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What Lies Beneath: Some Neurological Correlates of Nonsuicidal Self-injury

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Abstract: Generally regarded as being deliberate damage to the body in the absence of intent to die, and for purposes that are not socially sanctioned, nonsuicidal self-injury (NSSI) has been a subject of increasing concern in the area of public health over the past 20 years. Clinical and research evidence indicates that onset typically begins in early adolescence, although the data is less clear on the incidence of self-harm in the adolescent population in general. There is a general understanding, however, that one major function of NSSI is the regulation of emotions and managing distress, while self-punishment has also been reported as a further significant motive to self-harm. Whereas social and psychological factors contributing to risk for developing NSSI are now relatively well understood, neurological mechanisms involved in self-harming behaviours are less so. It is considered that an understanding of the underlying neural mechanisms involved in NSSI may provide a better explanation for the urges to self-harm, the role these mechanisms play in regulating emotions, and reasons why stopping the behaviour can be so difficult. This paper provides a brief summary of the current risk factors associated with NSSI, then reviews aspects of neurological correlates of self-harming behaviours with an emphasis on autonomic nervous system functioning.

Keywords: Nonsuicidal Self-injury, Autonomic Nervous System, Endogenous Opioids, Dissociation, Emotional Regulation

1. Introduction

Nonsuicidal self-injury (NSSI) is generally regarded in the literature as being the deliberate damage to the body in the absence of intent to die, and for purposes that are not socially sanctioned [1]. Although commonly associated with the term "cutting", the majority of young people engaged in repeated self-injury also report using multiple methods, for example, scratching, hitting, and burning [2, 3]. In addition, NSSI is associated with a range of mental health conditions, including major depressive disorder, anxiety disorders, PTSD, and eating disorders [2, 4]. NSSI is also strongly comorbid with Borderline Personality Disorder (BPD), however several authors have stressed the importance of not regarding the two as being synonymous [1, 4, 5].

Increasing interest in NSSI amongst researchers and clinicians over the past 20 years has tended to correspond with a reported increase in self-harming behaviours amongst both clinical and community populations of adolescents. From North American data Klonsky and colleagues [6] reported that onset typically occurred around 13 to 14 years of age, and that lifetime rates in adolescent populations were between 15-20%. More recent data [7] reported similar rates of self-injuring behaviours amongst non-clinical samples of adolescents (17%), but with clinical samples being significantly higher (30-45%).

A recent UK study led by Sally McManus [8] analysed data for presentations to hospital emergency departments (EDs) following NSSI from 2000 to 2014. The authors reported an increase in presentations in both sexes, but most notably in females aged 16 to 24 years, where the prevalence of self-harming behaviours increased from 6.5% in 2000 to 19.7% in 2014. A similar increase in incidence was reported in an Australian study, where Hiscock and colleagues [9] found that between 2008 and 2015 mental health presentations to Victorian EDs by children and adolescents increased by 6.5% per year, and that self-harm accounted for some 22.5% of these presentations.

With respect to the non-clinical population of adolescents who self-harm, its relatively low lethality generally resulted

in few presentations to medical services, making it difficult to accurately discern the exact prevalence of NSSI in this population [10]. Results from a survey conducted in Oxford [11] found that approximately 12% of adolescents had reported to hospital following acts of self-harm, but that "the largest proportion of acts of self-harm, possibly amounting to 80-90%, is invisible to professionals" (p. 434). Taken together, contemporary prevalence rates nevertheless pose major implications for public health. McManus [8], for example, expressed the concern that individuals who start to self-harm when young might adopt the behaviour as a long-term coping strategy, with the additional risk that self-harm could become normalized for young people.

There is also the finding that NSSI is the strongest known predictor of later suicide, even more so than other known risk factors such as suicidal ideation and depression [12]. Results from a long-term (M=11.4 years) follow-up study of self-harmers from Oxford [13] found a suicide rate of 2.6%, although a further 7.7% had also died from other causes. Unsurprisingly, repetition of deliberate self-harm significantly increased the risk of eventual suicide in both males and females. Whitlock [3], however, considered that the relationship between suicide and NSSI remained somewhat ambiguous, as in the vast majority of cases there was general agreement that NSSI was utilized to temporarily relieve distress rather than to signal an intention to end one's life. When it came to suicide risk, Klonsky and colleagues [6] nevertheless viewed NSSI as presenting double trouble, in that it increased the risk for both suicidal ideation and the ability to act on the ideation. Indeed, McManus [8] reported that, in addition to increasing prevalence of NSSI amongst adolescents, since 2010 there has also been an upward trend in suicides among people aged under 20 years in the UK.

