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## 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. The neural underpinnings of negative urgency are not fully understood, nor is the more general phenomenon of self-control failure in response to negative emotions. 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.

### Keywords

self-control; impulsivity; negative emotion; fMRI; negative urgency; lateral prefrontal cortex

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## 1. 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. Commonsense 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.

### 1.1 Negative Emotions and Self-Control

Self-control, the effortful inhibition of impulses, is the foundation of human society and individual success within it (Baumeister & Vohs, 2003, 2007; Duckworth, & Seligman, 2005; Tangney, Baumeister, & Boone, 2004). Negative emotions, such as anger, anxiety, fear, and sadness often reduce self-control (Cyders & Smith, 2008; Heatherton & Wagner, 2011; Schmeichel & Tang, 2015). For example, negative emotions impair executive functions necessary for self-control (Curci, Lanciano, Soleti, & Rimé, 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 & Wagner, 2011; Tice & Bratslavsky, 2000).

### 1.2 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 & Wagner, 2011; Wager, Davidson, Hughes, Lindquist, & Ochsner, 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, Robbins, & Poldrack, 2004; Chikazoe, Konishi, Asari, Jimura, & Miyashita, 2007). In these tasks, individuals inhibit a behavioral response (e.g., a button press) that has been made pre-potent or habitual through repeated execution (Gomez, Ratcliff, & Perea, 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, Harsay, Wessel, & Ridderinkhof, 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.

### 1.3 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, Falk, & Lieberman, 2011; Lopez, Hofmann, Wagner, Kelley, & Heatherton, 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 & 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, Braver, & Raichle, 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, Vohs, & Tice, 2007). 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., 2007).

#### 1.4 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 & Smith, 2008; Whiteside, & 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, DeWall, Metze, Walsh, & Lynam, 2011; Settles et al., 2013). Based on previous findings linking excessive inhibitory brain activity during negatively-valenced emotional situations to self-control failure (Chester & 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.

#### 1.5 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 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 & 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.

## 2. Material and Methods

### 2.1 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 assignment was 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’ 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 differ in gender distribution,  $\eta^2(1, N = 80) = 0.02$ ,  $p = .877$ , or age,  $t(78) = 1.05$ ,  $p = .296$ ,  $d = 0.24$ .

One male from the low urgency – high neuroticism group and one female from the high urgency – high neuroticism group terminated their MRI scans due to anxiety from being inside of the scanner. Analyses were then performed on the 78 remaining participants (40 high urgency, 38 low urgency).

## 2.2 Self-Report Measures

**2.2.1 Negative urgency**—Participants completed the 12-item negative urgency subscale of the UPPS-P Impulsivity Scale (Lynam, Smith, Whiteside, & Cyders, 2006; Whiteside & Lynam, 2001). This subscale captures dispositional individual differences in the tendency to act impulsively under conditions of negative affect. Each item was responded to along a 1 (Disagree Strongly) to 4 (Agree Strongly) Likert-type scale. Sample items include “when I am upset I often act without thinking” and “in the heat of an argument, I will often say things that I later regret.” This subscale has previously shown excellent internal reliability (Cyders & Smith, 2010). After reverse-scoring relevant items, all 12 responses were averaged together to create a negative urgency score for each participant that could range from 1 to 4.

**2.2.2 Neuroticism**—To measure neuroticism, the tendency to experience greater negative affect, participants completed the 10-item Neuroticism subscale of the Big Five Inventory (John & Srivastava, 1999). Each item was responded to along the same 4-point response scale that was used for the negative urgency subscale. After reverse-scoring relevant items, all 10 responses were averaged together to create a neuroticism score for each participant that could range from 1 to 4.

**2.2.3 Alcohol use**—To assess participants’ everyday alcohol use, participants completed two, online timeline follow-back calendars one month and twelve months after their scan (Sobell & Sobell, 1992). Participants were trained in how to complete the calendars during their initial laboratory visit. For each calendar, participants recorded how many alcoholic drinks they had consumed each day for the given month. A conversion chart was also provided which allowed participants to determine how many drinks a given amount of beer, wine, liquor, or malt liquor consisted of. The number of post-scan drinks were averaged across each day of the month because the number of days in a given month differed for each subject, yielding two, one-month and twelfth-month alcohol consumption scores.

**2.2.4. Self-control and sensation-seeking**—To provide evidence that alcohol use represented self-control failure and not other psychological processes such as sensation-seeking, participants completed measures of trait self-control (i.e., the 13-item Brief Self-Control Scale; Tangney et al., 2004) and the Sensation-Seeking subscale of the UPPS-P Inventory referenced above.

## 2.3 Procedure

**2.3.1 Intake session**—Participants arrived at our laboratory where they completed a battery of questionnaires, included the Brief Self-Control Scale, a demographics survey, and the Sensation-Seeking subscale of the UPPS-P Impulsivity Scale.

