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The Relationship Between Public Speaking Performance, Interpersonal Factors, and Endocrine and Inflammatory Response to Stress

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An abstract of
A thesis submitted to the Faculty of Emory College of Arts and Sciences
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Abstract

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Introduction: While physiological measures as well as self-report have been widely employed to examine what individual characteristics alter one's response to acute laboratory psychosocial stress challenges, measures of overt behavior have less commonly been utilized. Measures of overt behaviors, like physiological measures, may reveal important information about anxiety that is otherwise suppressed during self-report.

Methods: Speech form during a standardized acute psychosocial stress challenge (the Trier Social Stress Test [TSST]) was analyzed for speech errors using criteria previously outlined by Lewin et al., (1996). Speech errors were quantified as verbal errors (i.e. correlations, repetitions) as well as procrastinating verbalizations. Speech form was then related to subjective distress responses to the TSST (Profile of Mood States [POMS]), endocrine (cortisol) and inflammatory (interleukin [IL]-6) responses to the TSST as well as to trait features including depression (Beck Depression Inventory [BDI]) and anxiety (Beck Anxiety Inventory [BAI]), and state features including perceived life stress (Perceived Stress Scale [PSS]) and life satisfaction (Satisfaction With Life Scale [SWLS]).

Results: Speech errors were not found to be associated with changes in circulating concentrations of cortisol or IL-6 as a result of challenge with the TSST. However, speech errors were found to be positively associated with change in POMS total score from before to immediately following the TSST. BDI, PSS, and SWLS scores were found to be associated with increased speech errors during the task. No significant relationship was found between speech errors and BAI scores.

Conclusion: These results suggest that measures of speech performance during an acute laboratory psychosocial stressor may be related to subjective distress experience during the same challenge, as well as depression features, perceived life stress, and life satisfaction.

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1 Background

It is well established that various individual characteristics can cause alterations in physiological response to acute psychosocial stress. The effects of mood and anxiety disorders on this response have been especially well studied. Although findings regarding how depression and anxiety alone affect physiological response to psychosocial stress are inconsistent, numerous studies have shown that those with either depression or anxiety and early life adversity have altered physiological responses to psychosocial stress (Pariante & Lightman, 2008, Elzinga et al., 2010, Harkness et al., 2011). Age (Kudielka et al., 2004), as well as gender and menstrual cycle (Kirschbaum et al., 1999), have also all been found to have an effect on this physiological stress response. The effects of various personality factors on this physiological response have also been studied and numerous studies show that one's physiological response to psychosocial stress can to an extent be predicted from one's personality (Oswald et al., 2006, Pruessner et al., 1997).

Also well studied have been the effects of interpersonal factors on participants' perceived distress as a result of a psychosocial stressor. However, very few studies have looked at how overt behaviors during an acute psychosocial stressor or challenge in laboratory settings are affected by interpersonal factors and how the same stress-induced behaviors are related to physiological response and self-reported distress. With regard to a specific behavior during laboratory stress challenge, the number of studies looking at speech quality during psychosocial stressor challenge is even fewer as studies of overt behavior during psychosocial stress also have focused on facial expressions and gaze behavior (Lerner et al., 2007, Kleinke 1986). Those that have looked at how speech changes as a result of psychosocial stress have focused on the changes in speech form due to the stress as well as changes in speech content. Some studies have looked at how speech form is affected by anxiety with results revealing that highly anxious subjects pause more and pause for longer durations during public speaking tasks (Hofmann et al., 1997, Lewin et al., 1996). Subjects high in speech anxiety also spoke fewer words and made speeches rated as less fluent during a recorded speaking task (Baggett et al., 1996). As well, anxious subjects have also been shown to exhibit more sentence change, repetition of words, stutter, omission of parts of words, sentence incompletion and tongue slip (Kasl & Mahl, 1965). In addition to these changes in speech form, changes in speech content due to anxiety also have been examined with results revealing that highly anxious subjects make speeches that are less linguistically efficient (Zohar et al., 2003). No studies to our knowledge have looked at the possible associations between overt speech behavior and physiological response to psychosocial stress.

There are a number of reasons why direct measures of overt behavior should be utilized alongside verbal report and measures of physiological response in assessing anxiety, fear, and phobia during acute psychosocial stress. It has been noted that speech dysfluencies in social situations are involuntary and thus might reveal information about anxiety that the participant suppresses for the verbal report (Hofmann et al., 1997). Similarly, others have warned about the exclusive use of self-report in assessing anxiety during psychosocial stress, recommending a multidimensional approach (Eifert & Wilson, 1991).

The primary goal of this study was to examine the extent to which various interpersonal factors have an effect on speech quality during a laboratory public speaking psychosocial stress test (the Trier Social Stress Test [TSST]). Associations between speech quality and endocrine and inflammatory response to the TSST, as well as self-reported distress in response to the TSST, were also examined. Speech quality was assessed using measures outlined in Lewin et al., (1996). These measures include evaluating both verbal errors as well as procrastinating verbalizations.

