

10-5-2017

An experimental study of exposure-based and emotion-focused interventions: Strange bedfellows, or a match made in heaven?

Nikita Yeryomenko
University of Windsor

Follow this and additional works at: <https://scholar.uwindsor.ca/etd>

Recommended Citation

Yeryomenko, Nikita, "An experimental study of exposure-based and emotion-focused interventions: Strange bedfellows, or a match made in heaven?" (2017). *Electronic Theses and Dissertations*. 7308.
<https://scholar.uwindsor.ca/etd/7308>

This online database contains the full-text of PhD dissertations and Masters' theses of University of Windsor students from 1954 forward. These documents are made available for personal study and research purposes only, in accordance with the Canadian Copyright Act and the Creative Commons license—CC BY-NC-ND (Attribution, Non-Commercial, No Derivative Works). Under this license, works must always be attributed to the copyright holder (original author), cannot be used for any commercial purposes, and may not be altered. Any other use would require the permission of the copyright holder. Students may inquire about withdrawing their dissertation and/or thesis from this database. For additional inquiries, please contact the repository administrator via email (scholarship@uwindsor.ca) or by telephone at 519-253-3000ext. 3208.

An experimental study of exposure-based and emotion-focused interventions:

Strange bedfellows, or a match made in heaven?

By

Nikita Yeryomenko

A Dissertation
Submitted to the Faculty of Graduate Studies
through the Department of Psychology
in Partial Fulfillment of the Requirements for
the Degree of Doctor of Philosophy
at the University of Windsor

Windsor, Ontario, Canada

2017

© 2017 Nikita Yeryomenko

An experimental study of exposure-based and emotion-focused interventions: Strange bedfellows, or a match made in heaven?

by

Nikita Yeryomenko

APPROVED BY:

M. Lumley, External Examiner
Wayne State University

K. Milne
Department of Kinesiology

A. Scoboria
Department of Psychology

U. Kramer
Department of Psychology

A. Pascual-Leone, Advisor
Department of Psychology

June 15, 2017

DECLARATION OF ORIGINALITY

I hereby certify that I am the sole author of this thesis and that no part of this thesis has been published or submitted for publication.

I certify that, to the best of my knowledge, my thesis does not infringe upon anyone's copyright nor violate any proprietary rights and that any ideas, techniques, quotations, or any other material from the work of other people included in my thesis, published or otherwise, are fully acknowledged in accordance with the standard referencing practices. Furthermore, to the extent that I have included copyrighted material that surpasses the bounds of fair dealing within the meaning of the Canada Copyright Act, I certify that I have obtained a written permission from the copyright owner(s) to include such material(s) in my thesis and have included copies of such copyright clearances to my appendix.

I declare that this is a true copy of my thesis, including any final revisions, as approved by my thesis committee and the Graduate Studies office, and that this thesis has not been submitted for a higher degree to any other University or Institution.

ABSTRACT

Exposure therapy is considered to be the treatment of choice for anxiety disorders, but mechanisms of change underlying its effectiveness are currently being contested. Emotional processing theory postulates that habituation of fear is the primary predictor of change during exposure, while inhibitory learning theory challenges that and postulates that variability in the intensity of fear is the predictor of change. Moreover, recent evidence points towards verbalization of emotion as a predictor of change during exposure as well. The present study investigated these predictors using a sample of 41 college students in an analog experimental design in the context of an exposure task. The study found mixed support that verbalization of emotion plays a role in improving the behavioral outcomes after an exposure task. Habituation in fear, controlled for variability in the intensity of fear, predicted worse speech performance from before to after the experimental task, ($\beta = -.63, p = .001$), while variability in the intensity of fear, controlled for habituation, improved speech performance ($\beta = .54, p = .008$). At the same time, habituation in shame, when controlled for variability in the intensity of shame, predicted an improvement in state self-esteem from before to after the experimental task ($\beta = .31, p = .009$), while variability in the intensity of shame, when controlled for habituation in shame, predicted state self-esteem deterioration ($\beta = -.35, p = .038$). Physiological indices indicated that lower heart rate variability during the experimental task predicted worsening in self-esteem from before to after the task ($\beta = -.18, p = .034$), while habituation in fear ($r(39) = .40, p = .014$) and shame ($r(39) = .56, p < .001$) was positively correlated with parasympathetic nervous system activity when controlled for variability in these emotional states.

TABLE OF CONTENTS

DECLARATION OF ORIGINALITY	iii
ABSTRACT.....	iv
LIST OF TABLES	x
LIST OF FIGURES	xi
LIST OF APPENDICES.....	xii
CHAPTER 1. INTRODUCTION	1
<i>Exposure treatments</i>	1
<i>Emotional processing theory</i>	2
<i>Challenges to emotional processing theory</i>	3
<i>Inhibitory learning theory</i>	3
<i>Underlying neural mechanisms</i>	6
<i>The role of verbal processes in emotion processing</i>	11
<i>Core emotional processes in Social Anxiety Disorder</i>	13
<i>Summary of key issues and presenting research questions</i>	15
<i>The current study</i>	17
<i>Hypotheses</i>	18
CHAPTER 2. METHODS	19
<i>Sample</i>	19
<i>Procedure</i>	20

<i>The speech tasks</i>	23
<i>Ethical considerations</i>	24
<i>Measures</i>	24
<i>Intervention outcome measures</i>	24
<i>Social Interaction Anxiety Scale-6 (SIAS-6) and Social Phobia Scale-6 (SPS-6)</i>	25
<i>Brief Fear of Negative Evaluation Scale-Straightforward items (BFNE-S)</i>	25
<i>Brief Personal Report of Confidence as a Speaker (BPRCS)</i>	26
<i>State Self-Esteem Scale (SSES)</i>	26
<i>Social Performance Rating Scale, Modified (SPRS-M)</i>	26
<i>Process measures</i>	27
<i>Subjective Units of Distress Scale (SUDS)</i>	27
<i>State Shame and Guilt Scale (SSGS)</i>	28
<i>Physiological measures</i>	29
<i>Analysis</i>	30
<i>Assumptions testing</i>	32
 CHAPTER 3. RESULTS	32
<i>Inter-rater reliability for speech performance (SPRS-M)</i>	32
<i>Manipulation checks</i>	33
<i>Assumptions</i>	33
<i>Main analyses</i>	37
<i>Testing hypothesis 1: Do verbal processes affect outcomes?</i>	38
<i>Experimental group predicts somewhat better speech performance (SPRS-M)</i>	38
<i>Experimental group does not predict state self-esteem (SSES)</i>	39
<i>Hypothesis 1 results summary</i>	40
<i>Testing hypothesis 2: Do emotional processes predict outcomes?</i>	40
<i>Strategy for analysis of emotional processes</i>	40

<i>Ratings of distress (SUDS) predict speech performance (SPRS-M)</i>	41
<i>Ratings of distress (SUDS) do not predict state self-esteem (SSES)</i>	43
<i>Ratings of shame (SSGS) do not predict speech performance (SPRS-M)</i>	43
<i>Ratings of shame (SSGS) predict state self-esteem (SSES)</i>	43
<i>Hypothesis 2 results summary</i>	45
<i>Testing hypothesis 3: Do physiological indices predict outcomes?</i>	46
<i>Experimental group did not predict increase in LF/HF ratio</i>	46
<i>Lower LF/HF ratio predicts deterioration of state self-esteem (SSES)</i>	46
<i>HF component of HRV positively correlates with habituation in emotional states</i>	47
<i>Hypothesis 3 results summary</i>	48
CHAPTER 4. DISCUSSION	52
<i>Experiential group showed greater behavioral change as compared to Exposure group</i> ...	55
<i>Habituation and variability in emotional arousal predict change—but in opposite directions</i>	56
<i>Change in shame is an important predictor of emotional outcomes during exposure</i>	59
<i>General theoretical issues</i>	59
<i>Habituation and variability of emotion intensity have a complex relationship</i>	60
<i>Habituation and extinction are poorly differentiated</i>	61
<i>SUDS may measure multiple constructs</i>	63
<i>Emotional processes of shame, not just fear, can be important predictors of outcomes</i>	64
<i>Methodological implications</i>	65
<i>Clinical implications</i>	65
<i>Limitations and future directions</i>	66
<i>Conclusions</i>	70

REFERENCES	71
APPENDICES	83
<i>Appendix A:</i> Specific verbal instructions for the study.....	83
<i>Appendix B:</i> Correlation between process and outcome measures.....	86
<i>Appendix C:</i> Correlations between process measures	87
<i>Appendix D:</i> Correlations between outcome variables.....	88
<i>Appendix E:</i> Participant recruitment PRISMA flow chart.....	89
<i>Appendix F:</i> Basic demographic information for the sample	90
<i>Appendix G:</i> Means and standard deviations of outcome measures.....	91
<i>Appendix H:</i> Means and standard deviations of SUDS during the experimental task.....	92
<i>Appendix I:</i> Means and standard deviations of SSGS during the experimental task.....	93
<i>Appendix J:</i> Measures of average LF/HF ratio at different times during the study.....	94
<i>Appendix K:</i> Measures of average HF component at different times during the study	95
<i>Appendix L:</i> Study measures.....	96
<i>Appendix M:</i> Average and individual fear (SUDS) scores plotted over the course of the experimental tasks.....	103
<i>Appendix N:</i> Average and individual shame (SSGS) scores plotted over the course of the experimental tasks.....	104
<i>Appendix O:</i> Average and individual low frequency to high frequency (LF/HF) ratio scores plotted over the course of the experimental tasks.....	105

Appendix P: Average and individual high frequency (HF) component of recorded heart rate variability scores plotted over the course of the experimental tasks106

VITA AUCTORIS107

LIST OF TABLES

Table 1

*Multilevel variance inflation factor (MVIF) values of emotional process
predictors.....34*

Table 2

*Full and partial correlations between fear processes and speech
performance.....36*

Table 3

Full and partial correlations between shame processes and state self-esteem.....37

LIST OF FIGURES

Figure 1.

The experimental design graph.....22

Figure 2.

Effect of habituation of fear during combined experimental tasks on speech
performance.....49

Figure 3.

Effect of variability of fear during combined experimental tasks on speech
performance.....50

Figure 4.

Effect of habituation of shame during combined experimental tasks on state self-
esteem.....51

Figure 5.

Effect of variability in shame during combined experimental tasks on state self-
esteem.....52

LIST OF APPENDICES

APPENDIX A: Specific verbal instructions for the study	83
APPENDIX B: Correlation between process and outcome measures	86
APPENDIX C: Correlations between process measures	87
APPENDIX D: Correlations between outcome variables	88
APPENDIX E: Participant recruitment PRISMA flow chart	89
APPENDIX F: Basic demographic information for the sample	90
APPENDIX G: Means and standard deviations of outcome measures	91
APPENDIX H: Means and standard deviations of SUDS during the experimental task	92
APPENDIX I: Means and standard deviations of SSGS during the experimental task	93
APPENDIX J: Measures of average LF/HF ratio at different times during the study	94
APPENDIX K: Measures of average HF component at different times during the study	95
APPENDIX L: Study measures	96
APPENDIX M: Average and individual fear (SUDS) scores plotted over the course of the experimental tasks	103
APPENDIX N: Average and individual shame (SSGS) scores plotted over the course of the experimental tasks	104
APPENDIX O: Average and individual low frequency to high frequency (LF/HF) ratio scores plotted over the course of the experimental tasks	105

APPENDIX P: Average and individual high frequency (HF) component of recorded heart rate variability scores plotted over the course of the experimental tasks106

Introduction

Exposure treatments

Exposure treatments have been shown to be highly effective alone or in combination with cognitive restructuring for a range of anxiety disorders, and are considered to be the treatment of choice for some of them (Deacon & Abramowitz, 2004). Despite demonstrated and reliable success in treating anxiety, exposure treatments are highly underutilized in clinical practice. Research has shown that this underutilization is related to the perception by a large number of practicing clinicians that exposure treatments are harmful, intolerable, and unethical (Farrell, Deacon, Dixon, & Lickel, 2013). Contrary to common misconceptions, exposure therapy does not result in greater drop-out rates than other treatments and individuals who undergo exposure treatments usually find them tolerable (Wolitzky-Taylor, Viar-Paxton, & Olatunji, 2012). Likewise, there is no substantial evidence that exposure therapy is harmful when it is done appropriately. A study by Foa et al. (2002) found a temporary exacerbation of anxiety in some clients undergoing exposure therapy, but it was not predictive of treatment drop-out or treatment non-response. A more recent study of prolonged exposure therapy for chronic post-traumatic stress disorder in female victims of sexual and nonsexual assault has shown no evidence of any reliable exacerbation of trauma or depression symptoms among women in exposure treatments (Jayawickreme et al., 2014). In contrast, female survivors who were on the waitlist showed a reliable exacerbation of symptoms in the absence of treatment. Meta-analyses have shown that exposure strategies for social anxiety alone perform as well as cognitive restructuring, social skills training, or a combination of exposure with cognitive restructuring (Feske & Chambless, 1995; Deacon &

Abramowitz, 2004; Acarturk et al., 2009), and are sometimes superior (Gould, Buckminster, Pollack, Otto, & Yap, 1997). Thus, empirical evidence indicates that exposure treatments are both tolerable and effective.

Emotional processing theory

Arguably the most influential theory to date of how exposure therapy achieves its effect is the emotional processing theory (Foa & Kozak, 1986; Foa, Huppert, & Cahill, 2006). The theory postulates a model of learned fear, according to which the individual forms associations between a stimulus, a response, and their corresponding meaning representations (e.g., “this fear response helps deal with that dangerous stimulus”). The resulting adaptive *fear structures* accurately represent reality and enable the individuals to avoid danger. In some circumstances, however, maladaptive learning experiences, such as psychological trauma, can result in the formation of a pathological, or maladaptive, fear structure. In this structure the associations between stimuli, responses, and meaning representations inaccurately reflect the situation as dangerous when in reality it is not, leading to excessive or inappropriate responses intended to avert the danger that is not present.

According to emotion processing theory, a therapeutic change occurs through *emotional processing*, which involves two critical steps: (1) *initial fear activation*, and (2) *habituation of the fear structure* both within and between exposure sessions. Habituation, indexed by a reduction in fear over time, is believed to occur through the incorporation of new information about stimuli, responses, and meaning that is incompatible with it (Foa & Kozak, 1986). Initial fear activation occurs when an individual is exposed to cues that match closely to the fear structure, evidenced by physiological arousal and self-reported

anxiety that exceeds the pre-exposure levels. Furthermore the theory states that a decrease in fear within exposure sessions, and a decrease in peak fear levels between exposure sessions, indicates the habituation of fear, which serves as an index of successful emotional processing (Foa, Huppert, & Cahill, 2006).

Challenges to emotional processing theory

Recent years have seen the emergence of empirical evidence that does not support emotional processing theory's view on what the key therapeutic processes are. Initial fear activation, within-session and between-session habituation of fear were found to be inconsistently associated with symptom change in exposure treatments for anxiety disorders (Craske et al., 2008; Baker et al., 2010; Kircanski et al., 2012; Meuret, Seidel, Rosenfield, Hofmann, & Rosenfield, 2012; Wendt, Schmidt, Lotze, & Hamm, 2012). Furthermore, even when exposure treatment is initially successful, the previously habituated, or "unlearned", fear structure can be reactivated with new strength, especially in a context that is different from the treatment context, resulting in a *return of fear* (Tsao & Craske, 2000; Schiller & Phelps, 2011). Thus, while the success of exposure treatments is well-documented (as discussed above), the exact mechanisms of change remain uncertain.

Inhibitory learning theory

One of the newer explanations of the mechanisms behind exposure treatments is the idea of *inhibitory learning*. According to this approach, formerly "habituated" or "extinguished" fears are not actually unlearned, but rather are inhibited by new, competing learning (Craske, Treanor, Conway, Zbozinek, & Vervliet, 2014). This inhibitory process is thought to be central to extinction of learned fears (Craske et al.,

2008; Craske et al., 2014), and the success of exposure treatments as a consequence.

Craske and colleagues have proposed a number of changes based on this consideration to optimize exposure treatments. For example, in contrast to emotional processing theory's habituation approach, the inhibitory learning approach is not concerned with the reduction of fear during the exposure session (Craske et al., 2014).

Culver, Stoyanova, and Craske (2012) found that during exposure procedure for the severe fear of public speaking, two predictors of change at 2-week follow-up emerged: (a) greater variability in the levels of self-reported fear throughout the exposure predicted lower self-reported fear during the follow-up; (b) greater degree of within session habituation predicted lower speech duration, indicating worse performance, during the behavioral assessment task. The study additionally found an inconsistent association between physiological arousal (indexed by heart rate) and the outcomes in that on one hand, higher heart rate at the end of exposure predicted lower confidence in one's speaking ability during the one-week follow-up, while on the other hand predicting lesser amount of fear during that time as well.

Kircanski et al. (2012) found that after a series of exposure trials for contamination fears, the following predictors emerged at a 2-week follow-up: (a) greater within-session habituation of fear and greater between session habituation of arousal (indexed by heart rate) predicted less self-reported obsessions; (b) greater variance in self-reported fear during exposure predicted lower self-reported fear during the follow-up; (c) greater initial fear activation during exposure predicted higher overall levels of fear during the follow-up.

While the studies mentioned above did not find a reliable association between physiological arousal and changes in the participants during exposure tasks there may be a theoretical reason for its importance as a predictor. There is now converging evidence across multiple treatment approaches that moderate levels of arousal must be present in-treatment for lasting changes in emotional experience in psychotherapy, with too little or too much arousal disrupting the consolidation of new meaning, and the elaboration of new emotional meaning to compete with the old meaning (Hayes, Beck, & Yasinski, 2012), supporting the idea that sustained levels of physiological arousal may actually be therapeutic.

Newer evidence strongly suggests that variability in subjective fear plays a very important role in the exposure process. Variability in levels of fear may help enhance exposure by serving as a variety of internal contexts which are associated with extinction learning, and thus new inhibitory learning is more likely to be retrieved as variable levels of fear will be elicited in situations outside of the therapeutic exposure context, thereby generalizing the new inhibitory learning to contexts other than the exposure sessions and protecting against the “return of fear” (Craske et al., 2014). Another explanation for why variability in the intensity of fear is therapeutic is based on Rescorla-Wagner theory which states that the bigger the discrepancy between the expectation and reality, the stronger is the new learning, and thus variability in the intensity of fear may reflect changes in the expectancy of negative effect or harm (Kircanski et al., 2012). Thus, variability in the intensity of experienced fear is an *index* of inhibitory learning taking place with a variety of potential explanations of how it exerts its effects.

An alternative (and perhaps coexisting) possibility is that variability in fear intensity may represent increased access to more advanced emotional states. The theory behind emotion focused therapy stresses that when a client declares an emotional experience, or the researchers focus on it, there is actually a range of emotional experiences happening at the same time that are part of that person's immediate experience. The reason for that is that, according to this theory, any emotional experience is a dynamic system with a natural ebb and flow based on dominant and sub-dominant experiences. Where one emotion becomes less salient, the other, qualitatively different emotion, may move to the foreground of emotional experience (Pascual-Leone, Paivio, & Harrington, 2016). An example of that would be a situation in which an individual who is experiencing grief shifts from an experience of anger regarding the loss towards sadness, even as both emotions are co-existing aspects of that person's immediate experience. Thus, a conceptual leap from variability in one emotion correlating with an increased access to other, more advanced emotions, may be a distinct possibility.

Previous research found that an increased variance in qualitatively different emotional states and increased access to adaptive emotions over time was found to distinguish cases with good outcomes from cases with bad outcomes in emotion focused therapy (Pascual-Leone, 2009). Thus, a successful emotional change would not necessarily mean the absence of maladaptive emotional states, but rather an increased access to a more flexible repertoire of emotion. This flexibility would also imply a more nuanced appraisal of feared stimuli which may compete and inhibit the old maladaptive appraisals, as the underlying neural mechanisms may suggest.

Underlying neural mechanisms

Emotional experience in general appears to involve two distinct neural systems. The first system is responsible for the implicit, nonconscious, visceral, pre-verbal, or *bottom-up*, aspects of emotional experience and involves activation of the bilateral amygdala (Phelps, 2005; Ochsner et al., 2009), which is also responsible for the acquisition, storage, and expression of learned fear. The second system is involved in the explicit, conscious, conceptual, verbal, or *top-down*, aspects of emotional experience. This system involves activity of the left amygdala, as well as a conscious cognitive appraisal of the emotional significance of external or internal stimuli that engages the dorsomedial prefrontal cortex (dmPFC), activation of which is associated with a strong subjective experience of emotion (McRae et al., 2012). These neural systems are distinct, even though, in practice, they often interact: bilateral damage to the amygdala can prevent fear conditioning in humans as evidenced by lack of physiological arousal in the presence of a learned fear stimulus despite a conscious, verbal awareness, and understanding of the emotional significance of the fear stimulus, which is dependent on the intact hippocampal circuits (Phelps, 2005). LeDoux (2013) reported that a conscious feeling of fear originates in much the same way as other emotions, such as pride or shame. These feelings (i.e., fear, pride, shame) are generated via the cognitive processing of “raw neural materials” - in other words, through the interaction of the two neural systems that participate in the generation of the implicit-preverbal and explicit-verbal aspects of emotional experience.

