Biased Competition Favoring Physical Over Emotional Pain: A Possible Explanation for the Link Between Early Adversity and Chronic Pain

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ABSTRACT

Background: Early adversity predisposes to chronic pain, but a mechanistic explanation is lacking. Survivors of early adversity with chronic pain often seem impaired in their ability to be aware of, understand, and express distressing emotions such as anger and fear in social contexts. In this context, it has been proposed that pain may at times serve as a "psychic regulator" by preventing awareness of more intolerable emotions.

Method: This narrative review builds on the premise that physical pain and emotional pain are conscious experiences that can compete for selective attention. We highlight mechanisms whereby the consequences of early adversity may put emotional pain at a competitive disadvantage. A case history, supportive research findings, and an evidence-based neurobiological model are presented.

Results: Arising from abuse or neglect in childhood, impairments in the adult capacity to attend to and/or conceptualize the emotional meaning of felt distress may be associated with impaired engagement of the default network and impaired top-down modulation of affective response generation processes. Persistent and poorly conceptualized affective distress may be associated with reduced emotion regulation ability, reduced vagal tone, increased inflammation, and amplified nociceptive signals. Attention to physical pain may be reinforced by the temporary reduction in negative emotions that it causes.

Conclusions: These processes jointly promote biased competition favoring attention to physical pain and away from one's own emotions. They may constitute an unintentional analog of the phenomenon of self-injury in patients with borderline personality disorder in whom the intentional infliction of physical pain serves to downregulate intense emotional distress. Attending to, expressing, and understanding previously unacknowledged psychological distress unrelated to pain may facilitate recovery from chronic pain after early adversity. Mechanistic studies that can validate this clinically derived neurobiological hypothesis are urgently needed.

Key words: conceptualization, default network, early adversity, emotional pain, pain, psychotherapy.

INTRODUCTION

Chronic or persistent pain is a major health problem, estimated in 2010 to affect approximately 19% of the US population (1) and to cost US $635 billion in combined medical costs and lost productivity annually, exceeding the costs of cancer, heart disease, and diabetes (2). One of the reasons it is such a major problem is that there are few any effective treatments (3). An additional factor is that chronic pain is indeed a complex and heterogeneous condition and a single mechanism is not likely (4). The purpose of this article is to address one particular aspect of chronic pain—the observation that early life adversity increases the likelihood of its occurrence (5–7). Our goal is to produce an evidence-based model of possible brain mechanisms that would help explain this association. The justification for this model is derived from a combination of literature review and clinical observations, including those made during the psychotherapeutic treatment of patients with early adversity and chronic pain. Should the proposed mechanisms be verified in subsequent research, it could potentially pave the way for new modes of intervention.

Attempts to understand the mechanisms linking early adversity and chronic pain based on objective measures such as biological variables and behavioral observations must take into account a fundamental truth about chronic pain—it is a conscious experience. According to the International Association for the Study of Pain, "pain is an unpleasant sensory and emotional experience associated with actual or potential tissue damage" (8). Persistent pain is defined as pain every day or most days for the past 3 months (1), and, very similarly, chronic pain is generally defined as pain that lasts longer than 3 months (9). This definition of pain accepts that people do at times report pain for strictly psychological reasons and that, because reports of the latter can't always be distinguished from those due to a physical cause, they should be taken at face value (10). If a patient says that they are in pain, they are, no matter what objective tests reveal.

This creates major problems for the scientific study of pain: how can we know that we are engaging pain mechanisms and altering them if the only way we can measure pain is based on what people tell us? If one relies only on overt behavioral indicators of pain, as in studies in laboratory animals, the common human context

EAET = emotional awareness and expression therapy, **LEAS** = Levels of Emotional Awareness Scale

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in which such behavioral indicators are not present is not addressed. Our position is that once one acknowledges this limitation and accepts the fact that there is no adequate proxy or substitute for self-reported pain, this constitutes a foundational starting point for a new approach (11). It means that what we know about the brain basis of conscious experience becomes highly relevant. Specifically, conscious experience seems to reflect a limited cognitive resource, with competition for access to this resource (modulated by attentional amplification/suppression) between representations that are simultaneously activated across multiple brain systems at any given time (12–14). We place competition for conscious access and attentional amplification/suppression at the center of the perspective that we advance below.

A consequence of this new perspective is that we are limited in the kinds of data we can bring to bear in an attempt to provide an evidence-based account. One limitation is that we are seeking to obtain useful information while relying on self-reports of experience. A second limitation is that prospective research data are not available that link the concurrently assessed experience of children who have been subjected to abuse and neglect with their medical outcome as adults. Much of what we know is based on retrospective accounts of early adversity as well as the psychological characteristics of adult survivors of early adversity who are treated clinically in psychotherapy (15).

Because our focus is on early adversity as a factor that predisposes to chronic pain, we must consider how early adversity affects the way the brain represents different types of percepts and concept and how it affects the way these representations compete for attention. Early adversity itself is a heterogeneous phenomenon, including neglect and a wide variety of types of abuse, including physical, sexual, emotional, and verbal (16,17). For example, some forms of abuse involve physical pain, and early exposure to pain clearly alters brain mechanisms of pain processing later in life (18,19). Nevertheless, our starting point in this discussion is to hold that early adversity always involves *emotional pain*. Neglect, verbal abuse, and mistreatment of any kind hurt emotionally. This can in turn have important effects on learning and development in childhood (20). A cornerstone of our thesis is that early adversity predisposes to chronic pain in adulthood at least in part because early adversity reduces opportunities for learning to understand emotions through multiple pathways.

