

The data presented here show PAR availability at 70°S for one complete revolution of earth around the sun. The data may find applications in plant science, oceanography and marine biology in Antarctic region.

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Reversal of interferon-induced lymphokine-activated killer resistance in two murine cell lines by exposure to acid pH

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Interferon is known to augment the expression of MHC I antigens on a variety of tumour cell lines. In most cases, a simultaneous decline in the susceptibility of these tumour cells to natural killer (NK) cells and lymphokine-activated killer (LAK) cells is also observed. In the present communication, we have studied the LAK susceptibility and MHC I levels on two NK-resistant murine cell lines P815 and SP₂O. Treatment with interferon resulted in an increased MHC I expression as well as a decreased LAK susceptibility in both cell lines. A brief exposure of the interferon-treated tumour cells to citrate buffer (pH 3) resulted in a marked decline in the levels of MHC I and restoration of LAK susceptibility of the target cells. A direct role of MHC I antigens in determining the LAK

susceptibility of target cells is suggested by these results.

LYMPHOKINE-activated killer (LAK) cells are derived primarily from natural killer (NK) cells by interleukin-2 (IL-2) activation. There are qualitative differences between LAK cells and NK cells as the former are more efficient killers and lyse a wider range of target cells, including NK-resistant target cells. Quantitative expression of MHC I antigens on a tumour cell may be an important factor in determining its susceptibility to NK cells. In many systems, an inverse correlation between the quantitative MHC antigen expression on target cells and susceptibility to NK lysis has been demonstrated (reviewed by Ljunggren and Karre¹). Some recent studies have sought to investigate the role of target cell MHC class I antigen expression on LAK susceptibility of target cells. Wiebke *et al.*² have reported that a clear-cut correlation between enhanced MHC antigen expression and decreased LAK susceptibility was not observed in human tumour cell lines. Similar results were also reported by De Fries and Golub³, who observed that LAK susceptibility of certain human tumour cell lines following interferon treatment is not dependent on increased class I antigen expression. However, by depleting class I antigen expression by exposure to acid pH, Miyatake *et al.*⁴ reported that interferon-induced resistance to LAK lysis in cultured human gliosarcoma cells is, at least in part, due to enhanced levels of class I antigen expression.

In the present report, we have investigated the LAK susceptibility of two NK-resistant cell lines of murine origin, in which MHC I expression was initially boosted by interferon treatment and reduced thereafter by acid pH treatment: P815 (mastocytoma) and SP₂O (myeloma) cell lines used in this study were propagated in culture in RPMI-1640 supplemented with 10% FCS, 2×10^{-5} 2-mercaptoethanol, 300 μ g/ml glutamine and 60 μ g/ml gentamicin (complete medium). In order to generate LAK cells, spleen cells from C57B1/6 mice were cultured at 5×10^6 cells/ml with 200 U/ml of interleukin 2 (IL-2, a kind gift from Hoffmann La Roche, Nutley, NJ) in complete medium. After two days, cultures were split into two and supplemented with equal volumes of fresh medium and 200 U/ml IL-2. Cells harvested on day 5 from initiation of culture were washed and used as LAK effector cells.

Tumour cells were cultured (5×10^4 cells/ml in complete medium) with or without 200 U/ml recombinant murine interferon gamma for 48 h. After culture, the cells were harvested and washed. Portions of these IFN-treated tumour cells were subjected to a brief pH 3 treatment as described by Sugawara *et al.*⁵. Briefly, cell pellets were suspended in 0.5 ml of cold 0.2 M citric acid-Na₂PO₄ buffer (pH 3.0), containing 1 g/100 ml of bovine serum albumin. After 2 min,