Exploring the Pathophysiology of Emotion-Based Impulsivity: The Roles of the

Sympathetic Nervous System and Hostile Reactivity

1. Introduction

The tendency to behave impulsively when distressed, known as *negative urgency* (Cyders and Smith, 2008; Whiteside and Lynam, 2001), has emerged as a strong transdiagnostic risk factor for a broad range of maladaptive behaviors often exhibited by psychiatric patients (Dir et al., 2013). Examples include reactive aggression and intimate partner violence (Derefinko et al., 2011), substance abuse (Fischer et al., 2012; Kaiser et al., 2012), risky sexual behavior (Deckman and Nathan DeWall, 2011; Derefinko et al., 2014), non-suicidal self-injury (Bresin et al., 2012; Peterson et al., 2014), suicidal behavior (Anestis and Joiner, 2011), and binge eating and purging (Fischer et al., 2013; Wenzel et al., 2014). While often conceptualized as a personality trait and used as a predictor in statistical models, negative urgency has increasingly been recognized as a potential modifiable mechanistic variable of interest for interventions targeting these disorders, with evidence that negative urgency decreases over the course of a range of interventions for individuals with substance use problems, disordered eating, and emotion regulation difficulties (e.g., Delgado-Rico et al., 2012; Littlefield et al., 2015; Zapolski et al., 2016). Clarifying the physiological and psychological attributes that characterize negative urgency may help identify both individuals at greater risk and potential targets for intervention development.

Among the broader population of individuals predisposed to experiencing high levels of negative affect (or *neuroticism*), only some also report high levels of emotion-driven impulsivity (Cyders and Coskunpinar, 2010). Although more frequent experience of negative affect is a risk factor for negative affect-related impulsivity, impulsive behavior is only one of a wide range of potential responses to difficult emotions (Gratz

and Roemer, 2004). Little is known about the factors that may influence *which neurotic individuals display this impulsive behavior in the context of distress and therefore are at increased risk for the wide range of negative-urgency related problems*. Given experimental evidence that the sympathetic branch (SNS) of the autonomic nervous system plays a causal role in the connection between negative affect and emotion-related impulsive behavior (reviewed below), the present study examined whether elevated resting SNS activity strengthens the link between neuroticism and emotion-driven impulsivity, measured both by self-report and impulsive behavior during a stressful task. Given the behavioral and pharmacological interventions available that target elevated SNS activation (e.g., biofeedback, beta-blockers), understanding the role of SNS activation in negative urgency could have numerous clinical implications.

1.1 Pathophysiology of Urgency: A Role for the Sympathetic Nervous System?

Experimental work points to the potential for heightened SNS activation to facilitate emotion-driven impulsive behavior. SNS input at beta-adrenergic receptors increases amygdala reactivity (Hurlemann et al., 2010; van Stegeren et al., 2005) and is associated with poorer executive cognitive functioning in response to stress (Ramos and Arnsten, 2007). Similarly, beta-adrenergic receptor blockade, which reduces SNS activity, attenuates amygdala reactivity to emotional stimuli (Hurlemann et al., 2010) and improves executive cognitive functioning (e.g., working memory, switching) in stressful circumstances (Alexander et al., 2007; Roozendaal et al., 2004). At the behavioral level, alpha adrenergic blockade reduces or eliminates stress-induced alcohol- and food-seeking in rats (Lê et al., 2011), and multiple randomized controlled trials indicate that beta adrenergic blockade reduces aggression in humans (Goedhard et al., 2006;

Haspel, 2009). In sum, the experimental evidence suggests a potential facilitative role of heightened SNS activity in the pathophysiology of emotion-driven impulsivity.

Observational studies also suggest that SNS activity may be altered in clinical syndromes characterized by emotional impulsivity. Studies of individuals with borderline personality disorder, a psychiatric disorder characterized primarily by very high levels of neuroticism (Mullins-Sweatt et al., 2012) and impulsive behavior in response to negative emotion (Chapman et al., 2010; Peters et al., 2013), generally find greater tonic sympathetic activity (measured using cardiac pre-ejection period (PEP), a systolic time interval highly correlated with cardiac SNS innervation) in BPD patients compared to healthy controls (Ebner-Priemer et al., 2007; Kuo and Linehan, 2009; Weinberg et al., 2009). Taken together, findings suggest higher SNS arousal may contribute to emotion-driven impulsivity and associated syndromes.

