Exploring the Relationship between Alexithymia and Illness: Individual Differences in Physiological Reactivity and Symptom Reporting

Nelson J. Byrne

Department of Psychology

McGill University, Montreal

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Abstract

Alexithymia is a personality construct characterized by a deficit in the cognitive processing and regulation of emotion. The well documented association of alexithymia with illness has prompted investigations into pathways that might explain this association, such as physiological mechanisms or increased illness behaviour. The first aim of the present research was to explore the hypothesis that alexithymia has the potential to lead to disease because of peculiarities in physiological reactivity to emotional stimuli. The second aim was to examine the influence of alexithymia on self-report of somatic symptoms associated with a controlled medical event, and to extend assessment of cardiovascular reactivity to a qualitatively different emotional stressor in a quasi-naturalistic setting, thereby increasing generalizability. In Study One, film clips selected to induce positive and negative emotions were presented to high- and low-alexithymia men and women while physiological and cognitive-experiential facets of emotional responding were monitored. High-alexithymia males displayed reduced respiratory sinus arrhythmia in response to emotion induction, significantly different from the increase shown by others. This effect was independent of emotional valence or specificity. High-alexithymia individuals also reported lower ratings of positive emotions, greater confusion, and differences in thoughts related to evaluating and regulating their affective state. In Study Two, investigation of cardiovascular and emotional reactivity was extended beyond the laboratory to the blood donation clinic, which also provided a unique opportunity to evaluate the association between alexithymia and self-report of vasovagal symptoms. Alexithymia was positively associated with reported anxiety, pain, and vasovagal symptoms, and also with greater

increases in systolic blood pressure in anticipation of blood donation. Objective indices of vasovagal reactions did not concur with self-report, suggesting an increased tendency in individuals higher in alexithymia to report more somatic complaints. In sum, while these data provide support for associations between alexithymia and differences in autonomic reactivity, these associations do not all fit neatly within a hypo- or hyperarousal model. Further research is recommended using methods such as ambulatory monitoring to examine response to more naturally occurring emotional events. Continued investigation of illness behaviours such as symptom reporting as they relate to alexithymia is also warranted.

Résumé

L'alexithymie est un trouble de personnalité caractérisé par un déficit des processus cognitifs et de la régulation des émotions. Une association bien documentée entre l'alexithymie et la maladie a incité une investigation des pistes pouvant expliquer cette association, comme par exemple les mécanismes physiologiques ou l'augmentation des comportements maladifs. Le but premier de la présente recherche était d'explorer l'hypothèse que l'alexithymie ait le potentiel de mener à la maladie à cause de certaines particularités dans la réactivité physiologique aux stimuli émotionnels. Le but second était d'examiner l'influence de l'alexithymie sur l'auto-évaluation des symptômes somatiques associés à un événement médical contrôlé, et de poursuivre l'évaluation de la réactivité cardiovasculaire à un agent stressant qualitativement et émotionnellement différent dans un cadre quasi-naturaliste, de ce fait augmentant la généralisation. Dans la première étude, des séquences filmées, sélectionnées afin de produire des émotions positives et négatives, ont été présentées à des hommes et des femmes ayant des indices faibles et élevés d'alexithymie pendant que les aspects physiologiques et cognitifs-expérientiels de la réponse émotive étaient mesurés. Les hommes ayant un degré élevé d'alexithymie ont démontré une arythmie sinusale respiratoire réduite en réponse à la séquence émotive : cette réponse était significativement différente de l'augmentation respiratoire notée chez les autres participants. Cet effet était indépendant de la valence ou de la spécificité émotionnelle. Les individus ayant un degré élevé d'alexithymie ont aussi rapporté des évaluations basses de leurs émotions positives, des niveaux plus élevés de confusion, et des différences dans leurs pensées reliées à l'évaluation et à la régulation de leur état affectif. Dans la seconde étude, l'investigation de la réactivité cardiovasculaire et

émotionnelle a été étendue à l'extérieur du laboratoire vers un centre de donneurs de sang, ce qui fournit une opportunité d'évaluer une association entre l'alexithymie et l'auto-évaluation de symptômes vasovagaux. Les résultats ont démontré que l'alexithymie était associée positivement avec l'auto-évaluation de l'anxiété, de la douleur et de symptômes vasovagaux, et aussi avec des niveaux plus élevés de pression artérielle systolique lors de l'anticipation du don de sang. Des indices objectifs des réactions vasovagales ne coïncidaient pas avec l'auto-évaluation, suggérant une tendance plus prononcée chez les individus ayant un degré d'alexithymie plus élevée à rapporter plus de plaintes somatiques. En somme, bien que ces données démontrent un support pour une association entre l'alexithymie et les différences dans la réactivité autonome, ces associations ne s'accordent pas toutes soigneusement dans un model de hypo- ou hypervigilance. Des recherches supplémentaires utilisant des méthodologies telles que le monitorage ambulatoire sont recommandées afin d'examiner les réponses dans un cadre plus naturel élicitant des émotions. Des recherches plus poussées des comportements maladifs tels que les symptômes se rapportant à l'alexithymie seraient aussi justifiées.

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Contributions of Authors

I share authorship of both manuscripts presented on the following pages with my thesis supervisor, Dr. Blaine Ditto. While I am listed as first author of both papers, my contributions to each study differ slightly.

Study One was conceptualized, designed and executed by myself, with some assistance from a psychology honours student, Caroline Silverman, who I was responsible for supervising. A second honours student, Carol Zambrana, also helped with data collection later in the study. Data processing was done by myself with some assistance from volunteer research assistants. I was responsible for analysis of the data, in consultation with Rhonda Amsel and my thesis supervisor. Writing of the manuscript was done by myself, with guidance, suggestions, and some contributions from my supervisor.

As part of a larger study evaluating a treatment to reduce vasovagal symptoms in blood donors, for which Dr. Ditto is principal investigator, Study Two enlisted a team of paid research assistants. I participated fully in data collection, and conducted data analyses in consultation with Rhonda Amsel and my supervisor. I was responsible for preparation of the first draft of the manuscript with the guidance of my supervisor, and contributed substantially to two subsequent revisions through reanalyses of data and revisions to text.

Statement of Original Contributions

The research presented in the following studies contributes substantially to the literature on alexithymia in a number of ways.

Study One was among the first to consider respiratory sinus arrhythmia as part of the assessment of autonomic reactivity to emotional stimuli in alexithymics. In addition to including this previously neglected index of parasympathetic activity, Study One examined other physiological and cognitive-experiential components of emotional response in both males and females, under conditions of both positive and negative emotion induction, using stimulus materials that have been demonstrated to be effective for this purpose.

Study Two is the first reported investigation of cardiovascular reactivity in alexithymics outside of a laboratory setting, thus contributing to the generalizability of findings in this research area. The stressor is a personally relevant, controlled medical event, qualitatively different from stressors used in previous studies of this nature. Further, the induction of a limited set of symptoms in the blood donation setting provided a unique opportunity to explore patterns of symptom reporting as they relate to alexithymia, and permitted objective verification of self-reported somatic complaints.

Table of Contents

General Introduction

The idea that emotion can influence physical health is not new. Physicians and philosophers have posited since ancient times that excessive or unregulated affect can adversely influence both mental and bodily health. Hippocrates (ca. 460 B.C.) and his contemporaries theorized that both illness and emotional states could be attributed to alterations in the balance of four "humours": black bile, yellow bile, phlegm, and blood. Taking a less reductionistic approach, Galen (A.D. 131 – 201) stressed the importance of balancing the "non-naturals" including the "passions or perturbations of the soul." According to the doctrine of the non-naturals, which was incorporated in medieval medical texts, it was important for physicians to help patients keep their emotions in balance, for the sake of their bodies as well as their mental states (Jarcho, 1970). Although the scientific revolution of the nineteenth century led to a greater separation of the organic elements of disease from emotional malfunctioning, thus reinforcing a reductionistic biomedical model of disease, recently acquired knowledge of pathways through which the brain exerts its influence over the rest of the body has renewed interest in mind-body relations.

Of the many emotion-related constructs associated with health and disease, one that has received a considerable amount of attention over the past few decades is alexithymia. Literally translated from its Latin roots, alexithymia means "without words for feelings." While the term was first used by Sifneos (1973), the alexithymia construct has its origins in clinical observations and ideas described by Reusch (1948) and MacLean (1949) many years earlier. They proposed that bodily processes might be adversely affected by unmodulated states of emotional arousal, a consequence of deficits

in the capacity to represent emotions within the symbolic system of language. A cluster of characteristics related to difficulty with emotional identification and expression was observed in certain psychosomatic patients years later by Nemiah and Sifneos (Nemiah & Sifneos, 1970; Sifneos, 1967). They also observed an impoverished fantasy life and thought content characterized by a preoccupation with the details of objects and events in the external environment, similar to the *pensée opératoire* already described by Marty and de M'Uzan (1963). Nemiah, Freyberger and Sifneos' (1976) subsequent definition of the alexithymia construct thus included the externally oriented cognitive style and impoverished fantasy facets, as well as difficulty with identifying and describing feelings.

Taylor (1994) has proposed that the salient features of the alexithymia construct reflect a deficit in the cognitive processing and regulation of emotions. This is consistent with contemporary models of the cognitive development and organization of emotions. For example, according to Lane and Schwartz (1987), normal development of affect follows a sequence in which the emergence of symbolization and the acquisition of language lead to the formation of cognitive schemata of emotions of increasing complexity that gradually change the subjective experience of emotions from an awareness of bodily sensations and states of tension only to an awareness of blends of feelings and an ability to distinguish nuances of emotions. Higher degrees of alexithymia correspond to lower levels in this dimensional model. Bucci (1997b) notes, however, that the problem in alexithymia is not simply a lack of words for feelings, but rather a lack of symbols for somatic states.

Bucci (1997a) proposes a multiple code theory in which nonverbal and verbal representations of emotion develop sequentially. Nonverbal emotional schemata develop

first. These are comprised of subsymbolic processes involving the concrete sensory and motor systems, as well as symbolic imagery. The verbal emotional schemata develop later and are organized according to the symbolic format of language. The earlier stages of concrete sensory and motor processing are not abandoned when levels of formal, logical processing are attained. Taylor (2004) points out that, consistent with these theoretical formulations, the deficit underlying alexithymia is manifest not only as a difficulty in describing feelings in words, but also as an impoverished fantasy life, an associated utilitarian thought style, and a dissociation of the (subsymbolic) representations of sensory experiences and patterns of autonomic arousal from (symbolic) images and words.

In addition to these theoretical formulations, empirical evidence supports the claim that alexithymics have deficits in the cognitive processing and regulation of emotion. At least three studies that investigated differences in the perception and cognitive appraisal of nonverbal emotional stimuli have found that high-alexithymia individuals are less accurate in identifying posed facial expressions of emotion than are low-alexithymia individuals (Jessimer & Markham, 1997; Mann, Wise, Trinidad, & Kohanski, 1994; Parker, Taylor, & Bagby, 1993). Moreover, Lane et al. (1996) found that a higher alexithymia score correlated with lower accuracy rates on tasks that required matching verbal or nonverbal emotional stimuli (i.e., faces) with verbal or nonverbal emotional responses. Individuals who were categorized as alexithymic based on an empirically derived cutoff performed more poorly than other subjects on purely nonverbal matching, purely verbal matching, and mixed verbal-nonverbal matching. Lane et al. interpret their findings as being indicative of a general impairment in the capacity

for encoding and transforming emotional information. Collectively, the data from these studies support the view that alexithymia involves a deficit in symbolic representation of emotion, not simply an impairment in the use of emotion words.

With cognitive processing deficits limiting the ability to clearly and accurately identify and label emotion in themselves and others, it follows that emotion regulation strategies are also impaired in alexithymic individuals. Empirical findings, although limited, support this assertion. Schaffer (1993) developed an Affect Regulation Scale and used it to assess regulation strategies in a sample of adult psychiatric outpatients. Alexithymia was found to be positively associated with maladaptive styles of emotion regulation (e.g. bingeing on food, developing a headache), and negatively with adaptive strategies (e.g. trying to understand distressing feelings, talking to a caring person). Similarly, in a group of male parolees, Beckendam (1997) reported that alexithymia was associated with sexual and aggressive fantasies and behaviour, such as engaging in reckless activities and drinking alcohol, indicative of a maladaptive style of emotion regulation. Further evidence of an emotion regulation deficit in alexithymia has been inferred from the strong inverse relationship observed between alexithymia and emotional intelligence (Parker, Taylor, & Bagby, 2000), a construct that encompasses ability to reflect on emotions and use them in adaptive ways.

A surge in research on alexithymia over the past few decades is largely attributable to the development of brief, easily administered test instruments, in particular the widely used Toronto Alexithymia Scale (Bagby, Parker, & Taylor, 1994a; Taylor, Bagby, & Parker, 1992; Taylor, Ryan, & Bagby, 1986). However, the validity of much of this research rests upon the psychometric soundness of these instruments. A review of

assessment methods that have been used to identify alexithymic individuals is warranted before proceeding further.

Measurement of Alexithymia

The concept of alexithymia evolved from clinical observation of psychosomatic patients within a psychoanalytic tradition, a theoretical framework in which empirical research has been relatively neglected. Early attempts to develop measures proceeded hastily, and rigorous psychometric evaluation of some instruments did not occur until many years later. Measurement of alexithymia continues to be a contentious issue, with some of the most widely used measures still frequently criticized, and new ones still being introduced. As most measures of alexithymia have been reviewed previously (Bagby & Taylor, 1997b; Linden, Wen, & Paulhus, 1995; Taylor, Bagby, & Luminet, 2000), brief overviews of some the most commonly used instruments are presented below, along with a few more recently introduced measures with promising psychometric properties.

The Beth Israel Hospital Psychosomatic Questionnaire (BIQ), developed by Sifneos (1973), is a 17-item forced choice instrument completed by an interviewer. Of the 17 items, 8 key items are used in the rating of alexithymia. Adequate internal consistency (alpha coefficient = .72) has been reported but findings of test-retest reliability have been inconsistent, ranging from .20 to .72, depending on the population studied and with the higher coefficient obtained using a 4-point rating scale rather than forced choice (Keltikangas-Jarvinen, 1985; Sriram, Pratap, & Shanmugam, 1988). While most reports of interrater reliability have been high, these have varied with the experience of the raters and interview style (Linden et al., 1995), and have been criticized because of

other methodological inadequacies (Bagby & Taylor, 1997b). Numerous efforts have provided support for the validity of the BIQ, such as comparisons with scores on the Rotter Sentence Completion Task as an index of capacity to fantasize (Lesser, Ford, & Friedmann, 1979), or demonstrations of significant correlations with somatic complaint measures (Gardos, Schniebolk, Mirin, Wolk, & Rosenthal, 1984). Concurrent validity with the 26-item Toronto Alexithymia Scale has been reported in both clinical and nonclinical populations, across several languages, with correlations ranging from .39 to .77 (Taylor et al., 2000). Still, Linden et al. note that a lack of methodological rigour in many studies that have addressed the psychometric properties of the BIQ have weakened findings. Following Sriram et al.'s proposal with the goal of improving reliability and validity, Bagby, Taylor, and Parker (1994b) have developed a modified version of the BIQ by adding four new items for rating alexithymia, eliminating nonrelevant items, and changing rating from a dichotomous format to a 7-point Likert scale.

The Schalling-Sifneos Personality Scale (SPSS) is a 20-item self-report measure derived from the BIQ, with each item rated on a 4-point Likert scale, and with lower scores indicative of higher alexithymia (Apfel & Sifneos, 1979). Studies of internal consistency are detailed by Linden et al. (1995) and have yielded similar findings of inadequacy, with Cronbach's alpha generally ranging from .45 in a mixed student and community sample (Faryna, Rodenhauser, & Torem, 1986), to .57 in a group of 542 undergraduates (Bagby, Taylor, & Ryan, 1986). Test-retest reliability was reported to be good, with a correlation of .76 ($p < .001$) over a 2-week interval (Shipko & Noviello, 1984). Several factor-analytic studies have yielded congruence with the alexithymia construct (Blanchard, Arena, & Pallmeyer, 1981; Shipko & Noviello), but factors have

differed from study to study (Bagby & Taylor, 1997b), and lack of homogeneity has been a concern (Parker, Taylor, Bagby, & Thomas, 1991). Investigations of concurrent validity of the SPSS have found nonsignificant correlations with the BIQ and other alexithymia measures (Kleiger & Jones, 1980; Krystal, Giller, Jr., & Cicchetti, 1986; Paulson, 1985), or significant correlations of low magnitude (Bagby, Taylor, & Atkinson, 1988). Linden et al. review numerous validity studies, and conclude that behavioural and psychological correlates support at least moderate construct validity of the SPSS, but assessments of discriminant validity yielded equivocal findings. A revised version of the SPPS (Sifneos, 1986) failed to rectify the psychometric deficiencies of the original instrument.

The development of the 26-item Toronto Alexithymia Scale (TAS) followed a combined rational and empirical approach to test construction. Taylor, Ryan, and Bagby (1985) reviewed the literature and defined five content areas thought to reflect the substantive domain of the alexithymia construct. Forty-one test items were written based on these domains, and this pilot scale was administered to 542 undergraduate students. Following a series of factor and item analyses, 26 items were retained, and they clustered into four factors: 1) difficulty identifying and distinguishing between feelings and bodily sensations; 2) difficulty describing feelings; 3) reduced daydreaming; and 4) externally oriented thinking. Although alexithymia is seen as a dimensional construct, Taylor et al. (1988) have established cutoff scores for the TAS, with individuals scoring ≥ 74 considered alexithymic, and those scoring ≤ 62 considered nonalexithymic. Detailed reviews of studies addressing the reliability and validity of the TAS are available elsewhere (Bagby & Taylor, 1997b; Linden et al., 1995; Taylor et al., 2000). The psychometric properties of the TAS were generally superior to those of existing measures

at the time. Still, questions relating to the factor structure led to a revised version (Taylor et al., 1992) with a simplified two-factor structure, and shortly thereafter yet another revision, the twenty-item Toronto Alexithymia Scale (Bagby et al., 1994a; Bagby, Taylor, & Parker, 1994b). The remaining discussion of the TAS will focus on this most recent revision.

The TAS-20 is currently the most widely used instrument for measuring alexithymia. An initial validation study in 945 undergraduate students, and subsequent confirmations in student and psychiatric outpatient samples yielded a three-factor structure consistent with the theoretical roots of the alexithymia construct (Bagby et al., 1994a). That is, the first two factors were conceptually identical to those derived from the original TAS, namely difficulty identifying feelings (DIF) and difficulty describing feelings (DDF). The third factor was comprised of items assessing externally oriented thinking (EOT). Internal consistency coefficients were generally good (full scale Cronbach's alpha = .81), except for the EOT factor where alpha ranged from .64 to .66, and mean interitem correlations were comparatively low. Three week test-retest reliability was .77 in this student sample, and others have reported similar reliability in a psychiatric sample (Kooiman, Spinhoven, & Trijsburg, 2002). In reviewing the TAS-20, Koiiman et al. (2002) point out that although the three-factor structure has been replicated in many languages and cultures (e.g., Bach, Bach, de Zwaan, Serim, & Böhmer, 1996; Pandey, Mandal, Taylor, & Parker, 1996; Taylor, Bagby, & Parker, 2003), many EOT items had very low factor loadings and internal consistency tended to be unsatisfactorily low (alpha range .45 to .76) in most studies. Taylor et al. (2000) have argued that some claims of unstable factor structure (e.g., Erni, Lötscher, & Modestin, 1997; Loas, Otmani,

Verrier, Fremaux, & Marchand, 1996) can be attributed to the use of theory weak exploratory factor analyses rather than a confirmatory approach based on a priori models. The reliability and factorial validity of the TAS-20 has been confirmed in a large community sample of 880 men and 1053 women (Parker, Taylor, & Bagby, 2003), providing additional support for the validity of the three-factor structure and yielding internal consistency coefficients for the full scale and all individual factors above .70.

Support for the convergent validity of the TAS-20 has been provided by examining its relationship with measures of psychological-mindedness and need-for-cognition, yielding significant negative correlations as expected (Bagby et al., 1994b). Further, Bagby et al. (1994b) also reported theoretically meaningful correlations with several scales of the NEO Personality Inventory (Costa & McCrae, 1985), such as strong negative correlations with scales measuring openness to feelings and openness to fantasy, and nonsignificant correlations with agreeableness, conscientiousness, and excitement seeking, supporting the discriminant validity of the TAS-20. Evidence for concurrent validity was obtained by comparing TAS-20 scores obtained in a sample of 49 behavioural medicine outpatients to ratings by three clinicians on the BIQ (Bagby et al., 1994b), and cutoff scores were also established from this sample. Individuals scoring $\geq$ 61 are considered alexithymic, and those scoring $\leq$ 51 are considered nonalexithymic (Bagby & Taylor, 1997b).

As good as its psychometric properties appear to be when compared to some earlier instruments, the TAS-20 has not been without its critics. Vorst and Bermond (2001) argue that the TAS-20 does not provide a comprehensive operationalization of alexithymia since the factors of reduced fantasizing and reduced emotionalizing, which

have been described as essential alexithymia traits, are absent. Others concur that paucity of fantasy and imaginal thinking form an integral part of the original conceptualization of the construct (Kooiman et al., 2002; Sifneos, 1996). The developers of the TAS-20 argue, though, that the externally oriented thinking factor of the TAS-20 indirectly measures fantasizing (Bagby et al., 1994a), and that emotionalizing is not part of the original definition of alexithymia and should only be considered a correlate (Taylor et al., 2000).

Hoping to overcome what they perceived as shortcomings of the TAS-20, Bermond, Vorst, Vingerhoets and Gerritsen (1999) developed the 20-item Amsterdam Alexithymia Scale (AAS), designed to measure emotionalizing and fantasizing in addition to the three factors measured by the TAS-20. With the goal of developing two parallel versions of the questionnaire, 20 items were added to the AAS to produce the Bermond-Vorst Alexithymia Questionnaire (BVAQ; Vorst & Bermond, 2001). The BVAQ consists of five subscales, each comprised of eight items rated on 5-point Likert scales, with half the items negatively keyed. Vorst and Bermond found internal consistency to be at acceptable levels in Dutch, German, and English samples of university students, with total scale Cronbach's alpha at around .85, and mean for the subscales around .79. Principal components analyses in all three samples yielded comparable five-factor solutions. Results of confirmatory factor analyses yielded goodness of fit indices that Vorst and Bermond described as close to acceptable. However, in a 1999 study examining the replicability of the BVAQ factor structure in the English and French versions, Zech, Luminet, Rimé and Wagner (1999) found that most of the indices of goodness of fit in their confirmatory factor analyses were just below the standard criteria, although these improved when only the second set of 20 items was

used. Convergent validity data were also obtained by computing correlations between TAS-20 and comparable BVAQ subscale scores in a sample of 430 Dutch students (Vorst & Bermond). Additional validity data have most recently been gathered from a clinical sample (Müller, Bühner, & Ellgring, 2004), confirming a five-factor structure and providing additional convergent validity with the NEO Five-Factor Inventory (Costa & McCrae, 1992).

Some have argued that self-report may not be the best way to assess alexithymia. As Lane et al. (1996) point out, the alexithymia construct was developed based on observations of patients that appeared to lack the ability to evaluate their internal emotional states. They may be unaware of a deficit and be unable to report it themselves. Although it is standard testing practice to evaluate scores from any test instrument in the context of other relevant information, and common knowledge that assessment is improved when information is gathered from multiple sources using multiple methods, this is rarely done when assessing alexithymia. To encourage this practice, Haviland, Warren, and Riggs (2000) developed the Observer Alexithymia Scale (OAS), based on the assumption that alexithymics display behaviours that can be reliably reported by acquaintances and relatives. The 33 items that comprise the five subscales tap behaviours the developers report are key manifestations of alexithymia: interpersonal distance, lack of insight, somatising, lack of humour, and rigidity. In university student populations, internal consistency (coefficient alpha = .88 and .89) and 2-week test-retest reliability (.87) were good, and factor analyses supported an interpretable five-factor structure. Additional validity and reliability data have been obtained from clinical samples, and

suggest that the OAS is a promising tool (Haviland, Warren, Riggs, & Gallacher, 2001; Haviland, Warren, Riggs, & Nitch, 2002).

