Anxiety and its Effect on Sympathetic Nervous System Reactivity to Psychosocial Stress in Young Adults

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Abstract

Very few studies on sympathetic nervous system response to psychosocial stress take trait-like anxiety-related symptoms into account. Anxiety is characterized by unpleasant feelings of tension and apprehensive and worried thoughts. The present study reflects a secondary data analysis of an existing dataset to test hypotheses about trait anxiety's effects on the sympathetic nervous system response to a social stress induction, specifically levels of the sympathetic biomarker salivary alpha amylase (sAA). A total of 135 individuals participated in one of three conditions of the Trier Social Stress Test (non-stressful control condition, intermediate difficulty condition, very difficult negative evaluative condition), a negative evaluative social stress test, and provided repeated saliva samples for analysis. In addition, participants completed three questionnaires capturing different aspects of trait-like anxiety: the Overall Anxiety Severity and Impairment Scale, the Social Interaction Anxiety Scale, and the Anxiety Sensitivity Index which captures anxiety about physiological sensations. Based on previous research, I hypothesize that high in trait-like anxiety would predict higher alpha amylase reactivity to increasingly challenging levels of the Trier Social Stress Test. While increasing severity of stress condition significantly predicted greater sAA reactivity, contrary to expectations, none of the anxiety measures significantly predicted sAA reactivity alone or in interaction with stressor severity. The study found that anxiety did not cause significant difference in sAA production.

Keywords: anxiety, Trier Social Stress Test, social anxiety, SNS, stress

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Stress is characterized in the body in many different ways. Numerous neurotransmitters, enzymes, and hormones are secreted in a context of "flight or fight" response (Nater et al., 2005). The sympathetic nervous system, or SNS, is the system activated in these "flight or fight" responses. However, I am specifically interested in alpha amylase and its secretion during stress. Recent work shows that salivary alpha amylase (sAA) functions as a reliable and valid biomarker measuring stress reactivity in the sympathetic nervous system (Nater & Rohleder, 2009). It can be found in the saliva and is shown in greater amounts when people are exposed to environmental stress. This study accounts for differential variability when observing saliva samples, which is the phenomenon that people have different responses to the same stimuli (Monnery-Patris et al., 2015). However, only a minority of individuals have very negative responses to stress exposure, suggesting considerable variability across individuals.

It may be that pre-existing anxiety-related traits or symptoms account for some of their variability. In a study on stress as a predictor for depression in two groups of emerging young adults, the results showed evidence that two interpersonal forms of recent stress are significant unique predictors of major depressive episodes onsets during emerging adulthood (Vrshek-Schallhorn et al., 2015). Anxiety itself is defined as an emotional response involving apprehensive and worried thoughts and unpleasant feelings of tension. In addition, anxiety prompts avoidant and conservative behavior (Wilt, Oehlberg, & Revelle, 2011). The present study extends previous research on sympathetic reactivity to stress by looking at how different anxiety constructs, particularly social anxiety, can predict changes in the amount of alpha amylase secreted in response to a brief, controlled stressor in a laboratory environment.

The Sympathetic Nervous System

In the body, there are two stress response systems, sympathetic nervous system (SNS) and hypothalamic pituitary adrenal (HPA) axis. SNS governs the body's involuntary response to stressful or dangerous situations, the "flight or fight" response (Alshak & Das, 2019). One approach to probing the etiology of anxiety-related processes is examining sympathetic nervous system reactivity to stress exposure. The emergence of alpha amylase as a biomarker for stress reactivity may shed light on anxiety related processes (Nater & Rohleder, 2009). Salivary alpha amylase is an enzyme found in the saliva of humans that functions to begin the digestion of starches.

