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NEW THEORIES IN MIGRAINE PATHOPHYSIOLOGY

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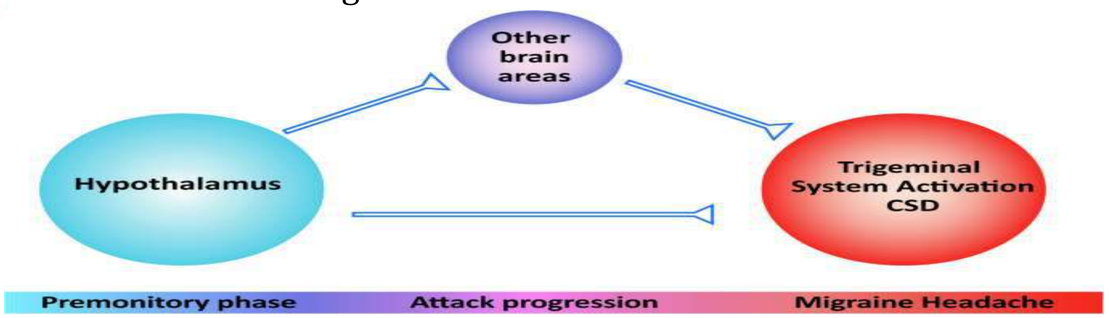
Introduction

Migraine is the third most prevalent disease in the world and affects 12% of the general population. It has recently been suggested that central neurochemical imbalance and low 5-HT levels facilitate the activation of the trigeminovascular nociceptive pathway, which therefore initiates migraine.



Keywords

Migraine, serotonin, trigeminovascular system, CGRP

Purpose

The aim of the study was to describe pathogenetic mechanisms of migraine according to the newest theories and scientific discoveries.

Material and methods

It was performed a systematic review on scientific papers concerning the role of serotonin, CGRP and cortical spreading depression in migraine development. After searching the PubMed, Hinari and Cochrane Library databases, a total of 247 papers were screened for relevance, but only 36 papers were selected for further analysis.

Results

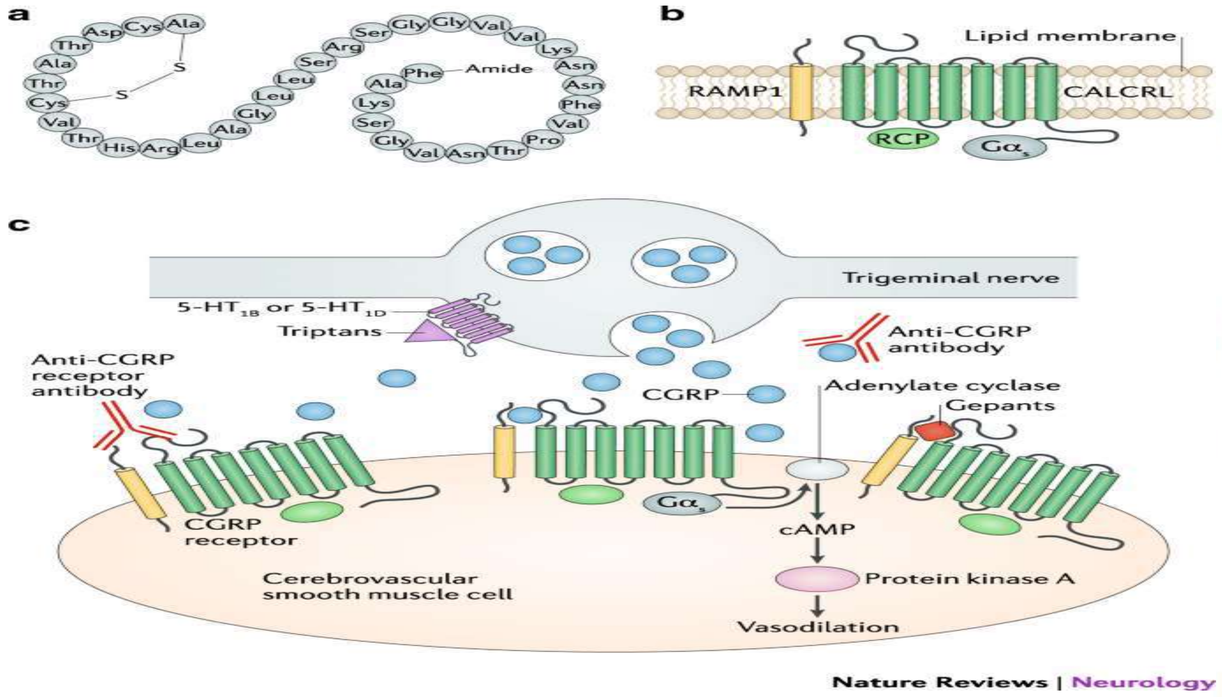


Figure 1. CGRP pathway (figure from *The Journal of Headache and Pain*)

It has been observed an increase in the amplitude of neuronal evoked potentials following the activation of inhibitory prejunctional 5-HT1B/1D autoreceptors and 5-HT decreased synthesis. The cortical spreading depression stimulated the trigeminovascular fibers and determined the release of CGRP, vasodilation and increased plasma protein extravasation.

Conclusions

Migraine depends on: a) activation of the trigeminovascular system with increased nociception, and b) dysfunction of CNS structures involved in the modulation of neuronal excitability and pain.