These dual systems participate in both *appraisal*, an evaluation of emotional significance of an external or an internal stimulus (Etkin, Egner, & Kalisch, 2011), as

well as *emotion regulation*¹, an arbitration between the conflicting appraisals or reappraisals of stimuli, and both automated preconscious as well as controlled conscious response through the inhibition of competing appraisals and action tendencies (Etkin et al., 2011). When presented with an emotional stimulus, a neural circuit consisting of the amygdala, dorsal Anterior Cingulate Cortex (dACC), and dorsomedial Prefrontal Cortex (dmPFC) is engaged in the detection, and conscious appraisal of emotional significance of that stimulus. Moreover, this neural circuit is recruited during the expression of physiological and action tendency fear response to that stimulus. Intentional reinterpreting, or reappraisal, of a stimuli's emotional meaning was found to activate both dACC and dmPFC (Kanske, Heissler, Schönfelder, Bongler, & Wessa, 2010). When there are competing appraisals of a stimulus, the amygdala, ventral Anterior Cingulate Cortex (vACC), and ventromedial Prefrontal Cortex (vmPFC) circuit is activated. This circuit inhibits competing appraisals and action tendencies, allowing for a course of action to take place.

There is a remarkable overlap between the neural circuits described above and those that are involved in the extinction of learned fear, which has led Quirk and Beer (2006) to suggest that extinction of learned fear is a special case of emotion regulation. In addition to its role in the expression of physiological and action tendency, the amygdala-dACC-dmPFC circuit is also involved in the conscious appraisal and reappraisal of a CS, while the amygdala-vACC-vmPFC circuit is involved in the inhibition of learned fear response. In the course of extinction learning, the vmPFC potentiates its connections to

¹ It is important to note that the meanings of these terms appear to be unique to the areas of neuroscience, and are somewhat distinct from the clinical field.

hippocampal regions, making extinction a very context-dependent process, accounting for the *return of fear* phenomenon (Quirk & Beer, 2006). Additionally, repeated exposure to feared stimuli was found to habituate the response of the amygdala with no corresponding change to dmPFC activity (Wendt et al., 2012), possibly elucidating why within-session habituation has not been a consistent predictor of long-term fear or symptom change, and further solidifying the idea that changes over the course of exposure can occur on one level but not on another. In other words, exposure to a feared situation may reduce the nonconscious fear reaction, but change little in the conscious appraisal of fear stimulus as threatening, thereby likely reducing overall therapeutic effectiveness.

Consistent with previous findings, a model of neurovisceral integration (Thayer & Lane, 2000) postulates that what is underlying anxiety disorders is a deficit in the inhibitory activity of parasympathetic nervous system. Central autonomic network (Bennarroch, 1993) directly influences heart rate variability by sending output to the sinoatrial node via stellate ganglia and the vagus nerve (Thayer & Lane, 2009). Structurally, the central autonomic network involves a variety of brain regions, among them the central nucleus of the amygdala, as well as anterior cingulate and vmPFC (Appelhans & Luecken, 2006). Thayer and Lane (2000) propose that this central autonomic network shares so many commonalities with the neural systems implicated in affective processing, such as the anterior executive region (Devinsky et al., 1995), and others, that they should be considered the same functional neural network. Indeed, the structural overlap between the central autonomic network and the brain regions involved in emotion regulation and fear extinction, which were reviewed earlier, is striking.

According to neurovisceral integration theory, prefrontal cortex (PFC) exerts inhibitory control over the central nucleus of amygdala. When PFC is deactivated, the net result is the activation, or disinhibition, of the central nucleus of amygdala, which subsequently leads to disinhibition of medullary cardioacceleratory circuits, leading to increased heart rate and decreased heart rate variability (Thayer & Lane, 2009). Thayer & Lane (2009) suggest that this common inhibitory circuit can be indexed by vagally mediated heart rate variability (HRV), that is, the high frequency (HF) component of HRV. Thus, they state, vagally mediated HRV can serve as an index of inhibitory control. Other researchers have also examined HRV as an index of emotion regulation (e.g., Thayer & Brosschot, 2005; Appelhans & Luecken, 2006; Fujimura & Okanoya, 2012; Williams et al., 2015). Thus, in the search for an index of inhibitory learning, heart rate variability appears to be a more promising candidate than heart rate, in general.

The literature reviewed above suggests that processing of the emotional stimuli during fear exposure involves two levels: a conscious level of verbal appraisals and a nonconscious level of physiological and action tendency reactions. In line with this reasoning, Catherall (2003) and LeDoux (2013) suggested that nonconscious and conscious systems would be affected in different ways by different interventions. Catherall (2003), proposed that a certain degree of habituation to distressing stimuli may need to be present so that excessive levels of arousal do not interfere with the conscious processing of emotion. LeDoux (2013), in turn, proposed that implicit system may be more responsive to exposure interventions operating through the extinction process, while the explicit system may be more responsive to verbal interventions aimed at changing explicit meaning.

The role of verbal processes in emotion processing

Given the dual systems underlying different aspects of emotional experience and the fact that they interact, it is not very surprising to find that even when exposure therapy that does not include any explicit verbal-cognitive reappraisal strategies it often nonetheless involves spontaneous cognitive changes. For example, exposure therapy often results in individuals' reduction of the explicit expectation of harm or threat when in the presence of what is called a conditioned stimulus, a “threat cue” which signals or predicts incoming harm or threat (Hofmann, 2008). The inverse is also true, in that explicit “cognitive” processes influence implicit “emotional” ones. Kircanski, Lieberman, and Craske (2012) have found that spider-phobic individuals who were verbalizing the negative emotions they experienced while approaching a live spider showed reduced physiological arousal compared to when they used neutral words or when they were instructed not to verbalize at all. Additionally, individuals who used more anxiety or fear words in this task had greater physiological reduction of fear during the exposure to a live spider. A similar study by Lieberman et al. (2007) found that labeling one's affect during exposure to negative images downregulated amygdala activity, indicating reduced arousal. Curiously, individuals with alexithymia, a condition characterized by difficulties in finding words for and verbalizing emotional experiences (Bermond et al., 2007) are particularly prone to low distress tolerance (Rose & Segrist, 2012). Furthermore, these individuals have less ability to regulate emotion through a strategy of cognitive reappraisal, where one consciously discusses and reformulates one's understanding of fearful stimuli (Pollatos & Gramann, 2012). One of the possible mechanisms at play here is that if indeed being able to articulate emotional experience verbally can downregulate

arousal, then not being able to do so may contribute to lower ability to regulate emotion as seen in alexithymia.

Evidence points that verbalization of emotion is an important variable that influences the course of exposure. The specificity of verbalization appears to play a particularly important part. Philippot, Vrielynck and Muller (2010) asked participants to give a 3-minute videotaped speech on a difficult topic chosen by the experimenters. Prior to the speech, the participants were asked a series of questions about their anticipation of giving the speech. The questions were designed to foster either a generic or a specific mode of processing. Individuals in the generic condition were asked about thoughts, emotions, and sensations that they typically experience in similar situations, while those in the specific condition were asked to specify as precisely as possible what thoughts, emotions, and sensations they experienced in the moment. The results indicated that at the end of the speech the participants in the specific processing group experienced less speech anxiety compared to the individuals in the generic processing or the control groups when rated by the judges or by self-report.

A different study investigated the effect of specific experiential mode of processing vs. an abstract-analytic mode on the emotional valence of thoughts after an impromptu speech task (Nilsson, Lundh, & Viborg, 2012). This crossover design study found that individuals who initially adopted a specific mode of processing, compared to those who initially adopted an abstract-analytic mode of processing, showed fewer negatively emotionally valenced thoughts at the end of the first post-intervention assessment. The results indicated a successful change of appraisal of emotional stimuli for the experiential self-focus group, making the stimuli less negative. There were no differences between the

groups at the second post-intervention assessment, after the self-focus tasks were switched, suggesting that experiential self-focus was primarily responsible for the change.

These studies taken together suggest that a specific, experiential, "in-the-moment" processing of immediate experience can enhance interventions that work with emotion, such as exposure, especially when the emotional experience itself is being labelled, reappraised, and elaborated on.

Core emotional processes in Social Anxiety Disorder

Fear of negative evaluation is thought of as being the core pathogenic feature for social anxiety: individuals with social anxiety believe that others are likely to think of them critically, and they place special importance on being evaluated in a positive light (Rapee & Heimberg, 1997). The fear of negative evaluation is triggered by social-evaluative situations where having an audience is the primary source of threat. However, it is critical to notice that the audience does not have to intentionally observe the socially anxious individual. For these people, anxiety and fear can be generated simply from the possibility that one might be perceived by someone, such as a stranger passing by on the street or the idea that someone might see one's picture, videotaped performance, etc. (Rapee & Heimberg, 1997).

Emotional processing theory postulates that the "fear of embarrassment" fear structure is underlying social anxiety disorder. In this type of fear structure, the meaning of threat is associated with a multitude of social stimuli and contexts (such as public speeches, neutral facial expressions). Verbal, physiological and behavioral responses to such stimuli (such as sweating, blushing, or avoiding eye contact) are likewise associated

with the risk of embarrassment and, in turn, tend to be interpreted as drawing criticism and increasing the likelihood of being rejected by others, further increasing the individual's anxiety. Fear structure in social anxiety is furthermore associated with high estimates of cost and probability of harm in social situations, such as expecting others to always be rejecting, or interpreting somewhat negative social events in catastrophic terms (Foa, Huppert, & Cahill, 2006). Thus, fear of embarrassment is the treatment target according to emotional processing theory in this context.

There is another contender for the role of the core pathogenic feature of social anxiety. Maladaptive shame is a social emotion characterized by a sense of self as flawed. Thus, maladaptive shame involves a global negative self-evaluation of oneself as being unacceptable in some interpersonal context (whether real or imagined) and is associated with a tendency to withdraw or hide (Lewis, 1971). Being prone to shame and the subsequent dysfunctional methods of coping with shame have been established as having a relationship with severity of psychopathology across multiple studies, and have been proposed as important mechanisms of disorder in depression and eating disorders (Candea & Szentagotai, 2013). Hedman, Ström, Stünkel, and Mörtberg (2013) found that individuals with social anxiety disorder, when compared to a control group of healthy participants matched on age and gender, showed more pronounced experiences of shame.

It is important to note that while maladaptive fear and shame are subjectively experienced in different ways, they are related. Both emotions are associated with a negative core sense of self and tendencies to withdraw or collapse rather than engage in any adaptive action tendency (Pascual-Leone & Greenberg, 2007). Thus, it is not surprising they both are prominently featured in social anxiety disorder.

In contrast to emotional processing theory, the theory based on emotion focused therapy (Shahar, 2013) states that the primary pathogenic feature in social anxiety disorder is *shame-anxiety*, a complex emotion which consists of maladaptive shame related to the negative core sense of self, with anxiety playing only a secondary role. This theory argues that primary emotions are fundamental reactions to an immediate situation or cue, while secondary emotions are reactions to primary emotions or cognitions, and therefore less central as a treatment target. According to this model of social anxiety, the secondary fear is about the risk of primary shame about being fundamentally flawed and unacceptable. Therefore, from the emotion-focused therapy treatment perspective, a successful treatment of social anxiety is predicated on activating and transforming the shame-based structure first and foremost, rather than changing anxiety and the associated fear structure (Shahar, 2013). Coming from a different treatment perspective, Fergus et al. (2010) found that during the course of exposure treatment a reduction in shame-proneness was related to changes in social anxiety disorder symptoms, further supporting the role of maladaptive shame as a core psychopathologic process. This critical role of shame supports the idea that that a successful treatment of social anxiety through exposure may not only require a change in the “fear structure,” but more importantly a change in the “shame structure.”

Summary of Key Issues and Presenting Research questions

The literature reviewed above indicates the central role of emotions during exposure to feared situations. The first line of evidence points out that verbal processing of emotions appears to influence emotion regulation efforts in some way. Explicit verbal labelling of emotions, in particular, has been found to down-regulate the implicit

physiological and behavioral reactions to threatening stimuli. The specificity and detail with which one verbalizes negative aspects of emotional experience has also been shown to reduce negative affect. Investigating this line of evidence further has important implications for exposure interventions, and potentially for other psychotherapy interventions as well.

The second line of evidence indicates that exposure treatments have a documented history of success, but certain emotional processes may be more important than others when it comes to determining the outcomes of exposure to feared situations. Habituation of fear has long been considered to be central to the therapeutic effects of exposure, which is in line with emotional processing theory. However, recent evidence points towards an inconsistent association between this habituation as an emotional process and change over the course of exposure, and instead points towards variability in the intensity of emotional states as the central therapeutic factor, which is more in line with inhibitory learning theory. Investigating these two emotional processes (i.e., habituation to emotion vs. variability in the intensity of emotion) as predictors of outcomes would be important in contributing to the evidence base of one theory over another.

The third line of evidence points towards the existence of a common inhibitory neural network involved in the regulation of all emotional states, including fear and shame. This network includes the amygdala-vACC-vmPFC neural circuit. Without the use of neuroimaging, the inhibitory activity of this neural circuit can only be inferred from the measurement of heart rate variability. However, such physiological measurement may shed further light on physiological correlates of implicit emotional processes, such as habituation and the variability in the intensity of emotional states.

A fourth line of evidence has to do with which emotion is central to exposure interventions. An emotion of fear has been considered central to exposure so far, although the emerging evidence indicates that other emotions have a role to play as well. In social anxiety disorder specifically, fear of embarrassment may be on par, or even secondary to, shame. It is important to understand whether that is the case to help guide future exposure research and interventions for social anxiety.

The current study

The present study aimed to investigate these lines of inquiry by recruiting university students with fear of public speaking to participate in an experimental design study. As part of the study, the participants were asked to come up with and deliver a videotaped speech on the topic of their choosing, with the understanding that the speech will later be evaluated by two expert raters. The participants were randomly assigned to one of three groups. The participants in the control group were asked to engage in a distraction word puzzle task. The participants in the exposure group were asked to watch their videotape for a total of three times while paying close attention to the video, although they were not informed of how many times they were to watch the recording. Finally, the participants in the experiential group were given the same instructions as the participants in the exposure group, except that after watching the video the participants were asked by the experimenter to verbally label and elaborate on their emotional experience. After the experimental manipulation (which comprised control, exposure, or experiential task) the participants were asked to come up with and deliver a second speech in the same way as before, except that it had to be on a different topic. The participants were not informed of the second speech to avoid the anticipation effects. The participants in all groups were

asked to fill out a series of measures before, after, and sometimes during the experimental task. The measures before and after the experimental task were aiming to assess behavioral and emotional changes, while the measures during the experimental task were aiming to measure emotional processes related to experiences of fear and shame, as well as indices of physiological activity.

Hypotheses

The following hypotheses were proposed by the study: (H1) there are greater behavioral and emotional changes in the experiential group, as compared to exposure group, or to the control group. It was predicted that the participants in the experiential group would have the most improvement in quality of their speech performance and state self-esteem, followed by the exposure group, followed by the control group. This hypothesis was examined through a comparison of change in measures from before the experimental task to after the experimental task while taking group membership into account. Additionally, (H2) process variables (i.e. initial activation, habituation and variability) related to fear and shame are predictive of change on the outcome measures. No specific predictions were made as to which emotional processes are more predictive of change given the mixed evidence to date. However, it was predicted that, irrespective of intervention, processes related to shame would be more predictive of the change in speech performance and state self-esteem outcome measures than experience of fear. These hypotheses were examined through the process component of the design. Finally, (H3) physiological indices of arousal are predictive of change on the outcome measures as well. It was predicted that higher heart rate variability during the task would predict better speech performance and state self-esteem at the conclusion of the study. No

specific prediction was made in regards to the high frequency component of measured heart rate variability, but it was expected that it would correlate with emotional process measures (i.e. both habituation, and variability in the intensity, of emotional states).

Methods

Sample

Participants were recruited from the Psychology Participant Pool at the University of Windsor, Ontario, and were awarded credit for their participation in accordance with the Participant Pool policy. Participants were recruited from the Undergraduate Participant Pool in two stages. During the first stage, to be eligible for the study, the participants had to answer “yes” to two screening questions: "Are you anxious in social situations?", and "Are you afraid of speaking in front of an audience?" Those participants who qualified could enter the online screening part of the study, where they were asked to fill out a basic demographics questionnaire as well as social anxiety screening measure. Those participants who fit the inclusion criteria were asked to come on campus to participate in the main part of the study in person.

To be included in the study, the participants had to satisfy the following criteria: (1) answer "yes" to both screening questions, as discussed above; (2) score at or above the cut-off scores on the Social Anxiety Disorder screening measures; and (3) not meet any of the exclusion criteria. As suggested by Antony and Swinson (2002), if a participant reported that he or she met the following criteria, he or she was excluded from the on-site study: (a) cardiovascular disease, high blood pressure, severe asthma or other respiratory disorder, hyperthyroidism or other respiratory disorder, or epilepsy; (b) current use of

benzodiazepines or beta-blocker medication; (c) excessive use of cannabis or alcohol in the past 30 days; (d) presence of suicidal ideation or self-harm in the past 30 days.

The study screened 318 participants online, and of those, 106 did not meet the cut-off criteria for social anxiety measures, and further 51 did not meet one or more of the other screening criteria (see Appendix E). The remaining 161 participants who met study criteria were contacted individually by email, and 45 of them came for the on-site study. Out of those who met in person, 4 individuals chose not to complete the study. All individuals who withdrew from the study chose to do so before or during the first speech task, which was part of the experimental protocol. The final sample consisted of 41 individuals, with an average age of 20.9 years ($SD = 3.9$), with 87.8% ($n = 36$) of participants female. In addition, 31.7% ($n = 13$) of the sample met diagnostic criteria for Social Anxiety Disorder based on the DSM-V criteria, as determined by a semi-structured interview at the beginning of the on-site visit. See Appendix F for additional demographic information.

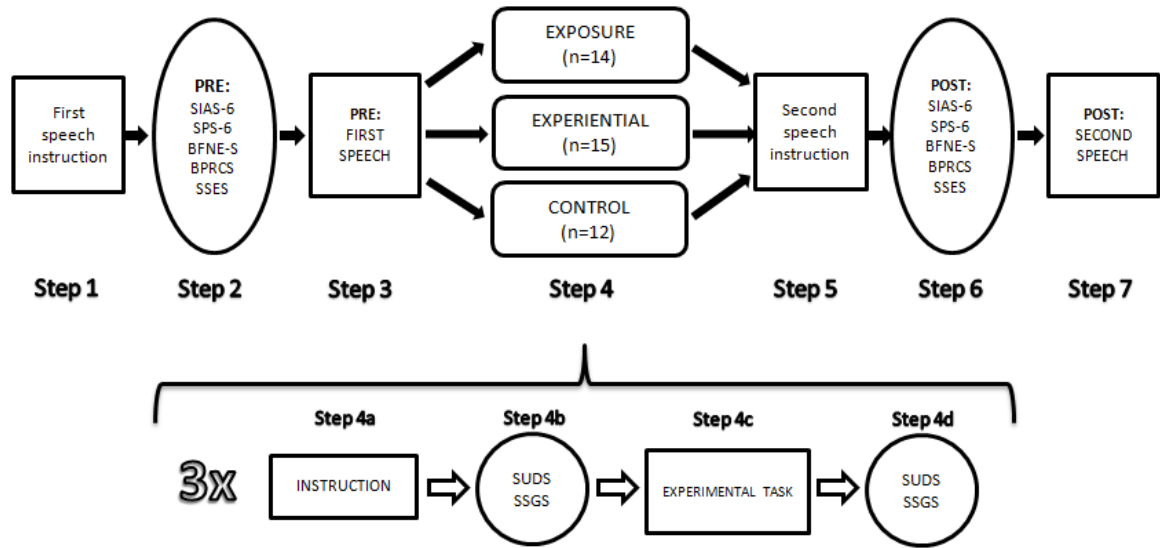
Procedure

The participants who qualified during the screening part of the study were invited for the on-site study which lasted approximately 60 minutes on average, and that took place on campus in a laboratory room, one participant at a time (see Figure 1 for a full design graph). Upon arrival, each participant was greeted by the experimenter and walked through the informed consent process and then randomly assigned to one of the three experimental groups. Irrespective of group assignment, after the consent forms were signed, audio recorder was turned on and recorded throughout the experimental session. The experimenter then took each participant to a separate room where the participant was

instructed on how to put on the heart rate chest strap, and then given time to change in privacy. After that, a 5-minute baseline period was recorded for the heart rate with the instruction to put away electronic devices and sit still while the experimenter stepped out of the room. Following that, the experimenter returned and asked the participant a series of questions from the semi-structured Social Anxiety Disorder interview.

