CRE-MSD Centre of Research Expertise for the Prevention of Musculoskeletal Disorders

Chronic Pain is a Different Kind of Injury

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Practitioners looking to improve workplace health and safety often try to prevent worker injuries, but worker behaviours are more motivated by the pain they experience on or off the job. While injuries can be mistakenly synonymous with pain, the two are distinct and independent.[1] The purpose of this position paper is to discuss how pain develops and identify some implications for workers experiencing chronic pain.

Key Messages

- Pain is processed in the brain, which is distinct from the injuries that people often associate with pain.
- Acute pain can develop into chronic pain even after a person recovers from an injury.
- Injury prevention can help prevent acute pain but cannot "fix" chronic pain.

Acute & Chronic Injuries/Pain

An injury is considered *acute* when a brief exposure is greater than tissues' injury tolerance.[2,3] Acute injuries are often traumatic and clearly linked to a specific exposure such as a sudden impact, lifting something heavy, or another clearly defined event. In contrast, an injury is considered *chronic* when a longer duration exposure reduces a tissue's injury tolerance to the point where a normally harmless event damages a compromised tissue.[2–5] Repetitive strain injuries,[6] overuse injuries,[7] fatigue fractures,[8] and other common workplace musculoskeletal disorders are usually categorized as chronic injuries. There are current actionable workplace guidelines, which consider these chronic injury pathways.[9–11]

It is important that despite acute and chronic injuries having different mechanisms, the pain from an acute injury feels similar to the pain from a chronic injury.[12,13] Injuries that hurt cause *acute pain* first, regardless of whether they were acute or chronic injuries. Acute pain is useful in that the experience of pain is linked to either quantifiable/measurable damage, or an exposure that could have caused this damage.[1,14,15] "Quantifiable damage" can be something that is visually apparent in the case of a disc herniation[16] or an inflamed tendon;[17] functionally apparent such as a rotator cuff tear[6] or osteoarthritis[18] causing reduced joint motion; or mechanically apparent in the case of a bony stress fracture.[19]

Acute pain may fluctuate over time but will usually dissipate as the injury heals;[15] however, there are several mechanisms that can cause the experience of pain to persist after a person recovers from the injury, resulting in the acute pain developing into chronic pain. *Chronic pain* is defined as pain lasting at least three to six months depending on the source.[20–23] Chronic pain is pain that is no longer associated with an injury or quantifiable damage: the disorder is the experience of pain rather than the physical damage to the tissues in the body where that pain seems to come from.[15,24,25] This distinction is important since modifying a workplace exposure to reduce tissue loads, for example, can prevent acute and chronic injuries, and therefore the development of acute pain, but it may not remove existing chronic pain or the change from acute to chronic pain. Additionally, acute and chronic pain may not feel distinct from one another,[12,26] sometimes acute and chronic injuries cannot be distinguished based on pain quality or intensity. This common use of the terms "acute" and "chronic" combined with workers associating pain with an injury (because injuries often hurt)[27] makes it difficult to separate the development of an injury from the development of pain. It is more useful to describe pain by its source instead of its duration: "acute" pain is described as "nociceptive" pain, and "chronic" pain is described as "neuropathic" pain.

Sensing Acute or Nociceptive Pain

Nociception is the term that describes how the nerves outside the brain alert us to potentially dangerous exposures.[28–30] Nociceptive neurons connect directly to the brain but are modified through spinal cord circuits that collect information from a variety of sources including other nearby tissues,[31] previous information from that tissue,[32] inflammation,[33] mood,[34] physical activity,[35] and memory[36]. The balance of this information comes from both inside and outside the brain and will increase or decrease the strength of the neurological signal before it reaches the brain. The brain then interprets the information it receives as pain or not-pain depending on the intensity of the incoming signal and the context that surrounds it.[28,30,37–39] In other words, the pain you experience is a brain-centric response that is different from both the potentially-dangerous stimulus that you have been exposed to and the neurological encoding of that potentially-dangerous stimulus (nociception). In acute pain, the brain's interpretation is based on the level of physical stimulus and interpreted in the context of additional information. An inflamed ankle is supposed to hurt more than an uninflamed ankle, just as stubbing your toe while upset should hurt more than stubbing that same toe while feeling positive. These complex nociceptive processes are important as they function to warn us to remove a dangerous or potentially dangerous exposure in order to prevent tissue damage or limit the severity of existing damage while incorporating the situation and other sensory information.[15]