Consistent with an expressed need for increased availability of valid and reliable instruments to supplement data obtained through self-report, Bagby, Taylor, Parker, and Dickens (2006) recently developed the Toronto Structured Interview for Alexithymia (TSIA). An initial set of 60 interview questions was written to reflect four facets of alexithymia – the three already measured by the TAS-20, and an additional fantasy and imaginal processes facet. Following a series of pilot tests and analyses on data obtained in community and psychiatric outpatient samples, 24 items were retained. Each question is scored along a 3-point continuum, coded 0, 1, or 2, and a set of prompts or probes accompany each question to elicit information to facilitate accurate rating. Principal component analysis and confirmatory factor analysis supported a hierarchical, four-factor structure. Two lower order scales, difficulty identifying feelings and difficulty describing feelings were considered to compose a higher order domain scale, labelled affect awareness. The two lower order scales of externally oriented thinking and imaginal processes composed a second higher order domain scale, labelled operative thinking. The full TSIA and the domain and facet scales generally demonstrated acceptable levels of interrater, internal, and test-retest reliability. Further, modest correlations with the TAS-20 provided some support for concurrent validity.

The Alexithymia-Illness Association

Much of the impetus for research on alexithymia has stemmed from its association with a wide array of somatic and psychiatric illnesses. These include classical psychosomatic diseases consistent with the origins of the alexithymia construct, but also

functional gastrointestinal disorders, and somatoform disorders. Alexithymia has also been found to be associated with certain psychiatric conditions, such as panic disorder and eating disorders (Taylor, 2000). As the research presented in subsequent sections investigates primarily the nature of the relationship between alexithymia and somatic illness, the following review will not include discussion of alexithymia's association with psychiatric disorders. Furthermore, only those studies that employed psychometrically sound test instruments to assess alexithymia, in most cases one of the three sequential versions of the Toronto Alexithymia Scale, will be considered.

One of the most widely cited studies linking alexithymia to health involved a prospective examination of risk of death by a group of Finnish researchers (Kauhanen, Kaplan, Cohen, Julkunen, & Salonen, 1996). The study sample consisted of 2297 men from 42 to 60 years of age who were participating in the Kuopio Ischemic Heart Disease Risk Factor Study. Over an average follow-up time of 5.3 years, all-cause mortality, including deaths due to diseases, injuries, suicides, and homicides, was determined by linkage to a national death registry. Alexithymia was assessed using a validated Finnish translation of the 26-item TAS. Based on alexithymia score, the study sample was divided into quintiles, with 60% of study participants in the first three quintiles categorized as nonalexithymic, 20.9% in the fourth quintile considered moderately alexithymic, and the 19.1% of men in the fifth quintile all scoring above the TAS cutoff score of 74 considered to be highly alexithymic. In age-adjusted survival analyses, men in the highest alexithymia quintile had a twofold greater risk of all-cause death and a threefold greater risk of death from accidents, injury, or violence relative to men in the three lowest quintiles. While these data do not shed much light on the reason for the

association between alexithymia and mortality, consideration of a large number of potential confounds makes these findings particularly interesting. The relationship between alexithymia and death was somewhat attenuated after adjustment for socioeconomic status, behavioural and biological risk factors, social support and marital status, prior diagnosis and perceived health status, or depressive symptoms, but relative risk of all-cause mortality remained significantly higher for those in the high alexithymia quintile. A surprising finding was the strong link between alexithymia and death from external causes, for which the researchers were not able to offer explanation because the low frequency of deaths in the sample did not permit a more detailed analysis.

Research linking alexithymia with illnesses long considered as psychosomatic remains relatively scarce. While several published studies support a robust association between alexithymia and essential hypertension, only a few of these are now considered to have employed psychometrically sound measures of alexithymia. Using the TAS-20, Todarello and colleagues (Todarello, Taylor, Parker, & Fanelli, 1995) compared the rate of alexithymia in a group of 114 hypertensive patients to that in groups of 113 psychiatric outpatients and 130 normal adults. A rate of 55.3% alexithymia was found in the hypertensive group, significantly greater than the rates of 32.7% and 16.3% in the psychiatric outpatients and normal controls, respectively. Although Todarello et al. reported that age and education level also significantly predicted TAS-20 scores, and that the hypertensive group was significantly older, these demographic variables did not differentially influence TAS-20 scores across the three groups. A second more recent study conducted by a Finnish group (Jula, Salminen, & Saarijärvi, 1999) investigated whether psychological distress symptoms, anger expression, and alexithymia were

associated with elevated blood pressure. Two hundred thirty-seven newly diagnosed yet untreated hypertensive men and women between 35 and 54 years of age were compared with an age and gender stratified random population sample of 146 normotensive men and women. Alexithymia rates of 57% in hypertensive men and 46% in hypertensive women were reported, compared to 18% and 9% in normotensive men and women, based on scores on the 26-item TAS with a cutoff of 74 points indicative of alexithymia. There were no differences between the hypertensive and normotensive groups on measures of psychological distress, including anxiety, depression, and hostility, or in anger expression. As might be expected, higher age, male gender, higher sodium intake and lower physical fitness were associated with increased blood pressure, but the association between alexithymia and hypertension was independent of these variables. Together, these epidemiological studies provide support for an association between alexithymia and hypertension that cannot be accounted for by demographic or behavioural factors that have been previously implicated in this condition.

Inflammatory bowel diseases (IBD) such as ulcerative colitis and Crohn's disease have also been traditionally regarded as classical psychosomatic diseases, so it is not unexpected that alexithymia appears to play a role here as well. Porcelli and colleagues (Porcelli, Zaka, Leoci, Centonze, & Taylor, 1995) compared rates of alexithymia in a group of 112 IBD patients, 89 with ulcerative colitis and 23 with Crohn's disease, and a group of 112 normal subjects matched for gender, age, and education. Based on scores on the TAS-20, a rate of 35.7% of alexithymia was reported in the IBD group, whereas only 4.5% of the controls were categorized as alexithymic. Notably, illness duration or level of disease activity were not related to alexithymia, inconsistent with one possible alternative

explanation, that alexithymia is just a reaction to illness. Although clinical levels of alexithymia were found in only slightly more than one third of the IBD patients, perhaps casting doubt on the notion that alexithymia was strongly associated with this disease traditionally seen as psychosomatic, it has been proposed that this may signify a subform of IBD that differs psychologically from other subforms (Taylor, 2004).

An alternative explanation for the finding of only a modest association between alexithymia and IBD is that alexithymia may be associated more strongly with functional somatic symptoms than with the psychosomatic diseases. To investigate this hypothesis, Porcelli and colleagues (Porcelli, Taylor, Bagby, & De Carne, 1999) compared rates of alexithymia in a group of 112 patients with functional gastrointestinal disorders (FGID) to rates in a group of 116 IBD patients and 112 healthy individuals. The FGID group was reported to be significantly more alexithymic than the IBD group, with 66% of the FGID group scoring above the established TAS-20 cutoff of ≥ 61, while rates of alexithymia in the IBD and normal groups were 38% and 4.5%, respectively. Significant group differences in TAS-20 scores remained even after controlling for significant covariate effects of depression, anxiety, gender, education, and gastrointestinal symptoms. Although several distinct disorders fall under the FGID umbrella, including irritable bowel syndrome, functional dyspepsia, and functional abdominal pain syndrome, Porcelli et al. elected to collapse all patients into a single group, citing the high degree of symptom overlap across FGID groups. Comparison of alexithymic characteristics between FGID groups may have added to understanding of the differences in psychological contributions to the symptom clusters that define each disorder. For IBD patients, though, the presence of alexithymia does not appear to be related solely to the

presentation of active symptoms. A subgroup of asymptomatic IBD patients still had significantly higher TAS-20 scores than a group of healthy controls.

Given the high frequency of alexithymia reported in patients with FGID, larger than for any other medical condition, a closer examination of this group of disorders is merited. The most prevalent and clinically significant subgroup within the FGID spectrum is irritable bowel syndrome (IBS), a disorder that cannot be explained by structural or biochemical abnormalities (Toner et al., 1998). Alexithymia has been found to play an intriguing role in mediating symptoms of IBS. In an investigation of predictors of treatment outcome (Porcelli et al., 2003), 112 patients with FGID, half of those with IBS, followed a usual course of treatment over a period of 6 months. Treatment varied case by case and involved one or more of the following: gastrointestinal medications, diet modifications, psychotropic medications, and sessions of counselling or brief psychotherapy. Measures of alexithymia, anxiety, depression, and gastrointestinal symptoms were administered pre- and posttreatment. Posttreatment, the group was divided into improved and unimproved based on preestablished criteria. Patients who did not respond to treatment had significantly higher levels of anxiety, depression, alexithymia, and gastrointestinal symptoms at baseline assessment than those in the improved group. Further, in regression analyses both alexithymia and depression emerged as significant predictors of treatment outcome, although alexithymia was the stronger predictor. In an additional study of the influence of alexithymia and psychopathology on the way in which individuals perceive and experience symptoms, FGID patients with comorbid psychiatric disorders who were referred to a gastroenterology clinic were compared to psychiatric patients with comorbid FGID who

were referred to a psychiatry outpatient service (Porcelli et al., 2004). FGID patients with psychiatric disorders were more alexithymic and had less psychopathology than psychiatric patients with FGIDs, but the severity and frequency of gastrointestinal symptoms did not differ. Thus, in addition to predicting treatment outcome, alexithymia appears to influence choice of treatment for FGID symptoms. In keeping with the proposal that alexithymic patients are prone to functional somatic symptoms because of a tendency to amplify and misinterpret the somatic sensations that accompany emotional arousal (Taylor, 2000), they may experience more severe somatic symptoms, and seek help from or be referred to a medical rather than a psychiatric setting.

The somatoform disorders, like functional gastrointestinal disorders, are characterized by the presence of somatic symptoms that cannot be adequately explained by organic findings. A meta-analytic review of the literature on alexithymia and somatisation was recently conducted by De Gucht and Heiser (2003). These authors reviewed a total of 35 articles published since 1985 that used one of the three sequential versions of the TAS to assess alexithymia. Evidence from these studies points to a small to moderate relationship between alexithymia and somatisation. When only the difficulty identifying feelings dimension of alexithymia is considered, though, results were unanimously indicative of a significant positive relationship with a mean correlation coefficient of .35, considered a moderate effect size. De Gucht and Heiser remark, however, that most studies have used questionnaires that only check for the presence of symptoms, not whether these symptoms are medically explained or not, perhaps leading to an overestimation of somatisation. The meta-analysis also examined cross-sectional studies that have compared level of alexithymia between individuals with and without a

somatoform disorder. All studies comparing somatoform conditions to healthy controls were found to have demonstrated a significantly higher degree of alexithymia in the somatoform condition, with effect sizes ranging from moderate to large. Those studies comparing somatoform to medical or psychiatric conditions did not yield conclusive evidence of increased alexithymia in somatoform-disordered patients, possibly due to failure to consider confounding demographic variables or overall level of psychopathology.

Pathways Linking Alexithymia and Illness

While it is clear that the preponderance of evidence points unequivocally to a relationship between alexithymia and illness, many questions relating to factors responsible for this relationship remain unanswered. Traditional views about mechanisms through which alexithymia may lead to illness are perhaps best reflected in the proposals offered by Bagby and Taylor (1997a). The impairment in emotion-processing and emotion-regulation strategies that underlie alexithymia, they explain, may lead to several possible outcomes. First, because of their limited subjective awareness and inability to cognitively process emotion, alexithymic individuals may tend to focus on, amplify, and misinterpret the somatic sensations that accompany emotional arousal. This may contribute to the development of hypochondriasis and somatisation disorder. Second, failure to successfully regulate distressing emotions might result in increased autonomic nervous system and neuroendocrine responses, creating conditions conducive to the development of somatic illness. Third, inability to modulate emotion through cognitive processing may lead to discharge of tension arising from unpleasant emotional states through impulsive acts. Together with a proneness to experience undifferentiated

negative affective states, this latter set of conditions sets the stage for the development of psychiatric conditions. Thus, according to these proposals, while the presence of alexithymia may increase the likelihood of illness development, the association between alexithymia and somatisation is not specific, as any number of somatic or psychiatric conditions may result.

Several possible pathways linking alexithymia and physical illness have been explicitly outlined and critiqued in a review by Lumley, Stettner and Wehmer (1996). The pathways considered are as follows: (a) alexithymia leads to organic disease through physiological mechanisms or unhealthy behaviours; (b) alexithymia leads to illness behaviour, such as overreporting of symptoms and excessive use of health care; (c) physical illness influences psychological functioning and leads to alexithymia; (d) sociocultural and biological factors comprise a third variable which cause both alexithymia and physical illness. Interestingly, Lumley et al. conclude that most evidence suggests alexithymia influences illness behaviour, but little support was found for a pathway involving physiological mechanisms that lead to organic disease. Elements of the first two pathways, namely physiological mechanisms and symptom reporting, are particularly relevant to the research presented here, and each will be discussed in turn.

Psychophysiological Findings

For many years, psychosomatic disease was conceptualized according to Freud's model of psychoneurotic pathology, and psychosomaticists approached treatment by attempting to identify and interpret conflicts over unconscious drive-related wishes (Taylor, 2004). Not all subscribed to this model. MacLean (1949) had observed a deficit in some psychosomatic patients' ability to express their emotions verbally. He speculated

that distressing emotions bypassed their usual processing point in the brain and, rather than finding expression in words, they found expression through autonomic pathways, thereby resulting in physiological changes that could then produce physical disease. Indeed, while variations on this theme remain prevalent today, and numerous findings of heightened physiological arousal support the hypothesis that alexithymia leads to organic disease through physiological mechanisms, the data remain equivocal. Although a substantial body of research has addressed alexithymia-related physiological differences, it has been plagued by methodological inconsistencies that have yielded conflicting results and rendered comparisons between studies difficult. Moreover, as discussed earlier, valid instruments to assess alexithymia have only become available within the past twenty years. The following discussion, limited primarily to studies that have employed the widely used TAS, focuses on some of the more noteworthy psychophysiological studies of alexithymia.

Wehmer, Brejnak, Lumley and Stettner (1995) exposed 72 undergraduate students, 30 males and 42 females, to a series of emotion-provoking slides while monitoring their heart rate and electrodermal responses. Alexithymia was assessed with the 26-item TAS, and also by analyzing the emotion word content of stories written to five TAT-like printed pictures. Each of 16 slides was presented for 10 s, and study participants then were asked to describe what they were feeling as they viewed each slide. Change scores from baseline were calculated for both physiological variables measured. The criterion for a stimulus-elicited electrodermal response was an increase in skin conductance of at least .25 μmhos of change within 3 s of slide onset. Using a median split to divide the sample into high and low alexithymia groups, Wehmer et al.

reported a trend for higher alexithymia scores to be associated with less heart rate increase and fewer electrodermal responses while viewing the slides. The high alexithymia groups had a higher baseline heart rate than the low groups, but this difference was statistically significant only with the emotion word measure of alexithymia. The authors concluded that their data provide little support for the view that alexithymia may be related to situationally induced autonomic overarousal. While the multimethod assessment of alexithymia certainly represents a strength of this study, the short duration of stimulus presentation and the restricted nature of the physiological measures may have limited findings.

The hypothesis of reduced autonomic activity in alexithymia was also tested in Linden, Lenz, and Stossel's (1996) study of cardiovascular reactivity to a variety of laboratory stress tasks. Eighty university students completed several questionnaires, including the 26-item TAS, and were then asked to participate in isometric handgrip, mental arithmetic, and negative affect provocation tasks while blood pressure and heart rate were monitored. Tercile groups of low, medium, and high alexithymia scorers formed the basis of subsequent analyses. The high alexithymia group displayed consistently smaller heart rate reactivity to the three tasks than either the medium or low alexithymia groups, but there were no corresponding differential blood pressure responses. Interestingly, the mean change values for both systolic (SBP) and diastolic blood pressure (DBP) during the negative affect provocation task suggest a nonsignificant trend in the opposite direction, with higher alexithymia scores corresponding to larger increases in blood pressure. Nevertheless, they reasonably conclude that their findings support a hypoarousal theory of alexithymia, suggesting that

alexithymia may be associated with a systemically lower arousal potential that does not distinguish between emotionally challenging or physically challenging tasks. As Linden et al. note, it is possible that individuals with high alexithymia scores experience reduced reactivity on other autonomic indices not measured in their study. Unfortunately, physiological measures were limited to heart rate and blood pressure, making it difficult to establish a more detailed picture of the relative contributions of sympathetic and parasympathetic systems to the observed responses.

With the goal of conducting a psychophysiological comparison between alexithymia and repressive coping, Newton and Contrada (1994) administered the 26-item TAS to 86 females who had been previously identified as low anxious, high anxious, or repressors. Following collection of baseline electrocardiogram and blood pressure data, participants were given 3 min to mentally prepare a speech about their least desirable personality characteristic. During subsequent delivery of the speech for 3 min, blood pressure readings were obtained at 60-s intervals, and electrocardiogram was recorded continuously. Based on a median split, participants were assigned to a high or low alexithymia group for purposes of analyses. There were no significant effects of alexithymia group for baseline heart rate or blood pressure readings, but the high alexithymia group exhibited significantly smaller heart rate increases during the speech task than the low alexithymia group. An interesting component of Newton and Contrada's study was the inclusion of subjective measures of affect, which they compared to physiological response by computing discrepancy scores. They reported that high alexithymics' reports of negative affect exceeded their heart rate responses, but the reverse was true for low alexithymics. Although Newton and Contrada did not set out

specifically to investigate autonomic reactivity patterns in alexithymics, their data do seem to bolster Linden et al.'s (1996) conclusion of hypoarousal.

Experience of both self-report and physiological deficits involving the arousal dimension of emotion in alexithymics has been confirmed by others. Roedema and Simons (1999) categorized 65 undergraduate psychology students, approximately equal proportions of men and women, as either alexithymic or control based on their scores on the 26-item TAS. Participants viewed a series of 21 colour slides, each for 6 s, representing positive, negative or neutral affective content while heart rate, skin conductance, and facial electromyographic data were collected. The slides were viewed a second time to collect emotion self-report data using the Self Assessment Manikin (Lang, 1980) and through generation of a list of words. High alexithymia individuals supplied fewer emotion-related words, and indicated less variation along the arousal dimension of the SAM. Further, although tonic levels of autonomic activity did not differ between groups, alexithymics produced fewer specific skin conductance responses and less heart rate deceleration than controls. No valence-related group differences were noted. While this study does have several methodological strengths, such as the inclusion of both negative and positive stimulus materials and assessment of subjective, expressive, and physiological responses, the same criticism can be made here as with Wehmer et al.'s (1995) work. It is unclear whether brief presentation of static images is sufficient to produce emotional responses of the duration and complexity required to effectively study the cognitive processing deficit associated with alexithymia. Despite this reservation, these data offer additional support for the hypothesis that alexithymia is associated with autonomic hypoarousal.

In contrast to the previously cited studies that generally indicated reduced reactivity in alexithymic individuals, Friedlander, Lumley, Farchione, and Doyal (1997) concluded that alexithymic and nonalexithymic groups did not differ in physiological reactivity or recovery to three different laboratory stressors. Friedlander et al. selected 42 alexithymic and 42 sex and race matched nonalexithymics from 1000 college students based on scores on the 26-item TAS. Following an autogenic relaxation exercise, participants were required to perform an interpersonal speech task, mental arithmetic, or passive viewing of disgusting scenes while heart rate, skin conductance, and blood pressure were monitored. Alexithymic individuals had tonically greater electrodermal activity and generally reported more arousal and displeasure than the nonalexithymic group. For the most part, groups did not differ in reactivity or recovery to the stressors, with the exception that alexithymic women had a smaller heart rate decrease than nonalexithymic women when viewing disgusting scenes. Explicit consideration of sex differences, categorization of alexithymic and nonalexithymic participants using empirically derived cutoff scores, and measurement of subjective responses as well as three different physiological variables are strengths of this study.

Based on physiological recordings obtained from 41 high- or low-alexithymia young adults before and after exposure to an emotionally arousing or neutral videotape, Stone and Nielson (2001) concluded that alexithymics' physiological response to arousal was intact. A 3-min videotape of oral surgery served as the arousing stimulus, and physiological measures included electrodermal activity and heart rate. Participants also completed an emotion adjective rating scale to provide data relating to the subjective experience of emotion. Both the high- and low-alexithymia groups exposed to the

arousing videotape experienced significant increases in heart rate and electrodermal activity, but high-alexithymia individuals did not report concomitant increases in emotional intensity, in contrast to their low-alexithymia counterparts. Stone and Nielson also reported baseline differences in electrodermal activity, with high-alexithymia participants showing higher readings. One argument that has sometimes been given to explain the lack of reactivity in alexithymics found in some studies is the increased levels of arousal observed at baseline, implying that no increase in physiological indices in response to arousing stimuli may be attributable to a ceiling effect. Yet, Stone and Nielson report increased tonic levels of arousal, but also intact reactivity. They conclude that their overall findings offer support for a decoupling between subjective and physiological arousal when alexithymics face emotionally negative stimuli, a proposal previously put forth by Martin and Pihl (1986).

One of the first findings of increased arousal in alexithymics identified using the 26-item TAS was reported by Infrasca (1997). Differences in electrodermal activity were examined in a group of 56 psychiatric outpatients, 27 males and 29 females, during a mental arithmetic task and while watching a short film depicting medical procedures. Alexithymic patients demonstrated higher levels of electrodermic activity at baseline and under both stress conditions. Since the analyses did not consider change from baseline in response to the stressors, no conclusions about reactivity can be made.

While the majority of research on physiological reactivity in alexithymics has been conducted with university student populations, Luminet, Rimé, Bagby and Taylor (2004) recently explored the relationship between alexithymia and emotional responding in 50 older adults. To test for habituation in physiological response, heart rate and blood

pressure measurements were obtained at both the initial exposure and reexposure to the stimulus, a 6-min television program excerpt depicting a woman coping with cancer. Higher scores on the difficulty describing feelings factor of the Bermond Vorst Alexithymia Questionnaire (BVAQ) were found to be associated with increased heart rate during the film presentation at both initial and reexposure. Heart rate at baseline was not significantly associated with any of the alexithymia factors. At initial exposure, higher scores on the BVAQ externally oriented thinking factor were associated with judgement of the movie as less negative and less important. Although Luminet et al. used the BVAQ to measure alexithymia rather than the more widely accepted TAS, their work is reviewed here because of multiple strengths in their research design and the accumulation of promising psychometric data in support of the BVAQ. Still, the use of a different instrument complicates comparison. Like most of the previously reviewed studies, Luminet et al.'s work can also be criticized for the limited assessment of physiological response, restricted to heart rate and blood pressure.

Older adults, ranging in age from 53 to 83 years, also comprised the sample in Waldstein, Kauhanen, Neumann, and Katzel's (2002) study of the relation of alexithymia to select psychosocial, psychophysiological and biomedical risk factors for cardiovascular disease. Alexithymic and nonalexithymic groups were defined by top and bottom quartiles of the distribution of TAS scores in 102 individuals, 76% male. The groups did not differ in resting cardiovascular parameters, heart rate reactivity, fasting glucose and lipoprotein lipids, body mass index, social desirability, or trait anger. Participants engaged in two tasks. The first was an anger recall task that involved detailed description of a personally relevant anger-provoking incident that was identified by the

participant. The second was a hypothetical role-play scenario involving delivery of a speech to the administrator of a nursing home where staff had mistreated a close relative. Alexithymics displayed significantly greater SBP and DBP responses to the anger recall task than did nonalexithymics, even after adjustment for trait anxiety, neuroticism, hostility, anger-in, perceived stress, or social support. Task-related emotional response did not differ between groups. Given the large number of potential confounding variables considered here, these data provide support for the view that alexithymia contributes independently to psychophysiological mechanisms that may increase risk of cardiovascular disease. One caveat of Waldstein et al.'s work is the disproportionate number of males in their sample, thus limiting assessment of the potential interaction effects of gender. While limiting assessment of physiological reactivity to heart rate and blood pressure was perhaps appropriate in an investigation of cardiovascular risk, more in depth evaluation of physiological responding may have enriched their findings.

While the majority of studies reviewed to this point have relied on measurement of heart rate, blood pressure, or electrodermal activity, either alone or in combination, an exception to this limited assessment of physiological response that has generally characterized the alexithymia literature is a study conducted by Neumann, Sollers, Thayer, and Waldstein (2004). In a group of 80 young women who had completed the 26-item TAS, blood pressure, heart rate, and other cardiovascular parameters derived from impedance cardiography and heart rate variability were evaluated at rest, during a 3-min anger recall task, and a 10-min recovery period. While the methodological details pertaining to the specific parameters assessed by Neumann et al. are beyond the scope of this review, the blunted cardiovascular reactivity observed for alexithymics during anger

recall was considered to have arisen from attenuated sympathetic activation and diminished vagal withdrawal. However, alexithymia was also related to significantly slower DBP recovery. The authors interpret their findings as indicative of an association between alexithymia and prolonged cardiovascular recovery from anger. Alexithymia was not related to higher tonic autonomic functioning, nor was it associated with self-reported state anger at rest or during the anger recall task. One limitation of Neumann et al.'s study is the restricted generalizability of findings due to an all female sample.