The term "emotional pain" is itself controversial (10), and this controversy gets to the heart of the issues at hand. The term as used here refers to affective distress that is so severe that it hurts (20). As with physical pain, it is impossible to determine with objective measurements whether someone is experiencing emotional pain: if a person says that they are experiencing emotional pain, they are. The objection to this is that it is misappropriating a word that is more properly used when there is a detectable physical cause. However, to insist on a strict separation is to maintain the perspective of Cartesian dualism that psychosomatic medicine specifically seeks to avoid. Moreover, no physical proof can be generated in either instance and the International Association for the Study of Pain definition acknowledges this. Biro (10) recommends accepting the impossibility of verifying the existence of pain or differentiating between psychological and physical causes. He proposes, consistent with some neuroimaging evidence that the anterior cingulate cortex may play a central role in representing pain and its aversiveness (21,22), that pain be categorized on a continuum from purely physical causation at one extreme to purely psychological causation at the other extreme (with all of the variations and combinations in between).

With this introduction of our assumptions and intended approach, we will now present an overview of a hypothesis derived from literature review and clinical observations about how emotional pain that is too difficult to process consciously may amplify/maintain somatic pain. This will be followed by a case example. The subsequent section will review empirical evidence in support of this hypothesis, followed by a section in which the brain basis of this hypothesized interaction between emotional and physical pain will be presented. We will conclude with a discussion of the implications of this proposal for research and clinical intervention.

HOW EARLY ADVERSITY MIGHT PREDISPOSE TO CHRONIC PAIN: THE BIASED COMPETITION HYPOTHESIS

Consider the plight of the mistreated child. If the parent is the perpetrator, the child will very likely have to deal with the emotional pain on its own on many occasions. We know from adults with early adversity that in some instances, which are commonly seen clinically, people will intentionally hurt themselves physically to distract attention and reduce the felt intensity of their emotional pain (23). It is therefore very reasonable to conclude that an abused child is motivated to minimize or avoid emotional pain to the extent that it can. How might the child do this?

Several tactics are likely. First, clinical observations suggest that children are often told that they are being treated as they are because they behaved badly. As a result, the child may attempt to be as perfect as they know how to be (24). Because the explanation for the mistreatment actually resides within the perpetrator, and bad behavior is not the actual cause or reason for the mistreatment, this strategy does not work, but the child has no way of knowing this. All the child knows is that despite their best behavior, the mistreatment and the pain persist. A second strategy is to remain vigilant for any signs of anger or other predictors of abuse that can enable the child to avoid (or at least prepare for) the next round of abuse (25). A third strategy is for the child to take advantage of the fact that there is competition for attentional resources and that if the competition can be influenced/controlled in some manner, then the emotional pain can be attenuated. Children learn, for example, that they can find some relief by distracting themselves, thinking of other things, or "tuning out" from their internal experience (26,27).

Another important consequence of early adversity pertains to learning the ability to mentally representing one's own emotions. Here, we focus on one particular aspect of mentally representing emotion, namely, representing the conceptual meaning of the affective responses one feels (i.e., representing emotion concepts). This conceptualization ability allows one to self-report feeling particular emotions and to understand and express those emotions adaptively. Developing this ability during early adversity may be hindered by the fact that in early childhood, emotions are interpersonally regulated (28). In the common case in which a parent is the perpetrator, it is a double whammy (29)—not only is the child abused but also the child does not get the mirroring, empathy, and compassion that it wants, needs, and deserves; the parent therefore does not help

regulate the child's emotions or help the child learn to recognize or regulate their own emotions in an adaptive manner (30,31). Specifically, given that the learned capacity to conceptualize and understand one's own emotions may be to a large degree dependent on this type of outside input from caregivers (usually parents) (32,33), poor conceptualization/understanding of emotions in adulthood can follow (e.g., see (34,35)).

Moreover, because of the lack of love and caring in the lives of abused and neglected children, this may lead to a deficit in positive emotion as well and thus decreased motivation to seek out close interpersonal relationships (36). Other sources of positive self-regard can be pursued (such as excellence in school or sports). Nevertheless, the foundational lack of caring and love can persist and be assuaged but may not be fully eliminated by other sources of good feelings. Thus, the early adversity survivor may stay quite attuned to signs of threat and danger and other predictors of impending harm, recognizing based on personal past experience when relationships are dangerous and threatening, but may become much less emotionally attuned and have relatively impoverished emotion knowledge when it comes to being close, attached, or loving with others, and may be less motivated to seek such relationships (37–39). This would make it more difficult to function effectively in interpersonal relationships and to benefit from the self-affirming and stress-reducing effects of secure attachment.

The general life strategy of many survivors of early adversity, therefore, may be to pursue interests that avoid or block out emotional pain (17). However, emotional pain cannot typically be avoided if one attempts to lead a normative occupational and social life. Work can get you only so far because there is a major social/interpersonal element to many if not most jobs. The inborn need for attachment and closeness with others can be denied or disavowed, but loneliness and lack of interpersonal validation can be painful. At times, emotional pain can be of such severity that self-induced physical pain is used as a last-ditch strategy to provide temporary relief (23).

One common clinical observation is that chronic pain is often preceded by an injury that causes acute pain (40). In the context of someone with early adversity, who adopts a life strategy of trying to minimize the felt intensity of his or her affective distress, this "unfortunate" occurrence can be found to serve a useful purpose. That is, the survivor may implicitly learn that attention to physical pain provides temporary relief from emotional pain, because of the fact that reduced attention to any given experience (including pain) can simultaneously reduce its perceived intensity (41–43). Through repeated experiences in which felt distress was reduced by increasing attention to pain, this pattern of avoidant attention would be reinforced and become habitual. This type of reinforcement/habitization process can occur in the absence of awareness (44–46), and the survivor need not understand that (or why) they have acquired such attentional habits. Reduced understanding of emotions in those with early adversity would also likely further promote reduced emotion-focused attention.