The autonomic nervous system (ANS) controls the salivary glands and releases sAA. In cases of high psychosocial stress, autonomic activation is high, thus increasing ANS activity. The SNS is a part of the ANS with sAA used as a marker of the SNS. This is evidence that sAA is a marker of the SNS due to the fact that the release of sAA from salivary glands is under local sympathetic nerves' control and increases when these pathways are stimulated (Nagy et al., 2015). In fact, sAA may offer advantages over another biomarker of autonomic reactivity that has been used previously, namely heart rate, because in contrast to heart rate, sAA offers an index of sympathetic rather than a combined index of both branches of the ANS, sympathetic, and parasympathetic. Thus, probing sAA reactivity may shed light on individual variability in sympathetic stress reactivity to stress exposure.

The Diathesis-Stress Model

One theoretical model that invokes individual variability in response to stressors is the diathesis-stress model. This diathesis-stress model suggests that an interaction between a predispositional vulnerability and stress can account for the onset of psychopathology (Monroe

& Simmons, 1991), but it can also be applied to study biological responses to stress. A study looked at how genetic influences on stress reactivity could provide insight into the depression risk mechanism (Avery and Vrshek-Schallhorn, 2016). Non-depressed young adults provided a DNA sample and completed either the control protocol or the negative evaluative Trier Social Stress Test. The results of the study supported a role of an interaction between genetics, stress reactivity, and depression risk, providing an example of how the diathesis-stress model works. Knowing the relationship between stress and the vulnerability to it will help to determine the physical reactivity a person can have to stress. Being aware of genetic and environmental factors that put those at a higher predisposition for things like stress makes researchers more aware of physiological responses. In defining the model, Monroe and Simmons also noted that the magnitude of events that increase vulnerability to stress and the quality of these events can help predict the disorders one might have a higher likelihood of experiencing. In looking at these events and their impact, we can theoretically find a relationship between biological reactivity to stress and a higher sensitivity to vulnerability for disorders. With an understanding of the diathesis-stress model, anxiety constructs can be used to predict biological reactivity to stress.

Anxiety-Related Traits and Symptoms

Anxiety refers to an emotional state including feeling of apprehension, nervousness, worry, and tension accompanied by physiological arousal (Spielberger, 2010). Symptoms refer to signs of anxiety individuals experience such as tension and worried or fearful thoughts that may wax and wane over time, while traits refer to longstanding, stable characteristics akin to personality. Anxiety-related traits and symptoms have previous been implicated in heightened stress-responding (Katsuragi et al., 1999). In this research, three anxiety constructs will be used to test how pre-existing anxiety-related constructs and stress exposure interact to predict

sympathetic reactivity to stress. The first construct is anxiety symptoms in young adults. Anxiety symptoms can include, but are not limited to, breathing rapidly, having difficulty controlling worry, sweating, trembling, feeling weak or tired, having trouble concentrating, and feeling nervous, restless, or tense (Mayo Clinic, 2018). For this study, I am particularly interested in observing these symptoms during the Trier Social Stress Test and how participants rate their experience of symptoms like these during the experiment. This will be most important to observing the effect of anxiety symptoms on sympathetic reactivity.

In addition to anxiety symptoms, I am particularly interested in social anxiety. Prior research has looked at the sAA secretion levels in people diagnosed with general social anxiety disorder. When compared to the control group, those with general social anxiety disorder had significantly higher diurnal salivary alpha amylase levels (Schumacher et al., 2013). Social anxiety, or social phobia, is defined as a marked and persistent fear of one or more social situations in which a person is exposed to people unfamiliar to them or to possible negative feedback or scrutiny by others in DSM-IV. It is often associated with significant impairment in social functioning as well as educational and occupational attainment (Caballo, Andres, & Bas, 1998). Social anxiety requires that an individual be doing something while others are watching and, to some extent, evaluating their behavior. This is particularly important to this study since participants will be undergoing a negative evaluative social stress test. Thus, social anxiety is of more interest than other anxiety constructs. However, there is still an interest in a person's sensitivity to anxiety.

Anxiety sensitivity is the fear of anxiety-related sensations. Those with anxiety sensitivity tend to harbor beliefs about the harmfulness of some bodily functions, like heart palpitations being a heart attack. It is also marked as being more aversive to episodes of fear and avoiding

things they believe may cause a panic attack (McNally, 2002). In addition, anxiety sensitivity is marked more by anticipatory anxiety rather than anxiety brought on by the actual stimuli. These three constructs of anxiety provides support to my hypothesis of how anxiety will effect sympathetic nervous system reactivity to psychosocial stress.