Ongoing research has provided valuable demographic data on populations of adolescents vulnerable to engaging in self-harming behaviours, however there is comparatively little information on those underlying neural mechanisms that play a role in pain and affect regulation, and that may contribute to the maintenance of NSSI. This paper provides a brief summary of what is known about the functions NSSI may serve, as well as antecedents to NSSI, then reviews aspects of neurological correlates of self-harming behaviours, with an emphasis on autonomic nervous system functioning.

2. Functions of NSSI

In addition to research investigating demographic data, attention had also been turning to understanding the motivation to self-harm. As Hooley and Fox [14] pointed out, most people are deterred by the idea of pain, especially self-inflicted pain. To engage in NSSI, therefore, required that the protective pain barrier first be overcome, particularly in instances of repeated self-harm. However, even given the possibility of altered pain perception among people who engaged in NSSI [15], this still did not explain the motivation to self-harm in the first place. Early clues were provided by Nock and Prinstein [16] who interviewed 108 adolescent psychiatric inpatients referred because of self-harming behaviour. These authors concluded that these adolescents engaged in their behaviours for "automatic reinforcement", which most commonly centred around reducing psychological distress and cognitive avoidance, i.e., refocusing attention away from negative thoughts and feelings.

There is now considerable academic literature examining factors that might explain the contemporary phenomenon of self-harm, including both theoretical approaches and empirical studies [7, 17, 18]. The function of NSSI as a means of regulating emotion and managing distress has received the strongest empirical support, with evidence suggesting that individuals engaging in self-harming behaviours are characterized by increased emotional reactivity, interpersonal sensitivity, and interpersonal problems. You and colleagues [7] also endorsed emotional reactivity – referring to the extent to which an individual experienced emotions strongly or intensely, and for a prolonged period of time before returning to baseline levels of arousal – as well as a lack of effective emotional regulation strategies, as being the most pronounced features of emotional dysregulation.

The literature therefore indicates that, with respect to stress management, high emotional reactivity and a diminished capacity to tolerate distress, coupled with poor effective emotional regulation strategies, increased the risk of using self-harming behaviours as a means of reducing intense negative emotions. As escape from negative emotions is reinforcing, You and colleagues [7] argued that when experiencing negative emotions individuals may engage in behaviours that provide immediate relief, at the expense of developing longer-term, more adaptive regulatory coping strategies. In support, there is evidence demonstrating that engagement in self-harming behaviours allows an individual to avoid unwanted emotions, with the subsequent relief and reduction in emotional arousal that NSSI brings, reinforcing the behaviour [19].

In addition to NSSI being a means of regulating distressing emotions, a cognitive function has also been investigated. Adolescents who engaged in NSSI, for example, have reported that the behaviour distracted them from unwanted thoughts [17], while there is also evidence indicating that individuals who self-harm showed a preferential attention to negative stimuli, and tended to neglect or diminish positive stimuli. The consequent negative thoughts, particularly if repetitive (i.e., ruminations), thus tended to amplify the negative meaning ascribed to stimuli, subsequently increasing distressing emotions [19]. In support, McKenzie and Gross [20] had previously reported on studies relying on retrospective recall that indicated the presence of high-activation negative emotions, such as anxiety and anger, as being direct precursors to acts of NSSI, and that these negative emotions were significantly decreased following NSSI, with the exception of an increase in shame and guilt.