In contrast, a number of studies have found seemingly opposite relations between SNS and generalized disinhibition, with suppressed baseline SNS characteristic of individuals with higher scores on unidimensional measures of impulsivity (Maniaci et al., 2017; Muñoz and Anastassiou-Hadjicharalambous, 2011), more errors on passive avoidance tasks (Muñoz and Anastassiou-Hadjicharalambous, 2011), and externalizing diagnoses in childhood (Beauchaine et al., 2001; Crowell et al., 2006), although findings are not consistent (Beauchaine et al., 2015; Karalunas et al., 2014; Musser et al., 2011). Several factors could explain these seemingly divergent findings. First, impulsivity is increasingly recognized as a multidimensional construct (Whiteside and Lynam, 2001), and suppressed tonic SNS findings may reflect forms of impulsivity not driven by emotion, such as a broader tendency toward disinhibited

action. Indeed, the aforementioned studies use measures of impulsive behavior (both self-report and behavior) not linked to distress. Second, heightened SNS activation may only translate into greater emotion-driven impulsivity among individuals who tend to experience more intense and difficult to control negative affect, suggesting the need to consider trait-level moderators such as neuroticism.

1.2 The Current Study

Despite the evidence linking urgency-related phenotypes to SNS functioning, no study to date has directly examined associations of negative urgency with tonic SNS functioning. We sought to clarify the role of tonic SNS in facilitating the link between neuroticism and negative urgency, including examining the potential mechanism of hostile reactivity to stressors. Higher tonic SNS activation was hypothesized to moderate the association of neuroticism with negative urgency, as well as with impulsive behavior in the context of a distressing behavioral task. These interactive effects were also examined on affective reactivity to the behavioral task, given the potential for differences in affective reactivity to relate to differences in emotion-driven impulsivity.

2. Method

2.1 Participants

Participants were 203 introductory psychology students who completed the study in partial fulfillment of course requirements and $80 payment. Due to missing or unusable physiological data for several participants, the final sample N was 194. Participants' ages ranged from 18-23 (M = 18.49 years, SD = 0.72), and 51% were female. The racial and ethnic characteristics were as follows: 78% White/Caucasian;

13% Black/African-American; 3% Asian; 3% Latinx; 2% Biracial; and 1% Other. "High

risk" participants were over-recruited to ensure sufficient variability in impulsive behavior

within this sample. Students completed a screening questionnaire during a mass testing

early in the semester. This measure assessed conduct problem behaviors prior to age

18, such as stealing, lying, and fighting. Those whose scores fell within the top 25% for

their gender were specifically invited to participate through email; in addition, the study

was listed on a website accessible to the general research pool.

2.2 Procedure

All study procedures were approved by the local institutional review board. The

current study was part of a larger longitudinal project designed to assess multiple

predictors of impulsive behavior in young adults; the data for the present study are

taken from the baseline visit. Other studies have been published using data from this

project (Derefinko et al., 2016a; 2016b; Roberts et al., 2014); none include the PASAT-

C task or examine the hypotheses in question. At the beginning of the session,

participants were fitted with electrodes for the collection of impedance cardiography

data, which were recorded using a custom bioamplifier from Mindware Technologies

(Bionex Model 50-371100-00; Gahanna, OH). The signal was digitized with Mindware

BioLab software at a sampling rate of 1 kHz. During the first session, participants

completed a two-minute physiological baseline (a measure of tonic levels of SNS

activity) while sitting quietly. Just prior to this baseline period, participants were given

the following instructions: "Right now we are going to gather a 'snapshot' of your

baseline state before the task begins. I need you to sit quietly for approximately two

minutes while I record your readings." Following these instructions, participants looked

at a blank computer screen with a simple black-and-white fixation point for two minutes.

Physiological measurements were also obtained during subsequent tasks (Derefinko et

al., 2016b) not included in the present study. Next, participants completed personality

measures, followed by a standardized laboratory stress task (see below).