The experimental procedure commenced and the participant was given the instructions for their first speech (Step 1) by the experimenter. Prior to and right after Step 1 the participant was asked to give a fear (SUDS) rating indicating how anxious he or she felt on a scale from 0 (not at all) to 100 (most anxious they have ever been). This procedure served as a manipulation check and aimed to establish whether the experimental procedures affected participant's anxiety. Following that, the participant was given a series of outcome questionnaires to fill out (Step 2). After the participant was finished with the questionnaires, Step 3 commenced. The experimenter told the participant that he or she would have 3 minutes to come up with a speech topic while the experimenter stepped out of the room. When the experimenter returned, the participant was given 3 minutes to deliver the speech in front of the video camera and the experimenter.

Figure 1. The experimental design.



After the participant delivered the speech, Step 4 commenced, which was the first step in the experiment where individuals received group-specific set of instructions for the task (see Appendix A for detailed instructions). There were three experimental groups in the protocol: control group, exposure group, and experiential group. Participants in all three groups followed the same general procedure: participants were given a group-specific set of instructions (Step 4a), after which they were asked to give their fear and shame ratings before the experimental task (Step 4b), after which they engaged in the group-specific experimental task (Step 4c), after which they were asked to give fear and shame ratings again (Step 4d). Participants in the control group were asked to solve a list of anagrams during the Step 4c. Participants in the exposure group were asked to watch the video of themselves giving the speech with the instruction to pay close attention. Participants in the experiential group were also asked to watch the video of themselves giving the speech and pay attention, and in addition to that after the video the participants were asked to verbally label and elaborate on emotions they experienced while watching

themselves on the recording (see Appendix A for detailed instructions participants received). This procedure (Steps 4a to 4d) was repeated a total of three times for each participant, and the participants were not informed of how many times they were to engage in the experimental task.

After completing the experimental task (Step 4), the participants in all groups received the same instructions for the remainder of the study. The participant was given instructions for their second speech (Step 5), and then asked to give a fear rating again. It is important to note that the participants were not informed that there was a second speech at any point in the study to avoid effects of anticipation. If a participant asked a question regarding upcoming procedures in the study, the experimenter told them that he cannot answer that question, but also noted that the study would be finished in the allotted amount of time and reminded the participant that they could end the study at any time without receiving penalty.

After the participant received the instruction regarding the second speech, he or she was asked to fill out the outcome questionnaires (Step 6), and prepare and deliver their second speech in the same way as before (Step 7), with a clarification that the second speech should be on a different topic than the first speech. Participants were then asked whether they wanted a debriefing on the study or wanted to ask any questions, and given a chance to do so.

The speech tasks. Speech videotaping procedures were modeled after the behavioral assessment tests (BATs), which are commonly used in social anxiety research and provide a semi-structured opportunity for observational evaluation of the participant's

quality of social performance, visibility of anxiety symptoms, as well as avoidance and escape behaviors (Hart, Jack, Turk, & Heimberg, 1999). In this paradigm, participants are often asked to confront a feared situation in a controlled environment, such as giving an impromptu videotaped speech in front of a small audience of confederates (Beidel, Turner, Jacob, & Cooley, 1989). In the proposed study participants were asked to give two 3-minute speeches in front of a video camera on the topic of their choosing. In prior research, physiological, cognitive, and behavioral data collected during the speech task was generally shown to have high test-retest reliability (Beidel et al., 1989), as well as sensitivity to the pre-to-post effects of treatment (Heimberg et al., 1990; Turner, Beidel, Cooley, Woody, & Messer, 1994). Thus, the speech task offers a flexible, semi-structured paradigm for subsequent measurements to be used.

Ethical considerations.

To address any concern about the safety of an exposure-based task, despite strong evidence that exposure interventions are generally as safe as other validated psychological interventions for the treatment of anxiety disorders, this study followed the guidelines suggested by Olatunji, Deacon, and Abramowitz (2009), and by Antony and Swinson (2002). To that end, the participants were informed of potential side-effects and benefits of challenging oneself with difficult emotional experiences, and reminded that they reserve the right to terminate the procedure at any time throughout the course of the session. Participants were also informed that if they find themselves feeling too uncomfortable, they can stop at any time.

Measures

Intervention outcome measures.

Social Interaction Anxiety Scale-6 (SIAS-6) and Social Phobia Scale-6 (SPS-6).

The SIAS/SPS-6 (Peters, Sunderland, Andrews, Rapee, & Mattick, 2012) is an adaptation of the widely used Social Interaction and Anxiety Scale (SIAS) and Social Phobia Scale (SPS; Mattick & Clarke, 1998), designed to provide a short screening and psychotherapy outcome tool for evaluating social anxiety. The SPS scale focuses on the assessment of fear of negative evaluation during routine activities, such as eating when others are present, while the SIAS focuses on fears of social interaction. The brief versions (of six items) used in this study correlated highly with the original versions (r 's = .88 - .94) at pre-, post-treatment, and 3-month follow-up, and showed convergent and discriminant validity. Both SIAS-6 and SPS-6 showed high diagnostic sensitivity (84.86 - 87.97) and specificity (86.05 - 97.67) in distinguishing individuals with social phobia from those without. Peters et al. (2012) suggested the following cut-offs for the presence of social anxiety disorder: a score of 7 or higher on SIAS-6, and a score of 2 or higher on SPS-6. While the short versions of these scales have not been used in single-session intervention studies as of yet, the full versions of SIAS and a shortened version of SPS were used as the screening measures in an experimental single-intervention study by Nilsson et al. (2012).

Brief Fear of Negative Evaluation Scale-Straightforward items (BFNE-S). The BFNE-S (Rodebaugh et al., 2004; Weeks et al., 2005) is an adaptation of Brief Fear of Negative Evaluation Scale (BNFE; Leary, 1983), which itself is an adaptation of the original Fear of Negative Evaluation Scale (Watson & Friend, 1969), a measure designed to assess the degree of fear of negative evaluation that is thought to be critical to the maintenance of social anxiety disorder. BFNE-S was found to have excellent internal

consistency ($\alpha = .90 - .96$), convergent validity with other measures of social anxiety, and discriminant validity with measures of mood. BFNE-S was found to have superior psychometric characteristics compared to BFNE-II (Carleton et al., 2007) and equivalent to BFNE-R (Carleton et al., 2006). It has been recommended over BFNE-R for a smaller gender difference in scoring (Carleton, Collimore, McCabe, & Antony, 2011).

Brief Personal Report of Confidence as a Speaker (BPRCS). The BPRCS (Hook, Smith, & Valentiner, 2008) is a 12-item adaptation of the 30-item Personal Report of Confidence as a Speaker (PRCS; Paul, 1966), and is a measure of fear of public speaking. BPRCS was found to have good internal consistency ($\alpha = .85$), good convergent validity with other measures of anxiety, and divergent validity with measure of depressed mood and sociability.

State Self-Esteem Scale (SSES). The SSES (Heatherton & Polivy, 1991) is a commonly used 20-item measure of self-esteem that is designed to capture experimentally-induced temporary deviations from a more global, stable view of one's self due to laboratory manipulations (Heatherton & Wyland, 2003). The SSES has a correlated 3 factor structure of performance, social and appearance self-esteem. The measure was found to have good internal consistency, $\alpha = .92$, as well as discriminant and construct validity (Heatherton & Polivy, 1991). Additionally, the SSES can successfully distinguish changes in mood from changes in state self-esteem (Bagozzi & Heatherton, 1994).

Social Performance Rating Scale, Modified (SPRS-M). The SPRS-M (Harb, Eng, Zaider, & Heimberg, 2003) is a standardized observer-rated measure of performance on a behavioral assessment test (described above) adapted from the Social Performance Rating

Scale (SPRS; Fydrich, Chambless, Perry, Buergener, & Beazley, 1998). The SPRS-M rates behavioral performance on four dimensions: gaze, voice quality, level of discomfort, and conversation flow. This modified version of the scale showed convergent and divergent validity with criterion measures of social anxiety (Harb et al., 2003).

For the purposes of this study, the measure was further modified to more closely correspond to the task the participants were asked to engage in, and to improve the reliability between the raters. A total of 82 videos (for 41 participants) were assigned a randomly generated 5-digit code, such that the raters could not infer which condition any given participant was in, or whether the speech was before or after the experimental task.

Process measures.

Subjective Units of Distress Scale (SUDS). The SUDS (Wolpe, 1958) is one of the most widely-used measures of anxiety to specific stimuli (Tanner, 2012). SUDS has the range of values from 0-100, although various ranges are used in practice (Antony & Swinson, 2002). SUDS was found to be a valid measure of both physical and emotional global discomfort (Tanner, 2012), and has been used as a measure of activation of fear structure (Foa & Kozak, 1986).

To test the effect of emotional processes on outcomes, this study used predictors suggested by Kircanski et al. (2012): (1) *Initial fear activation*, operationalized as the highest SUDS score during the experimental task; (2) *Within session habituation*, operationalized as the difference between the peak SUDS score and the final SUDS score at the end of the experimental task; (3) *Variance* in emotional process, operationalized as the standard deviation of SUDS during the experimental task. These variables were computed based on a total of six data points prior to and right after each iteration of the

experimental task, so as not to interfere with the experimental task itself (see Figure 1, steps 4b and 4d).

Like Culver et al. (2012), this study uses the term *habituation* to stay consistent with the terminology used in emotion processing theory and exposure literature in general, although the term here applies to the observation of attenuation in levels of fear over time rather than a hypothesized mechanism of change.

State Shame and Guilt Scale (SSGS). The SSGS (Marschall, Sanftner, & Tangney, 1994) is a 15 item, 3 subscale measure that evaluates the experience of shame, guilt, and pride as they occur in the moment (e.g. "I want to sink into the floor and disappear", "I feel small"). Only the Shame subscale will be used for this study. The Shame subscale has been demonstrated to have a good internal consistency ($\alpha = .82 - .89$).

Due to administrative error, the full SSGS Shame subscale (5 items) was completed by approximately half the sample ($n = 21$ out of 41). The rest of the sample only completed two items out of five, and subsequently, these two items were used to compute the overall SSGS Shame score². Examining the $n = 21$ subset, this abbreviated measure score correlated with the full scale score at an average of $r(20) = .96$ (range .94 to .98) across six time points, and therefore was deemed an acceptable substitution for the 5-item subscale as needed for the purposes of this study. Thus, total SSGS Shame subscale scores were computed from only two items for all 41 participants.

To test the effect of emotional processes on outcomes, process indices related to shame were created following the same logic as process indices related to fear: (1) *Initial*

² As indicated above, the inconsistency in participants' completion of the SSGS was due to an administrative error and as such was not a reflection of participant response patterns.

shame activation, operationalized as the highest SSGS score during the experimental task; (2) *Within session habituation*, operationalized as the difference between the peak SSGS score and the final SSGS score at the end of the experimental task; (3) *Variance* in emotional process, operationalized as the standard deviation of SSGS during the experimental task.

It is important to note that the term *habituation* of shame is used here as a convenient shorthand referring to an observation of a reduction in the intensity of shame over the course of the experimental task, rather than a hypothesized mechanism of change.

Physiological measures.

Heart rate was measured using a Polar S810i heart rate monitor which recorded R-R intervals for the duration of the study. A 5-minute resting heart rate baseline was assessed at the beginning of the study. Depolarization of the heart ventricles during heartbeat can be detected via electrocardiogram, and the resulting electrical signal (the R-wave of the QRS complex) can be recorded. The time between the two adjacent R-wave peaks (R-R intervals) indicates the passage of time between two adjacent heartbeats (Appelhans & Leucken, 2006). Because heartbeat in actuality is irregular, the time of R-R intervals varies substantially throughout the recording. Some form of measurement of variability in the length of the R-R intervals forms the basis of heart rate variability measurement. Generally speaking, these approaches fall into the time-domain and frequency-domain methods. The latter make use of Fast Fourier Transform to decompose the time signal into its constituent wavelengths (Task Force of The European Society of Cardiology and The North American Society of Pacing and Electrophysiology, 1996). Regardless of

measurement, reduced heart rate variability indicates a cardiovascular system which is not adaptable to environmental challenges and is associated with greater incidence of cardiovascular disease and mortality (Kemp & Quintana, 2013).

The current study examined a ratio of low to high frequency components of recorded heart rate variability (LF/HF), which is influenced by a combination of sympathetic and parasympathetic input, as an index of overall heart rate variability and flexibility in emotion regulation (Appelhans & Luecken, 2006). High frequency component (HF) of heart rate variability was examined as well, because it is mainly influenced by parasympathetic input and is thought to be an index of inhibitory activity of the prefrontal cortex (Thayer & Lane, 2009). Unlike SUDS and SSGS, which were measured right before and right after each iteration of the experimental task, heart rate variability was measured continuously during each iteration of the experimental task (see Figure 1, step 4c). It is important to note that heart rate variability was measured in three 3-minute intervals during each iteration of experimental task for any given experimental group. Such a short time interval for heart rate variability recording places a limitation on the accuracy and reliability of physiological findings.

Analysis

Results were analyzed using multilevel regression after Meuret et al. (2012) study which suggested this approach to analyzing similar type of data³. One of the key advantages of using multilevel regression is that it allows a way to model data change over time while simultaneously examining an experimental design as a multilevel data

³ An alternative approach using multiple regression (after Culver et al., 2012; Kirkanski et al., 2012) was also tried and produced very similar results.

structure so that experimental manipulation can be studied simultaneously with individual differences (Cohen et al., 2003). In this particular study, multilevel regression allowed a flexible approach to examining change across experimental interventions while simultaneously taking into account experimental group membership as well as dynamic changes within different emotional processes during the intervention. An additional advantage of this approach was that it allowed examining nested patterns of data simultaneously.

Full Maximum Likelihood (FML) procedure was used for all analyses, since it allows a direct comparison of both fixed and random effects of nested models (Hox, 2010). Robust error estimates of fixed effects are reported throughout. The statistical analysis strategy consisted of four steps. At each new step of regression a χ^2 deviance test was conducted to assess whether each new step results in a better fit of regression model to the data, and if the fit at any given step was not improved, no further steps in the analysis were taken. Step 1, the *intercept only model*, assessed the degree of intra-class correlation in the data. Step 2, the *random intercept and fixed slope model* with Time as level-1 predictor, assessed whether there was any overall change pre-to-post on a given measure for the individuals, and if this step failed to find significant change over time, no further analysis was conducted. Step 3 used level-2 predictors (group membership and process variables) to examine which variables influenced change over time on any given variable. Step 4 added *random slopes* to the model to test whether contextual variables exerted different effects on change pre-to-post for different individuals, i.e. whether the individuals' slopes varied significantly around the average slope. Random slopes were

modeled during the last step, since Hox (2010) suggested that the fixed effects model should have a good fit before the random effects are introduced.

Pre and post observations were entered at level 1 ($n = 82$), and were nested within individuals at level-2 ($n = 41$). Because HLM7 software does not automatically produce beta-weights, they were calculated from unstandardized coefficients using the formula $\beta = B * SD_{\text{predictor}} / SD_{\text{outcome}}$ (SSI Central, n.d.). All continuous predictors were centered around their respective grand means.

Assumptions Testing

The data were first explored using histograms and boxplots to determine presence of unusual cases. Model assumptions were checked for violations by examining model residuals at both levels for the intercept-only model, as well as the final model for each outcome variable. Normality of residuals was evaluated through Q-Q plots at both levels. Homoscedasticity was evaluated by a formal chi-squared test provided by the HLM7 software at level 1, and by plotting residuals against their predicted scores at both levels. Linearity was assessed by plotting the residuals against the predictor variables and superimposing a lowess fit line (Cohen et al., 2003). Presence of multicollinearity was tested by examining the Multilevel Variance Inflation Factor scores using the inverse of the predictor correlation matrix (Clark, 2013).

Results

Inter-rater reliability for speech performance (SPRS-M)

Two raters who were blind to the conditions (the experimenter and an undergraduate research assistant) each coded 100% of the videos using the SPRS-M scale. The raters underwent approximately three hours of training over the course of two

days on three-minute YouTube videos before rating the experimental videos. Inter-rater agreement was assessed using the intra-class correlation (ICC), which was computed in SPSS (two-way random, absolute agreement) following suggestions of Landers (2015). Inter-rater agreement for the videos was high, $ICC(2, 2) = .953$. After reliability was calculated, all disagreements were resolved by discussion that led to consensus between the raters. These minor changes produced a higher caliber data set that was then submitted for the analyses that follow.

Manipulation checks

A paired-samples t-test indicated that the instruction before the first speech task exerted the desired experimental effect, significantly raising the levels of self-reported fear from an average of 46.4 to 59.2 points on the SUDS scale, $t(40) = 8.016, p < .001$. In addition to that, the average levels of both self-reported fear and shame during the experimental task were not significantly different among the three groups, $F(2, 38) = .253, p = .778$, for fear, and $F(2, 38) = .404, p = .671$, for shame, respectively. These results suggest that the participants in the active control group were under approximately the same amount of stress while solving word puzzles as the participants who watched themselves on the videotape, despite being told they will not be evaluated on the task.

Assumptions

No pronounced violations of assumptions were detected. The model residuals appeared to be normally distributed on both levels, variances met the homoscedasticity requirement, and the relationship between the predictor variables and outcomes appeared to be linear. One anomaly, however, was found in the analysis: Within session habituation of fear was highly correlated with variability in fear levels (SUDS; $r = .92, p$

< .001). A similar high correlation was observed in relation to habituation and variability in shame (SSGS; $r = .76, p < .001$), although habituation of shame and variability in shame were less highly related than for fear.

These relationships between the predictors introduced a significant degree of multicollinearity in the model (see Table 1). It is important to note that multicollinearity does not violate any assumptions of the regression (Voss, 2004), but it does result in a number of complications. It is known to produce unstable (or context-specific) parameter coefficients with wide standard errors, leading to attenuated power in multiple regression (Cohen et al., 2003). Multilevel regression appears to suffer from the same problems, so that even relatively large regression coefficients run the risk of being found non-significant under substantial multicollinearity (Clark, 2013). While severe multicollinearity makes interpretation of individual coefficients difficult, it does not affect joint influence of the predictors when tested simultaneously (Cohen et al., 2003). Moreover, common remedies for collinearity such as dropping one of the correlated predictors or using the principal components regression create their own difficulties in interpretation (Voss, 2004). The only remedy chosen in this situation was centering the predictors.

Table 1

Multilevel variance inflation factor (MVIF) values of emotional process predictors.

	MVIF	
<u>Predictor</u>	<u>SUDS</u>	<u>SSGS</u>

Initial activation	1.194	2.544
Variability	7.612	3.842
Within session habituation	7.796	2.334

Note. SUDS = Subjective units of distress scale; SSGS =

State shame and guilt scale (shame subscale).

A closer inspection of the highly correlated predictors revealed that when they were entered into the model separately, they were not significant, but the significance changed drastically when they were entered together. An inspection of zero-order and partial correlations revealed the presence of statistical suppression both when fear process predictors were used (see Table 2) and when shame process predictors were used (see Table 3). Note that in both tables process variables are positively correlated with each other while also correlating with the outcome in opposite directions. In addition to that, note the change in the magnitude of effect when one process variable is correlated with the outcome while being controlled for the other process variable. Both of these signs are tell-tale marks of statistical suppression (Paulhus et al., 2004).

Statistical *suppression* occurs when the relationship between two predictor variables hides their true relationships with the outcome variable (Cohen et al., 2003). Likewise, Paulhus et al. (2004) suggest that when the relationship between the predictor variables results in a “suppression situation,” the indirect effects of variables are so strong that they overwhelm the direct effects. Paulhus et al. (2004) give an intuitive example of where one finds statistical suppression, involving the constructs of self-esteem and narcissism, two variables which positively correlate with each other. However, because their measure

of self-esteem inadvertently also measured narcissism, it was thus “contaminated” by it. This produced the puzzling net effect that self-esteem appeared to have slight positive, negative, and null correlations with antisocial behaviour across three samples. When self-esteem and narcissism were entered into regression jointly, their true relationship with antisocial behaviour was revealed: self-esteem had a strong negative correlation with antisocial behaviour, while narcissism had a strong positive correlation across all three samples – despite the fact that the variables were positively correlated with one another. More informative direct effects, given suppression, can only be revealed if the indirect path is controlled for. This can be accomplished by removing the shared variance between the predictors so that they do not “contaminate” each other by entering both predictors in the regression model as a block. The implications of this for interpreting the findings are further explored in the discussion section.

Table 2

Full and partial correlations between fear processes and speech performance

	Speech performance
Fear Habituation	-0.12
Fear Habituation †	-0.38*
Fear Variability	0.02
Fear Variability ‡	0.36*
	Fear Habituation
Fear Variability	0.93**

Note. SPRS-M = Social performance rating scale-modified; SUDS = Subjective

units of distress scale; * $p < .05$; ** $p < .01$; † controlled for variability; ‡ controlled for habituation. Note that fear process variables are positively correlated with each other but correlate in opposite directions with speech performance.