Shame and guilt lead directly to a further function of NSSI, that is, the need for self-punishment. Amongst

samples of US college students, for example, 32-43% of self-injurers reported self-punishment as a motive for their behaviour [21]. People who engaged in NSSI have been found to hold negative self-views as well as high levels of self-criticism, and self-criticism is significantly associated with endorsing self-punishment as a motivator for engaging in self-harming behaviours [14, 20]. Hooley and Fox [14] also hypothesised a relationship between self-criticism and pain, in that pain is likely perceived as being integral to punishment. Further, these authors posited that people who hold core beliefs about being bad or flawed may have less resistance to the idea of NSSI which, in turn, is more likely to remove a potential barrier to self-inflicted pain. In addition, reward expectations from engaging in an identity-affirming behaviour – punishing the bad self – are seen as likely playing a key role.

3. Antecedents to NSSI

Although there are still missing pieces in the puzzle of creating a stringent neurologically-based understanding of NSSI, it can nevertheless be argued that an understanding of the underlying neural mechanisms involved in self-harming behaviours allows for a better explanation of the urges leading to self-harm, the role self-harm plays for emotional regulation, and reasons why stopping the behaviour can be so difficult [22]. However, there remains the preliminary question of what factors may precipitate an urge to self-harm in the first place. A range of adverse childhood experiences has been advanced as predisposing engagement in NSSI [5, 23], while Nock [24] considered child maltreatment to be a distal factor of vulnerability to self-harm. This should not be regarded as deterministic, however, as child maltreatment can result in a range of psychiatric co-morbidities, and not necessarily NSSI.

Nevertheless, child maltreatment (abuse/neglect) increases the risk of altering autonomic nervous system (ANS) functioning, resulting in both sympathetic (SNS) and parasympathetic (PNS) based autonomic responses becoming sensitized, that is, altering the homeostasis of the systems mediating these responses. This, then, leaves a child vulnerable to developing persisting hyperarousal or dissociative related systems [25], together with a chronically dysregulated ANS that reacts *as if* the environment is threatening. A history of child maltreatment can therefore result in a brain focused on survival, i.e., with internal functioning focused on anticipating, preventing, or protecting against danger, rather than on an open engagement with the environment [26].

Consequent high ANS reactivity, together with the negative emotions generated by elevated ANS functioning, are likely proximal factors triggering self-harming behaviours. Trauma symptoms that generate elevated ANS functioning have been advanced as internal factors mediating the relationship between prior abusive experiences and NSSI [21, 27]. Given reports of the function of NSSI to relieve negative emotions, unpleasant or intrusive thoughts or feelings, and to release emotional pain [18, 20], Smith and colleagues argued that trauma symptoms, particularly those involving re-experiencing and hyperarousal, are sufficient to trigger self-harming behaviours.

Self-harming behaviours can also be triggered by factors in the social environment, such as relationship difficulties, feeling socially isolated, or help-seeking [18, 28]. Difficulties involving engaging adaptively with the social environment can be explained, at least in part, by faulty neuroception [29]. Defined as a neural process that enables humans to distinguish safe from dangerous social contexts, neuroception explains that in normally-regulated individuals the central nervous system (CNS) evaluates risk and matches neurophysiological states to the actual risk in the environment. When the environment has been appraised as being safe, for example, the defensive limbic structures are inhibited, enabling social engagement. Where defensive responses have become sensitized, however, the CNS may appraise the environment as being dangerous even when it is safe. This mismatch results in SNS-mediated states that support "fight or flight" or active avoidant responses, but not social engagement behaviours which, in turn, can impair the capacity to develop more adaptive problem-solving strategies. The result is the utilization of behaviours that are essentially reactive and directed towards providing immediate relief from emotional and psychological distress.

4. Autonomic Responses to Perceived Threat

Although acknowledging many definitions of stress consequent to exposure to threat, Stephen Porges [29] derived his concept of stress from the function of the ANS. The ANS has two branches, the sympathetic (SNS), and parasympathetic (PNS), whose responses are coordinated to meet shifts in both internal and external demands. In the absence of external challenges (e.g., perceived threat to self) the PNS optimizes the function of the internal viscera, while the SNS responds to external demands. In Porges' model the PNS essentially mediates homeostasis, whereas stress may be defined as the autonomic state that reflects a disruption of homeostasis through the subjugation of internal needs in response to external challenges.

