AGATHISFLAVONE ISOLATED FROM C*AESALPINIA PYRAMIDALIS LEAVES* INDUCES GROWTH INHIBITION OF GLIOBLASTOMA CELLS AND STIMULATES THE FOSFORILATION OF P42 AND P44 (ERK1/2) MAP KINASES.

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Malignant gliomas are very invasive, rapidly proliferating tumours and present a poor prognosis. Potential differentiation and/or apoptosis inducing factors can offer an alternative for the traditional chemotherapic treatments. Because cell growth and differentiation often involve p44/p42 mitogen-activated protein kinase (MAPK) pathway signaling, we explored MAPK signaling and growth response in the human high proliferative glioblastoma cell line GL-15 after treatment with the aghatisflavone (named AFCPB) extracted from C*aesalpinia pyramidalis* leaves. Cells (100,000 cells/plate) were grown in supplemented DMEM medium and treated with AFCPB (0.1-100μM) for 24-72h. Negative controls were treated with 0.5% DMSO. We observed, by ^{3}H-thymidin incorporation, that AFCPB at the higher concentration (100μM), revealed a efficient growth inhibition, that reached 78-83% after 72h exposure. Moreover, westernimmunoblot revealed that two main proteins, with an apparent molecular weight of 44 and 42kDa, were phosphorylated after 24h treatmnet with AFCPB. The increase in tyrosine phosphorylation was apparent following AFCPB at 1μM and the response was dose dependent up to 100μM. These results indicates that AFCPB induces growth inhibition of glioblastoma cells that may be related to fosforilation of p42/p44 (ERK1/2) MAP kinases. Supported by FAPESB and CNPq.