An important clinical caveat, which helps explain why such associations with emotional pain are not obvious, is that if the individual is asked about sources of distress in their emotional lives, which are typically interpersonal, such sources may not be reported (47). This is because the survivors may be hyperalert to cues or signs of danger (such as angry facial expressions) but often not to more complex interpersonal scenarios such as separation or loss. Recognition of such scenarios requires the ability to link them with emotional distress, but if life-long adaptations have been implemented to decrease emotional awareness with respect to troubling experiences such as separation and loss, this may not occur. However, if an astute clinician takes the time to obtain a careful history to determine the emotional context in which chronic pain is observed, particularly shortly after its onset, supportive evidence of this kind can often be obtained.

One of the legendary figures in psychosomatic medicine, George Engel, who was among the first to link early childhood adversity to chronic pain in adult life (48), made observations along similar lines and reached the conclusion that in certain pain prone patients, their pain seemed to serve as a "psychic regulator" (49). That is, a focus on physical pain seemed to serve as a type of "replacement" for emotional pain that was less tolerable.

Our model builds in part on Engel's prescient ideas. Similar to the "pain prone" individuals described by Engel, our primary aim is to establish the plausibility of the idea that in survivors of early adversity, patterns of attention to physical pain may become implicitly reinforced habits/strategies precisely because (1) they reduce the intensity of experienced emotional pain or distress and (2) emotions can be poorly understood in such individuals and therefore tend to be ignored. To illustrate, we will first provide a clinical example taken in part from a previously published case report by one of the authors (FSA) (50). After providing this example, we will then review work in cognitive neuroscience that supports the mechanistic plausibility of our proposed explanation for the link between early adversity and chronic pain.

CASE HISTORY

Martin (who provided consent for the case material presented in this article) was a married father of four children in his early 50s and a highly successful professional in the financial sector who requested psychotherapy to help with intractable pain and discomfort in his feet. Numbness in his toes began about 6 years earlier and progressed to involve persistent pain in his feet and legs. Three years later, he was diagnosed with peripheral neuropathy of unknown cause. An explanation for the pain in his lower extremities based on tissue damage or abnormalities could not be identified, and he was referred to psychotherapy under the assumption that it was stress related.

In psychotherapy, his extensive history of physical and emotional abuse in childhood was explored. His father was a well-loved high school teacher who would unpredictably fly into rages at home and regularly beat Martin, the youngest of five children, and his siblings with a belt. His older brother, who himself was bullied at school, would regularly beat Martin as well. Martin would deal with this physical abuse by "going internal" and doing his best not to feel the pain or show any outward signs of pain or distress (e.g., he remembers being determined not to cry). His relationship with his father was complex; although he was filled with fear and hatred during the beatings, he would also snuggle up to his father in bed at night when he was feeling ashamed or distressed and his father would comfort him. His father also had sore feet and Martin became quite skilled as a child at massaging his father's feet to help him feel better. His mother was a stoic "suck it up" person who did not protect him from the abuse and participated in the frequent mocking and shaming imparted by the family.

Martin dealt with this ongoing abuse by being driven, perfectionistic, extremely conscientious, and hypersensitive to others. He developed what he called a "hardened shell" that was difficult to pierce. Over time in psychotherapy, he felt that pinholes were being poked in this shell so that he could occasionally glimpse what was underneath, which he described as "ugly and bothersome" and "a beach ball of fear." "It's like this horrible experience that I find hard to believe actually happened. I know it happened. I lived through it. But there is a huge difference between knowing something and feeling it."

Martin was resilient and determined to find relief for the medically unexplained somatic pain that plagued him for decades. Martin's therapist was a relational psychoanalyst and pain and trauma specialist who saw Martin twice a week. Her approach to treating chronic pain was inspired and informed by the work of Sarno (51), who was familiar with Engel's insights. In sessions, she focused on being empathic and attuned to the nuances of his expressed emotion and the emotional implications of the situations he described—a kind of responsiveness he did not receive earlier in life. A major goal was to help Martin feel securely attached (39) to her (i.e., being comforted rather than rejected no matter what he said), which enabled him to share difficult and vulnerable feelings without shame or embarrassment and become more aware of his feelings and the meaning of his past and present experiences. Within the safety of the therapy relationship, he learned to identify, tolerate, and begin to regulate emotions that had been disconnected from his previous and current experience.

After 3 years of therapy there have been significant gains. He no longer feels that fear is what defines him. He is back to working out twice per week and playing golf weekly (including walking the course, which had previously been too painful). His medications, including duloxetine, gabapentin, and atomoxetine, have been discontinued. He continues to take lorazepam daily for anxiety but at lesser doses as he supplements this medication with breathing exercises, meditation, and prayer. He came to realize that he had been mistreating his own children because he was simply repeating what had been modeled for him as a child; he subsequently changed his parenting practices once he realized what he was doing and the effect it had on his children.

This case history therefore illustrates the following: (1) the adaptations to abuse that led him to become detached from and unable to experience his feelings; (2) the perfectionistic style he adopted as a reaction to the mistreatment that he received; (3) the insufficient parental empathy and lack of sufficient soothing he received in childhood; (4) the presence of long-standing physical symptoms for which a medical explanation could not be found; (5) the expansion of his repertoire of emotional experiences and his greater ability to feel, identify, and regulate his emotions as a result of empathic therapy that aimed to provide the ongoing experience of secure attachment; and (6) the reduction in pain and the expansion of healthy and adaptive behaviors coinciding with his improved awareness of his own and others' emotional experiences.

