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Q2 How do negative emotions impair self-control? A neural model of negative urgency

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ABSTRACT

Self-control often fails when people experience negative emotions. *Negative urgency* represents the dispositional tendency to experience such self-control failure in response to negative affect. Neither the neural underpinnings of negative urgency nor the more general phenomenon of self-control failure in response to negative emotions is fully understood. Previous theorizing suggests that an insufficient, inhibitory response from the prefrontal cortex may be the culprit behind such self-control failure. However, we entertained an alternative hypothesis: negative emotions lead to self-control failure because they *excessively* tax inhibitory regions of the prefrontal cortex. Using fMRI, we compared the neural activity of people high in negative urgency with controls on an emotional, inhibitory Go/No-Go task. While experiencing negative (but not positive or neutral) emotions, participants high in negative urgency showed greater recruitment of inhibitory brain regions than controls. Suggesting a compensatory function, inhibitory accuracy among participants high in negative urgency was associated with greater prefrontal recruitment. Greater activity in the anterior insula on negatively-valenced, inhibitory trials predicted greater substance abuse one month and one year after the MRI scan among individuals high in negative urgency. These results suggest that, among people whose negative emotions often lead to self-control failure, excessive reactivity of the brain's regulatory resources may be the culprit.

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Introduction

The opposite of the rational, regulated, and cool-headed person is the emotional, unbridled, and temperamental hot-head. Aversive feelings such as anger, sadness, and anxiety often disrupt individuals' attempts at self-control, resulting in impulsive behaviors and decisions. It remains uncertain how this happens. Common sense suggests that people who act rashly when they are upset fail to successfully inhibit their impulses because they are unmotivated or unable to do so. Yet just the opposite may be true: people may fail at self-control while they experience negative emotions because they *excessively* recruit inhibitory processes. The current paper tests these two competing predictions about why negative emotions undermine self-control.

Negative emotions and self-control

Self-control, the effortful inhibition of impulses, is the foundation of human society and individual success within it (Baumeister and Vohs,

2003, 2007; Duckworth and Seligman, 2005; Tangney et al., 2004). Negative emotions, such as anger, anxiety, fear, and sadness often reduce self-control (Cyders and Smith, 2008; Heatherton and Wagner, 2011; Schmeichel and Tang, 2015). For example, negative emotions impair executive functions necessary for self-control (Curci et al., 2013). Self-control breaks down in the face of such negative emotion because people fail to exert top-down inhibition of bottom-up emotional impulses (Heatherton and Wagner, 2011; Tice and Bratslavsky, 2000).

Self-control and the lateral PFC

Couched in a neural framework, self-control is thought to fail because the subcortical brain regions that promote negative affect (e.g., the amygdala) are not adequately regulated by brain regions that regulate them (e.g., the lateral prefrontal cortex; Heatherton and Wagner, 2011; Wager et al., 2008). Functional neuroimaging studies of inhibitory behavior using paradigms such as the Go/No-Go and Stop Signal tasks routinely show recruitment of the lateral prefrontal cortex, which fosters successful inhibition (Aron et al., 2004; Chikazoe et al., 2007). In these tasks, individuals inhibit a behavioral response (e.g., a button press) that has been made pre-potent or habitual through

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repeated execution (Gomez et al., 2007). Activity in the lateral prefrontal cortex during such inhibitory trials often spatially extends into the anterior insula, which plays less of a beneficial role in facilitating inhibitory behavior because it reflects the conscious awareness of inhibitory errors (Ullsperger et al., 2010). Taken together, established theory would predict that greater activity in the lateral prefrontal cortex would prevent self-control failures under conditions of negative emotions, and that any such self-regulatory impairment would result from an insufficient inhibitory response from this brain region.

Excessive PFC recruitment during negative affect

But what if self-control failure was due to excessive recruitment of the lateral prefrontal cortex? On the surface, such a possibility seems flimsy. Prior research supports the conventional hypothesis that self-control failure starts where inhibitory brain activity stops. For example, the less individuals recruited the lateral prefrontal cortex while they attempted to inhibit cravings, the more they went on to fail in controlling their urges (Berkman et al., 2011; Lopez et al., 2014). However, this relationship between the lateral prefrontal cortex and effective self-control appears to flip for regulatory situations characterized by negative affect. Indeed, greater lateral prefrontal activity during a socially painful event predicted impaired self-control both soon after the event and during the following week (Chester and DeWall, 2014). The question remains: why would greater inhibitory brain activity predict worse self-control?