Table 3

Full and partial correlations between shame processes and state self-esteem

	State self-esteem
Shame Habituation	0.06
Shame Habituation†	0.35*
Shame Variability	-0.21
Shame Variability‡	-0.40*
	Shame Habituation
Shame Variability	0.76**

Note. SSES = State self-esteem scale; SSGS = State shame and guilt scale (shame subscale); * $p < .05$; ** $p < .01$; † controlled for variability; ‡ controlled for habituation. Note that shame process variables are positively correlated with each other but correlate in opposite directions with state self-esteem.

Main Analyses

The analysis failed to find any changes from before the experimental task to after the task on either Brief Fear of Negative Evaluation-Straightforward items (BFNE-S) or Brief Personal Record of Confidence as Speaker (BPRCS) measures, so no further analyses were carried out on them. The same analyses were conducted for the Social

Performance Rating Scale – Modified (SPRS-M) and State Self-Esteem Scale (SSES).

The results indicated that the latter two measures showed significant change from before the experimental task to after the task, and this change was examined further. Findings related to each of these two latter outcome measures are presented in respective sections below. Correlations between outcome measures are presented in Appendix D, while respective means and standard deviations can be found in Appendix G.

Testing hypothesis 1: Do verbal processes affect outcomes?

Experimental group predicts somewhat better speech performance (SPRS-M).

Initial steps of the analysis indicated a significant change over time in speech performance. These were followed up with an analysis that simultaneously introduced two orthogonal contrasts. Contrast A compared the two active groups vs. the control group, while Contrast B specifically compared the experiential group vs. the exposure group. As expected, no significant group differences were found on their baseline speech performance, indicating that the random assignment into the groups worked as intended. Contrast A found that experiential and exposure groups taken together improved their speech performance to a greater degree, $\beta = .15$, $t(38) = 2.01$, $p = .052$, when compared to the Control group. These results indicate that processing the experience during the speech in some way made a difference over not processing that experience and using distraction instead. Even so, this difference was only marginally statistically significant, and the effect size was small. At the same time, contrast B indicated that the experiential group improved speech performance to a greater degree as compared to the exposure group, $\beta = .16$, $t(38) = 2.04$, $p = .048$, with a small effect size as well. These results indicate that verbally elaborating on the experience during the speech was more effective than simply

observing the videotape without explicitly talking about the experience. While these contrasts were each statistically significant individually (or were close to being significant), regression which included this set of contrasts did not pass the deviance test, $\chi^2(4) = 7.49, p = .111$, indicating that regression as a whole did not become more accurate when these two contrasts were included as compared to regression which did not take these contrasts into account. Thus, group membership offered an overall weak explanation of change in speech performance.

An additional post hoc analysis was also carried out to test whether diagnosable social anxiety affected the results of the previous analysis. This additional analysis indicated that having diagnosable social anxiety improved the accuracy of regression, $\chi^2(2) = 6.84, p = .032$, and indicated that participants with diagnosable social anxiety had worse first speech performance compared to rest of the sample, $\beta = -.38, t(37) = -2.83, p = .007$. Inclusion of this predictor rendered both orthogonal contrasts discussed above statistically non-significant, but otherwise did not change the interpretation of the results.

Experimental group does not predict state self-esteem (SSES).

The analysis found no effect of experimental group on SSES prior to the experimental task or over time. This indicates that the random assignment was successful and all three groups had the same level of state self-esteem prior to the experimental task. Moreover, subsequent change in state self-esteem from before to after the experimental task was unrelated to which group the individuals were assigned to. All further analyses involving fear and shame processes were carried out on the whole dataset irrespective of group assignment because, as mentioned before, participants did not differ in the levels of

fear and shame they experienced during the experimental task based on their group membership, including the control group.

An additional post hoc analysis was also carried out to test whether diagnosable social anxiety affected the results of the previous analysis. This additional analysis indicated that having diagnosable social anxiety improved the accuracy of regression, $\chi^2(2) = 15.76, p < .001$, and indicated that participants with diagnosable social anxiety had worse state self-esteem at the onset of the study compared to rest of the sample, $\beta = -.57, t(37) = -5.25, p < .001$. Inclusion of this predictor did not markedly change beta-weights, significance levels, or the interpretation of the results related to other predictors.

Hypothesis 1 results summary.

Overall, it appears that Hypothesis 1 was only weakly substantiated. There was evidence to indicate that exposure together with verbal labelling and elaborating on emotional experience may result in a slight improvement in speech performance when compared to exposure alone, but there was no evidence to indicate that such verbal processing influenced changes in state self-esteem.

Testing hypothesis 2: Do emotional processes predict outcomes?

Strategy for analysis of emotional processes.

Because the effects of experimental manipulation were ambiguous, and because the participants in the control group experienced the same amount of fear and shame during the word puzzle solving task as the other two groups did during the experimental interventions, all further analyses involving fear and shame processes were carried out on the whole dataset irrespective of group assignment. This analysis strategy was pursued to both examine the emotional processes themselves and their effects more closely and to

preserve statistical power. Means and standard deviations for emotional processes can be found in Appendix H for fear, and appendix I for shame.

Ratings of distress (SUDS) predict speech performance (SPRS-M).

Worse speech performance during the first speech was associated with greater initial fear activation during the experimental task, $\beta = -.45$, $t(37) = -3.22$, $p = .003$. These results indicate that individuals who did not perform well during the first speech were unsettled by their poor performance at the beginning of the experimental task. As mentioned earlier, there was an overall improvement in speech performance from before the experimental task to after the task, $\beta = .15$, $t(37) = 3.98$, $p < .001$, likely because of practice effects, and the following process predictors should be interpreted in the context of this overall improvement. Greater within session habituation of fear during the experimental task inhibited or reversed the prediction of improvement in speech performance, $\beta = -.63$, $t(37) = -3.54$, $p = .001$ (see Figure 2), suggesting that becoming more at ease during the task was detrimental for subsequent performance. Greater initial fear activation was related to improvement in speech performance from before the experimental task to after the task, $\beta = .16$, $t(37) = 2.13$, $p = .040$, and so was greater variability in fear during the experimental task, $\beta = .54$, $t(37) = 2.82$, $p = .008$ (see Figure 3), suggesting that individuals who felt unsettled at the beginning of the experimental task and who experienced more pronounced highs and lows of anxiety tended to deliver a better next speech, possibly due to experiencing the optimal amount of subjective anxiety that was mobilizing, rather than hindering. Further analysis found that including random slopes into the model did not improve the model fit.

An additional post hoc analysis was also carried out to test whether diagnosable social anxiety affected the results of the previous analysis. This additional analysis indicated that having diagnosable social anxiety correlated highly with initial fear activation, $r(39) = .41, p = .006$, but diagnosable social anxiety as a predictor did not improve the accuracy of regression, $\chi^2(2) = 3.44, p = .177$, and did not change the interpretation of the results.

In summary, an average participant improved his or her speech performance due to practice. The participants who performed poorly during the first speech were likely unsettled by the quality of their performance at the beginning of the experimental task, resulting in heightened anxiety. This initial anxiety appeared to benefit them as these participants tended to subsequently improve their performance. In fact, becoming less anxious and more at ease during the experimental task was detrimental to the subsequent speech performance, and may have reflected some participants' disengagement or boredom. The participants who experienced a greater range of anxiety responses, from weak to strong, during the experimental task tended to improve their subsequent speech performance the most.

Given the presence of statistical suppression, as discussed earlier, these results should be interpreted with extra caution. Because within session habituation and variance in fear levels create statistical suppression, their regression coefficients must be interpreted to mean, e.g., an effect of within session habituation of fear on speech performance, but *only* when controlled for variance in fear levels. Likewise, these results indicate an effect of variance in fear, but only when controlled for within session

habituation. Since initial fear activation was not involved in statistical suppression, its coefficient can be interpreted as usual.

Ratings of anxiety (SUDS) do not predict state self-esteem (SSES). Lower state self-esteem prior to experimental task was associated with greater initial fear activation at the onset of the experimental task, $\beta = -.58$, $t(37) = -2.41$, $p < .001$. No other fear process variables (i.e., within session habituation and variability in fear levels) were predictive of state self-esteem change from before the experimental task to after the task. These results indicate that individuals who had lower self-confidence prior to the experimental task were unsettled more easily at the onset of the task compared to the individuals who had higher self-confidence.

Ratings of shame (SSGS) do not predict speech performance (SPRS-M). Worse performance during the first speech was associated with a greater initial shame activation during the subsequent experimental task, $\beta = -.53$, $t(37) = -2.44$, $p = .021$. Within session habituation of shame and variability in shame were not predictive of change in performance from the first to the second speech. Further analysis found that including random slopes did not improve the model fit. No other processes related to shame emerged as good predictors of change in speech performance from first to second speech. These results indicate that individuals who did not perform well during the first speech were likely aware of their poor performance and were embarrassed by the quality of their performance at the onset of the experimental task.

Ratings of shame (SSGS) predict state self-esteem (SSES). Lower state self-esteem prior to the experimental task was associated with greater initial shame activation during the experimental task, $\beta = -.43$, $t(37) = -2.06$, $p = .047$. These results indicate that having

lower self-confidence led to being more easily embarrassed at the beginning of the experimental task. There was an overall worsening in state self-esteem from before the experimental task to after the task, $\beta = -.18$, $t(37) = -4.11$, $p < .001$, suggesting that the task may have served as a “reality check” for a number of individuals, resulting in an overall drop in self-confidence. The following predictors should be interpreted in the context this overall decrease. Higher within session habituation of shame served a protective function, slowing and potentially reversing any decreases in state self-esteem from before to after the task, $\beta = .31$, $t(37) = 2.77$, $p = .009$ (see Figure 4). However at the same time, higher variability in shame predicted further worsening of state self-esteem from before to after the experimental task, $\beta = -.35$, $t(37) = -2.16$, $p = .038$ (see Figure 5). In this sense, habituation of shame can be thought of as “protecting” or “buffering” against the detrimental effect of initial shame activation and variability in shame. Further analysis found that including random slopes did not improve the model fit.

An additional post hoc analysis was carried out to test whether diagnosable social anxiety influenced the results of the previous analysis. Diagnosable social anxiety did not significantly correlate with initial shame activation, $r(39) = .25$, $p = .107$. Including diagnosable social anxiety as a predictor improved the model fit, $\chi^2(2) = 14.63$, $p = .001$, and rendered predictive power of initial shame activation null. The results of this new model (which included diagnosable social anxiety as a predictor, but excluded initial shame activation to preserve degrees of freedom) indicated that individuals who are diagnosable with social anxiety tended to have lower state self-esteem at the onset of the task, $\beta = -.50$, $t(37) = -5.07$, $p < .001$, but also tended to improve their state self-esteem somewhat over the course of the study, $\beta = .16$, $t(37) = 1.90$, $p = .066$, although this

improvement was not statistically significant. Inclusion of diagnosable social anxiety as a predictor did not drastically influence the beta-weights or significance levels of habituation and variability in the intensity of shame as predictors, or the interpretation of other results.

Thus, an average participant experienced a temporary loss of self-confidence at the conclusion of the study. The participants who possessed less self-confidence prior to the experimental task were more easily embarrassed at the onset of that task. The participants who successfully managed their embarrassment during the task, however, appeared to not lose their self-confidence or only lost very little, while those who fluctuated between weak and strong feelings of embarrassment lost the most self-confidence. The same caution as mentioned previously applies to interpreting the coefficients of within session habituation of shame and variability in shame due to statistical suppression, while initial shame activation coefficient can be interpreted as usual.

Hypothesis 2 results summary.

Overall, it appears that Hypothesis 2 was partially substantiated. Both habituation and variability in the intensity of emotional states were predictive of change on behavioral and emotional outcome measures. A surprising finding was that the relationship between habituation and variability in the intensity of emotions turned out to be more complicated than initially thought.

The hypothesis was not substantiated to a degree in that shame did not appear to be more central than fear in the context of exposure task for individuals with fear of public speaking. However, somewhat in line with the hypothesis, shame appeared to be on par with fear in the sense that emotional processes related to shame predicted change in state

self-esteem, while emotional processes related to fear predicted change in speech performance quality. Moreover, this finding underlines the importance of multi-modal assessment strategy in the context of exposure to feared situations, since not including one of the modalities of assessment just mentioned would overlook one of the current findings.

Testing hypothesis 3: Do physiological indices predict outcomes?

Experimental group did not predict increase in LF/HF ratio.

There was a significant linear increase, $\beta = .23$, $t(77) = 3.80$, $p < .001$, for ratio of low to high frequency components of recorded heart rate variability (LF/HF) over the course of the experiment. This indicates there was an increase in LF/HF ratio from the 5-minute baseline to the first speech, followed by further increase after the experimental task to the second speech. However, participant membership in any of the experimental groups (including the control group) did not influence this increase. Thus, while the experimental task (including the control task) appeared to make an impact on the participants, resulting in increased physiological arousal over time, any differences between the experimental conditions were too subtle to be detected. Means and standard deviations of LF/HF ratios are presented in Appendix J.

Lower LF/HF ratio predicts deterioration of state self-esteem (SSES).

Change in LF/HF ratio was operationalized as a simple difference between LF/HF ratio during the first 3-minute experimental task (e.g. watching the video or solving the word puzzles) and the third, and last, 3-minute task. Change in LF/HF ratio during the experimental task did not predict changes on behavioral or emotional outcome measures from before the experimental task to after the task. Average LF/HF ratio was

operationalized as arithmetic average of LF/HF ratio for all three 5-minute experimental task segments. Higher average LF/HF ratio (indicating lower heart rate variability) during the experimental task predicted a worsening in state self-esteem from before the experimental task to after the task ($\beta = -.18$, $t(37) = -2.21$, $p = .034$). These results indicate that lower heart rate variability may indeed reflect poor emotion regulation capacity, as suggested by the literature (e.g. Appelhans & Luecken, 2006), which may lead to a temporary decrease in self-esteem due to difficulty regulating emotions arising out of the challenges the experimental task posed. It is important to note, however, that this particular analysis did not pass the deviance test, $\chi^2(2) = 5.36$, $p = .067$, and thus should be interpreted as very tentative.

HF component of HRV positively correlates with habituation of emotional states.

While high frequency (HF) component of heart rate variability (HRV) during the experimental task did not directly predict change on behavioral or emotion outcome measures from before the task to after the task, it significantly correlated with emotional processes variables. Given the statistical suppression between habituation and variability in emotion intensity, partial correlations were studied between HF component of HRV and these emotional process variables. The partial correlations revealed that HF component of HRV significantly correlated with habituation in fear ($r = .40$, $p = .014$; when controlled for variability in the intensity of fear) and shame ($r = .56$, $p < .001$; when controlled for variability in the intensity of shame) during the experimental task. At the same time, HF component of HRV negatively correlated with variability in the intensity of fear ($r = -.26$, $p = .117$; when controlled for habituation of fear) and variability in the intensity of shame ($r = -.48$, $p = .003$; when controlled for habituation of shame). While

one of the correlations did not reach statistical significance, correlations between HF component of HRV and variability in the intensity of emotions were in the consistently negative direction.

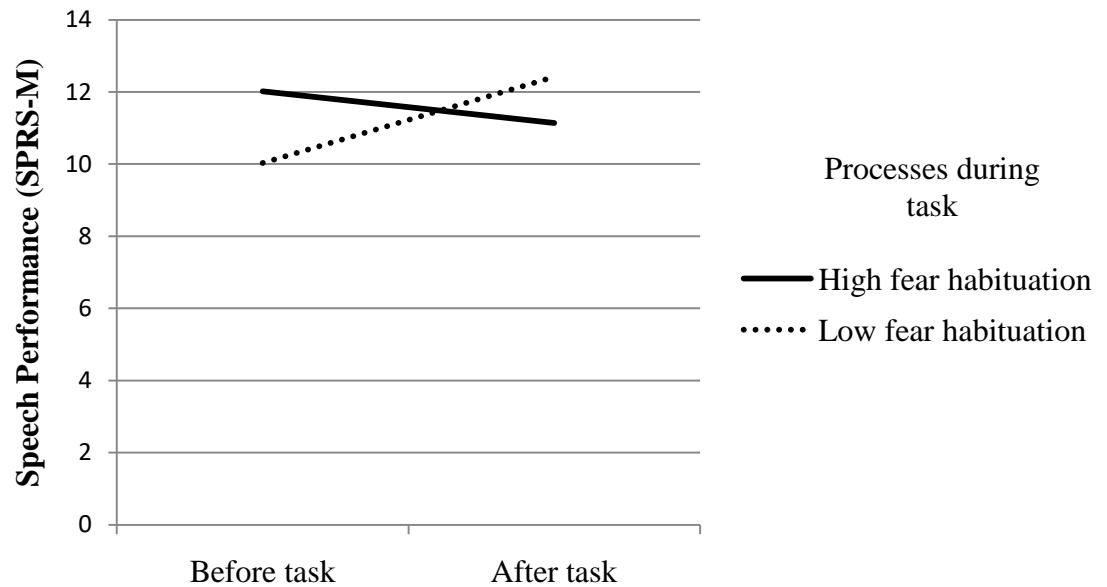
These results indicate that habituation, or decrease in the intensity of subjective emotional states appears to coincide with lessened physiological arousal due to the activation of parasympathetic nervous system. Conversely, variability in the intensity of emotional states coincided with increased physiological arousal due to parasympathetic withdrawal, but also potentially sympathetic activation, or both. Means and standard deviations of HF component of recorded heart rate variability are presented in Appendix K.

Hypothesis 3 results summary.

Hypothesis 3 was substantiated because higher LF/HF ratio, which indicates lower heart rate variability, correlated with a deterioration in state self-esteem from before to after the experimental task. This is consistent with the idea that lower heart rate variability indicates a deficit in emotional processing capacity, which would lead an individual to struggle coping with the demands posed by the experimental task.

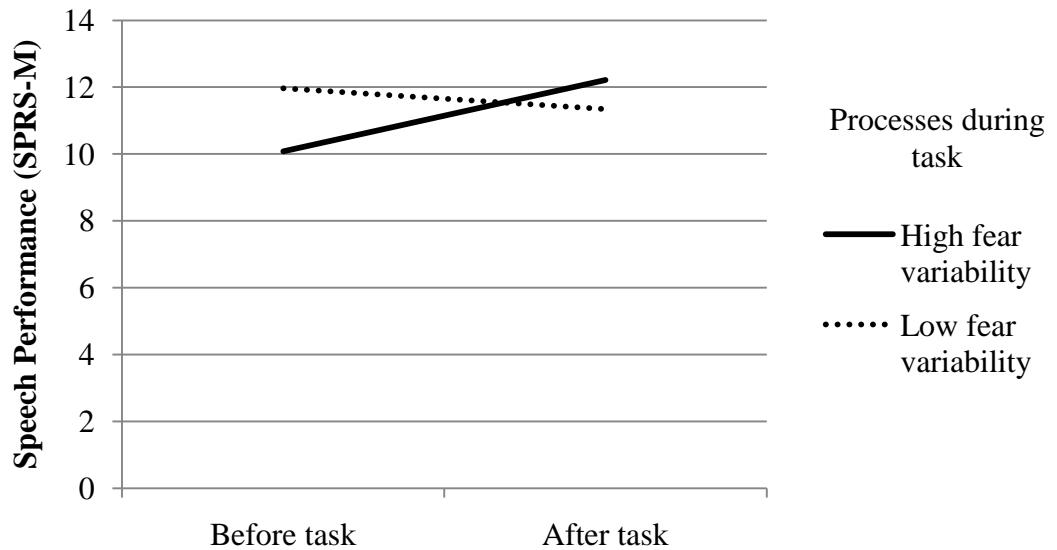
At the same time, the high frequency component of recorded heart rate variability positively correlated with habituation in the intensity of emotional states. This finding is consistent with the idea that habituation (in the sense of a reduction in the reported intensity of emotions) may reflect an inhibitory process involving the amygdala-vACC-vmPFC inhibitory circuit. Even so, this particular interpretation should be taken with considerable caution, since it is based on a theoretical inference, rather than neuroimaging results.

Figure 2. Effect of habituation of fear during combined experimental tasks on speech performance.



