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1: [Mol Oncol](#). 2009 Feb;3(1):77-83. Epub 2008 Nov 5.



Ebselen abrogates TNFalpha induced pro-inflammatory response in glioblastoma.

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We investigated the pro-inflammatory response mediated by TNFalpha in glioblastoma and whether treatment with organoselenium Ebselen (2-phenyl-1,2-benziselenazol-3[2H]one) can affect TNFalpha induced inflammatory response. Exposure to TNFalpha increased the expression of pro-inflammatory mediator interleukin IL-6, IL-8, monocyte chemoattractant protein-1 (MCP-1) and cyclooxygenase (COX-2). Treatment with Ebselen abrogated TNFalpha induced increase in pro-inflammatory mediators. Ebselen not only abrogated TNFalpha induced enhanced invasiveness of glioma cells by down-regulating matrix metallo proteinase (MMP-9) and urokinase plasminogen (uPa) activity, but also inhibited glioma cell migration. Treatment with Ebselen also down-regulated the enhanced ROS production of TNFalpha treated glioma cells. In addition, Ebselen induced DNA damage repair signaling response in glioma cells both in the presence and absence of TNFalpha. These studies indicate that together with its known ability to sensitize glioma cell to TNFalpha induced apoptosis, Ebselen can overcome TNFalpha induced pro-inflammatory mediators to prevent a build up of a deleterious pro-inflammatory tumor microenvironment.

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