Broadly, the triggering of anxiety in response to a perceived threat to self is initiated by the amygdala, located in the limbic region. The amygdala, in turn, triggers the hypothalamus which initiates an increase in SNS activity, while at the same time activating the locus coeruleus (LC). The release of increased amounts of norepinephrine (NE) by the LC directly assists in generating the total body response to threat. This up-regulation of the SNS results in an increase in heart rate, blood pressure, and respiration, as well as an increase in muscle tone and a sense of hypervigilance, and characterizes the classic "fight or flight" stress response [30, 31].

Under normal circumstances, once a threat has subsided,

the stress response is countered by hippocampal activation of the hypothalamic-pituitary-adrenal (HPA) axis resulting in the release of the hormone cortisol. Cortisol signals to the hypothalamus via a feedback loop that the corticotrophin-releasing hormone (CRH) no longer needs to be released, thus allowing the brain to begin the process of down-regulation. However, repeated activation of the stress response can result in neural circuits in the SNS becoming sensitized, with the threshold for SNS activation being reset and more easily triggered by external stressors. One consequence of this sensitized stress response is the development of an ongoing state of increased SNS arousal with its attendant hypervigilance, startle response, increased tension and behavioural irritability, together with poor attentional and sleep patterns [30].

Should the SNS mediated "fight or flight" response prove ineffective in overcoming the threat a second response, mediated by the PNS, may be activated. This response is generally activated when the threat is seen as being overwhelming and/or there is no chance of escape, and triggers the progress into a state of collapse and helplessness. PNS reactions characterize this phase. Vagal tone increases, leading to decreased heart rate, blood pressure, and a slowing of respiration, occasionally leading to stress fainting (syncope) [30]. Light-headedness and numbing are also commonly experienced. Whereas a regulated PNS helps restore internal balance, sleep, digestion, and immune system functioning, a dysregulated PNS, particularly when the dysregulation is chronic, can contribute to depression and fatigue [29].

This phase is characterised by a number of features that may have relevance for self-harming behaviours. First is the release of opiate peptides that act on opioid receptors throughout the CNS to produce a variety of effects, including pain relief, reduced anxiety, and enhanced mood. Data also suggests that endogenous opioids are involved in the down-regulation of the physiological aspects of emotion, including blood pressure and heart rate [32]. Endogenous opioids - β-endorphins and enkephalins in particular - diminish stress-induced autonomic and neuro-endocrine responses, thereby blunting the distressing affective components of pain, and may diminish the impact of stress by attenuating an array of physiological functions, including intense negative affective states. The widespread distribution of enkephalins throughout the limbic system, for example, is consistent with a direct role in the modulation of the stress response [32, 33].

Second, dissociative reactions dominate this phase. Partial dissociation involves a telescoping of the attentional field to concentrate on a narrow range of experience and the concomitant exclusion of other stimuli (both internal and external) from awareness and, to some extent, from accessibility [34]. More severe dissociation gives rise to a range of subjective experiences such as depersonalization, derealization, and emotional numbing. The activation of the dissociative response in the face of repeated threat can also lead to this response becoming sensitized, resulting in the development of a range of dissociative-related symptoms, such as withdrawal and detachment, as well as feelings of depersonalization and depression, becoming prominent in day-to-day life [25].

The specific symptoms that may develop following exposure to perceived threat can vary depending upon the nature, frequency, and intensity of the threat, together with the presence of attenuating factors such as family and/or community supports. Perry [25] noted from clinical observations that the capacity to dissociate in the face of high levels of threat appeared to be a differentially available response, that is, some people dissociate early, while others only in a state of complete terror. However, whereas the dissociative response appeared to be more commonly associated with females, clinical evidence suggested that responses to threat were commonly an admixture of both arousal and dissociation. Nevertheless, while both high autonomic arousal and dissociation may be acutely adaptive in the face of overwhelming threat, the consequent symptoms of both responses, particularly when chronic, become un-adaptive in a non-threat environment.

5. Neurological Correlates of NSSI

Referring to maltreated children, Stein and Kendall [31] wrote that some children "discover a seemingly paradoxical way to handle anxiety. By doing something physically traumatising or by provoking interpersonal conflicts, they can precipitate an autonomic nervous system crisis. The escalating intensity of arousal eventually triggers the brain's natural calming mechanisms including a sudden release of cortisol and endogenous opioids, inducing a calm, sometimes hypnotic-like or numbed state" (p. 114).