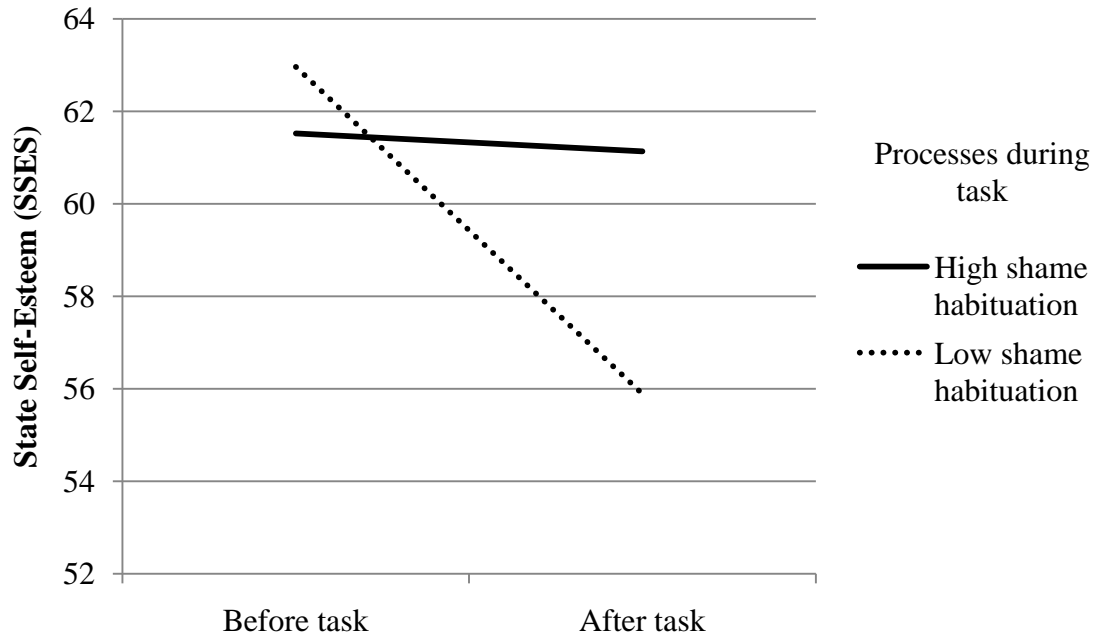
Notes. The graph is plotted for an average individual with either high or low habituation during the task ($\pm 1SD$ from the mean) at average levels of initial fear activation and variability in fear. Higher scores represent better performance. This graph shows that individuals who do not become more at ease during the task improve their speech performance, while those who experience a reduction of anxiety see a slight speech performance drop. The graph is based on $n = 41$ participants.

Figure 3. Effect of variability of fear during combined experimental tasks on speech performance.



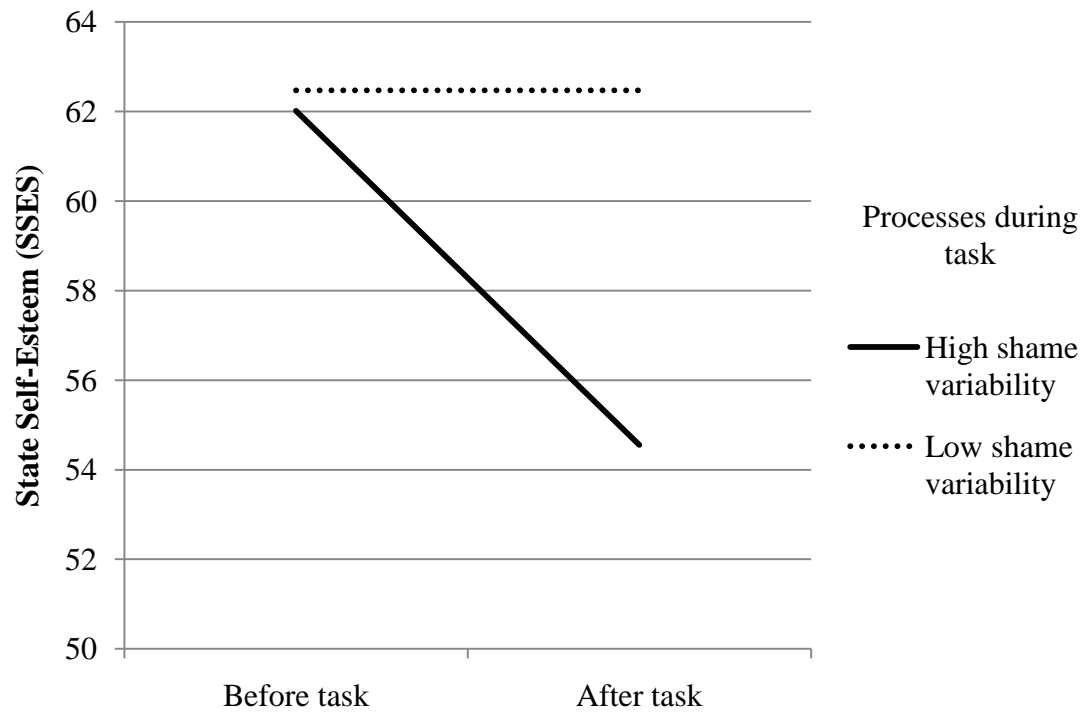
Notes. The graph is plotted for an average individual with either high or low variability of fear during the task ($\pm 1SD$ from the mean) at average levels of initial fear activation and within session habituation in fear. Higher scores represent better performance. The graph indicates that individuals who experience more pronounced highs and lows of anxiety during the task improve their speech performance, while those who have a more stable experience of anxiety see a slight speech performance drop. The graph is based on $n = 41$ participants.

Figure 4. Effect of habituation of shame during combined experimental tasks on state self-esteem.



Notes. The graph is plotted for an average individual with either high or low habituation of shame during the task ($\pm 1SD$ from the mean) at average levels of initial shame activation and average variability in shame. Higher scores represent better self-esteem. This graph shows that individuals who are able to regulate and reduce their experience of shame tend to keep confidence in themselves, while those who are not tend to lose that confidence. The graph is based on $n = 41$ participants.

Figure 5. Effect of variability in shame during combined experimental tasks on state self-esteem.



Notes. The graph is plotted for an average individual with either high or low variability of shame during the task ($\pm 1SD$ from the mean) at average levels of initial shame activation and within session habituation in shame. Higher scores represent better self-esteem. This graph indicates that individuals who are able to maintain an emotionally stable experience, in so far as shame is concerned, tend to preserve their self-esteem. In contrast, those who experience significant highs and lows in the intensity of feeling shame lose their confidence. The graph is based on $n = 41$ participants.

Discussion

Exposure treatments have been found to be an effective treatment for a variety of psychological disorders, but the mechanisms behind them remain somewhat elusive. Emotion processing theory's view (Foa & Kozak, 1986; Foa, Huppert, & Cahill, 2006) stressed the importance of within- and between-session habituation of fear as the principal therapeutic component, but the theory behind this mechanism of change has recently been questioned (i.e., Culver et al., 2012; Craske et al., 2014). Inhibitory learning theory proposed a different set of processes that occur during exposure to predict outcomes, among them, increased variability in fear and sustained physiological arousal. Verbal processes appear to influence the course of exposure as well, likely through the interaction between two neural streams that underlie both extinction and processing of emotions. The present study investigated whether verbalizing and elaborating on emotional experience contributes to changes over the course of an exposure task. Additionally, this study investigated a set of predictors of therapeutic change postulated by emotion processing theory and inhibitory learning theory, and additionally investigated whether the discrete emotion of shame was also important, as suggested by theory from emotion focused therapy. This was done in the context of a speech task in an analog sample of college students who reported both fear of public speaking and symptoms of social anxiety.

The study investigated whether verbal elaboration on emotional experience contributes to changes in behavioral and emotional outcomes during exposure to feared situations, as well as tested predictors of such change postulated by two competing theories, the emotion processing theory, and the inhibitory learning theory. The results offer mixed support for the hypothesis that verbal elaboration of emotional experience

during an exposure task improves the outcome of exposure-based interventions. The study also found some support for predictors postulated by both emotion processing theory and inhibitory learning theory, but also found that the relationship between these predictors may be more complicated than previously thought. Finally, this study found evidence that process predictors specifically related to shame may be as important as processes related to fear, but that effect may depend on the modality (i.e., method) by which outcomes are measured.

Physiological findings indicate that overall lower heart rate variability (HRV) during the task was associated with a decrease in state self-esteem from before the task to after the task. This finding is consistent with the theory that heart rate variability is an index of emotion regulation, and lower heart rate variability likely reflects deficits in emotion regulation capacity (Appelhans & Leucken, 2006), leading to a temporary deterioration of self-esteem due to difficulty managing challenges that the experimental task posed. In addition to that, vagally-mediated heart rate variability, indexed by high frequency component of HRV, correlated positively with habituation in both fear and shame (when controlled for variability in the intensity of these emotions). These findings raise an intriguing (albeit speculative) possibility that high frequency component of HRV reflects the engagement of the amygdala-vACC-vmPFC inhibitory circuit, meaning that habituation of emotional states, such as fear or shame, may be an index of inhibitory learning taking place, so long as variability in the intensity of emotional states is taken into account as well. It is important to note that this particular conclusion is based on extrapolation beyond the data produced by the current study.

Experiential group showed greater behavioral change as compared to Exposure group

Hypothesis 1 predicted greater cognitive and emotional changes in the experiential group (i.e., exposure plus experiential processing), as compared to exposure alone, or to the control group. This hypothesis was supported to a degree, in that while group membership was unable to predict changes in self-esteem, it did predict behavioral changes. First, the results confirm that all three groups were equal on the initial speech performance prior to the experimental manipulation. Second, experiential and exposure groups taken together improved in their speech performance over the control group, while experiential group improved to a greater degree as compared to exposure group. However, in both cases, the improvement effect size magnitude was small. In addition to that, diagnosable social anxiety differentiated individuals on the basis of the quality of their initial speech performance and state self-esteem at the onset of the study, with more socially anxious individuals being worse off in both categories.

While group membership overall was a weak predictor of change in speech performance, statistical power should be considered in the interpretation of this result. The true effect size of a single-session experimental manipulation in an analog sample with a relatively low severity of symptoms is likely to be very small, meaning that a very large sample is needed to obtain sufficient power. The study was only able to recruit 41 individuals, and with a sample size this modest the analysis comparing the groups was underpowered, resulting in inconclusive findings regarding the effects of experimental groups.

Habituation and variability in emotional arousal predict change - but in opposite directions

Hypothesis 2 predicted that variability in the subjective emotional states of fear and shame during the non-control experimental tasks will be more predictive of change on the outcome measures compared to traditional predictors (initial fear activation and within session habituation). Variability in the intensity of both fear and shame emotional processes during all three experimental tasks, including the control group, were predictive of changes on the outcome measures, although the relationships among these emotional processes and the outcomes appeared to be more complex than initially thought. While the participants improved their speech performance on average, likely due to practice effects, greater habituation of fear (i.e., reduced within-task experience of fear from its peak to the end of task) during the experimental task (when controlled for variability in fear levels) predicted worsening in speech performance. This somewhat counterintuitive finding might be explained by the idea of optimal arousal proposed by Catherall (2003) and Hayes et al. (2012). It is possible that individuals who habituated experienced a lower degree of arousal which was somewhat detrimental to their performance, while those who did not habituate experienced an optimal amount of arousal which nonetheless never reached levels high enough to be detrimental to performance. In contrast, greater variability in fear during the experimental task (when controlled for the simultaneous habituation of fear) predicted improvement in speech delivery. Diagnosable social anxiety was associated with lower self-esteem prior to the experimental task, higher fear at the onset of experimental task, and also with a slight

improvement in state self-esteem over the course of the study. This predictor otherwise did not significantly impact any other findings.

The findings discussed above are consistent with Culver et al. (2012) study, in which the participants could stop their speech at any point but were encouraged to deliver speeches lasting up to 2 minutes, which resulted in a behavioral measure of speech duration. That study found that when participants did not substantially habituate to fear within the session, it seemed to allow an improved behavioral performance and predicted participants delivering a longer speech at one week follow-up after the study. The current study also found that less habituation predicted higher overall quality of speech performance (because greater habituation predicted worse performance). However, both Culver et al. (2012) and Kircanski et al. (2012) found that variability in fear levels during exposure (i.e., the standard deviation of fear levels during the task), as measured on the SUDS scale, predicted lower fear levels during the follow-up, also measured by the SUDS scale. The current study further extended those findings, suggesting that greater variability in one's reported levels of fear is predictive of an outcome measure that was not SUDS-based. In other words, many of these prior studies used some variation of SUDS score to predict SUDS score in the future. In contrast, the current study used SUDS as a predictor of future outcome that was not also measured using some variation of SUDS. This difference in method adds confidence to the understanding that habituation and variability in the intensity of fear (as well as shame) are important predictors of behavioral and emotional outcomes.

The current study found no effect of habituation or variability in fear on either self-reported public speaking confidence or on speaker's negative thoughts related to how

others perceive him or her, echoing the findings of Culver et al. (2012). The latter study found no effect of either within session habituation or variability in fear on the confidence participants had in their speaking ability or on the fearful thoughts they experienced during public speaking. Thus, it is possible that experienced fear may be a better predictor for some modalities of outcome measurement (such as behavioral performance) than cognitive or emotional measures of anxiety over public speaking.

The gradual attenuation of arousal is a critical aspect of emotion processing theory, while inhibitory learning theory argues that any kind of highly aroused experience can lead to the eventual down-regulation of emotion. On one hand, the findings discussed above are not consistent with the emotion processing theory (Foa & Kozak, 1986) because habituation of fear was unexpectedly found to be hindering, rather than helpful, for the outcome of speech performance; meanwhile, greater variability in the intensity of fear predicted improvement in performance from before to after the experimental task, consistent with the inhibitory learning theory. This improvement may have been due to variability in fear levels reflecting a change in the expectancy of harm, or possibly due to being exposed to a variety of internal fear contexts or fear cues of different intensity, which are thought to strengthen new inhibitory learning and generalize it to different contexts (Craske et al., 2014). On the other hand, much in line with the emotion processing theory (although not directly predicted by it since this theory is only concerned with fear), habituation of shame during the exposure task was found to have beneficial effects on state self-esteem. At the same time greater variability in the intensity of shame proved to be detrimental, contrary to the inhibitory learning theory (with the caveat that, in the same way as emotion processing theory, inhibitory learning theory is

also concerned only with fear rather than shame). Thus, the findings of this study appear to partially support and partially refute both theories.

An inconsistent association between the traditional predictors postulated in emotion processing theory (e.g. within session habituation) and clinical outcomes has been clearly illustrated in prior research (i.e., Craske et al., 2008). Inhibitory learning in general is known to be highly context-dependent (Quirk & Beer, 2006), which may be a key to understanding this inconsistency. If all emotions indeed rely on the same general neural processes of *appraisal* and *regulation* (Etkin et al., 2011), where emotion regulation relies on the inhibitory input (Kanske et al., 2010), and extinction is indeed a special case of emotion regulation (Quirk & Beer, 2006), then the emergence and regulation of other emotions may be just as important in predicting the effects of exposure, serving as a vital internal context that so far has been largely overlooked.

Change in shame is an important predictor of emotional outcomes during exposure

Hypothesis 3 predicted that irrespective of intervention, the experience of shame is more predictive of the change on the behavioral and emotional outcome measures than experience of fear. It appears that the experience of fear and shame are processes that each predict change in outcomes, albeit on different measures of outcomes. Fear and shame were each somewhat predictive of functioning prior to the experimental manipulation, as evidenced by the relationship between social anxiety scores, first speech performance, and the initial activation of both these emotion states during the experimental task. While fear was the strongest predictor of changes in behavioral performance, shame was the strongest predictor of change in emotional well-being.

General theoretical issues

In light of the findings in this study, four issues may need to be addressed by future research. The first issue is that habituation and variability in the emotions of fear and shame were found to be highly correlated with each other. The second is that the terms *habituation* and *extinction* are poorly differentiated in the exposure therapy research literature. Third, the most commonly used measure of fear, the SUDS, may measure more than one construct. Fourth, most of the research has focused so far on studying processes related to fear, when other emotions, such as shame, may be as important. These issues are discussed in the sub-sections that follow.

Habituation and variability of emotion intensity have a complex relationship.

The relationship between within session habituation (i.e., the reduction of arousal from its peak to its last measurement within a given exposure session) and variability (i.e., variance of intensity of arousal within any given exposure session) in fear is complicated, in that they are positively correlated with one another but predict same outcomes (e.g. speech performance) in opposite directions. This finding may shed light on why within session habituation of fear has a troubled history as a predictor of clinical outcomes. *Within session habituation* is a construct that is often measured by a simple reduction from peak SUDS score and final SUDS score of each exposure session (Craske et al., 2008). *Emotional variability* has been measured as a standard deviation of SUDS scores averaged across exposure sessions (Kircanski et al., 2012). These measures are, unsurprisingly, highly correlated, because they are each calculated based on the presenting range of original SUDS scores, even though the constructs they measure remain distinct. Thus, it is possible that past literature studying these measures may have looked at the effects of *within session habituation* without simultaneously considering

emotion variability (and the other way around), and as a result, some instances of habituation likely occurred in the context of low emotion variability, while others were observed in the context of high emotion variability. Because it appears that these constructs predict change in opposite directions, the result of any analysis which does not take into account both variables at the same time may be a diluted, or even null, effect. This represents a statistical suppressor relationship, and in case it extends to other samples, future researchers should be cautioned that the effects of *within session habituation* of fear may be underestimated if the variability in fear intensity is not simultaneously considered. An additional consideration here is that a decrease from highest to last SUDS score, which a usual operational definition of within-session habituation, will automatically produce variability in measurement, thus the correlation. This may be especially true of the present study, since it measured SUDS over a relatively short period of time, which potentially did not give enough time for individuals' emotional experience to fluctuate substantially (although such fluctuation was observed, see Appendix M).

Physiological findings in this study furthermore suggest that different physiological processes underlie habituation and variability in the intensity of emotion. It appears that habituation in both fear and shame are correlated with inhibitory activity by the prefrontal areas, while variability in the intensity of these states are negatively correlated with it and may represent the influence of important contextual factors.

Habituation and extinction are poorly differentiated.

In actual practice, it is difficult to distinguish between habituation and extinction. In their article, Culver et al. (2012) say that they use the term *habituation* to stay consistent

with the literature terminology, while clarifying that a more accurate term is *extinction*. While this study follows the same convention, the word *habituation* here refers to an observation of attenuation of reported fear (or shame), rather than an underlying mechanism of change. The reason for this is that while *habituation* and *extinction* are theoretically distinct, distinguishing them by observing behavior or asking for self-report is probably impossible. *Habituation* is a non-associative form of learning that results in a decrease in response due to the repeated presentation of a stimulus, which is not the result of sensory adaptation or fatigue (Rankin et al., 2009). In contrast, *extinction* is an associative form of learning that is defined as a loss of responding to a fear cue when it is no longer followed by an aversive stimulus, because the cue loses its informative value and effectively becomes a meaningless signal (Bouton, 2002). Habituation has been proposed to be at least partially responsible for the extinction of learned fear (McSweeney & Swindell, 2002; Myers & Davis, 2007). While theoretically distinct, in practice the lines separating the two processes are blurry. The processes share multiple overlapping characteristics, such as spontaneous return of fear seen in the presence of a previously habituated or extinguished stimulus, or the negative exponential function shown by participants' responses during either habituation or extinction (McSweeney & Swindell, 2002). Neural mechanisms of extinction have recently received some attention (and overlap to a significant degree with neural mechanisms of emotion regulation; Quirk & Beer, 2006), but neural mechanisms behind habituation less so, and although it appears that both processes involve N-methyl-d-aspartate (NMDA) receptors, this area requires more research (Storsve, McNally, & Richardson, 2010). These points highlight the

practical problem in deciding whether the observed process that is used to predict outcomes is habituation, extinction, or perhaps even both.

SUDS may measure multiple constructs.

Another point is that while SUDS (i.e, units of “distress”) is purportedly a measure of “fear,” part of the inconsistency in how well it predicts outcomes may be due to different participants reporting different experiences by stating their SUDS score. LeDoux (2013) distinguishes between a conscious emotion of fear, which involves top-down processes and cognitive appraisal, and what he calls a *defensive organismic state* characterized by the stimulus-elicited behavioral responses (such as fighting, fleeing, or freezing) and physiological changes due to the activity of the autonomic nervous system, such as increase in heart rate and skin conductance, in response to a tacitly anticipated threat that is processed in part through the amygdala circuits. A close equivalent to *defensive organismic state* would be *global distress* in emotion focused therapy (not to be confused with emotion processing theory). While SUDS is a tried and true measure, in its current form it does not distinguish between a state of arousal with little cognitive involvement and a state of arousal where substantial cognitive processing of emotional material is taking place. In other words, qualitative and meaning-laden differences between distress in “high cognition” vs. “low cognition” are not differentiated using a single rating. One possibility to address this issue is to use widely available, affordable and convenient sport heart rate monitors to supplement SUDS measurement during exposure. For example, an individual who gives a high SUDS rating but whose heart rate variability is high or moderate may be engaged in more cognitive processing of “raw neural materials” (as LeDoux, 2013, calls it) compared to an individual who gives a high

SUDS rating and experiences low heart rate variability. This theoretical and somewhat speculative possibility may be worth investigating in further studies.