Based on what is known about the diathesis-stress model, I predict that participants high on the measures used are more likely to have higher alpha amylase reactivity to the Trier Social Stress Test (TSST), as a function of experimental condition, with increasing difference between people with lower versus higher anxiety traits as condition difficulty increases (control, intermediate, and challenge). It is possible that somewhat larger effects for the Social Interaction Anxiety Scale versus the other two constructs will be observed since the TSST is a social negative evaluative stressor; however, this study is unlikely to be powered to detect significant differences in interaction effects across the three constructs.

Method

Participants

Young adult participants enrolled in undergraduate psychology courses ranging from 18 to 30 years old were eligible to be tested in this study. There were 128 participants, with 44 control, 46 intermediate, and 38 in the challenge condition. Participants were screened through a mass screening procedure or by emailing the study coordinator. Exclusion criteria were: use of hormonal birth control, nicotine, corticosteroid medication, or psychotropic prescriptions (e.g., Adderall, antidepressants); being colorblind; history of head injury; being a non-native English speaker (due to the stressful nature of the speaking task); and current depression assessed with the depression section of a semi-structured clinical interview. 66 of the participants identified as males and 62 of the participants identified as females. No participants endorsed transgender or

gender fluid identity. Of the 128 participants, 49 identified as Causcasian (non-Hispanic), 42 identified as Black/African American, 18 identified as Biracial, 9 identified as Asian/Pacific Islander, 5 identified as other, 4 identified as Hispanic/Latino, and 1 identified as American Indian/Alaskin Native.

Materials

Saliva Samples. Saliva was sampled using passive drool into sterile, RNAse and DNAse free cryogenic vials. These vials were frozen at -20 degrees immediately following each session and stored until shipment in one group on dry ice via express courier to the University of Trier Biochemistry Laboratory for analysis.

Overall Anxiety Severity and Impairment Scale (OASIS). OASIS can assess the severity and impairment that is associated with any anxiety disorder(s) using a five item self-report (Campbell-Sills et al., 2009). Not only does OASIS measure the severity and frequency of anxiety, but it also measures the level of avoidance and interference from activities and social interactions that are associated with anxiety. It consists of 5 questions regarding anxiety and fear, capturing overall anxiety symptoms. OASIS uses a 0 to 4 scale, 0 referring to no symptoms or impairment to 4 referring to the most symptoms and impairment. An example of one of the questions on OASIS is “In the past week, how often have you felt anxious?”

Social Interaction Anxiety Scale (SIAS). SIAS examines social interactional anxiety, or the extreme distress that occurs when initiating and maintaining conversations with others (Brown et al., 1997). It consists of 20 statements regarding social anxiety that participants are supposed to rate whether they are characteristic or true for them. SIAS uses a 0 to 4 scale, with 0 being “not at all” and 4 being “extremely.” It works to gage generalized social fears, contributing

to an insight on social anxiety. The scale uses items like "I get nervous when I have to speak with someone in authority (teacher, boss, etc.)."

Anxiety sensitivity index. The anxiety sensitivity index is used to measure the fear of the fear response itself, a personality variable of anxiety sensitivity (Peterson & Heilbronner, 1987). The index uses a 16-item measure to assess how anxiety symptoms are believed to affect a person socially and somatically. The fear of fear that the index is measuring contributes to avoidance behavior, anxiety disorders, and fear learning. It uses a scale of 0-4 for these items to rate the frequency of their agreement with the statements, with 0 being "never" and 4 being "always." Anxiety sensitivity index uses items like "It scares me when my heart beats rapidly."

Three Condition Trier Social Stress Test.