**2.3.2 Go/No-Go task**—Participants arrived at the University of Kentucky’s Magnetic Resonance Imaging and Spectroscopy Center where they were screened for safety and comfort in the MRI environment and practiced the Go/No-Go fMRI task in an adjacent room prior to entering the MRI scanner. Participants entered the scanner where they completed a randomized, event-related design of an emotional go/no-go task while undergoing fMRI. Participants were instructed to press a button with their right thumb whenever they viewed the letter ‘M’ (Go trials) and to not press the button when they viewed the letter ‘W’ (No-Go trials). These letters were overlaid atop images from the International Affective Picture System (IAPS; Lang et al., 2008) that were selected based on pre-ratings of high, average, or low pleasantness to elicit positive, neutral, and negative affective valence, respectively (see Table 2 for stimuli ratings). Negative stimuli were diverse including frightening animals, human corpses, visibly distraught and suffering individuals, interpersonal violence, natural disasters, disgusting food and facilities. Social stimuli were included in all three conditions, and arousing images were included in the positive condition (e.g., breath-taking landscapes, daredevil acts) to prevent systematic bias. The task then possessed a 2 (response: Go vs. No-Go) by 3 (valence: negative vs. neutral vs. positive) within-subjects factorial design yielding 6 conditions.

The task consisted of 198 total trials each lasting 2.5 seconds (total duration: 8 minutes, 15 seconds), 132 of which were experimental trials and 66 were simple fixation trials. The onset of each trial was yoked to the temporal sampling interval of the scanner. Each experimental trial consisted of three elements. First, participants were cued to Go or No-Go while simultaneously being primed with an emotional image (1 second). Second, the response cue was replaced by a fixation cross while the emotional image remained. This allowed us to control for neural responses to the image itself. Third, the image disappeared and only the fixation cross remained on the screen (0.5 seconds). Of the 132 experimental trials, 93 were Go trials and 39 were No-Go trials, split evenly by emotional valence. The order of trials were randomized and held constant across participants.

**2.3.3 Follow-Up Surveys**—On the first day of the second month and thirteenth month from their MRI scan, participants received an internet survey that included a timeline follow back calendar (Sobell & Sobell, 1992) that assessed their past month of alcoholic drinking behavior.

## 2.4 fMRI Data

**2.4.1 Acquisition**—Functional brain images were collected on a 3-Tesla Siemens MAGNETOM Trio MRI scanner using a T2\*-weighted gradient echo planar imaging sequence with the following acquisition parameters: 64 × 64 matrix size, 224 × 224mm field of view, 28ms echo time, 2.5s repetition time, 40 3.5mm axial slices acquired, 90° flip angle, following a 3D shim, in interleaved order which allowed for whole brain coverage. A high-

resolution, 3D T1-weighted MPRAGE anatomical imageset was also acquired from each participant with the following parameters: 1mm isotropic voxels, 2.56ms echo time, 1.69s repetition time, 12° flip angle.

**2.4.2 Preprocessing**—The Oxford Center for Functional MRI of the Brain (FMRIB)'s Software Library (FSL version 5.0) was used to conduct all preprocessing and fMRI analyses (Jenkinson, Beckmann, Behrens, Woolrich, & Smith, 2012; Smith et al., 2004; Woolrich et al., 2009). Reconstructed functional volumes underwent head motion correction to the median functional volume and were skull stripped. Functional volumes underwent slice-timing correction, pre-whitening, were smoothed with a 5-mm FWHM Gaussian kernel, and were high-pass filtered (100 second cutoff). Non-brain structures were then stripped from reconstructed anatomical imageset.

**2.4.3 Analysis**—To analyze the Go/No-Go task, a fixed-effects general linear model was specified that modeled Go-Negative, Go-Neutral, Go-Positive, No-Go-Negative, No-Go-Neutral, and No-Go-Positive trials using a canonical double-gamma hemodynamic response function along with a temporal derivative. Three first-level contrasts within each participant compared the Go and No-Go conditions to one another, separately for each valence (e.g., No-Go-Negative > Go-Negative). The initial, one second portion of each trial in which the given image was displayed underneath a fixation cross was included as a nuisance regressor to account for effects of passively viewing each image. Further, all six motion parameters were modeled as nuisance regressors while fixation trials and the 0.5 second fixation screens at the end of each trial were left un-modeled.

The resulting contrast images from this analysis were first registered to native space structural volumes, spatially normalized to an MNI stereotaxic space template image, and resampled into  $2 \times 2 \times 2 \text{ mm}^3$  standard space. A group-level, mixed-effects general linear model was then performed which created group average maps for each contrast. Group level statistic images were fed into a 2 (high vs. low urgency) by 2 (high vs. low neuroticism) between-subjects general linear model in which each of the four groups defined during recruitment were modeled as regressors. High urgency groups were then contrasted against low urgency groups (high urgency > low urgency). The resulting  $Z$  (Gaussianized  $T/F$ ) statistic images were thresholded using clusters determined by  $Z > 2.3$  and a (family-wise error corrected) cluster significance threshold of  $p < .05$  across the entire brain (Heller, Stanley, Yekutieli, Rubin, & Benjamini, 2006; Worsley, 2001).

