THE PHYSIOLOGICAL RESPONSE TO IMPLICIT AND EXPLICIT FEAR FACES IN ALEXITHYMIA

by

Alison M. Gilbert

B.S., Cornell University, 2000

M.S., University of Pittsburgh, 2004

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This dissertation was presented

by

Alison M. Gilbert

It was defended on

April 23, 2008

and approved by

Ellen Frank, Ph.D., Professor

Mary L. Phillips, M.D., Ph.D., Professor

Michael Pogue-Geile, Ph.D., Associate Professor

Michael Sayette, Ph.D., Professor

Dissertation Advisor: Julie Fiez, Ph.D., Professor

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Alison M. Gilbert, PhD

University of Pittsburgh, 2008

Alexithymia is a form of disordered emotional processing associated with psychiatric illness and poor treatment outcomes. Its hallmarks are difficulty identifying, representing and evaluating emotional material. Research on alexithymia includes investigations of behavioral, physiological and neural responsivity. Findings within and between these different methodologies are inconsistent and often not well motivated. We use the neuroimaging literature to motivate a design that links behavior and physiology in alexithymia. The aims of this study are to determine: 1) whether individuals with alexithymia exhibit behavioral responses to emotional faces that differ from controls and whether there are corresponding changes in physiology, 2) whether behavioral and physiological responses in alexithymia are differentially influenced by an implicit versus explicit manipulation of emotional task context, compared to controls.

For Aim 1, we measure behavioral and physiological responses during two explicit facial categorization tasks: an emotion task requiring categorization of morphed emotional facial expressions, and an identity task requiring categorization of morphed facial identities. For Aim 2, we evaluate responses to emotional and neutral facial expressions in two task contexts: in an explicit task participants label the emotion of the face, while in an implicit task participants label the gender of the face. Using these experiments we determined whether undergraduates scoring

high (TAS \geq 61) or low (TAS \leq 51) on alexithymia (TAS-20) differ on accuracy, reaction time, heart rate and skin conductance.

Counter to prevailing theories of alexithymia as an appraisal problem, we found that alexithymia participants' task accuracy on emotional and non-emotional tasks is intact. We found less sensitivity to fearful faces during an emotion categorization task, but not a non-emotional identity categorization task, as we expected. In alexithymia, we found less sensitivity to emotion and altered heart rate reactivity during implicit and explicit tasks, compared to healthy controls. Counter to current conceptualizations of alexithymia as a difficulty engaging in top-down appraisal of emotional information, our findings suggest that alexithymia may be a problem generating physiological responses to emotional information. Based on our view of alexithymia, we revisit the current state of the alexithymia literature, provide suggestions for future research and propose potential therapeutic interventions.

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1.0 INTRODUCTION

1.1 OVERVIEW

Alexithymia (from the greek: a = lack, lexis = words, thymos = emotion) is a cognitive-affective disturbance that is characterized by a verbal and non-verbal difficulty recognizing and describing emotions of all types. The alexithymia construct emerged following attempts of clinicians and neuroscientists to characterize the emotional features of psychosomatic patients (Ruesch, 1948; Maclean, 1949). Clinical investigations were followed by the development of a construct that could address these features. In 1973, the term alexithymia was officially coined by Peter E. Sifneos. Sifneos used the term alexithymia to describe the subset of psychosomatic patients who were unable to articulate their emotions and experience benefits from psychoanalytic treatment (Sifneos, 1973). The clinical features of psychosomatic patients included inappropriate affect, lack of sensitivity and difficulty communicating (Sifneos, 1996).

Alexithymia is a clinically relevant construct that is associated with poor treatment outcomes in clinical groups, especially anxious and depressed patients (McCallum, Piper, Ogrodniczuk & Joyce, 2003; Ogrodniczuk, Piper, & Joyce, 2004, 2005). Thus, alexithymia poses an important challenge to current psychotherapeutic approaches that emphasize emotion identification as well as subsequent regulation. For example, alexithymia was a predictor of poor treatment outcome in a group of depressed patients who responded to interpretive or supportive

therapy; the effect of alexithymia symptoms was greater than that of baseline depression and anxiety symptoms, psychotherapy and medication treatment (Ogrodniczuk, Piper, & Joyce, 2004). Alexithymia is also associated with increased morbidity (i.e., cardiovascular disease) (Koh, Cho, Kim, Rho, Lee & Park, 2004; Kojima, Frasure-Smith & Lesperance, 2001) and disease-related mortality (Kauhanen, Kaplan, Cohen, Julkunen, & Salonen, 1996). Alexithymia is, therefore, a necessary focus of current psychobiological models of emotion identification and regulation as well as treatment of psychiatric illness.

The objective of this research is to investigate the physiological and behavioral correlates of alexithymia using a neuroscience approach. Before describing our experimental design we will review the alexithymia construct and its relationship with other constructs, such as personality and psychopathology.

1.1.1 Alexithymia: Historical development and association with other constructs

Over the past decade, researchers have generally agreed that the hallmark of alexithymia is a difficulty identifying feelings (DIF) and a difficulty describing feelings (DDF). This has resulted in the recent development of the most current and well-validated measure of alexithymia, the 20-item Toronto Alexithymia Scale¹ (TAS-20) (Bagby, Parker & Taylor, 1994a). The TAS-20 attempts to determine which individuals have a difficulty discriminating between emotions and physical sensations as well as verbalizing their own emotional experience. Alexithymia is also characterized by impairment in the ability to identify and describe the emotions of others, including their facial expressions (i.e., fear) (Jessimer & Markham, 1997; Lane et al., 1996,

¹ Studies in this review use the TAS-20 to assess alexithymia unless specified; TAS-26 is a 26 item measure that includes a fourth factor related to daydreaming.

2000; McDonald & Prkachin, 1990; Panday & Mandel, 1997; Parker et al., 1993, 2005), and in the ability to empathize with others (i.e., Haviland, Sonne & Kowert, 2004). Individuals with alexithymia have been also described as exhibiting constricted facial displays of emotion (McDonald & Prkachin, 1990). The intrapersonal and interpersonal difficulties of alexithymic individuals manifest as poor interpersonal functioning. For example, alexithymia has been associated with reduced perceived social support and a smaller social network (Lumley, Ovies, Stettner, Wehmer, & Lakey, 1996). Alexithymic patients are less likely to be married and report fewer close friendships and acquaintances (Kauhanen, Kaplan, Julkunen, Wilson, & Salonen, 1993).

Another feature of alexithymia is an externally oriented cognitive style (EXT) (Bagby, Parker & Taylor, 1994; Marty & de M'Uzanm, 1963). This domain is based on clinician reports that individuals with alexithymia prefer to describe the minute details of situations and physical symptoms rather than rely on imagination or introspection (Sifneos, 1996). For example, an individual with alexithymia may describe the action they take when confronted with a distressing circumstance rather than describe any accompanying feelings or fantasies.

The majority of research on alexithymia and sociodemographic variables has found that alexithymia is associated with male gender, increased age, low education and low socioeconomic status (Honkalampi, Saarinen, Hintikka, Virtanen & Viinamaki, 1999; Honkalampi, Hintikka, Laukkanen, Lehtonen & Viinamaki, 2001; Kokkonen et al., 2001; Lane, Sechrest, & Riedel, 1998; Salminen, Saarijarvi, Aarela, Toikka & Kauhenen, 1999; for contradictory evidence see Parker, Taylor, & Bagby, 2003, Bagby et al., 2004a). In another study, patients with alexithymia were also found to have a lower verbal and performance IQ (Honkalampi et al., 1999). In a sample of young adults in Finland the prevalence of alexithymia was 5.2% in females and 9.4%

in males (n= 5,028) (Kokkonen et al., 2001). In the general population of Finland, it was found that alexithymia is present in 17% of males and 10% of females (n = 1,285) (Salminen et al., 1998). In a British sample of undergraduates it was found that alexithymia was present in 18% of students, with higher rates in females compared to males (n = 371) (Mason, Tyson, Jones & Potts, 2005). In the current study we will match for gender since otherwise the number of men and women might differ between the two subject groups.

1.1.2 Alexithymia, defenses and coping

It has been suggested that alexithymia results from the use of ego defense mechanisms, such as repression, denial or reaction formation (Knapp, 1983; Parker, Taylor & Bagby, 1998; Vaslamatzis, 2003). The Defensive Style Questionnaire (DSQ) has been used to explore the relationship between alexithymia and ego defenses (Andrews, Sign & Bond, 1993). The DSQ measures maladaptive/immature defenses (including humour, suppression and sublimation), adaptive/mature defenses including regression, acting-out, passive-aggression and projection), and neurotic defenses (idealization, reaction formation, undoing and repression). It was found that alexithymia is positively correlated with maladaptive/immature defenses and negatively correlated with mature/adaptive defenses (Wise, Mann & Epstein, 1991, TAS-26; Parker et al., 1998). Furthermore, when the TAS-20 was used to classify individuals as alexithymic it was found that those categorized as alexithymic were more likely to use immature and neurotic defenses and less likely to use mature defenses (Parker et al., 1998).

Despite the correlation between alexithymia and ego defense mechanisms, it has been argued that these constructs can be distinguished (Lane, Sechrist, Reidel, Shapiro, & Kaszniak, 2000). For example, repression has been characterized by a suppression of emotional experience,

while alexithymia is considered a difficulty in the appraisal of emotional experiences (Aftanas, Varlamov, Reva & Pavlov, 2003; Parker et al., 1998; Taylor et al, 1997). Furthermore, it has been argued that alexithymia does not serve as an effective coping strategy because it is associated with poor health outcomes (Parker et al., 1998).

Evidence that alexithymia differs from a coping response, such as repression, has also been conducted by identifying groups of healthy individuals who use a "repressive-defensive" coping style. The repressive-defensive coping style is characterized by high scores on the Marlow Crown Social Desirability Scale and low scores on the Taylor Manifest Anxiety Scale. It was found that those who exhibited a repressive-defensive coping style scored low on measures of alexithymia and exhibited physiological responses that were associated with low levels of alexithymia (Myers, 1995; Newton & Contrada, 1994). Alexithymia has also been associated with a greater number of impairments on an emotion recognition task relative to individuals classified as using a repressive-coping style (Lane et al., 2000). In a related study, alexithymia was associated with a variety of coping strategies including immature defenses, avoidance oriented coping (i.e. distraction) and emotion coping (i.e. worrying, somaticising) (Parker et al, 1998). Based on these findings Parker et al (1998) proposed that alexithymia is a complex construct that may disrupt the ability to utilize appropriate coping strategies.

Others have suggested that one type of alexithymia, secondary alexithymia, serves as a coping response. More specifically, it has been proposed that secondary alexithymia is a state reaction or defense strategy that is invoked to cope with physical and psychiatric illness (Freyberger, 1977; Fukunishi, Kikuchi, Wogan, & Takubo, 1997 Wise, Mann, Mitchell, Hryvniak, & Hill, 1990- TAS-26). Secondary alexithymia may also account for findings of an association between symptoms of dissociation and alexithymia. Dissociation is a "disruption in

the usually integrated functions of consciousness, memory, identity, or perception (APA, 2000, pg. 519). Dissociative experiences have been characterized as coping strategies in response to stressful or painful experiences. Thus, dissociation may involve a difficulty in emotional awareness that is also characteristic of secondary alexithymia.

The Dissociative Experiences Questionnaire (DEQ) and Dissociation Questionnaire (DIS-Q) have been used to explore the relationship between dissociation and alexithymia. In a clinical and non-clinical population, individuals with pathological symptoms of dissociation exhibited higher alexithymia scores, relative to individuals without pathological levels of dissociation (Grabe, Rainermann, Spitzer, Gansicke & Freyberger, 2000). In a non-clinical, undergraduate, sample only, dissociative symptoms were associated with alexithymia. This relationship was partially mediated by current levels of stress (Elzinga, Bermond, & van Dyck, 2002). In contrast, scores on the DES failed to predict scores on the TAS-20 in a sample of psychiatric outpatients (Wise, Mann, & Sheridan, 2000). A related study found that there was no association between dissociation and alexithymia in psychiatric patients (Zlotnik et al., 1996). Furthermore, using factor analysis it was determined that dissociation and alexithymia are non-overlapping constructs in a non-clinical population (Lipsanen, Saarijarvi, & Lauerma, 2004).

Based on the current literature and mixed findings, it remains possible that alexithymia is a manifestation of inappropriate coping. The current investigation is focused on uncovering whether alexithymia is a construct that involves inappropriate arousal and/or appraisal of emotional experience as indicated by physiological responses. Whether this is a manifestation of inappropriate coping strategies is not the focus of the current investigation, but is a valid consideration for future research.

1.1.3 Alexithymia and psychopathology

The salient features of alexithymia, impairment in emotion regulation and appraisal, suggest that the construct may play an important role in, and improve our understanding of, medical and psychiatric illnesses of emotion regulation. Indeed, alexithymia has been associated with and has been described as a potential risk factor for psychiatric illnesses that are characterized by poor regulation of emotional experience. Associated psychiatric illnesses include anxiety, depression, PTSD, drug/alcohol use, eating disorders and somaticization (for review see Taylor et al., 1997).

Psychiatric research has also capitalized on the alexithymia construct as a way to better characterize difficulty identifying emotions of self and other that is characteristic of autism spectrum disorders (Berthoz & Hill, 2005; Hill, Berthoz & Frith, 2004). As suspected, autistic individuals have been found to exhibit higher alexithymia scores on the TAS-20. Likewise, the alexithymia construct has been used to enhance our understanding of schizophrenic symptoms. For example, a few studies have used measures of alexithymia to examine self-perceived deficits in expressive and receptive language, an impairment that may manifest as a difficulty labeling emotional experiences. Indeed, it has been found that scores on the Bonn Scale Receptive and Expressive Language Impairment Index were associated with the TAS-20 in a group of schizophrenic patients (Maggini, Raballo, Pelizza, Paini & Croci, 2003). Others have been interested in whether alexithymia is present in a population of schizophrenic patients, since both alexithymic and schizophrenic individuals have difficulties processing emotional experiences and faces (i.e., Schnieder et al., 2006; see Table 1). A recent study found that alexithymia was unrelated to negative symptoms of schizophrenia and course of illness. Furthermore, unlike symptoms of schizophrenia, alexithymia was stable from baseline to 12 months post treatment (Todarello, Porcelli, Grilletti & Bellomo, 2004). In a related study, it was found that alexithymia scores were significantly higher in paranoid compared to non-paranoid schizophrenics and that alexithymia was associated with language impairment. However, an inadequate measure of alexithymia was used (Stanghellini & Ricca, 1995).

A current debate in the literature is whether alexithymia is associated with psychiatric illnesses because their constructs overlap. This debate has been especially evident in the depression literature where strong associations have been found between alexithymia and depression (r = .42-.65) (i.e., Speranza, Corcos, Guilbaud, Loas, & Jeanmet, 2005; Honkalampi, Hintikka, Tanskanen, Lehtonen & Viinamaki, 2000; Lipsanen et al., 2004; Marchesi, Brusamonti, Maggini, 2000). For example, a recent study found that 3.2% of non-depressed versus 32.1% of depressed individuals in the general population were alexithymic (Honkalampi et al., 2000). Factor analytic methods have been used to examine the potential overlap between alexithymia and depression. Several studies have found that the TAS-20 and measures of depression (Hospital Anxiety and Depression Scale, Beck Depression Inventory, Zung-Self Rating Depression Scale) are non-overlapping (Marchesi et al., 2000; Muller, Buhner, & Ellering, 2003; Parker et al., 1991), while other research has yielded contradictory results using similar depression measures (Beck Depression Inventory) (Hintikka, Honkalampi, Lehtonen & Viinamaki, 2001; Lipsanen et al., 2004). One explanation for the potential overlap between depression and alexithymia is that negative affect promotes critical perceptions of self, thereby increasing the likelihood that individuals will report that they are poor at detecting emotions (Lumley, 2000). To account for these findings, we will exclude participants who have clinically significant symptoms of depression and related disorders. In a subset of participants we will also match for trait levels of neuroticism, which are associated with negative emotional states such as

depression (Trull & Sher, 1994; Bagby et al., 1994a), and trait levels of anxiety (Spielberger, 1983).

1.1.4 Alexithymia: State or trait

It has been posited that if alexithymia is a trait it should be stable and unaltered by affective states, including psychopathology and stress. In the clinical literature there is ongoing disagreement about whether alexithymia is considered a state or trait characteristic. One reason for the current debate in the alexithymia literature is that studies have used different methodological approaches (Luminet, Baby & Taylor, 2001). For example, in depressed patients, both cross-sectional and longitudinal approaches have been used to study alexithymia. Cross-sectional research has suggested that alexithymia is associated with a depressed state (i.e., Speranza et al., 2005; Honkalampi et al., 2000; Lipsanen et al., 2004; Marchesi et al., 2000). Longitudinal studies of alexithymia and depression have yielded mixed conclusions about whether alexithymia is a state or trait. For example, alexithymia (TAS-26) was stable at one year follow-up in a group of patients with anxiety and mood disorders (Salminen, Saarijarvi, Aairela & Tamminen, 1994). Saarijarvi et al (2001) found that alexithymia was associated with changes in depression and distress 1 year post treatment. Two longitudinal studies found a decline in both alexithymia and depression scores at 6 and 12 months post treatment, suggesting that alexithymia is related to affective state (Honkalampi et al., 2000; 2001). In a non-clinical undergraduate sample depression and anxiety symptoms waxed and waned along with exam-related stress, while symptoms of alexithymia remained unchanged (Martinez-Sanchez, Ato-Garcia, Adam, Medina & Espana, 1998).

In order to resolve inconsistencies in the literature, Luminet et al. (2001) examined the absolute and relative stability of alexithymia in a group of depressed patients. Absolute stability was defined as change in alexithymia over time, while relative stability was defined as the degree to which relative differences in alexithymia scores remain the same over time (Luminet et al., 2001). It was found that absolute stability was minimal relative to the change in depression symptoms post-treatment. In contrast, alexithymia scores exhibited relative stability- alexithymia at baseline significantly predicted alexithymia post-treatment when controlling for depressive symptoms. Based on this methodological approach, Luminet et al. (2001) concluded that alexithymia has a high degree of relative stability. However, small changes in absolute levels of alexithymia suggest that alexithymia may also have state-like properties.

Similar results were found when alexithymia symptoms were evaluated in a 5 year follow-up study and both relative and absolute stability were assessed (Saarijarvi et al., 2001; Saarijarvi, Salminen, & Toikka, 2006). The authors found relative, but not absolute stability of alexithymia. Scores at 5 year follow-up were predicted by baseline alexithymia scores, not depression symptoms, suggesting that alexithymia has relative stability and is trait-like. Alexithymia and depression scores decreased at 5 year follow-up and alexithymia and depression were correlated at baseline and follow-up. These results suggest that alexithymia behaves as an affective state modulated by depression (Saarijarvi, Salminen, & Toikka, 2006). In a related study, alexithymia was assessed prior to and following a cognitive stressor in a group of female undergraduates (Mikolajczak & Luminet, 2006). It was demonstrated that, following exams, alexithymia scores exhibited high relative stability and mean-level stability (the stability of the mean of the group), whereas psychological distress increased. Since small individual differences in alexithymia existed in this study, it remains possible that changes in affective state can

influence alexithymia. Indeed change in psychological distress accounted for 1% of change in alexithymia scores (Mikolajczak & Luminet, 2006).

Since alexithymia may be associated with changes in affective state, we will monitor these changes in the current study. We are most concerned with changes in anxiety level that may be experienced in our unfamiliar testing environment. Anxiety has also been strongly associated with alexithymia and psychiatric illnesses that often coincide with alexithymia (Berthoz, Consoli, Perez-Diaz, & Jouvent, 1999; Marchesi, Fonto, Balista, Cimmino & Maggini, 2005; Subric-Wrana, Bruder, Thomas, Lane & Kohle, 2005; see Taylor et al., 1997 for review). We will use the Spielberger State Anxiety Inventory (Spielberger, Gorsuch, & Lushene, 1983) to assess anxiety states.

1.1.5 Alexithymia and personality traits

Alexithymia is a multifaceted construct whose components are positively related to one another. As with other constructs, it can be expected that some facets of alexithymia will correlate with related constructs, such as personality traits (Taylor et al., 1997). Among clinical and non-clinical populations alexithymia is most likely a stable, trait-like characteristic (Luminet et al., 2001; Mikolajcczak & Luminet, 2006). In contrast to personality traits, the alexithymia construct emerged from clinical observations of patients during psychoanalytic treatment and as a means of understanding and addressing barriers to treatment. Despite this distinction between personality theory and the historical development of alexithymia, the potential overlap between these constructs has been examined.

It has been suggested that alexithymia incorporates aspects of the five-factor model, a well-validated and cross cultural model of personality. The five-factors include Neuroticism,

Extraversion, Openness, Agreeableness and Conscientiousness. Each of the five domains is composed of several facets, or sub-domains (NEO-PI; Costa & McCrae, 1992). In an undergraduate sample it was found that alexithymia was moderately positively correlated with Neuroticism (r = .27) including the facets Anxiety (r = .25), Depression (r = .36), Self-consciousness (r = .30) and Vulnerability (r = .35). Alexithymia was negatively associated with the Positive Emotion facet of Extraversion (r = -.36). Alexithymia was also negatively correlated with Openness (r = -.49) including the facets Fantasy (r = -.30), Aesthetics (r = -.29), Feelings (r = -.55), Actions (r = -.24) and Ideas (r = -.33) (Bagby et al., 1994b).

In a related study of undergraduates alexithymia was positively associated with Neuroticism and all its facets (r = .19 to .49) and negatively associated with Extraversion (r = .19) including the facets Warmth (r = -.19), Assertiveness (r = -.32), and Positive Emotion (r = .19). Alexithymia was negatively correlated with Competence (r = -.22) and Self-discipline (r = .22) (facets of Conscientiousness). Regression analyses revealed that only 20-29% of the variance in TAS-20 scores could be explained by scores on the NEO-PI. Predictors of TAS-20 scores included the Neuroticism factor and facets of Neuroticism (Vulnerability), Extraversion (Excitement-Seeking) and Openness (Feelings) (Zimmermann, Rossier, de Stadelhofen & Gaillard, 2005). The authors concluded that personality measures are associated with a vulnerability to emotional arousal and distress, low self-esteem and lack of emotional insight that is characteristic of alexithymia (Zimmermann et al., 2005).

A third study of undergraduate students found an association between alexithymia and the five personality factors (NEO-PI-Revised). A positive correlation was found between alexithymia and Neuroticism (r = .38) including the facets Depression (r = .44) and Self-consciousness (r = .31) and vulnerability (r = .39). A negative correlation was found between

alexithymia and Extraversion (r = -.36) including the facets Warmth (r = -.29), Assertiveness (r = -.34), Activity (r = -.30) and Positive Emotions (r = -.37). A negative correlation was found between alexithymia and Openness (r = -.41) including the facets Fantasy (r = -.26), Feelings (r = -.39), Actions (r = -.33) and Ideas (r = r.28). Alexithymia was negatively correlated with Altruism (r = .33), a facet of Agreeableness, and Competence (r = -.40), a facet of Conscientiousness (Luminet, Bagby, Wagner, Taylor & Parker, 1999).

Others have suggested that the three-factor model of personality is associated with and distinct from alexithymia. In a recent study of undergraduate students the Eysenck Personality Questionnaire (EPI: Eysenk & Eysenk, 1975) was used to assess the relationship between alexithymia and Extraversion, Neuroticism, Psychoticism (Parker et al., 1989). A low to moderate positive correlation was found between alexithymia and Neuroticism (r = .29) and Psychoticism (r = .17). A moderate negative correlation was found between alexithymia and Extraversion (r = .37). In addition to these correlations, a confirmatory factor analysis was used to determine the independence of these constructs. A four-factor model was constructed using Alexithymia, Extraversions, Neuroticism, and Psychoticism as factors. In this model alexithymia was moderately related to Neuroticism (r = .45) and Extraversion (r = .46), weakly related to Psychoticism (r = .19) and existed as a distinct construct (Taylor et al., 1997). Based on these findings it has been suggested that alexithymia combines elements of other personality factors and possesses unique properties.

In summary, research has found that alexithymia is associated with personality measures and that alexithymia is an independent construct. In this study, we will attend to personality measures that are most consistently associated with alexithymia, Neuroticism and Extraversion. We will account for personality traits by identifying participants who are matched on

Neuroticism and Extraversion. Matching for Neuroticism and Extraversion will allow us to identify participants who differ only in whether they are alexithymic.

1.1.6 Alexithymia and emotional intelligence

Salovey and Mayer (1990) suggested that alexithymia may be related to the verbal domain of emotional intelligence. Salovey & Mayer (1990) defined emotional intelligence as "the ability to monitor one's own and others' feelings and emotions, to discriminate among them and to use this information to guide one's thinking and actions (pg 189)." The author's relate emotional intelligence to social intelligence, a construct that incorporates intrapersonal and interpersonal intelligence. Indeed, alexithymia was negatively correlated (r = -.65) with a 33-item emotional intelligence scale (Schutte et al., 1998) and several components of a 65-item emotional intelligence scale that was administered to a clinical and non-clinical population (e.g. self-awareness, self-motivation, self-control, situational awareness, interpersonal empathy) (Fukunish et al., 2001). A negative correlation was also found between alexithymia and the Mayer-Salovey-Caruso Emotional Intelligence Test, which assesses performance on a variety of emotional tasks and situations (Lumley, Britta, Partridge, & Labouvie-Vief, 2005).

When a more comprehensive measure of emotional intelligence (BarOn Emotional Intelligence Quotient Inventory; EQ-i) was used a negative association was found between alexithymia and all facets of this measure (Parker, Taylor & Bagby, 2001). The EQ-i assesses intrapersonal (self-awareness, assertiveness, self-regard, self-actualization, independence) and interpersonal intelligence (empathy, relationship skills, social responsibility), adaptability (problem solving, reality testing, flexibility), stress management (stress tolerance, impulsive control, and mood (happiness and optimism). Despite the correlation between alexithymia and

the EQ-i, goodness of fit models determined that the alexithymia construct was distinct from that of the emotional intelligence construct (Parker et al., 2001).