Variability in levels of fear may indicate that other emotional experiences can become salient in individual's awareness, which would imply that more advanced emotion processing occurs, in the sense used by LeDoux (2013). If we take *fear* to mean *defensive organismic state* or *global distress*, then any emotion, even shame, is more advanced in its specificity of meaning. As discussed earlier, due to the dynamic nature of emotional experience dominant and sub-dominant emotions may coexist, and as the intensity of one emotion fades, another one may become more prominent in the immediate experience (Pascual-Leone, Paivio, & Harrington, 2016). If there is variability in *global distress*, there may be access to other emotions in the awareness, which is a possibility that can be investigated empirically in the future studies.

Emotional processes of shame, not just fear, can be important predictors of outcomes.

This study found that emotional processes (i.e. habituation and variability) related to shame emerged as important predictors on par with emotional processes related to fear, albeit in relation to different kinds of outcomes. While processes related to fear predicted behavioral outcomes (i.e. quality of speech performance), those related to shame predicted emotional ones (i.e. state self-esteem). This finding echoes somewhat a suggestion by Catherall (2003) and LeDoux (2013) that systems of different modalities (i.e. nonconscious vs. conscious) may be affected by different interventions, or perhaps, in the current case, different processes.

An additional possibility is that that certain emotions may hold special importance in relation to exposure treatment of certain disorders. Just as shame has been considered an important process in relation to social anxiety (Shahar, 2013), Kircanski et al. (2012) cite studies which state that in contamination-related obsessive compulsive disorder a feeling of disgust characterizes the condition as much as fear. Further research should examine this possibility.

Methodological implications

Two important methodological implications emerged from this research. The first one is the issue of multimodal measurement. In this study, emotional processes related to fear (i.e. its habituation and its variability), as suggested by both emotion processing and inhibitory learning theories, were found to be predictive of change on behavioral, but not emotional self-report measures. Meanwhile, indices related to shame were found to be predictive of change on emotional but not behavioral measures. The second methodological implication is related to choice of analytical strategy when examining these process variables. Given the possibility of statistical suppression between habituation and emotion variability in other samples, future studies looking to test emotion processing theory against inhibitory learning theory should follow the advice of Paulhus and colleagues (2004), which is to avoid using exploratory analysis strategies such as forward stepwise regression, because they are almost certain to overlook suppression. Thus, future work should take care to evaluate both habituation and variability in the intensity of emotion as predictors simultaneously as a precaution.

Clinical implications

One of the main clinical implications of the current study is a possibility that a reduction in subjective sense of distress or fear is not always a desirable or effective target during exposure treatment, while a focus on increasing the variability in intensity of these states is. These findings are mostly in line with the inhibitory learning theory (e.g. Craske et al., 2014), but also appear to mostly have implications in regards to behavioral outcomes while having little effect on emotional ones. At the same time, the focus on diminishing a subjective sense of shame may present itself as a desirable target in relation to improving the emotional outcomes of treatment. This finding is much in line with theory based on emotion focused therapy (Shahar, 2013) which treats shame as a core primary process maintaining social anxiety, while relegating fear to a secondary place. Of course, these findings may extend only as far as performance in social situations in individuals with social anxiety traits is concerned.

Limitations and Future Directions

The study has a number of limitations. The study utilized an analog design, so generalizing these findings to a clinical intervention should be done with caution. The second one has to do with recruitment and retention of participants (recall Appendix E). A high number of individuals ($n = 161$) screened online and meeting all study criteria showed initial interest in attending the current study on-site, but only 45 individuals actually followed through. In addition to that, 4 individuals who attended chose to terminate their participation in the study early. With respect to participant recruitment, it is difficult to explain why they chose to not further pursue the on-site part of the study. One possibility is that, given fear of evaluation which defines individuals with Social Anxiety Disorder (whether clinical or subclinical), some participants may have decided

that the study would be too stressful for them. On the other hand, an alternative explanation is that because most of the individuals recruited through the Psychology Participant Pool take part in studies because it is a course requirement, they could have chosen to obtain a quick credit by finishing the online part of the study without having the intention to follow through. That, coupled with availability of other online studies through the Participant Pool may explain low follow-through rate of the participants in the current study.

With regards to the dropout rate a dropout of almost 9% ($n = 4$) appears to be fairly high. However, the rate of the current study is still lower than Kircanski et al.'s (2012) study, which investigated exposure to contamination fears and had a dropout of 24% ($n = 12$). While the dropout rate in the current study may reflect the inherent challenge of asking individuals with fear of public speaking facing such speaking in some form, it may also reflect a success of ongoing informed consent process, which was carried out in this study with special rigor. The study recruited university students whose social anxiety was less severe than that typically observed in Social Anxiety Disorder, although 31.7% of the sample met the diagnostic criteria based on a semi-structured interview conducted at the beginning of the study. Nonetheless, while recruitment did target individuals with more severe social anxiety, it may be that potential participants may have avoided being involved in the study on account of fear of being in a situation where they can be negatively judged by other individuals (e.g., the experimenter), which is the hallmark of social anxiety. Thus, it is likely that the effect size for this study was small and limited the study's statistical power because individuals with more severe symptoms may have benefited more from the experimental task. Nevertheless, it is also possible that this study

may be studying a more universal process of *emotion regulation*, where the difference between a clinical and a non-clinical sample may be one of degree, rather than that of a kind.

Another unanticipated limitation in design was that while the instructions for the experiential group were specific, not all individuals in that condition followed them. For example, when one participant was asked about what emotions she experienced while watching herself give a speech on the video, she persisted (despite attempts at redirection) in describing her physical appearance and imperfections instead. Other individuals reported their thoughts rather than emotions, and so on. Thus, while the instructions were standard for all participants within any given group (similar to instructions in the study by Nilsson and colleagues, 2012), participants showed a wide range of idiosyncratic responses, some of which were not adherent to the intention of the assigned condition. If there is a strong effect of emotion verbalization on exposure, it was almost certainly diluted in these ways.

Related to that, another limitation of the study is that the participants in the control group experienced the same amount of anxiety during their “control” task as the participants did in the two other conditions. In other words, despite being told that they will not be evaluated on the control task, participants in that condition were as stressed solving timed word puzzles as were participants that were asked to watch themselves multiple times on video. Finally, while the findings of this study shed some light on how verbalization of emotion and changes in fear and shame affect outcomes related to social anxiety, the findings may be limited to short-term changes only because follow-up

measurement was beyond the current research focus and thus the study design did not incorporate it.

Finally, in regards to physiological recording, heart rate variability was calculated from a sports-grade equipment in recording intervals which were shorter than the 5-minute intervals suggested by the measurement guidelines (Task Force of The European Society of Cardiology and The North American Society of Pacing and Electrophysiology, 1996). Although the guidelines make provisions for shorter recording intervals, the current physiological recording can be judged as likely not being very accurate.

Further research should consider three main issues. The first one is that while it appears that verbalization of emotion may influence exposure (e.g., Kircanski, Lieberman, & Craske, 2012), which may be a special case of emotion regulation (Quirk & Beer, 2006), the current study found mixed evidence to support this notion, primarily because the participants did not adhere closely to the experimental instructions. Further studies should explore this topic in more depth.

The second issue is that given the complex relationship between habituation and variability in the intensity of emotional states, further research should follow Kazdin's (2009) suggestion on the study of mechanisms of change: that these variables should be studied in parallel, competing mediation analyses should be considered to further elucidate the relationship these predictors have with each other and various outcomes.

The third issue is that further research should try to incorporate physiological and neuroimaging data when it is possible or practical. While average heart rate was considered in previous studies (e.g. Culver et al., 2012), it yields relatively little information when compared to time-domain (e.g. SDNN, or the standard deviation of NN

intervals) and frequency-domain (e.g. LF/HF ratio, or the ratio of low frequency to high frequency components of heart rate signal) measures of heart rate variability, which are regarded as indices closely related to emotion regulation (Appelhans & Leucke, 2006). This physiological link, ideally recorded with ECG-accurate equipment, can connect exposure research to the broader study of emotion regulation.

Conclusions

Both habituation and variability in fear and shame emerged as significant predictors of change on behavioral and emotional measures in the course of an exposure task, supporting both inhibitory learning theory as well as indirectly supporting the emotion processing theory. Due to the apparent complex association between these predictors their effects during exposure seem to be dependent on each other and need to be studied further. It appears that both emotion processing theory and inhibitory learning theory may be correct to a degree. The current study suggests that habituation may be an index of inhibitory learning (or at the very least an index of inhibitory activity), while variability in the intensity of emotion may represent the influence of important contextual variables. Thus, these competing theories may be reconciled with future research which takes both predictors into account and investigates their physiological underpinnings.

References

- Acarturk, C., Cuijpers, P., van Straten, A., & de Graaf, R. (2009). Psychological treatment of social anxiety disorder: A meta-analysis. *Psychological Medicine, 39*(2), 241-254.
- Antony, M. M., & Swinson, R. P. (2002). *Phobic disorders and panic in adults: A guide to assessment and treatment*. Washington: American Psychological Association.
- Appelhans, B. M., & Luecken, L. J. (2006). Heart rate variability as an index of regulated emotional responding. *Review of General Psychology, 10*(3), 229-240.
- Bagozzi, R. P., & Heatherton, T. F. (1994). A general approach to representing multifaceted personality constructs: Application to self-esteem. *Structural Equation Modelling, 1*, 35-67.
- Baker, A., Mystkowski, J., Culver, N., Yi, R., Mortazavi, A., & Craske, M. G. (2010). Does habituation matter? Emotional processing theory and exposure therapy for acrophobia. *Behaviour Research and Therapy, 48*, 1139-1143.
- Beidel, D. C., Turner, S. M., Jacob, R. G., & Cooley, M. R. (1989). Assessment of social phobia: *Reliability of an impromptu speech task*. *Journal of Anxiety Disorders, 3*(3), 149-158.
- Bennaroch, E. E. (1993). The central autonomic network: Functional organization, dysfunction, and perspective. *Mayo Clinic Proceedings, 68*, 988-1001.
- Bermond, B., Clayton, K., Liberova, A., Luminet, O., Maruszewski, T., Bitti, P. E. R., Rimé, B., Vorst, H. H., Wagner, H., & Wicherts, J. (2007). A cognitive and an affective dimension of alexithymia in six languages and seven populations. *Cognition and Emotion, 21*(5), 1125-1136.

- Bouton, M. E. (2002). Context, ambiguity, and unlearning: Sources of relapse after behavioral extinction. *Biological Psychiatry*, *52*, 976-986.
- Carleton, R. N., Collimore, K. C., & Asmundson, G. J. G. (2007). Social anxiety and fear of negative evaluation: construct validity of the BFNE-II. *Journal of Anxiety Disorders*, *21*, 131–141.
- Carleton, R. N., Collimore, K. C., McCabe, R. E., & Antony, M. M. (2011). Addressing revisions to the Brief Fear of Negative Evaluation scale: Measuring fear of negative evaluation across anxiety and mood disorders. *Journal of Anxiety Disorders*, *25*(6), 822-828.
- Carleton, R. N., McCreary, D. R., Norton, P. J., & Asmundson, G. J. G. (2006). Brief fear of negative evaluation scale-revised. *Depression and Anxiety*, *23*, 297–303.
- Catherall, D. R. (2003). How fear differs from anxiety. *Traumatology*, *9*(2), 76-92.
- Clark, P. (2013). *The effects of multicollinearity in multilevel models* (Unpublished doctoral dissertation). Wright State University, Fairborn, Ohio.
- Cohen, J., Cohen, P., West, S. G., & Aiken, L. S. (2003). *Applied multiple regression/correlation analysis for the behavioral sciences* (3rd ed.). Mahwah, NJ: Lawrence Erlbaum Associates, Inc.
- Craske, M. G., Kircanski, K., Zelikowsky, M., Mystkowski, J., Chowdhury, N., & Baker, A. (2008). Optimizing inhibitory learning during exposure therapy. *Behaviour Research and Therapy*, *46*, 5-27.
- Craske, M. G., Treanor, M., Conway, C. C., Zbozinek, T., & Vervliet, B. (2014). Maximizing exposure therapy: An inhibitory learning approach. *Behaviour Research and Therapy*, *58*, 10-23.

- Culver, N. C., Stoyanova, M., & Craske, M. G. (2012). Emotional variability and sustained arousal during exposure. *Journal of Behavior Therapy and Experimental Psychiatry, 43*, 787-793.
- Deacon, B. J., & Abramowitz, J. S. (2004). Cognitive and behavioral treatments for anxiety disorders: A review of meta-analytic findings. *Journal of Clinical Psychology, 60*(4), 429-441.
- Devinsky, O., Morrell, M. J., & Vogt, B. A. (1995). Contributions of anterior cingulate cortex to behavior. *Brain, 118*, 279-306.
- Etkin, A., Egner, T., & Kalisch, R. (2011). Emotional processing in anterior cingulate and medial prefrontal cortex. *Trends in Cognitive Sciences, 15*(2), 85-93.
- Farrell, N. R., Deacon, B. J., Dixon, L. J., & Lickel, J. J. (2013). Theory-based training strategies for modifying practitioner concerns about exposure therapy. *Journal of Anxiety Disorders, 27*, 781-787.
- Fergus, T. A., Valentiner, D. P., McGrath, P. B., & Jencius, S. (2010). Shame- and guilt proneness: Relationships with anxiety disorder symptoms in a clinical sample. *Journal of Anxiety Disorders, 24*, 811–815.
- Feske, U., & Chambless, D. L. (1995). Cognitive behavioral versus exposure only treatment for social phobia: A meta-analysis. *Behavior Therapy, 26*, 695-720.
- Foa, E. B., & Kozak, M. J. (1986). Emotional processing of fear: Exposure to corrective information. *Psychological Bulletin, 99*(1), 20-35.
- Foa, E. B., Huppert, J. D., & Cahill, S. P. (2006). Emotional processing theory: An update. In B. O. Rothbaum (Ed.), *Pathological anxiety: Emotional processing in etiology and treatment* (3–24). New York: Guilford Press.

- Foa, E. B., Zoellner, L. A., Feeny, N. C., Hembree, E. A., & Alvarez-Conrad, J. (2002). Does imaginal exposure exacerbate PTSD symptoms? *Journal of Consulting and Clinical Psychology, 70*(4), 1022-1028.
- Fujimura, T., & Okanoya, K. (2012). Heart rate variability predicts emotional flexibility in response to positive stimuli. *Psychology, 3*(8), 578-582.
- Fydrich, T., Chambless, D. L., Perry, K. J., Buergener, F., & Beazley, M. B. (1998). Behavioral assessment of social performance: A rating system for social phobia. *Behaviour Research and Therapy, 36*, 995-1010.
- Gould, R. A., Buckminster, S., Pollack, M. H., Otto, M. W., & Yap, L. (1997). Cognitive-behavioral and pharmacological treatment for social phobia: A meta-analysis. *Clinical Psychology: Science and Practice, 4*(4), 291-306.
- Harb, G. C., Eng, W., Zaider, T., & Heimberg, R. G. (2003). Behavioral assessment of public-speaking anxiety using a modified version of the Social Performance Rating Scale. *Behaviour Research and Therapy, 41*, 1373-1380.
- Hart, T. A., Jack, M. S., Turk, C. L., & Heimberg, R. G. (1999). Issues for the measurement of social phobia. In H. G. M. Westenberg and J. A. den Boer (Eds.), *Social phobia: Recent trends and progress* (133-155). Amsterdam: Syn-Thesis Publishers.
- Hayes, A. M., Beck, J. G., & Yasinski, C. (2012). A cognitive behavioral perspective on corrective experiences. In L. G. Castonguay and C. E. Hill (Eds.), *Transformation in psychotherapy: Corrective experiences across cognitive behavioral, humanistic, and psychodynamic approaches* (85-102). Washington: American Psychological Association.

- Heatherton, T. F., & Polivy, J. (1991). Development and validation of a scale for measuring state self-esteem. *Journal of Personality and Social Psychology*, 60(6), 895-910.
- Heatherton, T. F., & Wyland, C. L. (2003). Assessing self-esteem. In S. J. Lopez & C. R. Snyder (Eds.), *Positive psychological assessment: A handbook of models and measures* (219-233). Washington: American Psychological Association.
- Hedman, E., Ström, P., Stükel, A., & Mörtberg, E. (2013). Shame and guilt in social anxiety disorder: Effects of cognitive behavior therapy and association with social anxiety and depressive symptoms. *PLoS ONE*, 8(4), e61713.
doi:10.1371/journal.pone.0061713
- Heimberg, R. G., Dodge, C. S., Hope, D. A., Kennedy, C. R., Zollo, L., & Becker, R. E. (1990). Cognitive behavioral group treatment of social phobia: Comparison to a credible placebo control. *Cognitive Therapy and Research*, 14, 1–23.
- Hofmann, S. G. (2008). Cognitive processes during fear acquisition and extinction in animals and humans: Implications for exposure therapy of anxiety disorders. *Clinical Psychology Review*, 28, 199-210.
- Hox, J. J. (2010). *Multilevel analysis: Techniques and applications* (2nd ed.) New York: Routledge.
- Jayawickreme, N., Cahill, S. P., Riggs, D. S., Rauch, S. A. M., Resick, P. A., Rothbaum, B. O., & Foa, E. B. (2014). Primum non nocere (first do no harm): Symptom worsening and improvement in female assault victims after prolonged exposure for PTSD. *Depression and Anxiety*, 31, 412-419.

- Kanske, P., Heissler, J., Schönfelder, S., Bongers, A., & Wessa, M. (2011). How to regulate emotion? Neural networks for reappraisal and distraction. *Cerebral Cortex*, *21*(6), 1379-1388.
- Kazdin, A. E. (2009). Understanding how and why psychotherapy leads to change. *Psychotherapy Research*, *19*, 418-428.
- Kemp, A. H., & Quintana, D. S. (2013). The relationship between mental and physical health: Insights from the study of heart rate variability. *International Journal of Psychophysiology*, *89*, 288-296.
- Kircanski, K., Lieberman, M. D., & Craske, M. G. (2012). Feelings into words: Contributions of language to exposure therapy. *Psychological Science*, *23*(10), 1086-1091.
- Kircanski, K., Mortazavi, A., Castriotta, N., Baker, A. S., Mystkowski, J. L., Yi, R., & Craske, M. G. (2012). Challenges to the traditional exposure paradigm: Variability in exposure therapy for contamination fears. *Journal of Behavior Therapy and Experimental Psychiatry*, *43*, 745-751.
- Landers, R.N. (2015). Computing intraclass correlations (ICC) as estimates of interrater reliability in SPSS. *The Winnower* 2:e143518.81744. DOI: 10.15200/winn.143518.81744
- Leary, M. R. (1983). A brief version of the Fear of Negative Evaluation Scale. *Personality and Social Psychology Bulletin*, *9*(3), 371-375.
- LeDoux, J. E. (2013). Coming to terms with fear. *Proceedings of the National Academy of Sciences of the United States of America*, *111*(8), 2871-2878.

- Lewis, H. B. (1971). Shame and guilt in neurosis. *Psychoanalytic Review*, 58(3), 419-438.
- Lieberman, M. D., Eisenberger, N. I., Crockett, M. J., Tom, S. M., Pfeifer, J. H., & Way, B. M. (2007). Putting feelings into words: Affect labeling disrupts amygdala activity in response to affective stimuli. *Psychological Science*, 18(5), 421-428.
- Marschall, D. E., Sanftner, J. L., & Tangney, J. P. (1994). *The State Shame and Guilt Scale (SSGS)*. Fairfax, VA: George Mason University.
- Mattick R. P., & Clarke, J.C. (1998). Development and validation of measures of social phobia scrutiny fear and social interaction anxiety. *Behaviour Research and Therapy*, 36, 455-470.
- McRae, K., Misra, S., Prasad, A. K., Pereira, S. C., & Gross, J. J. (2012). Bottom-up and top-down emotion generation: Implications for emotion regulation. *Social Cognitive and Affective Neuroscience*, 7, 253-262.
- McSweeney, F., & Swindell, S. (2002). Common processes may contribute to extinction and habituation. *Journal of General Psychology*, 129(4), 364-400.
- Meuret, A. E., Seidel, A., Rosenfield, B., Hofmann, S. G., & Rosenfield, D. (2012). Does fear reactivity during exposure predict panic symptom reduction? *Journal of Consulting and Clinical Psychology*, 80(5), 773-785.
- Moher D., Liberati A., Tetzlaff J., Altman, D.G., The PRISMA Group (2009). Preferred Reporting Items for Systematic Reviews and Meta-Analyses: The PRISMA Statement. *PLoS Med* 6(6): e1000097. doi:10.1371/journal.pmed1000097
- Myers, K. M., & Davis, M. (2007). Mechanisms of fear extinction. *Molecular Psychiatry*, 12, 120-150.

