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# When Thinking About Pain Contributes to Suffering: The Example of Pain Catastrophizing

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## INTRODUCTION

Pain is a complex, multi-faceted experience[91] that is shaped by a lifetime of learning processes and influenced by sensory, affective, cognitive, and sociocultural factors. A robust empirical literature documents the potent role negative thoughts about pain have on the experience of pain and pain-related suffering. Much of this literature has investigated pain catastrophizing (pCAT), documenting the impact of pCAT in both laboratory and clinical settings, across many different pain conditions including acute and chronic pain, and both cross-sectionally and longitudinally throughout the lifespan. While the name and conceptualization of pCAT are under debate[13; 17; 106] and definitions vary widely,[59; 105] we will use the phrase pCAT here, as this is the descriptor and name most often applied and measured in the existing literature. pCAT is subject to many of the same influences as pain; it also represents the endpoint of complex, lifelong learning processes that contribute to an appraisal of pain as threatening, overwhelming, and unmanageable. A recent qualitative and machine learning analysis of the available literature on pain-related suffering found that pCAT is one of eight multidimensional constructs comprising the current conceptual framework of pain-related suffering.[84]

As data accumulate and debates advance, the issues raised often circle around whether pCAT is an appraisal of the threatening nature of pain and one's ability to cope with pain, [47; 89; 99] or a pain coping strategy intended to regulate distress by engaging the social environment[104] or through maladaptive problem-solving strategies.[27] Most recently, pCAT has been proposed to be a repetitive negative thinking process with intertwined cognitive, affective, and behavioral features that may modulate distress triggered by pain. [37] Crombez[16] recommends using the alternative term "pain worry," as pCAT items frequently were classified in the categories of pain-related worry and pain-related distress.

Cognitive-affective responses to pain are common to these definitions of catastrophizing, and debate continues as to whether behavior should be included in its conceptualization.[99; 104]

This paper will address how negative thoughts about pain, in the form of pCAT, develop using a lifespan/developmental perspective and why these thoughts are important in understanding people's experience of pain, particularly chronic pain and suffering. We propose that pCAT is a mindset that evolves throughout development and may activate key neurobiological processes involved in amplifying pain-related signals. The evidence indicates this mindset about pain is learned in childhood and contributes to heightened pain and risk for chronic pain as children develop. We will integrate findings from the stress and pain literature with the extensive pCAT literature. The accumulated evidence suggests that learning during infancy and childhood interacts with the individual's genetic susceptibility to develop pCAT. Both pain and stress exposures during childhood shape neurobiological pain regulatory systems, and children who have learned pCAT show greater sensitivity to pain. For these individuals, we propose that repeated exposures to pain over time that intermittently activate pCAT likely contribute to further amplification of pain through changes in the neurobiological pain regulatory systems. This positive feedback loop contributes to pain sensitivity with each pain exposure (pain -> pCAT -> ↑pain -> ↑ pCAT.....). At some later time, often precipitated by a minor or major injury, or surgery, and possibly influenced by allostatic load, pCAT contributes to episodic or acute pain becoming persistent, potentially spreading across anatomic areas, and exacerbating pain-related suffering. This trajectory is not inevitable, as the impact of pCAT on pain and pain-related suffering can be enhanced or diminished by multiple factors. And finally, we will discuss directions for research to address key questions raised by this conceptualization of pCAT.

## What is known about the origins/development of pCAT (Nature vs. Nurture)?

The consistent individual differences seen in the experience of pain have sparked substantial research into genetic risk for common pain conditions,[125] and chronic overlapping pain conditions.[95] Many fewer investigations have examined the genetics of pain catastrophizing and the small number of available twin studies estimate heritability to be 36–37%,[6; 111] suggesting that more than 60% of the variability in pCAT is due to environmental influences. Phenotypically related factors show strong associations between pCAT and both anxiety sensitivity and fear of pain and a low association with neuroticism.[6] Specific genetic polymorphisms[52] are associated with pCAT or interact with pCAT,[100] suggesting that this vulnerability may overlap robustly with vulnerability to negative affect[6] yet the overlap with genetic vulnerability to pain sensitivity is more complex.[31; 124]

Environmental influences on the development of pain are largely attributable to learning across the lifespan, beginning at birth through direct experience and interactions with the social environment, primarily caregivers.[42] Newborns exhibited anticipatory elevations in heart rate[43] and distress[107] during preparation for venipuncture after experiencing multiple heel sticks, and during the first year of life infants learn from the caregiver's

response to their distress when receiving vaccinations.[88] Caregiver emotional availability, or the caregiver's ability to interpret and respond to the infant's cues, developed and stabilized over the first year [92], demonstrating that the first year is a critical time for both caregivers' and infants' learning about pain and coping with pain.