2.3 Pre-ejection Period (PEP).

SNS influences on the heart were quantified using impedance cardiography. PEP

was quantified as the period of time in milliseconds between the onset of ventricular

depolarization (Q-wave of ECG) and the opening of the aortic valve (B point of

dZ/dt waveform (Lozano et al., 2007). PEP reflects myocardial contractility and is

commonly used as an index of sympathetic cardiac control (Berntson et al., 2004).

Lower PEP values (i.e., shorter cardiac latencies) indicate higher cardiac sympathetic

activity*.* Tonic PEP was computed by taking the average level of PEP during the two-

minute period.

2.4 Psychological Measures

2.4.1 Neuroticism. The Five Factor Model Rating Form (Mullins-Sweatt, 2006)

asks participants to rate themselves on the 30 facets of the Five Factor Model of

personality. Individual facets were combined to construct the domain of Neuroticism. To

avoid criterion contamination, the impulsivity item was excluded from the measure in the

present study. The Neuroticism subscale (without the impulsivity item) demonstrated

good internal consistency in the current sample ($\alpha = 0.86$).

2.4.2 Emotion-Driven Impulsivity. Emotion-related impulsivity was measured

using the *negative urgency* subscale from the UPPS Impulsivity scale (Whiteside and

Lynam, 2001). This questionnaire provides a dimensional measure of the tendency to

engage in regretted action during or following experiences of negative emotion. Example items include, "It was hard for me to resist acting on my feelings" and "I often make matters worse because I act without thinking when I am upset". Internal consistency in the present sample was good ($\alpha = 0.89$).

2.5 Stress Reactivity.

The Paced Auditory Serial Addition Task – Computerized Version (PASAT-C; Lejuez et al., 2003) is a computerized serial addition task that has been validated and widely used as a standardized laboratory stress induction. The task involves the rapid addition of numbers, involving the recall of previously presented numbers no longer on the screen. Explosion sounds occur when participants make mistakes or fail to respond quickly. The task involves three levels. The first level is 115 seconds in duration and the second level is 175 seconds in duration, with presentation of task stimuli increasing in speed from the first to the second level. The third level occurs at the same speed as the second and lasts as long as 240 seconds, but participants are told they may quit the task whenever they choose. The time spent in the third, most stressful level of the PASAT prior to quitting is used in the present study as a behavioral measure of impulsive behavior (premature quitting), likely as an attempt to avoid further distress. In addition, the PASAT-C includes a pre- and post-task version of the Positive and Negative Affect Scale (10-item PANAS; Watson et al., 1988) that asks participants to rate the extent to which they are currently experiencing 10 different distress-related emotions using a visual analog scale. In the present study, we utilized the full Negative Affect score from the PANAS as a manipulation check to verify that the PASAT-C was indeed distressing. We also used pre-to-post change scores in the items "distressed",

"afraid", "hostile", and "ashamed" as affective outcomes, to examine outcomes across a

range of specific, potentially relevant affective states.

3. Results

3.1 Data Screening

Data were analyzed using SPSS 24. Descriptive statistics for primary study

variables are presented in Table 1. Screening for skewness and kurtosis revealed that

all variables fell within normal limits (Tabachnick and Fidell, 2000) with the exception of

PASAT-C quit time, which was heavily skewed. A log transformation was applied to this

variable, which was a sufficient correction to normalize residuals in later regression

analyses.

3.2 Manipulation Checks: Impact of PASAT on Affect

Next, manipulation checks examined whether the PASAT-C had functioned as a

stressor, increasing subjective distress. Consistent with previous reports, paired sample

t-tests revealed that the PASAT-C was associated with increased global self-reported

negative affect on the PANAS (Mean Difference Pre-to-Post = 1.21, $t(193) = 15.71$, $p <$

.001). This demonstrates that the PASAT-C functioned, as expected, as a subjectively

distressing task.

3.3 Zero-Order Correlations

Table 1 presents descriptive statistics for and zero-order correlations between

primary study variables. Female sex was associated with longer persistence on the

PASAT-C and no other variables; accordingly, female sex was only included as a co-

variate in subsequent models involving the PASAT-C[1]. Age was not associated with any

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[1] Inclusion of female sex in these models did not affect patterns of significance.

variables and therefore was not included in any subsequent analyses. Neuroticism was positively correlated with Negative Urgency; neither was correlated with PASAT-C quit time. Tonic PEP was not significantly correlated with any variables.

