

# Alexithymia, somatization and negative affect in a community sample

Phoebe E. Bailey, Julie D. Henry\*

*School of Psychology, University of New South Wales, Sydney, New South Wales 2052, Australia*

Received 20 December 2005; received in revised form 5 April 2006; accepted 23 May 2006

## Abstract

Whilst alexithymia has been consistently linked to somatization, two prominent theoretical models lead to opposite predictions as to which of two proposed typologies of alexithymia will be particularly associated with somatic symptom reporting. In the present study, participants were recruited from a cross-section of the general adult population ( $n=301$ ), and asked to complete a battery of self-report questionnaires which assessed alexithymia, somatization and negative affect. Collapsed across the whole sample, specific facets of alexithymia (enhanced fantasy life and difficulty identifying emotions) were significantly associated with somatization, and these pathways were perfectly mediated by negative affectivity. Further, type II alexithymia (impairment in the cognitive but not the affective dimension of alexithymia) was more predictive of somatization relative to type I alexithymia (impairment in both the cognitive and affective dimensions of alexithymia) and non-alexithymia (unimpaired in the cognitive and affective dimensions of alexithymia). The theoretical and clinical implications of these results are discussed.

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*Keywords:* Emotion; Depression; Anxiety; Somatic complaints

## 1. Introduction

Alexithymia is characterised by a reduction or absence of the tendency to engage in fantasizing and to consciously experience, describe and identify emotions, as well as an increased propensity toward externally oriented thinking (Nemiah and Sifneos, 1970). A prominent view is that these emotional deficits underlie failures in the capacity to recognize physical sensations as the somatic manifestations of emotions. Consequently, instead of dealing with the underlying emotion, these somatic sensations are focused on, resulting in somatosensory amplification, which may

then be misinterpreted as physical illness (Taylor et al., 1997, 1992).

Consistent with this possibility, a recent quantitative review which used Medline and PsycLIT searches to identify relevant studies, concluded that there is a small to moderate relationship between alexithymia and somatization (De Gucht and Heiser, 2003). However, prior studies which have investigated this relationship have almost exclusively used the Toronto Alexithymia Scale (TAS; Taylor et al., 1985) to index alexithymia. This is problematic because the TAS measures only three of the five putative characteristics of alexithymia: difficulty identifying emotions, difficulty describing emotions and externally oriented thinking. In addition to three subscales that correspond to the subscales of the TAS (identifying, verbalizing and analyzing, respectively), the more recently developed Bermond–Vorst Alexithymia Questionnaire

\*Corresponding author. Tel.: +61 2 9385 3936; fax: +61 2 9385 3641.

*E-mail address:* [julie.henry@unsw.edu.au](mailto:julie.henry@unsw.edu.au) (J.D. Henry).

(BVAQ; Vorst and Bermond, 2001) also includes measures of fantasizing (level of engagement in fantasies and day-dreams) and emotionalizing (capacity for emotional arousal). Vorst and Bermond argue that, whereas emotionalizing and fantasizing conjointly define the ‘affective’ dimension of alexithymia, identifying, verbalizing and analyzing together define the ‘cognitive’ dimension. Thus, it has been argued that, whilst the BVAQ is able to assess both the affective and cognitive dimensions of alexithymia, the TAS is only able to assess the latter (see Vorst and Bermond, 2001).

The aetiology of alexithymia remains unclear. Whilst some recent prospective studies support a social-developmental model (see Joukamaa et al., 2003; Lemche et al., 2004), according to Tabibnia and Zaidel (2004), at least three different neurological models have also been proposed. These identify (1) corpus callosum deficit (Miller, 1986; Parker et al., 1999, although for an alternate view, see Grabe et al., 2004), (2) right hemispheric deficit (Buchanan et al., 1980; Jessimer and Markham, 1997; Shipko, 1982) and (3) anterior cingulate dysfunction (Lane et al., 1997). In attempting to integrate this neurological research, Larsen et al. (2003) concluded that corpus callosum dysfunction is generally correlated with impairments in the cognitive but not the affective domain. This pattern of impairment (i.e., reduced emotional cognition with a sparing of the emotional experience) is referred to in the literature as type II alexithymia. In contrast, Larsen et al. argued that right hemispheric and frontal lobe deficits are generally correlated with impairments in both cognitive and affective characteristics, a deficit profile referred to as type I alexithymia. Larsen et al. further suggest that lesions to the corpus callosum are related to increased sympathetic activity and thus an increased tendency to experience and report somatic symptoms as a consequence of misinterpretation of the somatic sensations that accompany emotional arousal. In contrast, prefrontal lesions and right hemispheric deficits are associated with decreased physiological responses and, consequently, decreased risk of psychosomatic illness. Hence, Larsen et al.’s model clearly predicts that individuals presenting with type II alexithymia are more likely to somatize than are individuals presenting with type I alexithymia.

However, Lane and Schwartz’s (1987) levels of emotional awareness model asserts that the lowest levels of emotional awareness involve a focus on somatic sensations rather than consciously experienced emotions. Further, Lane et al. (1997) argue that transmission of interoceptive emotional information to the anterior cingulate cortex might permit completion of a feedback loop to modulate sympathetic arousal. Thus, failure of this transmission is argued to lead to exaggerated and persistent

sympathetic discharge and, consequently, somatization. Furthermore, the anterior cingulate cortex is located in the ventromedial frontal area of the brain (Larsen et al., 2003), which, as noted previously, has been particularly linked to type I alexithymia. Therefore, in the context of Lane and Schwartz’s (1987) model, type I (and not type II) alexithymia should be particularly related to somatization, which clearly contrasts with the predictions derived from Larsen et al.’s (2003) theoretical model. The first aim of the present study is to address which of these competing models is correct and, specifically, to assess whether type I, type II or non-alexithymia is more strongly associated with somatization.

Nevertheless, even if a strong relationship between type I and/or type II alexithymia and somatization can be demonstrated, the relationship may not be direct. In particular, it has been shown that alexithymia may represent a vulnerability factor which predisposes individuals to experience increased negative affect (NA), which in turn leads to medically unexplained symptoms (Lundh and Simonsson-Sarnecki, 2001). Thus, NA should be considered as a possible mediating factor when studying the relationship between alexithymia and somatization.

Whilst the externally oriented thinking subscale has consistently been found to not be significantly associated with NA (Modestin et al., 2004; Waller and Scheidt, 2004), some studies have found that difficulty identifying and describing emotions as indexed by the TAS are indirectly related to somatization via NA (Deary et al., 1997; Sayar et al., 2003). In contrast, De Gucht et al. (2004) found that, although alexithymia as indexed by Total TAS scores was not significantly associated with somatization once NA and positive affect were statistically controlled, further regression analyses of the separate subscales of the TAS revealed a significant direct relationship between somatization and the difficulty identifying emotions subscale. That is, as difficulty identifying emotions increased, level of somatization also increased independently of NA. Thus, there is conflicting evidence regarding the role of NA in the relationship between alexithymia and somatization. The second aim is therefore to assess whether specific facets of alexithymia are directly associated with somatization, or whether NA mediates any observed associations. This will be the first study which includes all five putative facets of alexithymia to conduct such an assessment.

## 2. Methods

### 2.1. Measures

Each potential participant received an introductory letter and a battery of counterbalanced self-report questionnaires,

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