Just as pain is learned and shaped throughout childhood,[14] pCAT is shaped throughout childhood by parents, information about threat,[4] painful experiences such as surgery,[12] and culture/language.[26] Children's and parents' pain-related cognitive, emotional, and behavioral responses bidirectionally influence one another in what has been described as a "dyadic dance,"[81] through both the child's own experience and their observation of others' experiences.[85] pCAT predicts negatively biased memory of post-surgical movement-evoked pain one year later [86], which may be influenced by parent-child discussions of the painful experience.[117] Children report greater anxiety after observing a parent taught to manipulate (described as "exaggerating") their expressions of pain during the cold-pressor test and, following exposure to these facial expressions, girls, as compared to boys, rated greater cold pressor pain.[3] Youth treated as preterm neonates in the intensive care unit (NICU) report higher pCAT and their mothers are more solicitous of their pain than either full term infants treated in the NICU or controls.[50] Threat appraisals and pCAT increased in children told the cold pressor task was "quite painful and difficult to deal with," although pain and fear were not impacted by this manipulation.[4]

These early-life interpersonal interactions between children and their social environments continue to play out as children develop, as, for example, parental expressions of pCAT correlate with functional outcomes, including school attendance.[72] In children undergoing major surgery, pre-operative parental pCAT, but not the child's own pCAT, predicted long-term outcomes.[90] When a child has chronic pain, parents' and children's pCAT scores are typically highly correlated.[25] Reciprocally, interventions in pediatric samples, even those delivered exclusively to children, reduce parental fear of pain[2] and pCAT.[46] Parents are of course not the only salient and powerful social influences for children and adolescents. Several controlled studies have demonstrated that showing strangers experiencing pain can enhance fear of pain[113] and catastrophizing cognitions.[48] These modeling effects were also evident on behavioral measures such as avoidance, highlighting the impact of social processes on pain as a motivational and protective system. Interpersonal interactions appear to continue shaping pCAT into adulthood, as patient and partner reports of catastrophizing reciprocally influence one another.[103] In addition, pCAT in adults can be modulated upward or downward by verbal suggestions from another person, and those changes in pCAT result in alterations in the perceived intensity of a painful stimulus.[64]

## What happens when a person engages in pCAT?

Human laboratory investigations of pain and pCAT provide the advantage of controlling the administration of calibrated stimuli and reveal large individual differences in pain,[32] and pCAT accounts in part for some of these differences in both healthy individuals[8] and surgical patients.[61] Among individuals who experience chronic pain, where pain generators are usually variable and difficult to quantify,[33] meta analyses report consistent associations of pCAT with pain and pain-related outcomes in both children[76] and adults.

[93] When pain becomes chronic, pCAT is associated with greater clinical pain [114] and greater pain-related suffering, including pain-related disability, depression and anxiety [93], internalized stigma [118], and opioid misuse.[54] Similar effects of pain catastrophizing are seen in children with chronic pain and across varying pain conditions.[35; 76]

Studies associate pCAT with neurobiological processes known to increase sensitization to pain and vulnerability to chronic pain. The neurobiological mechanisms associated with pCAT in both healthy and patient samples are nicely summarized elsewhere[8; 106] and include a range of systems involved in pain processing.[23] For example, pCAT is associated with human laboratory indices of central sensitization (temporal summation of pain and aftersensations).[11; 29] pCAT is also associated with conditioned pain modulation (CPM), a laboratory measure of the descending pain modulatory system, suggesting that pCAT may interfere with the optimal functioning of endogenous pain-inhibitory systems.[41; 120] Both high pCAT and low CPM represent inter-related and uniquely predictive risk factors for acute and long-term pain outcomes following surgery.[66] And finally, pCAT correlated with IL-6 elevations observed in response to laboratory pain testing in both healthy individuals and people with chronic pain, elevations that continued for hours following pain exposure. [28; 68]