Changes in PANAS affective items were generally uncorrelated with one another, with the exception that changes in the Distress item were positively correlated with changes in both the Hostility and Shame items. Neuroticism was associated with greater increases in Fear and Hostility during the PASAT, whereas Negative Urgency was only associated with greater increases in Hostility. PASAT-C quit time (reverse-scored so that higher scores represented earlier quitting/more impulsive behavior) was also associated with a greater increase in distress during the PASAT-C. PEP was not correlated with changes on PANAS affect items.

3.4 Interactive Effects of Neuroticism and SNS Activity on UPPS Impulsivity and PASAT-C Quit-Time

To test the primary study hypotheses, OLS hierarchical multiple regression models predicted Negative Urgency and quit-time on the PASAT-C from 1.) PEP (measuring SNS activity, with greater PEP indicating lower levels of SNS activation) and Neuroticism, and 2.) interactions of Neuroticism with PEP (Table 2). Models were estimated and probed using the Process (Hayes, 2013) macro in SPSS. PEP and Neuroticism were z-scored prior to use in models. To illustrate the nature of interactions, significant interactions between the continuous variables were probed for simple slope estimates at -1 SD and +1 SD of PEP.

Consistent with predictions, there was a significant interaction between Neuroticism and PEP predicting Negative Urgency (Figure 1). Specifically, shorter tonic

PEP (higher SNS activity) unmasked a strong positive link between Neuroticism and Negative Urgency (estimate for Neuroticism at -1 SD PEP = 0.66, t = 7.32, p < .001) that was less pronounced among those with longer tonic PEP (lower levels of SNS activity; estimate for Neuroticism at +1 SD PEP = 0.33, t = 3.66, p < .001). In sum, greater tonic sympathetic activity facilitated a relationship between Neuroticism and Negative Urgency, whereas Neuroticism was less robustly associated with Negative Urgency at lower levels of tonic sympathetic activity. The hypothesis that this pattern would extend to the behavioral outcome on the PASAT-C was not supported, with no significant interaction of Neuroticism with PEP predicting PASAT-C quit time (p = 0.13).

3.5 Interactive Effects of Neuroticism and SNS Activity on Forms of Affective Reactivity

Models predicting each of the four forms of PASAT-C-related affective reactivity (Distress, Fear, Hostility, and Shame) can be found in Table 3. Models predicted post-task affect scores, controlling for pre-task affect in Step 1, to model task-based reactivity. Then similar to the previous tests, Step 2 added PEP and Neuroticism and Step 3 the interaction of Neuroticism with PEP. Models revealed no significant interactive effects of Neuroticism and PEP on PASAT-C-related changes in distress or fear. However, PASAT-C-related changes in Hostility and Shame were predicted by the interaction of Neuroticism and PEP, in opposite directions (see Figure 2). Among individuals with *shorter* tonic PEP (higher SNS activity), higher levels of Neuroticism was associated with greater Hostility reactivity during the PASAT-C (estimate for Neuroticism at -1 SD PEP = 0.31, t = 2.97, p = .003), but was *not* significantly associated with Shame reactivity (estimate for Neuroticism at -1 SD PEP = -0.05, t = -

0.54, p = 0.59). However, among individuals with longer tonic PEP (lower levels of SNS activity), greater Neuroticism was associated with greater Shame reactivity during the PASAT-C (estimate for Neuroticism at +1 SD PEP = 0.25, t = 2.47, p = .02) and was not significantly associated with PASAT-C-related changes in Hostility (estimate for Neuroticism at +1 SD PEP = -0.01, t = -0.07, p = .95).