We hypothesized that there would be associations between physiological response to stress and speech quality. We also predicted that there would be associations between self-reported distress (determined by the Profile of Mood States [POMS]) and speech quality. We also predict that trait anxiety among other interpersonal factors will have an effect on speech form.

2 Methods

Procedures outlined here include stress test videos, various self-report assessments, and blood aliquots for measuring concentrations of immune and endocrine stress markers already collected as part of a larger parent study (The Emory CALM Study). The current project's focus was the assessment of stress test videos for speech quality in addition to statistical analyses of the relationship between speech quality and other study outcome variables.

2.1 Participants and self-report assessments

All participants were recruited from flyers posted throughout the Atlanta area as well as from ads placed in local newspapers. Participants were between the ages of 25 and 55, were medically healthy, and had no history of significant psychiatric illness. All participants also had a body mass index (BMI) less than 30. Demographic information of the participant population was representative of the greater metropolitan Atlanta area. All participants provided written informed consent. All completed the following questionnaires: Beck Depression Inventory (BDI), Beck Anxiety Inventory (BAI), Satisfaction With Life Scale (SWLS), and Perceived Stress Scale (PSS). Stress tests for all participants were between the 1 and 5 p.m. All study procedures were approved by the Emory University Institutional Review Board.

2.2 Trier social stress test (TSST)

The Trier social stress test (TSST) is a psychosocial laboratory stress test that reliably activates the hypothalamic-pituitary-adrenal (HPA) axis and the sympathetic nervous system (Kirschbaum et al., 1993), as well as inflammatory pathways (Pace et al., 2006). The test consists of both public speaking and mental arithmetic in front of an audience (Kirschbaum et al., 1993). Five minutes before the start of the test, participants were told specific instructions for the task. Participants were to take on the role of a job applicant and explain why they were the ideal candidate for a specific job to a committee of three people, who played the role of hiring managers. The job description was tailored to match each participant's interests and goals. Committee members were introduced to the participants as being specially trained to monitor nonverbal behavior. Participants were also told that the interview would be recorded and that voice frequency analysis of nonverbal behavior as well as video analysis would take place after the task. Participants had five minutes to prepare their speeches and then were asked to talk for five minutes in front of the committee. Upon completion of the speech, participants were then asked to serially subtract the number 13 from 1,022 as fast and accurately as possible and were asked to begin again if a mistake was made. After the task, subjects rested while blood draws were made. Blood draws were made additionally before the TSST, and time points for all blood draws are outlined in the following section.

2.3 Assessment of physiological response:

An indwelling venous catheter was placed in the antecubital vein of the non-dominant arm 90 minutes prior to the TSST to allow for acclimatization. Plasma cortisol concentrations were assessed prior to start of the stressor, and 15 min (T1), 30 min (T2), 45 min (T3), 60 min (T4), 75 min (T5), and 90 min (T6) after stressor initiation. As findings from a previous study from our group revealed a lag in IL-6 response (Pace et al., 2006), plasma IL-6 concentrations were assessed prior to the TSST and 90 min (T6) after the start of the stressor. Blood was collected before, during, and after the TSST into chilled EDTA-coated monovettes and centrifuged immediately. Plasma was stored at -80 °C until batch assay. Plasma IL-6 concentrations were measured by high-sensitivity enzyme-linked immunoassay (ELISA) (R&D Systems, Minneapolis, MN) and plasma cortisol concentrations were measured by ELISA (IBL International, Toronto, Ontario, Canada).

2.4 Assessment of subjective distress:

Distress levels were measured using the Profile of Mood States (POMS) both immediately prior to and following the TSST (McNair et al., 1992). General distress was measured by adding together scores on the tension-anxiety, depression-dejection, angerhostility, fatigue-inertia, and confusion-bewilderment subscales and subtracting scores on the vigor-activity subscale from this total.

2.5 Assessment of speech quality:

Speech quality assessment was carried out along the Lewin et al., (1996) paradigm. This includes seven measures of speech dysfluencies and two pause variables. A summary of the categories that were scored is in Table 1.

These categories will be examined as the speech dysfluencies and pause variables have been found to be consistent predictors of anxiety. A validation study (Krause & Pilisuk, 1961), which itself adapted the criteria from two similar earlier studies (Mahl 1956, Dibner 1956), found increased speech errors in these categories to be associated with anxiety.

2.6 Statistical analyses

Sample characteristics were evaluated descriptively by computing means and standard deviations for continuous variables. The relationship between speech errors and physiological as well as subjective distress responses to the TSST (expressed as changes from before to after the speech and math portion of the TSST) were analyzed using Pearson product-moment correlations.

3 Results

Demographic information for the sample is reported in Table 2.

