found on the surface of T leukemic cells was in fact bound to receptors for the Fc portion of IgG, since the reagents used would not double-label normal T cells binding ox red blood cells coated with an IgG anti-ox antibody, nor would they label the cells from acute leukemia that we have found will form rosettes with the above ox cells. Antibodies against leukemic cells may explain the finding of surface membrane immunoglobulin on these cells. Such cases, however, must be rare; we have not detected them in 64 other cases of acute lymphocytic leukemia screened in our laboratory, and it is very unlikely that such an antibody would be monoclonal in nature and produce a detectable IgG spike. Furthermore, 81% of the blast cells were labeled, not 100%, which might be expected were this to be an antibody against leukemic cells.

We remain confident enough of our conclusions to repeat the warning that TdT levels may be elevated in some B cell leukemias, and one case of so-called pre-B cell leukemia out of four recently described had moderately high levels of TdT activity.5

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REFERENCES

Neutrophil Products Inhibiting Cell Proliferation

To the Editor:

The paper by Herman et al. regarding neutrophil products that inhibit cell proliferation and their relation to granulocytic “chalone” induced in us a sense of deja vu. Experiments in other laboratories and our own about 15 years ago indicated that normal and CML leukocytes contained a factor that inhibited the uptake of 3H-TdR into target cells in vitro. The inhibitory factor was found to be the enzyme thymidine phosphorylase. This enzyme catalyzes the conversion of thymidine to thymine. Further evidence for degradation of thymine by leukocytes was also described. Thymidine phosphorylase activity was found in the medium after intact leukocytes were incubated and normal leukocytes were found to contain significantly higher levels of this enzyme than leukemic leukocyte. The enzyme was heat labile and non-dialyzable. Phosphate was a substrate. When the enzyme was preincubated with 3H-TdR before addition of the target cells, virtually complete inhibition of thymidine uptake was found.

We suspect that at least part of neutrophil-derived inhibitory factor(s) (NDIF) described by Herman et al. is this enzyme that can effectively convert tritiated thymidine to thymine and perhaps further degradation products no longer available for DNA synthesis. The heat-labile activity is certainly the most likely candidate for this. To the list of artifacts mentioned by Herman et al. that influence assays of chalone-like materials, including dilution of isotope with cold thymidine, changes in nucleotide pool size, interference with phosphorylation and transport, and cell death, we would add the presence of thymidine phosphorylase. The enzyme is also capable of degrading deoxyuridine, the uptake of which we also found to be inhibited by leukocyte thymidine phosphorylase. The observation by Herman et
REFERENCES

1. Herman SP, Golde DW, Cline MI: Neutrophil products that inhibit cell proliferation: Relation to granulocytic "chalone." Blood 51:207-219, 1978

Neutrophil Products Inhibiting Cell Proliferation: Reply

To the Editor:

The point made by Dr. Marsh and Dr. Perry is perfectly valid. The presence of thymidine phosphorylase is one more element that may influence an assay based on the inhibition of incorporation of tritiated thymidine.

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Neutrophil products inhibiting cell proliferation [letter]

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