3.6 Post-hoc Mediated-Moderation of the Association Between Neuroticism and Negative Urgency by Affect and Sympathetic Activity

To examine the potential role of reactivity in Hostility and Shame as mechanism underlying the PEP-moderated link between Neuroticism and Negative Urgency, a post-hoc moderated-mediation model was estimated using Process (Hayes, 2013) producing 95% bias-corrected confidence intervals (CIs) with 5000 bootstrap samples. Indirect paths were modeled from Neuroticism to Negative Urgency via changes in Hostility, and PEP was entered as a moderator of 1.) the effect of Neuroticism on affect change and 2.) the direct effect of Neuroticism on Negative Urgency. Significant mediated-moderation was demonstrated from Neuroticism to Negative Urgency via increased Hostility, moderated by PEP (95% CI index of moderated mediation via Hostility = -0.088 to -0.001; see Figure 3). Moderation of the path from Neuroticism to Hostility by PEP was significant (B = -0.16, p = .019). Probing the indirect effect from Neuroticism to Negative Urgency via increased Hostility demonstrated a significant path at low levels of PEP (high levels of SNS activation; 95% CI for indirect effect at -1 SD PEP = 0.005 to 0.147), but not at mean (95% CI for indirect effect at mean PEP = -0.0003 to 0.087) or high levels of PEP (95% CI for indirect effect at +1 SD PEP = -0.052 to 0.044). PEP also moderated the direct path from Neuroticism to Negative Urgency (B = -0.13, p =

.040), as well as a conditional effect of PEP on Negative Urgency (B = -0.13, p = .036).

A similar model was examined substituting changes in Shame for Hostility, given the

unexpected interaction found for Shame; however, no significant direct, indirect, or

moderated effects were found. In sum, only for individuals with shorter PEP intervals at

rest (higher tonic SNS activation), greater increases in Hostility accounted for part of the

relationship between Neuroticism and Negative Urgency.

4. Discussion

These findings add to a growing body of literature delineating the

pathophysiology of emotion-driven impulsivity. As predicted, baseline lower PEP

intervals interacted with neuroticism to predict higher self-reported negative urgency in

the present sample. These findings suggest that for individuals broadly more prone to

negative affect, those who also have higher levels of tonic SNS activation may be at

greatest risk for impulsive behavior when upset or stressed. In a post-hoc test, these

effects were accounted for in part by increased hostility in response to a stressful task,

suggesting that greater susceptibility to angry affect in particular may play a role in how

SNS activation facilitates vulnerability to affect-related impulsive behavior. This is

consistent with previous research demonstrating that anger, unlike most forms of

negative affect, is strongly linked to the behavioral approach system (Carver, 2004;

Harmon-Jones, 2003) and approach-related neural activation (Harmon-Jones and Allen,

1998; Herrero et al., 2010). This potential emotion-driven increase in approach

motivation could fuel the range of risky behaviors exemplified in negative urgency, such

as reactive aggression (Derefinko et al., 2011), risky sexual behavior (Deckman and

Nathan DeWall, 2011; Derefinko et al., 2014), and binge-eating (Fischer et al., 2013;

Wenzel et al., 2014). While the present study was conducted in a non-clinical sample, this model is also consistent with clinical findings of heightened tonic or baseline SNS activation in BPD (Ebner-Priemer et al., 2007) and bulimia nervosa (Monteleone et al., 2011), disorders characterized by heightened neuroticism, anger reactivity (Engel et al., 2007; Gardner et al., 1991), and emotion-driven impulsivity (Claes et al., 2005; Peters et al., 2013). Given that this moderated-mediation model was conducted as a post-hoc test, further research specifically designed to examine this model further is warranted.

PEP moderated the association between neuroticism and increased elicitation of both hostility and shame during the PASAT-C, in opposite directions. Individuals higher in neuroticism and with lower PEP (higher tonic SNS activity) reported greater increases in hostility following the task, whereas those with higher levels of neuroticism and higher PEP (lower tonic SNS) demonstrated greater increases in feeling ashamed. Anger-generating processes, such as anger rumination, have been shown to increase SNS activation (Ray et al., 2008); however, the present findings suggest that elevated tonic SNS activation may also facilitate hostile reactivity. Only increased hostile reactivity to the stressor was a significant factor in the larger moderated-mediation model predicting negative urgency; however, the higher neuroticism/lower SNS profile that may increase shame reactivity could in turn increase risk for other types of maladaptive behaviors and outcomes, perhaps including internalizing responses such as increases in avoidance and depressive symptoms.