Speech errors during the 5-minute speech portion of the TSST were analyzed according to six criteria. Total verbal errors (corrections, distortions, fragments, and repetitions), total procrastinating verbalizations (nonverbal intrusions, procrastinations, and qualifying phrases), as well as total verbal dysfluencies (total verbal errors plus total procrastinating verbalizations) were first computed. Subsequently, each of these cumulative variables was divided by total words spoken to compute three corresponding ratio variables.

Two raters independently rated four speeches apart from those included in the sample to assess reliability of the rating procedure. Pearson's correlation coefficient for the total number of speech errors was $r = .95$ ($r[4] = 0.952$, $df = 2$, $p < 0.048$).

Physiological and subjective distress measures before and during the TSST were aggregated for comparison to speech error variables. A variable representing initial change in cortisol due to the stressor was calculated by subtracting circulating cortisol concentrations prior to the speech from those thirty minutes after stressor start (i.e. delta cortisol). A variable representing change in IL-6 concentrations to the stressor was calculated by subtracting circulating IL-6 concentrations prior to the TSST from those after. Additionally, a variable representing initial change in subjective distress as a result of the stressor was calculated by subtracting POMS total score prior to the TSST from POMS total score immediately after.

3.1 Speech errors and interpersonal factors

Across the sample, depression features assessed with the BDI were found to be positively correlated with total verbal errors (r[31] = 0.337, df = 29, p = 0.032) (Figure 1) and total verbal dysfluencies (r[31] = 0.402, df = 29, p = 0.012). Of note, these variables' corresponding ratio variables were also positively correlated with depression feature (verbal errors: r[31] = 0.305, df = 29, p = 0.048, verbal dysfluencies: r[31] = 0.348, df = 29, p = 0.027). Additionally, perceived life stress assessed by the PSS was found to be positively correlated with total verbal errors (r[30] = 0.423, df = 28, p = 0.01) (Figure 2). As well, the verbal errors ratio was found to be positively correlated with perceived life stress (r[30] = 0.338, df = 28, p = 0.034). Perceived life stress was also positively correlated with total verbal dysfluencies (r[30] = 0.359, df = 28, p = 0.026), but the verbal dysfluencies ratio was found not to be correlated with perceived life stress (r[30] = 0.227, df = 28, p = 0.114). Life satisfaction (determined by SWLS scores) was found to be inversely correlated with both total verbal errors (r[31] = -0.493, df = 29, p = 0.002) (Figure 3) and total verbal dysfluencies (r[31] = -0.435, df = 29, p = 0.007) and the verbal errors ratio (r[31] = -0.454, df = 29, p = 0.005) as well as the verbal dysfluencies ratio were also found to be inversely correlated with life satisfaction (r[31] = -0.359, df = 29, p = 0.024). Interestingly, no significant correlations were found between state anxiety as assessed by the BAI and any of the summary variables (total verbal errors: r[29] = -0.231, df = 27, p = 0.114, total procrastinating verbalizations: r[29] = 0.176, df = 27, p = 0.181, total verbal dysfluencies: r[29] = 0.024, df = 27, p = 0.450).

3.2 Speech errors and physiological response to the TSST

Across all participants, both cortisol and IL-6 changed from baseline to after the start of the TSST (main effect of time for cortisol: F[1.777,49.745] = 9.976, $p < 0.001$; main effect of time for IL-6: F[1.602,44.865] = 39.934, $p < 0.001$) (Figures 4 and 5). Change in circulating cortisol concentrations in response to the TSST (i.e. from baseline to 30 mins after the start of the stressor) were found not be associated with any of the summary speech variables (total verbal errors: r [29] = -0.036, df = 27, $p = 0.426$, total procrastinating verbalizations: r[29] = 0.139, df = 27, $p = 0.236$, total verbal dysfluencies: r[29] = 0.097, df = 27, $p = 0.309$). Analyses of IL-6 response to the stressor (baseline to 90 mins after the start of the stressor) yielded similar results (total verbal errors: r[29] = -0.192, df = 27, $p = 0.159$, total procrastinating verbalizations: r[29] = -0.094, df = 27, $p = 0.314$, total verbal dysfluencies: r[29] = -0.166, df = 27, $p = 0.194$).

3.3 Speech errors and subjective distress response to the TSST

Change in TSST-induced subjective distress (determined by POMS total score) was found to be positively correlated with total number of verbal errors (r[31] = 0.319, df = 29, $p = 0.04$) (Figure 6). The verbal errors ratio was also found to be positively correlated with subjective distress response (r[31] = 0.316, df = 29, $p = 0.042$). To examine this relationship further, groups were formed using a median split of the total verbal errors variable. Demographic information for the groups formed is reported in Table 3. A very weak trend for main effect of group was observed on subjective distress throughout the stress test (F(1,52.32) = 2.169, $p = 0.152$).