- Nilsson, J-E., Lundh, L-G., & Viborg, G. (2012). Effects of analytical and experiential self-focus on rumination after a stress induction in patients with social anxiety disorder: A pilot study. *Cognitive Behaviour Therapy*, *41*(4), 310-320.
- Ochsner, K. N., Ray, R. R., Hughes, B., McRae, K., Cooper, J. C., Weber, J., Gabrieli J. D. E., & Gross, J. J. (2009). Bottom-up and top-down processes in emotion generation: Common and distinct neural mechanisms. *Psychological Science*, *20*(11), 1322-1331.
- Olatunji, B. O., Deacon, B. J., & Abramowitz, J. S. (2009). The cruelest cure? Ethical issues in the implementation of exposure-based treatments. *Cognitive and Behavioral Practice*, *16*, 172-180.
- Pascual-Leone, A. (2009). Dynamic emotional processing in experiential therapy: Two steps forward, one step back. *Journal of Consulting and Clinical Psychology*, *77*(1), 113-126.
- Pascual-Leone, A., & Greenberg, L. S. (2007). Emotional processing in experiential therapy: Why "the only way out is through." *Journal of Consulting and Clinical Psychology*, *75*(6), 875-887.
- Pascual-Leone, A., Paivio, S., & Harrington, S. (2016). Emotion in psychotherapy: An experiential-humanistic perspective. In D. Cain, S. Rubin, K. Keenan (Eds.) *Humanistic Psychotherapies: Handbook of Research and Practice (Second edition)*. (pp. 147-181). Washington, DC, US: American Psychological Association.
- Paul, G. L. (1966). *Insight vs desensitization in psychotherapy*. Stanford, CA: Stanford University Press.

- Paulhus, D. L., Robins, R. W., Trzesniewski, K. H., & Tracy, J. L. (2004). Two replicable suppressor situations in personality research. *Multivariate Behavioral Research, 39*(2), 303-328.
- Peters, L., Sunderland, M., Andrews, G., Rapee, R. M., & Mattick, R. P. (2012). Development of a short form Social Interaction Anxiety (SIAS) and Social Phobia Scale (SPS) using nonparametric item response theory: The SIAS-6 and the SPS-6. *Psychological Assessment, 24*, 66–76. doi:10.1037/a0024544
- Phelps, E. A. (2005). The interaction of emotion and cognition: Insights from studies of human amygdala. In L. F. Barrett, P. M. Niedenthal, & P. Winkielman (Eds.), *Emotion and Consciousness* (51-66). New York, NY: Guilford Press.
- Philippot, P., Vrielynck, N., & Muller, V. (2010). Cognitive processing specificity of anxious apprehension: Impact on distress and performance during speech exposure. *Behavior Therapy, 41*, 575-586.
- Pollatos, O., & Gramann, K. (2012). Attenuated modulation of brain activity accompanies emotion regulation deficits in alexithymia. *Psychophysiology, 49*(5), 651-658.
- Quirk, G. J., & Beer, J. S. (2006). Prefrontal involvement in the regulation of emotion: Convergence of rat and human studies. *Current Opinion in Neurobiology, 16*, 723-727.
- Rankin, C.H., Abrams, T., Barry, R. J., Bhatnagar, S., Clayton, D. F., Colombo, J., Coppola, G., Geyer, M. A., Glanzman, D. L., Marsland, S., McSweeney, F. K., Wilson, D. A., Wu, C., & Thompson, R. F. (2009). Habituation revisited: An updated

- and revised description of the behavioral characteristics of habituation. *Neurobiology of Learning and Memory*, 92, 135-138.
- Rapee, R. M., & Heimberg, R. G. (1997). A cognitive-behavioral model of anxiety in social phobia. *Behaviour Research and Therapy*, 35(8), 741-756.
- Rodebaugh, T. L., Woods, C. M., Thissen, D. M., Heimberg, R. G., Chambless, D. L., & Rapee, R. M. (2004). More information from fewer questions: the factor structure and item properties of the original and brief fear of negative evaluation scale. *Psychological Assessment*, 16, 169–181.
- Rose, P., & Segrist, D. J. (2012). Difficulty identifying feelings, distress tolerance and compulsive buying: Analyzing the associations to inform therapeutic strategies. *International Journal of Mental Health and Addiction*, 10, 927-935.
- Schiller, D., & Phelps, E. A. (2011). Does reconsolidation occur in humans? *Frontiers in Behavioral Neuroscience*, 5(24), 1-12.
- Shahar, B. (2013). Emotion-focused therapy for the treatment of social anxiety: An overview of the model and a case description. *Clinical Psychology and Psychotherapy*. Online publication. <http://dx.doi.org/10.1002/cpp.1853>
- SSI Central. (n.d.). *Standardized coefficients*. Retrieved from http://www.ssicentral.com/hlm/help7/faq/FAQ_Standardized_coefficients.pdf
- Storsve, A. B., McNally, G. P., & Richardson, R. (2010). US habituation, like CS extinction, produces a decrement in conditioned fear responding that is NMDA dependent and subject to renewal and reinstatement. *Neurobiology of Learning and Memory*, 93, 463-471.

- Tanner, B. A. (2012). Validity of global physical and emotional SUDS. *Applied Psychophysiological Feedback, 37*, 31-34.
- Task Force of The European Society of Cardiology and The North American Society of Pacing and Electrophysiology. (1996). Heart rate variability: standards of measurement, physiological interpretation and clinical use. *European Heart Journal, 17*, 354-381.
- Thayer, J. F., & Brosschot, J. F. (2005). Psychosomatics and psychopathology: Looking up and down from the brain. *Psychoneuroendocrinology, 30*, 1050-1058.
- Thayer, J. F., & Lane, R. D. (2000). A model of neurovisceral integration in emotion regulation and dysregulation. *Journal of Affective Disorders, 61*, 201-216.
- Thayer, J. F., & Lane, R. D. (2009). Claude Bernard and the heart-brain connection: Further elaboration of a model of neurovisceral integration. *Neuroscience and Biobehavioral Reviews, 33*, 81-88.
- Tsao, J. C. I., & Craske, M. G. (2000). Timing of treatment and return of fear: Effects of massed, uniform-, and expanding-spaced exposure schedules. *Behavior Therapy, 31*, 479-497.
- Turner, S. M., Beidel, D. C., Cooley M. R., Woody, S. R. and Messer, S. C. (1994). A multicomponent behavioral treatment for social phobia: Social Effectiveness Therapy. *Behaviour Research and Therapy, 32*, 381-390.
- Voss, D. S. (2004). *Multicollinearity*. Retrieved from <http://www.uky.edu/~dsvoss/docs/multic.pdf>
- Watson, D., & Friend, R. (1969). Measurement of social-evaluative anxiety. *Journal of Consulting and Clinical Psychology, 33*, 448-457.

- Weeks, J. W., Heimberg, R. G., Fresco, D. M., Hart, T. A., Turk, C. L., Schneier, F. R., et al. (2005). Empirical validation and psychometric evaluation of the brief fear of negative evaluation scale in patients with social anxiety disorder. *Psychological Assessment, 17*, 179–190.
- Wendt, J., Schmidt, L. E., Lotze, M., & Hamm, A. O. (2012). Mechanisms of change: Effects of repetitive exposure to feared stimuli on the brain's fear network. *Psychophysiology, 49*(10), 1319-1329.
- Williams, D. P., Cash, C., Rankin, C., Bernardi, A., Koenig, J., & Thayer, J. F. (2015). Resting heart rate variability predicts self-reported difficulties in emotion regulation: A focus on different facets of emotion regulation. *Frontiers in Psychology, 6*, 1-8. doi: 10.3389/fpsyg.2015.00261
- Wolitzky-Taylor, K. B., Viar-Paxton, M. A., & Olatunji, B. O. (2012). Ethical issues when considering exposure. In T. E. Davis III, T. H. Ollendick, & L-S. Öst (Eds.), *Intensive one-session treatment of specific phobias. Autism and child psychopathology series* (195-208). New York, NY: Springer Science + Business Media.
- Wolpe, J. (1958). *Psychotherapy by reciprocal inhibition*. Stanford, CA: Stanford University Press.

Appendix A

Specific verbal instructions for the study

ALL GROUPS

VIDEO INSTRUCTION PRE

After you fill out some questionnaires, I will ask you to pick a topic, anything you want, and give a short speech. You will have 3 minutes to prepare the speech, and 3 more minutes to deliver it. If you run out of things to say and need to change topics, you can do so, just try to keep talking for three minutes. This video camera will be recording the whole time. Later your video will be evaluated by two expert judges. If you get too distressed to continue the study, you can stop at any time by saying "stop" and we will stop. Remember, you can't take any written notes.

CONTROL GROUP

INSTRUCTION A

Now, there will be a few tasks for you to accomplish. In the next three minutes, solve as many word puzzles as you can. In these word puzzles the letters are mixed up. Just write down what word the letters really make next to the jumbled letters. You will not be graded on this task.

INSTRUCTION B

Let's do a second sheet of word puzzles. Like before, you will have 3 minutes, and you will not be graded.

INSTRUCTION C

Let's do the last sheet of word puzzles. The instructions are the same as before.

EXPOSURE GROUP

INSTRUCTION A

We will watch the recording multiple times. As you watch, try to notice when you feel self-conscious, embarrassed, or on the spot. Notice what feelings you have in your body, any urges to do anything or act in some way, or emotions that you feel. Remember that this is not a test. Just stay engaged with watching the video.

INSTRUCTION B

Let's watch the video again. As before, notice when you feel self-conscious, embarrassed, or on the spot. Notice what feelings you have in your body, any urges to do anything or act in some way, or emotions that you feel.

INSTRUCTION C

Let's watch it a final time. As before, notice when you feel self-conscious, embarrassed, or on the spot. Notice what feelings you have in your body, any urges to do anything or act in some way, or emotions that you feel.

EXPERIENTIAL GROUP

INSTRUCTION A

We will watch the recording multiple times. As you watch, try to notice when you feel self-conscious, embarrassed, or on the spot. Notice what feelings you have in your body, any urges to do anything or act in some way, or emotions that you feel. Remember that this is not a test. Just stay engaged with watching the video.

TASK A

Now I want to ask you some questions. These questions are not a test. I want us both to better understand what it was like for you watching yourself on the video.

- Describe what it felt like in your body while you were watching?
- Tell me about any thoughts or images that came into your mind while watching?
- Describe any feelings or emotions you had while watching?
- Tell me about anything else that you've noticed I haven't asked about?

INSTRUCTION B

Let's watch the video again. As before, notice when you feel self-conscious, embarrassed, or on the spot. Notice what feelings you have in your body, any urges to do anything or act in some way, or emotions that you feel.

TASK B

Let's see what has been going on for you this time.

- What feelings did you have while watching this time?
- See if you can put more words to these feelings?

- I wonder if these feelings tell you something about what you want, need, or deserve?

INSTRUCTION C

Let's watch it a final time. As before, notice when you feel self-conscious, embarrassed, or on the spot. Notice what feelings you have in your body, any urges to do anything or act in some way, or emotions that you feel.

TASK C

- What feelings came up this time?
- What has changed for you over the different times you watched the video?
- Of all the things you noticed during these exercises, what stands out to you as the most important?

ALL GROUPS

VIDEO INSTRUCTION POST

After you fill out another set of questionnaires, I will ask you to pick a different topic of your choosing and give another, different, speech. Like before, you will have 3 minutes to prepare and 3 minutes to deliver the speech. If you run out of things to say and need to change topics, you can do so, just try to keep talking for three minutes. If you find yourself too distressed to continue the study, you can stop at any time by saying "stop". Later on this speech will be evaluated by two expert judges as well. Remember, you can't take any written notes.

Appendix B

Correlation between process and outcome measures

Measure	SUDS initial activation	SUDS habituation	SUDS variability	SSGS initial activation	SSGS habituation	SSGS variability
SIAS/SPS Pre	.588**	.190	.157	.445**	.400**	.430**
SIAS/SPS Post	.575**	.159	.114	.534**	.424**	.493**
SPRS-M Pre	-.436**	-.141	-.181	-.378*	-.235	-.215
SPRS-M Post	-.324*	-.206	-.170	-.463**	-.330*	-.248
BFNE-S Pre	.464**	.084	.081	.290	.128	.259
BFNE-S Post	.528**	.157	.145	.490**	.268	.452**
BPRCS Pre	-.523**	-.271	-.307	-.543**	-.222	-.517**
BPRCS Post	-.625**	-.280	-.282	-.497**	-.247	-.426**
SSES Pre	-.517**	.012	.023	-.531**	-.369*	-.444**
SSES Post	-.463**	.048	.036	-.569**	-.291	-.515**

Note. SUDS = Subjective Units of Distress Scale; SSGS = State Shame and Guilt Scale; SIAS/SPS = Social Interaction Anxiety Scale-6/Social Phobia Scale-6 combined; SPRS-M = Social Performance Rating Scale-Modified; BFNE-S = Brief Fear of Negative Evaluation – Straightforward items; BPRCS = Brief Personal Record of Confidence as a Speaker; SSES = State Self-Esteem Scale. ** $p < .01$; * $p < .05$.

Appendix C

Correlations between process measures

	Average LF/HF ratio	SSGS initial activation	SSGS habituation	SSGS variability	SUDS initial activation	SUDS habituation	SUDS variability
Average HF component	-.854**	-.031	.346*	-.023	.065	.433**	.316
Average LF/HF ratio		-.066	-.394*	-.046	-.037	-.287	-.207
SSGS initial activation			.594**	.779**	.521**	.076	.063
SSGS habituation				.756**	.251	.244	.112
SSGS variability					.372*	.088	.081
SUDS initial activation						.403**	.377*
SUDS habituation							.932**

Note. SUDS = Subjective Units of Distress Scale; SSGS = State Shame and Guilt Scale. ** $p < .01$; * $p < .05$.

Appendix D

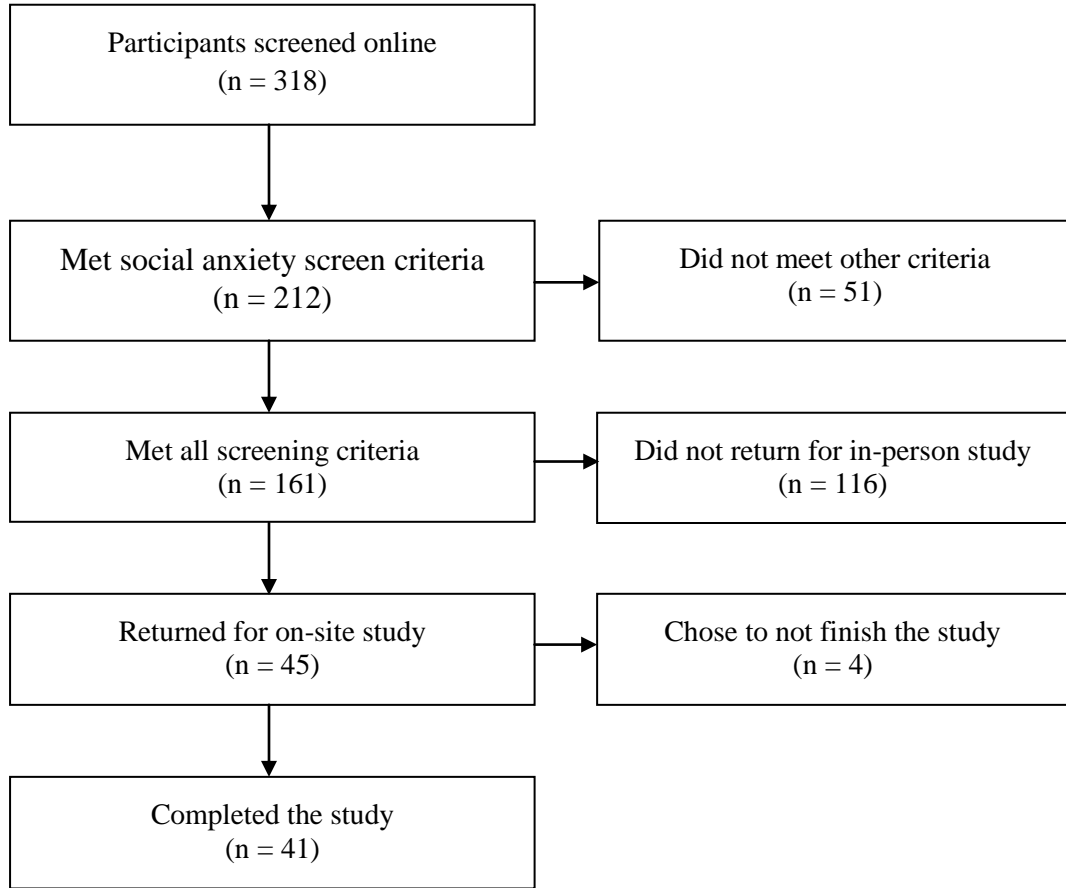
Correlations between outcome variables

	SIAS /SPS Pre	SIAS /SPS Post	SPRS- M Pre	SPRS- M Post	BFNE- S Pre	BFNE- S Post	PRCS Pre	PRCS Post	SSES Pre	SSES Post
SIAS/SPS Pre	1	.907**	-.303	-.278	.626**	.604**	-.477**	-.515**	-.640**	-.420**
SIAS/SPS Post		1	-.283	-.313*	.608**	.681**	-.487**	-.572**	-.658**	-.522**
SPRS-M Pre			1	.851**	-.067	-.211	.423**	.462**	.390*	.343*
SPRS-M Post				1	-.059	-.238	.412**	.461**	.415**	.348*
BFNE-S Pre					1	.869**	-.352*	-.339*	-.696**	-.604**
BFNE-S Post						1	-.472**	-.524**	-.694**	-.686**
BPRCS Pre							1	.811**	.328*	.374*
BPRCS Post								1	.398*	.408**
SSES Pre									1	.821**
SSES Post										1

Note. SIAS/SPS = Social Interaction Anxiety Scale-6/Social Phobia Scale-6 combined; SPRS-M = Social Performance Rating Scale-Modified; BFNE-S = Brief Fear of Negative Evaluation – Straightforward items; BPRCS = Brief Personal Record of Confidence as a Speaker; SSES = State Self-Esteem Scale. ** $p < .01$; * $p < .05$.

Appendix E

Participant recruitment PRISMA flow chart. Adapted from Moher et al. (2009).



Appendix F

Basic demographic information for the sample

Group	N	Female	SAD dx	SIAS/SPS-6	
				Pre	Post
Experiential	15	80.0% (n = 12)	20.0% (n = 3)	23.47 (8.48)	23.27 (8.89)
Exposure	14	92.9% (n = 13)	42.9% (n = 6)	23.57 (8.78)	24.50 (7.92)
Control	12	91.7% (n = 11)	33.3% (n = 4)	23.75 (6.54)	25.25 (7.33)
Total	41	87.8% (n = 36)	31.7% (n = 13)	23.59 (7.87)	24.27 (7.97)

Note. SAD dx = diagnosable Social Anxiety Disorder (DSM-V criteria); SIAS/SPS-6 = combined Social Interaction Anxiety Scale / Social Phobia Scale-6. Standard deviations are indicated in the brackets for SIAS/SPS-6.

Appendix G

Means and standard deviations of outcome measures

Group	N	SPRS-M		BFNE-S		PRCS		SSES	
		Pre	Post	Pre	Post	Pre	Post	Pre	Post
Experiential	15	10.47 (1.96)	11.93 (1.91)	28.07 (7.43)	27.93 (7.13)	3.67 (2.09)	3.13 (1.96)	64.33 (7.93)	59.00 (10.52)
Exposure	14	11.21 (2.83)	11.71 (2.70)	25.36 (6.45)	25.71 (6.33)	4.00 (2.54)	3.50 (2.41)	62.86 (10.45)	60.14 (10.17)
Control	12	11.50 (2.91)	11.67 (3.26)	30.25 (4.77)	29.50 (6.13)	3.17 (2.41)	3.17 (2.13)	58.92 (11.26)	56.00 (13.50)
Total	41	11.02 (2.54)	11.78 (2.56)	27.78 (6.56)	27.63 (6.59)	3.63 (2.31)	3.27 (2.12)	62.24 (9.87)	58.51 (11.20)

Note. SPRS-M = Social Performance Rating Scale-Modified; BFNE-S = Brief Fear of Negative Evaluation-Straightforward items; PRCS = Personal Record of Confidence as Speaker; SSES = State Self-Esteem Scale. Standard deviations for all measures are indicated in the brackets.

Appendix H

Means and standard deviations of SUDS during the experimental task

Group	N	SUDS					
		1st task iteration		2nd task iteration		3rd task iteration	
		Step 4b	Step 4d	Step 4b	Step 4d	Step 4b	Step 4d
Experiential	15	64.2 (26.7)	53.5 (26.1)	50.3 (25.5)	44.8 (26.9)	41.9 (24.9)	37.7 (22.8)
Exposure	14	66.9 (24.3)	59.6 (21.1)	56.1 (24.7)	49.9 (28.1)	47.9 (27.9)	46.7 (27.0)
Control	12	73.3 (20.0)	52.5 (20.6)	52.9 (22.2)	47.1 (23.2)	45.4 (22.3)	45.00 (24.3)
Total	41	67.8 (23.8)	55.3 (22.6)	53.02 (23.9)	47.2 (25.7)	45.0 (24.7)	42.9 (24.4)

Note. SUDS = Subjective Units of Distress Scale. Standard deviations are indicated in the brackets.