Of the studies reviewed here that have assessed physiological responding in alexithymics, four (Linden et al., 1996; Newton & Contrada, 1994; Roedema & Simons, 1999; Wehmer et al., 1995) provide support for a hypoarousal theory which posits that alexithymia is associated with decreased autonomic reactivity. Two studies (Friedlander et al., 1997; Neumann et al., 2004) reported attenuated reactivity for alexithymic women, while intact reactivity similar to that of low alexithymics was reported by another (Stone & Nielson, 2001). A hyperarousal model of alexithymia during stress is supported by two of the studies reviewed here (Luminet et al., 2004; Waldstein et al., 2002), although the validity of Luminet et al.'s findings is perhaps questionable, as the researchers used a relatively new, although promising, measure of alexithymia. The hypothesis that alexithymia is associated with increased tonic autonomic activity is supported by three of the aforementioned studies (Friedlander et al., 1997; Infrasca, 1997; Stone & Nielson, 2001), while seven others did not find clear baseline differences (Linden et al., 1996; Newton & Contrada, 1994; Roedema & Simons, 1999; Wehmer et al., 1995; Luminet et al., 2004; Neumann et al., 2004; Waldstein et al., 2002).

The preceding review has underlined some of the methodological differences and deficiencies that have hampered progress in determining which role, if any, differences in autonomic reactivity may play in explaining the association between alexithymia and somatic illness. Limited assessment of physiological variables, failure to explicitly consider gender influences, use of different assessment instruments, or differences in stimulus materials have yielded inconsistent findings. Yet, while findings relating alexithymia to physiological reactivity diverge when individual studies are compared, some interesting trends emerge if the overall picture is examined. For example, alexithymics do not seem to respond strongly to brief stressors. However, this does not seem inconsistent with the defining feature of alexithymia – a deficit in the cognitive processing of emotion. On the contrary, the data seem to suggest greater reactivity to longer, perhaps more personally relevant stressors (e.g. older adults shown a film about cancer, recall of a personally relevant anger-provoking incident), where the issue of emotion evaluation and regulation may require more resources. Finally, interpretation of findings has been driven by competing models of hyper- or hypoarousal that perhaps do not take into account the complexity of emotional responding.

Symptom Reporting

An alternative or complement to the proposal that alexithymia leads to illness through physiological mechanisms is based on alexithymia's established association with somatisation (De Gucht & Heiser, 2003). Unfortunately, the term somatisation has been variously defined, depending on theoretical origins, with some definitions encompassing the requirement of a comorbid psychiatric diagnosis, or entailing assumptions about etiology. For example, one common distinction is that made between "presenting

somatisation" and "functional somatisation." While the former phenomenon is secondary to psychological distress, the latter is a primary phenomenon characterized by medically unexplained symptoms (De Gucht & Fischler, 2002). Regardless, the common element among definitions is the presence of somatic symptoms that cannot be adequately explained by organic findings.

In the case of alexithymia, Lumley, Stettner and Wehmer (1996) have summarized some possible mechanisms through which medically unexplained symptoms may be manifested. People with alexithymia may be overly attuned to their bodies and may therefore notice otherwise benign sensations, or somatic sensations may result from the undifferentiated arousal that accompanies alexithymia. Subsequent focus on and amplification of these sensations may result in them being experienced as symptoms of physical illness, perhaps because of misattribution to an organic rather than psychological cause. Alternatively, a response bias of excessive symptom reporting may be present because of the overlap between alexithymia and neuroticism or negative affectivity (Lundh & Simonsson-Sarnecki, 2001; Wise & Mann, 1994).

Apart from the fundamental research goal of clarifying the nature of the relationship between alexithymia and illness, investigation of symptom reporting in alexithymia is also motivated by economics. Increased perceived symptoms can result in frequent use of health care services, increasing cost and depriving those with legitimate medical conditions of necessary treatment. As Jyväsjärvi et al. (1999) point out, a relatively small proportion of the population uses a disproportionately large share of health care services, and while these "frequent attenders" usually complain of somatic symptoms, they commonly have more psychiatric disturbances than other patients. In

their investigation of alexithymia, hypochondriacal beliefs, and psychological distress in this population, Jyväsjärvi et al. found that all three of these factors were associated with frequent attendance in men, but not in women.

Frequent use of health care may reflect an increase in perceived symptoms, but it does not permit conclusions about whether or not there is a real organic cause for these symptoms. Much of the literature on medically unexplained symptoms has relied on data obtained from checklists that cannot distinguish between actual pathophysiological change versus the illness behaviour of symptom reporting. Kauhanen and colleagues (Kauhanen, Kaplan, Cohen, Salonen, & Salonen, 1994) sought to address this shortcoming in their investigation of alexithymia and coronary heart disease (CHD) in a population-based random sample of 2297 middle-aged men in Eastern Finland. Scores on the Finnish translation of the 26-item TAS were associated with a prior diagnosis of CHD, but not with greater prevalence of ischemia on an exercise tolerance test. In men with a CHD diagnosis, alexithymia was associated with decreased artherosclerosis, as measured via ultrasonography of the carotid artery. Carotid artherosclerosis has been shown to be associated with the incidence of CHD, and is considered to be an important indicator of the pathophysiological process of the disease. Further, self-reports of exertion and symptoms during the exercise tolerance test were higher as TAS score increased. Thus, although alexithymia was associated with more frequent diagnosis of CHD, there was no evidence of an association with clinical or pathophysiological indicators of the disease, suggesting that the increased likelihood of diagnosis is attributable to increased symptom reporting in alexithymics. Interestingly, the association between alexithymia and CHD diagnosis did not hold among those with the most severe

progression of artherosclerosis. Similar conclusions about increased symptom reporting were reached by Lumley, Tomakowsky, and Torosian (1997) in two studies of the relationship of alexithymia to subjective and biomedical measures of disease. Although the findings of these aforementioned studies stand up to methodological scrutiny, some caution is generally warranted in interpreting medically unexplained symptoms. Failure to identify an underlying medical condition does not invariably imply that reported symptoms are not the result of some pathophysiological process that eludes current diagnostic methods.

The notion of somatosensory amplification is one that bears further attention, as without this process it is unlikely that the somatic sensations typically associated with emotional experience would be of sufficient intensity to elicit misattribution of the sensations to physical illness. In fact, the evidence related to physiological reactivity reviewed previously seems to point more to hypoarousal in alexithymia, and even when there has been increased reactivity, the size of the increase has been relatively small. The relationship between somatosensory amplification and alexithymia was addressed directly by Wise and Mann (1994) in 101 psychiatric outpatients, 53 men and 48 women, with diagnoses of mostly mood and anxiety disorders. A 10-item somatosensory amplification self-report inventory was administered along with the 26-item TAS, and other instruments including a measure of neuroticism. Partial correlation coefficients controlling for depression revealed a positive correlation between TAS score and somatosensory amplification, but when the sample was stratified by gender, the association was maintained only for females. Wise and Mann conclude that while

alexithymia and somatosensory amplification are interrelated, they occur within a broader setting of negative affectivity as measured by neuroticism.

Indeed, negative affectivity, neuroticism and alexithymia appear to interact in meaningful ways within the context of symptom reporting. In a sample of 377 primary care patients, neither neuroticism nor alexithymia were found to have a significant direct effect on medically unexplained symptoms (De Gucht, Fischler, & Heiser, 2004). Rather, the neuroticism effect was mediated by high negative affect and low positive affect, while the alexithymia effect was mediated by low positive affect. The authors suggest that neuroticism and alexithymia may be vulnerability factors that predispose people to experiencing negative affects or an inability to experience positive affects, which in turn may lead to symptoms. In contrast with general alexithymia, the difficulty identifying feelings factor of the TAS-20 did have a significant direct effect on medically unexplained symptoms. A similar result was obtained by Deary, Scott, and Wilson (1997). In their investigation of the contribution of multiple health related psychological constructs to medically unexplained symptoms, they reported that despite a large general latent trait of negative affectivity that underlay most of the measured variables, the difficulty identifying feelings and difficulty describing feelings facets of the TAS-20 comprised one factor in their structural equation model, and contributed independently to the variance in medically unexplained symptoms.

Rationale for Present Research

An overview of the literature suggests that evidence supporting the relationship between alexithymia and somatic illness is substantial, though the nature and direction of the association remains unclear. Numerous pathways have been identified that may

account for this relationship, including possible differences in physiological reactivity in alexithymics, or differences in illness behaviours such as symptom reporting. Closer examination of studies investigating these pathways reveal divergent findings, and a host of methodological concerns, not the least of which is the validity of instruments used to assess alexithymia.

Research that has addressed the question of whether physiological differences may predispose alexithymics to illness continues to improve, particularly since the development of the TAS-20. However, several methodological issues remain problematic. First, most previous studies have restricted measurement of physiological variables to heart rate, blood pressure, or skin conductance, or a combination of these. These measures provide information about sympathetic nervous system (SNS) response, or the response of end organs, such as the heart, that receive mixed input from both the SNS and the parasympathetic system (PNS). Yet, at the physiological level, emotional responding is known to involve a complex interaction of sympathetic and parasympathetic systems (Berntson, Cacioppo, & Quigley, 1991). Second, the fact that alexithymia is defined by a deficit in the cognitive processing of emotion is not often reflected in the choice of stimulus materials employed. These have frequently been nonemotional in nature, such as mental arithmetic, or of short duration, such as static images on slides. Even when attempts have been made to induce more enduring emotional responses, the focus has usually been restricted to negatively valenced materials. Further, even though emotional responding is known to include physiological, behavioural, and cognitive-experiential components, relatively few studies have evaluated more than one component.

Research has only recently begun to study the relationship between respiratory sinus arrhythmia (RSA), a response solely driven by the PNS, and emotion. RSA refers to rhythmic fluctuations in heart rate associated with respiration, which results from innervation by the tenth cranial nerve, the vagus. Heart rate increases with attenuation of vagal activity during inspiration, and slows with restored vagal input during expiration. The vagal fibers responsible for RSA originate in the medullary nucleus ambiguous and terminate in the sinoatrial node of the heart. Porges (1995) has proposed a polyvagal theory, arguing that nucleus ambiguous fibers evolved to facilitate the complex system of emotion responses seen in mammals. In brief, it has been proposed that cardiac vagal activity may serve as an organizing construct for individual differences in the expression and self-regulation of emotion. Respiratory sinus arrhythmia then, as an index of cardiac vagal activity, provides a noninvasive window into the relationship between this vagal circuit and affective experience.

Regarding the issue of appropriate stimulus selection for the evaluation of emotional responding, various methods of inducing emotion in the laboratory have been tested and reviewed (Westermann, Spies, Stahl, & Hesse, 1996). Induction of both positive and negative mood has been accomplished most successfully using film clips or stories. Methods for using films for this purpose, including a collection of excerpts shown to reliably induce particular emotions, have been elaborated by Gross and Levenson (1995; 1997).

Study One aimed to overcome some of the methodological limitations of previous attempts at evaluating the relationship between alexithymia and emotional responding. Film clips preselected to induce a variety of positive and negative emotions served as

stimulus materials, and multiple physiological variables were measured, including RSA. Evaluation of emotional responding was also multimodal, and included the cognitive-experiential as well as the physiological component.

In addressing the question of differences in symptom reporting associated with alexithymia, research has been hampered by the use of questionnaires that can only check for symptoms, not whether these symptoms are medically explained or not. Studies of symptom reporting that have endeavoured to identify biological indicators of disease are rare, perhaps because of the resources and effort required to conduct such a study on a large scale.

Study Two took advantage of a relatively standard set of symptoms that frequently occur in new blood donors to assess the influence of alexithymia on symptom reporting. Thus, rather than requiring medical examination and testing to rule out underlying pathophysiology, symptoms were induced in a group of otherwise healthy young adults. Further it aimed to increase generalizability of findings of altered physiological reactivity by studying cardiovascular response to the stress of blood donation in a quasi-naturalistic setting, a mobile clinic.

Study One

Manuscript Submitted for Publication

Alexithymia and Autonomic Reactivity to Emotion-Inducing Films

Nelson Byrne, B.Sc., B.Sc., and Blaine Ditto, Ph.D.

Department of Psychology, McGill University

Abstract

Objective: This study examined the effect of alexithymia on physiological and cognitive experiential facets of emotional responding. **Method:** Forty-two high and low alexithymia individuals, 21 males and 21 females, watched four film clips selected to induce positive or negative emotions. A variety of physiological measures were obtained, including respiratory sinus arrhythmia (RSA). Subjective experience was also assessed. **Results:** No alexithymia related baseline differences in physiological measures were found. High alexithymia males displayed reduced RSA in response to emotion induction rather than the increased RSA shown by others. High alexithymia individuals reported lower ratings of positive emotions, greater confusion, and differences in thoughts related to evaluating and regulating their affective state. **Conclusions:** Alexithymia is associated with differences in emotional responding at both the physiological and cognitive experiential levels. The importance of including indices of both sympathetic and parasympathetic activity in future research is noted.

Key Words: alexithymia; autonomic reactivity; respiratory sinus arrhythmia; emotional responding; emotion regulation

Alexithymia and Autonomic Reactivity to Emotion-Inducing Films

Alexithymia can be most concisely characterized as a deficit in the cognitive processing of emotion (1). First introduced by Sifneos (2), the term alexithymia referred to a subgroup of psychosomatic patients observed to have difficulty identifying and describing feelings. Subsequently elaborated by Nemiah et al. (3), this multifaceted personality construct is also considered to encompass difficulty differentiating emotions from physiological states, lack of fantasy, and a propensity toward externally oriented rather than psychologically minded thought (4).

Even when stringent cutoff scores are employed to categorize individuals as clinically alexithymic, they are frequently over represented in clinical populations. Notably, rates of alexithymia above 50% have been reported in hypertensive populations (5, 6). Porcelli et al. (7) found that alexithymics comprised 35.7% of a sample of inflammatory bowel disease patients Comparably high figures have been reported in populations suffering from panic disorder (8, 9). A review of the epidemiologic literature is provided by Taylor (10). This consistent reporting of increased frequency of psychiatric and somatic illness in alexithymics has fuelled research aimed at evaluating explanations for the observed associations.

As summarized by Friedlander et al. (11), it has been suggested that alexithymics experience affect, but they lack the ability to differentiate, cognitively elaborate, label and then regulate the affect by using psychological strategies usually employed by others, such as healthy emotional expression, or sharing of feelings. Consequently, their affect remains diffuse but active, accompanied by physiological arousal, and is experienced as a global, undifferentiated, negative subjective state. Friedlander et al. argue that this creates

a foundation that may contribute to the development of various disorders. Two mechanisms through which alexithymia may contribute to the development of disorders are: 1) physiological hyperarousal accompanied by a negative subjective state; 2) impairment of affect-regulation strategies aimed at reducing arousal and improving subjective functioning.

The hypothesis that altered physiological arousal leads to disease has stimulated numerous investigations of alexithymics' physiological responses, most often to stimuli presented in the laboratory. Yet several studies have either failed to find a relationship between alexithymia and reactivity to stressors, or have demonstrated sympathetic hyporeactivity. Friedlander and colleagues (11) compared heart rate and electrodermal responses of alexithymic and nonalexithymic groups to three different laboratory stressors: mental arithmetic, a speech task, viewing disgusting scenes. While alexithymics were found to have tonically greater electrodermal activity, findings did not support group differences in reactivity. Similarly, Linden et al. (12) reported reduced heart rate responses in high alexithymia individuals to three laboratory stress tasks (isometric hand-grip, mental arithmetic, and discussion of an anger-producing event) but blood pressure responses did not differ significantly with level of alexithymia. Roedema and Simons (13) recorded facial muscle, heart rate, and skin conductance activity during presentation of emotionally relevant slides. Individuals identified as alexithymic were found to produce fewer nonspecific skin conductance responses and showed less heart rate deceleration. Using impedance cardiography, Neumann et al. (14) found that alexithymia was associated with blunted sympathetic activation and diminished vagal withdrawal in women during an anger recall task. On the other hand, there have also been

reports of increased reactivity in alexithymics. Infrasca (15) found that alexithymia was associated with larger skin conductance responses to an emotional film. An association between alexithymia and increased heart rate reactivity was observed by Luminet and Rimé (16) with exposure to an emotion-inducing movie. Waldstein et al. (17) found that older alexithymics had larger blood pressure responses to anger provocation than age-matched controls. As these examples illustrate, findings from experimental studies have been inconsistent and difficult to compare, in part due to use of different stimuli, measurement of different physiological variables, and even the assessment of alexithymia. For example, although the preceding discussion was mostly limited to studies that employed the widely used Toronto Alexithymia Scale (TAS-20; [18, 19]), Linden et al. note that findings of hyperreactivity are somewhat more common when the Schalling-Sifneos Personality Scale is used to assess alexithymia, while findings of hyporeactivity are more commonly noted in studies that used the TAS-20.

The issue of different stimuli may be particularly important. Given that alexithymia is defined as a problem in emotional processing, it would be reasonable to expect differences in physiological reactivity particularly in response to emotional stimuli. While all of the previously noted stimuli may have elicited emotion to some degree, it is likely that some were more effective in this regard than others. For example, physiological responses to arithmetic tasks can be influenced by frustration and concern about doing poorly, but these issues are often mild and can be obscured by significant cognitive and motor influences on reactivity to such tasks. To minimize these issues, the present study employed emotional film clips, a well documented means of inducing affective states in the laboratory (20, 21). The study was the first to use several films to

induce both negative and positive changes in emotion to study differences in physiological reactivity in alexithymia, employing previously proven methodological standards (22-24).

Although most prior research on the autonomic correlates of alexithymia has emphasized possible differences in sympathetic activity, there is ample evidence that emotion-related autonomic responses include a complex pattern of sympathetic and parasympathetic activation (25). Moreover, it has been argued that changes in cardiac vagal activity in particular are associated with individual differences in the expression and regulation of emotion (26). For this reason, a number of physiological measures were obtained here, including cardiac respiratory sinus arrhythmia, an index of cardiac vagal activity. Finally, it was hoped that inclusion of measures of the cognitive-experiential component of emotional responding would broaden understanding of the cognitive processing deficit in alexithymics, and perhaps provide useful information to explain physiological differences.

Method

Participants

Forty-two individuals participated in the study, 21 with high- and 21 with low-alexithymia scores. Participants were drawn from a sample of 492 undergraduates who completed Toronto Alexithymia Scale (TAS-20;[18, 19]). The mean TAS-20 score of the screening group was 45.1 (SD = 10.6). To create a sample for the laboratory study with variability in alexithymia, the screening group was divided into terciles based on TAS-20 scores and individuals from the low- and high-alexithymia terciles were invited to participate. Eleven high-alexithymia females, 10 high-alexithymia males, 10 low-alexithymia females, and 11 low-alexithymia males participated in the study. There were no significant group differences in demographic characteristics (Table 1).

Film Stimuli

Sections of 14 commercial movies deemed likely to induce relatively distinct positive and negative emotions were selected for consideration in the present study, including some previously used by Gross and Levenson (22). Following their methodology, these excerpts were viewed by seven volunteers who rated each using a list of 16 emotion terms with 0 to 8-point Likert scales, adapted from Ekman, Friesen and Ancoli (27). The average interrater reliability was .80. Based on these ratings, two positive (POS1 and POS2) and two negative (NEG1 and NEG2) excerpts with relatively intense and distinct emotional content were selected.

POS1: This excerpt from the film *The English Patient* (Miramax Films, 1996) depicts a woman following a candlelit path to meet her lover and their ensuing romantic interaction. Duration: 3 min 59 s.

POS2: Set in a diner, this humorous scene from *When Harry Met Sally* (Columbia Pictures, 1989) shows a female character faking an orgasm in the presence of her male companion and astonished onlookers. Duration: 2 min 35 s.

NEG1: This scene from *Terms of Endearment* (Paramount Pictures, 1983) depicts a mother's grief as she watches her daughter die of cancer. Duration: 6 min 28 s.

NEG2: A terrified woman is attacked with a knife in her bathroom by her husband's jealous lover in this excerpt from *Fatal Attraction* (Paramount Pictures,1987). Duration: 5 min 12 s.

Apparatus

Audiovisual. The film clips were digitized and presented on a 17-inch colour monitor using Microsoft Power Point. This facilitated counterbalancing of the films and the incorporation of on-screen instructions. Participants were also videotaped during testing using a camera concealed behind a one-way mirror to permit later evaluation of the facial expression of emotion. These data will not be reported here.

Physiological. Continuous measurements of heart rate, high frequency heart rate variability, finger pulse amplitude, and skin conductance were obtained using a Grass polygraph. The electrocardiogram for measurement of heart rate and heart rate variability was obtained using disposable electrodes placed in bipolar configuration on opposite sides of the rib cage. The signal was analyzed by a Delta Biometrics Vagal Tone Monitor II (VTM-II; Bethesda, MD). After measuring the time between successive R waves, mean values of heart rate (in bpm) were produced every 30 s. The VTM-II produces measurements of high frequency heart rate variability ("vagal tone", in log units) by using a moving polynomial filter to quantify beat-to-beat heart rate variability in the

respiratory frequency band of 0.12 to 0.40 HZ, i.e., respiratory sinus arrhythmia. Details of the procedure can be found in Porges and Bohrer (28).

Skin conductance (in μSiemens) was measured using electrodes attached to the palmar surface of the middle phalanges of the first and third fingers of the nondominant hand. Finger pulse amplitude (in mV) was measured using a photoplethysmograph attached to the distal phalange of the second finger of the nondominant hand. These signals were digitized and reduced using CODAS software and a computer.

Blood pressure was measured using a Critikon 845XT automatic blood pressure monitor that was activated from the control room. Given the variable length of the film clips, the blood pressure cuff was manually triggered to inflate at 2.5 min intervals during the initial 5 min baseline period, at the beginning and end of each film clip and 1 min following the end of the clip.

Postfilm Questionnaires. Following each film clip, the participant completed the same emotion rating scale used for film selection (adaptation from Ekman, Friesen, and Ancoli (27) as used by Gross and Levenson (22)). The participant was asked to indicate how they currently feel using 16 emotion adjectives rated on a 0 (none) to 8 (most in my life) scale. Separate ratings of overall mood intensity and valence (unpleasant vs. pleasant) were also requested.

Following each film, the participant also completed the State Meta-Mood Scale (SMMS; (29, 30)), a state measure of monitoring, evaluating and regulating one's current mood. It consists of two scales: the Meta-Evaluation Scale (MES) and the Meta-Regulation Scale (MRS). The MES is comprised of four subscales measuring thoughts about the control and influence of the mood ("This mood will never change"), its clarity

("I know exactly how I am feeling), acceptance or rejection of the mood ("There's nothing wrong with it"), and typicality ("I feel this mood often"). The MRS is comprised of three subscales that measure thoughts about maintaining current mood ("I wouldn't want to change my mood"), dampening positive mood ("I'm trying to relax because it is too positive"), and repairing negative mood ("I'm imagining something better to improve my mood."). Mayer and Stevens (30) observed good internal consistency (.75 - .87) for the MES and MRS, and low correlations among their subscales. Convergent validity of the MES and MRS was also demonstrated with other measures, including the Toronto Alexithymia Scale.

Procedure

Participants were instructed to abstain from cigarettes and coffee for four hours prior to testing and from alcohol for 24 hours. They were also asked to avoid strenuous exercise in the hour before arriving at the laboratory. Upon arrival, they provided written informed consent and completed a brief health-screening questionnaire. Following attachment of the physiological recording equipment, the participant was seated at a classroom desk 1.5 m from a video monitor, in a room separated from the recording equipment and the experimenters by a one-way mirror. Ongoing instructions were provided on the monitor throughout the testing procedure.

To begin, the participant was asked to relax for 5 min while watching a blank screen to permit recording of baseline physiological measures. The film clips were then presented in counterbalanced order. As in previous research (31), each clip was preceded by a 30-s blank screen. During this period, participants were asked to clear their minds of any thoughts, feelings, or memories that may have been evoked by previous clips or other

events they had recently experienced. After the film clip there was an 8 min interfilm period. During the first 2 min of this period, participants were asked to sit quietly and think about how the preceding clip made them feel. Afterwards, the participant completed the emotion rating questionnaire and the SMMS. The procedure was repeated for each of the four film clips.