4 Discussion

The current study examined the relationship between physiological and subjective responses to an acute laboratory psychosocial stressor challenge and speech performance behaviors during the challenge. Associations between intrapersonal factors and speech performance behaviors were also assessed. Findings reported here provide insight in to what factors may contribute to acute psychosocial stress responses. Total verbal errors and total verbal dysfluencies during the stress task were found to be related to depression features, while no significant relationship was found between anxiety features and any of the summary speech error variables. Upon examining the relationship of speech errors to physiological response to the stressor, it was discovered that cortisol and IL-6 reactivity were not correlated with any of the speech errors summary variables. Analysis of the relationship between speech errors and subjective distress as a result of the stressor revealed that total verbal errors were correlated with change in subjective distress as a result of the stressor.

In contrast to a previous study that found depression features were not associated with physiological response to psychosocial stress (Harkness et al., 2011), depression features were found to be associated with increased number of verbal errors and verbal dysfluencies made during the TSST. Speech errors therefore might represent a more sensitive measure of the stress response that fluctuates more in accordance with depressive affect. Similar to previous studies that reported anxiety is not related to physiological response to psychosocial stress (Takahashi et al., 2005, Elzinga et al., 2010), anxiety was not found to be associated with the number of speech errors during the stressor. However, a previous study has also reported that those with diagnosed anxiety disorders make more speech errors than controls during a public speaking task (Hofmann et al., 1997). Our result, as well as that for depression feature, should be interpreted with caution as participants had to have no current or past history of a psychiatric condition, and thus, the ranges for both depression and anxiety feature could be limited, leading to false results. As many studies have found that depression or anxiety with early life adversity alters cortisol reactivity (Harkness et al., 2011, Elzinga et al., 2010), future research could examine how these factors affect speech errors during psychosocial stress.

Few studies have examined associations between overt behaviors and endocrine response to psychosocial stress. One previous study found that gaze behavior during stress challenge in healthy children was inversely correlated with cortisol reactivity, while vocal quality and discomfort were not (Hessl et al., 2006). To our knowledge, no previous study has examined the association between speech form and physiological response to psychosocial stress. The results of the current study were that speech errors were not associated with cortisol and IL-6 response to psychosocial stress. Our results suggest that speech errors may fluxuate independently of physiological response to psychosocial stress. However, it is also possible that other biomarkers of the physiological response to stress may associate better with speech errors. For example, changes of circulating plasma adrenocorticotropin hormone concentrations as a result of TSST challenge may provide a more sensitive index of HPA axis reactivity that cortisol. Likewise, other biomarkers of the inflammatory response may be more reactive to TSST challenge, including nuclear factor-kappa B pathway activation in peripheral blood mononuclear cells (Wolf et al., 2009). Also, we did not assess the relationship between speech errors and autonomic function. Future studies would do well to correlate heart rate responses to the TSST and speech errors, or heart rate variability and speech errors. Heart rate variability would provide insight in to parasympathetic versus sympathetic nervous system activation during the TSST. It is possible that parasympathetic withdrawal as a result of TSST challenge may be especially likely to associate with speech errors.

Previous studies examining speech errors during psychosocial stress challenge have examined changes in subjective anxiety as well as a result of the stressor. One previous study found that participants who experienced more subjective anxiety during a public speaking task also paused more and for longer durations as well as exhibited more filled pauses than controls during their speeches (Hofmann et al., 1997). As well, participants who experienced more subjective anxiety as a result of a public speaking task were also found to make speeches rated as less fluent (Baggett et al., 1996). Our results as well suggest an association between affective state and speech errors. Though further studies are required to more fully characterize the relationship between speech errors and physiological response to the stress, the results of the current study suggest that subjective distress may be more closely tied to speech form errors as this property of speech is probably more reliant on cognitive processes than physiological ones.

In rating participants' speech, this study only looked at the various components of speech form. However, additional characteristics of speech, such as speech content and speech quality, could also be employed to better analyze speech output. There exists an extensive literature on how various parameters of speech change as a result of stress. Speech parameters such as volume and pitch have been found to change as a result of stress (reviewed in Steeneken & Hansen, 1999). These measures of speech quality might be more closely associated with endocrine and inflammatory response to stress than other measures of speech output because such speech parameters could be more controlled by physiological processes than cognitive ones (e.g. sympathetic nervous system activity). In future studies, examining speech content and speech quality in addition to speech form could allow for a more complete analysis of changes in speech due to psychosocial stress.

The current study has a number of limitations worth noting. First, our sample consisted only of participants who had no past or current psychiatric condition. Thus, stress responsivity, as measured by all procedures in the current study, was probably confined to a limited range. Future studies including participants variedly affected by depression or anxiety are required to better characterize the relationship between speech errors and other measures of stress response. Second, the sample size for the current study was small. A larger sample size would have allowed for upper and lower quartiles to be formed to better analyze the relationship between speech errors and physiological and subjective distress responses to psychosocial stress. Additionally, the present study only examined the effects of a psychosocial stressor on speech. We would be as well interested in the effects of other types of stress (i.e. multi-tasking) on speech output.

In conclusion, the findings of this study suggest that speech performance during an acute psychosocial stressor may be related to subjective distress experienced during the same event, as well as to depression features and feelings of life satisfaction.