EVIDENCE SUPPORTING THE BIASED COMPETITION HYPOTHESIS

Links Between Early Adversity, Decreased Emotional Awareness, and Amplified Physical Pain

Retrospective studies have documented that early adversity is associated with a variety of physical and mental illnesses in adulthood, including chronic pain (5,7,16). Fiddler and colleagues (52) showed that early adversity was associated with a greater number of physical symptoms, including chronic pain, independent of whether physical symptoms were medically explained or not. A prospective study by Jones and colleagues (53) showed that adversity in childhood, such as prolonged separation from one's mother, was associated with the development of chronic widespread pain (similar to fibromyalgia) in adulthood. This is important evidence, in part because it speaks against a possible alternative explanation—that the association between early adversity and pain in adulthood is solely attributable to the latter's enhancement of the ability to retrospectively recall emotionally painful experiences in childhood (such as abuse and neglect).

A study of adoptees from Romania who immigrated to the United Kingdom demonstrated that compared with those raised in more traditional homes, children raised in orphanages in which physical but not emotional needs were attended to have severe impairments in theory of mind and executive processes (54). Among those from orphanages, the longer they lived in such facilities the more severe were these impairments. Similar findings have been obtained in children living in a boarding home in Turkey and children in foster care in the United States, including observed reductions in emotion understanding and theory of mind (55,56). These findings suggest that severe neglect is associated with major impairments in understanding the minds of others as well as impairments in self-regulation (also see (35,57,58)). They are also consistent with a rich clinical literature suggesting that emotions are interpersonally regulated in early childhood and that in the absence of mirroring and empathy, the capacity for emotional self-awareness and related emotion self-regulatory capacities is reduced (17,28).

In addition to cognitive abnormalities, abuse and neglect are now known to be associated with changes in the brain and peripheral physiology. Teicher and colleagues (59) have shown that there are changes in gray matter and connectivity patterns that correspond to the type of abuse that children experienced. For example, witnessing physical violence is associated with reduced gray matter in visual cortex (60), and a history of sexual abuse is associated with reduced gray matter in the genital area of somatosensory cortex (61). Regarding peripheral physiology, Lovallo and colleagues (62) showed that relative to matched participants without childhood adversity, heart rate and cortisol responses to the Trier Social Stress Test were reduced and that the reduction was greater in those with more severe early adversity. Similarly, in contrast to patients with simple phobia who show potentiated startle responses in aversive contexts, patients with long-enduring, pervasive apprehension/avoidance with broad anxiety and depression comorbidity (e.g., posttraumatic stress disorder secondary to cumulative trauma) showed startle responses that were paradoxically diminished in all aversive contexts (63). These findings are consistent with the interpretation that the brain and body naturally adapt to attenuate the experience of stress and distress associated with abuse and neglect.

Alexithymia, referring to impairment in the ability to identify and describe emotion, and differentiate emotion from bodily sensations, is known to be associated with greater chronic physical pain (64,65). Scores on the leading measure of alexithymia, a self-report measure called the 20-item Toronto Alexithymia Scale, also correlate highly with self-reported negative affect (66). One

study showed that alexithymia was associated with the affective but not sensory component of pain and that this association was mediated by depression (67), raising the question of whether reported negative affect (as on a depression inventory), and not an impairment in the ability to report or experience it, accounts for this association with pain (65). In a study of women with painful rheumatic conditions (fibromyalgia and rheumatoid arthritis) relative to healthy women, the patients scored higher on the 20-item Toronto Alexithymia Scale and lower on a related performance measure called the Levels of Emotional Awareness Scale (LEAS), which does not correlate with negative affect (68). In a related observation by Lackner (69) in patients with irritable bowel syndrome, lower LEAS scores were associated with more severe pain on a typical day. Thus, current evidence suggests that lower emotional awareness is associated with greater pain.

Furthermore, Lumley and colleagues (70) recently reported a randomized three-arm trial comparing an 8-week group-based "Emotional Awareness and Expression Therapy" (EAET) to cognitive behavioral therapy and to psychoeducation for fibromyalgia. EAET produced significantly greater increases in LEAS scores and significantly greater reductions in pain at the conclusion of treatment, and at 6-month follow up, relative to the other two arms. Similar findings of reduced pain were also obtained in another clinical trial of emotional awareness training in irritable bowel syndrome (71). Together, these findings strongly suggest that similar to the psychotherapeutic treatment of Martin, enhancing emotional awareness can improve pain.

Competition Between Emotional and Physical Pain

In the formulation above our claim is that survivors of early adversity come to discover that paying more attention to physical pain reduces the experienced intensity and awareness of affective distress arising from their social circumstances. A related phenomenon, which is unfortunately quite common in patients with borderline personality disorder (a condition often associated with early adversity and poor understanding/regulation of emotion (35,72–74)), is that of intentional self-injury. This is a clinical phenomenon in which people intentionally induce physical pain, such as by making superficial cuts in the skin with razor blades, for the purpose of relieving emotional pain or distress (23). An imaging study was performed in patients with borderline personality disorder and healthy volunteers in which emotional distress was induced and participants cut themselves superficially to induce pain (75). Imaging data revealed that self-induced pain (superficial cutting) reduced amygdala activity associated with top-down frontal lobe inhibition in patients with borderline personality disorder, whereas no such pattern was observed in healthy controls, either in reducing distress or in dampening amygdala activity. This study did not examine early adversity per se, but as noted previously, a strong association exists between borderline personality disorder and early adversity (72). This study provides corroborative and mechanistic evidence to support the claim that physical pain may at times be preferable to emotional pain in people who have experienced early adversity. It also supports the possibility that the mechanisms at work in early adversity may not operate in the same way in healthy volunteers, which may be due to the neuroanatomical and physiological adaptations made to early adversity.

