LN2 recognizes B-cell follicles in frozen tissue, while no reactivity can be observed using the same dilution in paraffin sections.

Second, in contrast to Ki-B3, those antibodies mentioned as more or less specific for B cells recognize peripheral T-cell lymphomas, whereas those more or less specific for T cells recognize especially low-grade B-cell lymphomas (our own observations). This restricts the applicability of these monoclonal antibodies in the differential diagnosis of peripheral T-cell and B-cell lymphomas. Like other antibodies recognizing antigens in formalin-fixed material, Ki-B3 also detects T-lymphoblastic lymphomas. But this crossreactivity is of little relevance because lymphoblastic lymphomas can be easily differentiated from other peripheral B-cell or T-cell lymphomas.

In our paper we did not state that LN1 and LN2 do not react with formalin-fixed tissue, but said that they recognize B-cell related antigens after B5 fixation. We mentioned this point because of the difference we found in the reactivity of these antibodies in fresh frozen tissue and after formalin fixation. If the reactivity of a monoclonal antibody designated to recognize a formalin-resistant antigen is dependent on the duration and the concentration or kind of fixative, then a negative result is useless. Such a difference in reactivity following different kinds of fixation was not found for Ki-B3.

A.C. FELLER
HEINZ J. RADZUN
MOHAMMAD R. PARWARESCH
H.H. WACKER
Department of Pathology
University of Kiel
Kiel, FRG

GERHARD MOLDENHAUER
Institute for Immunology and Genetics
German Cancer Research Center
Heidelberg, FRG

---

GLYCOPEPTIDES IIb AND IIa IN K562 CELLS

To the Editor:

Recently, Silver et al.1 as well as others2,3 reported that K562 cells, stimulated by phorbol-12-myristate-13-acetate (PMA), produce glycoprotein (GP) IIa but not GP IIb. In this respect, K562 cells distinguish themselves from several other cell types, including smooth muscle cells, endothelial cells, and megakaryocytes, which produce both GP IIa and a GP IIb-like molecule.4

The authors concluded therefore that in stimulated K562 cells only the GP IIa gene is expressed. We have shown previously5 that this view is incorrect (Fig 1). Our finding is clearly at variance with that of Silver et al.1 How to explain this discrepant observation is not clear at the moment. It is interesting to note that initially it was thought that cultured endothelial cells only produce GP IIa.5 It soon became clear that endothelial cells synthesize a GP IIb-like molecule as well.7,8 Apparently, for an as yet unclear reason, the synthesis of GP IIb by certain cell types can be overlooked.

It seems obvious now that K562 cells stimulated with PMA, as well as many other cell types, synthesize a GP IIb/IIa-like complex. The question whether GP IIa can be expressed in the absence of GP IIb clearly requires further investigation.

J.C. GILTAY
H.J. BRINKMAN
P.W. MODDERMAN
P.A.T. TETTEROO
J.A. VAN MOURIK
Central Lab of The Netherlands
Red Cross Blood Transfusion Service
Amsterdam

REFERENCES

To the Editor:

Giltay and colleagues immunoprecipitated a GPIIb-like molecule from lysates of phorbol ester-stimulated K562 cells. As we recently reported,1 we were unable to immunoprecipitate such a protein from these cells using either polyclonal antibodies against GPIIb or against GPIIIa (to immunoprecipitate a GPIIb/IIIa complex). From the intensity of the bands in Fig 2 of our paper and the bands in the Fig 1 supplied by Giltay et al, it is unlikely that we would overlook a GPIIb-like band. Furthermore, our result was confirmed using anti-GPIIb monoclonal antibodies and flow cytometry and is consistent with the recent report of Papayannopoulou et al.2 There are several potential explanations for the discrepancy noted by Giltay et al. First, there may be genetic variation between the K562 cell subline we studied and that studied by Giltay and coworkers. Second, the protein identified by these workers may not be GPIIb. Using cDNA encoding the full-length GPIIb sequence, we were unable to detect GPIIb mRNA transcripts in either unstimulated or phorbol ester-stimulated K562 cells.3 Moreover, we could not detect GPIIb mRNA transcripts in endothelial cells,4 a result also reported by Suzuki et al3 and Bray et al.4 The previously reported endothelial cell GPIIb-like protein is actually the α subunit of the vitronectin receptor, a heterodimer also containing GPIIIa.5 The mobility of the K562 cell GPIIb-like band in Fig 1 supplied by Giltay et al suggests that it is also the α subunit of the vitronectin receptor. Finally, whether GPIIIa can exist in the cell membrane without the formation of a heterodimer complex will require further investigation.

JOEL S. BENNETT
GASTON VILAIRE
MARGARET MCDONOUGH
Hematology-Oncology Section
Hospital of the University of Pennsylvania
Philadelphia, PA

SAMUEL M. SILVER
Hematology-Oncology
Simpson Memorial Institute
University of Michigan
Ann Arbor, MI

REFERENCES


SOLUBLE INTERLEUKIN-2 RECEPTOR IN CHILDHOOD NON-HODGKIN’S LYMPHOMA

To the Editor:

Recently, Pui et al presented some interesting findings in childhood non-Hodgkin’s lymphoma.1 Using an ELISA method to determine the serum levels of a soluble receptor for interleukin-2 (sIL2R), they conclude that an elevated sIL2R level is a more potent predictor of prognosis than clinical stage or lactase dehydrogenase (LDH) level. They use as controls an inclusive group of children ranging from 1 to 6 years of age with otitis media (median age: 3 years) and observe higher levels than the published norms for adults (404 to 942 U/mL). They speculate that the higher levels were stimulated by ear infections.

Using the same method for sIL2R analysis, we studied the sera of 122 normal children to prepare normal standards for childhood. The results are displayed in the scattergram (Fig 1) and illustrate that normal uninfected children have slightly higher than adult levels at
Glycoproteins IIb and IIIa in K562 cells [letter]
JC Giltay, HJ Brinkman, PW Modderman, PA Tetteroo and JA van Mourik