Table 1 Measures of quality of speech form to be used

	Pause Variables
Pause	Number of silent periods either within or between sentences. To be counted, pauses had to last more than 2 sec.
Pause length	Length of time spent in pause. Only pauses of 2 sec or more were timed.
	Verbal Errors
Correction	Number of corrections or a word or phrase. This category includes any verbalization that changes any previous verbalization to a different form.
Distortion	Number of mispronunciations, "tongue slips," neologisms, and spoonerisms (e.g. "scutterbotch" vs. "butterscotch")
Fragment	Number of incomplete words or sentences where the meaning intended is not conveyed.
Repetition	Preservation of parts of words, words, or phrases. (e.g. stuttering)
	Procrastinating Verbalizations
Nonverbal intrusion	Number of nonverbal utterances that interrupt speech. Laughing, coughing, heavy breathing, mumbles, "voice catches," lip smacks, sniffs, and sighs are included in this category.
Procrastination	Number of verbalizations that serve to delay speech. (e.g. "ah," "and," "hmm," "like," "uh," "um," "well," "you know")
Qualifying phrase	Number of phrases that serve to comment on the speaker's own speaking ability. (e.g. "I'm not a good speaker")

Note. Adapted from "Enduring Without Avoiding: Pauses and Verbal Dysfluencies in Public Speaking Fear," by M. R. Lewin, D. W. McNeil, and J. M. Lipson, 1996, *Journal of Psychopathology and Behavioral Assessment*, *18*, p. 392. Copyright 1996 by the Plenum Publishing Corporation.

Table 2 Descriptive information for the sample

	Demographic information
N	31
Age (S.D.)	30.65 (8.093)
Gender	12 Male (38.7%)
Race	24 White / Caucasian, 3 African-American, 3 Asian, 1 American Indian or Alaskan / African-American
Ethnicity	3 White / Caucasian, Hispanic
	Self-report questionnaires
BDI	2.84 (3.769)
BAI	3.38 (4.362)
PSS	18.43 (7.596)
SWLS	26.29 (5.028)
	Speech errors
Pause	1.94 (2.308)
Pause Length	6.063 (7.918)
Correction	7.45 (4.767)
Distortion	5.23 (3.509)
Fragment	0.39 (0.715)
Repetition	7.16 (5.757)
Nonverbal intrusion	9.13 (5.731)
Procrastinations	44.42 (18.873)
Qualifying phrase	0.05 (0.250)
Word count	681 (103.085)
	Summary speech variables
Total verbal errors	20.23 (12.777)
Total procrastinating verbalizations	53.61 (20.747)
Total verbal dysfluencies	73.84 (26.074)
Total verbal errors / Word count	0.030 (0.018)
Total procrastinating verbalizations / Word count	0.081 (0.036)
Total verbal dysfluencies / Word count	0.111 (0.043)