### 3. Results

#### 3.1 Behavioral Results

**3.1.1 Go/No-Go task**—The experimental software failed to record the responses of two participants (1 high urgency, 1 low urgency) for the Go/No-Go task. For the remaining 76 participants, the task recorded accuracy rates for Go and No-Go trials which were computed as a percentage of the total number of trials responded to correctly (i.e., a button press on Go trials, no button press on No-Go trials) over the total number of trials. This computation was performed separately for each affect condition, yielding six accuracy scores. One participant was deemed an outlier as their accuracy on No-Go-Negative trials was 6.49  $SDs$  below the

sample mean. All other participants were within one *SD*. This one outlier was excluded from all subsequent analyses.

Overall, average accuracy rates were extremely high, ranging from 97.13% to 99.40%, which is likely due to the slower pace of the task than is conventionally used (in order to more accurately estimate the BOLD response). Accuracy rates were characterized by a main effect of inhibition, such that across all affect conditions, participants were more accurate on Go trials,  $M = 99.14\%$ ,  $SD = 2.01\%$ , than No-Go trials,  $M = 97.44\%$ ,  $SD = 3.32\%$ ,  $F(1, 74) = 13.53$ ,  $p = .001$ ,  $\eta_p^2 = .155$ . There was no main effect of affect condition,  $F(2, 149) = 0.01$ ,  $p = .937$ ,  $\eta_p^2 = .000$ , or an interaction between affect and trial-type,  $F(2, 149) = 0.87$ ,  $p = .354$ ,  $\eta_p^2 = .012$ . Response latencies were also obtained for Go trials. Response latencies showed a marginally significant main effect of affect condition,  $F(1, 74) = 3.48$ ,  $p = .066$ ,  $\eta_p^2 = .045$ , which appeared to be driven by slower response times among positive trials,  $M = 573.80\text{ms}$ ,  $SD = 72.83\text{ms}$ , than neutral,  $M = 564.33\text{ms}$ ,  $SD = 75.76\text{ms}$ , or negative trials,  $M = 567.97\text{ms}$ ,  $SD = 74.32\text{ms}$ . No main effects of urgency group or interactions with urgency group were observed in these behavioral analyses. Thus, the high urgency group fared the same on the task as the low urgency group in regards to response latency and accuracy.

**3.1.2 Alcohol use**—Of the 78 original participants, eight failed to return the one-month post-scan alcohol consumption survey and 22 failed to return the twelfth-month survey. At one month after the scan, participants showed substantial variability in the post-scan month of alcohol use with the average number of daily drinks ranging from 0 to 4.39,  $M = 0.72$ ,  $SD = 0.92$ . At the twelfth month after the scan, participants' daily alcohol drinks ranged from 0 to 6.65,  $M = 0.78$ ,  $SD = 1.14$ . An outlier was identified from the twelfth month survey, who consumed 206 drinks over the month (5.21 *SDs* from the mean, all others within 2.5 *SDs*) and was excluded from all subsequent analyses involving alcohol consumption. Using multiple linear regression to control for both gender and neuroticism (via continuous neuroticism scores rather than group assignment), participants in the high urgency group reported substantially more alcohol consumption over the one month after their MRI scan,  $\beta = .31$ ,  $t(66) = 2.66$ ,  $p = .010$ , though the effect was non-significant for the twelfth month,  $\beta = .21$ ,  $t(51) = 1.50$ ,  $p = .139$ . One month and twelfth month alcohol calendars were strongly, positively correlated with each other,  $r(53) = .71$ ,  $p < .001$ . Supporting alcohol consumption as a proxy for self-control failure, and not other psychological processes such as sensation-seeking, alcohol consumption across both the one month and twelfth month follow-ups was associated with less trait self-control,  $\beta = -.44$ ,  $t(57) = -3.15$ ,  $p = .003$ , and not with sensation-seeking,  $\beta = -.04$ ,  $t(57) = -0.28$ ,  $p = .783$ .

## 3.2 Neuroimaging Results

**3.2.1 Whole brain results**—To assess the neural correlates of our Go/No-Go task prior to making any group comparisons, we conducted a whole-brain contrast between No-Go > Go conditions (across all three emotion conditions). This contrast revealed a typically-observed pattern of greater activation across both cingulo-opercular and fronto-parietal networks and lesser activity in the primary motor cortex and cerebellum (Supplemental Figure 1; Supplemental Table 1; Swick, Ashley, & Turken, 2011). A nearly identical pattern of activity



was observed when the same No-Go > Go contrast was constrained to the negative affect conditions of the task (Supplemental Figure 2; Supplemental Table 2).