Appendix I

Means and standard deviations of SSGS during the experimental task

Group	N	SSGS					
		1st task iteration		2nd task iteration		3rd task iteration	
		Step 4b	Step 4d	Step 4b	Step 4d	Step 4b	Step 4d
Experiential	15	4.9 (2.8)	4.5 (2.5)	4.6 (2.5)	3.9 (2.0)	3.9 (2.2)	3.6 (2.0)
Exposure	14	5.7 (2.8)	5.1 (2.2)	4.9 (2.0)	4.5 (2.5)	4.6 (2.5)	4.5 (2.4)
Control	12	6.5 (2.3)	4.8 (1.5)	4.4 (1.9)	3.9 (1.3)	3.8 (1.8)	4.4 (2.0)
Total	41	5.6 (2.7)	4.8 (2.1)	4.6 (2.1)	4.1 (2.0)	4.1 (2.2)	4.2 (2.1)

Note. SSGS = State Shame and Guilt Scale. Standard deviations are indicated in the brackets.

Appendix J

Measures of average LF/HF ratio at different times during the study

Group	N	LF/HF ratio					
		5-min Baseline	First Speech	1st task iteration	2nd task iteration	3rd task iteration	Second Speech
		2.26	3.56	1.79	1.96	2.07	3.46
Experiential	15	(1.48)	(2.50)	(1.61)	(1.77)	(2.19)	(3.01)
		1.88	3.54	1.51	2.09	2.04	3.46
Exposure	13	(1.27)	(3.11)	(0.91)	(1.33)	(1.28)	(2.37)
		2.40	2.76	2.36	2.18	2.22	3.38
Control	11	(1.38)	(1.49)	(1.57)	(1.84)	(1.86)	(2.11)
		2.17	3.33	1.86	2.06	2.10	3.44
Total	39	(1.36)	(2.46)	(1.41)	(1.62)	(1.79)	(2.51)

Note. LF/HF = a ratio of low to high frequency component of recorded heart rate variability. Standard deviations are indicated in the brackets.

Appendix K

Measures of average HF component at different times during the study

Group	N	HF					
		5-min Baseline	First Speech	1st task iteration	2nd task iteration	3rd task iteration	Second Speech
Experiential	15	0.34 (0.13)	0.27 (0.15)	0.41 (0.18)	0.39 (0.15)	0.42 (0.20)	0.27 (0.12)
Exposure	13	0.37 (0.14)	0.29 (0.16)	0.36 (0.11)	0.33 (0.10)	0.37 (0.16)	0.27 (0.11)
Control	11	0.31 (0.15)	0.27 (0.09)	0.33 (0.19)	0.34 (0.14)	0.39 (0.20)	0.27 (0.12)
Total	39	0.34 (0.14)	0.28 (0.14)	0.37 (0.6)	0.36 (0.13)	0.39 (0.19)	0.27 (0.11)

Note. HF = normalized high frequency component of recorded heart rate variability. Standard deviations are indicated in the brackets.

Appendix L

Study measures

Demographics questionnaire

Age. How old are you? _____

Gender. Please circle.: Female Male Specify if other: _____

Do you have any of the following conditions? Please circle.

1. Cardiovascular disease or high blood pressure? Y / N

If yes, please specify: _____

2. Severe asthma or other respiratory disorder? Y / N

If yes, please specify: _____

3. Hyperthyroidism or other endocrine disorder? Y / N

If yes, please specify: _____

4. Epilepsy? Y / N

Medication and substance use. Please circle.

1. Are you currently prescribed and taking any psychiatric medication?

Benzodiazepines? Y / N

Beta-blocker medication? Y / N

Other: _____

2. In the past 30 days, what was your average alcohol intake? Please circle.

I do not drink 1-4 drinks/wk 5-9 drinks/wk 10 or more drinks/wk

3. In the past 30 days, how often did you use cannabis? Please circle.

I didn't Less than weekly Almost every week Almost every day

4. In the past 30 days, have you used any other recreational drugs (e.g. LSD)? Y / N

Suicidal ideation and self-harm. Please circle.

1. In the past 30 days, have you had thoughts about killing yourself? Y / N

2. In the past 30 days, have you engaged in self-harming (e.g. cutting)? Y / N

SIAS-6 and SPS-6

Instructions: For each question, please circle a number to indicate the degree to which you feel the statement is characteristic or true of you. The rating scale is as follows:

	0	1	2	3	4
	Not at all characteristic or true of me	Slightly characteristic or true of me	Moderately characteristic or true of me	Very characteristic or true of me	Extremely characteristic or true of me
1. I have difficulty making eye contact with others.	0	1	2	3	4
2. I find it difficult mixing comfortably with the people I work with.	0	1	2	3	4
3. I tense up if I meet an acquaintance on the street.	0	1	2	3	4
4. I feel tense if I am alone with just one person.	0	1	2	3	4
5. I have difficulty talking with other people.	0	1	2	3	4
6. I find it difficult to disagree with another's point of view.	0	1	2	3	4
7. I get nervous that people are staring at me as I walk down the street.	0	1	2	3	4
8. I worry about shaking or trembling when I'm watched by other people.	0	1	2	3	4
9. I would get tense if I had to sit facing other people on a bus or train.	0	1	2	3	4
10. I worry I might do something to attract the attention of other people.	0	1	2	3	4
11. When in an elevator, I am tense if people look at me.	0	1	2	3	4
12. I can feel conspicuous standing in a line.	0	1	2	3	4

Note. Items 1–6 are from the Social Interaction Anxiety Scale (SIAS), and Items 7–12 are from the Social Phobia Scale (SPS).

(Peters, Sunderland, Andrews, Rapee, & Mattick, 2012)

Brief Fear of Negative Evaluation, Straightforward Items

(Carleton, Collimore, McCabe, & Antony, 2011; Rodebaugh et al., 2004; Weeks et al., 2005)

Please circle the number that best corresponds to how much you agree with each item.

	Not at all characteristic of me	A little characteristic of me	Somewhat characteristic of me	Very characteristic of me	Entirely characteristic of me
1. I worry about what other people will think of me even when I know it doesn't make any difference.	1	2	3	4	5
2. I am frequently afraid of other people noticing my shortcomings.	1	2	3	4	5
3. I am afraid that others will not approve of me.	1	2	3	4	5
4. I am afraid that other people will find fault with me.	1	2	3	4	5
5. When I am talking to someone, I worry about what they may be thinking about me.	1	2	3	4	5
6. I am usually worried about what kind of impression I make.	1	2	3	4	5
7. Sometimes I think I am too concerned with what other people think of me.	1	2	3	4	5
8. I often worry that I will say or do wrong things.	1	2	3	4	5

Score: _____

Personal Report of Confidence as a Speaker

(Hook, Smith, & Valentiner, 2008)

See if the following statements apply to you. Please circle the answer.

1. My hands tremble when I try to handle objects on the platform.
Y / N
2. I am in constant fear of forgetting my speech.
Y / N
3. While preparing a speech I am in a constant state of anxiety.
Y / N
4. My thoughts become confused and jumbled when I speak before the audience.
Y / N
5. Although I talk fluently with friends I am at a loss for words on the platform.
Y / N
6. The faces of my audience are blurred when I look at them.
Y / N
7. I feel disgusted with myself after trying to address a group of people.
Y / N
8. I perspire and tremble just before getting up to speak.
Y / N
9. My posture feels strained and unnatural.
Y / N
10. I am fearful and tense all the while I am speaking before a group of people.
Y / N
11. It is difficult for me to search my mind calmly for the right words to express my thoughts.
Y / N
12. I am terrified at the thought of speaking before a group of people.
Y / N

State Self-Esteem Scale

(Heatherton & Polivy, 1991)

This is a questionnaire designed to measure what you are thinking at this moment. There is of course no wrong answer for any statement. The best answer is what you feel is true of yourself at the moment. Be sure to answer all of the items, even if you are not certain of the best answer. Again, answer these questions as they are true for you RIGHT NOW.

1. I feel confident about my abilities.

1	2	3	4	5
Not At All	A Little Bit	Somewhat	Very Much	Extremely

2. I am worried about whether I am regarded as a success or failure.

1	2	3	4	5
Not At All	A Little Bit	Somewhat	Very Much	Extremely

3. I feel satisfied with the way my body looks right now.

1	2	3	4	5
Not At All	A Little Bit	Somewhat	Very Much	Extremely

4. I feel frustrated or rattled about my performance .

1	2	3	4	5
Not At All	A Little Bit	Somewhat	Very Much	Extremely

5. I feel that I am having trouble understanding things that I read.

1	2	3	4	5
Not At All	A Little Bit	Somewhat	Very Much	Extremely

6. I feel that others respect and admire me.

1	2	3	4	5
Not At All	A Little Bit	Somewhat	Very Much	Extremely

7. I am dissatisfied with my weight.

1	2	3	4	5
Not At All	A Little Bit	Somewhat	Very Much	Extremely

8. I feel self-conscious.

1	2	3	4	5
Not At All	A Little Bit	Somewhat	Very Much	Extremely

9. I feel as smart as others.

1	2	3	4	5
Not At All	A Little Bit	Somewhat	Very Much	Extremely

10. I feel displeased with myself.

	1	2	3	4	5
	Not At All	A Little Bit	Somewhat	Very Much	Extremely
11. I feel good about myself.	1	2	3	4	5
	Not At All	A Little Bit	Somewhat	Very Much	Extremely
12. I am pleased with my appearance right now.	1	2	3	4	5
	Not At All	A Little Bit	Somewhat	Very Much	Extremely
13. I am worried about what other people think of me.	1	2	3	4	5
	Not At All	A Little Bit	Somewhat	Very Much	Extremely
14. I feel confident that I understand things.	1	2	3	4	5
	Not At All	A Little Bit	Somewhat	Very Much	Extremely
15. I feel inferior to others at this moment.	1	2	3	4	5
	Not At All	A Little Bit	Somewhat	Very Much	Extremely
16. I feel unattractive.	1	2	3	4	5
	Not At All	A Little Bit	Somewhat	Very Much	Extremely
17. I feel concerned about the impression I am making.	1	2	3	4	5
	Not At All	A Little Bit	Somewhat	Very Much	Extremely
18. I feel that I have less scholastic ability right now than others.	1	2	3	4	5
	Not At All	A Little Bit	Somewhat	Very Much	Extremely
19. I feel like I'm not doing well.	1	2	3	4	5
	Not At All	A Little Bit	Somewhat	Very Much	Extremely
20. I am worried about looking foolish.	1	2	3	4	5
	Not At All	A Little Bit	Somewhat	Very Much	Extremely

State Shame and Guilt Scale, Shame subscale

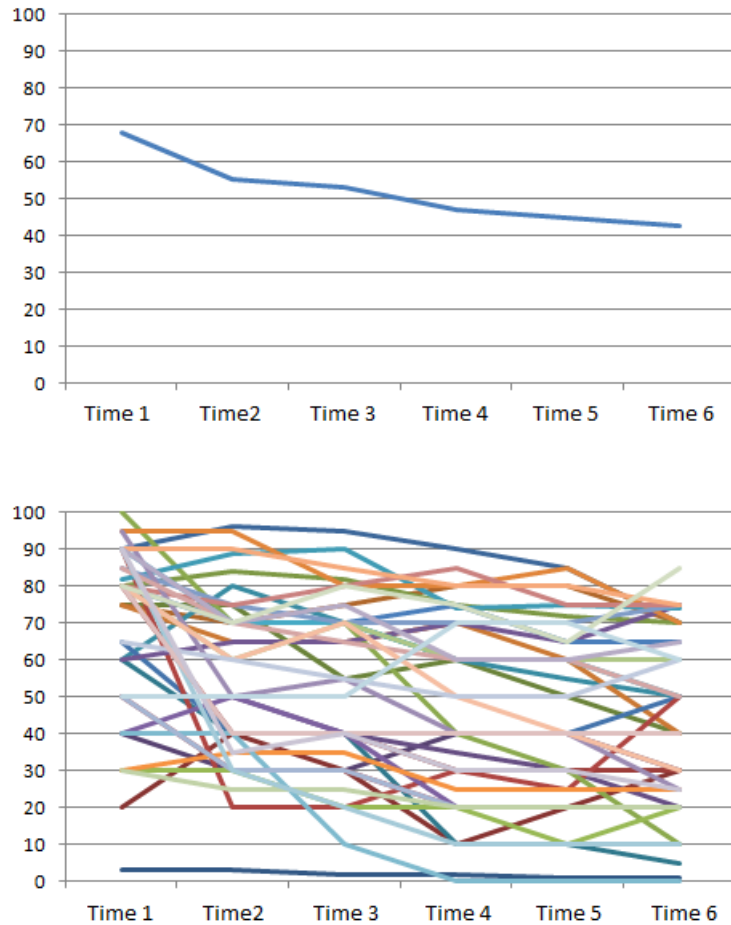
(SSGS; Marschall, Sanftner, & Tangney, 1994)

The following are some statements which may or may not describe how you are feeling right now. Please rate each statement using the 5-point scale below. Remember to rate each statement based on how you are feeling right at this moment.

	I do not feel this way at all		I feel this way somewhat		I feel this way very strongly	
1. I want to sink into the floor and disappear	1	2	3	4	5	
2. I feel small	1	2	3	4	5	
3. I feel like I am a bad person	1	2	3	4	5	
4. I feel humiliated, disgraced	1	2	3	4	5	
5. I feel worthless, powerless	1	2	3	4	5	

Appendix M

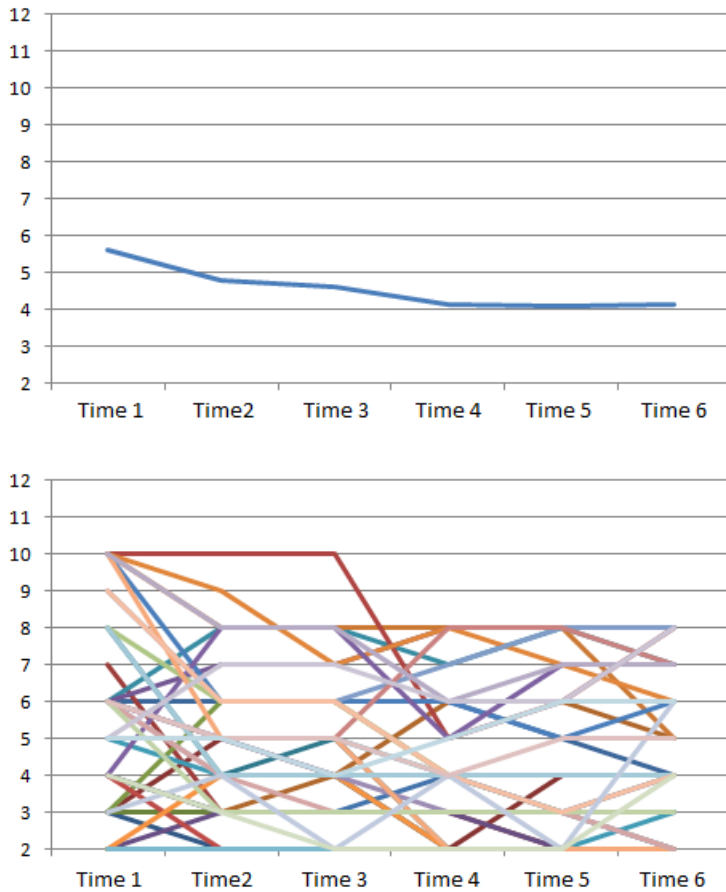
Average and individual fear (SUDS) scores plotted over the course of the experimental tasks.



Note. These scores were measured three times during Steps 4b and 4d of the study, resulting in six measurements. Average scores are plotted on the top and individual scores on the bottom of the graph.

Appendix N

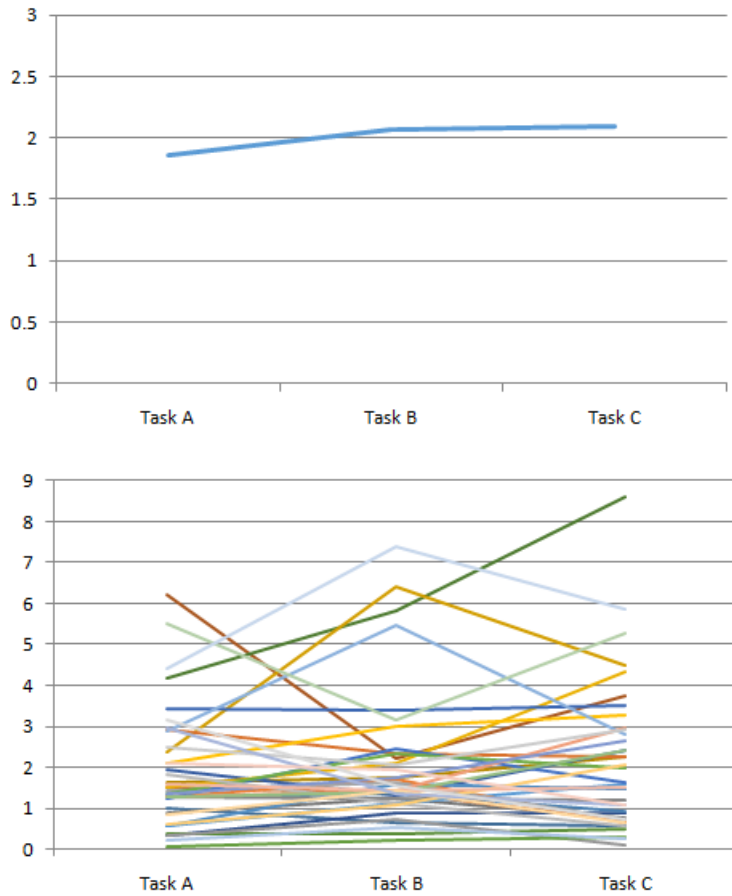
Average and individual shame (SSGS) scores plotted over the course of the experimental tasks.



Note. These scores were measured three times during Steps 4b and 4d of the study, resulting in six measurements. Average scores are plotted on the top and individual scores on the bottom of the graph.

Appendix O

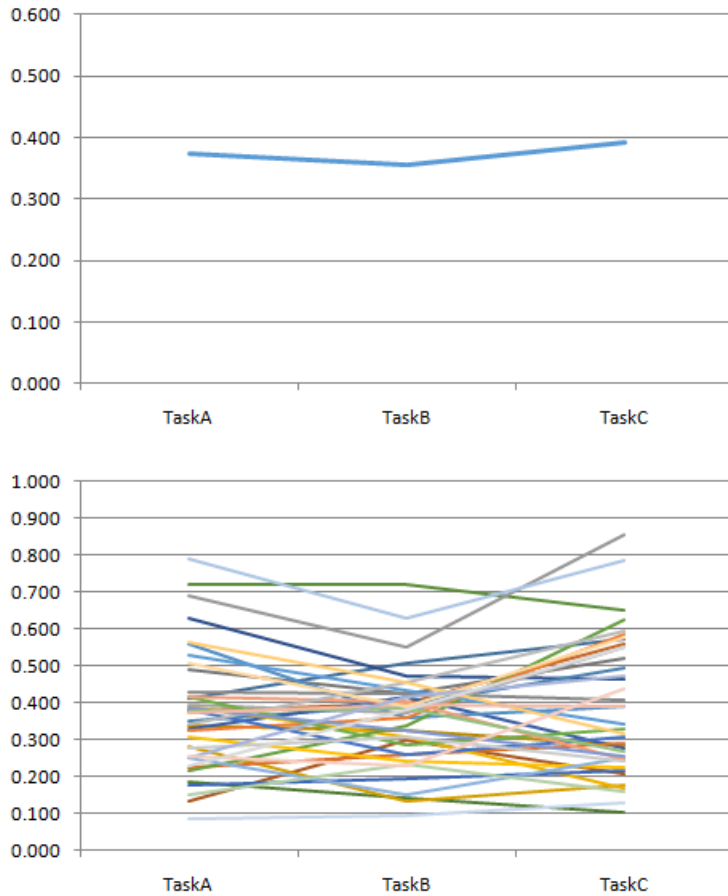
Average and individual low frequency to high frequency (LF/HF) ratio scores plotted over the course of the experimental tasks.



Note. These scores were measured three times throughout the Step 4c of the study. Average scores are plotted on the top and individual scores on the bottom of the graph.

Appendix P

Average and individual high frequency (HF) component of recorded heart rate variability scores plotted over the course of the experimental tasks.



Note. These scores were measured three times throughout the Step 4c of the study. Average scores are plotted on the top and individual scores on the bottom of the graph.

Vita Auctoris

NAME: Nikita Yeryomenko
PLACE OF BIRTH: Frunze, USSR
YEAR OF BIRTH: 1986
EDUCATION: York University, B.A., Toronto, ON, 2006-2009
University of Windsor, M.A., Windsor, ON, 2010-2012