Data Reduction and Analysis

Preliminary analyses revealed that film-related physiological change often did not occur until near the end of the film clip. This was not unexpected given that the events depicted in the clips usually built to an emotional climax. Thus, to highlight the impact of the films, it was decided to calculate average values of the physiological measures during the 30 s immediately following the end of the film clip. These were compared with average values obtained during the last 2 min of the 5-min prefilm baseline period. That is, for each dependent measure, four change scores were calculated by subtracting the baseline value from the value obtained at the end of each film. The primary statistical analyses were 2 (Gender) x 2 (Alexithymia) x 4 (Film) repeated measures analyses of covariance (ANCOVAs), using baseline value as the covariate. As a conservative measure, the effects of repeated measures were assessed using multivariate tests of significance.

Results

Physiological Data

While the film clips produced significant physiological changes from baseline on most variables measured (Table 2), relatively few significant group effects emerged from the ANCOVAs. Despite significant decreases in heart rate during some films, blood pressure remained generally unaffected. This may have been due to the fact that peripheral resistance, at least as indicated by finger pulse amplitude (FPA), appears to have increased. This suggests that physiological response to the films was not simple relaxation. Several responses indicating attention to the films were observed (Table 2).

The key finding involving alexithymia was a significant Alexithymia x Gender interaction effect in the ANCOVA of RSA, $F(1,36) = 7.87$, $p = .008$. In general, RSA increased while watching the films, especially during *When Harry Met Sally* and *Fatal Attraction,* the clips for which participants also gave the highest emotional intensity ratings. Contrary to this trend (see Figure 1), males with high alexithymia scores displayed a small decrease in RSA which was significantly lower than the increases displayed by low alexithymia males, $t(18) = 1.86$, $p = .04$, low alexithymia females, $t(17) = 1.67$, $p = .059$, and high alexithymia females, $t(18) = 2.94$, $p = .005$.

The skin conductance (GSR) and finger pulse amplitude ANCOVAs both produced interaction effects involving alexithymia that approached significance. The Alexithymia x Gender interaction effect approached significance in the skin conductance ANCOVA, $F(1,33) = 3.20$, $p = .083$. Reminiscent of the RSA results, males with high alexithymia scores exhibited the smallest increases in skin conductance while watching the films. Similarly, the Alexithymia x Film interaction effect in the finger pulse

amplitude ANCOVA approached significance, $F(3,30) = 2.38, p = .089$. This was due to slightly smaller reductions in pulse amplitude (indicating less vasoconstriction) in high alexithymia individuals while watching clips from *The English Patient* and *Fatal Attraction.*

Postfilm Questionnaire Data

The effectiveness of the film clips to elicit emotion was assessed using a 2 (Gender) x 2 (Alexithymia) x 4 (Film) x 16 (Emotion Adjective) ANOVA. The Film x Emotion Adjective interaction was significant, $F(45,1575) = 36.85, p < .001$, reflecting higher ratings of the emotions targeted by the clips. More interesting, the Alexithymia x Emotion Adjective interaction was also significant, $F(15,525) = 3.77, p < .001$. Follow-up tests revealed that, compared to their low alexithymia counterparts, participants in the high alexithymia group reported lower levels of several positive emotion adjectives. Amusement, $t(40) = -2.11, p < .05$, contentment, $t(40) = -2.20, p < .05$, and happiness, $t(39) = -2.03, p = .050$, were all rated significantly lower by high alexithymia individuals. Conversely, high alexithymia individuals reported greater confusion, $t(40) = 2.33, p < .05$. There were no significant effects involving alexithymia when intensity and pleasantness ratings were subjected to analysis of variance.

The MES data were analyzed using a 2 (Gender) x 2 (Alexithymia) x 4 (Film) x 4 (Subscale) ANOVA. A significant Alexithymia x Subscale interaction was observed, $F(3,36) = 12.05, p < .001$. While high and low alexithymia individuals did not differ in their ratings of how typical their feelings were after the films, and how much these feelings influenced their thinking, they did differ significantly in their acceptance, $F(1,40) = 6.33, p = .016$, and particularly their mood clarity, $F(1,40) = 37.47, p < .001$,

scores. That is, high alexithymia individuals appear to have been less accepting of, and less clear about their emotional states following the films than low alexithymia individuals.

Similar to the MES analysis, the ANOVA of MRS data also yielded a significant Alexithymia x Subscale interaction, $F(2,36) = 8.37$, $p = .001$. Participants in the high alexithymia group reported significantly higher ratings on the dampening subscale, $F(1,39) = 5.41$, $p < .05$, but scored lower on the maintenance subscale, $F(1,39) = 7.31$, $p = .010$. Compared to participants in the low alexithymia group, this suggested that high alexithymia participants reported more thoughts related to dampening positive mood, but their ratings of thoughts related to maintaining their current mood were lower. The group difference in the repair subscale scores was marginally significant, $F(1,39) = 3.96$, $p = .054$, with high alexithymia individuals reporting more thoughts about trying to repair negative mood.

Discussion

The fact that high alexithymia individuals, particularly males, seemed to be generally less physiologically reactive to emotional films, with decreased RSA as well as a tendency toward decreased sympathetic (GSR, FPA) reactivity, begs the question of what is different about how they process emotion at a cognitive level. Although the SMMS is not a widely used instrument and there is only limited published data to support its validity, the results may provide a context in which to interpret the physiological findings. The picture that emerges from the SMMS data suggests that, when faced with emotional information, alexithymics experience a state of confusion and nonacceptance of their affective state, with thoughts directed at diminishing their experience, regardless of the valence or specific emotion elicited. While it has been established that the construct of alexithymia is distinct from repression and defensiveness (12, 32), these results suggest that there may be some active inhibition occurring at a cognitive level. Consistent with this idea is the decrease in RSA observed in high alexithymia males, suggesting parasympathetic withdrawal. On the other hand, although high alexithymia males displayed significantly different RSA response to the films than the other groups, their overall degree of RSA decrease from baseline compared to no change only approached significance ($p = .07$). Of the four films, only the clip from *Terms of Endearment* elicited a decrease in RSA for the high alexithymia male group that was significantly different from zero, $t(8) = -2.49$, $p < .05$. Therefore, in contrast to the idea of inhibition of emotions, it is possible that the physiological findings are more consistent with an inability on the part of high alexithymia males to engage in cognitive processing of the films' emotional content and display more typical physiological responses.

The decrease in RSA displayed by high alexithymia males in contrast to the increase in RSA shown by the other groups has not been previously reported. In the one published study that did include a measure of RSA, a significant association between alexithymia and diminished vagal withdrawal was observed in women (14). In contrast to the present results, Neumann et al. found that higher alexithymia scores corresponded to smaller RSA decreases in response to a task that typically elicited decreases in RSA. Despite the apparent lack of congruence, both sets of results may be consistent with diminished psychological engagement in the tasks, in one case leading to relatively greater, and in another case leading to smaller parasympathetic withdrawal.

Given knowledge of the potential for complex interaction of the sympathetic and parasympathetic systems during emotional responding (33), it is perhaps not surprising that conclusions about hyper- or hyporeactivity have been inconsistent. With a few exceptions (13, 14, 34), the hyporeactivity hypothesis has been based on differences in heart rate reactivity, which is subject to both sympathetic and parasympathetic influences. Characterization of alexithymics as hyper- or hyporeactive is perhaps overly simplistic, and fails to capture the complexity of differences in physiological response to emotional stimuli that may occur in these individuals. More explicit consideration of gender and inclusion of indices of parasympathetic activity in future research would be prudent.

The finding that high alexithymia participants reported lower levels of amusement, contentment and happiness is consistent with the notion of an underlying tendency toward negative affectivity that some have reported characterizes these individuals (35, 36). Given high alexithymia individuals' higher ratings of confusion in response to the emotional stimuli, it is not surprising that their ratings of positive emotion

items were lower. The film stimuli did produce changes in autonomic activity, and as Davidson (37) points out, activity in the autonomic nervous system for which the subject has no explanation biases experience toward negative rather than positive affect. Using a protocol similar to the present study, Gross and Levenson (23) found that participants who were instructed to suppress their emotions reported less amusement, but not less sadness, in response to emotion-inducing film clips.

One potential limitation of the present study is that no attempt was made to categorize participants as clinically alexithymic or not. Based on the cutoff score of ≥ 61 for the TAS-20 determined by Bagby and Taylor (38) from a small clinical sample of 39, not all of the participants in the high alexithymia group were clinically alexithymic. However, using a cutoff score of ≥ 56 employed by Loas et al. (39) in a university student population, only three of the participants in the high alexithymia group could not be classified as clinically alexithymic. A reanalysis of the data using Taylor et al.'s more conservative cutoff scores to establish clinically alexithymic and non alexithymic groups did not change the results reported here.

Although the use of film clips to induce emotion has been shown to be effective, the clips did not elicit large changes or patterns in physiological activity consistent with research on specific physiological correlates of emotion (37). For example, the observed increases in respiratory sinus arrhythmia were more suggestive of a state of relaxed attention than states of sadness or fear. On the other hand, given that study participants were selected to represent high and low levels of alexithymia, a construct frequently associated with altered physiological response, it would be premature to attribute the atypical physiological responses solely to the stimuli. Moreover, the intensity and

complexity of affective states achieved using film clips in the laboratory is unlikely to accurately reflect emotion experienced in response to personally relevant events that occur in everyday life. Future research should consider other emotion induction techniques, such as recall of past emotional experiences (40). Ambulatory monitoring of physiological reactivity while individuals experience emotional events under more naturalistic circumstances may also yield more valid and generalizable results.

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References

1. Taylor GJ, Bagby RM, Parker JD. The alexithymia construct: A potential paradigm for psychosomatic medicine. Psychosomatics 1991;32:153-64.
2. Sifneos PE. The prevalence of "alexithymic" characteristics in psychosomatic patients. Psychother Psychosom 1973;22:255-62.
3. Nemiah JC, Freyberger H, Sifneos PE. Alexithymia: a view of the psychosomatic process. In: Hill OW, editor. Modern trends in psychosomatic medicine. vol 3. London: Butterworths; 1976. p. 430-9.
4. Lumley MA. Alexithymia, emotional disclosure, and health: A program of research. J Pers 2004;72:1271-300.
5. Jula A, Salminen JK, Saarijärvi S. Alexithymia: A facet of essential hypertension. Hypertension 1999;33:1057-61.
6. Todarello O, Taylor GJ, Parker JDA, Fanelli M. Alexithymia in essential hypertension and psychiatric outpatients: A comparative study. J Psychosom Res 1995;39:987-94.
7. Porcelli P, Zaka S, Leoci C, Centonze S, Taylor GJ. Alexithymia in inflammatory bowel disease. Psychother Psychosom 1995;64:49-53.
8. Parker JDA, Taylor GJ, Bagby RM, Acklin MW. Alexithymia in panic disorder and simple phobia: a comparative study. Am J Psychiatry 1993;150:1105-7.
9. Zeitlin SB, McNally RJ. Alexithymia and anxiety sensitivity in panic disorder and obsessive-compulsive disorder. Am J Psychiatry 1993;150:658-60.
10. Taylor GJ. Recent developments in alexithymia theory and research. Can J Psychiatry 2000;45:134-42.

11. Friedlander L, Lumley MA, Farchione T, Doyal G. Testing the alexithymia hypothesis: Physiological and subjective responses during relaxation and stress. J Nerv Ment Dis 1997;185:233-9.
12. Linden W, Lenz JW, Stossel C. Alexithymia, defensiveness and cardiovascular reactivity to stress. J Psychosom Res 1996;41:575-83.
13. Roedema TM, Simons RF. Emotion-processing deficit in alexithymia. Psychophysiology 1999;36:379-87.
14. Neumann SA, Sollers JJI, Thayer JF, Waldstein, SR. Alexithymia predicts attenuated autonomic reactivity, but prolonged recovery to anger recall in young women. Int J Psychophysiol 2004;53:183-95.
15. Infrasca R. Alexithymia, neurovegetative arousal, and neuroticism: An experimental study. Psychother Psychosom 1997;66:276-80.
16. Luminet O, Rime B, Bagby RM, Taylor GJ. A multimodal investigation of emotional responding in alexithymia. Cognition & Emotion 2004;18:741-66.
17. Waldstein SR, Kauhanen J, Neumann SA, Katzel LI. Alexithymia and cardiovascular risk in older adults: Psychosocial, psychophysiological, and biomedical correlates. Psychol Health 2002;17:597-610.
18. Bagby RM, Taylor GJ, Parker JDA. The twenty-item Toronto Alexithymia Scale: II. Convergent, discriminant, and concurrent validity. J Psychosom Res 1994;38:33-40.
19. Bagby RM, Parker JDA, Taylor GJ. The twenty-item Toronto Alexithymia Scale: I. Item selection and cross-validation of the factor structure. J Psychosom Res 1994;38:23-32.

20. Westermann R, Spies K, Stahl G, Hesse FW. Relative effectiveness and validity of mood induction procedures: A meta-analysis. Eur J Soc Psychol 1996;26:557-80.
21. Gerrards-Hesse A, Spies K, Hesse FW. Experimental inductions of emotional states and their effectiveness: A review. Br J Psychol 1994;85:55-78.
22. Gross JJ, Levenson RW. Emotion elicitation using films. Cognition & Emotion 1995;9:87-108.
23. Gross JJ, Levenson RW. Hiding feelings: The acute effects of inhibiting negative and positive emotion. J Abnorm Psychol 1997;106:95-103.
24. Philippot P. Inducing and assessing differentiated feeling states in the laboratory. Cognition & Emotion 1993;7:171-93.
25. Cacioppo JT, Berntson GG, Larsen JT, Poehlmann KM, Ito TA. The psychophysiology of emotion. In: Lewis R, Haviland-Jones JM, editors. The handbook of emotion. 2nd ed. New York: Guilford Press; 2000. p. 173-91.
26. Porges SW, Doussard-Roosevelt JA, Maita AK. Vagal tone and the physiological regulation of emotion. Monogr Soc Res Child Dev 1994;59:167-86, 250-83.
27. Ekman P, Friesen WV, Ancoli S. Facial signs of emotional experience. J Pers Soc Psychol 1980;39:1124-34.
28. Porges SW, Bohrer RE. The analysis of periodic processes in psychophysiological research. In: Cacioppo JT, Tassinary LG, editors. Principles of Psychophysiology. Cambridge: Cambridge University Press; 1990. p. 708-53.
29. Mayer JD, Gaschke YN. The experience and meta-experience of mood. J Pers Soc Psychol 1988;55:102-11.

30. Mayer JD, Stevens AA. An emerging understanding of the reflective (meta-) experience of mood. J Res Pers 1994;28:351-73.

31. Gross JJ, Levenson RW. Emotional suppression: Physiology, self-report, and expressive behavior. J Pers Soc Psychol 1993;64:970-86.

32. Paez D, Basabe N, Valdoseda M, Velasco C, Iraurgi I. Confrontation: Inhibition, alexithymia, and health. In: Pennebaker JW, editor. Emotion, disclosure & health. Washington, DC: American Psychological Association; 1995. p. 195-222.

33. Berntson GG, Cacioppo JT, Quigley KS. Autonomic determinism: The modes of autonomic control, the doctrine of autonomic space, and the laws of autonomic constraint. Psychol Rev 1991;98:459-87.

34. Wehmer F, Brejnak C, Lumley M, Stettner L. Alexithymia and physiological reactivity to emotion-provoking visual scenes. J Nerv Ment Dis 1995;183:351-7.

35. Lundh L-G, Simonsson-Sarnecki M. Alexithymia, emotion, and somatic complaints. J Pers 2001;69:483-510.

36. Wise TN, Mann LS. The relationship between somatosensory amplification, alexithymia, and neuroticism. J Psychosom Res 1994;38:515-21.

37. Davidson RJ, Gray JA, LeDoux JE, Levenson RW, Panksepp J, Ekman P. Is there emotion-specific physiology? In: Ekman P, Davidson RJ, editors. The nature of emotion: Fundamental questions. Series in affective science. New York: Oxford University Press; 1994. p. 235-62.

38. Bagby RM, Taylor GJ. Measurement and validation of the alexithymia construct. In: Disorders of affect regulation: Alexithymia in medical and psychiatric illness. Cambridge, UK: Cambridge University Press; 1997. p. 46-66.

39. Loas G, Fremaux D, Otmani O, Verrier A. Prevalence of alexithymia in general population: Study in 183 normal subjects and in 263 university students. [French]. Ann Med Psychol (Paris) 1995;153:355-7.

40. Prkachin KM, Williams-Avery R, Zwaal C, Mills DE. Cardiovascular changes during induced emotion: An application of Lang's theory of emotional imagery. J Psychosom Res 1999;47:255-67.

Table 1

Mean (SE) Baseline Characteristics of Groups

	Females		Males	
Alexithymia Group	Low (n=10)	High (n=11)	Low (n=11)	High (n=10)
Alexithymia Score	26.8 (.7)	65.9 (1.0)	29.6 (1.4)	58.9 (1.3)
Age (years)	21.8 (1.7)	19.8 (.2)	20.1 (.4)	20.1 (.8)
Body mass index (kg/m^2)	22.8 (1.2)	22.1 (1.1)	23.4 (.9)	22.4 (.8)
SBP (mmHg)	106.3 (3.5)	106.8 (3.0)	117.1 (3.3)	119.6 (3.7)
DBP (mmHg)	66.3 (1.9)	63.6 (2.9)	64.7 (2.4)	70.4 (2.3)
HR (bpm)	81.1 (3.5)	76.2 (3.5)	72.4 (3.0)	79.4 (2.9)
RSA (units)	6.4 (.4)	7.2 (.3)	6.8 (.2)	6.6 (.1)
GSR (μSiemens)	3.1 (1.3)	2.8 (.5)	2.0 (.2)	1.4 (.3)
FPA (mv)	2.4 (.5)	3.0 (.4)	2.9 (.6)	3.0 (.6)

Table 2

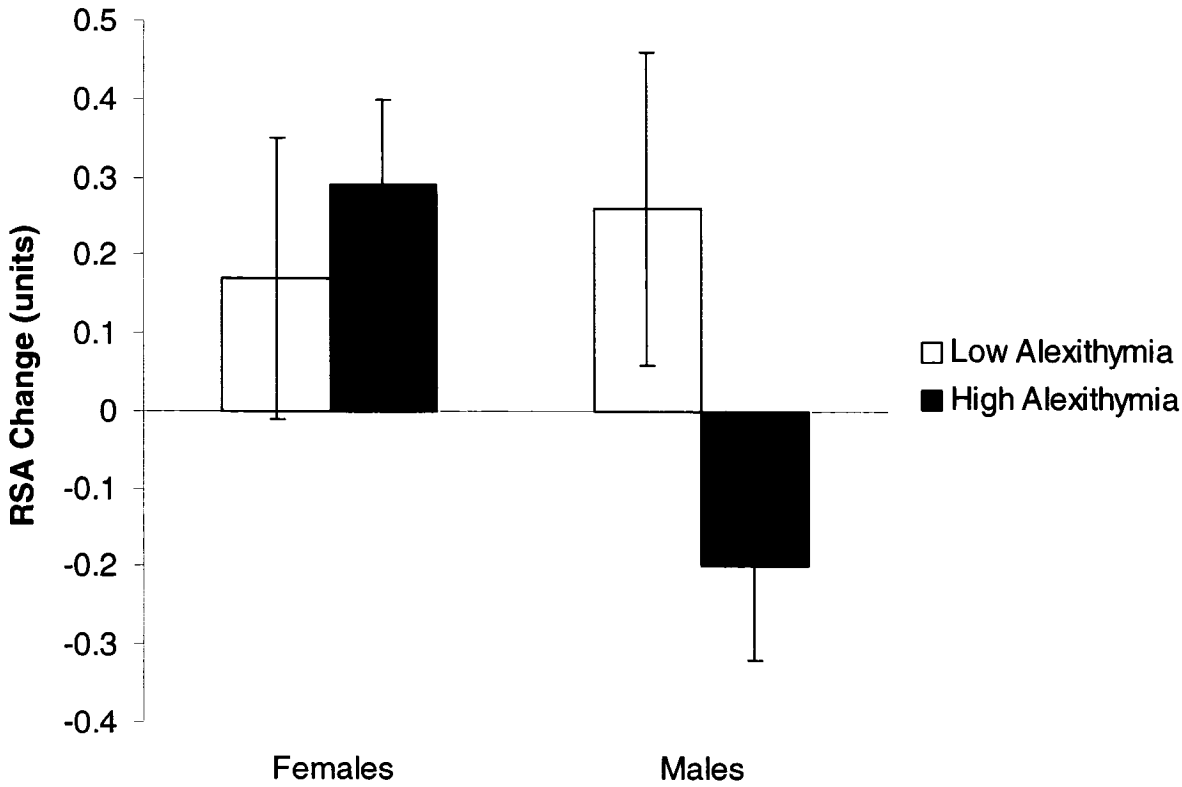
Mean (SE) Physiological Changes Produced by Film Clips

	English Patient	Harry Met Sally	Fatal Attraction	Terms of Endearment
SBP (mmHg)	0.0 (.9)	-0.3 (1.3)	0.8 (1.2)	2.2 (1.4)
DBP (mmHg)	1.2 (.9)	1.6 (.7)*	1.4 (.7)	0.7 (.9)
HR (bpm)	-0.6 (.8)	-3.1 (.6)*	-2.6 (.9)*	-0.5 (.7)
RSA (units)	.06 (.10)	.30 (.12)*	.24 (.12)*	-.02 (.10)
GSR (μSiemens)	13.2 (.7)*	12.1 (.7)*	13.3 (.7)*	12.9 (.7)*
FPA (mv)	-1.2 (.2)*	-1.1 (.2)*	-1.3 (.2)*	-1.0 (.2)*

* Significantly different from 0, $p < .05$.

Figure legends

Figure 1. Mean RSA change during emotion films for high and low alexithymia groups.


0.5
0.4
0.3
0.2
0.1
0
-0.1
-0.2
-0.3
-0.4
RSA Change (units)
Females
Males
Low Alexithymia
High Alexithymia

Bridge to Study Two

Although Study One found differences in autonomic response between high and low alexithymia individuals, the observed differences were relatively small and limited to one variable, respiratory sinus arrhythmia. While several methods have proven effective at eliciting affective changes in the laboratory, it is unclear whether these changes are of a magnitude that resembles emotional experiences of everyday life. Moreover, typical emotional stimuli used in laboratory settings, such as films, may be qualitatively different than events that occur in a naturalistic environment. For example, while a film clip depicting a medical procedure may produce a mild aversive reaction in some individuals, it is conceivable that undergoing such a procedure would produce a larger reaction, as it would be more personally relevant.

Given that a deficit in the cognitive processing of emotion is the defining feature of alexithymia, a barrage of emotional information over a period of several minutes is perhaps too complex for alexithymic individuals to comprehend. One might speculate that under such circumstances, rather than remaining cognitively engaged, they "tune out" and fail to experience the same level of arousal as nonalexithymic individuals. Unfortunately, at least in a laboratory setting, the differences in emotional quality triggered by different stressors remain poorly understood (Steptoe, 1985), as self-report indices of affect tend to correlate poorly with physiological changes. Yet, it is well established that different laboratory tasks are associated with different cardiovascular response patterns. In turn, as Linden, Rutledge and Con (1998) point out, these response patterns may reflect qualitative differences in emotional response, as well as varying intensities of cognitive challenge and needed effort at resolution. Given alexithymics'

deficit in processing emotion at a cognitive level, exposure to a stressor that typically elicits an emotional response but does not involve processing of complex emotional information may be more successful at inducing affective change in alexithymic individuals.

Blood donation is known to be an anxiety-provoking medical procedure, particularly for inexperienced donors, and increases in blood pressure are known to occur in the predonation waiting period (Adler, Ditto, France, & France, 1994). However, a fear or anxiety response to a medical procedure such as blood donation is likely qualitatively different, and probably less cognitively complex, than that involving an interpersonal encounter, for example. During blood donation, the stimuli inducing the fear can be quite concrete in nature, such as the approach of a sharp needle during venipuncture, or the sight of blood. When faced with such stimuli, alexithymics may respond differently than when required to process more complex emotional events.

Although Study One focused primarily on evaluating the hypothesis that alexithymia leads to organic disease through physiological mechanisms, this is only one of four potential pathways proposed by Lumley et al. (1996). A second proposed pathway implicates illness behaviour, such as symptom reporting, as an explanation for the alexithymia-illness association. Investigation of this pathway typically necessitates medical testing or extensive review of patient records to rule out underlying medical conditions that may account for self-reported symptoms.

The blood donation clinic may provide a convenient opportunity to evaluate differences in symptom reporting. Vasovagal reactions such as weakness, dizziness, lightheadedness, and nausea are relatively common occurrences (Meade, France, &

Peterson, 1996). Thus, a relatively limited set of somatic sensations is induced, serving as a useful manipulation to investigate patterns of symptom reporting as they relate to alexithymia. Additionally, the donation procedure entails painful procedures such as finger pricking and venipuncture, thereby permitting assessment of pain experience.

