



WEB OF SCIENCE



PATHOPHYSIOLOGY AND THE GENETIC BASIS OF PAIN



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Abstract

Introduction: International Association for Pain Study (IAPS) proposed the evaluation of pain level as the fifth vital sign to be followed throughout the hospitalization of a patient, beside temperature, breathing, heart rate and blood pressure. Pain is a warning signal to an endogenous or exogenous noxious stimulus and it represents an essential mechanism for survival. When the intensity or duration of pain exceed the expected limits (depending on the etiology of the underlying disease), pain becomes a harmful factor to physical and mental health of the individual.

Material and method:

It can be observed four components on painful phenomena: sensitive-sensory, mental motivational-emotional, psychic cognitive and somatic-vegetative.

Manifestations observed during pain can be classified into four classes with Fordyce model: nociception, pain, suffering and pain behavior.

Pain receptors (nociceptors) are divided into single-modal receptors (specific) activated by mechanical stimulation and multimodal receptors (non-specific) activated by the mechanical, thermal, chemical and biological stimulation.

In "Pain: past, present and future" - by Mogil J.S., published in June, 2012 – it is shown that the chronic pain is an example of interaction between gene and environment.

The COMT gene codifies the catecol-O-metil transferase. The GCH₁ gene controls the synthesis of an enzyme called GTP (cyclohyddrolase 1) that it is involved in dopamine and serotonin production.

The OPRM₁ gene codified the human opioid receptors.

Results and conclusions:

Future development of therapies that increase the pain threshold by acting on the gene COMT and therapies for hereditary increased sensitivity and motility disorder by acting on the gene GCH₁ could be a way for personalized treatment of pain.

Key words: gene, COMT, OPRM₁, GCH₁