**3.2.2 Group comparisons**—To test whether individuals high in urgency exhibited greater activity in their lateral prefrontal cortex during negatively-valenced inhibition, we conducted three group comparisons (High Urgency > Low Urgency) for each affect condition of the No-Go > Go within-subjects contrast. For the negative affect condition, High Urgency participants showed two clusters of greater inhibitory neural activation than controls that spanned multiple brain regions. These two clusters in the left and right hemispheres both included the anterior insula, dorsal striatum, and ventrolateral PFC (VLPFC; Figure 1; Table 3). Activated voxels were assigned to a given region based on the Harvard-Oxford Structural Probability Atlas. Voxels determined to be probabilistically within white matter pathways were not assigned to a given region as the interpretation of white matter activity is unclear. No clusters survived thresholding from the No-Go > Go contrasts of either the neutral or positive affect conditions.

To explore the relations between inhibitory neural activity under negative affect and behavioral measures, percent signal change values associated with brain activation were first extracted from all activated voxels across both clusters, yielding an average, inhibitory network value for each participant. Then, percent signal change values were extracted from each subregion of the clusters (i.e., left and right anterior insula, left and right VLPFC, dorsal striatum). The striatum was not dichotomized based on hemisphere as there was no clear theoretical prediction regarding hemispherical laterality in the striatum's role in inhibitory functioning. A psycho-physiological interaction (PPI) analysis was also conducted to assess group differences in functional connectivity between the VLPFC and other brain regions that may subservise the experience of negative affect (e.g., the amygdala). This analysis revealed no group differences in functional connectivity between either hemisphere of the VLPFC and other brain regions.

**3.2.3 Associations with inhibitory accuracy**—To assess how brain activity during negatively-valenced inhibitory (i.e., No-Go) trials was associated with actual inhibitory accuracy, we conducted a whole-brain regression analysis using identical thresholding parameters as used in the previous whole-brain group comparisons. In this analysis, we averaged accuracy rates for each participant by averaging across all No-Go-Negative trials. Brain activity from the No-Go-Negative > Go-Negative contrast was regressed onto these accuracy values to detect which brain regions showed functional associations, negative and positive, with task accuracy. The regression analyses were conducted separately for High and Low Urgency participants to detect different neural mechanisms through which each group achieved accuracy.

The results of the regression analyses revealed substantially different patterns of associations between inhibitory brain activity and inhibitory accuracy between the two Urgency groups (Figure 2; Tables 4 and 5). Among High Urgency participants, accuracy on No-Go-Negative trials was positively associated with large clusters of activity in traditionally regulatory and inhibitory regions of the prefrontal cortex (e.g., dorsal anterior cingulate cortex, DLPFC,

anterior insula, VLPFC; raw data depicted in Supplemental Figure 3). No such prefrontal clusters were observed among Low Urgency participants.

### 3.3 Multiple Mediation Analyses

To assess whether the greater activation of the inhibitory network explained the tendency for individuals high in urgency to experience self-control failures, we tested two bias-corrected, bootstrapped (at 1,000 samples), multiple mediation models (Preacher & Hayes, 2008) whereby each of the five subregions identified from the High > Low Urgency group contrast were modeled as mediators of the effect of negative urgency on one-month and twelfth-month alcohol use, separately. Continuous scores on our measure of neuroticism together with gender were included as covariates in all these analyses. Each analysis yielded a 95% confidence interval for the indirect effect of each of the five subregions (left and right anterior insula, left and right VLPFC, dorsal striatum) in which the exclusion of the value 0 within this interval indicated a significant indirect effect (Preacher & Hayes, 2008). Both mediation models indicated that within the inhibitory network, only the right anterior insula mediated the effect of negative urgency on alcohol consumption (Figure 3). Specifically, participants in the high urgency group showed greater right anterior insula activity which was then related to greater alcohol consumption.

## 4. Discussion

Inhibition in the service of self-control is a crucial capacity for effectively navigating the human world (Baumeister & Vohs, 2003, 2007). Despite the understanding that negative emotions impair inhibition, relatively little is known about how brain functioning can contribute to such self-control failure. Across our findings, we obtained support for an 'excessive inhibition' model of self-control failure due to negative emotions. People who tend to fail at self-control when they experience negative affect (i.e., those high in negative urgency) showed a more intense recruitment of inhibitory regions when they attempted to control their responses. This effect only held when participants were simultaneously observing an image intended to induce negative emotions.

The extent to which they recruited the prefrontal cortex was associated with greater inhibitory performance though only among individuals high in negative urgency, suggesting that such excessive inhibition is compensatory in nature. It is uncertain why these associations were not observed, to a lesser extent, among individuals low in negative urgency. Such compensatory over-activation of the PFC was not observed among individuals low in negative urgency. Our findings suggest that, for those who tend to respond to negative emotions with self-control failure, inhibitory regions are used inefficiently and excessively, which results in eventual self-regulatory fatigue (Baumeister et al., 2007). The excessive recruitment of the PFC appeared to be initially adaptive as it was associated with greater inhibitory accuracy, though in the longer term it appeared to predict self-regulatory failures such as alcohol abuse. Much like an athlete, individuals higher in negative urgency may benefit from training their self-regulatory exertions to be less excessive in order to retain such psychic resources for later self-regulatory challenges.