In summary, findings using emotional intelligence measures are consistent with the view that alexithymia involves a lack of emotional insight. The findings also suggest that alexithymia can be measured independently from emotional intelligence. Whether alexithymia represents an ideal measure of emotional intelligence is not the focus of the current investigation, but is a valid consideration for future research.

1.1.7 Measurement of alexithymia: Toronto Alexithymia Scale – 20 item

Over the past four decades researchers have designed a variety of measures to assess alexithymia. These measures have attempted to define the abnormal appraisal process and interpersonal style that is characteristic of alexithymia. The most current and well-validated measure of alexithymia is the Toronto Alexithymia Scale 20- item. This scale assesses three components: difficulty identifying feelings (DIF), difficulty describing feelings (DDF) and externally oriented thinking (EXT). Unlike the TAS-26 the TAS-20 scale eliminates "daydreaming," a factor that had high correlations with social desirability and was negatively correlated with EXT (Bagby et al., 1994a; Parker, Taylor & Bagby, 2003).

Construct validity and reliability has been established for the TAS-20 (Cronbach's alpha = .81; DIF = .78, DDF = .75, EXT = .66; test-retest reliability = .77). The three-factors of the TAS-20 have been replicated in undergraduate students, psychiatric populations, a large community sample, and across 17 different cultures (Belgium, Finland, France, Greece, Germany, India, Italy, Japan, Netherlands, Norway, Peru, Poland, Portugal, South Korea, Spain, Sweden, and Taiwan) (Parker, 1993; Parker et al., 2003; Taylor et al., 2003; Taylor et al., 1997).

Critics have argued that the three factor structure of the TAS-20 is best represented by two factors or has different item loadings than those on the TAS-26 (Davies, Stankow & Roberts, 1998; Erni, Lotscher & Modestin, 1997; Loas, Otmani, Verrier, Fremaux, & Marchand, 1996; Kojima et al., 2001; Kooiman, Spinhoven & Trijsburg, 2002; Zimmermann et al., 2005). One suggestion is that different methodological approaches yield different factor structures (Parker et al., 2003; Loas et al., 2001). For example, when data were reanalyzed using a confirmatory factor analysis the original three-factor structure of the TAS-20 was preserved (Parker et al., 2003; Loas et al., 2001). Other research suffers from important limitations. For example, Davies et al. (1998) failed to reverse score items and use a 5-point Likert scale to score the TAS-20 (Parker et al., 2003). In our study we will look across TAS-20 factors in order to adequately assess the alexithymia construct. We will also use the TAS-20 to determine whether our participants are high (TAS ≥ 61) or low (TAS-20 ≤ 51) on alexithymia.

1.1.8 Summary and approach

In summary, alexithymia is a multifaceted construct whose components are positively related to one another. However, as with other constructs, it can be expected that some facets of alexithymia will correlate with related constructs (Taylor et al., 1997). For purposes of this study we view alexithymia as a trait that could, potentially, be influenced by current affective state. We will assess one aspect of state-related distress, anxiety, using the Spielberger state anxiety scale. We will control for overlap with psychopathology by excluding participants who have clinically significant symptoms of depression and related disorders. In a subset of participants we will match our alexithymia and control groups for levels of trait anxiety, Neuroticism and Extraversion, traits that have been associated with risk for psychiatric illness as well as

alexithymia (Bagby et al., 1994a; Juylha & Isometsa, 2006; Luminet et al., 1999; Trull & Sher, 1994). Thus, our approach will allow us to detect differences in alexithymic and healthy controls that are independent of associated changes in affective state, psychopathology and personality constructs.

1.2 MOTIVATION AND THEORETICAL DEVELOPMENT OF THE PROPOSED WORK

Based on the current literature, alexithymia is a valid and independent construct with clinically significant implications. Research into the etiology of alexithymia includes investigations of behavioral, physiological and, more recently, neural responsivity. Findings within and between these different methodological approaches have yielded inconsistent conclusions and are often not well motivated. The following review identifies key variables that impact the relationship between behavior, physiology and neuroscience in order to develop a well-motivated, integrative, and innovative perspective that allows us to carry out the following study aims: 1) to determine whether individuals with alexithymia exhibit behavioral responses to emotional faces that differ from controls and whether there are corresponding changes in physiology, 2) to examine whether behavioral and physiological responses in alexithymia are differentially influenced by an implicit versus explicit manipulation of emotional task context, as compared to controls.

1.2.1 Behavioral and physiological findings

Behavioral findings. A number of studies of alexithymia have examined behavioral measures such as accuracy and reaction time in order to establish whether alexithymia is associated with impairment in the processing of emotion (see Table 1). These studies provide strong evidence that the ability to label and respond to emotional stimuli is affected in individuals with alexithymia.

Turning first to experimental studies involving the labeling of facial expressions, Kano et al. (2003) found that alexithymic men are less accurate at identifying a range (mild, moderate and intense) of sad faces compared to non-alexithymic men. Parker et al. (1993) determined that alexithymic individuals are less proficient at identifying a range of facial expressions (distress, contempt, surprise, interest, shame, anger, fear, disgust and enjoyment) relative to individuals without alexithymia. Jessimer & Markham (1997) also found that alexithymic individuals are less able to recognize a range of emotional faces (happy, surprise, sad, fear, disgust anger), as did Panday & Mandel (1997).

Verbal and physical expressions of emotion are also inaccurate in individuals with alexithymia. For example, relative to controls, individuals with alexithymia have difficulty matching verbal descriptions of faces and situations to emotional images (Lane et al., 1996; Panday & Mandel, 1997) and give verbal descriptions of emotional experience that are inconsistent with emotional situations or physiological responses (Friedlander et al., 1997; Leweke et al., 2004; Martin & Pihl, 1985; Stone & Nielson, 2001; Papciak et al., 1995; Whemer et al., 1995). Related work has found that, relative to controls, alexithymic individuals are less adept at engaging in posed facial expressions (McDonald & Prkachin, 1989), but are able to accurately label posed facial expressions.

Studies suggest that reaction time to emotional information may also reflect the affective impairment of alexithymic individuals. As alexithymia scores increase, implicit presentation of angry faces fails to influence reaction time in response to congruent versus incongruent emotional words or faces (Vermeulen, Luminet & Corneille, 2006). Likewise, alexithymic participants take longer to color name emotionally arousing words (i.e., victim, AIDS) relative to healthy controls (Parker et al., 1993).

Physiological findings. It is well-established that emotional stimuli can evoke changes in a number of physiological measures. For example, in healthy individuals changes in heart rate and skin conductance are associated with the viewing of emotional pictures (Lang et al., 1993). Given the strong link between the presentation of emotional stimuli and physiological reactivity, a natural conclusion is that physiological reactivity to emotional stimuli should be disordered in alexithymia, since the behavioral findings indicate that the ability to label and respond to emotional stimuli is affected. However, such a link has not been established. Instead, the physiology research on alexithymia has yielded a set of inconsistent findings, as described below (see Table 2).

Surprisingly, none of the studies that have investigated physiological responses in alexithymia have used the type of emotion identification tasks that have been explored behaviorally. The closest point of comparison comes from a handful of studies (n=6) that have examined the physiological response to emotional stimuli without asking subjects to explicitly engage in emotion identification. These studies have found increases, decreases and non-significant changes in physiology. Some of this variability may reflect the complexity of the stimuli, which were emotionally arousing pictures (e.g., images of injury, accident or starvation) or segments of emotionally arousing films (e.g., a videotape of the movie *The Shining*). Differences

in the task context may also be an important factor. In most cases, participants were simply asked to view the presented stimuli (Roedema & Simons, 1999; Stone & Nielson, 2001; Whemer et al., 1995). In some cases participants also engaged in a stress situation involving a speech task and/or effortful mental task within the same experimental session (Franz et al., 2003; Friedlander et al., 1997; Infrasca, 1997). These studies may have been confounded by carryover effects of emotional stress or cognitive factors, such as attention.

In another set of studies (n=7), the physiological responses of alexithymic individuals and controls were monitored while participants experienced cognitive and affective stressors (e.g., they were asked to engage in speech tasks, mental arithmetic, a situation provoking anger) (Fukunishi, Sei, Morita & Rahe, 1999; Linden et al., 1996; Martin & Phil, 1986; Neumann et al., 2004; Newton & Contrada, 1994; Papciak et al., 1985; Rabavilas, 1987). The majority of these studies (n = 4) found evidence for increased physiological arousal in alexithymic individuals relative to nonalexithymic individuals either during preparation for a stress task (Fukunishi, Sei, Morita & Rahe, 1999) or throughout an experiment in which an affective stressor was presented (Martin & Phil, 1986; Papciak et al., 1985; Rabavilas, 1987). However, three studies found decreased physiological arousal in alexithymic individuals versus controls during a stressful encounter (Newton & Contrada, 1994; Neumann et al., 2004) or throughout an experiment in which an affective stressor was presented (Linden et al., 1996).

Some of the mixed findings may reflect factors that influence physiology beyond emotion. These factors include baseline arousal and cognitive effects. For example, a subset of studies have found that alexithymia participants exhibit increased baseline heart rate (Infrasa, 1997; Papciak et al., 1985; Stone & Neilson, 2001; Whemer et al., 1995) or task-independent hypoarousal (Linden, Lenz, & Stossel, 1996) as well as increased baseline measures of anxiety (Martin & Phil, 1986;

Newton & Contrada, 1994). Furthermore, tasks that incorporate emotional anticipation within the context of a cognitive stressor may be subject to cognitive confounds, since factors such as attentional load can affect physiological measures. For instance, attention during cognitive tasks has been associated with heart rate deceleration (Jennings, 1986).

Summary. Unfortunately, the literature on physiological findings in alexithymia does not clearly converge with prior behavioral findings and there is a clear need for further research. There are several factors that may account for discrepancies between the behavioral and physiological research. One factor in prior work is that the stimuli and task designs used in the behavioral research do not correspond to those used in the physiological research. A second factor is that even within the physiological research, many of the studies have used nonstandardized stimuli, and task designs involving both cognitive and emotional stressors. The issue of potentially confounding cognitive effects is important, because there is clear evidence that changes in attention can induce changes in physiological measures (Jennings, 1986). A third factor is that many of the physiological studies have (6/7) used an unreliable measure of alexithymia (Infrasca, 1997; Linden, Lenz, & Stossel, 1996; Martin & Phil, 1986; Neumann et al., 2004; Newton & Contrada, 1994; Papciak et al., 1985; Rabavilas, 1987). In Experiment 1, our goal is to measure both behavior and physiology in the same experimental paradigm, in order to determine whether individuals with alexithymia exhibit behavioral responses to emotional faces that differ from controls and whether there are corresponding differences in physiological measures.

1.2.2 A neuroscience perspective

In addition to behavioral and physiological studies of alexithymia, there have also been studies that have utilized functional neuroimaging (see Table 3). A review of these studies provides insight into the neural mechanisms that may underlie alexithymia, which will motivate the design of a second experiment. Thus far, five studies have been conducting using neuroimaging to examine differences between alexithymic individuals versus controls. Neuroimaging studies have examined changes in blood flow (n=1) and metabolic activity (n=2) during implicit, passive viewing of emotional pictures. All three studies found abnormal activity in the medial prefrontal cortex (MPFC), but not the amygdala in alexithymic versus control participants (Berthoz et al., 2002; Kano et al., 2003; Leweke et al., 2004). Two other studies examined changes in blood flow during explicit recall of emotional experiences (Huber et al., 2002; Mantani et al., 2005). Again, both studies found abnormal activity in the MPFC, but not the amygdala, in alexithymic versus healthy controls. The localization of aberrant neural responses in alexithymia to the MPFC, and not the amygdala, is interesting because of the different roles that these two regions are thought to play in emotional processing, as discussed below.

The amygdala. Our current knowledge of neural circuitry suggests that the amygdala, part of the central autonomic nervous system, is an important bridge between emotional processing, and behavioral and physiological responsivity. The amygdala receives information from the thalamus and cortex, and sends output to the hypothalamus and autonomic control centers in the brainstem. In animals, the amygdala has been studied most extensively using fearful stimuli and conditioning paradigms (Amaral, Price, Pitkanen, & Carmichael, 1992). Fear stimuli have direct, bottom-up, access to the amygdala. For example, during conditioning of footshock to a tone, the mere presentation of a fear cue (tone) is associated with a rapid subcortical response occurring

within 20 milliseconds of cue presentation (for review see LeDoux, 2000). In humans, passive viewing or gender identification of emotional images (such as faces showing a fearful expression) is sufficient to activate the amygdala (Hariri, Bookheimer, & Mazziotta, 1999; Hariri, Mattay, Tessitore, Fera, & Weinberger, 2003; Lange et al., 2003; Morris et al., 1996, 1998; Williams et al., 2001; Whalen et al., 2001).

The strong interconnections between the amygdala, hypothalamus, and brainstem autonomic control centers allow it to be centrally involved in the coordination of physiological responses to arousing stimuli and events. Human work has found support for this connection. Amygdala lesions impair skin conductance responses to emotive stimuli, and they alter the relationship between skin conductance responses and ratings of emotional stimuli (Glascher & Adolphs, 2003). Imaging studies have examined the correlation between blood flow in the amygdala and the physiological response during presentation of fear faces. The majority of studies have focused on heart rate and skin conductance. Heart rate is an end-organ measure that is under both sympathetic and parasympathetic nervous system control. In healthy participants, a strong correlation has been found between blood flow in the bilateral amygdala and heart rate during passive viewing of emotional faces (Critchley et al., 2005). Skin conductance response is an electrodermal technique that assesses sympathetic nervous system cholinergic responses in the skin. This technique provides additional evidence that, in healthy controls, the amygdala is correlated with autonomic responses during emotional tasks. For example, blood flow increases in the amygdala are associated with skin conductance responses when fear-related emotional material is presented during tasks that do not require explicit emotion labeling (Hariri et al., 2003; Williams et al., 2004a, b; 2005a, b).

The MPFC. In the existing human neuroimaging literature on emotion processing there are several prevailing theories about the role of the MPFC. One view is that the MPFC detects emotional information and uses this information to *engage* an appraisal process. By this account, other factors such as attentional load may also play an important role in modulating the appraisal process (Kalisch, Korenfeld, Stephan, Weiskopf, Seymour & Dolan, 2006; Keightly, Winocur, Graham, Mayberg, Hevenor & Grady, 2003). An alternative view is that the MPFC is involved in *implementing* top-down control. For example, investigators have described alexithymia as a problem involving the "cognitive comprehension of emotion" (Berthoz et al., 2002). According to this view the MPFC accounts for a failure to yield the appropriate response to emotional information. A difficulty *implementing* top-down control seems to be the prevailing theory in the alexithymia literature (Lane et al., 2007, see for review).

According to our view that alexithymia involves difficulty *implementing* top-down control we focus our review of top-down control on the modulation of emotion via MPFC. Modulation of emotion may occur in a top-down fashion via reciprocal projections from higher-level cortical areas to subcortical brain regions, especially the amygdala. Our best model of top-down control comes, once again, from the animal literature on fear. This literature has focused upon the MPFC, which in humans is thought to correspond to the anterior cingulate cortex (Brodmann's areas 24, 25, 32). In animals, the MPFC affects the extinction of a conditioned fear response, but not acquisition of a fear cue (LeDoux, Romanski & Xagoraris, 1989; Milad & Quirk, 2002; Morgan, Romanski, LeDoux, 1993).

Medial prefrontal modulation of emotional information can occur through a direct impact on neuronal firing in the amygdala. Stimulation of neurons in the MPFC inhibits the activity of amygdala neurons (Quirk, Likhtik, Pelletier & Pare, 2003). In human studies, explicit labeling of

fear faces is associated with increased blood flow in the MPFC and decreased blood flow in the amygdala (Hariri et al., 2000; Winston, O'Doherty, & Dolan, 2003; for ventral lateral prefrontal cortex see Lange et al., 2003; Leiberman et al., 2007). Thus, there appears to be a "push-pull" relationship between MPFC and amygdala responses (Beauregard, Levesque, & Bourgouin, 2003; Ochsner, Bunge, Gross, & Gabrieli, 2002).

Physiological research suggests that there are two potential pathways through which topdown physiological modulation can occur. One pathway appears to be a serial connection between the MPFC and amygdala. Through this pathway, MPFC can suppress the activity the amygdala, which in turn modulates amygdala control over physiological and behavioral responsivity. In a second pathway, the MPFC appears to be capable of influencing physiological responsivity independently from the amygdala (Frysztak & Neafesy, 1994; Powell et al., 1994, 1997). Tracing studies have found that the MPFC has direct projections to regions within the cardiovascular system (Frysztak & Neafesy, 1994; Hurley, Herbert, Moga, & Saper, 1991; Saper, 1982; Terreberry & Neafsey, 1983, 1987; Yasui et al., 1985; for reviews see Craig, 2002; Saper, 2002). To add to this complexity, different subregions of the MPFC may influence different physiological components, with ventral and dorsal subdivisions of the MPFC linked to sympathetic versus parasympathetic responses, respectively. Based on such findings, different measures of physiological arousal (heart rate and skin conductance) in humans may be tied to different aspects of top-down control; though limited data in this area makes strong predictions premature (Matthews, Paulus, Simmons, Neleson & Dimsdale, 2004)

Summary. Findings from the neuroscience literature provide a potential mechanism and model for examining alexithymia. One way to conceptualize the role of the amygdala in emotional reactivity is to posit that it plays a fundamental role in generating bottom-up responses

to emotional stimuli. That is, stimuli that have either innate or learned emotional significance can gain direct access to the amygdala, even when they are not within the current focus of attention. Engagement of the amygdala can in turn lead to the generation of physiological responses, which may help to prepare the body for an appropriate response and which may help to provide a basis for the conscious recognition and identification of an emotional state (e.g., by serving as a somatic marker) (Damasio, 1996). In contrast, the MPFC appears to play a fundamental role in *implementing* the top-down control of emotion – e.g., in appraising emotional stimuli and in regulating physiological responses through direct connections to autonomic control systems and indirect influence via the amygdala. The fact that amygdala activation appears similar in controls and individuals with alexithymia has been interpreted as bottom-up processing of emotional information is normal in alexithymia. On the other hand, the fact that MPFC activation appears to differ between controls and individuals with alexithymia has been interpreted as top-down control of emotional information may be disordered in alexithymia (Berthoz et al., 2002; Huber et al., 2002; Kano et al., 2003; Lane et al., 1997; Leweke et al., 2004; Mantani et al., 2005). In Experiment 2, we examine these ideas by measuring the behavioral and physiological changes associated the presentation of emotional stimuli in implicit and explicit task contexts.

1.2.3 The current study

The current work has two aims. The first aim is to determine whether individuals with alexithymia exhibit behavioral responses to emotional faces that differ from controls and whether there are corresponding group differences in physiological responsivity. To address this aim we measure behavioral and physiological responses during two explicit facial categorization tasks:

an emotion task involving the categorization of morphed emotional facial expressions, and an identity task involving the categorization of morphed facial identities. The second aim is to examine whether behavioral and physiological responses in alexithymia participants are differentially influenced by task context, as compared to controls. To address this aim, we evaluate responses to emotional and neutral facial expressions in two task contexts: in an explicit task context participants will label the emotion of the presented stimulus, while in an implicit task context participants will label the gender of the presented stimulus. Using these two experimental designs we will determine whether undergraduate participants who score high $(TAS \ge 61)$ or low $(TAS \le 51)$ on a measure of alexithymia (TAS-20) differ on multiple outcome measures; these include accuracy, reaction time, heart rate and skin conductance.

In both experiments, we seek to provide a mechanistic understanding of alexithymia by focusing on fear, an affective domain that elicits consistent, correlated physiological and neuroanatomical changes in both animals and humans. Surprisingly, no physiological or neuroimaging study has examined fear in alexithymia, even though it affords the best opportunity to form a clear link to past animal research and functional imaging studies in humans.

1.2.3.1 Predicted results, Experiment 1

The emotional categorization task requires the classification of a set of morphed fearful facial expressions displaying different levels of emotional intensity. The emotion categorization task allows us to extract two main behavioral measures: sensitivity to emotional faces (calculated as the category boundary of the categorical curve), and variability in emotional labeling near the category boundary (calculated as the slope of the categorical curve). Physiologically, the response to stimuli at end-points of the categorical continuum can be contrasted in order to

determine the physiological correlates of mostly emotional compared to mostly non-emotional faces. Group comparisons of category boundary, slope and end-points of emotion categorization thus allow us to assess potentially subtle alterations in emotion identification and physiological arousal in alexithymia versus control subjects. We make the following predictions about behavioral and physiological responses measures in our emotion categorization task:

- a) In our behavioral measures, we expect that the location and slope of the categorical boundary in our emotion identification task will differ for individuals with alexithymia because individuals with alexithymia appear to be less sensitive and more variable in their emotional responses, when compared to controls.
- b) In our physiological measures, at the end-points of the emotion categorization task we expect individuals with alexithymia to show blunting of the physiological response to mostly fearful versus mostly neutral faces, because of less sensitivity to emotions, when compared to controls.

For our facial identity task, we can also extract behavioral estimates of the category bound and slope, and the physiological responses to the faces at the end-points of the continuum can also be measured. For this task we predict similar behavioral and physiological responses in alexithymia and control groups, since there is no reason to believe that alexithymia is a problem that would make it difficult to identify faces that are non-emotional. We make the following predictions about behavioral and physiological responses in our identity categorization task:

a) In our behavioral measures, we predict that the category boundary and the slope of these measures will be similar across groups.

b) In our physiological measures, we predict that the end-points of the identity categorization continua will not be distinguished by physiological responses in alexithymia participants, when compared to controls.

Finally, for both of our categorization tasks we can hone in on the physiological response to highly ambiguous faces near the category boundary. These faces are least consistently labeled and, thus, difficult for participants to classify. An increase in attention should exert a nonemotional influence on physiological measures. For instance, prior research indicates that heart rate should decelerate during the categorization of ambiguous versus non-ambiguous stimuli (Klorman, Weisenfeld & Austin, 1975). For heart rate, increased attention is associated with heart rate deceleration. For SCR, an ambiguous versus non-ambiguous faces should increase the responses. Prior research found increases in SCR during recall of novel images or words that require attention. For SCR, increased attention is associated with an increase in SCR (Andreassi, 2007). For the identification task, similar physiological responses to ambiguous faces (heart rate deceleration, SCR increase) should be found in both subject groups, unless individuals with alexithymia have a top-down problem with physiological regulation that extends outside of the emotional domain. For the emotion categorization task, we may see group differences for ambiguous versus unambiguous faces because attention is present in a changing emotional context.

1.2.3.2 Predicted results, Experiment 2

We borrowed from neuroscience studies that engage the amygdala and MPFC in order to understand the potential behavioral and physiological mechanisms of alexithymia. We developed a task design in which we manipulate task instruction by asking participants to identify the emotion or gender of a fearful or neutral face. Our explicit task instruction asks

participants to direct their attention to the emotion of the face so that they can label emotion, while our implicit task instruction asks participants to direct their attention to the gender of the face so that they can label gender. This task design has been used by a variety of neuroscience research groups to uncover the biological correlates of implicit and explicit processing (Critchley et al., 2000; Gorno-Tempini et al., 2001; Keightly et al., 2003; Lieberman et al., 2007; Lange et al., 2003; Morris et al., 1996; Veilleumier et al., 2002; Winston et al., 2003). In this literature, implicit tasks have been viewed as weighted towards bottom-up processing, while explicit tasks have been viewed as weighted towards top-down processing. In alexithymia, the neuroimaging literature points to normal function in the amygdala, but not the MPFC in alexithymia versus control participants (Berthoz et al., 2002; Huber et al., 2002; Kano et al., 2003; Leweke et al., 2004; Mantani et al., 2005). These findings suggest bottom-up processing may be unaffected while top-down processing may be altered in alexithymia.

Based on the neuroimaging literature, if bottom-up processing is intact then behavioral markers attributed to bottom-up processing should also be intact. Fortunately, a large literature on implicit processing in healthy controls provides a solid foundation for the following specific predictions about our behavioral measures:

a) Gender identification (implicit): In our behavioral measures, similar to controls we predict slower reaction time to fearful versus neutral faces during the implicit task because emotion is distracting (Gorno-Tempini et al., 2001). Additionally, since MPFC, related to regulation of responses to emotional faces (Harriri et al., 2000; Lieberman et al., 1997), may be impaired in alexithymia we may find an exaggerated, even slower reaction time, to fearful faces versus neutral faces during implicit tasks, when compared to controls. We do not predict group differences in task accuracy since gender labeling is not emotional.

b) Emotion identification (explicit): In our behavioral measures, similar to controls we predict faster reaction time to fearful versus neutral faces during explicit tasks, since the emotional signal can facilitate responding (Gorno-Tempini et al., 2001). Since alexithymia participants may be less able to use appraisal mechanisms that take advantage of somatic markers, the magnitude of faster reaction time to fearful faces versus neutral faces may be less (blunted) and task accuracy may be poorer in alexithymia versus control subjects.

Based on the neuroimaging literature, if bottom-up processing is intact then physiological markers attributed to bottom-up processing should also be intact. Unfortunately, there is little literature on the physiological correlates of implicit and explicit processing in healthy controls. However, the same reasoning from our behavioral predictions can be brought to bear on our physiological predictions. If bottom-up processing is intact, we predict the following for heart rate:

a) Gender identification (implicit): In control subjects, the emotional content of the fearful versus neutral faces should be distracting, thereby increasing the amount of attention necessary to perform the task. In controls, increased attentional demands serve to slow heart rate, thereby opposing the faster heart rate that should be induced by exposure to fearful versus neutral faces. In alexithymia, if bottom-up processing is intact participants are also vulnerable to distraction. Therefore this group should also be susceptible to the opposing influence of heart rate slowing associated with increased attentional demands. In fact, since appraisal has been considered to be more difficult in alexithymia, individuals with alexithymia may be subject to greater increases in the attentional demands of the task, when compared to controls. This increased attentional demand of emotional faces should serve to slow heart rate more for alexithymia versus control participants (see Figure 1).