The Trier Social Stress Test (TSST) is a standardized lab-based psychosocial stress induction. All three conditions of the TSST participate used here (control, intermediate, and negative evaluative challenge conditions) in two activities, a speech task and a math task, with varying levels of negative evaluation from members of the research team staff. Further, in all conditions, saliva samples were collected for sAA production at the beginning of the experiment, immediately following the experiment after any confederates left the room (+20 minutes), and after the debriefing (+45 minutes). Participants are told they will pick a topic from a box of papers and have 5-minutes to prepare a 5-minute speech. To begin, the facilitator pretends to turn on a camera focused on the participant. Participants are told that the video recording will be viewed by multiple people at a later date but are informed in the debriefing that this was not true. In the control and intermediate conditions participants are told they will not be evaluated, but participants in the challenge condition are told they will be evaluated. The participant picks their topic from the box, is given a piece of paper, and is told they have five minutes to prepare. At the

end of the five minutes, the paper is taken and the participant begins their speech. All participants were told to stand for the entirety of the speech to control for the influence of body position on sympathetic reactivity. In the control condition, the experimenter facilitated this part of the experiment out of the eye gaze of the participant. In the intermediate condition, there is one confederate facilitating this portion of the experiment who enters at the beginning of the Trier Social Stress Test. During the TSST, the facilitator in the intermediate section keeps a completely neutral face and body language; they give no positive or negative feedback. For participants in the challenge condition, they face two confederates during the TSST. The two confederates follow a script with non-verbal negative feedback to give during the social performance part of the TSST that conveys boredom or dissatisfaction with the speech. For example, at different parts of the speech the confederate was directed to roll their eyes or sigh. During all conditions the participant is reminded to continue speaking for the full five minutes if they pause too long. After the 5-minutes are up, the participant moves onto the second social event of the TSST. For this part, the participant has a mental arithmetic problem to work on for 5-minutes. They are asked to count backwards by 13 starting at 2,017. If a mistake is made they are asked to start over from 2,017. The challenge confederates continue their negative feedback during the second portion while the intermediate confederate remains neutral and the control condition remains led by the experimenter.

Manipulation Checks. Following the TSST, participants completed 3 items assessing the extent to which they felt evaluated overall, positively, and negatively.

Design

Data for this secondary analysis came from a larger study on stress reactivity. Participants completed one of three conditions of the Trier Social Stress Test, designed to vary in level of

exposure to negative evaluation (control, intermediate, or challenge), following pseudo-randomization. This means that participants were blind to the condition they were signing up for on Sona. Each participant completed self-report questionnaires measuring three different anxiety constructs.

Procedure

All procedures were approved by the Institutional Review Board of the University of North Carolina at Greensboro; participants provided signed, informed consent prior to the study, and signed permission to use the data following debriefing at the conclusion of each session. Participants completed a self-report questionnaire at the beginning of the study. All studies began between 1 and 4:30 in the afternoon to ensure that biomarker levels were not affected by the diurnal rhythm.

Statistical Approach

First, I examined whether groups differed in baseline biomarker levels or demographic characteristics. Next, several manipulation checks were examined whether the stress manipulation had the expected effect on perceived evaluation using several questions administered immediately following the TSST and on AUCI alpha amylase levels using an ANOVA. A linear contrast was used to test for the expected effect of TSST manipulation severity. Throughout analyses, reactivity in alpha amylase was indexed by calculating the area under the curve with respect to increase (AUCI using the three timepoints, which is an accepted measure of reactivity) (Pruessner, Kirschbaum, Meinlschmid, & Hellhammer, 2003). Alpha amylase was log transformed prior to AUCI calculation to address typically observed positive skew in the data.

For the primary analyses, I predicted sAA reactivity by entering it as the dependent variable in a multiple linear regression model with the following predictors: main effects of experimental condition (meaning level of stress acting on sAA by itself, coded as 0 for controls, 1 for the intermediate condition, and 2 for the negative evaluative condition), main effect of anxiety-related construct (meaning level of anxiety construct acting on sAA by itself), and the multiplicative statistical interaction of condition and anxiety-related construct. I centered all dimensional variables prior to calculating interaction terms. As planned, all models covaried main effects of three variables that have previously been shown to influence anxiety-related responding, which were gender identity, education of parents, and minority status. Gender Identity variable was coded with males as 1 and females as 0. For minoritized status, non-minority (white) was coded as 0 and minoritized status was coded as 1.

