Biopsychosocial approach to pain

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Abstract

The Author deals with the issue of subjective pain and correlates it with several factors, investigated by the biopsychosocial model according to various perspectives that interact with each other.

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In 1973 IASP, the highest association on pain studies, defined pain as an experience made of sensory and emotional dimensions. Therefore the biopsychosocial model is prevailing in the management of both acute and chronic pain. But yet in some cases only a biomedical approach which hinders the treatment and understanding of this human condition, can prevail. Actually psychologists and therapists who are not purely pain specialists, can hardly measure pain education, perceived competence and comfort of the therapist himself. As a result,basic essential knowledges on pain need to be integrated in Psychology University Courses. The experience of pain is influenced by biology, beliefs, cultures, mood, anxiety and environment. The biopsychosocial approach that deals with the multiple dimensions of chronic pain is the one that appears most promising to us. Monotherapy in relieving pain will soon be abandoned. As a matter of fact, a number of algologists show interest in Mindfulness. Awareness-based interventions for chronic pain are leaded by the principle that the practice of awareness brings an attenuation of the coupling between the sensory component of pain and the cognitive and emotional components of pain. In line with this principle, recent research demonstrates the neural mechanisms that support mindfulness-based pain reduction. It affects areas of the brain related to attention, introspection and cognitive and emotional processing. These components can amplify pain and contribute to disability. Diminishing the cognitive and emotional reactions of chronic pain through awareness is believed to reduce emotional distress and therefore suffering and disability. Discriminating the various dimensions of pain, the sensory component is measurable in time, space and intensity while the affective - emotional component requires different and complex parameters and tools. Cognitive assessments and affective aspects of pain are involved in the discriminative and motivational systems of cortical relevance. The affective part of pain, which is always present, is undoubtedly the complex articulated one, including both the cognitive and the emotional and motivational aspects. These factors have a surprisingly important influence on the perception of pain and these relationships reside in the connectivity of the brain regions that control the perception of pain, attention or expectation and emotional states.

The experience of pain

Functional MRI confirmed that the activity of the afferent and descending pain pathways is altered by the state of attention, positive and negative emotions, among many other factors not related to the nociceptive stimulus. The physiology of central pain amplification in the brain takes into account these important connections. Patients with chronic pain have alterations in brain regions involved in the cognitive and emotional modulation of pain. These regions are the insula, the anterior cingulate cortex and the prefrontal cortex. This complex inter-reaction may explain why those with cognitive distortion and psychological distress are at greater risk for chronic pain and central pain amplification. Between 30 and 60% of patients with chronic pain have comorbid depression. There is a two-way relationship between the presence and severity of pain and depression. In patients with chronic pain, the change in pain is always a predictor of the severity of the depression and vice versa. Furthermore, a biopsychosocial context is useful for conceptualizing individual differences in pain, the results relating to demographic factors associated with individual differences in pain. By definition pain is a subjective and highly personal experience, so much so that direct measurement of pain is impossible, rather we have to rely on individuals' self-reporting and, to some extent, their behavior, to provide a glimpse into the their experience. The experience of pain is carved by a mosaic of unique factors of the person, which makes the experience of pain completely individualized. That is, there are important and pervasive individual differences in pain, and these individual differences produce pain experiences that are completely unique to the person experiencing them (i.e. make the pain personal). Individual differences in pain are independent of the initial stimulus. Perhaps the simplest manifestation of individual differences is that an experimental stimulus delivered at a standardized intensity elicits reports of subjective pain that vary considerably between individuals. Interestingly, these differences in self-reported pain are corroborated by inter-individual differences in brain activation evoked by the same pain stimulus and are partly predicted by individual differences in brain morphology, suggesting that these individual differences are not simply a product of idiosyncrasies in pain reporting. These individual differences also emerge in the clinical setting. For example, pain reports following the same surgical procedure vary greatly between patients considering relative outcomes. Only the biopsychosocial model provides an ideal framework for conceptualizing individual pain differences. This model hypothesizes that the experience of pain is influenced by complex and dynamic interactions between multiple biological, psychological and social factors. It is important to emphasize that the set of biopsychosocial factors that contribute to the experience of pain and its expression vary considerably among people. Therefore, pain is sculpted from a mosaic of factors that is completely unique to each individual at any given time, and this mosaic must be considered to provide optimal pain treatment.