PLAUSIBLE NEUROCOGNITIVE MECHANISMS PROMOTING AMPLIFIED PHYSICAL PAIN AND REDUCED EMOTIONAL AWARENESS IN EARLY ADVERSITY

Cognitive/Biological Mechanisms

In this section, we seek to explain how, in the context of early adversity, biased competition between attention to physical versus emotional pain may be implemented within well-studied neurocognitive processes (Figure 1). What needs to be explained is how the effects of early adversity and the adaptations to it lead affected individuals to preferentially focus on physical pain (i.e., when simultaneously faced with social sources of emotional distress and bodily sources of physical pain in adulthood). As described previously, we place primary emphasis on two major factors: (1) reinforcement of attention to pain and (2) poor emotion conceptualization abilities (which can in turn result in prolonged, poorly understood, and poorly regulated unpleasant affective arousal (76,77)). Here, we will briefly highlight current bodies of work on mechanisms that may underlie these phenomena.

With respect to poor conceptualization ability, Bayesian models of perception may be particularly useful. These models (78) and the growing body of work supporting them (e.g., (79–89)), suggest that the brain infers the most likely interpretation of afferent sensory input (including afferent nociceptive and interoceptive input) based on the following three factors: (1) learned prior expectations, (2) the discrepancy (prediction-error) between those expectations and afferent input, and (3) learned, context-specific estimates of the relative reliability of those expectations and those prediction-error signals. Such models suggest that in the context of poor emotion concept learning in early adversity, low reliability estimates would be assigned to emotion concept representations—effectively reducing attention to and thoughts about emotions. Such models also entail that if an individual has learned a strong expectation for pain, and the estimated reliability of that expectation is higher than the estimated reliability of afferent nociceptive signals in that context, then such an individual's brain may infer (and therefore perceive/experience) the presence of pain, even in the absence of actual tissue damage ((90); also see (91)). Thus, in the case of survivors of early adversity, if there were repeated instances of physical pain in childhood, as was the case with Martin, there may therefore be a strong learned expectation that leads such survivors to attend more to physical pain and to perceive/interpret ambiguous afferent signals from the body as indicative of physical pain (also see (19,82,92,93)). Recent findings supporting Bayesian models of pain processing suggest that those with higher expectations for pain will experience more pain given the same afferent input (corresponding to differences in prediction-error processing within the anterior insula; (94,95)). Greater expectations for pain and reduced expectations for emotion can therefore combine to promote a biased competition favoring attention to pain and an inability to understand and regulate affective distress and its causes.

The resulting, poorly regulated affective distress is also likely associated with a shift in sympathetic/parasympathetic balance, such that parasympathetic tone is reduced and its anti-inflammatory effects are disinhibited (96–98). As a result, inflammation is enhanced, which can in turn heighten nociceptive processing and promote hyperalgesia (painful stimuli become more painful) and allodynia (benign stimuli become painful) at both central and