Only the right anterior insula mediated the greater alcohol use we observed among our high urgency group. The anterior insula is well-known for its role in maintaining interoceptive awareness of one's homeostatic state (Craig, 2009), and within the context of inhibitory tasks appears to subserve one's awareness of inhibitory errors (Ullsperger et al., 2010). This error awareness function appears to be more strongly associated with the right hemisphere of the anterior insula (Klein et al., 2007; Ullsperger et al., 2010). Although our high urgency group made no more inhibitory errors than did controls, they may have felt an acute subjective awareness of their perceived errors. This aversive state, when experienced chronically, may promote substance abuse as a means to ameliorate it. Interventions targeting successful inhibition (e.g., smoking cessation) might profit from reducing the awareness of self-control failures among certain individuals (e.g., those high in negative urgency). This deleterious role of the anterior insula dovetails nicely with research showing that an excessive focus on or awareness of failure, loss, or negative outcomes undermines performance across an array of regulatory domains, particularly under conditions of negative affect (e.g., performance anxiety; Higgins, 1998).

We also observed greater dorsal striatum activity among high urgency subjects when they attempted to inhibit a prepotent response under conditions of negative affect. The dorsal striatum's role in facilitating habitual, automatic responding (Grahn, Parkinson, & Owen, 2008) suggest that the 'Go' response, under conditions of negative affect, became more prepotent for individuals high in negative urgency. Dysfunctional striatal activity may be an important, though largely unexplored, contributor behind self-control failures. However, it is crucial to note that the inherent problems of reverse inference in functional neuroimaging studies (Poldrack, 2006; which are also present in almost all psychological research) undermine any certainty we can have as to which psychological processes are represented by the greater activation of the regions we observed.

Yet why would greater recruitment of regions that typically promote effective self-control lead to self-control failure in the face of negative affect? Negative emotions are extremely, even notoriously, difficult to regulate, which is likely an evolutionary adaptation that allows emotions to effectively shape our behavior to yield better outcomes (Baumeister, Vohs, DeWall, & Zhang, 2007). Thus, they require substantial executive, and therefore prefrontal, resources when human try to control them. Unlike cravings, which appear to be effectively regulated by greater inhibitory neural activity (e.g., Berkman et al., 2011), negative affect is experienced as aversive. The impulse to squash or inhibit these uncomfortable impulses may be far stronger. This excessive tendency to inhibit negative emotions may explain why we observe such aggravated inhibitory neural responses among those who routinely fail at self-control in these circumstances.

Our findings are part of a larger movement to understand the neurobiological underpinnings of negative urgency's deleterious effects on self-control and substance abuse. Negative urgency has been previously linked to greater inhibitory brain activity during negative affect (Cyders et al., 2014b) and urgency's link to alcohol abuse also appears to be mediated by activity in the ventromedial PFC during alcohol-related cues (Cyders et al., 2014a). The VMPFC is a well-established node in the brain's reward circuitry and plays a critical role in computing the value of a given stimulus (Gläscher, Hampton, & O'Doherty, 2009).

Integrating our findings with these, it may be that negative urgency interacts with negative emotions to produce substance abuse by (A) initially over-taxing regulatory resources in the lateral PFC that then (B) tips the regulatory balance in favor of the ventromedial PFC and reward. Future research should attempt to empirically substantiate this process model.

This study possessed several important limitations. First, the sample size was relatively large for a functional neuroimaging study yet likely still insufficient to obtain an ideal level of statistical power for the multiple mediation analyses we employed. Also, we adopted an extreme groups design that artificially dichotomized negative urgency. We hope that future research, with larger samples, will attempt to replicate our mediational findings while assessing urgency as a dimensional construct. Second, all effects reported in this manuscript were purely correlational and thus it is impossible to make any causal claims. Indeed, the directionality of these effects may be difficult to establish. It may be that excessive prefrontal recruitment contributes to the development of negative urgency and not vice-versa or even that these effects are bi-directional and recursive. Experimental manipulations of negative affect, in which individuals are made to react impulsively to negative affect, as well as longitudinal and developmental research on negative urgency are needed to bolster such directional claims. Third, our sample consisted of undergraduates who possessed subclinical levels of negative urgency. Future research might benefit from testing whether clinical populations in which negative urgency is a central facet (e.g., borderline personality disorder; Cyders & Smith, 2008) demonstrate a similar tendency to excessively recruit inhibitory brain regions. Finally, our Go/No-Go task used a fixed inter-stimulus-interval (ISI) instead of a jittered ISI. Jittered ISIs are efficient means to de-confound the BOLD response from data acquisition parameters, yet they undermine statistical power. We adopted a fixed ISI in order to maximize the power of our relatively smaller number of trials.