Maladaptive changes in both brain structure and function are seen in chronic pain and correlate with pCAT. Studies in adults with chronic pain[70] find that pCAT is associated with both functional and structural differences that include reductions in gray matter density [53] and altered processing in areas that contribute to spreading across anatomic areas.[30] Early work suggested associations between pCAT and the functional connectivity of the default mode network (DMN, a set of brain circuits that subserve self-referential cognition) with other networks participating in somatosensation, pain modulation, and attention/threat detection. More recent studies have shown that engaging in anxious and catastrophizing cognitions about pain activates the DMN[70] and confirmed that individuals reporting elevated pCAT also show amplified connectivity between the DMN and the salience and somatomotor networks.[30; 62; 63] Treatment-associated reductions in catastrophizing correlate with reductions in DMN connectivity.[69]

While caution is warranted when inferring causality from these associations, these findings, particularly those demonstrating longitudinal prediction of outcomes,[61; 86] are certainly intriguing and deserve continued investigation into the potential for a complex causal chain activated by pain in vulnerable individuals with elevated pCAT. One possible causal chain is presented in Figure 1. Caution is also warranted in regarding these neurobiological pathways to increased pain sensitivity as validation of pCAT, as current knowledge cannot untangle the nature[1] of the association between the construct of pCAT and these neurobiological processes.[75]

Experts in stress research have convincingly argued that models of stress can inform our understanding of pain, particularly chronic pain, based on the substantial overlap in knowledge of the neurobiology of stress and the neurobiology of pain,[74; 79] and stress

[1]**Footnote:** We thank an anonymous reviewer for noting this important distinction.

exposure enhances risk for pain chronification.[82] Various exposures, including early life stressors such as maternal separation in neonates, unpredictable sound, injury, and injection of low-dose inflammatory agents provide examples of priming, or the formation "...of molecular memory... that may adversely affect future responses to similar insults (p.195;[23])." Simply put, stressful and painful exposures have the potential to create these "molecular memories," including altered nociceptive processing in the brain, which serve as priming mechanisms for future hyperalgesia. Two primary brain networks, the reward system and the descending pain modulatory system (DPMS), play a major role in priming.[23] Early life stress exposure alters the DPMS thereby changing inhibition or facilitation of ascending nociceptive signals and the brain regions involved in processing the cognitive-affective dimensions of pain: the Anterior Cingulate Cortex (ACC), amygdala, and hypothalamus.[23]

Stress research has shown that maternal stress and cortisol during pregnancy influence the developing fetus such that the newborn's glucocorticoid stress response is elevated and the behavioral recovery from a blood draw is delayed.[22] Neonates under NICU care routinely experience both painful and stressful early exposures due to procedures and the typical disruption to mother-child bonding and attachment that occurs. Compared to healthy controls, preterm neonates treated in the NICU tested years later showed increased activation in response to moderately painful heat stimuli in the ACC and insula, areas involved in affective processing of painful stimuli.[51] A recent preclinical investigation primed animals with repeated noxious pin pricks that produced greater conditioned place aversion during contralateral non-noxious stimulation.[55] Since inhibition of ACC activity during priming reduced avoidance, avoidance was interpreted as consistent with "catastrophizing-like behaviors."

When the brain perceives threat, a network of physiological responses are activated in nonlinear networks that maintain allostasis yet, when overused, create the "wear and tear" of allostatic load.[23; 44; 79] In healthy adults, cold pressor pain stimulates a robust cortisol response that positively correlates with pain ratings.[18; 24] For individuals who catastrophize, laboratory findings suggest that pain is a highly provocative stressor that triggers an intense stress response. Cortisol responses to laboratory pain testing correlated with pCAT,[18] yet cortisol responses to a social stressor did not,[58] suggesting that pCAT potently shapes responses to painful stressors but may serve less strongly as a general indicator of stress response.[68] Lower cortisol reactivity has been associated with both higher self-reported stress and higher pCAT in older adults with chronic low back pain.[108] Lower diurnal variation in cortisol related to both greater leg pain and higher pCAT in lumbar surgery patients scheduled for surgery,[57] and back surgery patients who continue to report pain following lumbar fusion showed both a flattened cortisol awakening response and higher pCAT as compared to patients with a pain-free outcome.[39] The cortisol awakening response is sensitive to psychosocial factors such as stress, particularly chronic stress,[67] and has been shown to mediate the relationship between perceived stress and inflammation.[65]