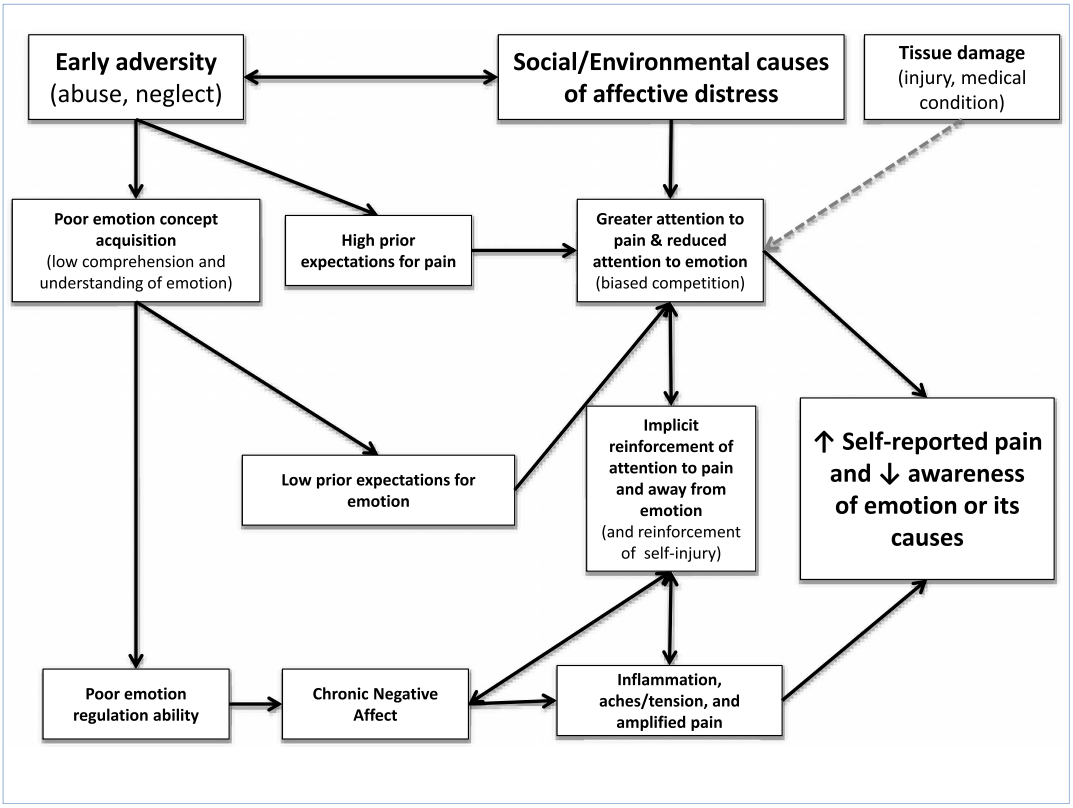

FIGURE 1. Depiction of the proposed psychological mechanisms described in the text. Early adversity is envisioned to hinder emotion concept learning and increase pain expectations. Poor emotion concept acquisition in turn leads to both reduced expectations for feeling emotions and poor emotion regulation ability, which in turns promotes greater levels of chronic (poorly understood) negative affect. Chronic negative affect can both induce tension, aches, and other sources of somatic pain and can also amplify afferent pain signals both centrally and peripherally (in part via increased expression of proinflammatory cytokines). With these effects in place, attention would be biased toward somatic pain and away from thought about emotion, creating a biased competition favoring pain at the forefront of awareness. In the presence of social sources of affective distress, attention to pain would reduce intensity/awareness of felt distress, which would in turn reinforce the habit of attending to pain. This mechanism is captured in the figure by illustrating that when both chronic negative affect and attention to somatic pain jointly interact with reinforcement learning mechanisms, the result would be the habitization of attention to pain. Similar reinforcement learning mechanisms could also promote self-injury behavior, because this can reduce felt affective distress in a similar manner. Whereas these mechanisms could operate in the absence of peripheral tissue damage, its presence would facilitate the initiation of these mechanisms. The gray dashed arrow emanating from the "Tissue damage" box signifies that it is facilitating but not essential to the proposed mechanisms. Color image is available only in online version (www.psychosomaticmedicine.org).

peripheral stages of processing (99–101). Through this mechanism, persistent and unrecognized/unregulated negative affective responses may therefore amplify/maintain perception of pain; however, these painful somatic experiences would not be recognized/understood as emotional in origin. Thus, in the presence of injury, perceived pain intensity would be amplified (hyperalgesia), and in the absence of injury, nonpainful stimuli may be perceived as painful (i.e., due to allodynia and strong top-down expectations regarding the presence of physical pain). Such influences would act in combination with the fact that persistent negative affective responses can themselves directly produce sources of bodily pain as well (e.g., diffuse aches and soreness from chronic muscle tension or altered sleep; (102))—the perceived intensity of which could also be amplified by the same mechanisms described previously.

Pain perception in those with early adversity may be further amplified by other attention-related mechanisms as well. Current neurocognitive models of conscious awareness (12) and the large body of evidence supporting them (e.g., (13,14)) suggest that conscious processing represents a limited resource that the brain must selectively allocate toward some representations over others. Attentional modulation represents one mechanism that enhances the strength of attended representations, suppresses the strength of unattended representations, and therefore promotes conscious processing of attended representations. As mentioned previously, the absence or deficit in emotion conceptualization processes would be expected to put emotion concept representations at a disadvantage in the competition for attentional resources relative to perceptual representations of pain. Under these circumstances, bodily sensations arising due to chronic negative affective responses may therefore be more likely to be perceived as ambiguous bodily sensations, interpreted as purely physically (i.e., not emotionally) painful, and the intensity of all sources of pain would be further amplified by pain-focused attention (43).

With respect to reinforcement learning mechanisms, leading models (46,103) and the large body of work supporting them (e.g., (104–107)) suggest that the selection of both cognitive actions

(such as attention allocation) and behavioral actions (such as inflicting self-injury) will occur with greater frequency when—in an individual's personal past experience—such actions have been reliably followed by either (1) increases in pleasant/rewarding experience or (2) decreases in unpleasant/distressing experience. As such, the reduction in felt affective distress when attention is selectively allocated to somatic pain will itself be reinforcing. Under these conditions, attention toward somatic pain would become frequent/habitual (i.e., due to negative reinforcement), because it would reliably reduce overall distress in the context of chronic negative affect (i.e., by removing attention from affective distress, and the amplifying effects it would otherwise have).

Similar negative reinforcement mechanisms may also account for self-injury in the context of early adversity (23). That is, if an individual learns that attention to somatic pain reduces their overall distress, they may also come to realize that self-injury effectively draws their attention toward somatic pain as well (i.e., pain is known to powerfully "grab" attention in a bottom-up manner (43,108)). Thus, because self-injury draws attention toward somatic pain, felt distress is likely to be reduced, which itself is reinforcing. Repeated self-injury may therefore become a habitual strategy for affect regulation in those with early adversity in a similar manner to the habitual attention allocation strategy described previously.

Role of Neural Networks

There is a large body of work on the neural basis of many of the elements of the explanation we propose (Figure 2). For example, one network of brain regions, called the default network, has been linked to the conceptualization processes that underlie emotion recognition, emotional awareness, and theory of mind abilities (109–113). Another network of brain regions, referred to as the limbic network, seems to play an important role in generating affective responses and associated visceromotor control processes (109,110,114). The amygdala and a network of brain regions called the salience network have been implicated in the automatic allocation of attention to pain and other affective stimuli relevant to homeostasis and metabolism (109,115). Finally, the basal ganglia, via interaction with sensorimotor network regions and with the midbrain dopamine system, have been implicated in reinforcement learning processes (46,116).

Therefore, it seems plausible to suggest that chronic negative affective responses (i.e., somatic/visceral responses) in those with early adversity are continually generated by the limbic network