- b) Emotion identification (explicit): In control subjects, emotional content (e.g., a fearful face) should induce a faster heart rate. This physiological change may serve as a "somatic marker" that facilitates responding; thereby reducing the attentional demands that can produce heart rate slowing. For this task, the emotional and cognitive factors both predict a relative shift towards faster heart rate for fearful vs. neutral faces. In alexithymia, if bottom-up processing is intact then the heart rate should be faster for fearful vs. neutral faces. However, problems with top-down processing may interfere with the capacity to use heart rate as a "somatic marker" that reduces the attentional demands associated with labeling a fearful vs. a neutral face. In this case the amount of attention allocated to fearful and neutral faces may be about the same. Overall, the difference between heart rate responses to fearful vs. neutral faces may be smaller in individuals with alexithymia, compared to controls because the faster heart rate associated with the bottom-up signal is not complemented by a reduction in the heart rate slowing associated with labeling a fearful versus neutral face (see Figure 2).
- c) Considering the patterns of data across the two tasks (gender identification and emotion identification), we can make predictions about the direction (not the magnitude) of the effect of emotion and attention on heart rate. We predict that both control subjects and individuals with alexithymia will exhibit slower heart rate during gender identification (implicit) versus emotion identification (explicit) processing. The effect of both tasks for individuals with alexithymia may produce even slower heart rates than healthy controls, because problems with top-down control increase the attentional demands associated with emotion regulation in the gender identification task, and fail to

yield, or blunt, the attentional benefits associated with the processing of somatic markers in the emotion identification task.

If bottom-up processing is intact, we predict the following for SCR:

- a) Gender identification (implicit): For SCR the same principles apply to our predictions, but the literature on SCRs suggests that both emotion and attention are linked to SCR *increases*. In control subjects, the emotional content of the fearful versus neutral faces should be distracting thereby increasing the amount of attention necessary to perform the task. In controls, increased attention demands serve to increase the number of SCRs, thereby increasing SCRs that should be induced by exposure to fearful versus neutral faces. In alexithymia, if bottom-up processing is intact, participants are also vulnerable to distraction. Therefore, this group should also be susceptible to the influence of increased SCR associated with increased attentional demands. In fact, since appraisal has been considered to be more difficult in alexithymia, individuals with alexithymia may be subject to even greater increases in the attentional demands of the task, when compared to controls. This increased attentional demand of emotional faces should serve to increase SCRs more for alexithymia versus control participants.
- b) Emotion identification (explicit): For SCR the same principles apply to our predictions, but the literature on SCRs suggests that both emotion and attention are linked to SCR *increases*. In control subjects, emotional content (e.g., a fearful face) should induce an increase in the number of SCRs. This physiological change may serve as a "somatic marker" that facilitates responding; thereby reducing the attentional demands associated with an increase in the number of SCRs. For this task, the emotional factor predicts a relative decrease in SCR for fearful vs. neutral faces for controls. In

alexithymia, if bottom-up processing is intact then the number of SCRs should increase for fearful vs. neutral faces. However, problems with top-down processing may interfere with the capacity to use SCR as a "somatic marker" that reduces the attentional demands associated with labeling a fearful vs. a neutral face. In this case the amount of attention allocated to fearful and neutral faces may be about the same. Overall, the difference between SCR responses to fearful vs. neutral faces may be larger in individuals with alexithymia, compared to controls because the increase in SCRs associated with the bottom-up signal is not complemented by a reduction in SCRs associated with labeling a fearful versus neutral face.

c) For SCR, we predict that both control subjects and individuals with alexithymia will exhibit increased SCRs during gender identification (implicit) versus emotion identification (explicit) processing. The effect of both tasks for individuals with alexithymia may produce an even greater number of SCRs than healthy controls, because problems with top-down control increase the attentional demands associated emotion regulation in the gender identification task, and fail to yield, or blunt, the attentional benefits associated with the processing of somatic markers in the emotion identification task.

2.0 METHOD

2.1 PARTICIPANTS

We screened 728 undergraduate students (18-28) participating in the psychology subject pool at the University of Pittsburgh. Psychology undergraduates provided written consent and completed a set of questionnaires that included the Toronto Alexithymia Scale 20-item, NEO PI-R Neuroticism and Extraversion scale (Costa & McCrae, 1992), Spielberger Trait Anxiety Inventory (Spielberger, Gorsuch, & Lushene, 1983), and handedness questionnaire (Annett, 1970). The NEO PI-R and STAI were administered in order to establish that behavioral and physiological responses are associated with alexithymia, rather than comorbid anxiety or vulnerability to psychopathology. Participants were awarded course credit for completing these study questionnaires.

We recruited a sample of 62 participants (30 controls, 32 alexithymia) from the undergraduates screened who met our inclusion/exclusion criteria. Participants met the following inclusion criteria at screening: 1) TAS-20 score was greater than or equal to 61 (alexithymia) or less than or equal to 51 (no alexithymia), and 2) right-handed. These participants were contacted by telephone and asked to provide verbal consent for their participation in a brief phone screen. The phone screen was used to determine whether participants met the following exclusion criteria: 1) current self-reported anxiety disorder, major depressive disorder, mania, attention-

deficit/hyperactivity disorder, psychosis or significant drug and/or alcohol use 2) self-reported history of anxiety disorder, major depressive disorder, mania, attention-deficit/hyperactivity disorder, psychosis or significant drug and/or alcohol use, 3) current and past use of psychotropic medication or medication that would affect cardiac function, and 4) self-reported history of hypertension, cardiovascular, or pulmonary disease. If exclusion criteria were met a study visit was arranged. If participants could be reached by e-mail only, they provided verbal consent for screening at the time of their study appointment. Symptom information and diagnoses for all participants was not confirmed by SCID diagnosis or relatives of the participant making this a limitation of the screening procedure.

Our strict set of exclusion criteria and our access to participant volunteers for the physiological task during the late spring and summer terms at the University of Pittsburgh made it necessary to relax our exclusion criteria. Seven participants total met our criteria. We considered six of these participants to be index cases given the prevalence of depression, anxiety and substance use in the college age population, and therefore decided to include them in our study (Dawson, Fredrick & Chou, 2005; Garlow et al., 2007; Schwartz, 2006). Three participants had been told by a professional that they had depression in the past. One participant reported an anxiety disorder diagnosis by a professional and was receiving medication treatment at the time of the study. Two participants had past alcohol/drug use that was a problem but neither endorsed current drug or alcohol problems. The seventh participant was judged to have met the initial intent of our exclusion criteria, which was to avoid cases of considerable psychopathology. This alexithymia participant was excluded from the study because of self-reported history of depression diagnosis in addition to current depression symptoms and current thoughts of suicide; the patient also had a history of medication treatment. This participant was

able to contract for safety and was given a referral for outpatient treatment as well as the phone contact for the emergency room at Western Psychiatric Institute and Clinic.

As a second level of recruitment and analysis we attempted to match a subset of alexithymia and control participants for Neuroticism, Extraversion, Anxiety (STAI) and gender in order to account for the association between alexithymia and other personality traits.

At study visit, the eligible participants consented to enroll in the physiological study. We enrolled sixty-one study participants (30 controls, 31 alexithymia). Four participants were excluded from the study during the physiology session. One participant was excluded because their TAS score was not at a criterion score of greater than or equal to 61; one participant was excluded because of mitral valve prolapse; one participant was excluded for oppositional behavior during the physiology experiment; one subject was excluded because during physiology recording they appeared to have a pattern of heart beats that was consistent with a "wandering p-wave". This participant was informed about this pattern and was encouraged to have this examined by their physician.

Participants withdrew from caffeine for 12 hours and alcohol for 24 hours prior to their testing session. Enrolled participants were paid \$20 for their participation in the study. The Institutional Review Board at the University of Pittsburgh reviewed this research.

2.2 DESIGN

Following enrollment participants engaged in a two-part experimental study session. During Experiment 1, participants were administered an identity categorization task and an emotion categorization task. In the identity and emotion tasks participants categorized a set of morphed

facial stimuli with different mixtures of two facial identities [identity A (George Clooney or Queen Elizabeth), identity B (David Schwimmer or Hillary Clinton)] or two facial expressions [expression A (neutral), expression B (fearful)]. We used a 3 x 2 design with face category (mostly face A stimuli [0%-20%], face A/B stimuli [30%-70%], mostly face B stimuli [80%-100%]) and group (alexithymia, control) as factors. Accuracy was the behavioral dependent measure for this experiment. Heart rate and skin conductance were the physiological dependent measures. During Experiment 2, we instructed participants to label the emotion of a neutral or fearful face (emotion identification), or to label the gender of a neutral or fearful face (gender identification). We used a 2 x 2 design with image type (neutral, fearful) and task (gender identification, emotion identification) as factors. Accuracy and reaction time were the behavioral dependent measures for this experiment. Heart rate and skin conductance were the physiological dependent measures.

2.3 PROCEDURE

At the start of the study session each participant was seated in a sound-attenuated temperature controlled room. Participants completed the state component of the Spielberger State Anxiety Inventory (SSAI) and then participated in a 7 minute baseline period of physiological assessment. During this time participants were seated in front of the computer and asked to relax and remain awake while their heart rate and skin conductance were recorded. Following the baseline period, participants spent approximately 60 minutes completing the experimental tasks (Experiment 1 and Experiment 2). Recovery data were collected for 7 minutes following the experimental tasks. During this time participants were seated in front of the computer and asked to relax and remain awake. Following the recovery period, participants were asked to once again complete the state

measure of the SSAI. Throughout the study session participants were instructed to remain as still as possible in order to minimize movement artifact. Physiological measures were acquired throughout the baseline, experimental task, and recovery periods.

2.3.1 Experimental tasks

2.3.1.1 Experiment 1: Categorical Processing

To examine categorical processing of facial identities (identity categorization task), participants viewed static images of famous faces (Rotshtein, Henson, Treves, Driver & Dolan, 2005). The identity task allowed us to attribute findings in our emotion categorization task to the specific effects of emotion responsivity rather than cognitive task effects (e.g., arousal due to difficulty). For this task, the identity of a face varied, but the intensity of the emotional expression remained constant. First, participants viewed original images of famous faces and were asked to indicate how well they know the face (i.e., I don't recognize the face at all; the face looks familiar) and to name the face, if possible. If the participant could not name the face they were told the identity of the face followed by a 60 second exposure to the face. This allowed participants to become familiar with the prototypical face that they then categorized.

For the identity categorization task participants viewed static faces of an original image of one face that morphed to an original image of a similar face. Specifically, we used an original image of Queen Elizabeth that morphs into an original image of Hilary Clinton and original image of George Clooney that morphs into an original image of David Shwimmer (Rotshtein et al. 2005). The two sets of identity morphs were presented at 10% intervals (11 levels) and each image was presented twice (n = 22 images) (Rotshtein et al., 2005). Each image was presented for 2 seconds. A fixation cross was presented during the interstimulus interval, which were jittered (10-12

seconds) to address concerns about habituation effects (Lawrence et al., 2004; Surguladze et al., 2005). Following the fixation cross subjects made a forced-choice identity response (i.e. Queen Elizabeth or Hilary Clinton).

To examine categorical processing of emotional faces (emotion categorization task), participants viewed static discrete or blended images of neutral or fearful faces (Ekman & Friesen, 1976; Young et al., 2002) on a computer monitor screen and were required to determine the emotion (neutral, fearful) of each face by pressing one of two designated response buttons on the computer keyboard.

Each stimulus was comprised of different expressions created by morphing a neutral and a fearful face (Russell et al., 2007). The identity of the face remained constant, but the intensity of the expression varied. The emotion morphs were presented at 10% intervals (11 levels) of emotional intensity. We presented two different facial identities (1 male, 1 female). The 22 images were randomly interleaved. Each image was presented for 2 seconds. A fixation cross was presented during the interstimulus interval, which was jittered (10-12 seconds) to address concerns about habituation effects (Lawrence et al., 2004; Surguladze et al., 2005).

2.3.1.2 Experiment 2: Task-dependent emotion processing

During Experiment 2, we manipulated task context by asking participants to label the emotion of a neutral or fearful face (emotion identification), or to label the gender of a neutral or fearful face (gender identification). For the emotion identification task, participants viewed static images of neutral or fearful faces (Ekman & Friesen, 1976; Young, Perrett, Calder, Sprengelmeyer, & Ekman, 2002; Tottenham, Borscheid, Ellertsen, Marcus, & Nelson, 2002) on a computer monitor screen and were required to determine the emotion (neutral, fearful) of each face by pressing one

of two designated response buttons on the computer keyboard. For the gender identification task, participants viewed a different set of static images of neutral or fearful faces on a computer monitor screen and were required to determine the gender (male, female) of each face by pressing one of two designated response buttons on the computer keyboard. For each participant, images were randomly assigned to either the emotion identification task or the gender identification task condition.

During the experiments a total of 60 validated facial expressions were presented. Sixty different facial identities with neutral (n=30; 15 female, 15 male) or fearful (n=30; 14 female, 16 male) expressions were used (Eckman & Friesen, 1976; Gur et al., 2002; Tottenham et al., in press). Each face was presented one at a time. Fearful faces were randomly interleaved with neutral faces. The gender identification task consisted of three blocks. Each block was composed of 30 randomly presented images that were displayed on the computer screen for 2 seconds. A fixation cross was presented during the interstimulus interval, which was jittered (10-12 seconds) to address concerns about habituation effects (Lawrence et al., 2004; Surguladze et al., 2005). Following the jittered interstimulus interval the participant was *prompted* to respond on the keyboard to the face stimulus. This response delay was necessary in order to control for the potential effects of movement on changes in heart rate and skin conductance. At the end of the block of gender labeling of facial expressions, there was a break, in which instructions for the emotion identification task was given. The emotion identification task also consisted of three blocks. Each block was composed of 30 randomly presented images that were displayed on the computer screen for 2 seconds. A fixation cross was presented during the interstimulus interval, which was jittered (10-12 seconds) to address concerns about habituation effects (Lawrence et al., 2004; Surguladze et al., 2005). Following the jittered interstimulus interval the participant was *prompted* to respond on the keyboard to the face stimulus.

In order to avoid effects of movement on heart rate and skin conductance in Experiment 2, the *immediate responses* to facial expressions, were acquired in a separate paradigm that followed the same design. For this "reaction time" paradigm a total of forty validated facial expressions were presented (Eckman & Friesen, 1976; Gur et al., 2002; Tottenham et al., in press). Forty different identities with neutral (n= 20; 9 female, 10 male) or fearful (n= 20; 10 female, 11 male) expressions were used. Each face was presented one at a time. Fearful faces were randomly interleaved with neutral facial expressions. The gender identification task consisted of three blocks. Each block was composed of either 21 or 19 randomly presented images that were displayed on the computer screen for 2 seconds. Participants were instructed to respond immediately to the face stimulus. A fixation cross was presented during the interstimulus interval, which was jittered (10-12 seconds) to address concerns about habituation effects (Lawrence et al., 2004; Surguladze et al., 2005). At the end of the block of gender labeling of facial expressions, there was a break, in which instructions for the emotion identification task was given. The emotion identification task also consisted of three blocks. Each block was composed of either 21 or 19 randomly presented images that were displayed on the computer screen for 2 seconds. Participants were instructed to respond immediately to the face stimulus. A fixation cross was presented during the interstimulus interval, which was jittered (10-12 seconds) to address concerns about habituation effects (Lawrence et al., 2004; Surguladze et al., 2005). This reaction time paradigm was presented either before or after the original paradigm for Experiment 2 in which participants were prompted to respond to each face.

2.3.2 Measures

2.3.2.1 Behavioral Measures:

During Experiment 1 and Experiment 2 participants responded to each face by pressing one of two designated response buttons on the computer keyboard. Keyed responses along with accuracy and reaction time of each response were collected and recorded using E-prime software (Psychological Software Tools, Inc.).

2.3.2.2 Physiological Measures

Heart Rate. An initial exfoliation was performed at three locations on the chest using alcohol swabs and ECG skin prep. Each participant was then fitted with adhesive electrodes in a three-lead configuration. Throughout the study, ECG was recorded using Grass Model 12 DC and AC amplifiers. The digitized (12 bit) signal was sampled at 1000 Hz. Data collection and analysis was conducted with a PC computer-based system (Mindware Technologies Ltd., Gahanna, OH). Heart rate was measured by acquiring resting and phasic changes in beat-to-beat interval. Our predictions focused on differences in inter-beat interval rather than the high and low frequency components of the ECG signal. We used the peak of the ECG R-wave in order to determine inter-beat interval. Data artifacts were assessed using computer editing and were then visually inspected and hand corrected. Missed beats were subdivided. In few instances, movement artifact was confounded with detection of R-waves and the relevant trial was eliminated.

Skin Conductance. An initial exfoliation was performed with soap and water on the surface of the palm of the non-dominant, left hand. Electrodermal Ag-AgCl electrodes designed for placement on the palm of the hand were filled with conductive paste and secured to the palm with electrode collars. Skin conductance was recorded using Grass Model 12 DC and AC amplifiers.

Grass amplifier was set at TC.8 so in order to examine phasic changes in SCR. A low pass filter was set at 35 Hz in order to eliminate frequencies above this physiological range. Readings of skin conductance were sampled at 1000 Hz and stored digitally on computer. Data collection and analysis was conducted using a PC computer-based system (Mindware Technologies Ltd., Gahanna, OH). The maximal SCR change in the period 1–3 s following a face-presentation was assigned as the event-related SCR to that face.

Outliers. We examined our behavioral and physiological data for potential outliers. Outlier criteria for behavioral and physiological measures were the following: 1) a standard deviation \geq 3.29 and, 2) a Cook's D greater than 4/n-k-1 (n = sample size; k = number of predictors) (Tabachnick & Fidell, 2001; Weinberg & Abromowitz, 2002,).

2.3.2.3 Psychological Measures

Toronto Alexithymia Scale 20-item (Bagby et al., 1994). The TAS-20 was administered at screening. Each questionnaire consists of 20 items on a one to five point Likert scale. The TAS-20 measures difficulty identifying feelings, difficult describing feelings, externally oriented thinking and also yields a total score. The total minimum score on this scale is 0 indicating no alexithymia and the maximum score is 100, indicating high levels of alexithymia. Established cut-off scores are: \geq 61 alexithymic, 52-60 intermediate, and \leq 51 non-alexithymic. It took approximately 5-10 minutes to complete this questionnaire (Bagby et al., 1994a).

The Spielberger State-Trait Anxiety Inventory (Spielberger et al. 1983). The Spielberger Trait Anxiety Inventory (STAI) was administered at screening. The Spielberger State Anxiety Inventory (SSAI) was administered prior to and following the experiment. Each questionnaire consisted of 20 items and measured current anxiety levels on a one to four point Likert scale. The total minimum score on this scale is 20 indicating no / low levels of anxiety; the maximum score

of 80 indicates high levels of anxiety. It took a total of approximately 5 minutes to complete the each assessment. For our second level recruitment and analysis of matched pairs, scores on the STAI were used as selection criteria.

The NEO-PI R Neuroticism and Extraversion Scale (Costa & McCrae, 1992). The NEO-PI R Neuroticism and Extraversion Scales were administered at screening. The NEO-PI R assesses five stable personality traits, including Neuroticism and Extraversion. Neuroticism is a tendency towards negative emotions such as sadness, fear and guilt. High scores on Neuroticism are associated with anxiety, depression and alexithymia (Trull & Sher, 1994; Bagby et al., 1994a). Individuals who score low on Neuroticism are considered calm and less reactive to everyday situations. The Neuroticism scale consists of 48 items, eight items for each of six domains: anxiety, anger hostility, depression, self-consciousness, impulsiveness and vulnerability.

Extraversion is a tendency towards being sociable, assertive, active and talkative. High scores on Extraversion are associated with these characteristics. Low scores are associated with introversion. Introversion is a tendency towards being reserved and independent. The Extraversion scale consists of 48 items, eight items for each of six domains: warmth, gregariousness, assertiveness, activity, excitement-seeking and positive emotions.

Self-reported responses on the NEO-PI R are made on a five-point Likert scale ranging from "strongly disagree" to "strongly agree". Scores on both scales are classified as very low, low, average, high and very high. Thirty-eight percent of individuals score in the average rage, 24% score in the high and low range, and 7% score in the very high and very low range on the NEO-PI R.

3.0 RESULTS

3.1 PARTICIPANTS

Among the 728 subject pool participants who were assessed, 532 were identified who met our initial screening criteria: 1) TAS \leq 51 (control) or TAS \geq 61 (alexithymia) and, 2) right handed. Twelve percent of the population were identified as alexithymic (64/532, 12%), which is within the range of past alexithymia studies (Kokkonen et al., 2001; Salminen et al., 1998; Mason, Tyson, Jones & Potts, 2005).

From our larger group of subject pool participants (n = 532) we identified a smaller study sample. One important consideration is whether our study sample is representative of the larger population. The following is a set of analyses conducted in order to determine whether our study sample of control and alexithymia participants is representative of the subject pool control and alexithymia participants. Our variables of interest were alexithymia, trait anxiety, extraversion, neuroticism and gender. We also determined study sample group differences on these measures.

First, we compared subject pool control participants not included in our study (n = 427) to our study sample of control participants (n = 28). We did not find group differences on TAS-20, trait anxiety, extraversion (t (453) < -1.20, p > .23) and gender (X^2 (1, N = 452) = .371, p = .54). In general, our control study sample seems to be representative of the subject pool with

the exception of differences in anxiety and neuroticism (t (453) = -2.48, p = .00). This is expected because we attempted to select for controls who would match our alexithymia group.

Turning to the alexithymia group, we compared subject pool alexithymia participants not included in our study (n = 34) to our study sample of alexithymia participants (n = 29). Subject pool alexithymia participants did not differ significantly from study alexithymia participants on TAS-20, trait anxiety, neuroticism and extraversion (t (62) >-1.25, p > .22). On gender, we found significant differences between subject alexithymia pool participants and our study sample of alexithymia participants ($X^2(1, N = 62) = 5.36$, p = .02). Our study sample of participants had more females compared to males, while the subject pool had two fewer females compared to males.

Next, we assessed group differences between subject pool control (n = 427) and alexithymia participants (n = 34) who were not selected for our study. We found group differences on TAS-20, trait anxiety, neuroticism and extraversion (t (459) > - 20.61, p = .00; alexithymia > control), but not gender (X^2 (1, N = 453) = 1.63, p = .20).

Finally, in our study sample of alexithymia (n = 29) and control (n = 28) participants we again conducted group comparisons on our variables of interest (see Table 4 for means/Std err.). We did not find significant between group differences in extraversion t (55) = 1.40, p = .17) or gender (X^2 1, N = 57) = 1.51, p = .22). We found significant group differences between TAS-20 (t (55) = -16.04, p = .00). The average TAS-20 score of our alexithymia and control group is similar to a subset of past study participants for whom TAS-20 scores have been reported (see Table 5). On the TAS-20 our alexithymia participants most often endorsed symptoms of difficulty identifying feelings (19.3 +/-7.2) and externally oriented thinking (19.5 +/- 4.6) when compared to difficulty describing feelings (14.8 +/- .7) (t (56) = -5.13, p = .00). In our study we

also found group differences in trait anxiety (t (54) = -4.39, p = .00) and neuroticism (t (55) = -2.89, p = .01). However, the degree of a group difference in anxiety and neuroticism is minimized in our study sample. To further account for the possible effects of anxiety and neuroticism traits we matched a subset of our study participants on these measures (see Table 6 for means/Std err.).

3.1.1 Baseline

3.1.1.1 State Anxiety

We assessed state anxiety prior to and following our two experiments using the Spielberger State Anxiety Scale. We were interested in determining whether our control and alexithymia study groups differ on measures of state anxiety from pre to post treatment. We conducted an ANOVA with anxiety (pre, post) x group (control (n=25), alexithymia (n=29) as factors and state anxiety score as our outcome measure. We were missing anxiety measures on four control participants.

We found a significant main effect of anxiety, in which anxiety declined in both groups from pre to post treatment (anxiety: F (1, 52) = 21.88, p = .00). We also found a trend towards a significant anxiety x group interaction (F (1, 52) = 3.83, p = .06), in which the alexithymia group experienced a greater decline in anxiety (t (52) = -3.82, p = .00) compared to the control group (t (52) = -2.37, p = .07). We found a significant group effect in which the alexithymia group endorsed more state anxiety symptoms compared to controls (F (1, 52) = 11.41, p = .00).

3.1.1.2 Heart Rate:

We examined group differences in heart rate at baseline for our control and alexithymia groups. We did not find significant group differences (t (54) = 1.38, p = .17). Baseline heart rate was not

correlated with ratings of pre-experimental state anxiety (r (56) = .155, p = .28). Also, we did not find a significant relationship between baseline heart rate measures and heart rate reactivity in Experiment 1 or Experiment 2 (r (56) > .263, p > .05) suggesting that our data does not violate the law of initial values. The law of initial values indicates that heart rate reactivity depends on the baseline level of heart rate. For example, a higher baseline heart rate has been associated with reduced reactivity (ceiling effect) as well as a greater opportunity for increased reactivity (Andreassi , 2007).

3.1.1.3 Skin Conductance

We examined group differences in the proportion of SCRs at baseline for our control versus alexithymia groups. We did not find significant group differences (t (54) = -.625, p = .53). Baseline SCRs were not correlated with ratings of pre-experiment state anxiety (r (51) = -.192, p = .18). The literature suggests that the law of initial values does not apply to measures of SCR (Andreassi , 2007).

3.2 EXPERIMENT 1: CATEGORICAL PROCESSING

3.2.1 Behavioral Findings

Hypothesis: In alexithymia compared to control participants, we predict that during emotion categorization behavioral responses to emotional faces will produce a slope that is less steep and more variable because of less sensitivity to emotion. During the identity task, we predict that the

category boundary and the slope of these measures will be similar across groups because this is a non-emotional task.

We first examined whether participants categorize a set of morphed facial stimuli with different mixtures of two facial expressions (neutral/fear) or two facial identities (identity A, identity B) (see Figure 3).