Adverse childhood experiences show a dose-dependent[82] association with the development of chronic pain[19] and pain-related disability,[7] primarily mediated through

affective disturbances such as anxiety[19] and depression.[15] While pain is clearly a stressor, there is a bidirectional relationship (pain, especially chronic pain[79] → stress, as well as stress, particularly chronic stress → pain[44]). This bidirectional relationship is complex, involves multiple mediating and moderating factors, and requires understanding of potential protective effects,[74] including optimism,[45; 108] positive affect,[34] and resilience.[38]

## A developmental model for the role of pCAT as a risk for persistent pain across the lifespan

The evidence we have reviewed suggests that genetics create a susceptibility to pCAT that is independent of pain sensitivity and likely related to the affective dimension of pain processing. Extensive work has identified both cognitive/attentional and affective/motivational components of pCAT using laboratory stimulation, brain imaging, and both healthy and clinical samples. How the neonate learns to experience and manage distress is critical[92] to forming their earliest responses to pain and lays the foundation for anticipating pain, forming beliefs about the threat value of pain, and forming appraisals of their ability to cope with pain throughout childhood. As children develop, the child's own accumulated experiences with pain[51] and stress,[79; 80] observing their parents' responses to their own chronic pain,[102] and parents' modeling of pCAT[49] likely shape the neurobiological pain regulatory systems particularly those that amplify pain.[23; 79] Peer and sibling interactions may also shape pCAT, as can the acute pain of surgery.[86; 94] As these formative experiences accrue throughout childhood, increased pain sensitivity contributes to expectations and anticipation of future amplified pain.

As this cycle continues into adulthood, changes in brain structures influence the functioning of networks responsible for the perception and allocation of attention to stimuli such as pain. The salience network that is responsible for threat detection shows unusually elevated connectivity with the DMN in the context of pCAT[30] which may reflect hypervigilance. As neurobiological priming mechanisms[23] are repeated with additional routine exposures to pain (e.g., headaches, stomach aches, and sports injuries) and stressors, the central processing of nociceptive signals becomes amplified, generating a positive feedback loop whereby learning and experience alter neurobiological processes. We propose that, in some individuals, these altered systems in turn further amplify both pain and pCAT (pain -> pCAT -> ↑ pain -> ↑ pCAT (priming); in the future: priming -> ↑↑ pain -> ↑ pCAT -> ↑↑ ↑ pain ->↑ pCAT (more priming)) creating hyperalgesia and reinforcing thoughts that pain is terrible, unmanageable, and may get worse.

Over time, previously non-painful stimuli become painful. This may contribute to an increasing frequency and unpredictability of pain; a recent systematic review and meta-analysis reported that individuals experiencing high state level of negative affect (i.e., fear of pain, pain-related anxiety) found unpredictable noxious stimuli more intensely painful than predictable stimuli.[87] At the time of a precipitating pain episode – minor or major strain, minor or major injury or surgery – the evidence suggests that the neurobiological pain system may be further amplified by pCAT in response to the precipitating painful episode and this likely contributes to the perpetuation, and often worsening, of pain that endures

beyond “normal” healing. With continued pain and in particularly vulnerable individuals, pCAT contributes to the spreading of pain to additional body sites.[30; 83; 121] As pain becomes chronic pCAT contributes to risk for greater pain and pain-related suffering,[96; 122] including deterioration in mood[40] and poor treatment response.

This trajectory is not inevitable, as factors such as sex,[60] resilience,[38] and optimism [5] influence the impact of pCAT on pain and pain-related suffering. There is strong evidence that pCAT is modifiable by a number of interventions, particularly for individuals reporting clinically significant pCAT, although the findings are less compelling for subclinical pCAT.[97] An initial test of a single session intervention targeting pCAT recently showed promising results, both in comparability to a more intensive approach[21] and long-term outcomes.[20]

We propose that these learned experiences with pain contribute to the anticipation, experience, and future expectation of overwhelming and unmanageable pain (pCAT). We also propose that pCAT is part of a complex causal chain in which, at some point in the development of pCAT, various neurobiological processes are activated, and others inhibited, perpetuating increased pain sensitivity. These changes in cognitive, affective, and neurobiological processing of pain may contribute to longer-term alterations that further enhance pain sensitivity for longer and longer periods of time, heightening risk for chronicity, as portrayed in Figure 2. Consistent with the aging literature, older[77] and elderly adults[78] report less pCAT relative to younger adults, which may be attributable to resilience and other age-related changes in expectations and mood.[77]

