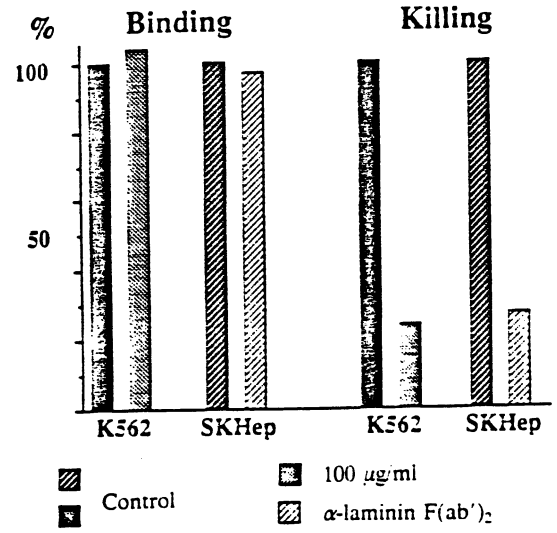

Fig. 3. Single-cell cytotoxicity assay. The effect of antilaminin $F(ab')_2$ (100 μg/ml) on target-cell binding and killing by Leu-19$^-$/CD3$^-$ LAK-cells in a 90-min agarose single-cell assay is shown.

also been identified on purified rat LAK-cells (A-LAK-cells) indicating possible homology between recognition structures expressed on LAK-cells from different species [7]. In this regard, it is interesting that the patterns of tumour target cells 'recognized' by rat and human LAK-cells are considerably overlapping.

We believe that the laminin like p48 protein is a receptor structure expressed on non-MHC-restricted cytotoxic cells of NK- or T-cell phenotype and that this structure is critically involved in their target-cell recognition/activation of cytotoxicity.

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N21.6 Phosphatidylinositol-anchored antigens defined by non-lineage mAb

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Cell-surface proteins are usually anchored to the membrane via a transmembrane hydrophobic peptide followed by a cytoplasmic hydrophilic peptide. Recently, a number of proteins have been described which have an unusual membrane anchor consisting of a phosphatidylinositol glycan (PIG) moiety which replaces a transmembrane hydrophobic peptide in the precursor. These proteins are released from the cell surface by phosphatidylinositol-specific phospholipase C (PI–PLC) [1]. We have reported both PIG-anchored and polypeptide-chain-anchored isoforms of LFA-3 and CD16 (FcRγIII).

In order to identify further antigens with PIG anchors we have compared the binding of antibodies to cells before and after PI–PLC treatment. The non-lineage panel was screened on three different cell types with ELISA and the results were confirmed by immunofluorescence flow cytometry (Table 1). Fifteen mAb showed a significant decrease in binding to cells after PI–PLC treatment. mAb N14 (BW209/2) and N87 (CLB/FcGran1) identify CD16 and N90 (MEM-15) identifies CD14; both of these proteins are known to be PIG-anchored.

The T-cell and activation panels were screened on the

Table 1. Effect of PI–PLC treatment on binding of Workshop non-lineage panel of antibodies

Workshop no.	mAb name	Per cent decrease after PI–PLC (control SLFI)*		
		Jurkat cells	Granulocytes	Monocytes
N007	143-30	93 (2.8)	66 (0.3)	75 (0.4)
N014	BW209/2	—	70 (3.0)	—
N029	WM68	100 (2.8)	—	100 (0.3)
N033	LO-MN25	97 (5.8)	—	86 (0.7)
N036	YTH53.1	96 (14.8)	—	—
N043	G26	33 (0.3)	—	33 (0.6)
N047	J4-57	99 (7.3)	—	100 (0.8)
N064	BRIC5	50 (3.4)	—	43 (0.7)
N068	Tü145	100 (4.2)	—	—
N084	WM63	—	—	100 (1.8)
N087	CLB/FcGran1	—	79 (4.3)	—
N090	MEM-15	—	—	69 (6.5)
N102	TS2/9	46 (0.9)	—	—
N113	K31	100 (4.2)	—	—
N125	F2B7.2	91 (2.2)	66 (0.3)	—

*SLFI, specific linear fluorescence intensity. —, not analysed. The SLFI were directly comparable between granulocytes and monocytes. Jurkat cells were analysed at a different time with different amplification settings. The SLFI without PI–PLC treatment is shown in parentheses.

Jurkat T-cell lymphoma. mAb A87 (JML-H105) and T18 (52-2B6) showed 66 and 79 per cent decrease in binding to Jurkat cells after PI–PLC treatment, respectively.

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