I planned to decompose any significant interaction effects by identifying the effect of anxiety within each experimental condition by itself. P-values <= .05 were considered statistically significant.

Results

Preliminary Analyses

First, screening for group differences indicated no concerns. Baseline alpha amylase did not significantly differ by group, F(2.193, 78.34) = 1.80. p = 0.169. This shows us that each group started out with the same alpha amylase levels and there was no significant difference that could alter the results. Parent education did not significantly differ by group (all ps > 0.169), nor did gender identification or identification with a minoritized racial/ethnic group (both c^2(2) <= .977, p >= 0.614); for group descriptive values, see Table 4.

Second, manipulation checks indicated that the three TSST conditions showed the expected significant linear contrasts on sAA, perceived overall evaluation, negative evaluation, and positive evaluation. Increasing TSST manipulation severity was associated with greater alpha amylase reactivity, $F(1,125)=4.613$, $p=.034$, greater perceived overall evaluation, $F(1,121)=24.514$, $p<.001$, and perceived negative evaluation, $F(1,103)=39.575$, $p<.001$, but lower perceive positive evaluation, $F(1,104)=27.067$, $p<.001$. Thus, I concluded that the TSST manipulation performed as anticipated and that sAA is sensitive to it.

Primary Analyses

Across all three models tested, there was consistently a significant main effect for condition severity (all *t*-values 1.997 to 2.202, all *p*-values .030 to .048), such that greater severity of stressor was associated with greater sAA reactivity. In no case did the trait-like anxiety indicator predict sAA reactivity either in main effect or in interaction with condition (all *p* values > .05). Full models are presented in Tables 2 through 4.

Discussion

The purpose of the present study was to examine the hypothesis that participants with trait-like anxiety expression would produce more salivary alpha amylase as negative evaluation level of a lab-based stress manipulation increased. If this were supported it would suggest that the sympathetic nervous system reactivity to stress exposure were associated with individual differences in symptoms of anxiety, and may underlie those symptoms. However, based on the results, despite a consistent effect of the stress induction on alpha amylase reactivity, there was no significant interaction between anxiety symptoms and stress induction severity level in predicting the production of salivary alpha amylase in response to stress exposure.

As seen in previous research conducted on stress and alpha amylase reactivity, our results are consistent with others' findings of a significant effect of stress exposure and sAA reactivity. Stroud et al. (2009) reported increased sAA secretion in a peer rejection paradigm in adolescents. In Nader and Rohleder (2009), the researchers found that, through a review of the prior research, there are multiple cases of sAA increases in response to psychologically stressful conditions, like mothers watching their children being exposed to a stressful task or an oral academic examination. In their study, they found few examples of non-significant changes in sAA productivity in response to stressful stimuli like a strange situation paradigm.

There are, however, several reasons why we may have failed to detect effects of anxiety related constructs on reactivity to a stress induction. The Trier Social Stress Test does focus on a social manipulation. Due to that, the stress induction could be the wrong match to the participant's type of anxiety. Despite our negative findings for social anxiety, if we did not have enough participants with elevations in social anxiety, and social anxiety is a stronger match to the social evaluation induction, we may be missing an effect. Future research could also address this by including other conditions more relevant to other aspects of anxiety, instead of only a social evaluation manipulation.

There could also the issue of the stress induction not being robust enough to cause enough of a range of physiological reaction to be able to detect differences between people. Despite this being the most robust social performance stressor we can expose participants to in the lab, other manipulations that use social rejection stress such as a simulated social media friend rejection could be an appropriate fit.