However, workplace exposures that are linked to injuries can modify the sensation of acute pain in unexpected ways. One major factor is *exercise-induced hypoalgesia*.[35,40,41] This process is initiated by chemical messengers that are released with the development of muscle fatigue.[42] This has been demonstrated to occur after repetitive upper limb work[43] and trunk flexion,[44] which are exposures common in manual materials handling, construction, and healthcare work. In isolation, exercise-induced hypoalgesia is immediate and localized,[43,45] meaning that its effects fade quickly after the exposure ends and only effects tissues and structures close to the active muscles. However, repetitive trunk flexion introduces a gradual stretching of tissues in the lower back called "creep" that can delay the desensitization for up to ten minutes and extend its duration.[44] Injuries that occur during this period of desensitization, either during the exercise or shortly after, might not feel painful until the effects of exercise-induced hypoalgesia wear off, as is observed in recreational and professional athletes.[46,47] While exercise-induced hypoalgesia is beneficial in the short-term to ensure high-exertion (and often dangerous) tasks are not catastrophically interrupted by pain, it can be a potential long-term issue as it can result more severe injuries.[46,47]

Constructing Chronic or Neuropathic Pain

Although our understanding remains incomplete, there are several cortical regions of the brain that appear to activate when healthy individuals experience pain. These include structures responsible for sensory processing,[48] executive functioning (i.e., mental processes that enable us to plan, focus attention, remember, and juggle multiple tasks),[49] emotion,[29] and memory.[36] Chronic or neuropathic pain may involve additional active structures,[48] altered connectivity between active structures,[25,50,51] physical changes to primary structures based on gray matter volume,[25,51,52] or a combination of all three. The cortical changes in individuals experiencing chronic or neuropathic pain can alter their perception of pain by skewing its severity or perceived location in the body. In addition to changes in the brain, nociceptive neurons in the spinal cord also show evidence of increased receptor production and more interconnected circuits in people living with chronic pain,[53] both of which can amplify existing nociceptive signals,[54] or cause non-nociceptive neurons (those activated by light touch) to activate nociceptive pathways.[55] This means that some individuals with chronic pain can experience pain without any incoming nociceptive signal because of the rewiring of and physical changes to the neurons in their brain and spinal cord.[25,37,38,48] These changes are called *neuroplastic* because the layout and connectivity of neurons has been altered and are difficult to reverse. This transition from nociceptive (acute) to neuropathic (chronic) pain can be initiated through the physical features of an exposure[22,24] or other non-exposure factors,[56] and can be continually reinforced in the absence of any risk factors once the neuroplastic changes have occurred.[57] All hope is not lost, for there is evidence that these neuroplastic changes can be reversed over time,[51,52,58] and many people who sustain injuries never develop chronic or neuropathic pain.[22,59–61] However, there will be cases where a worker's symptoms do not align with their physical exposures, and while the problem may not lie with a musculoskeletal injury, the physical alterations to their nervous systems can be considered a different kind of "injury".

Conclusion

Injuries are relatively easy to measure and have an established connection to workplace exposures. As such, they can often be effectively prevented and managed through the hierarchy of controls, workplace practices, work design considerations, or interventions. Pain is at least one step removed from an injury and can be influenced by factors both related and unrelated to the exposures it is often connected to through injury. While injury prevention is an effective way to manage pain development, the time course of injury and pain recovery are different, and workers who successfully recover from an injury may still experience pain in the absence of a clear exposure because of the neuroplastic changes associated with neuropathic pain.

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Position papers are funded by the Centre of Research Expertise for the Prevention of Musculoskeletal Disorders, which receives funding through a grant provided by the Ontario Ministry of Labour, Immigration, Training and Skills Development. The views expressed are those of the authors and do not necessarily reflect those of the Centre nor of the Province.

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