These authors are describing the switch, under induced stress, from dominant SNS functioning to PNS functioning. There is some research evidence [35] indicating that adolescents who self-harm demonstrated reduced vagally-mediated heart rate variability at rest (i.e., lower PNS activity), compared to age-matched controls, suggesting that adolescents engaging in self-harming behaviours are characterized by an ANS profile reflecting greater SNS dominance. Given reports of self-harm producing relief from emotional distress, and even being referred to as a positive experience providing a sense of gratification [18], the empirical evidence suggests that engaging in self-harming behaviour, such as cutting or burning, is sufficient to precipitate an autonomic crisis and trigger a switch to PNS functioning as described by Stein and Kendall [31].

5.1. NSSI and Dissociation

Associated with increased vagal tone, decreased heart rate and a slowing of respiration, PNS activation prompts passive coping strategies, such as physical and emotional disengagement, or dissociation. A review of 19 studies investigating the relationship between NSSI and dissociation in adolescents [10] found a positive association between the two in 17 of the studies. The reviewers noted difficulties with respect to differences in conceptualizing and measuring

dissociation between the various studies, but nevertheless concluded that the data confirmed a relationship between self-harming behaviours and dissociation, and additionally that the severity of dissociative experiences pointed to the severity and frequency of acts of NSSI.

Cernis and colleagues [10] also pointed out that the cross-sectional nature of the studies made it difficult to confidently predict causality, that is, whether engaging in self-harming behaviours was for the purpose of inducing dissociation or escaping from it. As engagement in self-harm typically occurs at a peak of negative affect it seemed a reasonable presumption that the function of NSSI was to trigger a PNS response that induced dissociation, manifesting in emotional numbness and feelings of relief. Indeed, McKenzie and Gross [20], for example, found that individuals reported a range of high-activation negative emotions prior to acts of NSSI, and that most of the emotional change accompanying NSSI occurred from high negative to low negative activation. These authors added that any positive affect that may have emerged through self-harm was likely via increases in low negative activation, such as calm, rather than via changes in positive activation.

Edmonson and colleagues [18] similarly reported that the majority of studies reviewed by them endorsed self-harm as a means of calming the self, relieving emotional pain, and producing a feeling of numbness when emotions became too strong. These findings are consistent with the function of self-harm being dissociation-inducing in order to down-regulate distressing negative affect. However, Edmonson and colleagues also found that some 20% of studies reported self-harm having a stimulating effect, for example, generating feelings and a sense of being alive, and regaining a sense of self. These findings support engaging in acts of self-harm as having the function of escaping a dissociative state by triggering SNS arousal in order to up-regulate affect.

Whereas hyper-arousal, mediated by increased SNS activity, has been well documented, particularly in relation to the effects of trauma [25, 26] the clinical manifestations of hypo-arousal (PNS dorsal-vagal-mediated states) have received less attention. Under normal circumstances, that is, in the absence of external challenges, the PNS optimizes the function of internal states and promotes physiological stability [29]. However, some individuals with a history of child maltreatment may develop a default PNS response to threat, resulting in a chronic hypo-aroused state. Symptoms typically include a lack of energy, flat affect, and feeling numb, “empty”, or “dead” [36]. The state of emotional “emptiness”, conceptualized as experiencing low positive affect, and encompassing descriptions of lack of feeling/sensation, has been found, together with dissociation, to be positively associated with NSSI in adolescents [37]. Results of this study indicated that the state of low arousal set the stage for engaging in acts of self-harm as a means of increasing the experience of feeling/sensation, and are consistent with the findings of Edmonson and colleagues [18] that some acts of self-harm have the function of escaping a dissociative state.

5.2. Role of Endogenous Opioids

The endogenous opioids are widely distributed throughout the central and peripheral nervous systems and are best recognized for their analgesic properties, as opioid analgesia involves a blunting of the negative affective component of pain [33]. In particular, β-endorphins and enkephalins, those opioids with a high affinity for Mu and δ-opioid receptors, are implicated in the modulation of pain, as well as reward and emotions [38]. Induced stress on the ANS, for example as a consequence of NSSI, activates the locus coeruleus-norepinephrine (LC-NE) system in parallel with the HPA axis – specifically the release of corticotropin-releasing hormone (CRH) – with this activation playing an integral role in initiating and maintaining arousal [39]. The opposing influences of CRH and endogenous opioids on LC activity must be finely balanced in order for the stress system to maintain homeostasis.

