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Prostaglandins Leukot Essent Fatty Acids. 1998 Mar;58(3):193-200.

## Apoptosis in human primary brain tumours: actions of arachidonic acid.

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### Abstract

It has been postulated that loss of proliferative control in tumour cells is a consequence of depletion of cellular arachidonic acid (AA) and that exogenous AA and n-6 fatty acids may restore control of proliferation. To test this hypothesis and to investigate the activity of AA, apoptosis in human primary brain tumour cells was analysed using flow terminal deoxynucleotide transferase uridine nick end-labelling (TUNEL). The effect of exogenous AA (30 microM) was analysed in collagenase-dispersed tissue from seven human primary brain tumours and in the normal brain tissue surrounding one of the tumours. Exogenous AA stimulated apoptosis in tumour tissue. A rapid three-fold increase in endonuclease activity was detected in tumour cells incubated with AA. The increase in apoptosis was significantly greater than the contemporary (< 15%) increase in necrosis detected using propidium iodide permeability and was greater than AA effects on normal brain tissue. These results are consistent with activation of the pathways of apoptosis by AA.

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