Finally, previous studies of cardiovascular reactivity have relied largely on measures obtained in the laboratory, and criticism has focused on failure to find generalizability of lab reactivity to the natural environment, highlighting limitations of traditional lab cardiovascular reactivity methodology (Schwartz et al., 2003). It was hoped that extending this investigation to a more naturalistic setting, with a somewhat more varied population, would increase generalizability of findings.

Study Two

Alexithymia, Cardiovascular Reactivity, and Symptom Reporting During Blood Donation

Nelson Byrne, B.Sc., B.Sc., and Blaine Ditto, Ph.D.

Department of Psychology, McGill University

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Abstract

Objective: With blood donation serving as a naturalistic stressor and a controlled medical event, the aim of this study was to examine emotional and cardiovascular reactivity, self-report of vasovagal symptoms, and perceived pain as a function of scores on the Toronto Alexithymia Scale (TAS-20). **Method**: Healthy young adult blood donors (N = 610) recruited at mobile blood collection clinics completed the TAS-20, pre- and postdonation measures of anxiety, postdonation measures of pain and vasovagal symptoms, and had their blood pressure and heart rate measured before and after giving blood. **Results:** Alexithymia score was positively associated with reported anxiety, pain, and vasovagal symptoms. Higher alexithymia was also associated with greater increases in predonation systolic blood pressure in anticipation of blood donation. In general, women and less experienced blood donors reported more vasovagal symptoms than men and more experienced donors, and this corresponded to higher rates of treatment by the nurses, more fainting, and fewer full units of blood obtained. However, despite more reports of vasovagal symptoms by alexithymic donors, alexithymia score was not related to these variables. **Conclusions:** The results suggest that individuals with higher alexithymia scores were more anxious in the blood donation setting and more prone to report physical symptoms in the absence of a clear difference in the medical outcome of the blood donation procedure.

Key words: alexithymia, symptom reporting, cardiovascular reactivity, blood donation, vasovagal reaction, pain.

BDRI = Blood Donation Reactions Inventory; **DBP** = diastolic blood pressure; **GLM** = general linear model; **HR** = heart rate; **SBP** = systolic blood pressure; **STAI** = Spielberger State-Anxiety Scale; **TAS-20** = Toronto Alexithymia Scale.

Alexithymia, Cardiovascular Reactivity, and Symptom Reporting During Blood Donation

The term alexithymia was coined by Nemiah and Sifneos (1,2) in the 1970s to describe patients presenting with psychosomatic concerns who were observed to have difficulty identifying and describing emotions. Additional features of this multifaceted personality construct include difficulty differentiating feelings from the bodily sensations of emotional arousal, a lack of fantasy resulting from constricted emotional abilities, and a pattern of externally oriented thinking. Unlike the concepts of repression and denial, in which identified emotions are kept below the surface of consciousness, alexithymics are thought to lack established emotional representation (3). More succinctly, alexithymia may be defined as a difficulty with cognitive processing of emotion (4).

Interest in the study of alexithymia has increased in recent years, in part as a result of mounting evidence of its association with a number of psychiatric and medical conditions such as hypertension (5,6), asthma (7), chronic pain syndrome (7), functional gastrointestinal disorders (8), alcoholism and substance abuse (9), anxiety disorders (10,11), and eating disorders (12–14). One particularly striking finding is that alexithymia predicted all-cause mortality after 5 years, even after controlling for demographic and medical risk factors (15). A recent review of the epidemiologic literature is provided by Taylor (16).

Although the association between alexithymia and illness is robust, the nature of this relationship is unclear. Possible pathways linking alexithymia and physical illness have been outlined by Lumley and colleagues (17,18). These include: 1) alexithymia leads to organic disease through physiological mechanisms or unhealthy behaviours; 2)

alexithymia leads to illness behaviour such as overreporting of symptoms and excessive use of health care; 3) physical illness influences psychologic functioning and leads to alexithymia; and 4) sociocultural and biologic factors comprise a third variable, which causes both alexithymia and physical illness.

The hypothesis that alexithymia leads to organic disease through physiological mechanisms is based on findings of altered physiological arousal, which may result from impaired regulation of emotion in alexithymics (4). In general, results point to greater physiological arousal (e.g., higher heart rate and skin conductance) at rest and, consistent with their reports of lower emotional reactivity to stressors, smaller physiological responses to stimuli such as watching emotional slides (19–22).

An alternative explanation for the link between alexithymia and illness stems from the finding that alexithymics tend to report more symptomatology in the absence of illness, perhaps in lieu of emotional complaints (25). Significant positive correlations have been reported between alexithymia and measures of somatisation and hypochondriasis (26, 27), possibly as a result of alexithymics' focusing on, amplifying, and misinterpreting somatic sensations that accompany emotional arousal. In psychiatric populations, two studies found that alexithymics reported more somatic complaints than nonalexithymic patients (28, 29). Kauhanen and colleagues (30) found greater reports of symptoms such as nausea, dizziness, heart palpitations, and headaches among those higher in alexithymia. Alexithymia has also been found to be positively associated with reports of experimental (31) and clinical (32) pain. It remains unclear whether symptom reporting is the result of higher rates of illness in alexithymics or whether illness is more

frequently diagnosed because misinterpretation and amplification of somatic sensations leads to increased healthcare utilization.

The blood donation clinic provides an interesting environment in which a number of these issues can be studied. First, particularly for inexperienced donors, blood donation can be a stressful experience that is associated with increases in anxiety and blood pressure in the predonation waiting period (37). In the present study, changes in anxiety and cardiovascular activity in relation to alexithymia were studied. Second, the nature of the donation procedure is such that vasovagal reactions are not uncommon. The relatively controlled induction of a limited set of somatic sensations served as a useful manipulation to investigate differences in symptom reporting as they relate to alexithymia. Finally, several fairly controlled painful procedures are conducted as part of the blood donation procedure, providing an opportunity to assess pain experience. The present study examined self-reported anxiety, cardiovascular reactivity, symptom reporting, and reported pain in blood donors varying in alexithymia.

Method

Participants

As part of a larger study concerning the prediction and prevention of vasovagal reactions to blood donation, 1560 donors were recruited over an 18-month period at mobile blood donor clinics held by the provincial blood collection agency at universities and colleges throughout the Montreal area. Participants for the present study were 610 accepted donors, 263 males and 347 females, who were randomly assigned to the no-treatment control group. Alexithymia was assessed with the widely used 20-item Toronto Alexithymia Scale (TAS-20 (35, 36)). There were no significant associations, or anything approaching significance (all $p > .20$), between alexithymia score (mean = 42.1 ± 10.0) and age (mean = 22.4 ± 7.2 years), number of prior blood donations (mean = 3.6 ± 7.3), or body mass index (mean = 23.7 ± 3.8 kg/m2).

Procedure

After registering at the clinic, prospective participants were directed to a research assistant. Once informed consent had been obtained, they completed a short predonation questionnaire that included an abbreviated version of the Spielberger State-Anxiety Scale (STAI (33)) and demographic questions about age, height, weight, and number of previous blood donations. Blood pressure was measured twice using a B-D Assure manual inflate digital blood pressure monitor (Becton, Dickinson and Co., New Jersey) while seated with the arm supported at heart level. These monitors use the oscillometric principle to measure blood pressure and, according to the manufacturer, are accurate to within ± 3 mm Hg. In cases in which there was a notable discrepancy between the first and second readings, blood pressure was measured a third time.

People were then seated in a waiting area before being called by a nurse for a health screening. During the screening, a blood sample was obtained by pricking the fingertip with a disposable lancet. Those deemed eligible to give blood proceeded to the first available donation chair and 450 mL of blood was drawn. Immediately after the blood draw, the attending nurse completed a brief questionnaire concerning issues such as the difficulty of needle insertion, whether the donor's chair had been reclined to treat vasovagal symptoms, and whether a full unit of blood was obtained. With assistance, donors then moved to a rest area where they remained seated on a donation chair for approximately 10 min before being met by a research assistant to have blood pressure measurements taken a second time. A set of postdonation questionnaires was provided at this point, which donors completed while consuming refreshments. These questionnaires included the Blood Donation Reactions Inventory (34), a self-report instrument designed to assess vasovagal reactions during blood donation, and the TAS-20. As well, the STAI was administered a second time. Finally, visual analog ratings of pain produced by the predonation finger prick and the venipuncture required to draw blood were requested.

Data Reduction and Analyses

Two predonation and two postdonation blood pressure readings for each subject were averaged to yield one systolic (SBP) and one diastolic (DBP) value at each time point. Similarly, pre- and postdonation heart rate (HR) values were obtained by averaging two HR readings at each time point. To reduce the positive skewness of the Blood Donation Reactions Inventory (BDRI) data, a log transformation (log[BDRI + 1]) was applied to raw scores as in previous research (34). Number of previous blood donations,

used as a continuous independent variable representing donation experience, was also subjected to a log transformation to better approximate a normal distribution.

Primary data analyses were conducted within the general linear model (GLM) framework using Systat statistical software (Systat Software, Inc., Point Richmond, CA). Alexithymia, defined as total TAS-20 score, and experience were used as continuous independent variables. Gender was entered as a dichotomous independent variable, along with the interaction effects among gender, alexithymia, and experience. To more closely examine the effects of the blood donation procedure on physiological change in relation to alexithymia, separate analyses of pre- and postdonation blood pressure and HR were conducted, as well as analyses of pre- to postdonation change scores in SBP, DBP, and HR.

Dichotomous yes–no variables such as whether the nurse reclined the donor's chair to treat vasovagal symptoms and whether a full unit of blood was obtained were analyzed using logistic regression equations using gender, previous donation experience, and alexithymia as predictor variables.

Results

Anxiety Ratings

The analysis of predonation anxiety scores yielded significant main effects of donation experience ($F[1,583] = 4.83$, $p = .028$) and alexithymia ($F[1,583] = 11.31$, $p = .001$). Higher predonation anxiety was reported in those with less donation experience and higher alexithymia scores. The analysis of postdonation anxiety scores produced only a similar significant main effect of alexithymia ($F[1,589] = 8.04$, $p = .005$). In general, predonation anxiety scores were significantly higher than postdonation scores ($t[589] = 9.50$, $p < .001$).

Cardiovascular Activity and Change

There were no significant effects involving alexithymia in the analyses of pre- or postdonation blood pressure or HR, or DBP and HR change scores. However, the analysis of systolic blood pressure change scores produced a significant effect of alexithymia ($F[1,593] = 5.02$, $p = .025$). SBP was generally higher while people were waiting to give blood compared with the postdonation refreshment period ($t[600] = 14.91$, $p < .001$). This difference was greater among those with higher alexithymia (Fig. 1). The effect remained significant with addition of postdonation SBP, which might be considered the "baseline" in this situation, as well as whether a full unit of blood was obtained as covariates in the model ($F[1,591] = 4.75$, $p = .030$).

Pain Ratings and Vasovagal Symptom Reporting

Analysis of the fingerprick pain ratings yielded only a significant main effect of alexithymia ($F[1,590] = 4.42$, $p = .036$). Higher alexithymia scores were associated with

higher pain ratings. There were no significant effects in the analysis of the venipuncture pain ratings.

The analysis of BDRI scores produced significant main effects of gender ($F[1,597] = 5.12$, $p = .024$), donation experience ($F[1,597] = 6.33$, $p = .012$), and alexithymia ($F[1,597] = 4.85$, $p = .028$). Women, people with less previous donation experience, and individuals with higher alexithymia scores reported more donation-related symptoms such as dizziness, weakness, and so on.

Medical Characteristics of the Blood Donations

There were no significant effects in the analyses of the nurse's rating of the ease of needle insertion or time in the donation chair. None of the logistic regression equations predicting whether a needle adjustment was required, whether the donor's chair was reclined, whether they fainted, or whether a full unit of blood was obtained produced significant effects of alexithymia. This occurred despite the fact that gender and previous blood donation experience, which were also associated with BDRI scores, were significantly related to whether the donor's chair was reclined to treat a vasovagal reaction, whether the donor fainted, and whether a full unit of blood was obtained (Table 1). Men and more experienced blood donors were less likely to faint or have their chairs reclined and more likely to produce a full unit of blood.

Discussion

Higher symptom reporting, including higher pain ratings, was associated with alexithymia. In addition to more physical symptom reporting, alexithymia was associated with higher self-reported anxiety and a greater difference in pre- and postdonation SBP. Given the fairly controlled nature of "injury" and blood loss produced by this medical intervention, known to produce a predictable set of symptoms, the present results provide some interesting information about the relationship between alexithymia and symptom reporting.

The underlying reason for higher symptom reporting by alexithymics has been the subject of considerable discussion. Some have argued that the reports are a reflection of real underlying illness given the associations between alexithymia and many medical disorders, whereas others have suggested that characteristics such as hypochondriasis or somatic attention mediate the relationship. The cluster of potential symptoms elicited during blood donation includes several that are potentially visible to attending staff, e.g., fainting. Although outright fainting was rare (3%), it was not associated with alexithymia score. There was also no association between alexithymia score and whether the nurse believed it necessary to treat the donor by reclining their chair, despite higher symptom reports by alexithymics. This contrasted with the effects of donation experience and gender, in which there was a concordance between higher symptom reports among less experienced donors and women and various objective measures of outcome such as chair reclining. Thus, although the sensations experienced by donors with higher alexithymia scores were construed as significant symptoms according to their BDRI ratings, they were not considered to be sufficiently severe by attending staff to warrant reclining the

donation chair. This suggests that alexithymics have an exaggerated inclination to attend to somatic sensations. At a minimum, they suggest a somewhat greater "disconnect" between "objective" physiological state and symptom reports, although the explanation for this disconnect is unclear.

Given that alexithymia, by definition, involves reduced reporting of emotion, the positive association between alexithymia and STAI scores is somewhat surprising. However, in examining responses to individual items in the five-item abbreviated STAI, it was found that this was the result of donors higher in alexithymia reporting feeling less comfortable, calm, relaxed, and pleasant, but not more anxious than donors lower in alexithymia. Thus, these results, as well as the pain data, seem to provide additional evidence of a tendency to report physical discomfort as opposed to negative emotion.

The limited nature of the cardiovascular data makes it difficult to interpret these results, although the apparently greater increase in predonation systolic blood pressure among alexithymics is intriguing. Because most (19–22) but not all (e.g., (24)) previous studies of physiological reactivity in alexithymics have indicated a tendency for reduced rather than exaggerated physiological reactivity to stress, it seems unlikely that this reflects a general tendency for high SBP reactivity. It may have been related to the very salient nature of the "stressor," i.e., anticipation of venipuncture with a large needle and loss of a significant amount of blood, and consistent with what appears to have been a greater state of psychologic distress in this environment, even if it was described in physical terms.

That said, another challenge in the interpretation of the SBP finding is the choice of the appropriate baseline period. For several reasons, we view the SBP finding as

indicative of a greater increase in SBP during the stressful predonation period in alexithymics as opposed to a greater decrease after donation. First, both SBP and reported anxiety were significantly higher before than after donation. This was true regardless of whether a full unit of blood was obtained from the donor. Second, measurements of resting blood pressure were obtained in the laboratory in a small subsample of these donors (N = 28) with similar equipment. There was no difference in postdonation and resting laboratory SBP (mean = 112.3 versus 112.4 mm Hg), whereas predonation SBP was significantly higher than resting laboratory SBP (mean = 116.2 versus 112.3 mm Hg; $t[27] = 2.08$, $p = .024$). Finally, researchers commonly operationalize the anticipation period before an aversive event (e.g., electric shock, public speaking, dental examinations) as the stress period and the recovery period as the baseline. The previously discussed results are consistent with this view in the present case. However, this issue needs to be considered with special caution in the present case, especially given the loss of blood inherent in the blood donation procedure.

There are a number of other limitations to the study beyond the lack of an unambiguous baseline period. For example, although there were no significant differences in key demographic characteristics such as age and previous blood donation experience between those high and low in alexithymia, the fact that the present research was not the primary focus of the larger intervention trial meant that limited information was available about other possible characteristics that may have distinguished those high and low in alexithymia. It seems unlikely that there were any obvious nonpsychologic confounds that could explain the results, but this issue should be examined in closer detail. More important, the possible involvement of other psychologic constructs should

be addressed in future research. For example, it is possible that the present results could be explained by a general neuroticism or tendency toward negative affectivity associated with alexithymia as opposed to alexithymia per se. Alexithymia is an important psychologic construct that is related to a number of health outcomes. Further research is required to specify the mechanisms of these links and its relations to other health-related personality constructs.

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References

1. Nemiah JC, Fricchione GL, Sifneos PE. Alexithymia: a view of the psychosomatic process. In: Hill OW, ed. Modern Trends in Psychosomatic Medicine, vol 3. London: Butterworths; 1976:430–9.
2. Sifneos PE. The prevalence of 'alexithymic' characteristics in psychosomatic patients. Psychother Psychosom 1973;22:255–62.
3. Lane RD, Sechrest L, Reidel R, Weldon V, Kaszniak A, Schwartz GE. Impaired verbal and nonverbal emotion recognition in alexithymia. Psychosom Med 1996;58:203–10.
4. Taylor GJ, Bagby RM, Parker JD. The alexithymia construct: a potential paradigm for psychosomatic medicine. Psychosomatics 1991;32:153–64.
5. Todarello O, Taylor GJ, Parker JDA, Fanelli M. Alexithymia in essential hypertension and psychiatric outpatients: a comparative study. J Psychosom Res 1995;39:987–94.
6. Jula A, Salminen JK, Saarijärvi S. Alexithymia: a facet of essential hypertension. Hypertension 1999;33:1057–61.
7. Dirks JF, Robinson S, Dirks DL. Alexithymia and the psychomaintenance of bronchial asthma. Psychother Psychosom 1981;36:63–71.
8. Porcelli P, Taylor GJ, Bagby RM, De Carne M. Alexithymia and functional gastrointestinal disorders: a comparison with inflammatory bowel disease. Psychother Psychosom 1999;68:263–9.

9. Rybakowski J, Ziolkowski M, Zasdzaka T, Brzezinski R. High prevalence of alexithymia in male patients with alcohol dependence. Drug Alcohol Depend 1988;21:133–6.
10. Parker JDA, Taylor GJ, Bagby RM, Acklin MW. Alexithymia in panic disorder and simple phobia: a comparative study. Am J Psychiatry 1993;150:1105–7.
11. Zeitlin SB, McNally RJ. Alexithymia and anxiety sensitivity in panic disorder and obsessive-compulsive disorder. Am J Psychiatry 1993;150: 658–60.
12. Bourke MP, Taylor GJ, Parker JD, Bagby RM. Alexithymia in women with anorexia nervosa: a preliminary investigation. Br J Psychiatry 1992; 161:240–3.
13. de Groot JM, Rodin G, Olmstead MP. Alexithymia, depression, and treatment outcome in bulimia nervosa. Compr Psychiatry 1995;34: 53–60.
14. Schmidt U, Jiwany A, Treasure J. A controlled study of alexithymia in eating disorders. Compr Psychiatry 1993;34:54–8.
15. Kauhanen J, Kaplan GA, Julkunen J, Salonen JT. The association of alexithymia with all-cause mortality: prospective epidemiologic evidence. Psychosom Med 1994;56:149.
16. Taylor GJ. Recent developments in alexithymia theory and research. Can J Psychiatry 2000;45:134–42.
17. Cohen SC, Rodriguez MS. Pathways linking affective disturbances and physical disorders. Health Psychol 1995;14:374–80.
18. Lumley MA, Stettner L, Wehmer F. How are alexithymia and physical illness linked? A review and critique of pathways. J Psychosom Res 1996;41:505–18.

19. Wehmer F, Brejnak C, Lumley M, Stettner L. Alexithymia and physiological reactivity to emotion-provoking visual scenes. J Nerv Ment Dis 1995;183:351–7.
20. Newton TL, Contrada RJ. Alexithymia and repression: contrasting emotion- focused coping styles. Psychosom Med 1994;56:457–62.
21. Linden W, Lenz JW, Stossel C. Alexithymia, defensiveness and cardiovascular reactivity to stress. J Psychosom Res 1996;41:575–83.
22. Friedlander L, Lumley MA, Farchione T, Doyal G. Testing the alexithymia hypothesis: physiological and subjective responses during relaxation and stress. J Nerv Ment Dis 1997;185:233–9.
23. Waldstein SR, Kauhanen J, Neumann SA, Katzel LI. Alexithymia and cardiovascular risk in older adults: psychosocial, psychophysiological, and biomedical correlates. Psychol Health 2002;17:597–610.
24. Infrasca R. Alexithymia, neurovegetative arousal and neuroticism. An experimental study. Psychother Psychosom 1997;66:276 –80.
25. Paez D, Basabe N, Valdoseda M, Velasco C, Iraurgi I. Confrontation: inhibition, alexithymia, and health. In: Pennebaker JW, ed. Emotion, Disclosure & Health. Washington, DC: American Psychological Association; 1995:195–222.
26. Bagby RM, Taylor GJ, Ryan DP. Toronto Alexithymia Scale: relationship with personality and psychopathology measures. Psychother Psychosom 1986;45:207–15.
27. Deary IJ, Scott S, Wilson JA. Neuroticism, alexithymia and medically unexplained symptoms. Pers Individ Dif 1997;22:551–64.
28. Taylor GJ, Parker JD, Bagby RM, Acklin MW. Alexithymia and somatic complaints in psychiatric out-patients. J Psychosom Res 1992;36:417–24.

29. Wise TN, Mann LS. The attribution of somatic symptoms in psychiatric outpatients. Compr Psychiatry 1995;36:407–10.

30. Kauhanen J, Julkunen J, Salonen JT. Alexithymia and perceived symptoms: criterion validity of the Toronto Alexithymia Scale. Psychother Psychosom 1991;56:247–52.

31. Nyklíček I, Vingerhoets AD. Alexithymia is associated with low tolerance to experimental painful stimulation. Pain 2000;85:471–5.

32. Dalton JA, Feuerstein M. Fear, alexithymia, and cancer pain. Pain 1989; 38:159–70.

33. Spielberger CD. Manual for the State-Trait Anxiety Inventory (STAI). Palo Alto, CA: Consulting Psychologists Press; 1983.

34. Meade MA, France CR, Peterson LM. Predicting vasovagal reactions in volunteer blood donors. J Psychosom Res 1996;40:495–501.

35. Bagby RM, Taylor GJ, Parker JDA. The twenty-item Toronto Alexithymia Scale: II. Convergent, discriminant, and concurrent validity. J Psychosom Res 1994;38:33–40.

36. Bagby RM, Parker JDA, Taylor GJ. The twenty-item Toronto Alexithymia Scale: I. Item selection and cross-validation of the factor structure. J Psychosom Res 1994;38:23–32.

37. Adler P, Ditto B, France CR, Frances A. Cardiovascular reactions to blood donation in offspring of hypertensives and normotensives. J Psychosom Res 1994;38:429–39.

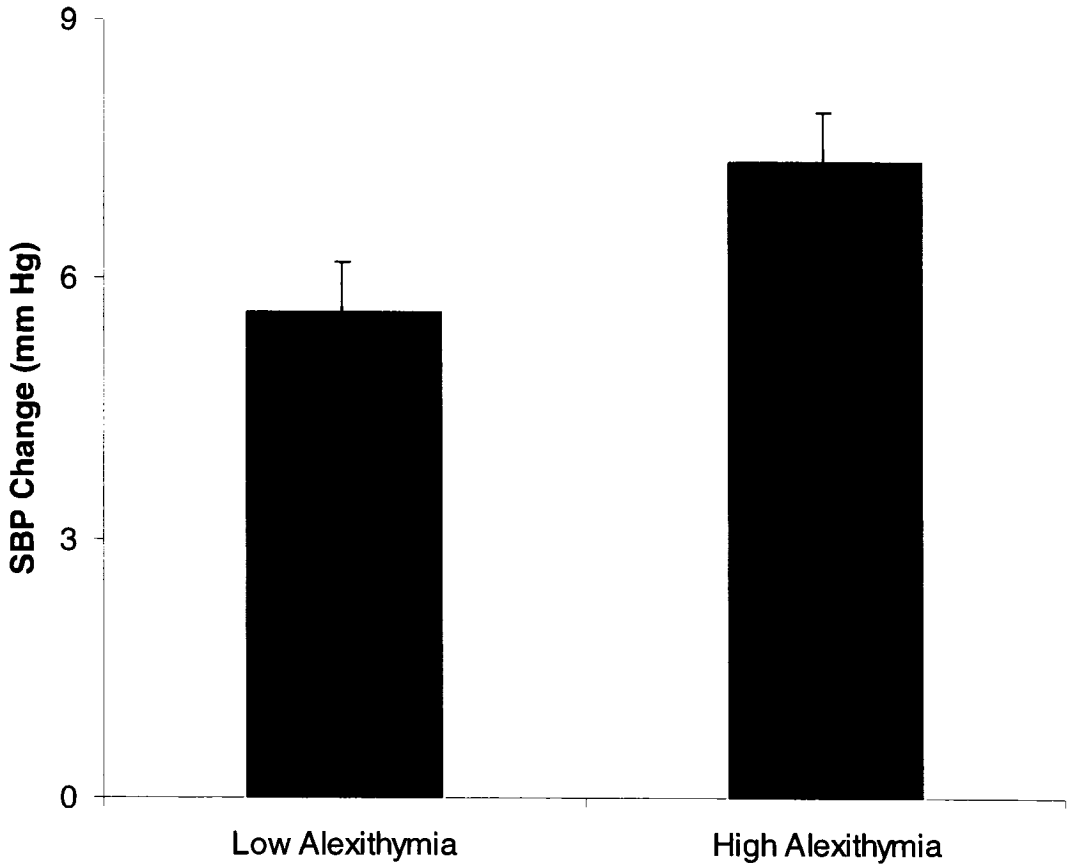
Table 1

Predictors of Dichotomous (Yes/No) Variables

	Odds Ratio (Probability)		
Variable	Gender	Alexithymia	Donation Experience
Chair reclined	0.43 (0.035)	0.99 (0.741)	0.10 (0.001)
Donor fainted	0.15 (0.013)	0.99 (0.603)	0.21 (0.040)
Full unit obtained	2.42 (0.066)	1.00 (0.840)	9.27 (0.002)

Figure legends

Figure 1. Change in systolic blood pressure (predonation systolic blood pressure – postdonation systolic blood pressure) based on median split of alexithymia.