Across subject groups, the general pattern was an s-shaped plot, as expected when stimuli can be perceived categorically. In the identity task, faces that contained a high percentage of face A stimuli (i.e., David Schwimmer) were classified as face A and faces that contained a high percentage of face B stimuli (i.e., George Clooney) were classified as face B. Faces that appeared to be an even mixture of face A/B stimuli were sometimes classified as face A (i.e., David Schwimmer) and sometimes classified as face B (i.e., George Clooney). In the emotion task, faces that appeared to contain a high percentage of face A stimuli (neutral expression), were classified as neutral and faces that appeared to contain a high percentage of face B stimuli (fearful expression), were classified as fearful. Faces that appeared to be an even mixture of face A/B stimuli, (neutral/fearful), were sometimes classified as neutral and sometimes classified as fearful.

To assess group differences in categorical processing of identity and emotion, participants' categorical responses were modeled using a logistic regression function that allowed us to estimate the slope and category boundary of the transition between face A and face B. For each participant we determined the best fitting slope (- intercept/beta) and category boundary (beta). We then used an independent samples t-test to examine group differences in slope and category boundary for the identity and emotion tasks. We found no group differences

in category boundary (t (54) < 1.40, p > .17) or slope (t (55) < .590, p > .56) for either the identity or the emotion tasks.

Overall, these results suggest that alexithymia and control participants do not differ in how they categorize a set of morphed faces and emotional identities. In our matched groups we found a similar pattern of results for both tasks (see Appendix A). This finding is surprising since the alexithymia literature suggests that alexithymia participants should have difficulty labeling emotions.

3.2.2 Heart Rate

Hypothesis: Using a categorical task we predict that we can evoke physiological responses during a behavioral paradigm. For the identification task, similar physiological responses to ambiguous faces (heart rate deceleration, SCR increase) should be found in both subject groups, unless individuals with alexithymia have a top-down problem with physiological regulation that extends outside of the emotional domain. For the emotion categorization task, we may see group differences for ambiguous versus unambiguous faces because attention is present in a changing emotional context.

We were interested in whether the physiological signal in response to the categorical labeling of identity and emotion in alexithymia is the same as that of controls. We used inter beat interval (IBI) as our measure of the heart rate response to the presentation of faces. This measure was continuously acquired throughout the experiment, and so in order to focus our analysis on the critical issue of physiological reactivity in alexithymia we took a number of data reduction steps both within and between trials. We were particularly interested in capturing the early vagal response, a parasympathetic response that is thought to reflect attention. The vagal

response can occur as early as within one beat of stimulus presentation (Bernston, Quigley & Lozano, 2007). Therefore, we chose to implement a reactivity measure in which, for each of our stimuli, we computed the average mean difference between the three IBIs following stimulus presentation (IBI 4,5,6) and the two IBIs preceding the stimulus presentation (IBI 1,2). This time window best captured the baseline data from all participants as well as their reactivity to facial expressions (Critchley et al., 2005). We reason that this measure should capture stimulus-specific changes in heart rate to the onset of facial expressions, and thus serve as an informative measure of physiological reactivity (see Figure 4).

One limitation of our study design is that we had only two trials of each experimental condition. In order to gain statistical power we decided to take advantage of the categorical perception of our face stimuli. Similar to past research, we reasoned that we could collapse across trials in which the stimuli were similarly categorized by participants (Kircher et al., 2001). Specifically, we conducted data averaging across trials involving stimuli at the far endpoints of the identity and emotion continua, where faces contained a high percentage of face A stimuli (100/0, 90/10, 80/20) or a high percentage of face B stimuli (0/100, 10/90, 20/80). We also averaged across "ambiguous" trials that fell near the boundary of the identity and emotion continua, where participants were inconsistent in how they labeled faces with a more even mixture of face A stimuli and face B stimuli (70/30, 60/40,50/50,40/60, 30/70). This created three stimulus levels for the identity and emotion tasks (mostly face A, mostly face B, ambiguous faces).

Our dependent measure of reactivity was submitted to an ANOVA with group (control, alexithymia) and stimulus type (mostly face A, mostly face B, face A/B) as factors. In the identity task analysis we excluded one participant who met outlier criteria. Data from two

additional participants were not included because of technical problems. None of the participants in the emotion task analysis were outliers and there were no technical problems.

In the identity task we found a main effect of stimulus type (F (2, 54) = 27.31, p = .00). We conducted follow-up t-tests to further asses these stimulus effects. We found that IBI was significantly increased (slowed heart rate) for the ambiguous (face A/B) as compared to the non-ambiguous (mostly face A, mostly face B) stimulus conditions (t (55) > 6.11, p = .00), whereas the two non-ambiguous stimulus conditions were not significantly different from each other (t (55) = -.18, p = .86). Our ANOVA did not yield significant group differences (F (1, 54) = .04, p = .85) or a significant stimulus type x group interaction (F (2, 54) = 2.13, p = .12). These results provide evidence that alexithymic individuals exhibit the normal heart rate deceleration in response to cognitive tasks requiring attention (Jennings, 1986). In our matched groups we found a similar pattern of results for the identity task (see Appendix A).

In the emotion task we also found a main effect of stimulus type (F (2, 56) = 3.47, p = .03). In follow-up t-tests, we found that IBI was significantly increased (slowed heart rate) for the ambiguous (face A/B) as compared to the non-ambiguous, mostly face B stimulus condition (t (57) = 2.82, p .01). Unlike the identity task, no difference was found for the ambiguous (face A/B) compared to the non-ambiguous, mostly face A stimulus condition (t (57) = 1.04, p = .30). The two non-ambiguous stimulus conditions were not significantly different from each other (t (57) = 1.42, p = .16). Our ANOVA for the emotion task did not reveal a significant effect of group (F (1, 56) = 1.85, p = .18) or a significant stimulus type x group interaction, (F (2, 56) = 2.19, p = .12). We believe this pattern of stimulus effects reflects opposing effects of cognitive and emotional influences on heart rate (see Figure 5). As noted above, the increasing difficulty of categorizing ambiguous faces may serve to slow heart rate in response to ambiguous stimuli. On

the other hand, in the emotion task, the degree of emotional expression that increases as stimuli progress from mostly face A stimuli to mostly face B stimuli should serve to speed up heart rate.

Since our data suggest that physiological reactivity in the emotion task can be a mixture of cognitive and emotional influences, we conducted further analyses in which we narrowed in on heart rate reactivity to mostly face A stimuli versus mostly face B stimuli in control and alexithymia participants through a secondary ANOVA with stimulus type (mostly face A, mostly face B) and group (control, alexithymia) as factors. We reasoned that the end-points (mostly face A, mostly face B) of the emotion continuum are cognitively equivalent in difficulty, but emotionally non-equivalent. We found a trend towards a significant stimulus type x group interaction (F (1, 56) = 3.38, p = .07), with a trend towards faster heart rate for face B stimuli compared to face A stimuli for the control group (t (28) = 1.93, p = .06) but not the alexithymia group (t (28) = -.173, p = .71). In our matched groups we found a similar pattern of results for the emotion task (see Appendix A).

Overall, we found our expected task effects. For both groups, during our identity task we found heart rate slowing during labeling of non-emotional ambiguous faces that require attention. For controls, during our emotion task we found opposing effects of emotion and attention in which heart rate becomes slower in response to increased attention to ambiguous faces and becomes faster in response to increases in emotional intensity. The intensity of this response in our sample of healthy controls was similar to the attentional response in a sample healthy controls that participated in a similar emotion paradigm (Daneree et al., 2006). In alexithymia participants, during our emotion task we found the normal heart rate slowing related to an increase in attention, but a pattern in which heart rate does not become faster with an increase in emotional intensity (see Figure 5). These patterns appear to be consistent with the

hypothesis that alexithymia and control groups differ on emotional tasks. In our matched groups we found a similar pattern of results for both the identity and the emotion tasks, suggesting that these effects may not be explained by differences in anxiety and neuroticism (see Appendix A).

3.2.3 Skin Conductance

Hypothesis: We hypothesize that our SCR effects will be similar to those found with heart rate, since increases in SCR and faster heart rate have been associated with the amygdala response to fearful faces (Hariri et al., 2003; Williams et al., 2004a,b; 2005a,b). In contrast to heart rate deceleration, attention may increase SCR responses (for review see Andreassi, 2007).

Our measure of the skin conductance response (SCR) to categorical processing was continuously acquired throughout the experiment. In order to focus our analysis on the critical issue of physiological reactivity in alexithymia we took a number of data reduction steps both within and between trials. We were particularly interested in capturing the response to the presentation of each face stimulus. We began by determining, for each trial, whether an SCR occurred. This was done using a standard amplitude criterion threshold of .05 microsiemens in a 1-3 second time window (Dawson, Schell & Fillion, 2007). Since a large number of participants did not consistently exhibit SCRs (14/58) we did not proceed with SCR amplitude as our outcome measure and instead implemented a count-based outcome measure. For each participant, our count-based measure was calculated as the percentage of trials with an SCR for each of our facial expression conditions. We reason that count-based reactivity should capture SCR linked to the onset of facial expressions and serve as our single dependent measure of physiological reactivity.

As with our heart rate data, we took advantage of the categorical perception of face stimuli (face A, face B, face A/B). Specifically, we collapsed across trials at the far end-points (mostly face A, mostly face B) and near the middle (face A/B) of the identity and emotion continua for each subject. This created three stimulus levels for the identity and emotion task (mostly face A, mostly face B, face A/B). Our dependent measure, percent of SCRs, was submitted to an ANOVA with group (control, alexithymia) and stimulus type (mostly face A, mostly face B, face A/B) as factors. In the identity task, none of the participants were outliers and there were no technical problems. In the emotion task analysis, we excluded one participant who met outlier criteria. Three participants were excluded for technical problems.

In the identity task we did not find an effect of stimulus type (F (2, 55) = 2.28, p = .11). However, follow-up t-tests revealed a significantly larger percent of SCRs during labeling of ambiguous stimuli (face A/B) as compared to the non-ambiguous (mostly face B) stimulus conditions (t (56) > -2.23, p = .03), and a trend for ambiguous stimuli (face A/B) as compared to the non-ambiguous (mostly face A) stimulus condition (t (56) = -1.72, p = .09). The percent of SCRs did not differ for mostly face A stimuli compared to mostly face B stimuli (t (56) = .29, p = .93), which is expected. Our ANOVA did not yield significant group differences (F (1, 55) = .26, p = .61) or a significant stimulus type x group interaction (F (2, 55) = .60, p = .55). In general, we observed a pattern in which labeling ambiguous faces that require greater attention was associated with a greater physiological response (increase SCR) when compared to labeling non-ambiguous faces (see Figure 6). In our matched groups we found a trend in the same direction (see Appendix A).

In the emotion task we found a trend towards an effect of stimulus type (F (2, 53) = 2.77, p = .07). Follow-up t-tests revealed a significantly larger percent of SCRs during labeling of

ambiguous stimuli (face A/B) as compared to the non-ambiguous (mostly face B) stimulus condition (t (54) > 2.20, p = .03), but no significant difference was found for the ambiguous stimuli (face A/B) compared to the non-ambiguous (mostly face A) stimulus condition (t (54) > .33, p = .74). We did not find a stimulus x group interaction (F (1, 53) = 2.23, p = .14) or a significant effect of group (F (2, 53) = .322. p = .72). This pattern of findings is consistent with out heart rate data.

Given that we found significant stimulus effects in our heart rate data, and that our emotion task may contain opposing cognitive and emotional influences, we conducted a secondary ANOVA with stimulus type (mostly face A, mostly face B) and group (control, alexithymia) as factors. We found a trend towards a stimulus effect (F (2, 53) 3.29, p = .07) with a greater proportion of SCRs to face B compared to face A. This pattern of results is expected since past research suggests a positive relationship between viewing fearful faces and SCRs (Williams et al., 2001) (see Figure 6). We did not find a significant group effect (F (1, 53) = 2.17, p = .15) or a significant stimulus type x group interaction (F (2, 53) = .44, p = .51). In our matched groups we did not find significant effects of stimulus type, group or a stimulus type x group interaction for the emotion task (see Appendix A).

Our SCR results show similarities to our heart rate findings. Our identity task yielded the expected effects of attention during labeling of ambiguous faces which was an increase in SCR. Our emotion task yielded the expected pattern of both attention and increasing emotional intensity, which was an increase in SCR (see Figure 6). However, unlike our findings with heart rate, this effect did not differ by group.

3.3 EXPERIMENT 2: TASK-DEPENDENT EMOTION PROCESSING

In Experiment 1, both our identity and emotion tasks yielded significant differences in processing of ambiguous faces (face A/B) compared to faces at endpoints of the identity and emotion continuum (mostly face A, mostly face B). This result suggests that similar physiological changes associated with increases in task difficulty may be normal in alexithymia. However, when we compared the physiological responses to end-point stimuli in the emotion task, the pattern of heart rate data suggested that our alexithymia group may be less sensitive to fearful versus neutral faces relative to controls. We were interested in whether this nonsignificant pattern could be explained by difficulty labeling emotional information or difficulty detecting emotional information. In order to test this question, we turned to the neuroscience literature. In alexithymia the neuroscience research suggests healthy amygdala-related bottom-up processing, but potential problems with MPFC-related top-down processing. To study whether bottom-up processing is intact in alexithymia, we enlisted an implicit/explicit processing paradigm used in past neuroscience research to explore potential bottom-up and top-down mechanisms. We examined the effects of gender labeling of fearful and neutral faces (emotion detection) versus the effects of emotion labeling of fearful and neutral faces (emotion appraisal).

We first assessed potential group differences between behavioral and physiological responses to neutral faces. We found no group differences across our measures of accuracy, reaction time, heart rate and skin conductance (p > .05). Past research has also found that behavioral responses to neutral faces do not differ between alexithymia and control participants (Kano et al., 2003). This suggests that behavioral and physiological reactivity is related to emotion and that alexithymia participants are not just hyper or hypo reactive to all stimuli.

3.3.1 Behavioral Results

Hypotheses: If bottom-up processing is intact in alexithymia then, like controls, we predict that for both subject groups the behavioral responses to emotional stimuli will be facilitated (faster, more accurate) when the task can benefit from bottom-up emotional arousal (e.g., explicit emotion identification tasks). In contrast, behavioral responses to emotional stimuli should be adversely affected (slower, less accurate) when the bottom-up emotional information serves to interfere or distract with the task goals (e.g., identification of gender).

We manipulated task context by instructing participants to label the emotion of a neutral or fearful face (emotion identification), or to label the gender of a neutral or fearful face (gender identification).

To address our primary question of whether emotion processing differs between alexithymia and control subjects, as our dependent variable we computed the emotional reactivity of each participant. We defined emotional reactivity as the average difference in accuracy for fear versus neutral faces. This was assessed in a 2 x 2 ANOVA with task context (emotion identification, gender identification) and group (alexithymia, control) as factors. In this analysis we excluded one participant who met outlier criteria. Data from one additional participant were not included because of technical problems.

We did not find significant group differences in task accuracy (F (1, 55) = .826, p = .37). There was not a significant main effect of task (F (1, 55) = .144, p = .706) or task x group interaction (F (1, 55) = .193, p = .66) (see Figure 7). We obtained the same pattern of results when this analysis was repeated with matched groups (see Appendix A).

Overall, we did not find the expected group difference in accuracy during explicit labeling of emotion. These findings are surprising because they are at odds with the behavioral

literature, which has found that individuals with alexithymia have problems labeling emotions. However, the results are consistent with the findings from Experiment 1 (see Figure 1), where similar response curves were found in an emotion categorization task for individuals with alexithymia, as compared to controls. Our matched groups yielded consistent results as well (see Appendix A)

We next examined reaction time in order to determine whether alexithymia participants differ from controls in their speed of responding to fearful faces during emotion versus gender labeling. Since we were particularly interested in a measure that would capture group differences in reaction time to fearful faces, we chose to implement a reactivity measure in which, for each of our stimuli, we computed the average mean difference between fearful versus neutral faces.

Our dependent measure of reactivity was submitted to a 2 x 2 ANOVA with task (emotion identification, gender identification) and group (alexithymia, control) as factors. For technical reasons we did not have reaction time data from all 30 controls (n = 20 controls).

This analysis yielded a trend towards a significant task effect (F (1, 47) = 3.72, p = .06) in which reaction time to fearful faces slows down during gender identification, but is speeded up during emotion identification. The direction of these effects is consistent with our predictions, and with prior studies that have examined implicit and explicit emotional processing in normal controls (see Figure 8).

We did not find a significant task x group interaction (F (1, 47) = 2.80, p = .16), or a significant group effect (F (1, 47) = .005, p = .95). While this is consistent with our prediction that individuals with alexithymia and controls would show similar effects of task context, upon visual inspection of our data we observed a crossover pattern suggesting that alexithymia

participants are less reactive to fearful faces across both tasks. Follow-up one-sample t-tests were conducted in order to examine whether reactivity to fearful faces was significantly different from zero in controls and alexithymia participants. In controls, we found a trend towards significant reactivity to fearful faces for both the emotion identification (t (19) = -1.86, p = .08) and gender identification tasks (t (19) = 1.94, p = .07), while this trend was not found in alexithymia participants across both the emotion identification (t (28) = .224, p = .82) and gender identification (t (28) = -.259, p = .80) tasks. We obtained non-significant results in our matched groups (p > .30) (see Appendix A).

Our pattern of increased reactivity in controls, but not alexithymia participants suggests a possible emotion blunting in alexithymia that could be related to problems generating physiological responses to emotions (see Figure 8). This finding is consistent with the physiological differences we observed to end-point stimuli in Experiment 1. Taken together, these results run counter to the results that would be predicted if bottom-up processing is intact in alexithymia.

3.3.2 Heart Rate

Hypothesis:

a) Gender identification (implicit): In control subjects, the emotional content of the fearful versus neutral faces should be distracting, thereby increasing the amount of attention necessary to perform the task. In controls, increased attentional demands serve to slow heart rate, thereby opposing the faster heart rate that should be induced by exposure to fearful versus neutral faces. In alexithymia, if bottom-up processing is intact participants are also vulnerable to distraction. Therefore this group should also be susceptible to the opposing influence of heart

rate slowing associated with increased attentional demands. In fact, since appraisal has been considered to be more difficult in alexithymia, individuals with alexithymia may be subject to greater increases in the attentional demands of the task, when compared to controls. This increased attentional demand of emotional faces should serve to slow heart rate more for alexithymia versus control participants (see Figure 1).

- b) Emotion identification (explicit): In control subjects, emotional content (e.g., a fearful face) should induce a faster heart rate. This physiological change may serve as a "somatic marker" that facilitates responding; thereby reducing the attentional demands that can produce heart rate slowing. For this task, the emotional and cognitive factors both predict a relative shift towards faster heart rate for fearful vs. neutral faces. In alexithymia, if bottom-up processing is intact then the heart rate should be faster for fearful vs. neutral faces. However, problems with top-down processing may interfere with the capacity to use heart rate as a "somatic marker" that reduces the attentional demands associated with labeling a fearful vs. a neutral face. In this case the amount of attention allocated to fearful and neutral faces may be about the same. Overall, the difference between heart rate responses to fearful vs. neutral faces may be smaller in individuals with alexithymia, compared to controls because the faster heart rate associated with the bottom-up signal is not complemented by a reduction in the heart rate slowing associated with labeling a fearful versus neutral face (see Figure 2).
- c) Considering the patterns of data across the two tasks (gender identification and emotion identification), we can make predictions about the direction (not the magnitude) of the effect of emotion and attention on heart rate. We predict that both control subjects and individuals with alexithymia will exhibit slower heart rate during gender identification (implicit) versus emotion identification (explicit) processing. The effect of both tasks for individuals with

alexithymia may produce even slower heart rates than healthy controls, because problems with top-down control increase the attentional demands associated emotion regulation in the gender identification task, and fail to yield, or blunt, the attentional benefits associated with the processing of somatic markers in the emotion identification task.

We were interested in whether the physiological signal in response to the categorical processing of identity and emotion in alexithymia is the same as that of controls. Inter beat interval (IBI) was our measure of heart rate response to face processing and was continuously acquired throughout the experiment. In order to focus our analysis on our critical question about emotional task context and physiological reactivity in alexithymia we took a number of data reduction steps both within and between trials. We computed the mean difference between the three IBIs following face stimulus presentation (IBI 4,5,6) and the two IBIs preceding face stimulus presentation (IBI 1,2). This time window best captured the baseline data from all participants as well as their reactivity to facial expressions (Critchley et al., 2005). We next determined the difference between heart rate reactivity in response to fearful versus neutral faces. This served as the outcome measure for our emotion and gender identification task conditions.

Our dependent measure of reactivity was submitted to a 2 x 2 ANOVA with task (emotion identification, gender identification) and group (alexithymia, control) as factors. In this analysis we excluded two participants who met outlier criteria. Data from two additional participants were not included because of technical problems.

We found a significant task x group interaction (F (1, 53) = 8.01, p = .01). Follow-up t-tests found a trend towards a difference between reactivity during gender and emotion tasks in controls (t (25) = -1.91, p = .07) and a significant difference in alexithymia participants (t (28) = 2.13, p = .04). As shown in Figure 9, the interaction shows a cross-over pattern, a finding that

runs counter to our predictions that the direction (if not magnitude) of physiological changes associated with our task context manipulation would be similar in individuals with alexithymia and controls. Follow-up one-sample t-tests were conducted in order to examine reactivity to fearful faces in controls and alexithymia participants. In controls, we found a trend towards faster heart rate reactivity to fearful faces during emotion identification (t (25) = 1.92, p = .07) but not slower heart rate reactivity to fearful faces during gender identification (t (25) = -1.15, p = .26). These findings are in accord with our predictions, since the increased task attention associated with responding to emotional faces in the explicit task context should create a decelerating influence on heart rate that opposes the accelerating influence associated with the bottom-up processing of a fearful stimulus.

In alexithymia participants this pattern of reactivity to fearful faces was reversed for emotion identification (t (28) = 1.59, p = .12) and gender identification tasks (t (28) = -1.69, p = .10), but not significant (see Figure 9). We did not find a significant main effect of task (F (1, 53) = .03, p = .86) or group (F (1, 53) = .001, p = .97). By our predictions, we should find slower heart rate reactivity for the explicit versus implicit task, as we found for controls. If anything, this slowing should be exaggerated because individuals with alexithymia should be experiencing more attentional demands during the implicit task and less benefit of attention with the explicit task. Instead, our reactivity data appear to be consistent with a problem with bottom-up processing. In our matched groups, the pattern of results was in the same direction, suggesting that our results are unrelated to anxiety and neuroticism (see Figure A6).

Overall, our results suggest an interaction in which both healthy controls and alexithymia participants may exhibit task-dependent processing, though in opposite directions (see Figure 9). The direction of predicted effects in healthy controls was supported. In

alexithymia greater attentional demands may account for a pattern of heart rate slowing in response to fearful faces during the explicit task. Context effects may account for heart rate increases during gender identification. Overall, our heart rate data suggest that alexithymia may involve problems with bottom-up emotion detection necessary for emotion processing.

3.3.3 Skin Conductance

Hypothesis:

- a) Gender identification (implicit): For SCR the same principles apply to our predictions, but the literature on SCRs suggests that both emotion and attention are linked to SCR *increases*. In control subjects, the emotional content of the fearful versus neutral faces should be distracting thereby increasing the amount of attention necessary to perform the task. In controls, increased attention demands serve to increase the number of SCRs, thereby increasing SCRs that should be induced by exposure to fearful versus neutral faces. In alexithymia, if bottom-up processing is intact, participants are also vulnerable to distraction. Therefore, this group should also be susceptible to the influence of increased SCR associated with increased attentional demands. In fact, since appraisal has been considered to be more difficult in alexithymia, individuals with alexithymia may be subject to even greater increases in the attentional demands of the task, when compared to controls. This increased attentional demand of emotional faces should serve to increase SCRs more for alexithymia versus control participants.
- b) Emotion identification (explicit): For SCR the same principles apply to our predictions, but the literature on SCRs suggests that both emotion and attention are linked to SCR *increases*. In control subjects, emotional content (e.g., a fearful face) should induce an increase in the number of SCRs. This physiological change may serve as a "somatic marker"

that facilitates responding; thereby reducing the attentional demands associated with an increase in the number of SCRs. For this task, the emotional factor predicts a relative decrease in SCR for fearful vs. neutral faces for controls. In alexithymia, if bottom-up processing is intact then the number of SCRs should increase for fearful vs. neutral faces. However, problems with top-down processing may interfere with the capacity to use SCR as a "somatic marker" that reduces the attentional demands associated with labeling a fearful vs. a neutral face. In this case the amount of attention allocated to fearful and neutral faces may be about the same. Overall, the difference between SCR responses to fearful vs. neutral faces may be larger in individuals with alexithymia, compared to controls because the increase in SCRs associated with the bottom-up signal is not complemented by a reduction in SCRs associated with labeling a fearful versus neutral face.

c) For SCR, we predict that both control subjects and individuals with alexithymia will exhibit increased SCRs during gender identification (implicit) versus emotion identification (explicit) processing. The effect of both tasks for individuals with alexithymia may produce an even greater number of SCRs than healthy controls, because problems with top-down control increase the attentional demands associated emotion regulation in the gender identification task, and fail to yield, or blunt, the attentional benefits associated with the processing of somatic markers in the emotion identification task.

Our measure of SCR to emotional faces was continuously acquired throughout the experiment. In order to focus our analysis on our critical question about emotional and physiological reactivity in alexithymia we took a number of data reduction steps both within and between trials. We were particularly interested in capturing the response to the presentation of each face stimulus. We began by determining, for each trial, whether an SCR occurred. This was

done using a standard amplitude criterion threshold of .05 microsiemens in a 1-3 second time window (Dawson, Schell & Fillion, 2007). Since a large number of participants did not consistently exhibit SCRs (5/54) we did not proceed with SCR amplitude as our outcome measure and instead implemented a count-based outcome measure. For each participant, our count-based measure was calculated as the percentage of trials with an SCR for each of our facial expression conditions (neutral, fearful). We next determined the difference between SCR to fearful versus neutral faces. This served as the outcome measure for our emotion and gender identification task conditions.

Our dependent measure of reactivity was submitted to a 2 x 2 ANOVA with task (emotion identification, gender identification) and group (alexithymia, control) as factors. In this analysis we excluded one participant who met outlier criteria. A second participant was excluded who met outlier criteria for all other behavioral and physiological measures in Experiment 2. Data from four additional participants were not included because of technical problems.