Contrary to predictions, the interactive pattern of findings was not replicated for PASAT-C quit time. PASAT-C quit-time also did not demonstrate a significant association with negative urgency. It may capture a different aspect of impulsivity such

as lack of perseverance, the tendency to give up on boring or difficult activities

prematurely (Whiteside and Lynam, 2001), or may reflect low distress tolerance but not

negative urgency per se. This may also be affected by the lack of contingencies used

with the task, with no consequences applied to individuals who quit prematurely, which

may not adequately model the rash or regrettable action component of negative

urgency. Willingness to quit the task early might also reflect lower agreeableness (with

agreeable people trying harder to persist with the requested task and disagreeable

individuals demonstrating less concern for the research) for some individuals, rather

than impulsivity or difficulty tolerating distress, especially given the over-recruitment for

conduct problems for this sample. Future research should examine these interactive

effects with other types of behavioral impulsivity tasks and across a broader range of

samples. Findings to date across impulsivity research also generally demonstrate

surprisingly low concordance between self-report and task-based measures (Cyders

and Coskunpinar, 2012; 2011), so this null finding may reflect a common broader

assessment issue. While the null findings for the PASAT-C warrants further

investigation given that PASAT-C scores have demonstrated modest associations with

psychopathology and real-world outcomes in a limited set of studies (Leyro et al., 2010),

self-reported negative urgency is a more robust predictor of a wider range of real-world

dysregulated behavior (Berg et al., 2015; Settles et al., 2012).

**4.1 Does the Pathophysiology of Emotion-Related Impulsivity Differ from that of
Non-Affective Impulsivity?**

While preliminary and conducted in a non-clinical sample, the effects of tonic

SNS activity on negative urgency in the present sample may help to clarify conflicting

results in the literature regarding the role of the SNS in impulsivity. In contrast to the

deleterious associations of higher tonic SNS activity in urgency-related phenotypes and

facilitating the link between neuroticism and emotion-driven impulsivity in the present

study, an extant body of literature suggests that lower tonic levels of SNS activity, as

well as reduced SNS reactivity, characterizes clinical groups exhibiting non-affective

impulsivity (i.e., lack of premeditation and sensation-seeking), such as attention deficit

hyperactivity disorder, conduct disorder, and antisocial personality disorder (Beauchaine

et al., 2013; Lorber, 2004). While on the surface, these forms of impulsive behavior can

appear similar, they may involve different underlying biological alterations. Clearly

differentiating between forms of impulsive behavior and symptoms in future research

that utilizes clinical samples, as well as conducting studies clearly contrasting these

forms of impulsive behavior, may clarify some of the divergent findings on physiology

and impulsivity and related psychopathology.

4.2 Limitations

 Several limitations of the present study should inform future work in this area.

First, the cross-sectional nature of our sample precludes any conclusions about causal

effects of the SNS on negative urgency. Experimental work examining the impact of

beta adrenergic receptor blockade across various behavioral manifestations of negative

urgency (e.g., aggression, self-injury, binge eating, substance abuse) would provide a

clearer test of these hypotheses. Second, the present study utilized a student sample

over-recruited for risky behavior; the results may not be fully generalizable to clinical

samples. However, use of this sample allowed for examination of the constructs across

a broader range than would be present in a purely clinical sample. Third, this sample

was relatively small for the more complex models presented, with only 51% power to find some of these effects. Further work is needed to examine these models with more powerful designs. Also, only tonic SNS activation was assessed in the present study; further research should attempt to replicate these findings while adding assessment of SNS reactivity to various types of tasks and stimuli. Finally, the period of psychophysiological measurement was relatively short compared to other similar studies, lasting only two minutes; future research should examine physiological functioning over longer periods.

4.3 Conclusion

Among individuals with greater vulnerability to negative affect, those with generally elevated SNS arousal may be predisposed to hostile responses to stress and emotion-driven impulsive behavior. In contrast, those with generally lower SNS arousal may be more likely to display shame responses to stress. Further work is needed to examine how experimental manipulations of the SNS may influence affective reactivity and negative urgency and how elevated SNS activity may contribute to the expression of impulsive behavior and psychopathology in daily life.

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