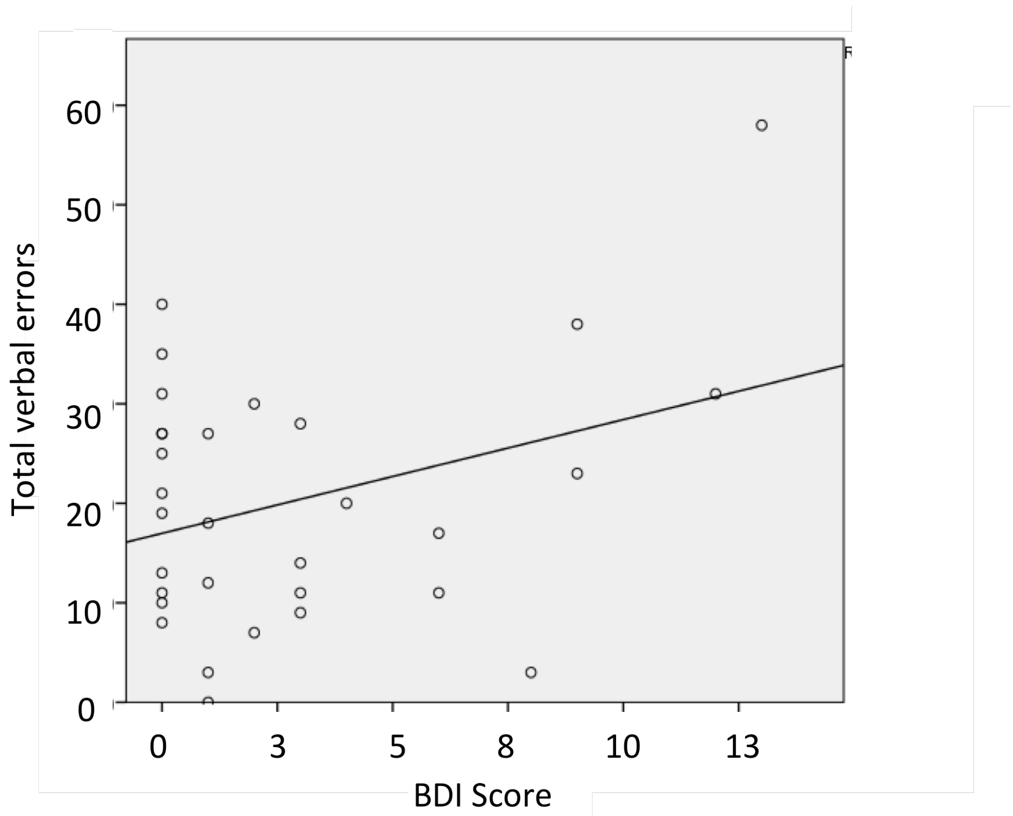

Figure 1 Total verbal errors are positively associated with depression features

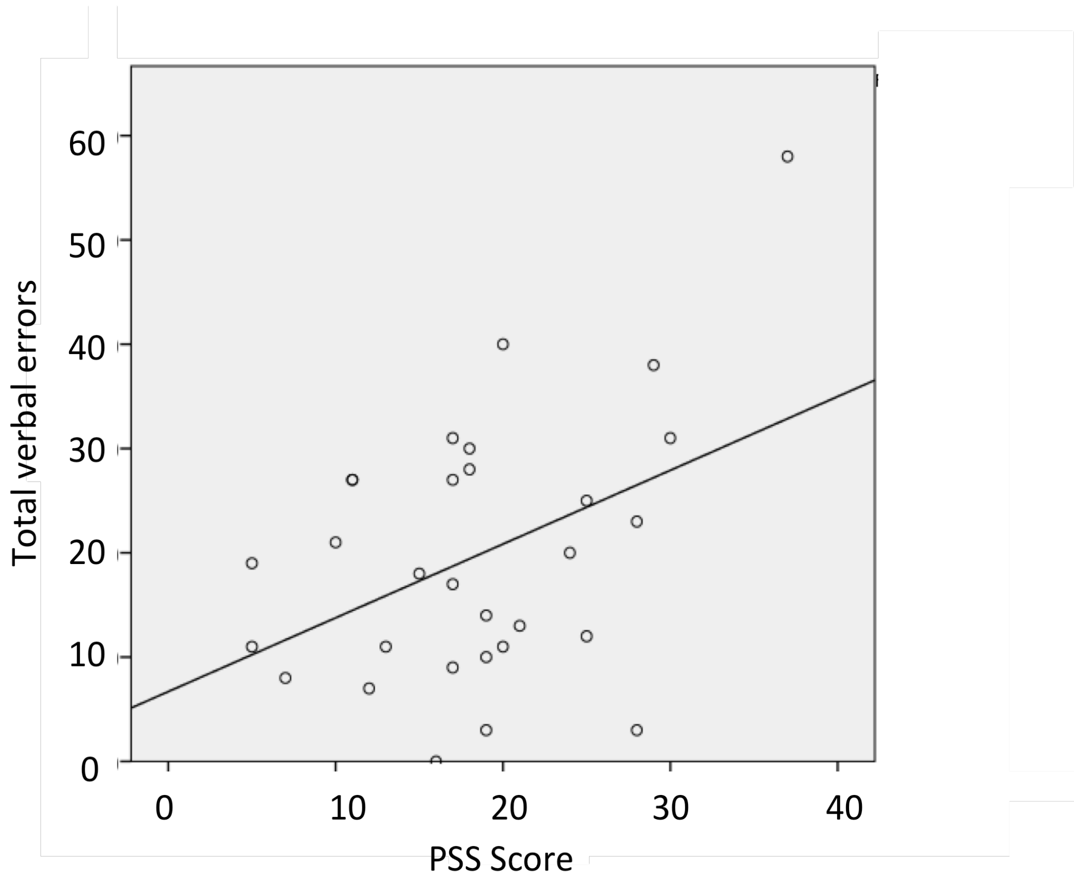

Figure 2 Total verbal errors are positively related to perceived life stress

