB³</td>
<td>Remission</td>
<td>Anti-TLAA 90% E-rosettes</td>
</tr>
<tr>
<td>11/22/77</td>
<td>PB³</td>
<td>Relapse</td>
<td>Anti-TLAA 90% E-rosettes</td>
</tr>
<tr>
<td>1/30/78</td>
<td>PB³</td>
<td>Remission</td>
<td>Anti-TLAA 80% E-rosettes</td>
</tr>
<tr>
<td>2/27/78</td>
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</tr>
<tr>
<td>3/30/78</td>
<td>PB³</td>
<td>Relapse</td>
<td>Anti-TLAA 100% E-rosettes</td>
</tr>
</tbody>
</table>

³ PB, peripheral blood; BM, bone marrow.

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**Chart 1.** Quantitative absorption of monkey anti-TLAA (A) and monkey anti-la sera (B) with AMML cells. Details of the absorption procedure are given in "Materials and Methods." The number of absorbing cells shown along the abscissa represents the total number of cells used. The absorbed sera were tested by cytotoxicity assay; the titer is recorded as the highest dilution of serum giving at least a 30% increase in lysis over normal serum controls.
with 125I using lactoperoxidase, solubilized in Triton X-100, and used in radioimmunoprecipitation and polyacrylamide gel electrophoresis studies. The cell preparation used for labeling contained 5% E-rosette-forming cells and consisted of 98% blasts. These blasts showed strong reactivity for both TLAA and Ia antisera (Table 1). As shown in Chart 4, both the TLAA and Ia-like antigens could be precipitated from this antigen preparation. These reduced precipitates were electrophoresed on 7.5% SDS-gels so that both the TLAA and Ia antigens could be shown. Determination of TLAA molecular weight is not accurate on these gels, and therefore the TLAA value is listed as M.W. >150,000. When similar precipitates were electrophoresed on 4% SDS-gels, the molecular weight of the TLAA was 160,000 to 170,000, as we have reported previously (3, 4). An additional antigen (M.W. 94,000) was precipitated from these cells by absorbed antisera to HSB. Work from this laboratory (2) suggests that this antigen is similar to the common ALL-associated antigen described by others (6, 20, 25). The antigenic sites on the M.W. 94,000 moiety may be arranged in a fashion which does not result in cytolysis after reaction with antibody and complement, since a number of cells (NALM-1 and some non-B, non-T ALL cells) have been found which are negative by cytotoxicity assay but which possess this antigen by radiolabeling and SDS-gel studies (2). The common ALL antigen has been reported to be present in some cases of acute nonlymphocytic leukemia by other investigators (6, 10).

**DISCUSSION**

In this study, a high percentage of cells from 20 patients with AMML or CML-BC was shown to react with both anti-TLAA and anti-la antisera. In most cases where E-rosette studies were done, less than 10% of the target cell suspension formed E-rosettes. Quantitative absorption of the anti-TLAA serum with cells from an AMML donor showed that these cells could remove reactivity of the antiserum for HSB and thymocytes. Similarly, when the absorbed serum was tested by radioimmunoprecipitation and SDS-polyacrylamide gel electrophoresis using 125I-lactoperoxidase-labeled, detergent-solubilized peripheral blood lymphocytes, the peak at M.W. 170,000 was removed. Thus, by several criteria, the T-cell-associated antigen detected on AMML cells seems to be the TLAA described in our other reports (3–5). Quantitative absorption of the anti-la serum with AMML cells followed by either microcytotoxicity assay or radioimmunoprecipitation and SDS-polyacrylamide gel electrophoresis showed that Ia-like antigens were present on these cells. In addition, both TLAA and Ia antigens could be precipitated from radiolabeled, detergent-solubilized leukemic blasts from an AMML patient. Using a double fluorescence technique, over 80% of the blast cells from one AMML patient and one CML-BC patient were shown to express both TLAA and Ia antigens.

While a number of other investigators have reported the presence of T- and B-cell markers on the same cell (9, 11, 21, 23, 24), most of this work has involved isolated case studies of patients with various lymphoproliferative disorders. In most instances, this dual reactivity was based on mixed rosette studies using E-rosettes and C3 rosettes (erythrocytes coated with antibody and complement). In one instance (11), E-rosettes were combined with an assay for surface immunoglobulin.