This analysis resulted in non-significant effects [(task: F (1, 52) = .174, p = .68); group (F (1, 52) = .027, p = .87) task x group (F (1, 52) = .071, p = .79)], suggesting no differences in the percentage of SCRs in response to fear minus neutral faces for the emotion identification and gender identification tasks (see Figure 10). Follow-up one-sample t-tests were conducted in order to examine reactivity to fearful faces compared to zero, in controls and alexithymia participants. In controls, we did not find significant reactivity to fearful faces during emotion (t (24) = -.765, p = .45) and gender identification (t (24) = -1.58, p = .13) tasks. In alexithymia, we did not find significant reactivity to fearful faces during emotion (t (28) = -1.40, p = .17) and gender identification tasks (t (28) = -1.46, p = .15).

One concern in the SCR literature is that SCRs may diminish or disappear with repeated exposure to an emotional stimulus (habituation) (Dawson, Schell & Fillion, 2007). We conducted statistical tests in order to confirm that our counterbalanced presentation of face stimuli was not subject to habituation effects. We conducted a 2 x 2 x 2 ANOVA with task order (emotion identification administered first, gender identification administered first), task (emotion identification, gender identification) and group (alexithymia, control) as factors. Our outcome measure was percent of SCRs to fear minus neutral faces. We found no significant main effects of task, group or task order and no interactions between these factors (F $(1, 50) \le 1.43$, $p \ge .24$). In our matched groups, we obtained the same non-significant results with a similar pattern of effects (see Appendix A).

Overall, we found alexithymia participants do not differ from controls on SCR, suggesting that SCR may be intact. While this may be a valid conclusion, on the other hand, controls did not exhibit an SCR increase to fearful faces. Our lack of SCR effect in controls is unexpected since healthy bottom-up responses to fearful faces have been associated with increases in SCR (Hariri et al., 2003; Williams et al., 2004a,b; 2005a,b). This failure to replicate prior findings in controls leads us to question the reliability of our measure.

4.0 DISCUSSION

In this study we set out to accomplish two aims: 1) to determine whether individuals with alexithymia exhibit behavioral responses to emotional faces that differ from controls and whether there are corresponding changes in physiological measures, 2) to examine whether behavioral and physiological responses in alexithymia are differentially influenced by an implicit versus explicit manipulation of emotional task context, as compared to controls. We conducted two experiments in order to assess each goal. The first experiment involved an emotion and an identity categorization task in which we examined behavioral and physiological responses to faces. The second experiment involved implicit and explicit processing tasks that allowed us to examine bottom-up and top-down influences on the behavioral and physiological responses to fearful versus neutral faces. We were sensitive to the potential role of traits related to alexithymia, including anxiety and neuroticism, by controlling for these factors in a matched groups analysis. Baseline physiological arousal and state anxiety were also examined in order to consider their potential impact on our outcome measures. Our findings point us towards an account of alexithymia in which appraisal processes are intact but the generation and/or detection of physiological signal is altered. This conclusion runs counter to findings in neuroimaging literature, which would suggest that brain regions involved in emotion detection are intact functionally, while brain regions linked to emotion appraisal are altered functionally. This conclusion also contradicts the general notion that alexithymia involves a difficulty in identifying

emotions. In the following discussion, we elaborate on these conclusions and how they fit into the current state of the alexithymia literature.

4.1 STUDY POPULATION

Our research is focused on participants who have difficulty processing emotions but for whom this process may not interfere with mood. The majority of neuroimaging studies (4/5) have focused on a similar population of participants who are free from psychiatric illness including anxiety and depression (Berthoz et al., 2002; Kano et al., 2003; Leweke et al., 2004; Mantani et al., 2005). In contrast, in the physiology literature few studies have excluded participants with a psychiatric history (Franz et al., 2003; Friedlander et al., 1997; Stone & Nielson, 2001). In fact the majority of research has focused on all comers (Martin & Phil, 1986; Newton & Contrada, 1994; Naring; Fukunishi, Sei, Morita & Rahe, 1999; Parker et al., 1993; Roedema & Simons, 1999; Whemer et al., 1995) or a specific psychiatric population (Infrasca, 1997, Rabavilis, 1987). The inconsistency in study population across neuroimaging and physiological studies may account for mixed findings in the literature. Since there is considerable overlap between alexithymia and mood symptoms (Taylor, Bagby & Parker, 1997, for review) future research using our categorical processing and implicit/explicit task manipulations could examine alexithymia in depressed and/or anxious participants in order to determine whether behavioral and physiological results generalize to these populations.

4.2 BASELINE FINDINGS

Past research has determined that individuals with alexithymia are likely to report that they are in an anxious state during experimental tasks (Martin & Phil, 1986; Newton & Contrada, 1994). We examined whether our alexithymia and control groups differ on a measure of self-reported state anxiety. Consistent with past research we found that alexithymia participants report greater state anxiety relative to controls prior to our experimental manipulation, and that reported state anxiety declined more in alexithymia versus control participants at the end of the study. Since our experiments were counterbalanced we do not believe that this decline in self-reported anxiety affected our Experiment 1 and Experiment 2 disproportionately.

Interestingly, we did not find group differences in baseline heart rate; while, prior studies have found mixed results (group differences: Infrasca, 1997; Papciak et al., 1995; Stone & Neilson, 2001; Whemer et al., 1995; no difference: Newton & Contrada, 1994; Roedema & Simons, 1999; hypoarousal: Linden, Lenz, & Stossel, 1996). For baseline SCR, we did not find differences between alexithymia and control participants, which is similar to past research studies (Rabavilis, 1987; Roedema & Simons, 1999). We also assessed the potential relationship between self-reported state anxiety and baseline heart rate and skin conductance. As expected, baseline differences in self-reported pre-experiment state anxiety were not correlated with baseline measures of skin conductance or baseline heart rate in either alexithymia or control participants. Though we did not find group differences in the relationship between self-reported anxiety and physiological reactivity (p = .65), findings from past research motivate closer examination of this relationship in alexithymia. Past research has found dissociation between self appraisal and change in heart rate in alexithymia compared to control subjects. For example, it has been found that alexithymia participants are not as adept at reporting a change in their heart rate when

compared to controls, and that high alexithymia participants tend to be less at ease during experiments despite normal heart rate (Manatini et al., 2005; Naring & van der Staak, 1995; Papciak et al., 1998). Chronic problems generating physiological responses to emotion could "decouple" self-reported anxiety and physiology, requiring alexithymia participants to be highly attuned to external cues (Martin & Phil, 1986).

We did not find baseline difference in heart rate or SCR for the control and alexithymia groups suggesting that these are not confounds of our reactivity data. We found baseline differences in state anxiety in alexithymia compared to control participants. We determined whether anxiety confounds our heart rate and skin conductance measures by examining whether anxiety at baseline and physiological measures at baseline are correlated. Since we did not find a correlation between these measures in either group we can conclude that a difference in state anxiety does not confound our data. Self-reported state anxiety at baseline was not significantly correlated with heart rate or SCR for either alexithymia or control participants. The relationship between self-reported anxiety and physiological reactivity did not differ between groups. Given the literature on decoupling of self-reported anxiety and physiological state and alexithymia, this relationship deserves additional evaluation and consideration.

Why might we find a difference in anxiety for alexithymia compared to control participants? One suggestion is that a problem generating a "somatic marker" of emotion creates unpredictability. In prior work on fear conditioning, it has been shown that a decrease in the ability to predict an aversive event is associated with an increase in general anxiety. In this study, the experiment context may have served as a cue (Grillon, Morgan, Davis & Southwick, 1998). By extension, if internal emotions (somatic markers) are absent in alexithymia, these individuals may turn to the external context as a cue that helps to predictions about the environment.

4.3 EXPERIMENT 1: CATEGORICAL PROCESSING

In Experiment 1, we attempted to determine whether individuals with alexithymia exhibit behavioral responses to emotional faces that differ from controls and whether there are corresponding changes in the physiological responses to emotional faces.

For the identity categorization task we predicted that our experimental groups would exhibit similar behavioral and physiological responses, since this task involves only non-emotional faces. As predicted, we did not find group differences in the location and slope of the category boundary. As expected, we also did not find group differences in heart rate or SCR at the endpoints of the identity continua. Our prediction of heart rate slowing during labeling of ambiguous identities was also supported, suggesting that the normal physiological impact of increased attention during cognitive tasks may be intact in alexithymia. This finding is in line with our suggestion that individuals with alexithymia are physiologically less sensitive to emotional, but not cognitive information. Interestingly, the effect of increased attention was an increase in the number of SCRs for both groups. This is consistent with past research suggesting that SCR is sensitive to cognitive demands and may increase with increased attention and novelty (Dawson, Schell and Filion et al., 2007).

For the emotion categorization task, we predicted that the location and slope of the category boundary would differ for individuals with alexithymia, when compared to controls. We found that the location and slope of the category boundary was the same for both groups, suggesting that emotion labeling in alexithymia is intact. This result is surprising and may reflect the specifics of our task. For example, participants were given unlimited time to respond to faces, which may have minimized group differences in task accuracy. In support of this conclusion, in a recent study, alexithymic individuals were more likely to detect a difference between negative

(anger, sad and fear) and neutral faces when a stimulus is displayed for a longer duration (3 seconds versus 1 second). Also, our task involved presentation of two faces for each level of emotional intensity (0-100%) which may have limited our power to detect group difference in task accuracy. A reaction time measure, speeded task conditions, or the use of a larger stimulus set may help to reveal group differences. Physiologically, we predicted that end-points of the emotion categorization task would be blunted in response to mostly fearful versus mostly neutral faces in alexithymia participants because they would be less sensitive to emotion, when compared to controls. Our heart rate findings reveal a pattern that appears to be consistent with this prediction. For the SCR measure, we found increases in response to mostly fearful faces in healthy controls, thereby replicating findings from past research (Hariri et al., 2003; Williams et al., 2004a, b; 2005a,b). Surprisingly, in contrast to our heart rate data, we did not find evidence for group differences in the SCR for mostly fearful versus mostly neutral faces.

Predictions about emotion categorization of ambiguous faces in alexithymia participants can be informed by our findings during the identity categorization task. Physiological responses during the identity task suggest that ambiguous faces are associated with a slowing of heart rate and increase in SCR in both healthy controls and alexithymia participants. For the emotion categorization task, we predicted that the increase in task difficulty during classification of highly ambiguous faces near the category boundary would result in group differences in which there is an opposing impact of emotional-cognitive demands. Specifically, heart rate slowing during identification of ambiguous faces may work in opposition to the faster heart rate we expect in healthy controls in response to emotional face. In alexithymia the emotional-cognitive balance may be shifted because of problems processing emotions. Our predictions about processing of ambiguous faces during emotion categorization seem to be supported by changes in heart rate. We

found that heart rate was blunted during labeling of ambiguous faces that increase in fear intensity, in alexithymia participants compared to control participants. As predicted, we suspect that the opposing influence of emotion and cognition serves to slow heart rate. The proportion of SCRs was also reduced near the category boundary, but we did not find a significant group effect.

One reason that we may not have detected group differences in SCR during emotion categorization in Experiment 1 is that, unlike heart rate, SCR is correlated with changes in sympathetic nerve activity and is modulated by cholinergic receptors. SCR and heart rate do not always yield consistent results. For example, greater sustained attention has been associated with heart rate deceleration and SCR increases (e.g. Roedema & Simons, 1991; Stone & Nielson, 2001). During the emotion task, this relationship is further complicated by opposing emotional and cognitive demands. Both SCR and heart rate have important links to brain pathways including the amygdala, hippocampus, and frontal cortex. Further exploration of the role of cortical-subcortical interactions on the synaptic transmitters that mediate heart rate and SCR may be useful in further understanding these inconsistencies. Other explanations for our SCR results include individual differences in skin moisture and transduction from nerve to skin moisture to electrode. Additionally, past research has found small effects sizes when comparing individuals with psychopathology to healthy controls on measures of SCR, suggesting that group differences are difficult to detect (Dawson, Schell and Filion, 2007). It is unlikely that anxiety and neuroticism account for our inability to detect group differences in SCR, since we did not find a significant correlation between SCR and trait measures (r (53) > -.190, p > .17). Furthermore, our matched group analysis of skin conductance during emotion categorization also failed to yield group differences.

In summary, findings from Experiment 1 lead us to conclude that behavioral responses in alexithymia participants are intact during both emotional and non-emotion tasks. Physiological responses to non-emotional tasks also seem to be intact in alexithymia. In contrast, physiological responses to emotional tasks appear to be blunted in alexithymia. Furthermore, the combined effect of increased emotional intensity and increased attention during the emotion categorization task highlights the delicate interplay between emotion and cognition and its impact on physiological responses in both individuals with alexithymia and healthy controls.

4.4 EXPERIMENT 2: TASK-DEPENDENT EMOTION PROCESSING

Experiment 2, we attempted to gain a mechanistic understanding of alexithymia using implicit tasks that are weighted towards bottom-up processing and explicit tasks that are weighted towards top-down processing. Based on the neuroimaging literature we hypothesized that bottom-up processing in alexithymia is intact and that top-down appraisal is altered.

Our findings in healthy controls seem to be consistent with our prediction that processing of implicit versus explicit tasks enlists different physiological and behavioral signals. Our healthy control data suggests that our manipulation of implicit and explicit processing is effective. In Experiment 2, our implicit task is weighted towards bottom-up processing and our explicit task is weighted-towards top-down processing. Our findings and conclusions acknowledge the complex interplay between emotional and cognitive processes during both implicit and explicit tasks that assess accuracy, response time and physiology. This task design is similar to that of a Stroop task in which individuals are asked to read a word and either use or ignore congruent or incongruent information about the color of the word. In this task the response that is produced is influenced by

two processes that are color naming and word reading. These processes are not stochastically independent (Hillstrom & Logan 1997). Likewise, in Experiment 2 of our study, participants try to use emotional information during explicit processing and not use emotional information during gender identification. To some extent these processes involve both bottom-up and top-down processing. Some have argued that it is necessary to use specific algorithms in order to distinguish task manipulations involving automatic (implicit) and intentional (explicit) processing (Jacoby et al., 1991). Others have argued that dissociating automatic and intentional tasks is not appropriate when the outcome being measured depends on both processes (Hillstrom & Logan 1997). In our study, we employ automatic and intentional tasks whose outcomes depend on the complex interplay between emotional and cognitive processes during both implicit and explicit tasks. Since much of the affective neuroscience literature has employed task manipulations involving implicit and explicit processing, future research should consider whether it is necessary to employ specific task parameters that control for the effects of each process.

Behaviorally, in alexithymia, we expected to find problems labeling emotions during the explicit task. In contrast to our prediction, performance was intact during emotion labeling. This finding is consistent with findings from Experiment 1. In contrast to our results, several studies found poor performance on emotion labeling tasks in participants with alexithymia (Jessimer & Markham, 1997; Kano et al., 2003; Lane et al., 1996; Panday & Mandel, 1997; Parker et al., 1993). Our findings are partially in line with one study in which alexithymia participants were able to accurately label but not accurately engage in facial poses of emotional expressions (McDonald and Prkachin, 1990).

While accuracy for alexithymia participants was intact, our reaction time measures allowed us to make predictions about potential bottom-up and top-down influences. In healthy controls past

research found faster reaction time to fearful faces during explicit compared to implicit tasks. We based our predictions in alexithymia on the literature in healthy controls. In alexithymia, we predicted intact bottom-up processing in which reaction time during implicit tasks involves greater effort in top-down regulation that serves to slow heart rate when compared to controls. In alexithymia we also predicted intact bottom-up processing in which reaction time during explicit tasks involves problems labeling emotions rather than generating an emotional response. In this case we would expect to find less top-down regulation of emotion and therefore, faster reaction time to fearful faces that may be exaggerated, in alexithymia versus control participants. In alexithymia, however, we found a pattern in which reaction time to fearful faces appeared to be blunted and task-independent. This finding suggests that, in alexithymia participants, emotion does not modulate reaction time in response to task demands and that generating emotion (bottom-up) is not intact. Prior studies have found similar results. One study found that emotion fails to influence reaction time during implicit tasks in participants with alexithymia (Vermeulen, Luminet & Corneille, 2006); and a second study found that a greater time window provides the best opportunity for accurate emotion labeling during explicit tasks (Panday & Mandel, 1997). Taken together these behavioral findings appear to be consistent with the view that alexithymia is a problem of bottom-up processes related to generating emotions, in which there is less sensitivity to emotional information. Past research also highlights that task difficulty is an important factor in determining reaction time.

According to our behavioral data alexithymia may be a problem involving an altered bottom-up signal. Physiologically, we determined whether this was the case.

We predicted that bottom-up processing is intact in alexithymia. If this were the case then, like controls, alexithymia participants would be vulnerable to distraction and susceptible to the opposing influence of heart rate slowing associated with increased attentional demands. If appraisal is more difficult in alexithymia, individuals with alexithymia would be subject to greater increases in the attentional demands of the task, when compared to controls. This increased attentional demand of emotional faces would serve to slow heart rate more for alexithymia versus control participants.

Our findings were not consistent with this prediction, since we found faster heart rate in alexithymia relative to healthy controls and importantly the direction of the task effect was actually opposite to our predictions. We believe that our results are consistent with a problem with bottom-up processing in alexithymia. The faster heart rate during gender identification may suggest that alexithymia participants do not generate a distracting bottom-up signal for fearful versus neutral faces. Though highly speculative, we suggest that alexithymia participants may even benefit from emotional cues because emotional expressions may provide context that is associated with gender identity. During the gender identification task, we found that alexithymia participants are most accurate at identifying male faces when they are fearful (alexithymia group: male fear > male neutral, female fear, female neutral (t (26) > -2.22, p < .035), while healthy controls are equally accurate at identifying male and female faces when they are fearful or neutral [(male fear = male neutral, female fear, female neutral t (26) < 1.74, p > .095) (alexithymia versus controls for male fear t (52) = -2.56, p = .013)].

For the emotion identification task, we again predicted that bottom-up processing is intact. In this case, we might expect that, in alexithymia, problems with top-down processing interfere with the normal capacity to use heart rate as a "somatic marker" that reduces the attentional demands associated with labeling a fearful vs. a neutral face. The amount of attention allocated to fearful and neutral faces would not differ. Overall, the difference between heart rate

responses to fearful vs. neutral faces would be smaller in individuals with alexithymia, compared to controls because the faster heart rate associated with the bottom-up signal would not be complemented by a reduction in the heart rate slowing associated with labeling a fearful versus neutral face.

Our findings were not consistent with this prediction, since we did not find blunted responses to fearful versus neutral faces in alexithymia. We believe that our result could be explained by problems with bottom-up processing in alexithymia. Our results during the emotion task may indicate problems generating emotions which create a small physiological signal to fearful and neutral faces. Smaller signal in response to faces also may require greater attention to external cues. Increased attention might serve to slow heart rate, especially for fearful versus neutral faces for alexithymia participants relative to controls. However, in the case of the emotion task, the increased attention that may explain the heart rate slowing to fearful versus neutral faces may be independent from the effects of difficulty, which were the same for fearful and neutral faces during the reaction time task.

We did not find effects of fearful faces on SCR in controls or in alexithymia participants during Experiment 2. These SCR findings were unexpected, since past research has found correlations between passive viewing of emotional faces and images and SCR (Hariri et al., 2003; Williams et al., 2004a, b; 2005a,b). In Experiment 1, we did find significant increases in SCR for both control and alexithymia participants during labeling of mostly fearful faces. Since Experiment 1 and Experiment 2 were counterbalanced we do not believe that the differences in our SCR findings across Experiment 1 and Experiment 2 can be related to habituation effects. One possibility is that our SCR measure was not sensitive to the implicit/explicit paradigm used for this study.

Our results for Experiment 2 appear to be consistent with a bottom-up view of alexithymia. This view is best informed by current theories of emotion processing in which physiology provides information necessary to engage in adaptive behaviors and decision making (Bechara, Damasio, Tranel & Demasio, 1996). Based on this idea, the blunted reaction time and the altered physiological response in alexithymia may suggest that alexithymia participants are not generating a "somatic marker" necessary to make timely decisions about emotion and gender during implicit and explicit tasks (Bechara, Damasio, Tranel & Damasio, 1996). Past research has found that lesions of the amygdala impair the physiological response necessary to avoid aversive decisions during experimental conditions and in real life (Bechara, Damasio, Damasio & Lee, 1999). Interestingly, individuals with alexithymia are able to accurately label emotions, despite problems with physiological arousal and reaction time. This result may suggest that the ability to use MPFC/top-down processing to make decisions is intact. MPFC/top-down processing allows for the integration of somatic information along with information about context (Bechara, Damasio, Damasio & Lee, 1999). Context and attention may be particularly important compensatory mechanisms in alexithymia because of an altered/dampened bottom-up signal.

4.5 ALEXITHYMIA AS A PROBLEM OF BOTTOM-UP PROCESSING

Our experimental results suggest that alexithymia is a problem of bottom-up processing. During Experiment 1, we found that labeling of emotional and non-emotional faces is intact. We also found a pattern in which physiological arousal is blunted during categorization of emotional, but not cognitive stimuli in alexithymia. During Experiment 2, we found that labeling of emotional faces during emotion identification is intact. We also found a pattern of blunted behavioral

responses across both implicit and explicit task contexts, as well as a pattern of altered physiological responses to implicit and explicit task context relative to controls.

Our view that alexithymia is a problem of bottom-up processing may be an alternative view relative to the current thinking that alexithymia is a problem of emotional appraisal (Taylor et al., 2000). It also seems to be in opposition to other literature suggesting that alexithymia is a coping response (Parker, Taylor & Bagby, 1998). Coping through repression or related defense mechanisms would suggest over-regulation of emotion. Our findings suggest problems generating, rather than regulating emotion. Likewise, our findings differ from neuroimaging studies that indicate problems with MPFC-related appraisal rather than amygdala-related emotion detection. How can our results be reconciled with past research?

4.5.1 Neuroimaging

The neuroimaging literature provides the most consistent findings related to alexithymia and motivates our initial study predictions. Returning to this literature, studies required participants to make judgments about emotion (Berthoz et al., 2002; Kano et al., 2003). None of these experiments involved labeling a range of faces that elicit fear. Anderson et al., (2003a) suggested that the automaticity of the amygdala response is specific to fearful expressions and not to other salient signals that convey biologically relevant information. In this sense, our study may provide an important window into emotion detection in alexithymia that has yet to be examined. Any glaring problems detecting emotions should be apparent during our manipulation. Indeed, we found a pattern of altered physiological arousal during implicit and explicit tasks, which are weighted towards problems with emotion detection and emotion appraisal respectively.

Furthermore, the biological significance of fearful stimuli may allow behavior and physiology to transcend potential problems with top-down processing that have been found in past studies.

The neuroimaging literature puts forth a complex story of MPFC-amygdala interactions. In animals, stimulation of the MPFC elicits a decrease in activity of neurons in the central nucleus of the amygdala, which has output to brain regions important for eliciting a physiological response (Quirk et al., 2003). A recent human study enlisted a fear extinction paradigm in order to examine activity in the ventral MPFC. During extinction learning and increased blood flow in the ventral MPFC and a decrease in skin conductance during retention of the extinguished response was found. Amygdala blood flow was positively correlated with skin conductance during conditioning and extinction. Amygdala blood flow and skin conductance decreased with extinction learning. Amygdala blood flow and skin conductance also diminished with retention of the extinguished response. This pattern of ventral MPFC-amygdala response is again consistent with a "push-pull" relationship (Phelps, Delgado, Nearing & LeDoux, 2004). In human studies, explicit labeling of fear faces is associated with increased blood flow in the MPFC and decreased blood flow in the amygdala (Hariri et al., 2000; Winston, O'Doherty, & Dolan, 2003; Lange et al., 2003; Leiberman et al., 2007).

Based on the animal and human literature, we might expect this "push-pull" relationship in alexithymia. Prior neuroimaging studies of alexithymia have found that MPFC is altered, but studies report no difference in amygdala processing (Berthoz et al., 2002; Kano et al., 2003; Leweke et al., 2004; Huber et al., 2002; Mantani et al., 2005). This literature does not acknowledge the well-established MPFC-amygdala interaction in which change in one is associated with change in the other.

What can be said of the pattern of non-significant amygdala findings in the neuroimaging literature on alexithymia? In controls, the neuroimaging literature suggests that an increase in MPFC is associated with a decrease in amygdala response. MPFC-related regulation of the amygdala response may, in fact, serve to bring amygdala activation back to baseline levels. In alexithymia, if amygdala-related bottom-up processing is not engaged then there may be less need for MPFC-related regulation of emotion. Without emotion regulation there may not be an opposing force to counteract amygdala activity, and we might find no change in amygdala function. Based on this model, when a group comparison between controls and alexithymia participants is made we might find no difference in amygdala function. Another reason for the non-significant difference in amygdala activity in the neuroimaging literature may be that the amygdala is susceptible to imaging artifact and habituation effects, which may pose a challenge to neuroimaging studies (Lange et al., 2003). In addition to our speculations about amygdala-MPFC interactions in alexithymia, this question prompts further neuroimaging studies in alexithymia participants that use implicit and explicit tasks to provoke a push-pull relationship.

Another way to reconcile our findings with past neuroimaging research is to consider paradigms that might present additional opportunities to target alexithymia symptoms. Unlike past research, in our study we did not specifically present personally relevant material or probe "self-awareness" by asking participants to describe how each fearful expression made them feel following or during the experiment. Instead both of our experiments relied on external forced-choice judgments of either emotion or gender. Self-awareness has been linked to a related network of prefrontal brain regions, including the MPFC. Had we probed this more specifically then, perhaps, we may have found physiological alterations suggesting an MPFC-related disruption in alexithymia (Lane, Ahern, Schwartz & Kaszniak, 1997).

4.5.2 Physiology

Turning to the physiology literature, another question is whether we can equate our findings of bottom-up processing difficulties in alexithymia with past physiological studies. Again, the strength of our study design is that, unlike past research it accomplishes two goals: 1) assessing behavioral and physiological correlates of alexithymia using a set of comparable cognitive and emotional tasks and 2) enlisting known biological mechanisms important for emotion detection and emotion appraisal.

