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PEANUT LECTIN RECEPTORS ON THE SURFACE OF EPITHELIAL CELLS OF CARCINOMAS AND DYSPLASTIC LESIONS OF HUMAN BREAST

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The binding of peanut lectin (PNL) at the apical membrane of surface epithelial cells in explants from carcinomas and dysplastic lesions of human breast, grown in organ culture, was studied cytochemically with horseradish peroxidase as a marker. Both in dysplastic and neoplastic lesions, irregular binding of PNL on the surface of cells was present. The dysplastic lesions showed more regular and intensive labelling in comparison with carcinomas. The surface of cells of carcinomas was either completely devoid of receptors for PNL or only solitary cells bound PNL. The binding of PNL in aldehyde fixed or unfixed explants was similar, with partial endocytosis of label in the latter.

MEC

4-EPIDOXORUBICIN IN THE THERAPY OF BREAST AND OVARIAN CANCER

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24 patients with advanced and recurrent breast cancer were treated with 4-epidoxorubicin at the respective dose of 60mg/m² every 28 days. Out of 22 patients evaluated, 2 achieved CR (9%) and 7 PR (31%). In 8 cases previously not treated with chemotherapy, 4/8 showed response. Side effects were leukopenia, alopecia, nausea and vomitus but no cardiotoxicity.

21 patients with advanced ovarian cancer were treated with the combination CEP (Cyclophosphamide, 4-epidoxorubicin, cisplatin). 6 patients achieved CR and 6 PR. Remission rate = 57%. Side effects included vomiting, myelosuppression and alopecia. No severe cardio- or nephrotoxicity was documented.

Our results seem to indicate that the therapeutic activity of 4-epidoxorubicin in mono- or combination therapy is similar to doxorubicin. CEP is an effective and relatively well tolerated regimen in the treatment of advanced ovarian cancer.

MEE

THE EFFECT OF INHIBITION OF N-HYDROXY-2-ACETYLAMINOFLUORENE (N-OH-AAF) SULPHATION ON THE INITIATION OF γ -GLUTAMYLTRANSEPTIDASE (γ -GT) FOCI IN RAT LIVERJ. H. N. Meerman
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The formation of the unstable N-O-sulphate ester of N-OH-AAF in rat liver is associated with its hepatocarcinogenicity. However, in some other organs devoid of sulphotransferase activity, tumours are also induced by N-OH-AAF. Therefore the exact role of sulphation of N-OH-AAF in carcinogenicity is as yet unknown. We have inhibited sulphation of N-OH-AAF with pentachlorophenol (PCP) during the initiation phase in the initiation-promotion model for liver tumour induction as developed by Solt and Farber (Cancer Res. 43, 188, 1983). Inhibition of sulphation of N-OH-AAF by PCP prevents the formation of covalently bound adducts of N-OH-AAF to proteins, RNA and DNA in the rat liver while formation of other adducts (not formed by sulphation) is not affected (Meerman *et al.*, Carcinogenesis 2, 413, 1981).

We now report that inhibition of sulphation by PCP does not prevent the initiation of hyperplastic, γ -GT positive foci (regarded as preneoplastic lesions) by N-OH-AAF in the rat liver. This suggests that other DNA-adducts, not formed by sulphation, are responsible for the initiation of these foci.
