

2.4 | Supplemental review of plausible “stress response” mechanisms

In this supplemental review, we identify plausible “stress response” mechanisms that potentially underlie the stress process theoretical expectations (e.g., *stress deficit*; *stress amplification*) reviewed in our manuscript. Though some readers might find this supplementary review of helpful for conditionally interpreting results of our descriptive analyses, we present it primarily with the hopes that it encourages future theoretical elaborations, precise predictions about mechanisms, and severe tests of those predictions.

2.4.1 | Stress deficit: “Fight or flight” mechanisms

Despite differences in emphasis and explanandum, the core arguments of the stress process model and general strain theory overlap substantially (cf. Van Gundy, 2002; Slocum, 2010). Hence, for simplicity, both are collectively referred to hereafter as stress process theories. To summarize, stress process theories posit that exposure to stressors can result in subjective psychological experiences with stress and subsequent detrimental emotional and behavioral outcomes (*stress deficit*). These theories also identify chronic stress exposure as particularly problematic, and they suggest that individuals in disadvantaged groups or social locations might be especially vulnerable to stressors, stress, and their consequences (*stress clustering and stress amplification*).

However, the compatibility of these stress process models with findings from a growing body of research on biophysiological mechanisms underlying human stress response appear at present to be developed more precisely for depression than for crime. That is, stressful events or circumstances, which are at the heart of these theories, are known to generally trigger a series of automatic processes in the human body collectively referred to as the “stress response system” (cf. Schwartz et al., 2023; Schwartz and Allen, 2024). Often colloquially described as “fight or flight” responses, these processes include the release of hormones such as epinephrine (adrenaline) and cortisol, which results in increased heart rate, blood pressure, and breathing, as well as sharpened senses, increased energy from the release of stored glucose, and suppressed immune system functioning. Together, these reactions trigger a hypervigilant state characterized by increased activation of the brain’s sensory network and initial suppression of higher order cognitive processing (e.g., executive functioning). These changes are then typically followed by the normalization of cognitive brain functions due to the slow effects of corticosteroids. (For detailed descriptions of neurobiological responses to acute stress, see Hermans et al., 2014; Van Oort et al., 2017).

The “high alert” state induced by acute stress is generally viewed as adaptive in that it “may support an optimal response to stressful situations and subsequent recovery in healthy individuals” (Van Oort et al., 2017: 293). However, acute stress may also trigger less optimal responses under some conditions. For example, if exaggerated, these processes might result in a continuous state of hypervigilance, such as that observed in individuals suffering from posttraumatic stress disorder. Moreover, the increased connectivity between the brain’s sensory network and “default mode network” (DMN) documented under acute stress conditions also resembles brain activation patterns seen in individuals with depressive symptoms, which may indicate the existence of distinct neuropsychological mechanisms linking acute stress and depression (Qin et al., 2009).

It is arguably less clear precisely how typical neurobiological stress response processes might increase the likelihood of aggressive or criminal behavior. Stress-induced DMN activation

may manifest in maladaptive patterns of depressive rumination rather than adaptive forms of reflective rumination (Hamilton, Chen, and Gotlib, 2013), which might in turn increase the likelihood of criminal coping. Stress-induced changes in DMN activation also appear to be accompanied by reductions in higher-order cognitive processing (cf., Qin et al., 2009; Hermans et al., 2014) as well as altered processing of emotional stimuli and rewards or pain (Van Oort et al., 2017). Together, these effects may trigger the negative emotional and conditional criminogenic coping processes outlined in general strain theory.

Yet, experimental research aimed at documenting how acute stress affects decision-making in specific tasks has generated mixed findings (e.g., Buchanan and Preston, 2014; Starcke and Brand, 2016; Porcelli and Delgado, 2017; Ethridge et al., 2020). At this point, experimental research seems to suggest that acute stress might affect immediate situational criminal intentions by affecting valuations of rewards and costs, increasing reward-seeking and risk-taking decisions, and shifting individuals from goal-directed to habit-based decision-making. Additionally, recent research on workplace stress and decision-making among police and correctional officers suggests that experiencing acutely stressful and uncertain life-or-death situations on the job may cause physiological stress responses that, in turn, may increase the likelihood of stress-induced aggressive or violent reactive responses (Baldwin et al., 2022; Schwartz and Allen, 2024).

2.4.2 | Stress amplification: Chronic stress exposure mechanisms

Another shared tenet of stress process theories is the expectation that chronically experiencing subjectively potent stressors is detrimental to individual wellbeing in the long-term. For example, Pearlin & colleagues (1981) suggest that individuals who experience subjectively salient and “tenacious” (i.e., chronic) stressors are especially at risk of suffering depressive symptoms and other internalizing problems. Similarly, Agnew (2001; 2013) posits that individuals who experience subjectively severe and chronic strains are especially likely to react with criminal coping.