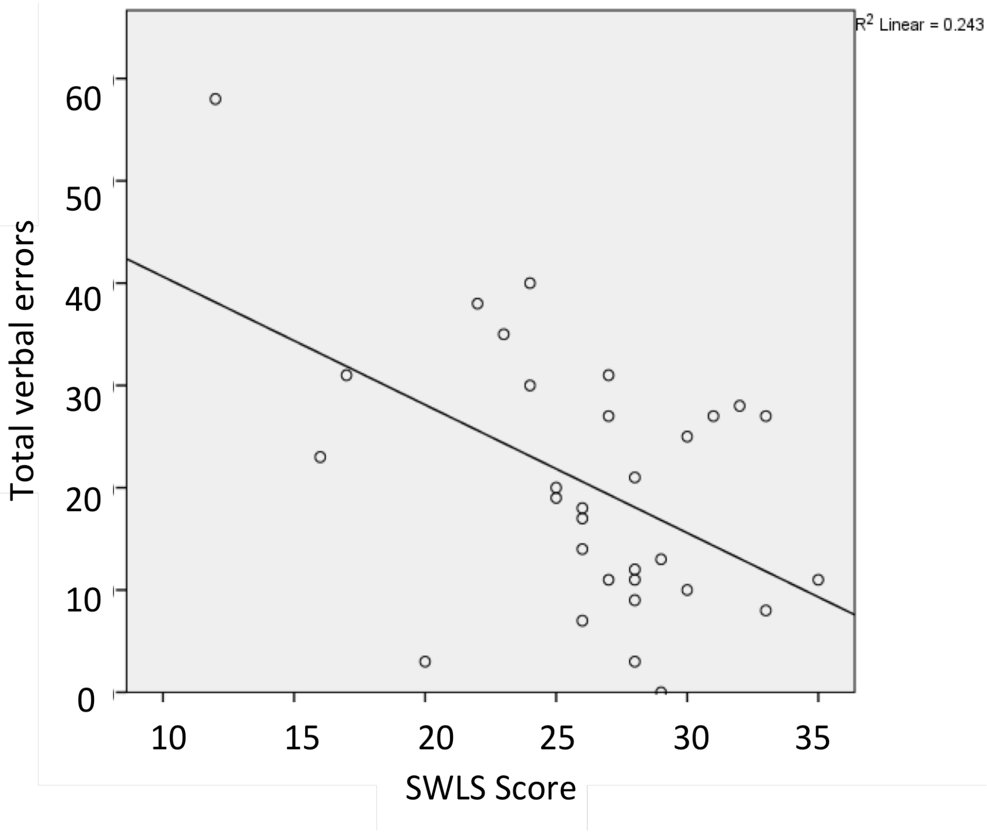

Figure 3 Total verbal errors are negatively correlated with life satisfaction

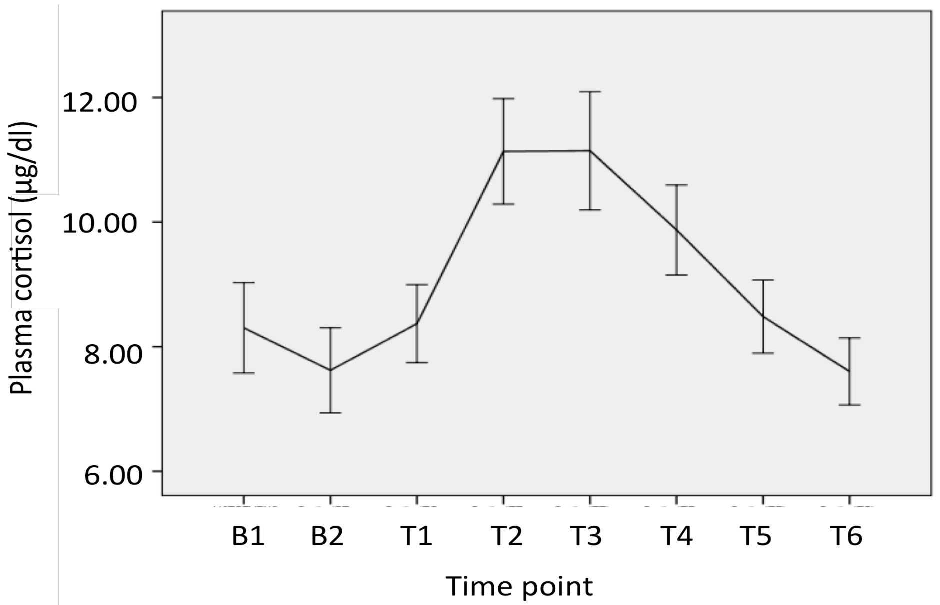

Figure 4 Circulating cortisol concentrations throughout the TSST procedure (mean ± S.E. of mean)