Despite these limitations, our pattern of findings fit well with contemporary theories of self-control, which demonstrate that intense self-control exertion predicts substantial self-control failure (Baumeister et al., 2007). An apt analogy for our results may be that of weight lifters who expend their muscular energy on the first few lifts, leaving them weakened and unable to complete their set of lifts. This model of compensatory yet excessive inhibition then has clear implications for interventions and therapies designed to ameliorate self-control failures (e.g., mood disorders, substance abuse, violence). Teaching individuals how to avoid under- and over-regulation of their impulses should yield substantial gains in improving self-control. Self-control requires a delicate balance and it appears to be very unfortunate when it is tipped too far in the direction of inhibition.

## Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

## Acknowledgments

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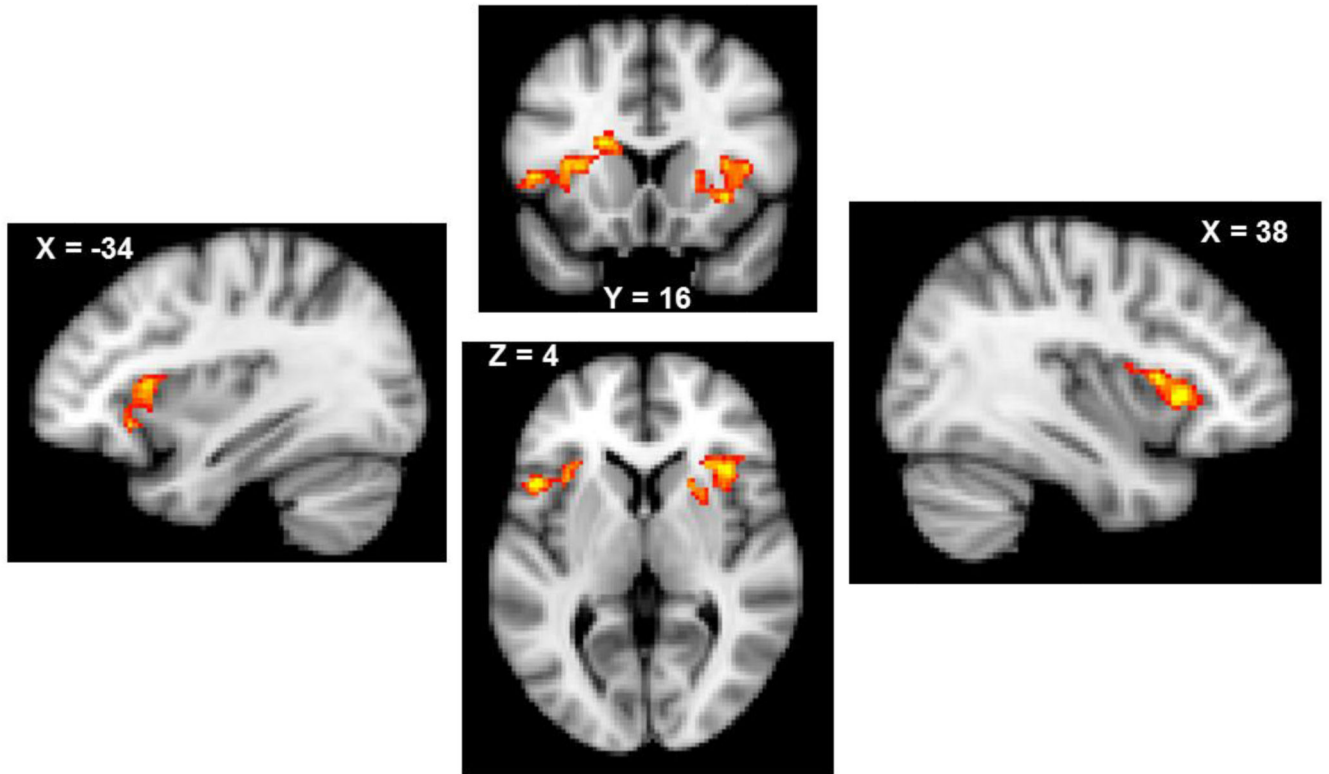
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### Highlights

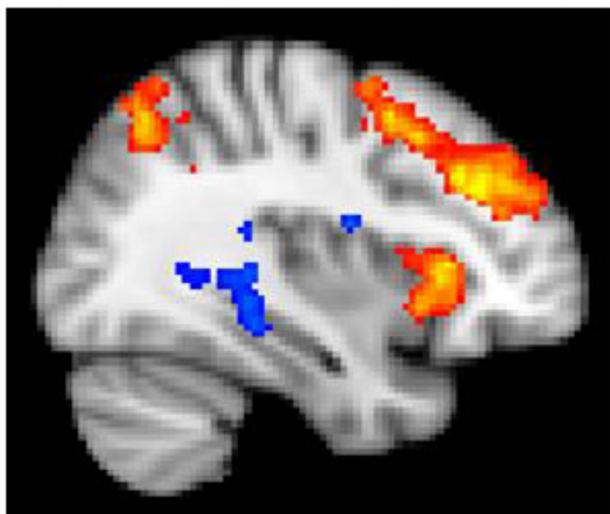
- Urgency predicts greater PFC activity during negative-valence inhibition
- Greater PFC activity compensated for urgency's inhibitory deficits
- Greater insular response predicted alcohol abuse after the scan



