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Dynamics of reactive oxygen intermediate production in human glioma: n-6 essential fatty acid effects.

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Abstract

BACKGROUND: Reactive oxygen intermediates (ROIs) are important signals controlling cell growth and cell death. Local essential fatty acid (EFA) deficiencies in tumour cells may limit tumour ROI generation. This deficiency may be rectified by the addition of exogenous EFA.

MATERIALS AND METHODS: The n-6 EFA effects on tumour ROIs were analysed in terms of kinetics, dose-response and individual cell type responses using flow cytometry of intracellular 2',7'-dichlorofluorescein oxidation. ROI formation in 30 gliomas and five paired samples of normal brain tissue, > 500 000 cells per specimen, was analysed every 10 s for 0-25 min.

RESULTS: Tumour cell basal ROI was lower than normal brain tissue ROI from the same subjects ($P < 0.00002$). Normal and tumour cell ROIs were stimulated by 4-40 micromol L⁻¹ n-6 EFAs, arachidonic acid (AA) and gamma-linolenic acid (GLA). The stimulated ROI rate was exponential, with the maximum dependent on EFA concentration and tumour grade.

CONCLUSIONS: EFAs stimulated tumour cells more than normal cells ($P < 0.0000017$, $n = 71$) and increased ROIs in glial fibrillary acidic protein-positive cells in tumours. This indicated high sensitivity of glioma cell ROIs to n-6 EFAs.

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