Pain marker

When considering individual difference factors, it is important to distinguish the characteristics of the individual that are statistically associated with pain responses (i.e. markers) from biological and psychosocial mechanisms that directly influence pain responses. In particular, some markers may reflect the underlying mechanisms of pain, while others do not. Examples of the former include demographic factors, such as gender, race / ethnicity, and age. Although each of these variables has been associated with pain responses, they reflect agents on the mechanisms that affect pain rather than the mechanisms themselves. That is, an individual's sex does not directly affect pain, rather the sex differences in pain reflect the effects of other biological and psychosocial processes (e.g., sex hormones, inflammatory responses, gender roles, pain management). Alternatively, a study could evaluate pain-related biological markers, in which case the biological marker represents both an individual difference factor and a potential mechanism that directly affects pain. Therefore, although individual differences in pain response present challenges for the scientist and physician, they also provide important opportunities. Indeed, studying the factors contributing to individual differences can provide important insights into pain mechanisms, which can lead to the development of new treatments. In addition, incorporating an understanding of individual differences in clinical pain assessment and diagnosis can allow the physician to select patient-tailored treatments, thereby improving treatment outcomes.

Considering the sex differences, the abundant epidemiological evidence shows that chronic pain is more common among women than men. Women are at greater risk for the most common chronic pain conditions, including migraine and tension-type headache, low back pain, fibromyalgia and widespread pain, temporomandibular disorders, irritable bowel syndrome and osteoarthritis. If sex differences are examined in the severity of acute and chronic pain, inconsistent and small data emerge. For all standard measures of experimental pain sensitivity, women exhibit greater sensitivity than men, including pain threshold (the minimum intensity of the stimulus required to produce pain), pain tolerance (the maximum intensity of the stimulus that an individual is willing to tolerate) and suprathreshold stimulus assessments. In particular, the extent of the sex difference varies considerably between studies and between pain measures and stimulus modalities, but the direction of the difference is highly consistent. Furthermore, the women showed a greater temporal sum of pain (a measure of transient central sensitization) and less conditioned pain modulation (a measure of endogenous pain inhibition), suggesting a modulatory balance of pain that is more strongly tuned towards pain facilitation versus pain inhibition among women. Conversely, in response to prolonged and repeated thermal stimuli, females exhibited greater habituation than males, suggesting a stronger inhibitory pain response to these types of stimuli. Multiple mechanisms have been proposed to explain these sex differences in pain, including the effects of sex hormones,

differences in endogenous opioid function, cognitive / affective influences. Given the aging of the world population, if the experience of pain changes with age it has attracted increasing attention in recent years. The patterns of pain prevalence throughout life are complex and vary according to the pain condition. In short, the prevalence of joint pain, pain in the lower limbs and neuropathic pain tends to increase with age. General chronic pain increases in prevalence until middle age, by which time the prevalence rises. In contrast, pain conditions such as headache, abdominal pain, back pain and temporomandibular disorders show a peak of prevalence in the third to fifth decade of life, after which their frequency decreases. It is important to note that these epidemiological results are almost exclusively based on cross-sectional studies that could influence the results. In addition to the prevalence of pain, several studies have examined age-related changes in the severity and impact of pain. Older people reported less acute pain intensity in some studies but not others. Similarly, age-related differences in the intensity and impact of chronic pain have not been consistently demonstrated.
Age-related changes in responses to experimental pain have been extensively studied. Taken together, these results suggest that older adults show less sensitivity to brief skin pains (eg heat pain threshold); however, sensitivity to more sustained painful stimuli that impact deeper tissues increases with age.

It has been hypothesized that a variety of biopsychosocial factors contribute to these age-related changes in pain processing. First, many pain-related diseases increase in frequency with age (eg diabetes, osteoarthritis, many forms of cancer, neurological diseases), which can contribute to an increase in pain among the elderly. Additionally, many of the biological changes that underlie aging may also contribute to increased clinical pain and altered pain modulatory balance, including systemic inflammation, oxidative stress, impaired automatic function, and changes in neuronal structure and function. . Furthermore, the psychosocial changes that occur with age could also have an impact on pain. Reductions in cognitive function, sleep quality, and social support are all common in the elderly, and these factors are also associated with increased pain. In particular, insufficient pain treatment in the elderly is common, which could further contribute to increased pain in this population.
The biopsychosocial model does not simply propose that factors from the biological, psychological and social domains exert important influences on pain. Perhaps the most important aspect of the model is its insistence that these different groups of factors interact to create the experience of pain. Although often overlooked in pain research, identifying and ultimately understanding these interactions is critical to elucidating the mechanisms that drive pain in different groups and individuals. The influence of psychological stress on pain could be mediated by specific biological processes, such as increased outflow of the sympathetic nervous system or increased inflammation. Another common type of interaction is an additive association, in which the combination of two individual

difference factors, each of which increases the risk of pain, produces a stronger effect than both factors alone. For example, if both the female gender and a particular genetic profile increase the risk of chronic pain, then the combination of being women and having a particular genetic profile would produce a greater risk than having one or the other. Sometimes the effect of a factor depends on the presence or absence of another factor. In this case, we may find that while both the female sex and the genetic profile are risk factors for pain, the association between the gene and pain is different for females and males. The genetic factor may increase the risk of pain in females but decrease the risk in males.

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