To the Editor:

The report by Claxton et al.1 describes a close correlation between the type of aberrant mRNA transcripts for PML/RAR-α in 21 patients with acute promyelocytic leukemia (APL) and expression of CD2, a surface antigen of the Ig superfamily of cellular adhesion molecules that is generally associated with a T-lymphocyte phenotype. Since we have recently studied a number of patients with APL using a battery of surface markers and reverse transcription polymerase chain reaction (RT/PCR) assay for PML/RAR-α, we repeated this evaluation on a considerably larger data set. For this analysis, we used probes recently described3 to analyze mRNA extracted from leukemic bone marrow cells of 52 patients who presented to this center at the time of initial diagnosis or after relapse from cytotoxic chemotherapy.

Flow cytometry was performed on Ficoll-Hypaque-separated mononuclear cells stained with a panel of commercially available monoclonal antibodies. Only fresh, nonfrozen samples were processed. We found no correlation between the type of PML/RAR-α transcript and CD2 expression. In our 52 patients, 16 cases showed pattern A (5′ in Claxton et al) and 36 cases showed pattern B (3′ in Claxton et al). Of the pattern A cases, 8 cases were positive for CD2 (ie, expression >20%) and 8 cases were negative. Of the pattern B cases, 12 were positive for CD2 expression and 24 were negative. Moreover, using the score test for the logistic regression model, no correlation was noted between the type of PML/RAR-α transcript and CD2 (with percent positivity expressed as a continuous variable) (P = .37). We also attempted to correlate the RT/PCR pattern of PML/RAR-α expression with several other parameters, including initial leukocyte count, expression of CD13 and CD33, presence or absence of disseminated intravascular coagulation, M3 or M3 variant morphology, and presence or absence of the newly described “retinoic acid syndrome.” However, none of these correlations were significant when tested individually. We did note that transcript type and CD13 expression together were correlated with the total leukocyte count; however, this association does not appear clinically informative. (This data set will be provided upon request to the authors.)

Although the presence of “aberrant surface phenotypes” has now been reported in a large proportion of patients with acute myeloblastic leukemia,4 we conclude that CD2 expression is not related to the type of breakpoint fusion in APL.

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REFERENCES

CD2 expression and PML/RAR-alpha transcripts in acute promyelocytic leukemia [letter; comment] [see comments]

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