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Prostaglandins in human brain tumors.

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Abstract

It has been recently observed that arachidonic acid (AA) metabolites may modulate many of the mechanisms involved in tumor growth and metastasis. In order to clarify the role played in human brain tumors, authors have determined AA metabolic profiles in 63 surgical specimen of human intracranial tumors (mostly neuroepithelial tumors and meningiomas). The five metabolites via the cyclooxygenase pathway (PGD₂, PGE₂, TxB₂, PGF_{2a}, 6-Keto-PGF_{1a}) were measured by high resolution gas chromatography-mass spectrometry after "ex vivo" metabolism of endogenous AA by tumor homogenates. The overall synthesis capacity of AA metabolites widely varied among different oncotypes, and, except in two cases of dermoid cysts, was higher than in normal brain tissue. AA metabolism seems more active in neuroepithelial tumors with the highest grade of anaplasia; some changes in the percentage of each metabolite is evident when anaplastic features changed. Thromboxane B₂ was the most represented and 6-Keto-PGF_{1a} the less abundant metabolite. Meningiomas and neuroepithelial tumors showed different relative proportion of AA metabolites which have in some cases reported to positively or negatively affect tumor growth. In histological subgroups of meningiomas AA metabolites synthesis capacity did not show any statistical difference. In the six cases of brain metastasis there is a wide range of overall synthesis capacity, with predominant synthesis of thromboxane B₂ and prostaglandin E₂, while the percentage of prostaglandin D₂, reported as antimetastatic, is very low.

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