To the Editor.

As discussed by Maslak and others, our report showed an association between CD2 expression and PML gene breakpoint in a series of molecularly analyzed acute promyelocytic leukemia (APL) patients. In this work we also described novel probes for Southern analysis and a reverse transcription polymerase chain reaction (RT-PCR) assay and observed a correlation between CD2 expression and the diagnosis of APL. Additional surface phenotype data on APL and other forms of AML is presented in a report in press. These studies are directed at the identification of coexpression of rare or abnormal combinations of surface antigens (eg, CD2 and myeloid markers such as CD13 or CD33). Given that most of these data have been accumulated without the benefit of direct two-color coexpression analysis, we have chosen to require a 10% overlap of marking levels for the secure demonstration of coexpression. Thus, for CD2 and CD33, the %CD2 + %CD33 would need to be greater than 110% to identify a case as coexpressing these antigens. This ensures that at least 10% of gated cells express both antigens. It must be noted that this approach will miss many cases who might be shown by two-color studies to have coexpression.

Maslak et al from Memorial Hospital describe in their letter their large series of APL patients and show no correlation between CD2 expression and PML breakpoint as determined by RT-PCR. They have kindly provided this data set to us for review so that we may understand the apparent difference between these two series of patients. The Memorial data are extensive, but lack information on other T-cell antigens that would allow exclusion of contamination of the blast gated population with T lymphocytes. Otherwise, their methodology appears similar to ours and therefore some conclusions are possible. By our 10% overlap criteria (above), 6 of 7 of our 5' breakpoint patients and 1 of 11 of our 3' patients express CD2 on promyelocytes. In contrast, 6 of 14 5' patients and 11 of 28 3' patients in the Memorial series express this antigen. Applying this group's 20% expression criterion (which may allow T-cell contamination to contribute false positives), 5 of 7 of our 5' cases and 1 of 11 of our 3' cases express CD2, as opposed to 8 of 14 5' and 11 of 28 3' leukemias at Memorial. We note that, when the 20% CD2 expression data of the two series of patients is pooled, the $\chi^2$ test still shows a significantly more frequent CD2 expression in 5' cases ($P = .02$).

It is clear from both visual inspection and $\chi^2$ analysis of these figures that the two series show disparate results. At present, we are
unable to explain this difference. It is possible that ethnic, genetic differences between the two series populations (eg, we have a large number of Hispanic patients) might account in part for the difference. Ultimately, larger series and two-color coexpression analysis should be performed to address this issue. This work has been initiated here.

REFERENCES


CD2 expression and the PML-RAR gene [letter; comment]

D Claxton, C Reading and A Deisseroth