Our comparison with past research focuses on heart rate which has been measured across all studies. At baseline, a condition independent from task effects, our findings are consistent with a subset of studies that found no difference in baseline heart rate even though anxiety at baseline is higher (Franz et al., 2003; Newton & Contrada, 1994; Roedema & Simon, 1999). During explicit tasks, our finding of a pattern of heart rate slowing in alexithymia is consistent with several studies (Linden et al., 1996; Neumann et al., 2004; Newton & Contrada, 1994). However these studies involved emotion provocation and did not account for the effects of both emotion and cognition on heart rate. The majority of past explicit studies of alexithymia are not consistent with our results. These studies examined primarily emotional stressors and found increases in heart rate relative to controls (Fukunishi, Sei, Morita & Rahe, 1999; Martin & Phil, 1986; Papciak et al., 1985; Rabavilas, 1987). During implicit tasks, our finding of a pattern of heart rate acceleration is inconsistent with past research in which less heart rate deceleration has been found in alexithymia participants versus control participants while passively viewing images of emotional scenes (Friedlander et al., 1997; Roedema & Simons, 1999; Whemer et al., 1995). In our study, we propose that the pattern of heart rate acceleration we observed during the implicit gender

identification task is related to using task context as a compensatory strategy related to problems generating emotions in alexithymia.

In our study, we found changes in heart rate in response to fearful compared to neutral faces. Our outcome measure for heart rate was inter-beat interval. This measure alone does not delineate the sympathetic and parasympathetic components of heart rate. Few studies have examined the physiological correlates of MPFC regulation. In animals it has been suggest the dorsal MPFC (BA 25) is associated with a parasympathetic response while ventral MPFC (BA 32) is associated with a sympathetic response (Powell et al., 1994). In humans, the few studies that have examined the contributions of parasympathetic and sympathetic activity to brain function have yielded mixed results (Gianaros et al., 2004; Matthews, Paulus, Simmons, Neleson & Dimsdale, 2004). Future research could further examine the relative sympathetic and parasympathetic contributions to brain function and how this relates to the appraisal and generation of emotional information in individuals with alexithymia and healthy controls.

In summary, our task-independent baseline measures are consistent with a subset of research findings. In contrast, task-related physiological findings are not comparable with past research, which is most likely related to differences in task design that may yield a different balance between cognitive influences on physiological measures. Based on this reassessment of physiological studies we suggest that future studies also use tasks that are directly informed by basic neuroscience. We also suggest measurement of the sympathetic and parasympathetic components of heart rate in order to delineate the contributions of MPFC to physiological reactivity.

4.6 ALEXITHYMIA VIEWPOINTS

Based on our findings we believe that alexithymia is a physiological problem rather than a cognitive problem. This view assumes that appraisal processing involves *implementing* top-down, MPFC-related control. While we believe that our results are consistent with a bottom-up model there is another potential explanation that may also be consistent with our results. As previously mentioned, appraisal processing may reflect detection and engagement of emotional processing that is influenced by attentional load. Consistent with the latter conceptualization, past research suggests lateral prefrontal cortex may be important for "elaborating" on MPFC-related appraisal processes (Kalisch, Korenfeld, Stephan, Weiskopf, Seymour & Dolan, 2006). In a recent study, an increase in blood flow in the dorsal prefrontal cortex was found during explicit labeling of facial expressions. Using a functional connectivity analysis a correlated was found between blood flow in the dorsal lateral prefrontal cortex, MPFC and amygdala during this task (Keightly, Winocur, Graham, Mayberg, Hevenor & Grady, 2003). The relationship between dorsal lateral prefrontal cortex and MPFC may suggest that attentional demands are part of the explicit emotion labeling process. In this case, an explicit emotion labeling task requiring attention might serve to slow heart rate, while an implicit gender labeling tasks requiring relatively less attention might serve to speed up heart rate. This result might then lead us to conclude that alexithymia is a problem of top-down processing related to *detecting and engagement* of emotional information.

The strongest test of these views, *implementation* versus *detection and engagement*, is a manipulation of attention. Attention may be manipulated using covert presentation of stimuli that do not require attention. Unfortunately, studies using covert tasks have yielded mixed findings and somewhat weak effects that may be related to differences in methodological approach (e.g., Phillips et al., 2004, for discussion). One concern about using covert tasks in our population of

alexithymia participants is that our results may be diminished. Other ways of reconciling these views is to incorporate into the current work a dual-task that might divide attentional demands.

4.7 TREATMENT APPROACHES

Our conclusion that alexithymia appears to be related to problems with bottom-up processing allows us to speculate about potential treatment approaches. A variety of evidence-based clinical interventions focus on labeling emotions, including Interpersonal Psychotherapy (IPT), Cognitive Behavioral Therapy (CBT) and Dialectical Behavior Therapy (DBT). In IPT, labeling of emotions is encouraged when describing the relationship between mood and interpersonal relationships. There is some evidence that difficulties identifying feelings may improve in a sample of depressed patients receiving IPT, though additional research is necessary (Gilbert et al., November 2006). In CBT, emotion labeling is a component of examining the relationship between feelings, thoughts and actions. Unlike IPT and CBT, in DBT, emotion labeling is targeted during skills training. Naming emotions is incorporated into a "model for describing emotions" (Linehan, M, 1993). During DBT skills training, this model is used to teach patients about the process of naming emotions. Patients are taught that this process has several components. The "prompting event" leads to an emotion and can occur automatically and without thought or as part of a person's thoughts, behaviors and physiological responses. The "interpretation" of the emotion follows the prompting event. Interpretation is when an individual appraises and considers the event. Subsequent to interpretation, or as a direct result of a prompting event, changes in the brain and body, as well as the senses occur. These contribute to the experience of emotion. Emotions are then expressed through facial movements, body language and physical action before the emotion can, finally, be named (Linehan, M., 1993). Training patients about the specific pathways that lead to emotion may be a useful way to draw attention to subtle physiological changes. It may also be useful in helping individuals to read the context of emotional experiences so as to maximize the experience of emotion.

Past research has found that DBT, which includes education about emotional awareness, or "mindfulness," has been effective in addressing symptoms of dissociative disorders, in which patients experience emotional numbing (e.g. Simpson et al., 2004). Although alexithymia and dissociative disorders are psychometrically non-overlapping and differ somewhat in their neural correlates (Phillips & Sierra, 2003), these disorders may have some common characteristics. Specifically, similar to depersonalization we found heightened anxiety and decreased physiological detection (heart rate) in alexithymia. Problems detecting emotions in both disorders may suggest that DBT is a useful intervention for improving the ability generate physiological responses in alexithymia.

Interestingly, in alexithymia, we found that emotions can be labeled. The problem may be a difficulty generating emotions. Based on this finding our suggestion is that evidence-based interventions incorporate biofeedback. On-line physiological assessments during emotional experience may help individuals with alexithymia increase their level of physiological response and reduce the effort that may be necessary to appraise this response. Furthermore, our study suggests that individuals with alexithymia might benefit from using the social context to guide emotion responding. We would suggest that treatment interventions might also emphasize the use of social context to facilitate physiological experiences of emotion.

REFERENCES

- Andreassi, J.L. (2007). *Psychophysiology: Human behavior and physiology response* (5th ed.) Mahwah, NJ: Lawrence Erlbaum associates.
- Aftanas, L. I., Varlamov, A.A., Revan, N.V., & Pavlov, S. V. (2003). Disruption of early event-related theta synchronization of human EEG in alexithymics viewing affective pictures. *Neuroscience Letters*, *340*(1), 57-60.
- Amaral, D., Price, J., Pitkanen, A., & Carmichael, S. (1992). Anatomical organization of the primate amygdaloid complex. In J. Aggleton (Ed.), *The Amygdala: Neurobiological Aspects of Emotion, Memory, and Mental Dysfunction* (pp. 1-66): Wiley-Liss, Inc.
- American Psychiatric Association. (2000). Diagnostic and statistical manual of mental disorders, 4th ed., text revision. Washington, DC: Author.
- Andrews, G., Sign, M., Bond, M. (1993). The Defense Syle Questionnaire. *Journal of Nervous and Mental Disease*, 181, 246-261.

- Annett, M. (1970). A classification of hand preference by association analysis. *British Journal of Psychiatry*, 61, 301-321.
- Bagby, R., Parker, J., & Taylor, G. (1994a). The Twenty-Item Toronto Alexithymia Scale

 I: Item selection and cross-validation of the factor structure. *Journal of Psychosomatic**Research, 38(1), 23-32.
- Bagby, R., Taylor, G., & Parker, J. (1994b). The Twenty-Item Toronto Alexithymia Scale- II: Convergent, discriminant, and concurrent validity. *Journal of Psychosomatic Research*, 38(1), 33-40.
- Beauregard, M., Levesque, J., & Bourgouin, P. (2001). Neural correlates of conscious self-regulation of emotion. *Journal of Neuroscience*, 21(18), 165RC.
- Bechara, A., Damasio, H., Damasio, A.R., & Lee, G.P. (1999). Different contributions of the human amygdala and ventromedial prefrontal cortex to decision-making. *Journal of Neuroscience*, 19(13), 5473-81.
- Bechara, A., Tranel, D., Damasio, H., & Damasio, A.R. (1996). Failure to respond autonomically to anticipated future outcomes following damage to prefrontal cortex.

 Cerebral Cortex, 6(2), 215-25.

- Bernston, G.G. Quigley, K.S. & Lozano, D. (2007): Cardiovascular psychophysiology.

 In J.T Cacioppo., L.G. Tassinary, G.G. Bernston (Eds)., *Handbook of Psyhcophysiology* (3rd ed.) (pp. 182-210). New York: Cambridge University Press.
- Berthoz, S., Artiges, E., Van de Moortele, P.-F., Poline, J.-B., Rouquette, S., Consoli, S. M., & Martinot, J.-L. (2002). Effect of impaired recognition and expression of emotions on frontocingulate cortices: An fMRI study of men with alexithymia. *American Journal of Psychiatry*, 159(6), 961-967.
- Berthoz, S., Consoli, S., Perez-Diaz, F., & Jouvent, R. (1999). Alexithymia and anxiety: compounded relationships? A psychometric study. *European Psychiatry*, *14*(7), 372-378.
- Berthoz, S. & Hill, E. L. (2005). The validity of using self-reports to assess emotion regulation abilities in adults with autism spectrum disorders, European Psychiatry, 20, 291-298.
- Britton, J. C., Taylor, S. F., Sudheimer, K. D., & Liberzon, I. (in press, corrected proof). Facial expressions and complex IAPS pictures: Common and differential networks.

 NeuroImage.
- Cohen, J. (1988). *Statiscal power analysis for the behavioral sciences* (2nd Ed.). Hillside, New Jersy: Lawrence Erlbaum Associates.

- Costa, P. J., & McCrae, R. (1992). *The NEO Personality Inventory Revised*. Odessa, Florida: Psychological Assessment Tools.
- Craig, A. (2002). How do you feel? Interoception: The sense of the physiological condition of the body. *Nature Reviews Neuroscience*, *3*, 655-666.
- Critchley, H. D., Rotshtein, P., Nagai, Y., O'Doherty, J., Mathias, C. J., & Dolan, R. J. (2005).

 Activity in the human brain predicting differential heart rate responses to emotional facial expressions. *NeuroImage*, 24(3), 751-762.
- Davies, M., Stankov, L. & Roberts, R. D. (1998). Emotional Intelligence: In search of an elusive construct. *Journal of Personality and Social Psychology*, 75, 989-1015.
- Devine, H., Stewart, S. H., & Watt, M. C. (1999). Relations between anxiety sensitivity and dimensions of alexithymia in a young adult sample. *Journal of Psychosomatic Research*, 47(2), 145-158.
- Dawson, D.A., Grant, B.F., Stinson, F.S., & Chou, P.S. (2005). Psychopathology associated with drinking and alcohol use disorders in the college and general population. *Drug and Alcohol Dependence*, 77, 139-150.

- Dawson, M.E., Schell, A.M. & Fillion, D.L. (2007):The electrodermal system.In J.T Cacioppo., L.G. Tassinary, G.G. Bernston (Eds)., *Handbook of Psyhcophysiology* (3rded.) (pp. 159-181). New York: Cambridge University Press.
- Eckman, P., & Friesen, W. (1976). *Pictures of Facial Affect*. Palo Alto, CA: Consulting Psychologists Press. Elzinga, B.M., Bermond, B., & van Dyck, R. (2002). The relationship between dissociative proneness and alexithymia. *Psychotherapy and Psychosomatics*, 71,104-111.
- Erni, T. Lotscher, K., & Modestin, J. (1997). Two-factor solution of the 20-item Toronto Alexithymia Scale Confirmed. *Psychopathology*, *30*, 335-340.
- Eysenck, H. J. & Eysenck, S. B.G. (1975). *Manual of Eysenck Personality Questionnaire*.

 San Diago, CA: Educational and Industrial Testing Service.
- Franz, M., Schaefer, R., & Schneider, C. (2003). Psychophysiological response patterns of high and low alexithymia under mental and emotional load conditions. *Journal of Psychophysiology*, 17(4), 203-213.
- Freyberger, H. (1977). Supportive psychotherapeutic techniques is primary and secondary alexithymia. *Psychotherapy and Psychosomatics*, 28, 337-342.

- Friedlander, L., Lumley, M., Farchione, T., & Doyal, G. (1997). Testing the alexithymia hypothesis: Physiological and subjective responses during relaxation and stress. *Journal of Nervous and Mental Diseases*, 185(4), 233-239.
- Frysztak, R., & Neafsey, E. (1994). The ffect of medial frontal cortex lesions on cardiovascular conditioned emotional responses in the rat. *Brain Research*, *643*, 181-193.
- Fukunishi, I. Kikuchi, M., Wogan, J., & Takubo, M. (1997). Secondary alexithymia as a state reaction in panic disorder and social phobia. *Comprehensive Psychiatry*, 38(3), 166-170.
- Fukunishi, I., Sei, H., Morita, Y., & Rahe, R. H. (1999). Sympathetic activity in alexithymics with mother's low care. *Journal of Psychosomatic Research*, 46(6), 579-589.
- Fukunishi, Wise, Sheridan, Shimani, Otake, Utsuki, & Uchiyanma (2001). Association of emotion intelligence with alexithymic characteristics. *Psychological Reports*, 89, 651-658.
- Garlow, S.J., Rosenberg, J., Moore, D., Haas, A.P., Koestner, B., Hendin, H. et al. (2007).

 Depression, desperation and suicidal ideation in college students: Results from the American foundation for suicide prevention college screening project at Emory University. *Depression and Anxiety*, 0, 1-7.

- Gianaros, P., van der Veen, F., & Jennings, R. (2004). Regional cerebral blood flow correlates with heart period and high-frequency heart period variability during working memory tasks: Implications for the cortical and subcortical regulation of cardiac autonomic activity. *Psychophysiology*, *41*, 521-530.
- Gilbert, A. M., Lanza di Scalea, T., Cyranowski, J., Swartz, H.A., Frank, E. (November, 2006). *Impact of Interpersonal Psychotherapy on Toronto Alexithymia Scale-20 Scores.* Poster abstract presented at the 2nd International Conference on Interpersonal Psychotherapy, Toronto, Canada.
- Glascher, J., & Adolphs, R. (2003). Processing of the arousal of subliminal and supraliminal emotional stimuli by the human amygdala. *Journal of Neuroscience*, 23(32), 10274-10282.
- Gorno-Tempini, M. L., Pradelli, S., Serafini, M., Pagnoni, G., Baraldi, P., Porro, C., Nicoletti, R., Umita, C., & Nichelli, P. (2001). Explicit and incidental facial expression processing: An fMRI Study. *NeuroImage*, *14*(2), 465-473.
- Grabe, H-J., Rainermann, S., Spitzer, C., Gansicke, M., & Freyberger, H.J., (2000). The relationship between dimensions of alexithymia and dissociation. *Psychotherapy and Psychosomatics*, 69, 128-131.

- Grillon C., Morgan C.A., 3rd, Davis, M., Southwick, S.M. (1998). Effects of experimental context and explicit threat cues on acoustic startle in Vietnam veterans with posttraumatic stress disorder. *Biological Psychiatry*, 44(10):1027-36.
- Gur et. al. (2002). Journal of Neuroscience Methods, 115, 137-143.
- Hariri, A., Bookheimer, S., & Mazziotta, J. (2000). Modulating emotional responses: Effects of a neocortical network on the limbic system. *Neuroreport*, 11(1), 43-48.
- Hariri, A. R., Mattay, V. S., Tessitore, A., Fera, F., & Weinberger, D. R. (2003). Neocortical modulation of the amygdala response to fearful stimuli. *Biological Psychiatry*, *53*(6), 494-501.
- Hariri, A. R., Tessitore, A., Mattay, V. S., Fera, F., & Weinberger, D. R. (2002). The amygdala response to emotional stimuli: A comparison of faces and scenes. *NeuroImage*, 17(1), 317-323.
- Haviland, M. G., Sonne, J. L., Kowert, P. A. Alexithymia and Psychopathy: Comparison and Application of California Q-set Prototypes. *Journal of Personality Assessment. Vol* 82(3) *Jun* 2004, 306-316.
- Herba, C.M., Heining, M., Yojng, A. W., Browning, M., Benson, P.J., Phillips, M.I., &

- Gray, J. A. (in press). Conscious and nonconscious discrimination of facial expressions. *Visual Cognition*.
- Hill, E., Berthoz, S., & Frith, U. (2004). Brief report: Cognitive processing of own emotions in individuals with autistic spectrum disorders and their relatives.

 *Journal of Autism and Developmental Disorders, 34(2), 229-235.
- Hillstrom, A.P. & Logan, G.D. (1994). Process dissociation, cognitive architecture, and response time: comments on Lindsay and Jacoby (1994). *Journal of Experimental Psychology and Human Perception and Performance*, 5, 1561-78.
- Hintikka, J., Honkalampi, K., Lehtonen, J., & Viinamaki, H. (2001). Are alexithymia and depression distinct or overlapping constructs? A study in the general population. *Comprehensive Psychiatry*, 42(3), 234-239.
- Honkalampi, K., Hintikka, J., Laukkanen, E., Lehtonen, J. & Viinamaki, H. (2001).

 Alexithymia and depression: A prospective study of patients with major depressive disorder. Psychosomatics, 42(3), 229-234.
- Honkalampi, K., Hintikka, J., Saarinen, P., Lehtonen, J. & Viinamaki, H. (2000). Is alexithymia a permanent feature in depressed patients? Results from a 6-month follow-up study. Psychotherapy and Psychosomatics, 69, 303-308.

- Honkalampi, K., Hintikka, J., Tanskanen, A., Lehtonen, J., & Viinamaki, H. (2000).

 Depression is strongly associated with alexithymia in the general population. Journal of Psychosomatic Research, 48, 99-104.
- Honkalampi, K., Saarinen, P., Hintikka, J., Virtanen, V., & Viinamaki (1999). Factors associated with alexithymi in patietns suffering from depression. *Psychotherapy and Psychosomatics*, 68, 270-275.
- Huber, M., Herholz, K., Habedank, B., Thiel, A., Muller-Kuppers, M., Ebel, H., Subic-Wrana,
 C., Kohl, K., & Heiss, W. (2002). Different patterns of regional brain activation during emotion stimulation in alexithymics in comparison with normal controls [German].
 Psychotherapie and Psychosomatik Medizinische Psychologie, 52(11), 469-478.
- Hurley, K., Herbert, H., Moga, M., & Saper, C. (1991). Efferent projections of the infralimbic cortex of the rat. *Journal of Comparative Neurology*, 308(2), 249-276.
- Infrasca, R. (1997). Alexithymia, neurovegetative arousal and neuroticism. *Psychotherapy and Psychosomatics*, 66, 276-280.
- Izard, C.E. (1971). *The faces of emotion*. New York, Appelton-Century Crofts.
- Jacoby, L.L. (1991). A process dissociation framework: Separating automatic from intentional uses of memory. *Journal of Memory and Language*, 30,513-541.

- Jennings J.R. (1986): Bodily changes during attending. In Coles M.G.H. Donchin E. Porges S.W. (Eds)., *Psychophysiology: Systems, Processes, and Applications*. (pp 268-289). New York, Guilford
- Jessimer, M., & Markham, R. (1997). Alexithymia: A right hemisphere dysfunction specific to recognition of certain facial expressions. *Brain and Cognition*, *34*, 246-258.
- Juylha, P. & Isometsa, E. (2006). The relationship of neuroticism and extraversion to symptoms of anxiety and depression. *Depression and Anxiety*, 23(5), 281-289.
- Kano, M., Fukudo, S., Gyoba, J., Kamachi, M., Tagawa, M., Mochizuki, H., Itoh, M., Hongo,
 M., & Yanai, K. (2003). Specific brain processing of facial expressions in people with alexithymia: an H215O-PET study. *Brain*, 126(6), 1474-1484.
- Kauhanen, J., Kaplan, G.A., Cohen, R.D., Julkunen, J. & Salonen, J.D. (1996). Alexithymia and risk of death in middle-aged men. *Journal of Psychosomatic Research*, 41(6), 541-549.
- Kauhanen, J., Kaplan, G.A., Julkunen, J., Wislon, T.W., Salonen, J.T. (1993). Social factors in alexithymia, *Comprehensive Psychiatry*, 34(5), 330-335.
- Kircher T.T., Senior C., Phillips M.L., Rabe-Hesketh, S., Benson, P.J., Bullmore, E.T. et al. (2001). Recognizing one's own face. *Cognition*. 78(1):B1-B15.

- Klorman R., Wiesenfeld A.R., & Austin, M.L. (1975). Autonomic responses to affective visual stimuli. *Psychophysiology*, 12(5):553-60.
- Knapp, P.H. (1983). Emotions and bodily changes: A reassessment. In L. Temoshok, C. VanDyke, & L.S. Zegans (Eds.) *Emotions in health and illness: Theoretical and research*foundations (pp. 15-27). New York: Grune & Stratton.
- Koh, K.B. Cho, S.Y., Kim, J.W., Rho, K.S., Lee, S.H. & Park, I.H. (2004). The relationship of anger expression and alexithymia with coronary artery stenosis in patients with coronary artery diseases. *Yonsei Medical Journal*, 45(2), 181-186.
- Kojima, M. Frasure-Smith, N. & Lesperance, F. (2001). Alexithymia following myocardial infarction: psychometric properties and correlates of the Toronto Alexithymia Scale. *Journal of Psychosomatic Research*, 51(3), 487-495
- Kokkonen, P., Karvonen, J.T., veijola, J., Laksy, K., Jokelainen, J., jarvelin, M-R., & Joukamas, M. (2001). Prevalence and sociodemographic correlates of alexithymia in a population sample of young adults. *Comprehensive Psychiatry*, 42(6), 471-476.
- Kooiman, C. G., Spinhoven, P. & Trijsburg, R. W. (2002). The assessment of alexithymia: A critical review of the literature and a psychometric study of the Toronto Alexithymia Scale-20. *Journal of Psychosomatic Research*, *53*, 1083-1090.

- Lane, R., Reiman, E., Ahern, G., & Thayer, J. (2001). Activity in the medial prefronal cortex correlates with vagal component of heart rate variability during emotion. Paper presented at the 10th Annual Rotman Research Institute Conference, Toronto, Ontario.
- Lane, R.D., Sechrest, L. & Riedel, R. (1998). Sociodemographic correlates of alexithymia.

 *Comprehensive Psychiatry, 39(6):377-85.
- Lane, R., Sechrest, L., Reidel, R., Weldon, V., Kaszniak, A., & Schwartz, G. (1996).
 Impaired verbal and nonverbal emotion recognition in alexithymia. *Psychosomatic Medicine*, 58, 203-210.
- Lane, R., Sechrest, L., Riedel, R., Shapiro, D., & Kaszniak, A. (2000). Pervasive emotion recognition deficit common to alexithymia and the repressive coping style. *Psychosomatic Medicine*, 62, 492-501.
- Lang, P.J., Greenwald, M.K., Bradley, M.M., Hamm, A.O. (1993). Looking at pictures: affective, facial, visceral, and behavioral reactions. *Psychophysiology*, 3, 261-73.
- Lange, K., Williams, L. M., Young, A. W., Bullmore, E. T., Brammer, M. J., Williams, S. C.
 R., Gray, J. A., & Phillips, M. L. (2003). Task instructions modulate neural responses to fearful facial expressions. *Biological Psychiatry*, 53(3), 226-232.

- Lawrence, N. S., Williams, A. M., Surguladze, S., Giampietro, V., Brammer, M. J., Andrew, C., Frangou, S., Ecker, C., & Phillips, M. L. (2004). Subcortical and ventral prefrontal cortical neural responses to facial expressions distinguish patients with bipolar disorder and major depression. *Biological Psychiatry*, *55*(6), 578-587.
- LeDoux, J. (2000). Emotion circuits in the brain. Annual Review of Neuroscience, 23, 155-184.
- LeDoux, J., Iwata, J., Cicchetti, P., & Reis, D. (1988). Different projections of the central amygdaloid nucleus mediate autonomic and behavioral correlates of conditioned fear. *Journal of Neuroscience*, 8(7), 2517-2529.
- LeDoux, J., Romanski, L., & Xagoraris, A. (1989). Indelibility of subcortical emotional memories. *Journal of Cognitive Neuroscience*, 1, 238-243.
- 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.
- Linehan, M. M., (1993). Skills training manual for treating borderline personality disorder, New York: Guilford.

- Leweke, F., Stark, R., Milch, W., Kurth, R., Schienle, A., Kirsch, P., Stingl, M., Reimer, C., & Vaitl, D. (2004). Patterns of neuronal activity related to emotional stimulation in alexithymia [German]. *Psychotherapie and Psychosomatik Medizinische**Psychologie, 54(12), 437-444.
- Linden, W., Lenz, J. W., & Stossel, C. (1996). Alexithymia, defensiveness and cardiovascular reactivity to stress. *Journal of Psychosomatic Research*, 41(6), 575-583.
- Lipsanen, T., Saarijarvi, S., & Lauerma, H. (2004). Exploring the relations between depression somatization, dissociation and alexithymia overlapping or independent constructs? *Psychopathology*, *37*, 200-206.
- Loas, G., Otmani, O., Verrier, A., Fremaux, D. & Marchand, M.P. (1996). Factor analysis of the French version of the 20-item Toronto Alexithymia Scale (TAS-20). *Psychopathology*, 29(2), 139-144.
- Loas, G., Corcos, M., Stephean, P., Pellet, J., Bizouard, P., Venisse, J-L. et al. (2001). Factorial structure of the 10-item Toronto Alexithymia Scale Confirmatory factor analysis in nonclinical and clinical samples. *Journal of Psychosomatic Research*, 50, 255-261.