While the biophysiological (e.g., “fight or flight”) mechanisms linking acute stressful events and conditions to depression and crime are somewhat clear, the exact mechanisms through which chronic stress exposure might cause depression and crime are less clear and likely more complex (cf. Juruena, Cleare, and Young, 2018; Mayer, Lopez-Duran, Sen, and Abelson, 2018; Tielbeek et al., 2018). One possibility is that chronic stress causes dysregulation of the hypothalamic-pituitary-adrenal (HPA) axis, or the biological stress-response system, which can manifest as hypersensitivity or an inability to effectively react, to subsequent stress. Put simply, under chronic stress, the HPA-axis presumably overstimulates the stress-response system and its associated “high alert” state. Though consequences are varied, such overstimulation presumably can result in “allostatic overload,” which is marked by energy depletion, a weakened immune system, neuronal damage in brain regions associated with stress response, and a general reduction in the body’s ability to effectively respond to and recover from stress (Juster, McEwen, and Lupien, 2010; McEwen, 2004; Miller, Chen, and Zhou, 2007). Theoretically, then, depression and crime might represent internalized and externalized overreactions to stress that are caused by a hypersensitivity to stressful stimuli stemming from chronic stress-induced HPA-axis dysregulation (Juruena 2014; Wells et al. 2017).

Early life and chronic stress have also been linked to other interrelated changes in brain structure, neural functioning, and epigenetic processes that may increase risks of depression and

aggressive or criminal behavior (Heim and Binder, 2012; Pechtel and Pizzagalli, 2011; McEwen et al., 2015; Sandi and Haller, 2015). Moreover, consistent with Agnew's (2006b) general strain theory, depression and crime might emerge from complex negative feedback loops involving individualized attempts to internally or externally cope with the fatigue, negative emotions, and psychophysiological toll of experiencing chronic stress (cf. Jackson and Knight, 2006; Jackson, Knight, and Rafferty, 2010).

2.4.3 | Amplification or desensitization in disadvantaged communities?

If stress-process theories are correct about chronic stress exposure increasing vulnerability to stress, then living in socioeconomically disadvantaged urban communities should amplify individuals' risks of experiencing depression and crime (*stress amplification*; cf. Agnew 1992: 60-61; 2001: 334; Thoits, 1995). After all, residents of such communities tend to be more chronically exposed to potent stressors such as poverty, physical decay, social disorder, and victimization (*stress clustering*). Seemingly consistent with these perspectives, correlative evidence suggests that individuals living in disadvantaged communities display more physiological indicators of allostatic overload, have poorer health, and report higher levels of subjective distress (Robinette, Charles, Almeida, and Gruenewald, 2016; Shulz et al., 2012). Moreover, correlative evidence shows individuals living in disadvantaged urban communities are at greater risk of experiencing depression, crime, and other psychopathological outcomes (Sampson, Morenoff, and Gannon-Rowley, 2002; Silver, Mulvey, and Swanson, 2002; Matheson et al., 2006).

Assuming stress process theories are correct, higher rates of depression and crime in low SES urban areas might be explained by chronic exposure to stress in these communities. However, such community-level correlations might instead spuriously reflect alternative causal processes such as selective migration (e.g., Dohrenwend and Dohrenwend, 1974; Sariaslan et al., 2013; 2016) or reverse causality (Schwartz et al., 2020). A study by Newbury and colleagues (2017) found that, net of genetic selection mechanisms, youths' subjective perceptions of neighborhood adversity were potent processive stressors that have serious implications for psychotic experiences. This study lends some credence to the notion that chronic exposure to stress in disadvantaged communities may be detrimental to mental health.

In contrast, research on crime paints a more mixed picture, with some studies even calling into question whether living in disadvantaged communities reliably increases the risks of criminality. For example, using data from the Project on Human Development in Chicago Neighborhoods, Fagan and Wright (2012) find that the association between neighborhood disadvantage and delinquent or violent behavior may not be as robust as is often expected. Likewise, long-term follow-up evidence from the "Moving to Opportunity" (MTO) experimental study challenges the notion that moving to less stressful neighborhoods reduces crime (Ludwig et al., 2013). Rather, Ludwig and colleagues (2013) conclude that "youth outcomes may be more affected by contemporaneous neighborhood conditions *than accumulated exposure to neighborhood environments*" [emphasis added], thus calling into question whether chronic exposure to the stresses of living in disadvantaged communities indeed causes crime.