9
6
3
0
SBP Change (mm Hg)
Low Alexithymia
High Alexithymia

General Discussion

The preceding studies aimed to contribute to the body of research that focuses on the investigation of the relationship between alexithymia and illness. More specifically, two pathways proposed to link alexithymia with illness were examined. Study One addressed the hypothesis that alexithymia leads to organic disease through physiological mechanisms by evaluating autonomic reactivity to emotion-inducing stimuli in the laboratory. Study Two extended the examination of physiological reactivity to a more naturalistic setting, and involved a qualitatively different stressor. Moreover, it also dealt with the question of symptom reporting as it relates to alexithymia. Overall, findings support the view that both pathways may contribute to the alexithymia-illness association, but it is important to interpret these data within a larger context.

From previous studies of alexithymia and autonomic nervous system (ANS) regulation, two opposing models have emerged. The hyperarousal model proposes that increased sympathetic nervous system (SNS) reactivity is characteristic of the alexithymic response to emotion-inducing stimuli, and elevated tonic levels of sympathetic activity may also be present. Although greater parasympathetic (PNS) withdrawal may be implicit in the hyperarousal model as well, it has almost never been directly assessed. Conversely, the hypoarousal model posits that alexithymia is associated with dampened subjective emotional experience and attenuated SNS reactivity to emotional stressors. As reviewed previously, there has generally been more support for this latter model.

The finding of an association between alexithymia and greater predonation systolic blood pressure (SBP) reactivity in Study Two would be consistent with a

hyperarousal model. Reports of hyperreactivity such as this have been relatively rare. In fact, increased SBP reactivity among alexithymic individuals identified with the Toronto Alexithymia Scale (TAS) has been reported on only one occasion, by Waldstein et al. (2002), in a group of older adults during an anger recall task. Increased heart rate in response to a distressing film was also reported by Luminet et al. (2004), again in older adults, with alexithymia measured using the Bermond-Vorst Alexithymia Questionnaire. It is interesting that the donation-related anxiety elicited in Study Two, and anger, the focus of Waldstein et al.'s study, are purported to be the best candidate emotions for promoting reactivity and contributing to disease (Lovallo & Gerin, 2003). Findings of significant associations between alexithymia and reactivity in older groups may have been facilitated by known age-related increases in SBP reactivity (Uchino, Holt-Lunstad, Bloor, & Campo, 2005) and the positive association of alexithymia with age (Pasini, Chiaie, Seripa, & Ciani, 1992; Salminen, Saarijärvi, Äärelä, Toikka, & Kauhanen, 1998). The fact that the sample in Study Two was comprised primarily of healthy young adults makes the hyperreactivity finding particularly interesting.

Interpretation of the physiological data in Study One is rendered somewhat more complicated as the results do not appear to fit neatly into a hyper- or hypoarousal model. The hyporeactivity label has generally been applied when no change in heart rate, influenced by both SNS and PNS input, or more rarely, when no change in a specific index of SNS activity has been observed. Including indices of PNS activity, such as respiratory sinus arrhythmia (RSA), changes somewhat the traditional notion of hyporeactivity. The nature of emotional responding is such that it is possible to observe increased vagal activity, classically associated with relaxation, while at the same time

find increased sympathetic activity, typically interpreted as arousal, or other complex interactions involving varying degrees of SNS and PNS response. In Study One, while the magnitude of RSA change was not significantly greater than zero, it did differ significantly from the response observed in the other groups, and tended to decrease rather than increase. Interpretation of this result within the hypo- / hyperreactivity framework would lead to the conclusion that the high alexithymia male group was hyporeactive, since the overall RSA change value across all films did not differ significantly from zero, and no changes in heart rate, blood pressure, or specific indices of SNS activity were observed. However, evidence now argues against the utility of simple SNS-hyperarousal models (Kamarck & Lovallo, 2003). Instead, focus has shifted to models that characterize cardiovascular reactivity as a function of two dimensions, sympathetic and parasympathetic, emphasizing the balance of contributions from each (Berntson, Cacioppo, Quigley, & Fabro, 1994).

The difference in SBP reactivity findings in Studies One and Two raise some questions about the nature of the emotional stressors used in each study. Whereas alexithymia was associated with greater predonation SBP increase in Study Two, no similar effect was observed in response to the film clips in Study One. There are a few possible explanations for this discrepancy. As explained previously, alexithymia is characterized by a deficit in emotional processing. Thus, for alexithymic individuals, complex emotional information such as films that portray emotion-related nuances of voice and facial expression may be difficult to decipher. The result may be a lack of engagement, leading to diminished physiological reactivity. However, when faced with an anxiety-provoking unambiguous stressor that elicits a more basic "fight or flight"

response, such as venipuncture, minimal emotional processing is required, and concomitant physiological arousal occurs. For alexithymic individuals, a reduced capacity to engage in effective emotion regulation strategies may amplify and prolong this arousal. These remain somewhat speculative arguments, though, and additional investigation would be required to determine how varying particular qualities of an emotional stressor may influence response, and to clarify alexithymia-related limitations in emotion regulation strategies.

An alternative and more parsimonious explanation for the observed discrepancy in SBP reactivity between the two studies is the temporal difference in blood pressure readings in relation to the emotional stressors in each case. It is possible increases in blood pressure occurred in anticipation of stimulus presentation in Study One as well, but given the rationale and design of the study, obtaining readings at that time point was not considered relevant.

Neither Study One nor Study Two found significant alexithymia-related baseline differences in any of the physiological variables measured. This is generally consistent with the findings in the psychophysiological literature reviewed earlier, with seven out of ten studies reporting no clear baseline differences. While it is possible, but unlikely, that baseline measures may have been confounded by the hemodynamics of blood donation in Study Two, the lack of baseline differences in Study One is more difficult to contest, particularly given the wide array of measures obtained.

In terms of subjective experience of emotion, Study One did not find alexithymia-related differences in intensity or valence ratings, although ratings of certain positive emotion adjectives were lower. Interestingly, this corresponds to the peculiarities

observed in anxiety ratings in Study Two, where donors higher in alexithymia reported feeling less comfortable, calm, relaxed, and pleasant, but not more anxious than donors lower in alexithymia. This apparent tendency for alexithymic individuals to underreport positive emotions is supported by others' findings of a robust negative correlation between alexithymia and positive affect (Lundh & Simonsson-Sarnecki, 2001). Additional evidence for differences in alexithymic individuals' evaluation and regulation of emotional experience is provided by Study One. The pattern observed suggests that, regardless of the valence of the emotion elicited, the experience of the alexithymic individual in the face of emotional information is one of subjective discomfort, and regulation strategies at the cognitive level seem to revolve around escaping this discomfort by changing the emotional experience. This type of analysis, focused on thoughts underlying evaluation and regulation strategies in alexithymic individuals, has not been reported elsewhere.

The symptom reporting data obtained in Study Two are consistent with the findings of many other studies that have found an association between alexithymia and reporting of somatic complaints (De Gucht & Heiser, 2003), or associations with experimental (Nyklíček & Vingerhoets, 2000) and clinical (Dalton & Feuerstein, 1989) pain. Unlike many previous studies that have neglected to adequately rule out medical explanations for perceived symptoms, the unique advantage of the approach used in Study Two is the opportunity it provided to evaluate the legitimacy of symptoms without the need for a more resource-intensive prospective approach. While it is true that no specific medical testing was conducted to investigate possible underlying pathology, the screening process at the clinics excludes donors with a variety of medical conditions.

Further, a sample comprised primarily of young adults and the induction of a limited set of symptoms characteristic of a vasovagal reaction limited the likelihood that reported symptoms could be attributed to disease processes. The increased anxiety reported by donors higher in alexithymia in conjunction with more symptoms and greater pain offers additional support for the argument that their perception of symptoms was attributable to psychological rather than physical factors.

Implications of Findings

Individual differences in physiological responses to environmental events have long been of interest in psychosomatic medicine, based largely on the assumption that they serve as a window into psychological and physiological processes implicated in disease development. More specifically, cardiovascular reactivity differences may serve as a marker for development of hypertension or coronary heart disease (Manuck, 1994). The strongest interpretation of the reactivity hypothesis posits that the effects of exaggerated reactivity accumulate during daily life and play a causal role in disease risk. However, evidence supporting the generalizability of lab-based cardiovascular reactivity to responses in the natural environment is limited (Schwartz et al., 2003). Alternatively, if the observed differences in reactivity do not generalize beyond the lab, they may still serve as markers of vulnerability, but would not play a causal role (Manuck, Kasprowicz, & Muldoon, 1990). Evidence attesting to the predictive validity of cardiovascular reactivity for development of hypertension over the long term, or for development of some preclinical disease states, is reviewed by Treiber et al. (2003).

Although measures of ANS reactivity are frequently derived from assessment of cardiovascular functioning, the implications of individual differences in ANS response

may extend beyond prediction of cardiovascular disease. To illustrate, in a line of research that examined the effects of brief psychological stressors on multiple aspects of autonomic, neuroendocrinologic, and immunologic responses, Cacioppo and colleagues (Cacioppo, 1994; Cacioppo et al., 1995) found that individual differences in heart rate reactivity predicted neuroendocrine and immune reponses. It thus appears that a coordinated regulatory mechanism governs the interaction and response of multiple systems to stressors. It is not surprising, then, that others have also documented alexithymia-related differences in neuroendocrine and immune response (e.g., Corcos et al., 2004; Dewaraja et al., 1997; Spitzer, Brandl, Rose, Nauck, & Freyberger, 2005).

If a proclivity to seek care is a consequence of the association between alexithymia and symptom reporting observed in Study Two, then this finding may have some implications for health care services. Some evidence suggesting that alexithymia is associated with frequent use of health services is provided by Jyväsjärvi et al. (1999), but this was true only for men. Lumley and Norman (1996) also found that, even when controlling for somatic complaints, the difficulty identifying feelings subscale of the 20-item TAS was linked to increased use of outpatient treatment. The externally oriented thinking dimension of alexithymia, however, was independently associated with decreased use of outpatient treatment. At the very least, the symptom reporting results of Study Two offer some confirmatory evidence for the positive association between alexithymia and medically unexplained symptoms observed by others (De Gucht & Heiser, 2003). Taken further, they may suggest a need for more comprehensive psychological assessment in frequent attenders in the interest of efficient allocation of health care resources.

Limitations

The assessment of alexithymia through self-report has some inherent limitations related to the nature of the alexithymia construct, in part defined by impaired introspective abilities. The implication is that the presence of the alexithymia trait may make it difficult to accurately judge one's ability to monitor and report on internal emotional states (Lane, Ahern, Schwartz, & Kaszniak, 1997; Lundh, Johnsson, Sundqvist, & Olsson, 2002). Unfortunately, there remains a lack of psychometrically sound methods of measuring alexithymia that do not rely on self-report. It is hoped that the introduction of some new observer-rated measures such as the Observer Alexithymia Scale (Haviland, Warren, & Riggs, 2000), or structured interviews like the Toronto Structured Interview for Alexithymia (Bagby, Taylor, Parker, & Dickens, 2006), may help overcome the limitations of self-report instruments.

One common oversight in many studies of alexithymia may be a failure to consider the influence of intervening variables. This is particularly true in research that has examined the association between alexithymia and medically unexplained symptoms, where some have argued that a response bias of excessive symptom reporting may be present because of the overlap between alexithymia and neuroticism or negative affectivity (Lundh & Simonsson-Sarnecki, 2001; Wise & Mann, 1994). For example, Lundh and Simonsson-Sarnecki found that alexithymia showed no association with somatic complaints in a community sample of 137, 25 to 51 year-old individuals when trait anxiety and depression were controlled. Overlap between the difficulty identifying feelings dimension of the TAS-20 and anxiety has also been reported in a sample of patients with depressive and anxiety disorders (Marchesi, Brusamonti, & Maggini, 2000).

Given this potential for overlap, it is not clear to what extent anxiety versus alexithymia may have been responsible for the increase in self-reported vasovagal symptoms observed in Study Two. Deary et al. (1997) maintain that alexithymia contributes independently to the prediction of medically unexplained symptoms, beyond negative affectivity, while some have implicated neuroticism or positive affect as mediating variables (De Gucht et al., 2004; Wise & Mann, 1994). Still others have found neuroticism and alexithymia to be independent (Vingerhoets, Van Heck, Grim, & Bermond, 1995).

To the extent that the data presented here are derived primarily from a young adult university student population, the generalizability of results is limited. This has been an issue for most psychophysiological investigations of alexithymia, although some recent studies have begun to target other age groups (e.g., Luminet et al., 2004; Waldstein et al., 2002). One potential difficulty that arises in a student population is increased levels of psychological mindedness (Lane, Sechrest, & Riedel, 1998), a construct that is negatively correlated with alexithymia (Bagby & Taylor, 1997b), thereby reducing the frequency of clinically alexithymic individuals in the sample. In the sample of 610 blood donors in Study Two, only 37 individuals, or 6% of the sample, scored above the TAS-20 cutoff of 61 that is indicative of clinical alexithymia (Bagby & Taylor, 1997b). Still, the finding of a significant association between alexithymia and SBP reactivity in this sample suggests that alexithymia need not be at a clinical level to influence cardiovascular reactivity, supporting the notion of alexithymia as a dimensional rather than categorical construct.

Future Research

To address the problem of generalizability, psychophysiological investigation of alexithymia must move from the laboratory to more naturalistic settings and extend beyond the usual pool of undergraduate psychology students. Regarding the latter, the issue may be more than simply generalizability of results. Restricting study to student samples that are typically healthy, younger, and more psychologically minded than the general population may reduce the likelihood of obtaining significant findings. While some epidemiological and psychometric studies have been conducted in community samples, this has not been the norm for psychophysiological research in this area. Inclusion of clinical populations such as patients with functional gastrointestinal disorders, in which alexithymia is known to be prevalent, would also be advantageous.

Additional methodological issues relating to the nature of the stressors that have typically been used in studying reactivity remain to be solved. It is frequently acknowledged that many tasks commonly used in the laboratory to elicit reactivity, such as mental arithmetic or the cold pressor, lack ecological validity (van Doornen & Turner, 1992). Alexithymia is a deficit in emotional processing, and much of human emotional experience occurs within the context of social interaction. Ideally, improved laboratory-to-field generalization of physiological response could best be accomplished through studying emotional arousal associated with social interaction in real-world settings. Technological advances in ambulatory physiological recording devices and handheld computers have made this a reliable and practical approach to data collection. An alternative to the ideal of ambulatory assessment is role-played interpersonal interaction in the laboratory (Waldstein, Neumann, Burns, & Maier, 1998). Indeed, Linden et al.

(1998) have presented a convincing case for the utility of social stressors in physiological reactivity research.

If a better understanding of physiological responding in alexithymics is to be achieved, measurement of this response must become more comprehensive. For example, impedance cardiography is a noninvasive methodology that provides data enabling computation of a number of indices of cardiac performance such as preejection period, stroke volume, and total peripheral resistance. Taken together with blood pressure and heart rate variability measures, a detailed picture of ANS reactivity can be established. In addition to assessment of reactivity through computation of pre- to poststimulus change scores, further effort should be directed to evaluation of anticipatory reactivity and patterns of recovery. However, assessment of physiological functioning should not be limited to the cardiovascular system, as response to stressors also involves interactions among the autonomic, neuroendocrine and immune systems. To accomplish research that requires expertise from areas as diverse as social psychology to psychophysiology, endocrinology and immunology, collaborative efforts will be necessary. Not only will this improve study design, but diversity of perspectives will encourage a balanced approach to interpretation and reporting of findings.

General References

Adler, P., Ditto, B., France, C. R., & France, J. (1994). Cardiovascular reactions to blood donation in offspring of hypertensives and normotensives. *Journal of Psychosomatic Research, 38,* 429-439.

Apfel, R. J. & Sifneos, P. E. (1979). Alexithymia: Concept and measurement. *Psychotherapy and Psychosomatics, 32,* 180-190.

Bach, M., Bach, D., de Zwaan, M., Serim, M., & Böhmer, F. (1996). Validierung der deutschen version der 20-item Toronto-Alexithymia-Skala bei normalpersonen und psychiatrischen patienten. *Psychotherapie, Psychosomatik Medizinische, Psychologie, 46,* 23-28.

Bagby, R. M. & Taylor, G. J. (1997a). Affect dysregulation and alexithymia. In G. J. Taylor, R. M. Bagby, & J. D. Parker (Eds.), *Disorders of affect regulation: Alexithymia in medical and psychiatric illness* (pp. 26-45). Cambridge, UK: Cambridge University Press.

Bagby, R. M. & Taylor, G. J. (1997b). Measurement and validation of the alexithymia construct. In G. J. Taylor, R. M. Bagby, & J. D. Parker (Eds.), *Disorders of affect regulation: Alexithymia in medical and psychiatric illness* (pp. 46-66). Cambridge, UK: Cambridge University Press.

Bagby, R. M., Taylor, G. J., & Atkinson, L. (1988). Alexithymia: A comparative study of three self-report measures. *Journal of Psychosomatic Research, 32,* 107-116.

Bagby, R. M., Taylor, G. J., Parker, J. D., & Dickens, S. E. (2006). The development of the Toronto Structured Interview for Alexithymia: Item selection, factor structure, reliability and concurrent validity. *Psychotherapy and Psychosomatics, 75,* 25-39.

Bagby, R. M., Taylor, G. J., & Ryan, D. P. (1986). The measurement of alexithymia: Psychometric properties of the Schalling-Sifneos personality scale. *Comprehensive Psychiatry, 27,* 287-294.

Bagby, R. M., Parker, J. D. A., & Taylor, G. J. (1994a). The twenty-item Toronto Alexithymia Scale: I. Item selection and cross-validation of the factor structure. *Journal of Psychosomatic Research, 38,* 23-32.

Bagby, R. M., Taylor, G. J., & Parker, J. D. A. (1994b). The twenty-item Toronto Alexithymia Scale: II. Convergent, discriminant, and concurrent validity. *Journal of Psychosomatic Research, 38,* 33-40.

Beckendam, C. C. (1997). *Dimensions of emotional intelligence: Attachment, affect regulation, alexithymia and empathy.* Doctoral dissertation, The Fielding Institute, Santa Barbara, CA.

Bermond, B., Vorst, H. C. M., Vingerhoets, A. J. J. M., & Gerritsen, W. (1999). The Amsterdam Alexithymia Scale: Its psychometric values and correlations with other personality traits. *Psychotherapy and Psychosomatics, 68,* 241-251.

Berntson, G., Cacioppo, J., Quigley, K., & Fabro, V. (1994). Autonomic space and psychophysiological response. *Psychophysiology, 31,* 44-61.

Berntson, G. G., Cacioppo, J. T., & Quigley, K. S. (1991). Autonomic determinism: The modes of autonomic control, the doctrine of autonomic space, and the laws of autonomic constraint. *Psychological Review, 98,* 459-487.

Blanchard, E. B., Arena, J. G., & Pallmeyer, T. P. (1981). Psychometric properties of a scale to measure alexithymia. *Psychotherapy and Psychosomatics, 35,* 64-71.

Bucci, W. (1997a). *Psychoanalysis and cognitive science: a multiple code theory.* New York: Guilford.

Bucci, W. (1997b). Symptoms and symbols: a multiple code theory of somatization. *Psychoanalytic Inquiry, 17,* 151-172.

Cacioppo, J. T. (1994). Social neuroscience: Autonomic, neuroendocrine, and immune responses to stress. *Psychophysiology, 31,* 113-128.

Cacioppo, J. T., Malarkey, W. B., Kiecolt-Glaser, J. K., Uchino, B. N., Sgoutas-Emch, S. A., Sheridan, J. F., Bernston, G. G., & Glaser, R. (1995). Heterogeneity in neuroendocrine and immune responses to brief psychological stressors as a function of autonomic cardiac activation. *Psychosomatic Medicine, 57,* 154-164.

Corcos, M., Guilbaud, O., Paterniti, S., Curt, F., Hjalmarsson, L., Moussa, M., Chambry, J., Loas, G., Chaouat, G., & Jeammet, P. (2004). Correlation between serum levels of interleukin-4 and alexithymia scores in healthy female subjects: preliminary findings. *Psychoneuroendocrinology, 29,* 557-562.

Costa, P. T. & McCrae, R. R. (1985). *The NEO Personality Inventory manual.* Odessa, FL: Psychological Assessment Resources.

Costa, P. T. & McCrae, R. R. (1992). *Revised NEO Personality Inventory (NEO-PI-R) and NEO Five-Factor-Inventory (NEO-FFI) professional manual.* Odessa, FL: Psychological Assessment Resources.

Dalton, J. A. & Feuerstein, M. (1989). Fear, alexithymia, and cancer pain. *Pain, 38,* 159-170.

De Gucht, V. & Fischler, B. (2002). Somatization: A critical review of conceptual and methodological issues. *Psychosomatics, 43,* 1-9.

De Gucht, V., Fischler, B., & Heiser, W. (2004). Neuroticism, alexithymia, negative affect, and positive affect as determinants of medically unexplained symptoms. *Personality and Individual Differences, 36,* 1655-1667.

De Gucht, V. & Heiser, W. (2003). Alexithymia and somatisation: A quantitative review of the literature. *Journal of Psychosomatic Research, 54,* 425-434.

Deary, I. J., Scott, S., & Wilson, J. A. (1997). Neuroticism, alexithymia and medically unexplained symptoms. *Personality and Individual Differences, 22,* 551-564.

Dewaraja, R., Tanigawa, T., Araki, S., Nakata, A., Kawamura, N., Ago, Y., & Sasaki, Y. (1997). Decreased cytotoxic lymphocyte counts in alexithymia. *Psychotherapy and Psychosomatics, 66,* 83-86.

Erni, T., Lötscher, B., & Modestin, J. (1997). Two factor solution of the 20-Item Toronto Alexithymia Scale confirmed. *Psychopathology, 30,* 335-340.

Faryna, A., Rodenhauser, P., & Torem, M. (1986). Development of an analog alexithymia scale. *Psychotherapy and Psychosomatics, 45,* 201-206.

Friedlander, L., Lumley, M. A., Farchione, T., & Doyal, G. (1997). Testing the alexithymia hypothesis: Physiological and subjective responses during relaxation and stress. *Journal of Nervous & Mental Disease, 185,* 233-239.

Gardos, G., Schniebolk, S., Mirin, S. M., Wolk, P. C., & Rosenthal, K. (1984). Alexithymia: Towards validation and measurement. *Comprehensive Psychiatry, 25,* 278-282.

Gross, J. J. & Levenson, R. W. (1995). Emotion elicitation using films. *Cognition and Emotion, 9,* 87-108.

Gross, J. J. & Levenson, R. W. (1997). Hiding feelings: The acute effects of inhibiting negative and positive emotion. *Journal of Abnormal Psychology, 106,* 95-103.

Haviland, M. G., Warren, W. L., Riggs, M. L., & Gallacher, M. (2001). Psychometric properties of the Observer Alexithymia Scale in a clinical sample. *Journal of Personality Assessment, 77,* 176-186.