- Luminet, O., Bagby, M.R., & Taylor, G. J. (2001). An evaluation of the absolute and relative stability of alexithymia in patients with major depression. *Psychotherapy and Psychosomatics*, 70, 254-260.
- Luminet, O., Bagby, M.R., Wagner, H., Taylor, G. J., & Parker, J.D.A. (1999).

 Relations between alexithymia and the five-factor model of personality: A facet-level analysis. *Journal of Personality Assessment*, 73(3), 345-358.
- Lumley, M.A. (2000). Alexithymia and negative affect. Journal of Psychosomatic Medicine, 49, 51-54.
- Lumley, M.A., Gustavson, R., Partridge, T. & Labouvie-Vief, G. (2005). Assessing alexithymia and related emotional ability constructs using multiple methods: Interrelationships among measures. *Emotion*, *5*(3), 329-342.
- Lumley, M.A., Ovies, T., Stettner, L., Wehmer, F., & Lakey, B. (1996). Alexithymia, social support and health problems. *Journal of Psychosomatic Research*, 41(6), 519-530.
- Lundh, L.-G., & Simonsson-Sarnecki, M. (2002). Alexithymia and cognitive bias for emotional information. *Personality and Individual Differences*, 32, 1063-1075.
- Maclean, P.D. (1949). Psychosomatic disease and the "visceral brain"; recent developments bearing on the Papez theory of emotion. *Psychosomatic Medicine*, *11*, 338-353.

- Maggini, C., Raballo, A., Pelizza, L., Paini, M., & Croci, R. (2003). Subjective experience of language impairment and psychopathology in schizophrenia, *Psychopathology*, *36*, 17-22.
- Mann, L., Wise, T., Trinidad, A., & Kohanski, R. (1994). Alexithymia, affect recognition, and the five-factor model of personality in normal subjects. *Psychological Reports*, 74, 563-567.
- Mantani, T., Okamoto, Y., Shirao, N., Okada, G., & Yamawaki, S. (2005). Reduced activation of posterior cingulate cortex during imagery in subjects with high degrees of alexithymia: A functional magnetic resonance imaging study. *Biological Psychiatry*, 57(9), 982-990.
- Marchesi, C., Brusamonti, E., Maggini, C. (2000). Are alexithymia, depression, and anxiety distinct constructs in affective disorders? *Journal of Psychosomatic Research*, 49, 43-49.
- Marchesi, C., Fonto, S., Balista, C., Cimmino C., & Maggini, C., (2005). Relationship between alexithymia and panic disorder: A longitudinal study to answer an open question. *Psychotherapy and Psychosomatics*, 74, 56-60.

- Martinez-Sanchez, F., Ato-Garcia, M., Adam, E. C., Medina, T. B. H. & Espana, J.J.S. (1998).

 Stability in alexithymia levels: A longtiudinal analysis on various emotional answers.

 Personality and Individual Differences, 24(6), 767-772.
- Martin, J., & Phil, R. (1986). Influence of alexithymic characteristics on physiological and subjective stress responses in normal individuals. *Psychotherapy and Psychosomatics*, 45, 66-77.
- Marty, P. & de M'Uzanm, M. (1963). La 'pensee operatoire.' Revue Française de Psychanalyse, 27 (suppl.), 1345-1356.
- Mason, O., Tyson, M., Jones, C., & Potts, S. (2005). Alexithymia: Its prevalence and correlates in a British undergraduate sample. *Psychology and Psychotherapy:*Theory, Research, and Practice, 78, 113-125.
- Matthews, S.C., Paulus, M.P., Simmons, A.N., Nelesen, R.A., Dimsdale, J.E. (2004). Functional subdivisions within anterior cingulate cortex and their relationship to autonomic nervous system function. *Neuroimage*, 22(3), 1151-6.
- McCallum, M., Piper, W.E., Ogrodniczuk J.S., & Joyce, A.S. (2003). Relationships among psychological mindedness, alexithymia and outcome in four forms of short-term

psychotherapy. *Psychology and Psychotherapy: Theory, Research and Practice*, 76(2), 133-144.

- McDonald, P., & Prkachin, K. (1990). The expression and perception of facial emotion in alexithymia: A pilot study. *Psychosomatic Medicine*, 52, 199-210.
 - Meyers, L.S. (1995). Alexithymia and repression: The role of defensiveness and trait anxiety, *Personality and Individual Differences*, 19(4), 482-492.
- Mikolajczak, M. & Luminet, O. (2006). Is alexithymia affected by situational stress or is it a stable trait related to emotion regulation? *Personality and Individual Differences*, 40, 1399-1408.
- Milad, M. R., & Quirk, G. J. (2002). Neurons in medial prefrontal cortex signal memory for fear extinction. *Nature*, 420(6911), 70-74.
- Morgan, M., Romanski, L., & LeDoux, J. (1993). Extinction of emotional learning:

 Controlbution of medial prefrontal cortex. *Neuroscience Letters*, *163*, 109-113.
- Morris, J., Friston, K., Buchel, C., Frith, C., Young, A., Calder, A., & Dolan, R. (1998). A neuromodulatory role for the human amygdala in processing emotional facial expressions. *Brain*, *121*(1), 47-57.

- Morris, J., Frith, C., Perrett, D., Rowland, D., Young, A., Calder, A., & Dolan, R. J. (1996).

 A differential neural response in the human amygdala to fearful and happy facial expressions. *Nature*, 383, 812-815.
- Muller, J., Buhner, M., & Ellegring, H. (2003). Relationship and differential validity of alexithymia and depression: A comparison of the Toronto Alexithymia and Self-rating Depression Scales, *Psychopathology*, *36*, 71-77.
- Nemiah, J.C. (1977). Alexithymia: Theoretical considerations. *Psychotherapy and Psychosomatics*, 28, 199-206
- Neumann, S. A., Sollers, I., John J., Thayer, J. F., & Waldstein, S. R. (2004). Alexithymia predicts attenuated autonomic reactivity, but prolonged recovery to anger recall in young women. *International Journal of Psychophysiology*, *53*(3), 183-195.
- Newton, T., & Contrada, R. (1994). Alexithymia and repression: Contrasting emotion-focused coping styles. *Psychosomatic Medicine*, *56*, 457-462.
- Ochsner, K., Bunge, S., Gross, J., & Gabrieli, J. (2002). Rethinking feelings: An fMRI study of the cognitive regulation of emotion. *Journal of Cognitive Neuroscience*, 14(8), 1215-1229.

- Ogrodniczuk, J.S. Piper, W.E. & Joyce, A.S. (2005). The negative effect of alexithymia on the outcome of group therapy for complicated grief: what role might the therapist play? *Comprehensive Psychiatry*, 46(3), 206-213.
- Ogrodniczuk, J.S. Piper, W.E. & Joyce, A.S. (2004). Alexithymia as a predictor of residual symptoms in depressed patients who respond to short-term psychotherapy. *American Journal of Psychiatry*, 58(2), 150-161.
- Papciak, A., Feuerstein, M., & Spiegel, J. (1985). Stress reactivity in alexithymia: Decoupling of physiological and cognitive responses. *Journal of Human Stress*, 11(3), 135-142.
- Parker, J., Taylor, G., & Bagby, R. (1993). Alexithymia and recognition of facial expressions of emotion. *Psychotherapy and Psychosomatics*, *59*, 197-202.
- Parker, P., Prkachin, K., & Prkachin, G. (2005). Processing of facial expressions of negative emotion in alexithymia: The influence of temporal constraint. *Journal of Personality*, 73(4), 1087-1107.
- Parker, J.D.A., Taylor, G.J., & Bagby, M.R. (1989). The alexithymia construct: Relationship with sociodemographic variables and intelligence. *Comprehensive Psychiatry*, 30(5), 434-441.

- Parker, J.D.A., Taylor, G. J., & Bagby, M. (1998). Alexithymia: relationship with ego defense and coping styles. *Comprehensive Psychiatry*, 39(2), 91-98.
- Parker, J.D.A., Taylor, G. J., & Bagby, M. (2001). The relationship between emotional intelligence and alexithymia. Personality and Individual Difference, 30, 107-115.
- Parker, J.D.A., Taylor, G. J. & Bagby, M.R. (2003). The 20-item Toronto Alexithymia Scale

 III. Reliability and factorial validity in a community population. Journal of

 Psychosomatic Research, 55, 269-275.
- Phelps, E. A., Delgado, M. R., Nearing, K. I., & LeDoux, J. E. (2004). Extinction learning in humans: Role of the amygdala and vmPFC. *Neuron*, *43*(6), 897-905.
- Phillips, M.L. & Sierra, M. (2003). Depersonalization disorder: a functional neuroanatomical perspective. *Stress*, 6(3), 157-65.
- Pollack, S., & Kistler, D. (2002). Early experience is associated with development of c ategorical representations for facial expressions of emotion. *Proceedings of National Academy of Sciences*, 99(13), 9072-9076.
- Powell, D., Chachich, M., Murphy, V., McLaughlin, J., Tebbutt, D., & Buchanan, S. (1997).

 Amygdala-prefrontal interactions and conditioned bradycardia in the rabbit.

 Behavioral Neuroscience, 111(5), 1056-1074.

- Powell, D., Watson, K., & Maxwell, B. (1994). Involvement of subdivisions of the medial prefrontal cortex in learned cardiac adjustments in rabbits. *Behavioral Neuroscience*, 108(2), 249-307.
- Powell, D. A., & Ginsberg, J. P. (2005). Single unit activity in the medial prefrontal cortex during Pavlovian heart rate conditioning: Effects of peripheral autonomic blockade.

 *Neurobiology of Learning and Memory, 84(3), 200-213.
- Quirk, G. J., Armony, J. L., & LeDoux, J. E. (1997). Fear conditioning enhances different temporal components of tone-evoked spike trains in auditory cortex and lateral amygdala. *Neuron*, *19*(3), 613-624.
- Quirk, G. J., Likhtik, E., Pelletier, J. G., & Pare, D. (2003). Stimulation of medial prefrontal cortex decreases the responsiveness of central amygdala output neurons. *Journal of Neuroscience*, 23(25), 8800-8807.
- Rabavilas, A. (1987). Electrodermal activity in low and high alexithymia neurotic patients.

 *Psychotherapy and Psychosomatics, 47, 101-104.
- Roedema, T., & Simons, R. (1999). Emotion-processing deficit in alexithymia. *Psychophysiology*, 36(3), 379-387.

- Ruesch, J. (1948). The infantile personality; the core problem of psychosomatic illness.

 *Psychosomatic Medicine, 10, 134-44.
- Russell, T.A., Reynaud, E., Kucharska-Pietruea, K. Ecker, C., Benson, P.J., Zelaya, F. et al., (2007). Neural responses to dynamic expressions of fear in schizophrenia. *Neuropsychologia*, 45, 107-123.
- Saarijarvi, S., Salminen, J.K. & Toikka, T. (2006). Temporal stability of alexithymia over a 5-year period in outpatients with major depression. Psychotherapy and *Psychosomatics*, 75, 107-112.
- Salminen, J.K., Saarijarvi, S., Aairela, E., & Tamminen, T. (1994). Alexithymia state or trait? One-year follow-up study of a general hospital psychiatric consultation outpatients. *Journal of Psychosomatic Research*, 38(7), 681-685.
- Salminen, J.K., Saarijarvi, S., Aarela, E., Toikka, T. & Kauhanen, J. (1998). Prevalence of alexithymia and its association with the sociodomographic variables in the general population of Finland. *Journal of Psychosomatic Research*, 46(1), 75-82.
 - Salovey, P. & Mayer, J.D. (1990). Emotional Intelligence. *Imagination, Cognition and Personality*, 9, 185-211.

- Saper, C. (1982). Reciprocal parabrachial-cortical connections in the rat. *Brain Research*, 242(1), 33-20.
- Saper, C. (2002). The central autonomic nervous system: Conscious visceral perception and autonomic pattern generation. *Annual Review of Neuroscience*, 25(433-469).
- Schneider, F., Gur, R.C., Koch, K., Backes, V., Amunts, K., Shah, N.J., Bilker, W., Gur, R.E., Habel, U. (2006). Impairment in the specificity of emotion processing in schizophrenia. *American Journal of Psychiatry*, 163(3), 442-447.
- Schutte, N.S., Malouff, J.M., Hall, L.E., Haggerty, D.J., Cooper, J.T., Golden, C.J. & Dornheim, L. (1998). Development and validation of a measure of emotional intelligence. *Personality and Individuals Difference*, 25, 167-177.
- Schwartz, A.J. (2006). Are college students more disturbed today? Stability in the acuity and qualitative character of psychopathology of college counseling center clients: 1992-1993 through 2001-2002. *J Am Coll Health*, *54*(6):327-37.
- Sifneos, P. E. (1973). The prevalence of alexithymic characteristics in psychosomatic patients. *Psychotherapy and Psychosomatics*, 22, 255-262.
- Sifneos, P. E. (1996). Alexithymia: Past and Present. *American Journal of Psychiatry*, 153(7), 137-141.

- Simpson, E.B., Yen, S., Costello, E., Rosen, K., Begin, A., Pistorello, J. (2004). et al. Combined dialectical behavior therapy and fluoxetine in the treatment of borderline personality disorder. Journal of Clinical Psychiatry, 65(3):379-85.
- Speranza, M., Corcos, M., Guilbaud, O., Loas, G. & Jeammet, P. (2005). Alexithymia, personality and psychopathology. *American Journal of Psychiatry*, *162*(5), 1030-1031.
- Spielberger, R., Gorsuch, R., & Lushene, P. (1983). *Manual for the state trait anxiety inventory*. Palo Alto, CA: Consulting Psychologists Press.
- Stone, L., & Nielson, K. (2001). Intact physiological responses to arousal with impaired emotional recognition in alexithymia. *Psychotherapy and Psychosomatics*, 70, 92-102.
- Surguladze, S., Brammer, M. J., Keedwell, P., Giampietro, V., Young, A. W., Travis, M. J.,
 Williams, S. C. R., & Phillips, M. L. (2005). A differential pattern of neural response toward sad versus happy facial expressions in major depressive disorder. *Biological Psychiatry*, 57(3), 201-209.
- Taylor, G. J., Bagby, M. R., & Parker, J.D.A. (2003). The 20-item Toronto Alexithymia Scale IV: Reliability and factorial validity in different languages and cultures. *Journal of Psychosomatic Research*, 55, 277-283.

- Tabachnick, B.G. & Fidell, L.S. (2001). *Using Multivariate Statistics, Fourth Edition*. Needham Heights, MA: Allyn & Bacon.
- Terreberry, R., & Neafsey, E. (1983). Rat medial frontal cortex: A visceral motor region with a direct projection to the solitary nucleus. *Brain Research*, 278, 245-249.
- Terreberry, R., & Neafsey, E. (1987). The rat medial prefrontal cortex projects directly to autonomic regions of the brainstem. *Brain Research Bulletin*, 19, 639-649.
- Todarello, O., Porcelli, P., Grilletti, F., & Bellomo, A. (2004). Is alexithymia related to negative symptoms of schizophrenia? A preliminary longitudinal study. *Psychopathology*, 38, 310-314.
- Tottenham, N., Borscheid, A., Ellertsen, K., Marcus, D., & Nelson, C. (2002). Categorization of facial expressions in children and adults: Establishing a larger stimulus set [Abstract]. *Journal of Cognitive Neuroscience*, 14(Suppl.), S74.
- Trull, T., & Sher, K. (1994). Relationship between the five-factor model of personality and Axis

 I disorders in a nonclinical sample. *Journal of Abnormal Psychology*, 103, 350-360.
- Vaslamatzis, G. (2003). Alexithymia: Contemporary theory and research data. *Psychiatriki*, 14(1), 6-7.

- Vermeulen, N., Luminet, O., & Corneille, O. (2006). Alexithymia in the automatic processing of affective information: Evidence from the affective priming paradigm. *Cognition and Emotion*, 20(1), 64-91.
- Wehmer, F., Brejnak, C., Lumley, M., & Stettner, L. (1995). Alexithymia and physiological reactivity to emotion-provoking visual scenes. *Journal of Mental andNervous Diseases*, 183(6), 351-357.
- Weinberg, S.L., & Abromowitz, S.K. (2002). *Data analysis for the behavioral sciences using SPSS*. New York: Cambridge University Press.
- Whalen, P., Shin, L., McInerney, S., Fischer, H., Wright, C., & Rauch, S. (2001). A functional MRI study of human amygdala responses to facial expressions of fear versus anger. *Emotion*, *1*(1), 70-83.
- Williams, L., Das, P., Liddell, B., Olivieri, G., Peduto, A., Brammer, M., & Gordon, E. (2005a). BOLD, sweat and fears: fMRI and skin conductance distinguish facial fear signals. *Neuroreport*, *16*(1), 49-52.
- Williams, L., Liddell, B., Kemp, A., Bryant, R., Meares, R., Peduto, A., & Gordon, E. (2005b). Amygdala-prefrontal dissociation of subliminal and supraliminal fear. [Electronic Version]. *Human Brain Mapping*.

- Williams, L., Liddell, B., Rathjen, J., Brown, K., Gray, J., Phillips, M., Young, A., & Gordon, E. (2004a). Mapping the time course of nonconscious and conscious perception of fear: An integration of central and peripheral measures. *Human Brain Mapping*, 21, 64-74.
- Williams, L. M., Brown, K. J., Das, P., Boucsein, W., Sokolov, E. N., Brammer, M. J., Olivieri,
 G., Peduto, A., & Gordon, E. (2004b). The dynamics of cortico-amygdala and autonomic activity over the experimental time course of fear perception. *Cognitive Brain Research*, 21(1), 114-123.
- Williams, L. M., Phillips, M. L., Brammer, M. J., Skerrett, D., Lagopoulos, J., Rennie, C., Bahramali, H., Olivieri, G., David, A. S., Peduto, A., & Gordon, E. (2001). Arousal dissociates amygdala and hippocampal fear responses: Evidence from simultaneous fMRI and skin conductance recording. *NeuroImage*, *14*(5), 1070-1079.
- Winston, J. S., O'Doherty, J., & Dolan, R. J. (2003). Common and distinct neural responses during direct and incidental processing of multiple facial emotions. *NeuroImage*, 20(1), 84-97.
- Wise, T. N., Mann, L.S., Mitchell, D., Hryvniak, M., & Hill. B. (1990). Secondary alexithymia: An empirical validation. *Comprehensive Psychiatry*, 31(4), 284-288.

- Wise, T.N., Mann, L.S., & Sheridan, M.J. (2000), Relationship between alexithymia, dissociation, and personality in psychiatric outpatients. Psychotherapy and *Psychosomatics*, 69(3), 123-127.
- Yasui, Y., Itoh, K., Takada, M., Matani, A., Kaneko, T., & Mizuno, N. (1985). Direct cortical projections to the parabrachial nucleus in the cat. *Journal of Comparative Neurology*, 234(1), 77-86.
- Young, A., Perrett, D., Calder, A., Sprengelmeyer, R., & Ekman, P. (2002). *Facial Expressions of Emotion Stimuli and Tests (FEEST)*. Bury St. Edmunds, England: Thames Valley Test Company.
- Zimmermann, G., Rossier, J., de Stadelhofen, F. M. & Gaillard, F. (2005). Alexithymia assessment and relations with dimensions of personality. European Journal of *Psychological Assessment*, 21(1), 23-33.
- Zlotnick, C., Shea, M.T., Pearlstein, T., Simpson, E., Costello, E., & Begin, A. (1996). The relationship between dissociative symptoms, alexithymia, impulsivity, sexual abuse and self-mutilation. *Comprehensive Psychiatry*, *37*(1), 12-16.

APPENDIX: MATCHED GROUPS

We selected a group of alexithymia (n = 13) and control (n = 12) participants from

our study group who were matched on anxiety, extraversion and neuroticism (t (23) < 1.1635, p

> .116) and gender (X² (N = 23) = .326, p = .57). Our matched groups remained non-significantly

different despite small differences in subject number for tasks in which data was missing because

of outlier exclusion or technical problems. Using our matched groups and the same set of study

analyses we re-examined our data in an effort to account for the potential confounds of anxiety

and neuroticism.

Experiment 1: Categorical Processing

Behavior:

We did not find group differences in category boundary for the emotion (t (23) =

.397, p = .69) and the identity task (t (23) = 2.06, p = .05). We did not find group differences

between the slopes for either the identity (t (23) = .281, p = .78) or the emotion tasks (t (23) = .281, p = .78)

1.17, p = .27) (see Figure 11).

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Heart Rate:

In the identity task we found a main effect of stimulus type (F (2, 22) = 7.71, p = .00). We conducted follow-up t-tests to further asses these stimulus effects. We found that IBI was significantly increased (slowed heart rate) for the ambiguous (face A/B) as compared to the non-ambiguous (mostly face A, mostly face B) stimulus conditions (t (23) > 3.27, p < .00), whereas the two non-ambiguous stimulus conditions were not significantly different from each other (t (23) = .855, p = .40). Our ANOVA yielded a trend towards group differences (F (1, 22) = 3.60, p = .07) but, importantly, and a non-significant stimulus type x group interaction (F (2, 22) = .848, p = .43).

In the emotion task we found a trend towards a main effect of stimulus type (F (2, 22) = 3.01, p = .06). We conducted follow-up t-tests to further asses these stimulus effects. We found a trend towards a significantly increased IBI (slowed heart rate) for the ambiguous (face A/B) as compared to the non-ambiguous (mostly face B) stimulus conditions (t (23) > 1.97, p < .06), whereas we did not find a significant difference between the ambiguous face (face A/B) as compared to the non-ambiguous (mostly face A) (t (23) = .605, p = .55). The two non-ambiguous stimulus conditions were not significantly different from each other (t (23) = .855, p = .40). We did not find significant stimulus x group (t (23) = .905, p = .41) or group effects (t (23) = .810, p = .38). Our secondary ANOVA yielded a trend for the effect of stimulus type (F (1, 22) = 3.29, p = .08) in which the heart rate for face B versus face A did not differ for controls (t (10) = 1.78, p = .10) or alexithymia groups (t (12) = .474, p = .64). Our secondary ANOVA did not yield

significant stimulus x group (F (1, 22) = 1.65, p = .21) or group effects (F (1, 22) = .510, p = .49) (see Figure 12).

Skin Conductance

In the identity task we did not find significant effects of stimulus type (F (2, 22) = 1.46, p = .24). Follow-up t-tests revealed a trend towards a significant difference between ambiguous (face A/B) as compared to face B (t (23) = -1.87, p = .07). We did not find significant effects of ambiguous (face A/B) compared to face A, or the two non-ambiguous stimulus conditions (mostly face A, mostly face B) (t (23) > -1.32, p > .200)

Our ANOVA yielded non-significant effects of group (F (2, 22) = .016, p = .90) and a non-significant stimulus type x group interaction (F (2, 22) = .418, p = .66).

This analysis resulted in non-significant effects [(stimulus type: F (1, 22) = 1.58, p = .22) group (F (1, 22) = .158, p = .69) task x group (F (1, 22) = .470, p = .63)]. Our follow-up t-tests did not reveal significant differences between ambiguous (face A/B) and non-ambiguous (mostly face A, mostly face B) faces (t (23) < 1.637, p > .11). Our secondary ANOVA with non-ambiguous faces (mostly face A, mostly face B) and group as factors also resulted in non-significant effects (F (2, 22) < 1.99, p > .17) (see Figure 13).

Experiment 2: Task-dependent Processing

Behavior:

Our accuracy data analysis resulted in non-significant effects [task: (F (1, 19) = .113, p = .74), group: (F (1, 19) = .3.05, p = .09) task x group: (F (1, 19) = .024, p = .88)] (see Figure 14).

Our reaction time data also yielded non-significant effects [task: F (1, 19) = .347, p = .56) group: F (1, 19) = .040, p = .84, task x group: (F (1, 19) = .756, p = .39)]. Upon visual inspection we observed a crossover pattern. In this small sample we confirmed that there were not differences in reactivity in controls (t (7) < -.247 > .81) or alexithymia participants (t (12) < -1.15, p > .271) (see Figure 15).

Heart Rate:

We found a significant task x group interaction (F (1, 22) = 9.55, p = .005). In controls, we found faster heart rate reactivity to fearful faces during emotion identification and slower heart rate reactivity to fearful faces during gender identification. In alexithymia participants this pattern of reactivity to fearful faces was reversed. Follow-up t-tests found a trend towards a significant difference between reactivity during gender and emotion tasks in controls (t (12) = 2.07, p = .06) and alexithymia participants (t (10) = 2.27, p = .05). Follow-up one sample tasks were conducted in order to examine reactivity to fearful faces in controls and alexithymia participants. In controls, we found no difference in reactivity to fearful faces during

emotion identification (t (10) = -1.13, p = .286), but significantly slower heart rate reactivity to fearful faces during gender identification (t (10) = 3.34, p = .01). In alexithymia participants this pattern of reactivity was reversed for emotion identification (t (12) = -1.14, p = .27) and gender identification tasks (t (12) = 1.86, p = .09). We did not find a significant main effect of task (t (1, 22) = .144, p = .71) or group (F (1, 22) = .010, p = .92) (see Figure 16).

Skin Conductance.

This analysis resulted in non-significant effects [(task: F (1, 22) = .185, p = .67); group: (F (1, 22) = .849, p = .37) task x group: (F (1, 22) = 1.09, p = .31)], suggesting no differences in the percentage of SCRs in response to fear minus neutral faces for the emotion identification and gender identification tasks (see Figure 17).