Other studies also call into question the claim that stress is more criminogenic for individuals in disadvantaged groups. For instance, one recent international test of general strain theory (Botchkovar, Tittle, and Antonaccio, 2013) reported overall support for general strain theory's hypothesized processes across all SES subgroups yet failed to find more pronounced

strain-coping associations among lower SES respondents. Another recent study (Wright and Fagan, 2013) using the PHDCN data found associations between youths' violent behavior and prior exposure to child abuse – a particularly criminogenic stressor according to general strain theory – were in fact *weaker* in disadvantaged communities. The authors speculated that violence saturation in disadvantaged communities may cause residents to become more tolerant of violence and more desensitized to its potential consequences. Moreover, their finding is consistent with other research showing that chronic exposure to violence may result in "desensitization" and attenuated associations with emotional distress (Farrell and Bruce, 1997), depressive symptoms (Fitzpatrick, 1993), and risks of criminal behavior (Zimmerman and Messner, 2011).

2.4.4 | Amplification or desensitization mechanisms: It varies?

Recall, stress process theories posit that chronic stress exposure should heighten vulnerability to stressors and amplify subjective stress and its consequences (cf. Pearlin et al., 1981; Agnew, 1992; Thoits, 1995). Thus, evidence of "desensitization" in disadvantaged communities, or of attenuated associations between chronic exposure to potent stressors like violence and outcomes such as crime or depression, poses a challenge to stress process theories. However, when considered alongside findings from research on systematic individual and community-based differences in stress responding, these observed desensitization effects are not altogether inexplicable for stress perspectives – though they do suggest the possible existence of alternative biological mechanisms to those underlying popular amplification arguments.

First, researchers have indeed documented substantial inter-individual variability in stress-response system activation (Bartolomucci et al., 2005; Miller, Chen, and Zhou, 2007). Moreover, some evidence suggests that stress only affects decision-making among a select group of stress "responders," or individuals who show marked increases in cortisol levels in response to acute stress (Buckert et al. 2014). Interestingly, however, heightened stress response and consequences for decision-making may be more likely when individuals experience "processive stress" resulting from social and situational interpretations as opposed to "systemic" stress resulting from physiological threats that require less cognitive processing (Starcke and Brand, 2016). If accurate, this implies that social and situational stressors might be more salient to depression and crime than oft-observed "objective" or chronic stressors usually linked to disadvantaged groups and communities.

Second, a recent study observed that lower socioeconomic status (SES) men were better able to resist negative emotions in response to stressors, whereas higher SES individuals with greater psychological resources exhibited greater systemic inflammation, or stress biomarkers indicative of higher stress-induced allostatic load (Elliot and Chapman 2016). This finding may suggest that (at least moderately) high levels of stress are quite common in financially and socioemotionally stable adults and, hence, chronic stress may be normative for many people in modern societies and not typically criminogenic or maladaptive (see also Cousijn et al., 2012).

Third, stress habituation research shows marked declines in biological stress-response system activation following repeated exposure to stressors, at least for some subjects (Herman, 2013; Natelson et al., 1988). This implies that chronic exposure to stress may mitigate rather than exacerbate the neurobiological and associated emotional or behavioral consequences of subsequent stress.

Together, these findings offer reasons to believe that stress effects are conditional on individuals' genetic and social predispositions, histories with stress exposures, and subjective experiences of stress. Put simply, when faced with similar stressful events or circumstances, some individuals may have stronger biopsychological reactions than others, leading to subjective differences in the experience of stress and of stress-induced outcomes. Moreover, this work gives reason to doubt whether chronic exposure to stressful community conditions is as universally detrimental as stress process theories assume. In fact, some evidence to the contrary points to chronic stress exposure possibly alleviating subsequent experiences with otherwise stressful events and mitigating their detrimental consequences.

2.4.5 | Summary

Overall, the documented biophysiological consequences of acute stress appear to include greater risks of depression, as expected by the stress process model. Acute stress might also increase risks of stress-induced criminal coping in ways that are consistent with general strain theory, perhaps by amplifying emotional processing, reducing the perceived costs of crime, inhibiting the rational contemplation of costs, or triggering emotionally reactive "fight" (adaptive survival) behaviors under acute stress. Irrespective of exact mechanisms underlying these *stress deficit* expectations, we examine comparable associations between stress and negative emotions throughout as a benchmark for assessing relative magnitudes of correlations between stress and criminal intent.

Also, stress-process theories claim that residence in disadvantaged communities is marked by chronic stress exposure (*stress clustering*) and that, regardless of the specific mechanisms, chronic stress exposure should amplify the consequences of stress. This *stress amplification* expectation implies stronger stress correlations with negative emotions and criminal intent in these social locations, as chronic stressors are perceived as frequent, potent, and more unjust and as stress proliferation presumably increases allostatic load for residents in these locations. Alternatively, there are some reasons to expect prolonged exposure to stressful community conditions instead might cause stress desensitization or habituation at the neurobiological systems level and an acclimation to stress at the emotional and behavioral levels. This competing expectation implies systematically weaker stress/outcome associations might be expected in communities characterized by higher levels of stress.

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