Haviland, M. G., Warren, W. L., Riggs, M. L., & Nitch, S. R. (2002). Concurrent validity of two observer-rated alexithymia measures. *Psychosomatics, 43,* 472-477.

Haviland, M. G., Warren, W. L., & Riggs, M. L. (2000). An observer scale to measure alexithymia. *Psychosomatics, 41,* 385-392.

Infrasca, R. (1997). Alexithymia, neurovegetative arousal, and neuroticism: An experimental study. *Psychotherapy & Psychosomatics, 66,* 276-280.

Jarcho, S. (1970). Galen's six non-naturals. *Bulletin of the History of Medicine, 44,* 372-377.

Jessimer, M. & Markham, R. (1997). Alexithymia: a right hemisphere dysfunction specific to recognition of certain facial expressions. *Brain and Cognition, 34,* 246-258.

Jula, A., Salminen, J. K., & Saarijärvi, S. (1999). Alexithymia: A facet of essential hypertension. *Hypertension, 33,* 1057-1061.

Jyväsjärvi, S., Joukamaa, M., Väisänen, E., Larivaara, P., Kivelä, S.-L., & Keinänen-Kiukaanniemi, S. (1999). Alexithymia, hypochondriacal beliefs, and psychological distress among frequent attenders in primary health care. *Comprehensive Psychiatry, 40,* 292-298.

Kamarck, T. W. & Lovallo, W. R. (2003). Cardiovascular reactivity to psychological challenge: Conceptual and measurement considerations. *Psychosomatic Medicine, 65,* 9-21.

Kauhanen, J., Kaplan, G. A., Cohen, R. D., Julkunen, J., & Salonen, J. T. (1996). Alexithymia and risk of death in middle-aged men. *Journal of Psychosomatic Research, 41,* 541-549.

Kauhanen, J., Kaplan, G. A., Cohen, R. D., Salonen, R., & Salonen, J. T. (1994). Alexithymia may influence the diagnosis of coronary heart disease. *Psychosomatic Medicine, 56,* 237-244.

Keltikangas-Jarvinen, L. (1985). Concept of alexithymia: The prevalence of alexithymia in psychosomatic patients. *Psychotherapy and Psychosomatics, 44,* 132-138.

Kleiger, J. H. & Jones, N. F. (1980). Characteristics of alexithymic patients in a chronic respiratory illness population. *The Journal of Nervous and Mental Disease, 168,* 465-470.

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.

Krystal, J. H., Giller, E. L., Jr., & Cicchetti, D. V. (1986). Assessment of alexithymia in post-traumatic stress disorder and somatic illness: Introduction of a reliable measure. *Psychosomatic Medicine, 48,* 84-94.

Lane, R. D., Sechrest, L., & Riedel, R. (1998). Sociodemographic correlates of alexithymia. *Comprehensive Psychiatry, 39,* 377-385.

Lane, R. D., Ahern, G. L., Schwartz, G. E., & Kaszniak, A. W. (1997). Is alexithymia the emotional equivalent of blindsight. *Biological Psychiatry, 42,* 834-844.

Lane, R. D. & Schwartz, G. E. (1987). Levels of emotional awareness: a cognitive developmental theory and its application to psychopathology. *American Journal of Psychiatry, 144,* 133-143.

Lane, R. D., Sechrest, L., Reidel, R., Weldon, V., Kaszniak, A., & Schwartz, G. E. (1996). Impaired verbal and nonverbal emotion recognition in alexithymia. *Psychosomatic Medicine, 58,* 203-210.

Lang, P. J. (1980). Behavioral treatment and biobehavioral assessment: Computer applications. In J. B. Sidowski, J. H. Johnson, & E. A. Williams (Eds.), *Technology in mental health care delivery systems* (pp. 119-137). Norwood, NJ: Ablex.

Lesser, I. M., Ford, C. V., & Friedmann, C. T. H. (1979). Alexithymia in somatizing patients. *General Hospital Psychiatry, 1,* 256-261.

Linden, W., Lenz, J. W., & Stossel, C. (1996). Alexithymia, defensiveness and cardiovascular reactivity to stress. *Journal of Psychosomatic Research, 41,* 575-583.

Linden, W., Rutledge, T., & Con, A. (1998). A case for the usefulness of laboratory social stressors. *Annals of Behavioral Medicine, 20,* 310-316.

Linden, W., Wen, F., & Paulhus, D. L. (1995). Measuring alexithymia: Reliability, validity, and prevalence. In J. N. Butcher & C. D. Spielberger (Eds.), *Advances in personality assessment* (pp. 51-95). Hillsdale, NJ: Lawrence Erlbaum Associates, Inc.

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,* 139-144.

Lovallo, W. R. & Gerin, W. (2003). Psychophysiological reactivity: Mechanisms and pathways to cardiovascular disease. *Psychosomatic Medicine, 65,* 36-45.

Luminet, O., Rime, B., Bagby, R. M., & Taylor, G. J. (2004). A multimodal investigation of emotional responding in alexithymia. *Cognition and Emotion, 18,* 741-766.

Lumley, M. A. & Norman, S. (1996). Alexithymia and health care utilization. *Psychosomatic Medicine, 58,* 197-202.

Lumley, M. A., Tomakowsky, J., & Torosian, T. (1997). The relationship of alexithymia to subjective and biomedical measures of disease. *Psychosomatics, 38,* 497-502.

Lumley, M. A., Stettner, L., & Wehmer, F. (1996). How are alelxithymia and physical illness linked? A review and critique of pathways. *Journal of Psychosomatic Research, 41,* 505-518.

Lundh, L.-G., Johnsson, A., Sundqvist, K., & Olsson, H. (2002). Alexithymia, memory of emotion, emotional awareness, and perfectionism. *Emotion, 2,* 361-379.

Lundh, L.-G. & Simonsson-Sarnecki, M. (2001). Alexithymia, emotion, and somatic complaints. *Journal of Personality, 69,* 483-510.

MacLean, P. D. (1949). Psychosomatic disease and the "visceral brain": recent developments bearing on the Papez theory of emotion. *Psychosomatic Medicine, 11,* 338-353.

Mann, L. S., Wise, T. N., Trinidad, A., & Kohanski, R. (1994). Alexithymia, affect recognition, and the five-factor model of personality. *Psychological Reports, 74,* 563-567.

Manuck, S. B. (1994). Cardiovascular reactivity in cardiovascular disease: 'Once more unto the breach.'. *International Journal of Behavioral Medicine, 1,* 4-31.

Manuck, S. B., Kasprowicz, A. L., & Muldoon, M. F. (1990). Behaviorally-evoked cardiovascular reactivity and hypertension: conceptual issues and potential associations. *Annals of Behavioral Medicine, 12,* 17-29.

Marchesi, C., Brusamonti, E., & Maggini, C. (2000). Are alexithymia, depression, and anxiety distinct constructs in affective disorders? *Journal of Psychosomatic Research, 49,* 43-49.

Martin, J. B. & Pihl, R. O. (1986). Influence of alexithymic characteristics on physiological and subjective stress responses in normal individuals. *Psychotherapy & Psychosomatics, 45,* 66-77.

Marty, P. & de M'Uzan, M. (1963). La "pensée opératoire". *Revue Française de Psychoanalyse, 27 (suppl.),* 1345-1356.

Meade, M. A., France, C. R., & Peterson, L. M. (1996). Predicting vasovagal reactions in volunteer blood donors. *Journal of Psychosomatic Research, 40,* 495-501.

Müller, J., Bühner, M., & Ellgring, H. (2004). The assessment of alexithymia: psychometric properties and validity of the Bermond-Vorst alexithymia questionnaire. *Personality and Individual Differences, 37,* 373-391.

Nemiah, J. C., Freyberger, H., & Sifneos, P. E. (1976). Alexithymia: a view of the psychosomatic process. In O. W. Hill (Ed.), *Modern trends in psychosomatic medicine* (pp. 430-439). London: Butterworths.

Nemiah, J. C. & Sifneos, P. E. (1970). Affect and fantasy in patients with psychosomatic disorders. In O. W. Hill (Ed.), *Modern trends in psychosomatic medicine* (pp. 26-34). London: Butterworths.

Neumann, S. A., Sollers, J. J. I., 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,* 183-195.

Newton, T. L. & Contrada, R. J. (1994). Alexithymia and repression: Contrasting emotion-focused coping styles. *Psychosomatic Medicine, 56,* 457-462.

Nyklíček, I. & Vingerhoets, A. J. J. M. (2000). Alexithymia is associated with low tolerance to experimental painful stimulation. *Pain, 85,* 471-475.

Pandey, R., Mandal, M. K., Taylor, G. J., & Parker, J. D. A. (1996). Cross-cultural alexithymia: Development and validation of a Hindi translation of the twenty-item Toronto Alexithymia Scale. *Journal of Clinical Psychology, 142,* 1150-1155.

Parker, J. D., Taylor, G. J., & Bagby, R. M. (2000). The relationship between emotional intelligence and alexithymia. *Personality and Individual Differences, 30,* 107-115.

Parker, J. D., Taylor, G. J., Bagby, R. M., & Thomas, S. (1991). Problems with measuring alexithymia. *Psychosomatics, 32,* 196-202.

Parker, J. D. A., Taylor, G. J., & Bagby, R. M. (1993). Alexithymia and the recognition of facial expressions of emotion. *Psychotherapy and Psychosomatics, 59,* 197-202.

Parker, J. D. A., Taylor, G. J., & Bagby, R. M. (2003). The 20-Item Toronto Alexithymia Scale III. Reliability and factorial validity in a community population. *Journal of Psychosomatic Research, 55,* 269-275.

Pasini, A., Chiaie, R. D., Seripa, S., & Ciani, N. (1992). Alexithymia as related to sex, age, and educational level: Results of the Toronto Alexithymia Scale in 417 normal subjects. *Comprehensive Psychiatry, 33,* 42-46.

Paulson, J. E. (1985). State of the art alexithymia measurement. *Psychotherapy and Psychosomatics, 44,* 57-64.

Porcelli, P., Affatati, V., Bellomo, A., De Carne, M., Todarello, O., & Taylor, G. J. (2004). Alexithymia and psychopathology in patients with psychiatric and functional gastrointestinal disorders. *Psychotherapy and Psychosomatics, 73,* 84-91.

Porcelli, P., Bagby, R. M., Taylor, G. J., De Carne, M., Leandro, G., & Todarello, O. (2003). Alexithymia as a predictor of treatment outcome in patients with functional gastrointestinal disorders. *Psychosomatic Medicine, 65,* 911-918.

Porcelli, P., Taylor, G. J., Bagby, R. M., & De Carne, M. (1999). Alexithymia and functional gastrointestinal disorders: a comparison with inflammatory bowel disease. *Psychotherapy and Psychosomatics, 68,* 263-269.

Porcelli, P., Zaka, S., Leoci, C., Centonze, S., & Taylor, G. J. (1995). Alexithymia in inflammatory bowel disease. *Psychotherapy and Psychosomatics, 64,* 49-53.

Porges, S. W. (1995). Orienting in a defensive world: Mammalian modifications of our evolutionary heritage. A polyvagal theory. *Psychophysiology, 32,* 301-318.

Reusch, J. (1948). The infantile personality. *Psychosomatic Medicine, 10,* 134-144.

Roedema, T. M. & Simons, R. F. (1999). Emotion-processing deficit in alexithymia. *Psychophysiology, 36,* 379-387.

Salminen, J. K., Saarijärvi, S., Äärelä, E., Toikka, T., & Kauhanen, J. (1998). Prevalence of alexithymia and its association with sociodemographic variables in the general population of Finland. *Journal of Psychosomatic Research, 46,* 75-82.

Schaffer, C. E. (1993). *The role of adult attachment in the experience and regulation of affect.* Doctoral dissertation, Yale University, New Haven, CT.

Schwartz, A. R., Gerin, W., Davidson, K. W., Pickering, T. G., Brosschot, J. F., Thayer, J. F. et al. (2003). Toward a causal model of cardiovascular responses to stress and the development of cardiovascular disease. *Psychosomatic Medicine, 65,* 22-35.

Shipko, S. & Noviello, N. (1984). Psychometric properties of self-report scales of alexithymia. *Psychotherapy and Psychosomatics, 41,* 85-90.

Sifneos, P. E. (1967). Clinical observations on some patients suffering from a variety of psychosomatic diseases. *Acta Medicina Psychosomatica, 7,* 1-10.

Sifneos, P. E. (1973). The prevalence of "alexithymic" characteristics in psychosomatic patients. *Psychotherapy and Psychosomatics, 22,* 255-262.

Sifneos, P. E. (1986). The Schalling-Sifneos personality scale revised. *Psychotherapy and Psychosomatics, 45,* 161-165.

Sifneos, P. E. (1996). Alexithymia: Past and present. *American Journal of Psychiatry, 153,* 137-142.

Spitzer, C., Brandl, S., Rose, H. J., Nauck, M., & Freyberger, H. J. (2005). Gender-specific association of alexithymia and norepinephrine/cortisol ratios. A preliminary report. *Journal of Psychosomatic Research, 59,* 73-76.

Sriram, T. G., Pratap, L., & Shanmugam, V. (1988). Towards enhancing the utility of the Beth Israel Hospital Psychosomatic Questionnaire. *Psychotherapy and Psychosomatics, 49,* 205-211.

Steptoe, A. (1985). Theoretical bases for task selection in cardiovascular psychophysiology. In A. Steptoe, H. Rueddel, & H. Neus (Eds.), *Clinical and methodological issues in cardiovascular psychophysiology* (pp. 6-15). New York: Springer Verlag.

Stone, L. & Nielson, K. A. (2001). Intact physiological response to arousal with impaired emotional recognition in alexithymia. *Psychotherapy and Psychosomatics, 70,* 92-102.

Taylor, G. J. (1994). The alexithymia construct: conceptualization, validation, and relationship with basic dimensions of personality. *New Trends in Experimental and Clinical Psychiatry, 10,* 61-74.

Taylor, G. J. (2000). Recent developments in alexithymia theory and research. *Canadian Journal of Psychiatry, 45,* 134-142.

Taylor, G. J. (2004). Alexithymia: 25 years of theory and research. In I. Nyklíček, L. Temoshok, & A. Vingerhoets (Eds.), *Emotional expression and health: Advances in theory, assessment and clinical applications* (pp. 137-153). New York: Brunner-Routledge.

Taylor, G. J., Bagby, R. M., & Luminet, O. (2000). Assessment of alexithymia: Self-report and observer-rated measures. In R. Bar-On & J. D. Parker (Eds.), *The handbook of emotional intelligence: Theory, development, assessment, and application at home, school, and in the workplace* (pp. 301-319). San Francisco: Jossey-Bass.

Taylor, G. J., Bagby, R. M., & 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.

Taylor, G. J., Ryan, D., & Bagby, R. M. (1985). Toward the development of a new self-report alexithymia scale. *Psychotherapy and Psychosomatics, 44,* 191-199.

Taylor, G. J., Bagby, R. M., & Parker, J. D. A. (1992). The Revised Toronto Alexithymia Scale: Some reliability, validity, and normative data. *Psychotherapy and Psychosomatics, 57,* 34-234.

Taylor, G. J., Bagby, R. M., Ryan, D. P., Parker, J. D. A., Doody, K., & Keefe, P. (1988). Criterion validity of the Toronto Alexithymia Scale. *Psychosomatic Medicine, 50,* 500-509.

Taylor, G. J., Ryan, D. P., & Bagby, R. M. (1986). Toward the development of a new self-report alexithymia scale. *Psychotherapy & Psychosomatics, 44,* 191-199.

Todarello, O., Taylor, G. J., Parker, J. D. A., & Fanelli, M. (1995). Alexithymia in essential hypertension and psychiatric outpatients: A comparative study. *Journal of Psychosomatic Research, 39,* 987-994.

Toner, B. B., Stuckless, N., Ali, A., Downie, F., Emmott, S., & Ackman, D. (1998). The development of a cognitive scale for functional bowel disorders. *Psychosomatic Medicine, 60,* 492-497.

Treiber, F. A., Kamarck, T. W., Schneiderman, N., Sheffield, D., Kapuku, G., & Taylor, T. (2003). Cardiovascular reactivity and development of preclinical and clinical disease states. *Psychosomatic Medicine, 65,* 46-62.

Uchino, B. N., Holt-Lunstad, J., Bloor, L. E., & Campo, R. A. (2005). Aging and cardiovascular reactivity to stress: Longitudinal evidence for changes in stress reactivity. *Psychology and Aging, 20,* 134-143.

van Doornen, L. J. P. & Turner, J. R. (1992). The ecological validity of laboratory stress testing. In J. R. Turner, A. Sherwood, & K. C. Light (Eds.), *Individual differences in cardiovascular response to stress* (pp. 63-83). New York: Plenum.

Vingerhoets, A. J., Van Heck, G. L., Grim, R., & Bermond, B. (1995). Alexithymia: A further exploration of its nomological network. *Psychotherapy & Psychosomatics, 64,* 32-42.

Vorst, H. C. M. & Bermond, B. (2001). Validity and reliability of the Bermond-Vorst Alexithymia Questionnaire. *Personality and Individual Differences, 30,* 413-434.

Waldstein, S. R., Kauhanen, J., Neumann, S. A., & Katzel, L. I. (2002). Alexithymia and cardiovascular risk in older adults: Psychosocial, psychophysiological, and biomedical correlates. *Psychology & Health, 17,* 597-610.

Waldstein, S. R., Neumann, S. A., Burns, H. O., & Maier, K. J. (1998). Role-played interpersonal interaction: Ecological validity and cardiovascular reactivity. *Annals of Behavioral Medicine, 20,* 302-309.

Wehmer, F., Brejnak, C., Lumley, M., & Stettner, L. (1995). Alexithymia and physiological reactivity to emotion-provoking visual scenes. *Journal of Nervous & Mental Disease, 183,* 351-357.

Westermann, R., Spies, K., Stahl, G., & Hesse, F. W. (1996). Relative effectiveness and validity of mood induction procedures: A meta-analysis. *European Journal of Social Psychology, 26,* 557-580.

Wise, T. N. & Mann, L. S. (1994). The relationship between somatosensory amplification, alexithymia, and neuroticism. *Journal of Psychosomatic Research, 38,* 515-521.

Zech, E., Luminet, O., Rimé, B., & Wagner, H. (1999). Alexithymia and its measurement: Confirmatory factor analyses of the twenty-item Toronto Alexithymia Scale and the Bermond-Vorst Alexithymia Questionnaire. *European Journal of Personality, 13,* 511-532.

Appendix A

Ethics Approval Forms

Appendix B

Publication Reprint

Psychosomatic Medicine, *67*, 471-475

Alexithymia, Cardiovascular Reactivity, and Symptom Reporting During Blood Donation

NELSON BYRNE, BSc, AND BLAINE DITTO, PHD

Objective: With blood donation serving as a naturalistic stressor and a controlled medical event, the aim of this study was to examine emotional and cardiovascular reactivity, self-report of vasovagal symptoms, and perceived pain as a function of scores on the Toronto Alexithymia Scale (TAS-20). **Method:** Healthy young adult blood donors (N = 610) recruited at mobile blood collection clinics completed the TAS-20, pre- and postdonation measures of anxiety, postdonation measures of pain and vasovagal symptoms, and had their blood pressure and heart rate measured before and after giving blood. **Results:** Alexithymia score was positively associated with reported anxiety, pain, and vasovagal symptoms. Higher alexithymia was also associated with greater increases in predonation systolic blood pressure in anticipation of blood donation. In general, women and less experienced blood donors reported more vasovagal symptoms than men and more experienced donors, and this corresponded to higher rates of treatment by the nurses, more fainting, and fewer full units of blood obtained. However, despite more reports of vasovagal symptoms by alexithymic donors, alexithymia score was not related to these variables. **Conclusions:** The results suggest that individuals with higher alexithymia scores were more anxious in the blood donation setting and more prone to report physical symptoms in the absence of a clear difference in the medical outcome of the blood donation procedure. **Key words:** alexithymia, symptom reporting, cardiovascular reactivity, blood donation, vasovagal reaction, pain.

BDRI = Blood Donation Reactions Inventory; **DBP** = diastolic blood pressure; **GLM** = general linear model; **HR** = heart rate; **SBP** = systolic blood pressure; **STAI** = Spielberger State-Anxiety Scale; **TAS-20** = Toronto Alexithymia Scale.

INTRODUCTION

The term alexithymia was coined by Nemiah and Sifneos (1,2) in the 1970s to describe patients presenting with psychosomatic concerns who were observed to have difficulty identifying and describing emotions. Additional features of this multifaceted personality construct include difficulty differentiating feelings from the bodily sensations of emotional arousal, a lack of fantasy resulting from constricted emotional abilities, and a pattern of externally oriented thinking. Unlike the concepts of repression and denial, in which identified emotions are kept below the surface of consciousness, alexithymics are thought to lack established emotional representation (3). More succinctly, alexithymia may be defined as a difficulty with cognitive processing of emotion (4).

Interest in the study of alexithymia has increased in recent years, in part as a result of mounting evidence of its association with a number of psychiatric and medical conditions such as hypertension (5,6), asthma (7), chronic pain syndrome (7), functional gastrointestinal disorders (8), alcoholism and substance abuse (9), anxiety disorders (10,11), and eating disorders (12–14). One particularly striking finding is that alexithymia predicted all-cause mortality after 5 years, even after controlling for demographic and medical risk factors (15). A recent review of the epidemiologic literature is provided by Taylor (16).

Although the association between alexithymia and illness is robust, the nature of this relationship is unclear. Possible pathways linking alexithymia and physical illness have been outlined by Lumley and colleagues (17,18). These include: 1) alexithymia leads to organic disease through physiological mechanisms or unhealthy behaviors; 2) alexithymia leads to illness behavior such as overreporting of symptoms and excessive use of health care; 3) physical illness influences psychologic functioning and leads to alexithymia; and 4) sociocultural and biologic factors comprise a third variable, which causes both alexithymia and physical illness.

The hypothesis that alexithymia leads to organic disease through physiological mechanisms is based on findings of altered physiological arousal, which may result from impaired regulation of emotion in alexithymics (4). In general, results point to greater physiological arousal (e.g., higher heart rate and skin conductance) at rest and, consistent with their reports of lower emotional reactivity to stressors, smaller physiological responses to stimuli such as watching emotional slides (19–22)

An alternative explanation for the link between alexithymia and illness stems from the finding that alexithymics tend to report more symptomatology in the absence of illness, perhaps in lieu of emotional complaints (25). Significant positive correlations have been reported between alexithymia and measures of somatization and hypochondriasis (26,27), possibly as a result of alexithymics' focusing on, amplifying, and misinterpreting somatic sensations that accompany emotional arousal. In psychiatric populations, two studies found that alexithymics reported more somatic complaints than nonalexithymic patients (28,29). Kauhanen and colleagues (30) found greater reports of symptoms such as nausea, dizziness, heart palpitations, and headaches among those higher in alexithymia. Alexithymia has also been found to be positively associated with reports of experimental (31) and clinical (32) pain. It remains unclear whether symptom reporting is the result of higher rates of illness in alexithymics or whether illness is more frequently diagnosed because misinterpretation and amplification of somatic sensations leads to increased healthcare utilization.

The blood donation clinic provides an interesting environment in which a number of these issues can be studied. First,

From the Department of Psychology, McGill University, Montreal, Quebec, Canada.

Address correspondence and reprint requests to Blaine Ditto, PhD, Department of Psychology, McGill University, 1205 Dr. Penfield Ave., Montreal, QC H3A 1B1 Canada. E-mail: blaine.ditto@mcgill.ca

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particularly for inexperienced donors, blood donation can be a stressful experience that is associated with increases in anxiety and blood pressure in the predonation waiting period (38). In the present study, changes in anxiety and cardiovascular activity in relation to alexithymia were studied. Second, the nature of the donation procedure is such that vasovagal reactions are not uncommon. The relatively controlled induction of a limited set of somatic sensations served as a useful manipulation to investigate differences in symptom reporting as they relate to alexithymia. Finally, several fairly controlled painful procedures are conducted as part of the blood donation procedure, providing an opportunity to assess pain experience. The present study examined self-reported anxiety, cardiovascular reactivity, symptom reporting, and reported pain in blood donors varying in alexithymia.

METHOD

Participants

As part of a larger study concerning the prediction and prevention of vasovagal reactions to blood donation, 1560 donors were recruited over an 18-month period at mobile blood donor clinics held by the provincial blood collection agency at universities and colleges throughout the Montreal area. Participants for the present study were 610 accepted donors, 263 males and 347 females, who were randomly assigned to the no-treatment control group. Alexithymia was assessed with the widely used 20-item Toronto Alexithymia Scale (TAS-20 (35,36)). There were no significant associations, or anything approaching significance (all $p > .20$), between alexithymia score (mean = 42.1 ± 10.0) and age (mean = 22.4 ± 7.2 years), number of prior blood donations (mean = 3.6 ± 7.3), or body mass index (mean = 23.7 ± 3.8 kg/m^2).