Ia-like antigens have been reported by other investigators on cells from some acute myelogenous leukemia and CML-BC patients using both heteroantisera (7, 22) and alloantisera (8) to these antigens. Winchester et al. (26) precipitated peaks at M.W. 37,000 and M.W. 28,000 from leukemic myeloblasts using an anti-la serum; absorption of the antiserum with leukemic myeloblasts removed serological reactivity when the antiserum was tested on B-cell lines. In addition, normal granulocytic cells have been shown to express Ia antigens at an early stage of differentiation (26). The majority of the positive
cells in normal marrow were myeloblasts with some positive promyelocytes. In one of the few other studies in which acute nonlymphocytic leukemia and CML-BC cells were tested with anti-T-cell and anti-la sera, Janossy et al. (12) reported that these cells were negative with the T-cell antisera. However, their T-cell antiserum was prepared in rabbits against monkey thymocytes and absorbed with acute myeloid leukemia cells; thus, the specificity of their reagent may be different from our anti-TLAA antisera.

Antisera raised against non-B, non-T ALL cells have been reported to detect a CALL antigen with a molecular weight near 100,000 (6, 25). While this antigen has been reported chiefly on cells from non-B, non-T ALL patients, there are reports of its occurrence on cells from some acute myelocytic leukemia patients (6, 10) as well as on cells from CML-BC patients (6, 12, 20), some T-cell ALL patients (20), and some T-lymphoblastoid cell lines (6). Data from this laboratory (2) suggest that the antigen near M.W. 96,000 precipitated from the radiolabeled AMML cells by absorbed antisera to HSB is similar to the CALL antigen (Chart 4). However, it may be that there are at least 2 distinct antigens in this region between M.W. 94,000 and M.W. 100,000 and that the tube method does not resolve them. Current radiographic SDS-polyacrylamide gel electrophoresis studies using the discontinuous system of Laemmli (13) and slab gel electrophoresis may help to resolve this point. The possible discrepancy between our data and previously reported data on distribution of the CALL antigen may result from differences in methods, since most earlier work involved immunofluorescence and cytotoxicity assays rather than radiolabeling and gel electrophoresis.

NALM-1 and non-B, non-T ALL cells were negative by cytotoxicity assay with the anti-HSB sera, yet the M.W. 94,000 antigen was detected by radioimmunoprecipitation and SDS-gel studies on these cells. Either the antibodies to this antigen are not cytotoxic or the antigen distribution is such that the cells are not lysed. Additional absorption of the antiserum with 5 x 10^6 thymocytes/ml removes cytotoxic reactivity against all cells tested, yet the M.W. 94,000 antigen can still be detected by radioimmunoprecipitation and SDS-gel electrophoresis on positive cells. Thus, the absorbed antiserum to HSB are serologically T-cell specific, although an additional antigen can be detected on some cells by gel electrophoresis.

Several serological reports have previously appeared from this laboratory on antigens of cells from AMML and CML-BC patients (17, 18). Simian antisera to both lymphocytic and myelogenous leukemia cells were shown to lyse cells from AMML donors. Since all antisera were absorbed with human WBC, any potential TLAA reactivity would have been removed. Some of the leukemia antisera used in these earlier studies may have been detecting la-like antigens or the CALL antigen. However, cross-absorption studies (17, 18) indicate that there may be other antigen(s) detected by these sera which do not fit the pattern of previously described surface membrane antigens. The anti-T- and anti-B-cell sera which were shown to react with AMML and CML-BC patients in an earlier report (17) are different from the ones used in this study. The molecular nature of the antigens detected by these earlier antisera were not characterized by labeling and electrophoresis studies. The earlier report (17) that absorption with AMML or CML-BC cells removed anti-B-cell but not anti-T-cell reactivity may be a reflection of the number of cells used for absorption. Quantitative absorption of anti-TLAA serum with AMML cells required >5 x 10^6 cells/ml to significantly reduce its cytotoxic titer; in the earlier report, the anti-T-cell serum was absorbed with only 2 x 10^6 AMML cells/ml.

Our current and past data indicate that cells from most AMML and CML-BC patients express la-like antigens and a T-cell-associated antigen simultaneously (3, 17). Thus, leukemic cells from these patients may be representative of a marrow stem cell with characteristics of both B- and T-cells. The TLAA-la-positive leukemic cells from most AMML and CML-BC patients do not form a high percentage of E-rosettes. However, recent studies by us have demonstrated that 80 to 100% of peripheral blood T-lymphocytes from normal donors when cultured on ‘conditioned’ medium can form E-rosettes and express la antigens as well (15). It may be that some cells have the genetic information for expression of both T-cell and HLA-D (la) antigens, but the quantitative expression of these antigens is influenced by cellular differentiation events. When these cells are activated or become malignant, they may then by dedifferentiation show quantitative differences in the expression of T-cell or la-associated antigens.

Abramson et al. (1) have already provided evidence that some murine stem cells give rise to both myeloid and lymphoid cells, including B- and T-lymphocytes, while other stem cells are more restricted in their differentiation pathway. Since at least one of these antigens (la) can be considered a differentiation-type antigen and the TLAA shows restricted tissue and cellular distribution, membrane marker studies may help to define the stem cell origin of some of the human leukemias.

REFERENCES


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