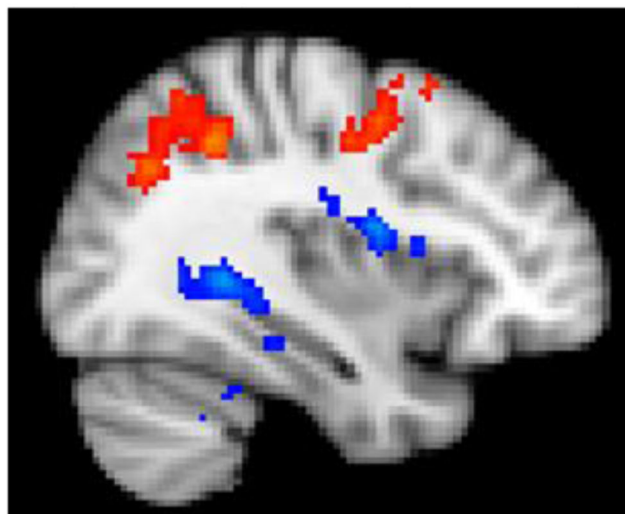
**Figure 1.** Axial, coronal, and left and right sagittal views of the two clusters of activation that were greater for the High Urgency group as compared to the Low Urgency group from the No-Go-Negative > Go-Negative contrast. MNI coordinates are provided in white text. Activated voxels range from red ( $Z = 2.3$ ) to yellow ( $Z = 3.62$ )



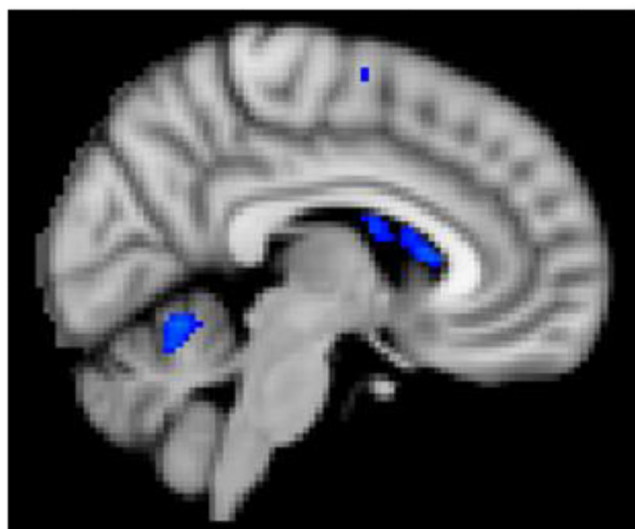
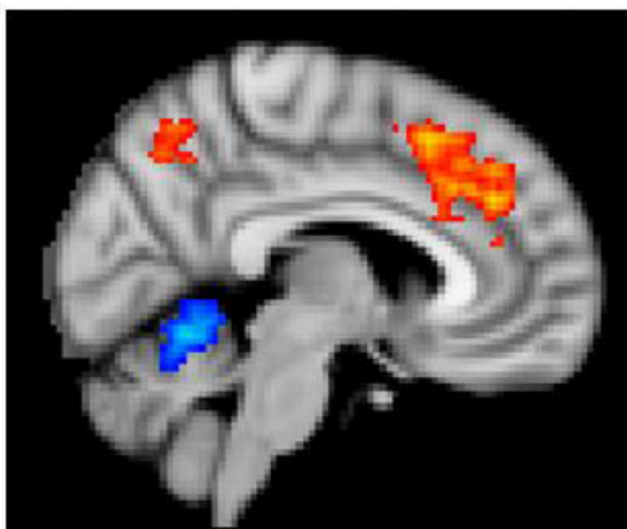
## High Urgency