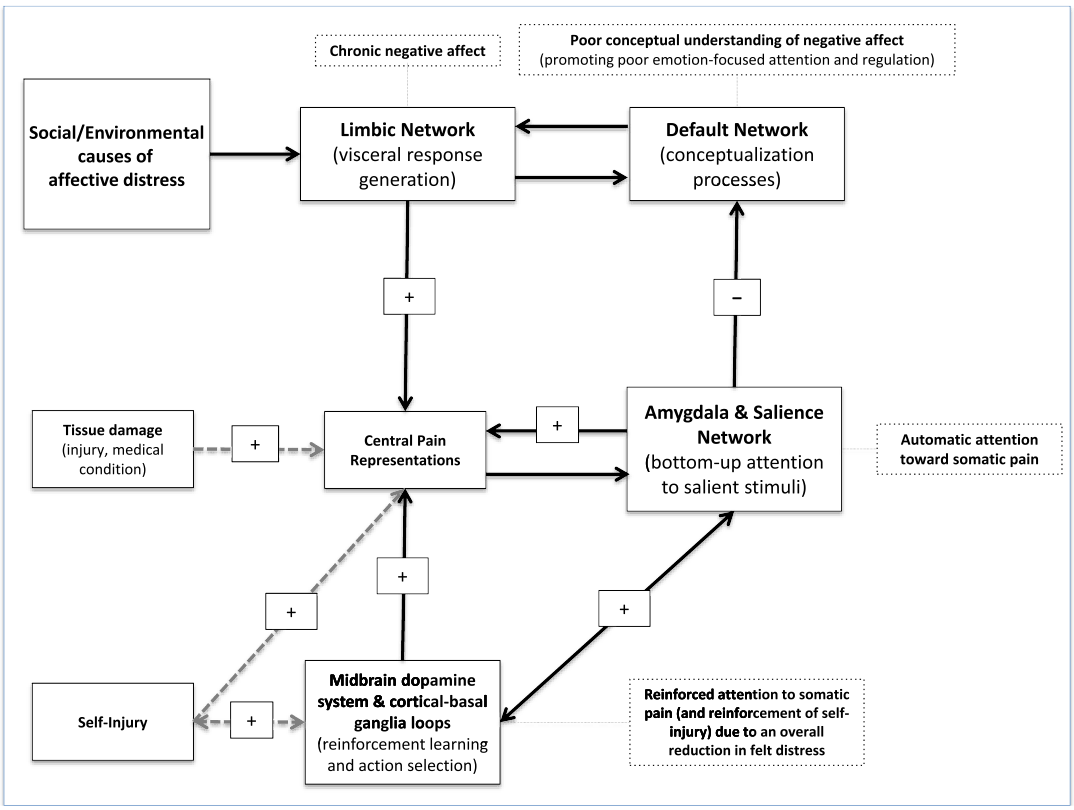

FIGURE 2. Depiction of the proposed neural basis of the psychological mechanisms depicted in Figure 1. The limbic network is envisioned as serving the function of initiating/maintaining chronic negative affective responses, which in turn amplify pain perception. The default network is envisioned as serving the function of representing emotion concepts, where this capacity may be underdeveloped in those with early adversity—leading to poor regulation of chronic negative affect. The amygdala and salience network are implicated in drawing attention toward (and amplifying the perception of) somatic pain. Interactions between cortical-basal ganglia loops and the midbrain dopamine system are implicated in reinforcement/habit learning, and may reinforce a habit of attention toward pain when this reliably reduced intensity/awareness of affective distress. The gray dashed arrows emanating from the "Tissue damage" box and "Self-injury" box signify that these are facilitating but not essential to the proposed mechanisms. Color image is available only in online version (www.psychosomaticmedicine.org).

and that such processes remain poorly regulated by default network conceptualization processes (i.e., due to lack of recognition of the emotional nature of resulting afferent somatic/visceral signals) that normally allow recognition/understanding of that negative affect and its causes. It also seems plausible to suggest that self-inflicted somatic pain activates the amygdala and salience network and diverts attention away from social sources of negative affect—leading to temporary reductions in overall felt distress. Finally, the basal ganglia (and their interactions with the other neural systems described previously) may mediate the resulting negative reinforcement/habitization of attention to somatic pain and of self-injurious actions.

DISCUSSION

The purpose of this article has been to propose a mechanistic explanation for the association between early adversity and chronic pain. The core observations upon which our proposed mechanistic explanation is based are that patients with early adversity often seem to have deficits in their capacity to understand their own emotions and have been shown to benefit, with reduced pain, from better attending to, understanding, and expressing distressing emotions unrelated to pain. These core observations came from individual case studies of patients with chronic pain and early adversity (49–51) and from randomized clinical trials of patients with painful functional somatic syndromes (70,71) who not uncommonly experienced early adversity (117). They provided the foundation for the Biased Competition hypothesis, which states that in light of these deficits, attention to physical pain attenuates the experienced intensity of even more painful emotional experiences (or may in some cases prevent conscious recognition/awareness of this affective distress altogether). Through repeated reinforcement, this pattern of attention allocation then becomes habitual and promotes both greater awareness of pain and reduced awareness/understanding of emotion in those with early adversity.

Should the proposed psychological, neurobiological, and peripheral physiological mechanisms be established in future research, they create the opportunity for new primary and secondary preventative interventions for chronic pain, such as those implemented by Lumley and colleagues (70,71). Moreover, given that the association between early adversity and poor outcomes in a variety of health domains is also well established (5), and yet the reasons for this more general association are also poorly understood (118), insights from this model regarding the peripheral consequences of deficits in the conceptualization and regulation of affective responses may also potentially be applicable in other health contexts associated with early adversity.

Given the current state of knowledge, however, it is important to place the Biased Competition hypothesis in context. The actual prevalence of the phenomenon we describe here is not well established. The prevalence of chronic pain in the general population is approximately 19% (1), the prevalence of early adversity is estimated to be as high as 40% (119), and by one estimate early adversity increases the likelihood of pain in adulthood relative to those without early adversity with an effect size of 0.33 (6). Thus, the proposed mechanism can account for only a subset of chronic pain patients.

It is also important to consider that deficits in emotion concept representation due to early adversity discussed previously may be attenuated by individual differences in the personal circumstances of patients. For example, other family members, such as siblings or grandparents, or other people outside the family, such as friends, teachers or coaches, could provide support and empathy and opportunities to learn to understand emotions. Such factors could provide a new window into the assessment of resilience factors and would help explain why many people do not have adverse mental or physical health consequences from early adversity.

