Headache. 2006 Sep;46(8):1291-5. doi: 10.1111/j.1526-4610.2006.00538.x.

Repression of stimulated calcitonin gene-related peptide secretion by topiramate

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PMID: 16942474 DOI: 10.1111/j.1526-4610.2006.00538.x

Abstract

Objective: The goal of the proposed research was to determine the effect of topiramate on basal and stimulated release of calcitonin gene-related peptide (CGRP) from trigeminal ganglia neurons.

Background: CGRP is implicated in migraine headaches. Clinical evidence supports topiramate as an effective migraine prophylactic. In this study, the connection between topiramate and CGRP expression was investigated.

Methods: Primary cultures of rat trigeminal ganglia were utilized to determine the effects of topiramate on CGRP release stimulated by a depolarizing stimulus (KCl), nitric oxide, and/or

protons. The amount of CGRP secreted into the culture media was determined using a CGRP-specific radioimmunoassay.

Results: Treatment of trigeminal cultures with KCl, nitric oxide donor S-nitroso-N-acetylpenicillamine, or protons (pH 5.5 media) caused a marked increase (3 to 5 fold) in the amount of CGRP release. Topiramate treatment repressed KCl-stimulated CGRP release in a time- and concentration-dependent manner. However, topiramate did not alter the amount of unstimulated or basal CGRP released from trigeminal neurons. In addition, topiramate inhibited nitric oxide and proton mediated CGRP secretion.

Conclusions: Findings from these studies demonstrate that topiramate can directly repress the stimulated release of CGRP from sensory trigeminal neurons. We propose that topiramate's ability to prevent migraine attacks may involve inhibition of CGRP secretion from trigeminal neurons.

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