## Low Urgency

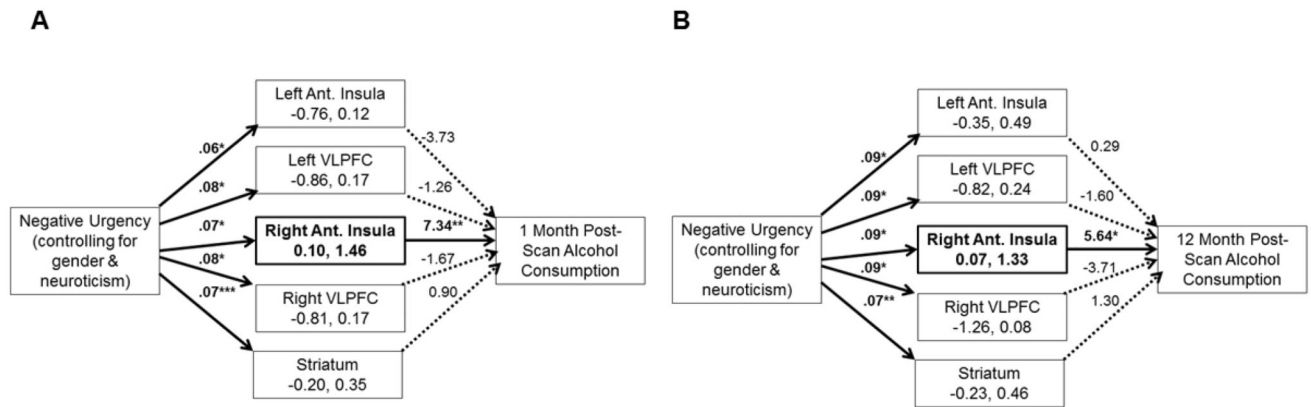


X = 36



X = 6

Figure 2. Whole brain regression results in which accuracy scores from No-Go-Negative trials were regressed onto brain activity from the No-Go-Negative > Go-Negative contrast, separately for High and Low Urgency groups. Red-yellow voxels indicate positive associations and blue voxels indicate negative associations. Correlated voxels range from red/dark-blue ( $Z = +/-2.3$ ) to yellow/light-blue ( $Z = +/-5.00$ )



**Figure 3. Bootstrapped multiple mediation model through which the effect of High Urgency on greater alcohol consumption for the (A) first and (B) twelfth months after the scan was mediated by activity in the right anterior insula from the No-Go-Negative > Go-Negative contrast. Values included in the mediator boxes are 95% confidence intervals of each indirect effect. \*p < .05, \*\*p < .01, \*\*\*p < .001**

**Table 1**  
**Demographics by negative urgency (listed below as ‘Urgency’) groups**

Group	N	Females	Age	Urgency	Urgency	Neuroticism
			<i>M(SD)</i>	<i>M(SD)</i>	Range	<i>M(SD)</i>
High Urgency	41	28	18.87(1.22)	2.81(0.19)	2.55-3.25	2.38(0.42)
Low Urgency	39	26	18.63(0.71)	1.54(0.33)	1.00-2.08	2.16(0.55)

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**Table 2**  
**IAPS stimuli pleasantness and arousal ratings by condition. Ratings could range from 1 (not very arousing/pleasant) to 9 (very arousing/pleasant). The ‘Vs. Midpoint’ column presents results from one-sample t-tests that compared ratings against the midpoint of the scale (i.e., 5)**

Valence	# of Stimuli	<i>M</i> ( <i>SD</i> )	Range	Vs. Midpoint	Arousal <i>M</i> ( <i>SD</i> )
Negative	44	3.04(0.55)	2.43-3.63	$t(43) = -23.70^{***}$	5.26(0.71)
Neutral	44	5.04(0.52)	4.25-5.90	$t(43) = 0.51$	3.45(1.05)
Positive	44	7.57(0.36)	6.84-8.34	$t(43) = 47.78^{***}$	4.91(1.03)

\*  
 p < .05,

\*\*  
 p < .01,

\*\*\*  
 p < .001

**Table 3**  
**Activated clusters from the High Urgency > Low Urgency group contrast of the No-Go-Negative > Go-Negative trial contrast**

<b>Sub Regions</b>	<b>Voxels (3mm<sup>3</sup>)</b>	<b>Peak Z</b>	<b>Peak MNI coordinates (x,y,z)</b>
Anterior Insula, VLPFC, Dorsal Striatum	540	3.59	-48, 14, 4
Anterior Insula, VLPFC, Dorsal Striatum	614	3.62	38, 20, 2

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**Table 4**  
**Whole brain regression results from the No-Go-Negative > Go-Negative contrast**  
**(accuracy scores from No-Go-Negative trials as regressor) among High Urgency**  
**participants only. Coordinates are in MNI space**

Region(s)	Voxels	Peak Z	x	y	z
----Negatively Correlated----					
Postcentral Gyrus, Inferior Parietal Cortex, Posterior Insula, Precentral Gyrus	4,555	-5.6	-50	-20	48
Cerebellum	730	-4.58	8	-52	-12
Inferior Parietal Cortex	696	-3.74	60	-18	18
----Positively Correlated----					
Precuneus	843	4.24	12	-56	42
DLPFC	1,281	4.64	30	34	32
Dorsal Anterior Cingulate Cortex / SMA	681	3.75	4	18	48
Cuneus	563	3.96	-10	-78	-4
Anterior Insula, VLPFC	534	4.19	32	22	-2
Anterior Insula, VLPFC	408	3.74	-36	18	-8
TPJ	556	3.75	48	-42	14

**Table 5**  
**Whole brain regression results from the No-Go-Negative > Go-Negative contrast**  
**(accuracy scores from No-Go-Negative trials as regressor) among Low Urgency**  
**participants only. Coordinates are in MNI space**

Region(s)	Voxels	Peak Z	x	y	z
----Negatively correlated----					
Postcentral gyrus, Inferior parietal cortex, posterior insula, caudate	11,844	-5.89	-54	-20	50
Cerebellum	1,099	-5.66	22	-50	-22
Precentral gyrus	580	-4.11	-4	-24	46
----Positively correlated----					
Lateral occipital cortex	745	3.72	44	-58	42
Precentral gyrus	578	3.67	24	16	60

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