First, greater inhibitory brain recruitment likely reflects a compensatory strategy for counter-acting self-regulatory deficits. Second, neuroimaging studies have suggested that cognitive and emotional processing may be integrated in the lateral PFC (Gray et al., 2002). In this manner, negative affect may compete with and therefore hijack neural circuitry necessary for effective inhibition. Finally, the deleterious effect of negative affect on self-control is possibly due to the tendency of self-control resources to be ‘fatigued’ after greater use (Baumeister et al., 2007b). Thus, negative affect may tax regulatory resources, rendering individuals less able to engage in self-control. The aversive nature of negative affect may also consume a significant portion of the lateral prefrontal cortex’s inhibitory ability, leaving less regulatory capacity for self-control. This temporal component of the excessive recruitment model is crucial as exacerbated prefrontal recruitment during negative affect may initially be adaptive, resulting in down-regulation of negative affect and effective behavior modification. However, in the longer term, such excessive recruitment is likely to result in self-regulatory fatigue and failure, as predicted by major theories of self-control (e.g., Baumeister et al., 2007a, b).

Individual differences in self-control failure during negative emotions

Individuals vary in the extent to which negative emotions impair their self-control efforts, resulting in impulsive actions and choices. This behavioral tendency is termed *negative urgency*, the dispositional tendency to respond to negative emotions with impulsive and rash acts (Cyders and Smith, 2008; Whiteside and Lynam, 2001). Negative urgency is a facet of impulsivity that predicts problematic outcomes (e.g., intimate partner violence, substance abuse) above-and-beyond other features of impulsivity, such as sensation-seeking (e.g., Derefinko et al., 2011; Settles et al., 2012). Based on previous findings linking excessive inhibitory brain activity during negatively-valenced emotional situations to self-control failure (Chester and DeWall, 2014), we expected that negative urgency would be associated with an excessive (and not insufficient) recruitment of the lateral prefrontal cortex during negative-valenced instances of inhibitory effort. Further, we predicted that such exaggerated activity in these prefrontal regions would predict self-control failure.

Present study

The literature lacks substantial support for the hypothesis that the excessive recruitment of the lateral prefrontal cortex during the experience of negative emotions leads to self-control failure. Moreover, no prior work has examined whether this excessive recruitment model may underpin the inhibitory deficits of negative urgency. To fill this gap in the literature, we hypothesized that (A) individuals high in negative urgency would show more lateral PFC activity during an inhibitory task than individuals low in negative urgency, (B) this group difference would only hold under inhibitory conditions of negative affect, and (C) that the more that individuals high in negative urgency recruited the lateral PFC, the more impaired their inhibitory behavior would be. For this last prediction, we sought to extend our findings outside of the laboratory and assess whether lateral PFC activity would predict self-control failures in the form of alcohol use following the experiment. Specifically, we hypothesized that activation of the lateral PFC would mediate the effect of negative urgency on greater alcohol abuse.

To test these hypotheses, we selected two groups of individuals based on whether they reported relatively high or low negative urgency (see Material and methods for more detail). We crossed this extreme-groups design with relatively high and low levels of neuroticism (the tendency to experience negative affect on a daily basis; John and Srivastava, 1999) to control for this potential group confound. Though negative urgency and neuroticism share many features (e.g., emotional lability), urgency represents a behavioral tendency towards rash acts that is distinct from neuroticism. These four groups of approximately 20 people underwent functional magnetic resonance imaging (fMRI) while they completed an inhibitory, Go/No-Go task under negative, neutral, and positive emotional valences. Finally, participants reported their daily alcohol consumption (a proxy for self-control failure) one month and twelve months after their MRI scan.

Material and methods

Participants

Potential participants were recruited from an introductory psychology participant pool. To prevent issues with comfort and safety in the MRI scanning environment and to ensure the quality of our fMRI data, participants were excluded for any of the following conditions: body-mass-index greater than 30, claustrophobia, color blindness, psychoactive medication use, psychological or neurological pathology, a history of seizures, or suspected pregnancy. To be recruited, potential participants also had to report that they had previously consumed alcohol to ensure the presence of variability on our alcohol consumption measure. Participants were recruited into one of four groups based on a 2 (high vs. low negative urgency) by 2 (high vs. low neuroticism) factorial design. ‘High’ and ‘low’ group assignments were determined by scores from the upper and lower halves of the sampling distribution, respectively. This extreme groups design was selected to maximize statistical power and was not intended to reflect clinically-significant thresholds in negative urgency.

Data were acquired from 80 healthy, right hand dominant undergraduate students who received course credit and money for their participation (see Table 1 for demographics). Regarding ethnic diversity, our sample was 77.6% White, 13.2% Black, 6.6% Asian, and 2.6% ‘other’. Participants in the high urgency groups reported significantly greater urgency, $t(78) = 21.50, p < .001, d = 4.78$, and marginally higher neuroticism, $t(78) = 1.98, p = .052, d = 0.44$, than participants in the low urgency groups. Validating our use of the terms ‘high’ urgency and ‘low’ urgency, participants in the high urgency groups reported urgency levels above the midpoint of the scale (i.e., 2.5), $t(39) = 10.54, p < .001, d = 2.33$, and low urgency groups reported urgency levels below the midpoint of the scale (i.e., 2.5), $t(39) = -18.44, p < .001, d = -4.17$. High and low negative urgency groups did not

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