## Summary and Future Directions

This synthesis demonstrates why these thoughts are important in understanding some people’s experience of pain, and the evidence suggests that pCAT contributes to pain-related suffering and may alter the neurobiological systems that modulate pain. Most of this evidence is based on “subclinical” pCAT and all health care providers integrating pCAT into their assessment or treatment of a person in pain need to be equipped to respectfully discuss pCAT and provide tools and support to help the person in pain manage this negative cognitive-affective mindset.[106; 110] Patients whose providers use labels such as “catastrophizer” feel dismissed and blamed[13; 119] when pCAT is not explained carefully and compassionately.[110]

While the evidence presented is compelling, crucial issues require investigation for the field to progress further. These include refining conceptual framework(s), developing modern measurement tools, designing pre-clinical models, and developing and testing interventions that investigate primary prevention. Debate continues about the conceptual framework that should guide future investigations of pCAT. The fear-avoidance model[115] has generated a wealth of knowledge about the development and maintenance of pain-related disability highlighting avoidance as a critical determinant of outcomes primarily in musculoskeletal pain.[36] pCAT plays an essential role in the FAM model with a recent meta- analysis finding that pCAT is more strongly associated with outcomes such as pain-related disability, negative affect, anxiety, and depression than the other key FAM constructs of fear of pain and pain vigilance.[93] This model is focused on avoidance and pain-related disability,

which are critically important outcomes for individuals with chronic pain[112] and are likely influenced by pCAT via multiple pathways (e.g., central amplification of pain intensity, hypervigilance to pain cues, expectations related to increases in pain with movement/activity, etc.). New or refined models need to capture the bidirectional, dynamic nature of the pain-pCAT relationship and integrate the numerous factors known to influence both the development and the impact of pCAT across time, including throughout childhood[94] and into adulthood.[56] In addition, future interventional studies may benefit from active efforts to optimize strategies to reduce the link between pCAT and behavioral avoidance.[101]

Another essential project involves developing the next generation measure of the pCAT construct that has been captured in the existing evidence. We believe stepwise and systematic progress in developing new measurement tools will ensure that the wealth of information developed about pCAT is not lost and that new approaches build on this knowledge base. Amtmann and colleagues[1] have proposed an alternative measure, titled Concerns About Pain scale, which may serve as a valuable starting point for this process. Webster and colleagues[119] initiated the “Rename Pain Catastrophizing” study as a patient-centered project designed to get input on the term from various stakeholders and others have also recently argued[13] that “language matters”, advocating for a change in terminology to recognize the social and cultural damage created by the pCAT term. Any new measure needs to include both dispositional and situational versions, as both the child[26] and adult literature demonstrate the value of both components and hint that state measures might be more valuable in understanding changes with experience.[116]

Preclinical human experiments need to continue to tackle the vital challenge of causality. Studies manipulating pCAT in both healthy and clinical volunteers are encouraging,[64; 71; 98] though findings are inconsistent.[98] Some investigations used statistical modeling to establish that changes in situational pCAT precede changes in pain, whereas changes in pain do not precede changes in pCAT.[9; 10] Other studies manipulated pCAT using pain control/anti-CAT statements and demonstrated reductions in pain,[109] and others have used hypnotic suggestions to increase and decrease pCAT in the same subject, demonstrating resulting increases and decreases in pain.[64] These findings suggest that, though the experience of pain activates pCAT (by definition), changes in pCAT contribute prospectively to changes in the experience of pain in a positive feedback loop. Experimentation needs to continue to investigate causal chains (i.e., invoke and manipulate pCAT in healthy individuals while investigating the neurobiological causal chain our developmental model delineates). Preclinical basic experiments may also have utility;[55] however, only if refinements of conceptual models include behaviors,[99; 104] distinguishing pCAT behaviors from pain behaviors.

And finally, this developmental model promotes the concept of early intervention, during childhood or adolescence when pCAT is forming and pain exposures are possibly priming the individual to be increasingly vulnerable to the negative effects of pCAT on pain and pain-related suffering. Exciting work demonstrating the impact of a 30-minute pain neuroscience lecture to middle schoolers[73] and a 90 min virtual group educational intervention for parents and child surgical candidates[123] suggest that relatively low-

cost interventions, delivered in childhood and adolescence, may have potential for wide dissemination and broad, possibly lifelong, impact.