There is evidence indicating that repeated stress, for example, through engaging in repeated episodes of self-harming behaviour, may tip the balance towards opioid regulation of the stress response [38, 39]. As reported by Valentino and Van Bockstaele [39], in contrast to acute stress where CRH excitation predominates and opioids act to temper this, with repeated stress the influence of CRH is diminished and Mu function in the LC is enhanced. A dysfunctional bias towards opioid neuronal regulation – increased opioid influence - may render individuals tolerant to opioid analgesia, that is, may lead to an increase in pain tolerance. Although an increase in pain tolerance may enable an individual to more readily engage in NSSI [14, 38], this in itself does not provide a sufficient explanation for the motivation to self-harm. It is possible, under conditions of increased opioid influence, that the induced stress of self-harm on the ANS engaged in response to high levels of negative affect, triggers a switch to PNS functioning and an opioid-mediated sense of calm consistent with the dissociation-inducing function of NSSI [10, 20].

This picture is far from clear, however, as research has reported finding both elevated and reduced levels of endogenous opioids in individuals engaging in NSSI [35, 38, 39]. Whereas elevated levels of endogenous opioids protect against the negative consequences of LC hyperactivity [33], reduced levels are hypothesized to bias regulation towards CRH-mediated excitation and hindering recovery of neuronal activity after stress, expressed as an exaggerated and more enduring activation of the LC-NE system [39]. Increased autonomic arousal associated with elevated LC-NE activity may therefore function as a proximal stimulus to self-harm in an attempt to down-regulate stressful negative affect. Kaess and colleagues [35] made the point that lower levels of β-endorphins and enkephalins should result in reduced activity at Mu and δ-reception sites, leading to predictions of increased pain perception sensitivity. These authors hypothesized, however, that while stress is associated with increased opioid activity, low levels of endogenous opioids may result

in Mu and δ-reception sites becoming sensitive to opioids, which could potentially create a context in which pain analgesia may be reported. It was nevertheless acknowledged that it was difficult to integrate such findings into a coherent theoretical framework at the present time [35].

The picture is further complicated when considering NSSI for the purposes of self-punishment. Pain sensitivity is a complex construct, driven by psychological and biological mechanisms [35]. A meta-analysis of 32 studies, for example, found that individuals engaging in NSSI reported higher pain thresholds and greater pain tolerance when compared to healthy controls [15]. These authors also found a tendency for elevated pain endurance, that is, a willingness to endure pain over time after the onset of pain; all of which suggests the presence of elevated opioid levels resulting in an increase in opioid analgesia and pain tolerance [38]. However, Hooley and Fox [14] remind us that people who engaged in NSSI tended to report higher levels of self-criticism, as well as negative self-views and self-dissatisfaction. Moreover, self-criticism was significantly associated with endorsing self-punishment as a motivation for engaging in NSSI.

Thus, with respect to self-punishment at least, whatever role endogenous opioids may play in the regulation of pain, there is likely a significant psychological mechanism involved in the motivation to self-harm. Hooley and Fox [14] considered that a cognitive style that involved high levels of self-criticism or self-hatred may be important for understanding NSSI, essentially because self-criticism or self-hatred removes a potential barrier to self-injury. It may therefore not be so much a matter of greater pain tolerance for those who self-injure in order to self-punish, but as the finding of greater pain endurance suggests, pain may be perceived as something that is deserved for those who feel they are bad or worthless, so are therefore willing to endure pain for longer. Although there may be an affective benefit for those who self-harm in order to self-punish – and here Hooley and Fox have reported that mood improvement via self-punishment appeared specific to those with elevated self-criticism, shame, or guilt – NSSI may nevertheless serve a different function from those who engage in self-harm in order to regulate their affect by seeking to either induce or escape from a dissociative state.

