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THE SUPPORT MECHANISM FOR CORTISOL METABOLISM BY LYMPHOCYTES: EFFECT OF CANCER PATIENTS' PLASMA

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Lymphocytes are capable of metabolizing cortisol to three metabolites which lack the immunosuppressive effect of their precursor. In our present work, we noted a linear correlation between glucose concentrations and the rate of cortisol metabolism by lymphocytes (LCM) in vitro. The LCM support mechanism and the effect of 1) normal plasma, 2) cancer patients' plasma, 3) AIDS patients' plasma and 4) seminal plasma were investigated further. We observed that: a) the effect of glucose on LCM was not influenced by insulin; b) almost all the utilized glucose was converted to lactate; c) lactate and ATP inhibited LCM; d) both NADPH and NADH exerted a direct positive effect on LCM in disrupted cells, but not in intact cells; e) glucose-6-phosphate and isocitrate reduced $NADP^+$ but only after the addition of the nucleotide; f) the plasmas of cancer patients, AIDS patients and seminal plasma showed a reduced effect upon LCM. This effect was not followed by reduction of glucose utilization. We conclude that the metabolism of cortisol by lymphocytes is complex, involving both endogenous and exogenous factors.

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CORRELATION OF ANTITUMOUR PROPERTIES OF PLATINUM COMPOUNDS WITH THEIR REACTIVITY TOWARD DNA: A SPECTROSCOPIC STUDY

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Interaction of mammalian DNA with cis-dichlorodiammine-platinum (II), its trans isomer, chlorodiethylenetriamineplatinum (II) chloride, and several platinum cytostatics of the second generation (derived from both platinum (II) and platinum (IV)) was investigated by means of measurement of circular dichroic spectra and denaturation-renaturation properties. It was found that the binding of antitumour active platinum compounds induced only minor disturbances of the premelting nature in the DNA secondary structure, whereas the inactive compounds induced formation of more extensive denatured regions in DNA molecules even at low binding levels.

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DESTRUCTION OF METASTATIC TUMOUR CELLS BY IMMUNOLOGICAL MEANS

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A reticulum cell sarcoma line which originally arose spontaneously in the uterus of an old (C57BL/Ka x C3H/He)F_1(BCF_1) mouse was shown either to metastasize to visceral organs upon s.c. back transplantation in both syngeneic and nude (Balb/c nu/nu) mice or it was accepted by semi-syngeneic (C57BL/6N x C3H/He)F_1 ($6HF_1$) mice with no metastasis formation. The tumour killed the former two hosts by metastatic growth even when grafted tumour was completely resected two weeks after implantation. In an attempt to control the metastatic growth by immunological means, variously treated spleen cells of either $6HF_1$ or Balb/c nu/+ mice were given to either BCF_1 or nude mice a few days after the removal of s.c. grafted tumours which had been carried for two weeks. All mice receiving spleen cells of either intact or non-specifically stimulated donors died of advanced metastases within 60 days, while the majority of mice given spleen cells from donors specifically immunized with tumour cells were free of metastases at 120 days. The fact that the protective effect of immune spleen cells was eliminated by pre-treatment of the cells with anti-Thy 1 serum indicated that the cells responsible for metastatic cell destruction are specifically activated T-lymphocytes.