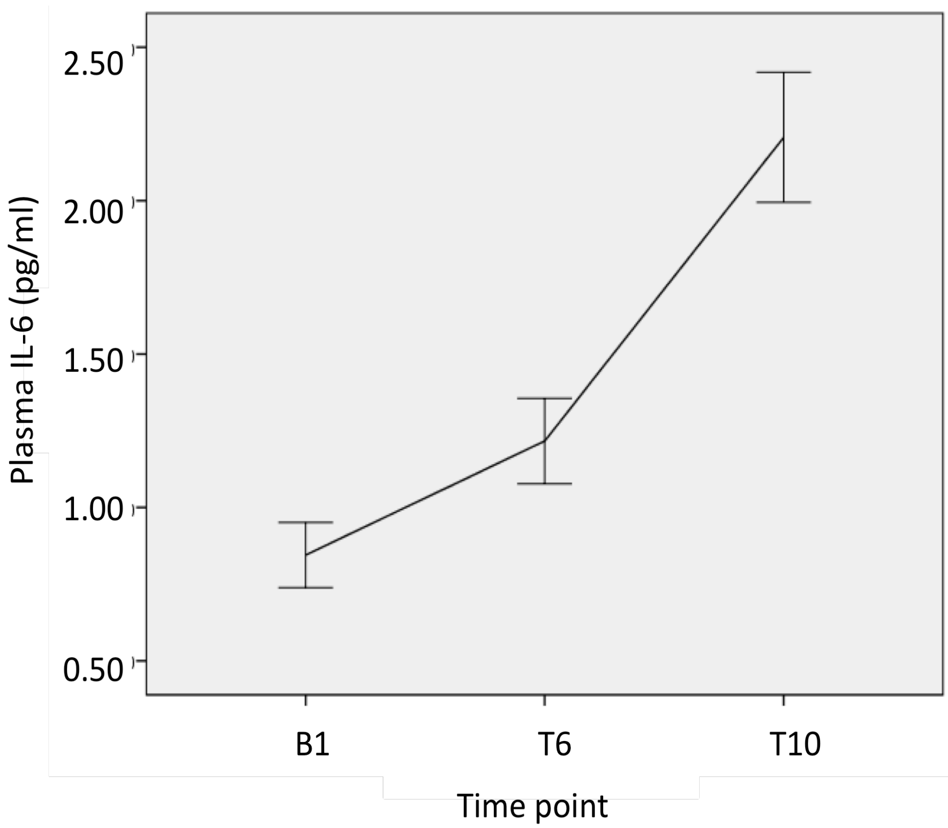

Figure 5 Circulating IL-6 concentrations throughout the TSST procedure (mean ± S.E. of mean)

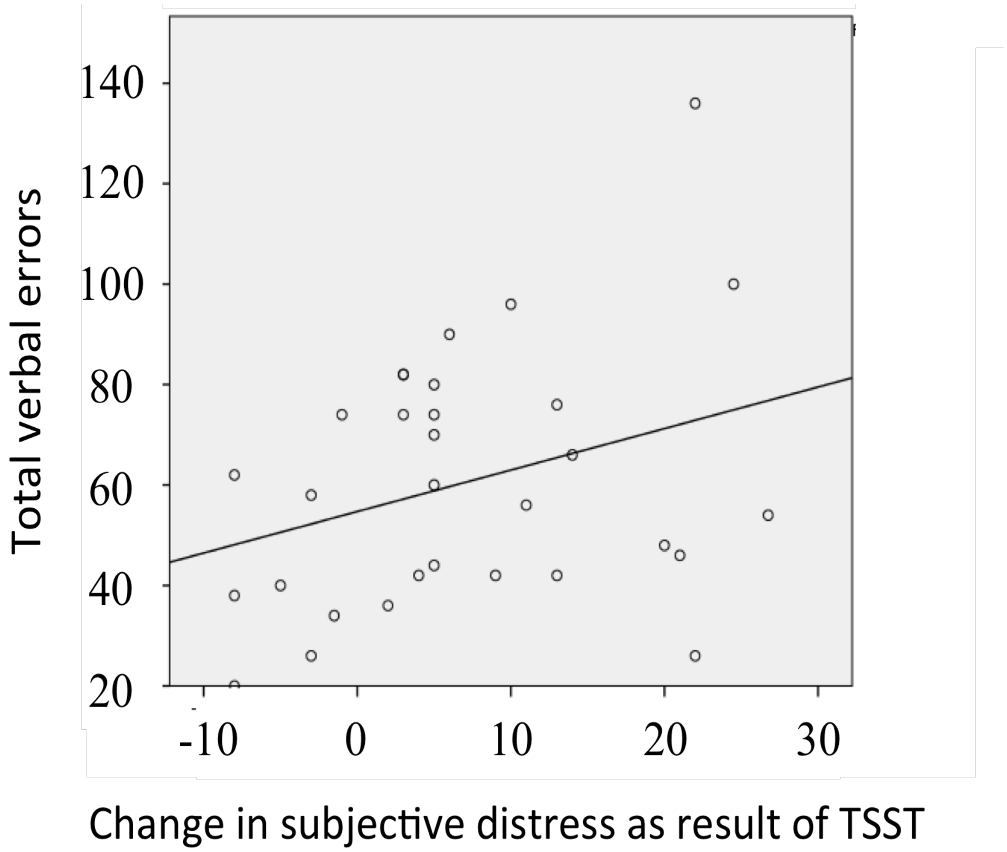

Figure 6 Total verbal errors is positively correlated with stress-induced subjective distress

Table 3 Demographic information for groups formed by median split of the total verbal errors variable

	Total Verbal Errors	
	Fewer Errors	More Errors
N	15	16
Age	27.87 (2.669)	33.25 (10.459)
Gender	2 Male (13.3%)	10 Male (62.5%)
Race	13 White / Caucasian, 1 African-American, 1 American-Indian or Alaskan / African-American	11 White / Caucasian, 2 African-American, 3 Asian
Ethnicity	1 White / Caucasian, Hispanic	2 White / Caucasian, Hispanic

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