separation and functional studies on cord T subpopulations should shed more light on the problem.

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REFERENCES

To the Editor:

We appreciate the comments of Drs Gerli and Rambotti on our article. Their findings of circulating cells in cord blood bearing the phenotypes OKT3 \textsuperscript{+} OKT8\textsuperscript{-} and OKT3 OKT4\textsuperscript{-} is of obvious importance and reflects the increasing complexity of T cell ontogeny defined by monoclonal antibodies. We unfortunately did not look for this particular phenotype in our study. We did, however, find a significant percentage of cells (25\% ± 17\%) bearing the phenotype OKT4\textsuperscript{-} OKT8\textsuperscript{-}, showing an immature phenotype. The validity of the methodology we used for the double-labeling study was verified by the absence of cells double labeling with OKT4 and OKT8 in adult blood.

The reagents described by the authors may well be more refined, but were not available to us and are probably not available to most groups working in this field. With the reagents available to us, our methodology was appropriate and its accuracy was tested.

We cannot explain the discrepancy between our results on OKT6 labeling of cells and those of Gerli and Rambotti, and Maccario et al. Our results in ten of the cord bloods tested were confirmed using an Ortho (Raritan, NJ) cytofluorograph, which excludes observer error in recognizing positively labeled cells. In one specimen of cord blood we examined, OKT6\textsuperscript{-} cells were not evident. In all others, however, OKT6\textsuperscript{-} cells were present in significant numbers (24\% ± 8\%).

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TRANSLOCATION (1;3) IN MYELODYSPLASTIC DISORDERS

To the Editor:

We have read the Concise Report by Moir et al about a new translocation t(1;3) (p36;q21) in myelodysplastic disorders.\textsuperscript{1}

We observed a similar karyotypic abnormality in a male patient, 80 years old, diagnosed in June 1979 with a sideroblastic anemia (30\% ringed sideroblasts). During 45 months of evolution, a monocytosis was observed in peripheral blood (45\%) and in bone marrow (37\%). A cytogenetic study done on a marrow sample showed a translocation (1p;3q) (p35;q21) by GTG banding in thirty metaphases analyzed (Fig 1).

Abnormalities of the long arm of chromosome 3 have been reported in association with high platelet counts.\textsuperscript{2 4} The authors hypothesized that the long arm of chromosome 3 may be involved in megakaryocytic maturation and platelet production.

In our patient the platelet count was initially 99 x 10\(^9\)/L and rose to 658 x 10\(^9\)/L at the time of the cytogenetic study (March 1983). This anomaly affecting q21 is similar to that reported by Bernstein et al\textsuperscript{5} and Mecucci and Van den Berghe,\textsuperscript{1} although in their cases the anomaly was t(3q\textsuperscript{-}, 3q\textsuperscript{+}). The breakpoint in band q21 is the same as that observed by Golomb et al,\textsuperscript{5} Norrby et al,\textsuperscript{6} and Carbonell et al.\textsuperscript{7} Patient 1 reported by Moir et al\textsuperscript{8} showed a high platelet count also.

Might the q21 band or a portion of the chromosome near it be the common area affected in these patients with thrombocytosis independently of the type (inversion or translocation) of structural alteration?

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Translocation (1,3) in myelodysplastic disorders [letter]

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