It should also be considered that in some patients with severe chronic pain, the impairment in emotional awareness could be caused by the pain itself. A potential explanation for this is that the medial prefrontal cortex, a key node in the default network involved in conceptualization of affective responses (109,120,121), tends to vary in its activity inversely with peripheral physiological arousal (122,123). Such arousal could occur by virtue of attention to social sources of emotional distress or could occur because of the arousal associated with the pain itself. However, when considering evidence that the capacity for concept-level representation of emotion requires adequate mirroring and empathy in childhood (which is deficient in many survivors of early adversity (17,28)), we believe that in many cases of chronic pain associated with early adversity, the emotion conceptualization deficit comes first. That said, this does not preclude the possibility that the reverse process could make it even more difficult for patients to improve their functioning in this area. The combination is more likely when the basic capacity for emotion conceptualization (and for other related mentalization processes) is more fragile.

It is noteworthy that the perspective offered here is consistent with current models of the transition from acute to chronic pain. It is widely recognized that mechanisms in the central nervous system, such as central sensitization due to chronic nociceptive stimulation and/or a deficit in the descending inhibition of nociceptive pathways, are both associated with the transition to chronic pain (124–126). There is also neuroimaging evidence that chronic pain has a stronger affective component in the central nervous system than acute pain (127). The Biased Competition hypothesis highlights an implicit reinforcement-based contribution to the persistent attentional focus on the experience of physical pain, as ostensibly unwanted as it is, that may not be appreciated by patients or clinicians. This attentional focus on pain could play a role in central sensitization and may be particularly relevant given the growing body of evidence (reviewed by Garcia-Larrea and Bastuji (128)) for unconscious nociceptive processing and for the role of attentional mechanisms in moderating whether unconscious pain representations become consciously accessible (i.e., supporting the same neurocognitive models of conscious access described previously in relation to emotion (14)). The idea that implicit reinforcement processes play a role in the transition from acute to chronic pain is also consistent with previous work that has found that differences in corticostriatal functional/structural connectivity (implicated in both cognitive-emotional and reinforcement processes) predict this transition (125,126).

IMPLICATIONS FOR RESEARCH

The Biased Competition hypothesis fundamentally involves amplification of physical pain by affective responses that are not experienced/recognized as such. We have previously discussed the neural basis of various phenomena that could be referred to as "unconscious emotion" and in that context discussed how it

could interact with physical pain (76,77,129). One example could be the potential role of implicit affective responses (generated by limbic network structures) in amplifying ascending nociceptive signals centrally (i.e., in addition to the peripheral inflammatory amplification mechanisms discussed previously (100,101,130)). To investigate this further in the context of neuroimaging, one would ideally need to implement a paradigm in which affective responses that are not consciously experienced/recognized interact with experimentally induced pain. Such a paradigm has not to our knowledge been investigated yet, although promising methods to operationalize unconscious emotion have been developed (77,131,132). This type of paradigm could also build on previous work that has shown that classical conditioning processes occurring outside of awareness can promote hyperalgesia (133,134).

Such research would come closer to the phenomena we describe here if it involved survivors of early adversity and affective stimuli that pertained to emotionally charged areas in the patient's life that were difficult to process, such as interpersonal contexts involving separation or loss (135). One possible design would be to expose participants to spoken words or statements that were emotionally painful while giving them the opportunity to self-administer a mildly painful shock or not. Because auditory stimuli would be more difficult to avoid than (for example) visual stimuli (i.e., where one could simply close their eyes), if the auditory stimuli were sufficiently painful emotionally, one would predict that the individual would self-administer physical pain to avoid/minimize attention to the associated affective distress and its interpersonal source/meaning (i.e., where the intensity of shocks could be controlled by the individual). This type of design could build on previous work that has shown that individuals will choose to self-administer shocks rather than sit alone with their thoughts (136,137). Based on the Biased Competition model, one might predict that this effect would be stronger (e.g., participants choosing more frequent/intense shocks) in those with early adversity and/or in those currently feeling intense distress and that this behavior would be more strongly reinforced over time in such individuals. In a neuroimaging context, one would predict that a focus on identifying what emotion one is feeling would activate the default network whereas a focus on physical pain would activate pain matrix regions (and somatosensory cortex in particular; (43,108,138–140)).

There are several additional areas where future research is needed. The EAET treatment method by Lumley and colleagues (70,71), which has been demonstrated to reduce pain in fibromyalgia and irritable bowel syndrome, could provide a very useful context for additional mechanistic research. In addition to evaluating emotional awareness deficits and their differential modifiability by psychotherapy, measures of early adversity should be added as a moderating variable. This treatment would also provide a context for functional brain imaging research, both using resting state and task-based approaches as just described, aimed at biased competition, both before and after treatment. Within that context, ambulatory measures of inflammation and physiological responses indicative of emotional arousal (141), implicit measures of emotional responding (142), self-reports of pain, and emotion regulation and coping skills (especially avoidance versus emotional approach coping; (143,144)) would all be expected to change in predictable ways as a function of treatment progress.

Research in laboratory animals could be used to evaluate whether exposure to early adversity amplifies the intensity of distress in social circumstances such that exposed, but not nonexposed, animals would choose to self-administer physical pain rather than continue exposure to the social source of distress (and evaluate whether this behavior becomes reinforced over time). Although self-reports of experienced distress or pain would not be possible, this would provide greater opportunities for examining molecular, genetic, neural, and pharmacological mechanisms.

CONCLUSIONS

Our intent in this article has been to provide a conceptual framework explaining the possible role of poor emotion concept learning (poor emotional awareness) and implicit attention-based adaptations/strategies for dealing with the emotional pain of early adversity that could contribute to and amplify chronic physical pain. The research proposed here could ultimately lead to additional methods of intervention that could reduce the prevalence of chronic pain.

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