Short Communication

Indian Journal of Biology
Volume 1 Number 1, January - June 2014

Revisiting the Role of Biology in Pain: Living or Leaving?

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Abstract

The objective of this short communication paper was to reappraise the role of biology in pain, its reception, perception, processing and reaction from an evidence-informed overview of studies found in PubMed. There were studies relating Biology of pain with anatomy, physiology, pathophysiology and molecular biology. Ethnicity was representing inter-individual variability in pain threshold and pain tolerance as a marker of biology. Biology-based assessment and treatment of pain was reported in studies on bone cancer pain, mucosal pain, fibromyalgia and chronic low back pain. There is need to explore the biologically based mechanisms in future studies of pain sciences.

Keywords: Pain biology; Pain sciences; Pain mechanisms; Therapeutic biology; Clinical biology.

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Anatomy

Structurally, central nervous system (CNS) glial cells play a role in neuronal functioning including pain processing and they are capable of recruitment at sites of CNS damage, and also to "act at a distance." When the nervous system initiates signals that alter the function of these glial cells, these cells in turn release factors that regulate neuronal function.[1]

Physiology

Functionally, upregulation of certain genes such as IEG, c-fos, Fos B, FRA-1, FRA-2, Jun B, Jun D, NGFI-A associated with slower kinetics was demonstrated in studies on molecular biology of pain.[2]

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Pathophysiology

Pathophysiologically, systems biology approaches utilizing high throughput technologies (genomics, transciptomics, lipidomics, and proteomics) are applied to clinical as well as experimental pain models to discover novel pain mediators. Some of the approaches include co-expression network generating algorithms, which infer potential associations/interactions between molecules and build networks based on these interactions. One such example is the protein-protein interaction networks that allow the lists of potential targets generated by these different platforms to be analyzed in their

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biological context.[3]

Molecular Biology

Clinically, Gene-based neuromodulation (cell type-specific targeting, expression control, and gene transfer) can be achieved through neuronal delivery of transgenes capable of altering synaptic function, which can be applied to control of pain and address the root causes of pain.[4]

Ethnicity

Rahim-Williams *et al* examined the ethnic group differences and their contributing factors in experimental pain response by conducting a systematic literature review of 26 studies using experimental pain stimuli to assess pain sensitivity across multiple ethnic groups.The majority of studies included comparisons between two racial groups: African Americans (AA) and non-Hispanic Whites (NHW), with AA demonstrating lower pain tolerance and pain thresholds. Those potentially important ethnic/racial group differences in experimental pain perception suggested the role of biology in pain and its perception.[5]

Biology-based Assessment and Treatment

Bone Cancer Pain

Bone cancer pain was explained by Goblirsch *et al* as follows; "mechanisms that drive bone cancer pain include tumor-directed osteoclast-mediated osteolysis, tumor cells themselves, tumor-induced nerve injury, stimulation of transient receptor potential vanilloid type 1 ion channel, endothelin A, and host cell production of nerve growth factor; current and future therapies include external beam radiation, osteoclast-targeted inhibiting agents, anti-inflammatory drugs, transient receptor potential vanilloid type 1 antagonists, and antibody therapies that target nerve growth factor or tumor angiogenesis."[6]

Mucosal Pain

Miaskowski *et al*[7] explained the biology of mucosal pain from anatomical evidence of oral mucosa containing primary afferent nociceptors that respond to thermal, mechanical, and chemical stimuli; and pathological evidence of occurrence of inflammation during the initial phase of mucosal injury caused by stomatotoxic chemotherapy or radiation therapy.

Fibromyalgia

Staud described the mechanisms of fibromyalgia (FM) from a biological perspective: on the role of central nervous system pain processing abnormalities in FM, including central sensitization and inadequate pain inhibition; the role of peripheral tissues as relevant contributors of painful impulse input that might either initiate or maintain central sensitization; and the contribution from persistent or intense nociception which can lead to neuroplastic changes in the spinal cord and brain, resulting in central sensitization and pain.[8]

Chronic Low Back Pain

Although exercises are routinely prescribed for people with chronic low back pain, Ryan *et al* found that addition of pain biology education as an adjunctive intervention was better in producing immediate changes in pain and self-reported function assessed using visual analogue scale and Roland Morris Disability Questionnaire respectively, with no

between-group differences with education-alone group at 3-months follow-up.[9]

There were studies relating Biology of pain with anatomy, physiology, pathophysiology and molecular biology. Ethnicity was representing inter-individual variability in pain threshold and pain tolerance as a marker of biology. Biology-based assessment and treatment of pain was reported in studies on bone cancer pain, mucosal pain, fibromyalgia and chronic low back pain. There is need to explore the biologically based mechanisms in future studies of pain sciences.

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