Additionally, although this sample is larger than many that utilize the TSST, we may have failed to detect an effect of anxiety related constructs on reactivity to a stress induction due

to sample size. It is possible there was a fairly small effect size of an interaction but there were not enough participants to provide statistical power to detect a small effect, a possible false negative finding. In addition, there might not have been enough participants with sufficiently severe anxiety to see the expected effect. The sample was not a clinical sample, that is, a sample with a diagnosed anxiety disorder, so an effect could be hard to detect among a small, non-clinical population. Last, there was no emphasis on social anxiety with the sample. Given that it is a social manipulation, a clinical subsample with diagnosed social anxiety could help to observe a greater effect.

Limitations

Despite several strengths, including using a controlled lab-based stress manipulation to ensure a known "dose" of stress exposure, and using an objective biomarker, this study is not without limitations. One of the limitations of the study is that the participants are recruited from a student population which may be high functioning. It was also not a clinical population. Another limitation of the study was that only salivary alpha amylase was used as a biomarker of the Sympathetic Nervous System. Further research could look at ECG pre-ejection period to gauge the contractility of the heart, a gold standard measure of SNS functioning, or other biomarkers of SNS reactivity.

Conclusions

Taken together, these results suggest that anxiety does not influence sympathetic functioning in interaction with stressor severity, however, consistent with prior research, increasing stress severity produced clear effects on increased sympathetic functioning. Future research could further probe whether other mental disorders, like depression, have any effect on salivary alpha amylase production.

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Table 1. Description of variables across three conditions

		Condition	Mean (or N if stated)	Standard Deviation
sAA production		00	4.4820	.81023
		1.00	4.7686	.84376
		2.00	4.5145	.65510
		Total	4.5947	.78539
Average Parent Education		00	5.0909	1.85921
		1.00	5.1413	1.51900
		2.00	5.6316	1.68733
		Total	5.2695	1.69518
ASI item mean		00	.4235	.37466
		1.00	.4563	.44283
		2.00	.4383	.29467
		Total	.4397	.37749
OASIS item mean		00	.3591	.55670
		1.00	.3348	.54005
		2.00	.1947	.28087
		Total	.3016	.48602
SIAS item mean		00	.6761	.37284
		1.00	.6951	.51037
		2.00	.7344	.39247
		Total	.7002	.42988
Gender status	0 Female	00	N = 22	
		1.00	N = 22	
		2.00	N = 18	
	1 Male	00	N = 22	
		1.00	N = 24	
		2.00	N = 20	
Minoritized Status	0 white	00	N = 16	
		1.00	N = 16	
		2.00	N = 17	
	1 minoritized	00	N = 28	
		1.00	N = 30	
		2.00	N = 21	

Table 2. Final Model Regression results for the Overall Anxiety Severity and Impairment Scale (OASIS) and Stress Induction Predicting sAA Reactivity

	Unstandardized B	Standard Error	T score	p-value
Gender Identity	1.713	2.246	.763	.447
Average education of parents	.716	.671	1.068	.288
Minoritized Status	4.373	2.305	1.898	.060
Centered Stress Condition	2.814	1.409	1.997	.048
Centered OASIS item mean	-1.548	2.652	-.584	.560
OASIS x stress	1.251	3.490	-.359	.721

Table 3. Final Model Regression results for the Social Interaction Anxiety Scale (SIAS) and Stress Induction Predicting sAA Reactivity

	Unstandardized B	Standard Error	T score	p-value
Gender Identity	1.775	2.232	.796	.428
Average education of parents	.725	.660	1.098	.274
Minoritized Status	4.240	2.341	1.811	.073
Centered Stress Condition	3.047	1.384	2.202	.030
Centered SIAS Item Mean	-3.407	2.588	-1.317	.190
SIAS x stress	2.252	3.708	.607	.545

Table 4. Final Model Regression results for the Anxiety Severity Index (ASI) and Stress Induction Predicting sAA Reactivity

	Unstandardized B	Standard Error	T score	p-value
Gender Identity	1.573	2.232	.705	.482
Average education of parents	.723	.675	1.070	.287
Minoritized Status	4.257	2.314	1.840	.068
Centered Stress Condition	2.977	1.394	2.136	.035
Centered ASI Item Mean	-1.109	3.055	-.363	.717
ASI x stress	.566	4.240	.133	.894