We must embrace the idea that patients experiencing pCAT are not “exaggerating” their pain. These individuals experience pain as more threatening and appear to mount a robust, possibly stimulus-specific stress response to pain which may intensify their experience of pain. The data we have reviewed suggest that these individuals may be experiencing greater pain as a result of sensitized neurobiological processing of nociceptive signals. This hypothesis deserves detailed investigation in order to identify relevant components of the causal chain(s) linking pCAT and greater pain,[75] including cognitive, affective, and neurobiological components. A person’s experience of pain today is a product of the many prior exposures and learning experiences that have shaped their cognitive, affective, and pain modulatory systems. As do all people who experience pain, people with pain who catastrophize deserve our respect and compassion so that they can trust that we will listen carefully to their experience and work with them to find the best treatment(s) for their pain.

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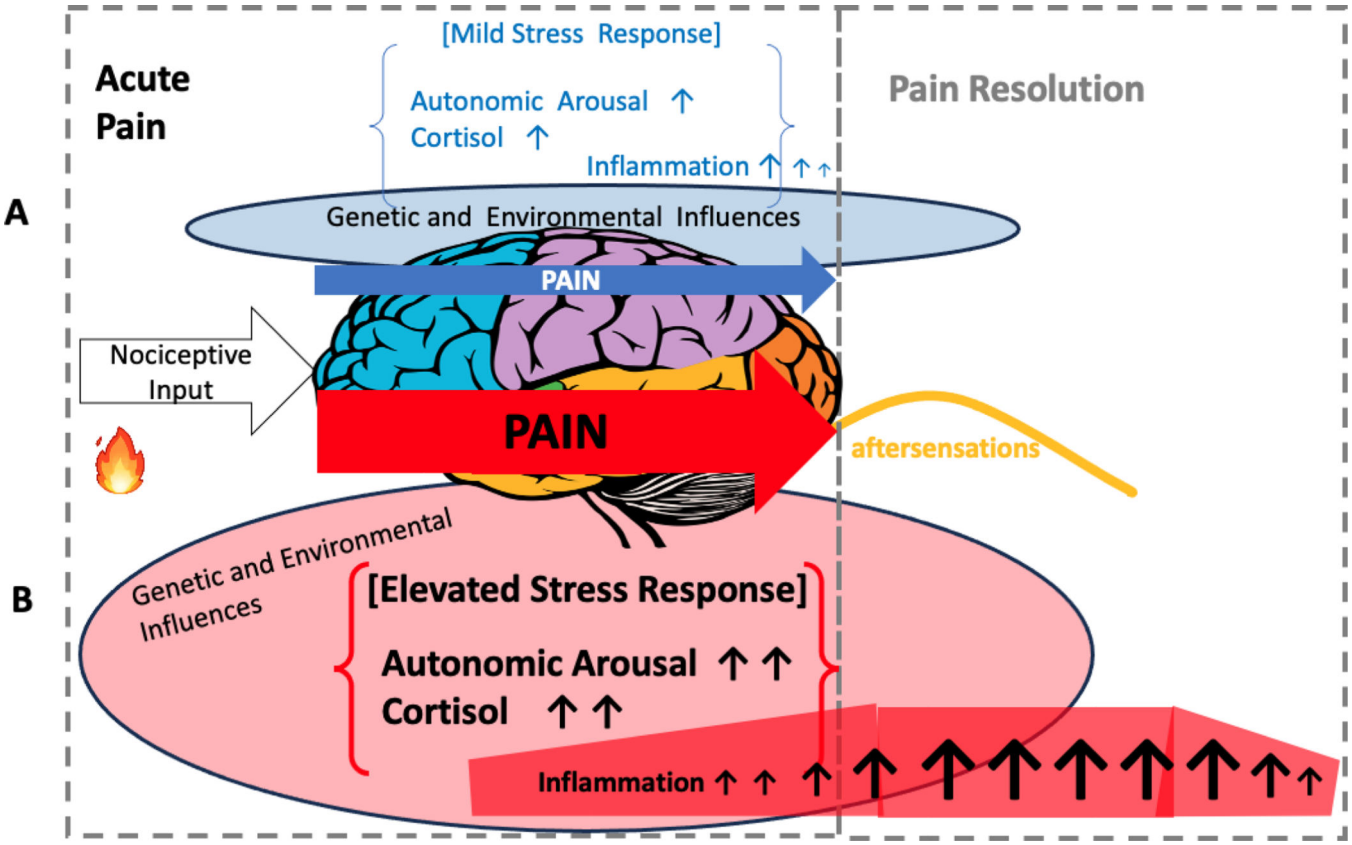


**Figure 1:**

Neurobiological mechanisms associated with pCAT in laboratory studies using calibrated stimuli are likely influenced by genetic and environmental/learning factors. (A) depicts an individual low in catastrophizing who demonstrates a mild stress response to pain with rapid resolution; (B) depicts an individual medium or high in catastrophizing who demonstrates an elevated stress response with slow resolution, including immediate aftersensations and also elevated inflammation for hours following termination of the painful stimulus. Moderating factors such as genetics and environmental exposures, including stressors, interact with pCAT and influence the magnitude of the stress response to pain.

