x 10^4 (T47D) and 1.9 x 10^5 (MD-MB-231) per cell. Ratios between binding at the cellular surface and intracellular binding were between 1 : 2 and 1 : 3 except for SK-OV-3 which exhibited similar binding for both locations. Down regulation was found for all cell lines with between 20 nM and 100 nM of EGF. The combined data suggest that EGF modulates proliferation of gynaecological carcinoma cells by affecting its own receptor.

DIVERGENT EFFECTS OF γ-INTERFRON ON ADAPTIVE AND NON-ADAPTIVE IMMUNE DEFENSE MODULATE METASTATIC SPREAD OF MELANOMA CELLS

M.Zöller and A.Strubel

Institute of Nuclear Medicine, German Cancer Resarch Center, Heidelberg, F.R.G.

The correlation between γ-interferon (IFN) treatment, susceptibility to non-adaptive and adaptive immunity and metastatic spread was evaluated in the B16 melanoma system. Treatment of B16 with IFN abrogated lysability by NK cells and macrophages and metastaic capacity was increased (by 5 to 10 fold the number of lung nodules). On the other hand, IFN-induced enhancement of MHC antigen expression resulted in both an increased susceptibility towards cytotoxic T cells and an enhanced cytotoxic potential of T-cells stimulated by IFN-treated B16. This was reflected in a significant reduction of metastatic nodules and prolongation of survival time of mice immunized with IFN-treated B16 cells, regardless of whether they were challenged with treated or untreated cells. Thus, loss of NK cell- and macrophage-susceptibility of IFN-treated B16 led to increased metastasizing capacity, but IFN-treatment in combination with immunization significantly reduced metastatic spread, pointing to a dominance of the T cell effect.