Table 1: Behavior

Study	Task Design		sk /pe	Behavio	r
		I	E	Accuracy	Reaction Time
Jessimer & Markham, 1997	Happy, surprise, sad, fear, disgust, anger (Ekman & Friesen, 1976)		X	A < C All emotions	NA
Kano, 2003	Happy, sad, anger, neutral 33% (mild), 67% (moderate) 100% (intense)		X	A < C Sadness and disgust	NA
Lane et al., 2000	PAT (Happy, surprise, sad, fear, disgust, anger, neutral)		X	Hi A < Med A < Lo A All emotions	NA
Lane et al., 1996	PAT (Happy, surprise, sad, fear, disgust, anger, neutral)		X	Hi A < Med A < Lo A All emotions	NA
Mann et al., 1994	Happy, surprise, sad, fear, disgust, anger (Ekman & Friesen, 1976)		X	A < C All emotions	NA
McDonald & Prkachin,1990	Happy, surprise, sad, fear, disgust, anger (Ekman & Friesen, 1976)		X	A < C posed response to negative slides; posed happiness expression	NA
Panday & Mandel, 1997	Happy, surprise, sad, fear, disgust, anger, neutral (Ekman & Friesen, 1976)		X	A < C (trend) A < C verbal expressions	A < C
Parker et al., 1993	Distress, surprise, fear, disgust, anger, contempt, interest, shame, enjoyment (Izard, 1971)		X	A < C except distinguishing between shame and disgust	NA
Parker et al., 2005	Sad, fear, anger, neutral (Ekman & Friesen, 1976)		X	A < C 1 sec exposure A = C 3 sec exposure	A < C
Vermeulen, et al., 2006	Happy, sad, anger; positive/negative words	X		NA	A < C anger as a prime; for word and faces
Lundh & Simonsson- Sarnecki, 2002	Illness, negative and neutral words	X		NA	A < C for illness versus negative words

Note: I = implicit; E = explicit; A= alexithymia; C = controls; NA= Not available; PAT = 140-item task in which participants match sentences and words, faces and words, sentences and faces, and faces and emotional scenes (Lane et al., 2000).

Table 2: Physiology

Study	Task Design	Task	к Туре	Physio	logy
	U	Implicit	Explicit	SCR	HR
Friedlander et al., 1997	Passive viewing of disgusting images	X		A > C	A > C
Roedema & Simons, 1999	Passive viewing of IAPS	X		A < C	A > C
Whemer et al., 1995	Passive viewing of emotional images (i.e. injury, accident)	X		A < C	A < C
Franz et al., 2003	Passive viewing of horror film clips	X		A < C	A < C at baseline
Infrasca, 1997	Passive viewing of emotional films (i.e., injury, surgery)	X		A > C	
Stone & Nielson, 2001	Passive viewing of oral surgery video	X		A > C	A = C
Newton & Contrada, 1994	Speech task		X		A < C
Rabavilas, 1987	Stress task using novel tones		X	A > C	
Martin & Pihl, 1985	Stress quiz		X		A > C
Fukunishi et al., 1999	Stress task- mental arithmetic		X		A > C
Papciak et al., 1985	Stress quiz		X		A > C
Linden et al., 1996	Social interaction; hand grip, mental arithmetic		X		A < C
Neumann et al., 2004	Recall of angry events		X		A < C

Note: A = alexithymia; C = control; IAPS = International Affective Picture System, a set of emotional images/scenes that have been rated for their pleasantness (positive, negative, neutral valence) and intensity (high, low arousal).

Table 3: Neuroimaging

Study	Task Design	Task	Type	Function	
		Implicit	Explicit	MPFC	Amygdala
Berthoz et al., 2002	IAPS Passive viewing	X		A < C negative images (BA 9) A > C positive images (BA 24,32)	A = C
Kano et al., 2003	Happy, sad, anger, neutral 33% (mild), 67%(moderate), 100% (intense)	X		A < C (Anger – Neutral) (BA 24)	A = C
Leweke et al., 2004	IAPS (fear, disgust, neutral) Passive viewing	X		A < C (Disgust- Neutral)	A = C
Huber et al., 2002	Emotional memories happy, sad, neutral		X	A < C (Happy-Neutral; Sad-Neutral) (BA 31, 32)	A = C
Mantani et al., 2005	Emotional imagery of events: past (happy, sad, neutral) and future (happy, sad, neutral)		X	A < C (Past Sad)	A = C

Note: A = alexithymia; C = controls; BA = Brodmann's area (given when available); IAPS = International Affective Picture System, a set of emotional images/scenes that have been rated for their pleasantness (positive, negative, neutral valence) and intensity (high, low arousal).

Table 4: Demographic, behavioral and physiological means and standard errors for all participants

Experiment 1: Categorical Processing

Demographics

Group	N	Gender	TAS-20	STAI	NEO_N	NEO_E
Alexithymia	29	7M	66.1 (.80)	63.4(2.0)	107.7(4.7)	121.0(3.4)
Control	28	10M	42.6 (1.2)	53.0(1.3)	90.1(4.0)	113.8(3.9)

Baseline Physiology:

Group	Baseline
Alexithymia	788.4(21.3)
Control	831.0 (25.6)

Emotion Task

Group	Slope	CatBound	Heart Rate				SCR	
			A	A/B	В	A	A/B	В
Alexithymia	1.17(.23)	46.3(1.5)	.98 (5.4)	10.7(3.6)	3.3(4.7)	.21(.04)	.20(.04)	.28(.05)
Control	1.37(.25)	46.6(1.8)	17.5(7.1)	18.6(4.5)	-1.2(6.4)	.15(.04)	.14(.03)	.19(.04)

Identity Task

Group	Slope	CatBound	Heart Rate			SCR		
			A	A/B	В	A	A/B	В
Alexithymia	.96(1.1)	48.1(1.1)	19.2(6.0)	38.4(5.5)	13.9(4.5)	.20 (.04)	.24 (.04)	.15 (.04)
Control	.98(1.1)	50.3(1.2)	10.6(6.4)	46.5(5.1)	17.9(4.6)	.20(.06)	.28 (.04)	.23 (.04)

Experiment 2: Task-dependent Processing

Gender Identification (Implicit Task)

Group	RT		Accu	ıracy	Heart	Rate	SO	CR
	Fear	Neutral	Fear	Neutral	Fear	Neutral	Fear	Neutral
Alexithymia	1108.89(38.3)	1103.08(39.5)	.45(.02)	.47(.02)	10.7 (3.4)	16.5(3.5)	.15(.03)	.19(.03)
Control	1047.47(54.5)	1002.4(59.6)	.53(.03)	.51(.02)	22.7(5.5)	15.3(4.9)	.14(.03)	.17(.04)

Emotion Identification (Explicit Task)

	· · · · · · · · · · · · · · · · · · ·							
Group	R	RT	Accı	ıracy	Hear	t Rate	SC	R
	Fear	Neutral	Fear	Neutral	Fear	Neutral	Fear	Neutral
Alexithymia	1056.49(33.5)	1064.82(33.3)	.93(.01)	.93(.01)	17.0(3.0)	10.3(3.1)	.19 (.03)	.22(.04)
Control	963.93(71.6)	1015.07(67.5)	.92(.02)	.90(.03)	18.5(4.5)	25.3(5.5)	.16 (.03)	.20 (.04)

Table 5: Alexithymia studies using the TAS-20 and reporting participant mean and SD

Study	Type	Measure	Alexithymia	Control	Psychopathology
			Mean (SD)	Mean (SD)	
Panday & Mandel,	Behavior	TAS-20	69 (1.5)	33 (1.5)	none
1997					
Parker et al., 1993	Behavior	TAS-20	60.06 (6.09)	34.67 (5.55)	All comers
Franz et al., 2003	Physiology	TAS-20	56.7 (4.3)	32.9 (7.7)	none
Stone & Nielson, 2001	Physiology	TAS-20	≥54 (highest = 75)	\leq 37 (lowest =23)	none
Kano et al., 2003	Neuroimaging	TAS-20	64.2 (3.6)	40.5 (5.7)	none
Berthoz et al., 2002	Neuroimaging	TAS-20	61.12 (5.2)	33.12 (3.40)	none

Table 6: Demographic, behavioral and physiological means and standard errors for matched participants

Demographics

Group	N	Gender	TAS-20	STAI	NEO_N	NEO_E
Alexithymia	13	3M	66.0 (1.7)	58.1 (3.0)	85.0 (8.1)	120.0 (5.2)
Control	12	4M	41.6 (2.1)	52.0 (2.3)	98.8 (7.9)	123.4 (4.3)

Baseline Physiology:

Group	Baseline
Alexithymia	766.3 (25.1)
Control	827.3 (46.9)

Experiment 1: Categorical Processing

Emotion Task

Group	Slope	CatBound		Heart Rate	;	SCR		
			A	A/B	В	A	A/B	В
Alexithymia	1.08 (.31)	46.4 (1.7)	6.9 (7.5)	13.6 (4.2)	2.7 (6.5)	.24 (.06)	.25 (.06)	.36 (.07)
Control	1.65 (.42)	47.9 (3.3)	22.6 (8.5)	21.3 (9.4)	07 (11.0)	.24 (.09)	.24 (.08)	.27 (.08)

Identity Task

Group	Slope	CatBound		Heart Rate		SCR			
			A	A/B	В	A	A/B	В	
Alexithymia	1.03 (.31)	46.9 (1.7)	31.9 (8.6)	42.1 (8.7)	21.3 (5.4)	.24 (.04)	.32 (.05)	.27 (.07)	
Control	1.15 (.32)	51.8 (1.6)	6.2 (11.7)	33.1 (8.2)	5.6 (4.2)	.29 (.10)	.36 (.07)	.23 (.10)	

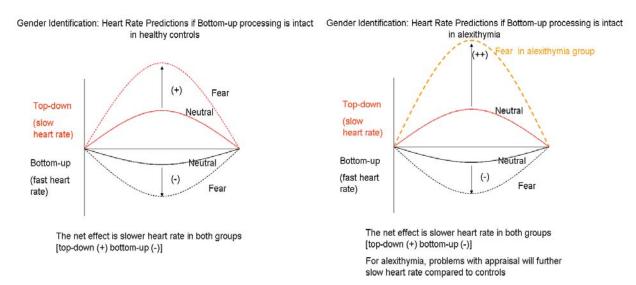
Experiment 2: Task-dependent Processing

Gender Identification (Implicit Task)

Group	R	Accuracy		Heart Rate		SCR		
	Fear	Neutral	Fear	Neutral	Fear	Neutral	Fear	Neutral
Alexithymia	1153.8 (65.7)	1129.96 (60.0)	.42 (.03)	.45 (.02)	12.2 (4.5)	19.1 (5.9)	.20(.04)	.22(.04)
Control	1120.0 (100.6)	1131.3 (101.9)	.58 (.06)	.51 (.03)	18.2 (3.7)	4.9 (4.3)	.21(.06)	.23(.07)

Emotion Identification (Explicit Task)

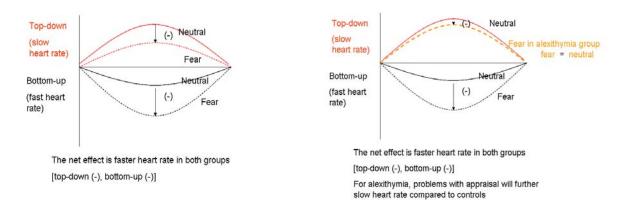
Group	RT		Accuracy		Heart Rate		SCR	
	Fear	Neutral	Fear	Neutral	Fear	Neutral	Fear	Neutral
Alexithymia	1037.7 (48.3)	1087.3 (43.7)	.93 (.01)	.94 (.03)	21.0 (4.9)	9.6 (3.1)	.17(.04)	.24(.04)
Control	1110.1 (143.4)	1107.1 (146.6)	.96 (.02)	.88 (.05)	14.3 (6.8)	24.2 (6.8)	.21(.06)	.21(.06)



Red lines = top-down processing; dotted red lines = fear, black lines = bottom-up processing; dotted black lines = fear; yellow dashed line = fear during top-down processing in alexithymia

Figure 1: Gender Identification: schematic drawing representing heart rate predictions for control and alexithymia participants if bottom-up processing is intact

Hypothesis: Gender identification (implicit): In control subjects, the emotional content of the fearful versus neutral faces should be distracting, thereby increasing the amount of attention necessary to perform the task. In controls, increased attentional demands serve to slow heart rate, thereby opposing the faster heart rate that should be induced by exposure to fearful versus neutral faces. In alexithymia, if bottom-up processing is intact participants are also vulnerable to distraction. Therefore this group should also be susceptible to the opposing influence of heart rate slowing associated with increased attentional demands. In fact, since appraisal has been considered to be more difficult in alexithymia, individuals with alexithymia may be subject to greater increases in the attentional demands of the task, when compared to controls. This increased attentional demand of emotional faces should serve to slow heart rate more for alexithymia versus control participants.



Red lines = top-down processing; dotted red lines = fear, black lines = bottom-up processing; dotted black lines = fear; yellow dashed line = fear during top-down processing in alexithymia

Figure 2: Emotion Identification: schematic drawing representing heart rate predictions for control and alexithymia participants if bottom-up processing is intact

Hypothesis: Emotion identification (explicit): In control subjects, emotional content (e.g., a fearful face) should induce a faster heart rate. This physiological change may serve as a "somatic marker" that facilitates responding; thereby reducing the attentional demands that can produce heart rate slowing. For this task, the emotional and cognitive factors both predict a relative shift towards faster heart rate for fearful vs. neutral faces. In alexithymia, if bottom-up processing is intact then the heart rate should be faster for fearful vs. neutral faces. However, problems with top-down processing may interfere with the capacity to use heart rate as a "somatic marker" that reduces the attentional demands associated with labeling a fearful vs. a neutral face. In this case the amount of attention allocated to fearful and neutral faces may be about the same. Overall, the difference between heart rate responses to fearful vs. neutral faces may be smaller in individuals with alexithymia, compared to controls because the faster heart rate associated with the bottom-up signal is not complemented by a reduction in the heart rate slowing associated with labeling a fearful versus neutral face.

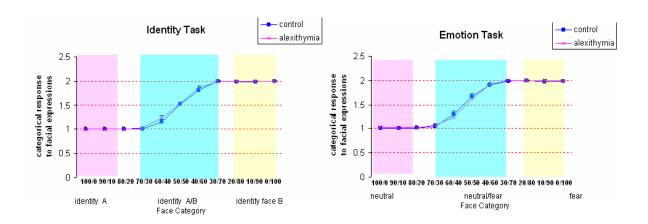


Figure 3: Experiment 1, Plot of behavioral responses

S-shaped plot of behavioral responses to faces ranging from identity A to identity B in the identity condition and from neutral (face A) to fearful (face B) in the emotion condition for alexithymia and control groups. On the x-axis is the degree to which the face represents face A versus face B (%face A / %face B). The shaded pink area represents responses to stimuli with a high percentage of face A stimuli (100/0, 90/10, 80/20). The shaded blue area represents responses to a more even mixture of face A/B stimuli (70/30, 60/40, 50/50, 40/60, 30/70). The shaded yellow area represents responses to stimuli with a high percentage of face B stimuli (80/20, 90/10, 100/0). The y-axis represents the behavioral response to the face as belonging to one of two categories (1 = face A response; 2 = face B response). On the y-axis, the red dotted line at y = 1.5 represents an equal portion of face A and face B responses.

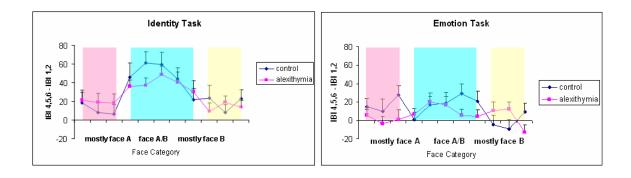
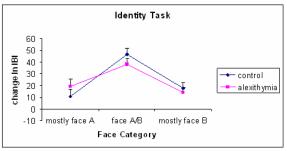


Figure 4: Experiment 1, Plot of heart rate responses at each IBI

Plot of heart rate responses to faces ranging from mostly face A to mostly face B in the identity condition and from neutral (face A) to fearful (face B) in the emotion condition for alexithymia and control groups. On the x-axis is the degree to which the face is labeled face A versus face B (mostly face A, face A/B, mostly face B). The shaded pink area represents responses to stimuli with a high percentage of face A stimuli (100/0, 90/10, 80/20). The shaded blue area represents responses to a more even mixture of face A/B stimuli (70/30, 60/40, 50/50, 40/60, 30/70). The shaded yellow area represents responses to stimuli with a high percentage of face B stimuli (80/20, 90/10, 100/0). The y-axis represents the average mean difference between the three IBIs following stimulus presentation (IBI 4,5,6) and the two IBIs preceding the stimulus presentation (IBI 1,2).



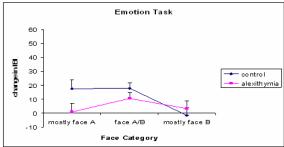
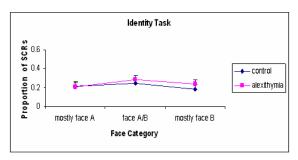


Figure 5: Experiment 1, Plot of heart rate responses

Plot of heart rate responses to faces ranging from mostly face A to mostly face B in the identity condition and from neutral (face A) to fearful (face B) in the emotion condition for alexithymia and control groups. On the x-axis is the degree to which the face is labeled face A versus face B (mostly face A, face A/B, mostly face B). The y-axis represents the change in IBI (IBI 4,5,6 – IBI 1,2). Values above the x-axis indicate slower heart rate, while values below the x-axis indicate faster heart rate.



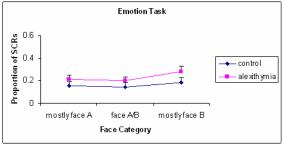


Figure 6: Experiment 1, Plot of skin conductance responses

Plot of the proportion of skin conductance responses (SCRs) to faces ranging from mostly face A to mostly face B in the identity condition and from neutral (face A) to fearful (face B) in the emotion condition for alexithymia and control groups. On the x-axis is the degree to which the face is labeled face A versus face B (mostly face A, face A/B, mostly face B). The y-axis represents proportion of SCRs.

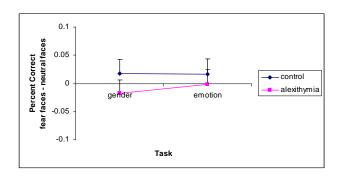


Figure 7: Experiment 2, Plot of task accuracy

Plot of task accuracy in response to fearful versus neutral faces. On the x-axis is the task (gender = gender identification/implicit task; emotion = emotion identification/explicit task). On the y-axis is the percent correct responses to fearful versus neutral facial expressions. Values above the x-axis indicate greater accuracy to fearful versus neutral faces, while values below the x-axis indicate poorer accuracy to fearful versus neutral faces.

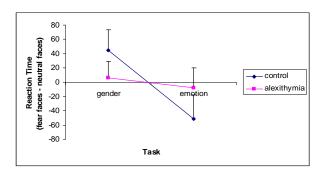


Figure 8: Experiment 2, Plot of reaction time

Plot of reaction time in response to fearful versus neutral faces. On the x-axis is the task (gender = gender identification/implicit task; emotion = emotion identification/explicit task). On the y-axis is the reaction time in response to fearful versus neutral facial expressions. Values above the x-axis indicate slower reaction time to fearful versus neutral faces, while values below the x-axis indicate faster reaction time to fearful versus neutral faces.

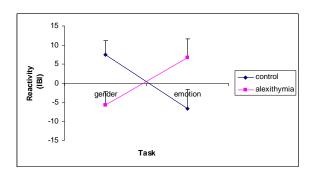


Figure 9: Experiment 2, Plot of heart rate reactivity

Plot of heart rate reactivity in response to fearful versus neutral faces. On the x-axis is the task (gender = gender identification/implicit task; emotion = emotion identification/explicit task). On the y-axis is the heart rate reactivity (measures as IBI 4,5,6- IBI 1,2) in response to fearful versus neutral facial expressions. Values above the x-axis indicate slower heart rate to fearful versus neutral faces, while values below the x-axis indicate faster heart rate to fearful versus neutral faces.

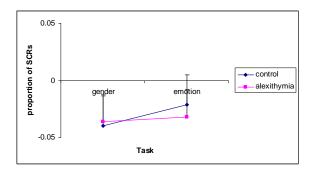


Figure 10: Experiment 2, Plot of skin conductance responses

Plot of Skin Conductance Response (SCR) in response to fearful versus neutral faces. On the x-axis is the task (gender = gender identification/implicit task; emotion = emotion identification/explicit task). On the y-axis is the proportion of skin conductance responses to fearful versus neutral facial expressions. Values above the x-axis indicate a greater number of SCRs to fearful versus neutral faces, while values below the x-axis indicate a smaller number of SCRs to fearful versus neutral faces.

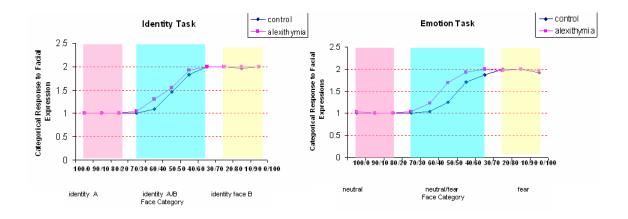
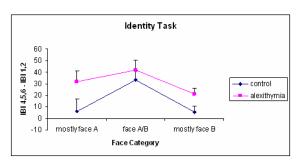


Figure 11: Experiment 1, Plot of behavioral responses in matched pairs

S-shaped plot of behavioral responses to faces ranging from identity A to identity B in the identity condition and from neutral (face A) to fearful (face B) in the emotion condition for matched groups. On the x-axis is the degree to which the face represents face A versus face B (%face A / %face B). The shaded pink area represents responses to stimuli with a high percentage of face A stimuli (100/0, 90/10, 80/20). The shaded blue area represents responses to a more even mixture of face A/B stimuli (70/30, 60/40, 50/50, 40/60, 30/70). The shaded yellow area represents responses to stimuli with a high percentage of face B stimuli (80/20, 90/10, 100/0). The y-axis represents the behavioral response to the face as belonging to one of two categories (1 = face A response; 2 = face B response). On the y-axis, the red dotted line at y = 1.5 represents an equal portion of face A and face B responses.



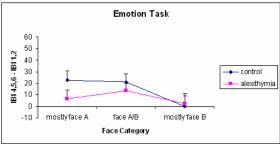


Figure 12: Experiment 1, Plot of heart rate responses in matched pairs

Plot of heart rate responses to faces ranging from mostly face A to mostly face B in the identity condition and from neutral (face A) to fearful (face B) in the emotion condition for matched groups. On the x-axis is the degree to which the face is labeled face A versus face B (mostly face A, face A/B, mostly face B). The y-axis represents the change in IBI (IBI 4,5,6 – IBI 1,2). Values above the x-axis indicate slower heart rate, while values below the x-axis indicate faster heart rate.

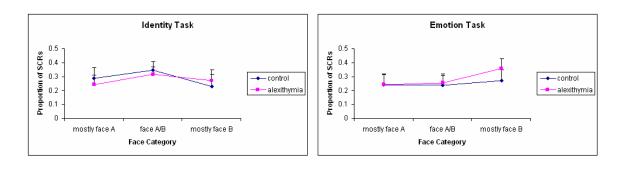


Figure 13: Experiment 1, Plot of skin conductance responses in matched pairs

Plot of the proportion of skin conductance responses (SCRs) to faces ranging from mostly face A to mostly face B in the identity condition and from neutral (face A) to fearful (face B) in the emotion condition for matched groups. On the x-axis is the degree to which the face is labeled face A versus face B (mostly face A, face A/B, mostly face B). The y-axis represents proportion of SCRs.

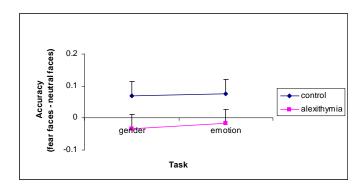


Figure 14: Experiment 2, Plot of task accuracy in matched pairs

Plot of task accuracy in response to fearful versus neutral faces for matched groups. On the x-axis is the task (gender = gender identification/implicit task; emotion = emotion identification/explicit task). On the y-axis is the percent correct responses to fearful versus neutral facial expressions. Values above the x-axis indicate greater accuracy to fearful versus neutral faces, while values below the x-axis indicate poorer accuracy to fearful versus neutral faces.

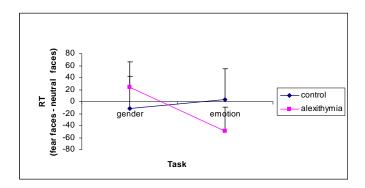


Figure 15: Experiment 2, Plot of reaction time in matched pairs

Plot of reaction time in response to fearful versus neutral faces for matched groups. On the x-axis is the task (gender = gender identification/implicit task; emotion = emotion identification/explicit task). On the y-axis is the reaction time in response to fearful versus neutral facial expressions. Values above the x-axis indicate slower reaction time to fearful versus neutral faces, while values below the x-axis indicate faster reaction time to fearful versus neutral faces.

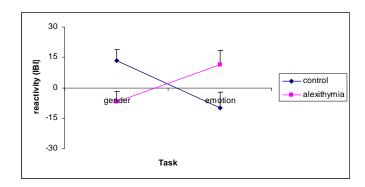


Figure 16: Experiment 2, Plot of heart rate reactivity in matched pairs

Plot of heart rate reactivity in response to fearful versus neutral faces for matched groups. On the x-axis is the task (gender = gender identification/implicit task; emotion = emotion identification/explicit task). On the y-axis is the heart rate reactivity (measures as IBI 4,5,6- IBI 1,2) in response to fearful versus neutral facial expressions. Values above the x-axis indicate slower heart rate to fearful versus neutral faces, while values below the x-axis indicate faster heart rate to fearful versus neutral faces.

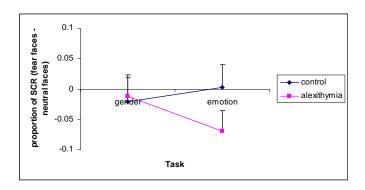


Figure 17: Experiment 2, Plot of skin conductance responses in matched pairs

Plot of Skin Conductance Response (SCR) in response to fearful versus neutral faces for matched groups. On the x-axis is the task (gender = gender identification/implicit task; emotion = emotion identification/explicit task). On the y-axis is the proportion of skin conductance responses to fearful versus neutral facial expressions. Values above the x-axis indicate a greater number of SCRs to fearful versus neutral faces, while values below the x-axis indicate a smaller number of SCRs to fearful versus neutral faces.