6. Summary

NSSI in adolescent populations has become a subject of increasing concern over the past 20 years, with contemporary prevalence rates posing major implications for public health. There is a general consensus that a major function of NSSI is the regulation of strong negative affect, with the hypothesis advanced [7] that engaging in behaviours that provided immediate relief from negative emotions came at the expense of developing longer-term, more adaptive regulatory coping strategies which, in turn, tended to make NSSI a default strategy when experiencing emotional distress. In similar vein Hasking and colleagues [19] posited that if an individual came to expect NSSI to reduce emotional distress they would be more likely to self-injure when feeling distressed, with the subsequent reduction in arousal likely reinforcing and maintaining the behaviour.

There is evidence to suggest that individuals who self-harm appear to show an altered stress response [22], although as this author has pointed out, due to a lack of longitudinal studies it is difficult to say with certainty whether such alterations to the stress response systems occur as a result of engaging in self-harming behaviours, or whether these alterations existed as a prior vulnerability, for example, as a consequence of earlier adverse childhood experiences. Either way, the clinical impact of stress derives from the ability of repeated or chronic stressors to produce enduring dysfunctions in the stress response systems such that they become over-active, or are not counter-regulated [33]. One consequence of an altered stress response is reflected in an up-regulated SNS resulting in a range of stress-based symptoms characteristic of the classic "fight or flight" response [30]. It is hypothesized that additional stress on the ANS as a result of acts of self-harm is sufficient to trigger a switch to PNS functioning, prompting passive coping strategies and emotional and cognitive calmness, which is consistent with the dissociation-inducing function of NSSI [10, 20].

The link between dissociation and NSSI is complex however, and at times conflicting, as there is also evidence supporting a dissociation-avoiding function associated with self-harming behaviours [18]. In these circumstances it is hypothesized that chronic stressors have resulted in a sensitized PNS, reflected in a hypo-aroused state of general passivity, flat affect, and feelings of emotional emptiness. Engaging in self-harming behaviours is now seen as having the function of triggering SNS arousal in order to up-regulate affect and increase a sense of feeling/sensation [36, 37].

The role of endogenous opioids is equally complex. Having a major role in blunting the negative affective component of pain, endogenous opioids are released as part of the stress response to intense sensations, but their effects on the stress response system are kept in balance by the opposing influences of CRH on the LC-NE system [33]. Chronic stress, however, can produce enduring modifications in neural circuits that can result in opioid-mediated inhibition, and may render individuals tolerant to opioid analgesia [39]. One implication is that over time individuals may need to self-harm more, or more severely, in order to achieve the same effects. Less easily understood is the finding that there can be both elevated as well as reduced levels of endogenous opioids in individuals engaging in NSSI, as well as a lack of critical knowledge on what influences these differing levels of opioids may have on real-world NSSI episodes [35, 38]. Thus, while endogenous opioids have a role to play in pain modulation, the mechanism(s) by which they act in relation to NSSI are yet to be fully understood.

Finally, Hasking and colleagues [19] remind us that the motivation to self-injure, and the many processes that precede the behaviour, do not happen in a vacuum but in a complex interplay of external and internal cognitive and

emotional experiences. McKenzie and Gross [20] had similarly concluded that it was unlikely there was one single mechanism underlying the diverse phenomena gathered together under the umbrella term of NSSI, and that a particular challenge for future research would be to more precisely define underlying psychological and physiological mechanisms. More recently Kaess and colleagues [35] also stressed the importance of ongoing research in this area, including that with a stronger focus on the real-world clinical implications of the neurobiology involved in self-harming behaviours. For example, these authors stressed the potential value of longitudinal studies. Much is now known about how childhood adversity can lead to adverse biological alterations, but there are no longitudinal studies to date investigating biological factors x environment interactions over time that could shed light on important questions of risk and resilience with regards to NSSI. Keeping in mind the fact that accumulating data show that clinically-relevant NSSI in particular is generally associated with high levels of comorbidity, including mood disorders, anxiety disorders, PTSD, eating disorders, and substance abuse [2, 4, 35], from a treatment perspective it may be most efficacious to place self-harming behaviours within the context of the whole clinical picture, and to plan treatment approaches accordingly.

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