Procedure

After registering at the clinic, prospective participants were directed to a research assistant. Once informed consent had been obtained, they completed a short predonation questionnaire that included an abbreviated version of the Spielberger State-Anxiety Scale (STAI (33)) and demographic questions about age, height, weight, and number of previous blood donations. Blood pressure was measured twice using a B-D Assure manual inflate digital blood pressure monitor (Becton, Dickinson and Co., New Jersey) while seated with the arm supported at heart level. These monitors use the oscillometric principle to measure blood pressure and, according to the manufacturer, are accurate to within ±3 mm Hg. In cases in which there was a notable discrepancy between the first and second readings, blood pressure was measured a third time.

People were then seated in a waiting area before being called by a nurse for a health screening. During the screening, a blood sample was obtained by pricking the fingertip with a disposable lancet. Those deemed eligible to give blood proceeded to the first available donation chair and 450 mL of blood was drawn. Immediately after the blood draw, the attending nurse completed a brief questionnaire concerning issues such as the difficulty of needle insertion, whether the donor's chair had been reclined to treat vasovagal symptoms, and whether a full unit of blood was obtained. With assistance, donors then moved to a rest area where they remained seated on a donation chair for approximately 10 minutes before being met by a research assistant to have blood pressure measurements taken a second time. A set of postdonation questionnaires was provided at this point, which donors completed while consuming refreshments. These questionnaires included the Blood Donation Reactions Inventory (34), a self-report instrument designed to assess vasovagal reactions during blood donation, and the TAS-20. As well, the STAI was administered a second time. Finally, visual analog ratings of pain produced by the predonation finger prick and the venipuncture required to draw blood were requested.

Data Reduction and Analyses

Two predonation and two postdonation blood pressure readings for each subject were averaged to yield one systolic (SBP) and one diastolic (DBP) value at each time point. Similarly, pre- and postdonation heart rate (HR) values were obtained by averaging two HR readings at each time point. To reduce the positive skewness of the Blood Donation Reactions Inventory (BDRI) data, a log transformation (log[BDRI + 1]) was applied to raw scores as in previous research (34). Number of previous blood donations, used as a continuous independent variable representing donation experience, was also subjected to a log transformation to better approximate a normal distribution.

Primary data analyses were conducted within the general linear model (GLM) framework using Systat statistical software (Systat Software, Inc., Point Richmond, CA). Alexithymia, defined as total TAS-20 score, and experience were used as continuous independent variables. Gender was entered as a dichotomous independent variable, along with the interaction effects among gender, alexithymia, and experience. To more closely examine the effects of the blood donation procedure on physiological change in relation to alexithymia, separate analyses of pre- and postdonation blood pressure and HR were conducted, as well as analyses of pre- to postdonation change scores in SBP, DBP, and HR.

Dichotomous yes–no variables such as whether the nurse reclined the donor's chair to treat vasovagal symptoms and whether a full unit of blood was obtained were analyzed using logistic regression equations using gender, previous donation experience, and alexithymia as predictor variables.

RESULTS

Anxiety Ratings

The analysis of predonation anxiety scores yielded significant main effects of donation experience ($F[1,583] = 4.83$, $p = .028$) and alexithymia ($F[1,583] = 11.31$, $p = .001$). Higher predonation anxiety was reported in those with less donation experience and higher alexithymia scores. The analysis of postdonation anxiety scores produced only a similar significant main effect of alexithymia ($F[1,589] = 8.04$, $p = .005$). In general, predonation anxiety scores were significantly higher than postdonation scores ($t[589] = 9.50$, $p < .001$).

Cardiovascular Activity and Change

There were no significant effects involving alexithymia in the analyses of pre- or postdonation blood pressure or HR, or DBP and HR change scores. However, the analysis of systolic blood pressure change scores produced a significant effect of alexithymia ($F[1,593] = 5.02$, $p = .025$). SBP was generally higher while people were waiting to give blood compared with the postdonation refreshment period ($t[600] = 14.91$, $p < .001$). This difference was greater among those with higher alexithymia (Fig. 1). The effect remained significant with addition of postdonation SBP, which might be considered the "baseline" in this situation, as well as whether a full unit of blood was obtained as covariates in the model ($F[1,591] = 4.75$, $p = .030$).

Pain Ratings and Vasovagal Symptom Reporting

Analysis of the fingerprick pain ratings yielded only a significant main effect of alexithymia ($F[1,590] = 4.42$, $p = .036$). Higher alexithymia scores were associated with higher pain ratings. There were no significant effects in the analysis of the venipuncture pain ratings.

The analysis of BDRI scores produced significant main

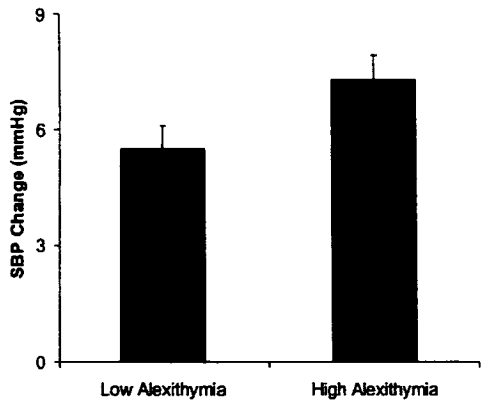

Figure 1. Change in systolic blood pressure (predonation systolic blood pressure − postdonation systolic blood pressure) based on median split of alexithymia.

effects of gender (F[1,597] = 5.12, p = .024), donation experience (F[1,597] = 6.33, p = .012), and alexithymia (F[1,597] = 4.85, p = .028). Women, people with less previous donation experience, and individuals with higher alexithymia scores reported more donation-related symptoms such as dizziness, weakness, and so on.

Medical Characteristics of the Blood Donations

There were no significant effects in the analyses of the nurse's rating of the ease of needle insertion or time in the donation chair. None of the logistic regression equations predicting whether a needle adjustment was required, whether the donor's chair was reclined, whether they fainted, or whether a full unit of blood was obtained produced significant effects of alexithymia. This occurred despite the fact that gender and previous blood donation experience, which were also associated with BDRI scores, were significantly related to whether the donor's chair was reclined to treat a vasovagal reaction, whether the donor fainted, and whether a full unit of blood was obtained (Table 1). Men and more experienced blood donors were less likely to faint or have their chairs reclined and more likely to produce a full unit of blood.

DISCUSSION

Higher symptom reporting, including higher pain ratings, was associated with alexithymia. In addition to more physical symptom reporting, alexithymia was associated with higher self-reported anxiety and a greater difference in pre- and postdonation SBP. Given the fairly controlled nature of "injury" and blood loss produced by this medical intervention, known to produce a predictable set of symptoms, the present results provide some interesting information about the relationship between alexithymia and symptom reporting.

The underlying reason for higher symptom reporting by alexithymics has been the subject of considerable discussion. Some have argued that the reports are a reflection of real underlying illness given the associations between alexithymia and many medical disorders, whereas others have suggested that characteristics such as hypochondriasis or somatic attention mediate the relationship. The cluster of potential symptoms elicited during blood donation includes several that are potentially visible to attending staff, e.g., fainting. Although outright fainting was rare (3%), it was not associated with alexithymia score. There was also no association between alexithymia score and whether the nurse believed it necessary to treat the donor by reclining their chair, despite higher symptom reports by alexithymics. This contrasted with the effects of donation experience and gender, in which there was a concordance between higher symptom reports among less experienced donors and women and various objective measures of outcome such as chair reclining. Thus, although the sensations experienced by donors with higher alexithymia scores were construed as significant symptoms according to their BDRI ratings, they were not considered to be sufficiently severe by attending staff to warrant reclining the donation chair. This suggests that alexithymics have an exaggerated inclination to attend to somatic sensations. At a minimum, they suggest a somewhat greater "disconnect" between "objective" physiological state and symptom reports, although the explanation for this disconnect is unclear.

Given that alexithymia, by definition, involves reduced reporting of emotion, the positive association between alexithymia and STAI scores is somewhat surprising. However, in examining responses to individual items in the five-item abbreviated STAI, it was found that this was the result of donors higher in alexithymia reporting feeling less comfortable, calm, relaxed, and pleasant, but *not* more anxious than donors lower in alexithymia. Thus, these results, as well as the pain data, seem to provide additional evidence of a tendency to report physical discomfort as opposed to negative emotion.

The limited nature of the cardiovascular data makes it difficult to interpret these results, although the apparently greater increase in predonation systolic blood pressure among alexithymics is

TABLE 1. Predictors of Dichotomous (Yes/No) Variables

	Odds Ratio (Probability)		
Variable	Gender	Alexithymia	Donation Experience
Chair reclined	0.43 (0.035)	0.99 (0.741)	0.10 (0.001)
Donor fainted	0.15 (0.013)	0.99 (0.603)	0.21 (0.040)
Full unit obtained	2.42 (0.066)	1.00 (0.840)	9.27 (0.002)

intriguing. Because most (19–22) but not all (e.g., (24)) previous studies of physiological reactivity in alexithymics have indicated a tendency for reduced rather than exaggerated physiological reactivity to stress, it seems unlikely that this reflects a general tendency for high SBP reactivity. It may have been related to the very salient nature of the "stressor," i.e., anticipation of venipuncture with a large needle and loss of a significant amount of blood, and consistent with what appears to have been a greater state of psychologic distress in this environment, even if it was described in physical terms.

That said, another challenge in the interpretation of the SBP finding is the choice of the appropriate baseline period. For several reasons, we view the SBP finding as indicative of a greater increase in SBP during the stressful predonation period in alexithymics as opposed to a greater decrease after donation. First, both SBP and reported anxiety were significantly higher before than after donation. This was true regardless of whether a full unit of blood was obtained from the donor. Second, measurements of resting blood pressure were obtained in the laboratory in a small subsample of these donors of these donors (N = 28) with similar equipment. There was no difference in postdonation and resting laboratory SBP (mean = 112.3 versus 112.4 mm Hg), whereas predonation SBP was significantly higher than resting laboratory SBP (mean = 116.2 versus 112.3 mm Hg; $t[27] = 2.08$, $p = .024$). Finally, researchers commonly operationalize the anticipation period before an aversive event (e.g., electric shock, public speaking, dental examinations) as the stress period and the recovery period as the baseline. The previously discussed results are consistent with this view in the present case. However, this issue needs to be considered with special caution in the present case, especially given the loss of blood inherent in the blood donation procedure.

There are a number of other limitations to the study beyond the lack of an unambiguous baseline period. For example, although there were no significant differences in key demographic characteristics such as age and previous blood donation experience between those high and low in alexithymia, the fact that the present research was not the primary focus of the larger intervention trial meant that limited information was available about other possible characteristics that may have distinguished those high and low in alexithymia. It seems unlikely that there were any obvious nonpsychologic confounds that could explain the results, but this issue should be examined in closer detail. More important, the possible involvement of other psychologic constructs should be addressed in future research. For example, it is possible that the present results could be explained by a general neuroticism or tendency toward negative affectivity associated with alexithymia as opposed to alexithymia per se. Alexithymia is an important psychologic construct that is related to a number of health outcomes. Further research is required to specify the mechanisms of these links and its relations to other health-related personality constructs.

REFERENCES

1. Nemiah JC, Fricchione GL, Sifneos PE. Alexithymia: a view of the psychosomatic process. In: Hill OW, ed. Modern Trends in Psychosomatic Medicine, vol 3. London: Butterworths; 1976:430–9.
2. Sifneos PE. The prevalence of 'alexithymic' characteristics in psychosomatic patients. Psychother Psychosom 1973;22:255–62.
3. Lane RD, Sechrest L, Reidel R, Weldon V, Kaszniak A, Schwartz GE. Impaired verbal and nonverbal emotion recognition in alexithymia. Psychosom Med 1996;58:203–10.
4. Taylor GJ, Bagby RM, Parker JD. The alexithymia construct: a potential paradigm for psychosomatic medicine. Psychosomatics 1991;32:153–64.
5. Todarello O, Taylor GJ, Parker JDA, Fanelli M. Alexithymia in essential hypertension and psychiatric outpatients: a comparative study. J Psychosom Res 1995;39:987–94.
6. Jula A, Salminen JK, Saarijärvi S. Alexithymia: a facet of essential hypertension. Hypertension 1999;33:1057–61.
7. Dirks JF, Robinson S, Dirks DL. Alexithymia and the psychomaintenance of bronchial asthma. Psychother Psychosom 1981;36:63–71.
8. Porcelli P, Taylor GJ, Bagby RM, De Carne M. Alexithymia and functional gastrointestinal disorders: a comparison with inflammatory bowel disease. Psychother Psychosom 1999;68:263–9.
9. Rybakowski J, Ziolkowski M, Zasdzaka T, Brzezinski R. High prevalence of alexithymia in male patients with alcohol dependence. Drug Alcohol Depend 1988;21:133–6.
10. Parker JDA, Taylor GJ, Bagby RM, Acklin MW. Alexithymia in panic disorder and simple phobia: a comparative study. Am J Psychiatry 1993;150:1105–7.
11. Zeitlin SB, McNally RJ. Alexithymia and anxiety sensitivity in panic disorder and obsessive-compulsive disorder. Am J Psychiatry 1993;150: 658–60.
12. Bourke MP, Taylor GJ, Parker JD, Bagby RM. Alexithymia in women with anorexia nervosa: a preliminary investigation. Br J Psychiatry 1992; 161:240–3.
13. de Groot JM, Rodin G, Olmstead MP. Alexithymia, depression, and treatment outcome in bulimia nervosa. Compr Psychiatry 1995;34: 53–60.
14. Schmidt U, Jiwany A, Treasure J. A controlled study of alexithymia in eating disorders. Compr Psychiatry 1993;34:54–8.
15. Kauhanen J, Kaplan GA, Julkunen J, Salonen JT. The association of alexithymia with all-cause mortality: prospective epidemiologic evidence. Psychosom Med 1994;56:149.
16. Taylor GJ. Recent developments in alexithymia theory and research. Can J Psychiatry 2000;45:134–42.
17. Cohen SC, Rodriguez MS. Pathways linking affective disturbances and physical disorders. Health Psychol 1995;14:374–80.
18. Lumley MA, Stettner L, Wehmer F. How are alexithymia and physical illness linked? A review and critique of pathways. J Psychosom Res 1996;41:505–18.
19. Wehmer F, Brejnak C, Lumley M, Stettner L. Alexithymia and physiological reactivity to emotion-provoking visual scenes. J Nerv Ment Dis 1995;183:351–7.
20. Newton TL, Contrada RJ. Alexithymia and repression: contrasting emotion-focused coping styles. Psychosom Med 1994;56:457–62.
21. Linden W, Lenz JW, Stossel C. Alexithymia, defensiveness and cardiovascular reactivity to stress. J Psychosom Res 1996;41:575–83.
22. Friedlander L, Lumley MA, Farchione T, Doyal G. Testing the alexithymia hypothesis: physiological and subjective responses during relaxation and stress. J Nerv Ment Dis 1997;185:233–9.
23. Waldstein SR, Kauhanen J, Neumann SA, Katzel LI. Alexithymia and cardiovascular risk in older adults: psychosocial, psychophysiological, and biomedical correlates. Psychol Health 2002;17:597–610.
24. Infrasca R. Alexithymia, neurovegetative arousal and neuroticism. An experimental study. Psychother Psychosom 1997;66:276–80.
25. Paez D, Basabe N, Valdoseda M, Velasco C, Iraurgi I. Confrontation: inhibition, alexithymia, and health. In: Pennebaker JW, ed. Emotion, Disclosure & Health. Washington, DC: American Psychological Association; 1995:195–222.
26. Bagby RM, Taylor GJ, Ryan DP. Toronto Alexithymia Scale: relationship with personality and psychopathology measures. Psychother Psychosom 1986;45:207–15.

27. Deary IJ, Scott S, Wilson JA. Neuroticism, alexithymia and medically unexplained symptoms. Pers Individ Dif 1997;22:551–64.
28. Taylor GJ, Parker JD, Bagby RM, Acklin MW. Alexithymia and somatic complaints in psychiatric out-patients. J Psychosom Res 1992;36:417–24.
29. Wise TN, Mann LS. The attribution of somatic symptoms in psychiatric outpatients. Compr Psychiatry 1995;36:407–10.
30. Kauhanen J, Julkunen J, Salonen JT. Alexithymia and perceived symptoms: criterion validity of the Toronto Alexithymia Scale. Psychother Psychosom 1991;56:247–52.
31. Nyklicek I, Vingerhoets AD. Alexithymia is associated with low tolerance to experimental painful stimulation. Pain 2000;85:471–5.
32. Dalton JA, Feuerstein M. Fear, alexithymia, and cancer pain. Pain 1989; 38:159–70.
33. Spielberger CD. Manual for the State-Trait Anxiety Inventory (STAI). Palo Alto, CA: Consulting Psychologists Press; 1983.
34. Meade MA, France CR, Peterson LM. Predicting vasovagal reactions in volunteer blood donors. J Psychosom Res 1996;40:495–501.
35. Bagby RM, Taylor GJ, Parker JDA. The twenty-item Toronto Alexithymia Scale: II. Convergent, discriminant, and concurrent validity. J Psychosom Res 1994;38:33–40.
36. Bagby RM, Parker JDA, Taylor GJ. The twenty-item Toronto Alexithymia Scale: I. Item selection and cross-validation of the factor structure. J Psychosom Res 1994;38:23–32.
38. Adler P, Ditto B, France CR, Frances A. Cardiovascular reactions to blood donation in offspring of hypertensives and normotensives. J Psychosom Res 1994;38:429–39.

Appendix C

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Appendix D

Twenty-Item Toronto Alexithymia Scale:

English and French Versions

TAS-20

Using the scale provided as a guide, indicate how much you agree or disagree with each of the following statements by circling the corresponding number. Give only one answer for each statement.

Circle 1 if you STRONGLY DISAGREE
Circle 2 if you MODERATELY DISAGREE
Circle 3 if you NEITHER DISAGREE NOR AGREE
Circle 4 if you MODERATELY AGREE
Circle 5 if you STRONGLY AGREE

1. I am often confused about what emotion I am feeling.

1	2	3	4	5
Strongly Disagree	Moderately Disagree	Neither Disagree Nor Agree	Moderately Agree	Strongly Agree

2. It is difficult for me to find the right words for my feelings.

1	2	3	4	5
Strongly Disagree	Moderately Disagree	Neither Disagree Nor Agree	Moderately Agree	Strongly Agree

3. I have physical sensations that even doctors don't understand.

1	2	3	4	5
Strongly Disagree	Moderately Disagree	Neither Disagree Nor Agree	Moderately Agree	Strongly Agree

4. I am able to describe my feelings easily.

1	2	3	4	5
Strongly Disagree	Moderately Disagree	Neither Disagree Nor Agree	Moderately Agree	Strongly Agree

5. I prefer to analyze problems rather than just describe them.

1	2	3	4	5
Strongly Disagree	Moderately Disagree	Neither Disagree Nor Agree	Moderately Agree	Strongly Agree

6. When I am upset, I don't know if I am sad, frightened, or angry.

1	2	3	4	5
Strongly Disagree	Moderately Disagree	Neither Disagree Nor Agree	Moderately Agree	Strongly Agree

7. I am often puzzled by sensations in my body.

1	2	3	4	5
Strongly Disagree	Moderately Disagree	Neither Disagree Nor Agree	Moderately Agree	Strongly Agree

8. I prefer to just let things happen rather than to understand why they turned out that way.

1	2	3	4	5
Strongly Disagree	Moderately Disagree	Neither Disagree Nor Agree	Moderately Agree	Strongly Agree

9. I have feelings that I can't quite identify.

1	2	3	4	5
Strongly Disagree	Moderately Disagree	Neither Disagree Nor Agree	Moderately Agree	Strongly Agree

10. Being in touch with emotions is essential.

1	2	3	4	5
Strongly Disagree	Moderately Disagree	Neither Disagree Nor Agree	Moderately Agree	Strongly Agree

11. I find it hard to describe how I feel about people.

1	2	3	4	5
Strongly Disagree	Moderately Disagree	Neither Disagree Nor Agree	Moderately Agree	Strongly Agree

12. People tell me to describe my feelings more.

1	2	3	4	5
Strongly Disagree	Moderately Disagree	Neither Disagree Nor Agree	Moderately Agree	Strongly Agree

13. I don't know what's going on inside me.

1	2	3	4	5
Strongly Disagree	Moderately Disagree	Neither Disagree Nor Agree	Moderately Agree	Strongly Agree

14. I often don't know why I am angry.

1	2	3	4	5
Strongly Disagree	Moderately Disagree	Neither Disagree Nor Agree	Moderately Agree	Strongly Agree

15. I prefer talking to people about their daily activities rather than their feelings.

1	2	3	4	5
Strongly Disagree	Moderately Disagree	Neither Disagree Nor Agree	Moderately Agree	Strongly Agree

16. I prefer to watch "light" entertainment shows rather than psychological dramas.

1	2	3	4	5
Strongly Disagree	Moderately Disagree	Neither Disagree Nor Agree	Moderately Agree	Strongly Agree

17. It is difficult for me to reveal my innermost feelings, even to close friends.

1	2	3	4	5
Strongly Disagree	Moderately Disagree	Neither Disagree Nor Agree	Moderately Agree	Strongly Agree

18. I can feel close to someone, even in moments of silence.

1	2	3	4	5
Strongly Disagree	Moderately Disagree	Neither Disagree Nor Agree	Moderately Agree	Strongly Agree

19. I find examination of my feelings useful in solving personal problems.

1	2	3	4	5
Strongly Disagree	Moderately Disagree	Neither Disagree Nor Agree	Moderately Agree	Strongly Agree

20. Looking for hidden meanings in movies or plays distracts from their enjoyment.

1	2	3	4	5
Strongly Disagree	Moderately Disagree	Neither Disagree Nor Agree	Moderately Agree	Strongly Agree

TAS-20vf

Instructions : Indiquez, en utilisant la grille qui figure ci-dessous, à quel point vous êtes en accord ou en désaccord avec chacune des affirmations qui suivent. Il suffit de mettre une croix (X) à la place appropriée. Ne donnez qu'une réponse pour chaque assertion : (1) désaccord complet, (2) désaccord relatif, (3) ni accord ni désaccord, (4) accord relatif, (5) accord complet.

	Désaccord complet 1	Désaccord relatif 2	Ni accord ni désaccord 3	Accord relatif 4	Accord complet 5
1. Souvent, je ne vois pas très clair dans mes sentiments.	...	...	...	...	...
2. J'ai du mal à trouver les mots qui correspondent bien à mes sentiments.	...	...	...	...	...
3. J'éprouve des sensations physiques que les médecins eux-mêmes ne comprennent pas.	...	...	...	...	...
4. J'arrive facilement à décrire mes sentiments.	...	...	...	...	...
5. Je préfère analyser les problèmes plutôt que de me contenter de les décrire.	...	...	...	...	...
6. Quand je suis bouleversé(e), je ne sais pas si je suis triste, effrayé(e), ou en colère.	...	...	...	...	...
7. Je suis souvent intrigué(e) par des sensations au niveau de mon corps.	...	...	...	...	...
8. Je préfère simplement laisser les choses se produire plutôt que de comprendre pourquoi elles ont pris ce tour.	...	...	...	...	...
9. J'ai des sentiments que je ne suis guère capable d'identifier.	...	...	...	...	...
10. Etre conscient de ses émotions est essentiel.	...	...	...	...	...
11. Je trouve difficile de décrire mes sentiments sur les gens.	...	...	...	...	...
12. On me dit de décrire davantage ce que je ressens.	...	...	...	...	...
13. Je ne sais pas ce qui se passe à l'intérieur de moi.	...	...	...	...	...
14. Bien souvent, je ne sais pas pourquoi je suis en colère.	...	...	...	...	...
15. Je préfère parler aux gens de leurs activités quotidiennes plutôt que de leurs sentiments.	...	...	...	...	...
16. Je préfère regarder des émissions de variétés plutôt que des dramatiques.	...	...	...	...	...
17. Il m'est difficile de révéler mes sentiments intimes même à mes amis très proches.	...	...	...	...	...
18. Je peux me sentir proche de quelqu'un même pendant les moments de silence.	...	...	...	...	...
19. Je trouve utile d'analyser mes sentiments pour résoudre mes problèmes personnels.	...	...	...	...	...
20. Rechercher le sens caché des films ou des pièces de théâtre perturbe le plaisir qu'ils procurent.	...	...	...	...	...