Dimethylfumarate

Dimethylfumarate (DMF), a drug used in the treatment of psoriasis and multiple sclerosis, has been shown to limit the growth of melanoma cells. The ability of DMF to inhibit the Rel protein has been used to explain the antioncogenic properties of this drug.

DMF appears to have a promising role in the treatment of malignant brain neoplasms. DMF reduced proliferation rate, generated cell lysis, decreased the expression of NF-κB, and restricted the growth of CD133 cells in gliomas. This suggests that DMF may be considered for further antitumor studies, and provide a new treatment modality for brain tumors [1)].

Previous reports have shown that dimethylfumarate (DMF) can activate the Kelch-like ECH-associated protein 1-nuclear factor erythroid 2-related factor 2-antioxidant responsive element (Keap1-Nrf2-ARE) system in vivo and in vitro, which leads to the downregulation of oxidative stress and inflammation.

DMF administration resulted in abatement of the development of early brain injury and cognitive dysfunction in this prechiasmatic cistern SAH model. This result was probably mediated by the effect of DMF on the Keap1-Nrf2-ARE system [2)].

1)
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2)
Liu Y, Qiu J, Wang Z, You W, Wu L, Ji C, Chen G. Dimethylfumarate alleviates early brain injury and secondary cognitive deficits after experimental subarachnoid hemorrhage via activation of Keap1-Nrf2-ARE system. J Neurosurg. 2015 Jan 23:1-9. [Epub ahead of print] PubMed PMID: 25614941.