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Subject: Schädlichkeit von Alkohol in Topicals

Posted by [glockenspiel](#) on Thu, 14 Sep 2006 10:40:29 GMT

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Darüber wird ja wild gestritten, wie ist denn diese Studie zu sehen? Gilt das auch für topische Anwendungen?

[http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&mp;db=pubmed&dopt=Abstract&list\\_uids=15605983](http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&mp;db=pubmed&dopt=Abstract&list_uids=15605983)

Chronic ethanol treatment enhances inflammatory mediators and cell death in the brain and in astrocytes.

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Inflammatory processes and cytokine expression have been implicated in the pathogenesis of several neurodegenerative disorders. Chronic ethanol intake induces brain damage, although the mechanisms involved in this effect are not well understood. We tested the hypothesis that activation of glial cells by ethanol would induce stimulation of signaling pathways and inflammatory mediators in brain, and would cause neurotoxicity. We used cerebral cortex from control and chronic ethanol-fed rats, which received ethanol-liquid diet for 5 months and cultured of astrocytes exposed to 75 mM ethanol for 7 days. Our results demonstrate that chronic ethanol treatment up-regulates iNOS, COX-2 and IL-1beta in rat cerebral cortex and in cultured astrocytes. Under both experimental conditions, up-regulation of these inflammatory mediators and IL-1RI concomitantly occurs with the stimulation of IRAK and MAP kinases, including ERK1/2, p-38 and JNK, which trigger the downstream activation of oxidant-sensitive transcription factors NF-KB and AP-1. These effects were associated with an increased in both caspase-3 and apoptosis in ethanol-fed rats and in astrocytes exposed to ethanol. In conclusion, chronic ethanol treatment stimulates glial cells, up-regulating the production and the expression of inflammatory mediators in the brain, and activating signalling pathways and transcription factors involved in inflammatory damage and cell death.

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