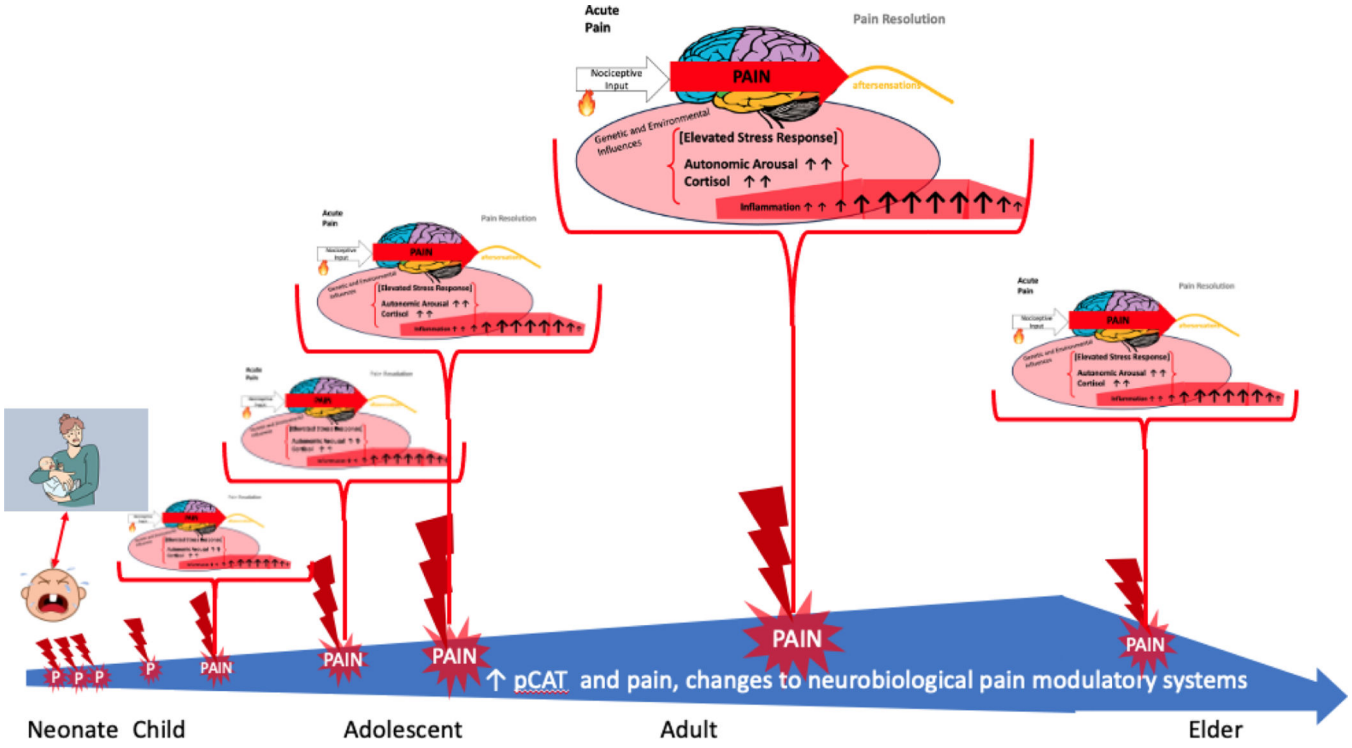


**Figure 2:**

A developmental model for the role of pCAT in contributing vulnerability to persistent pain across the lifespan. While this model focuses on learning from exposures to pain and pCAT and changes to the neurobiological pain modulatory systems (detailed above in 1B) that accumulate over time resulting in increased pain sensitivity, the role of pCAT in contributing to pain-related suffering is also substantial. As noted in the text, a number of factors likely modulate the relationship between pCAT, pain, and pain-related suffering.