The Spanish Journal of Psychology
2003, Vol. 6, No. 1, 51-59

1138-7416

Alexithymia – State or Trait?

Francisco Martínez-Sánchez, Manuel Ato-García, and Beatriz Ortiz-Soria
University of Murcia

Alexithymia refers to a specific disturbance in emotional processing that is manifested by difficulties in identifying and verbalizing feelings and a tendency to focus on and amplify the somatic sensations that accompany emotional arousal. Alexithymia is conceptualized both as an affect-deficit disorder and a continuous personality variable. The main purpose of the present study was to investigate the stability levels of alexithymia with regard to changes in emotional distress levels caused by university exams. We tested 20 university students at four different times, before and after the exams. Alexithymic features and self-reported emotional distress (trait anxiety and physical symptoms) were measured. Whereas emotional distress measures changed significantly during the diverse phases, the level of alexithymia remained unchanged. We therefore conclude that alexithymia represents a constant trait.
Keywords: emotion, alexithymia, stress, anxiety

La alexitimia describe un trastorno en el procesamiento emocional, manifestado mediante una marcada dificultad para identificar y expresar afectos, así como una tendencia a amplificar las sensaciones somáticas ligadas a la activación emocional. La alexitimia es conceptualizada tanto como un trastorno en la regulación afectiva, como una variable de personalidad. El principal objetivo de este trabajo es investigar la estabilidad temporal de los niveles de alexitimia en relación a los cambios experimentados en el malestar emocional causado por los exámenes universitarios. Evaluamos 20 universitarios en cuatro ocasiones diferentes, antes y después de los exámenes. Se evaluaron los niveles de alexitimia y de malestar emocional (ansiedad y sintomatología somática). Los resultados mostraron que mientras que las medidas de malestar emocional cambiaron significativamente durante las diversas fases, el grado de alexitimia permaneció inalterable. Se concluye afirmando que la alexitimia constituye un rasgo estable de personalidad.
Palabras clave: emoción, alexitimia, estrés, ansiedad

Correspondence concerning this article should be addressed to Francisco Martínez-Sánchez, Departamento de Psicología Básica y Metodología. Facultad de Psicología, Edificio Luis Vives, Universidad de Murcia, Apartado 4021, 30080 Murcia (Spain). E-Mail: franms@fcu.um.es

The term *alexithymia* (from Greek, literally, "lack of words for emotions"), coined by Sifneos in 1973, refers to a specific disturbance in affective-emotional processing, which has the following salient features: (a) difficulty in identifying and describing feelings and emotions verbally; (b) difficulty in distinguishing between feelings and somatic sensations that accompany emotional arousal; and (c) externally-oriented thinking and impaired symbolic activity (Taylor, 2000; Taylor, Bagby, & Parker, 1997). Recent research has pointed out that individuals with alexithymia not only have difficulty to express emotions verbally but also a deficit in the cognitive processing of emotions (Berenbaum & Prince, 1994; Jessimer & Markham, 1997; Martínez-Sánchez & Marín, 1997; Parker, Taylor, & Bagby 1993; Suslow, 1998). Consequently, emotions remain undifferentiated and poorly regulated (Taylor, Bagby, & Parker, 1991).

Alexithymia is conceptualized as a stable personality trait that correlates positively with neuroticism (Pandey & Mandal, 1996), depression (Hendryx, Haviland, & Shaw, 1991), and anxiety (Bagby, Taylor, & Atkinson, 1988). It is distributed normally and should be regarded as continuous and not discrete; it is not a disease state, strong empirical support revealing a stable personality trait, rather than just a consequence of psychological distress. Alexithymia is a unique personality construct, which is represented by a cluster of traits across the dimensions of the Five-Factor model of personality (Luminet, Bagby, Wagner, Taylor, & Parker, 1999). Some authors argue that alexithymia could also be considered as a resultant state of depression and/or anxiety, as well as the effect of some chronic psychopathological and somatic disorders (Hendryx, Haviland, Shaw, & Henry, 1994; Horton, Gewirtz, & Kreutter, 1992).

It has been hypothesized that this limited emotional awareness and cognitive processing of affects leads to prolonged and amplified physiological arousal and neurovegetative reactivity to stress, thus potentially disturbing the autonomic, pituitary-adrenal and immune systems (Infrasca, 1997; Martin & Pihl, 1986; Papciak, Feuerstein, & Spiegel, 1985). Dysregulation or heightened activation of the autonomic nervous system may explain the proneness to "functional" somatic disorders in individuals described as being alexithymic. In addition, alexithymia is regarded as one of several possible risk factors that seem to increase susceptibility to organic disease and to certain types of unhealthy behavior, and to a biased perception and reporting of somatic sensations and symptoms (Lumley, Stettner, & Wehmer, 1996; Lumley, Tomakowsky, & Torosian, 1997).

In this context, there is little information about the stability levels of alexithymia over time. Its variability margin as a function of situational contingencies also remains unknown, as well as the range of variance in responses to questionnaires of emotional and somatic symptoms.

Several reports (de Groot, Rodin, & Olmsted, 1995; Freyberger, 1977; Keltikangas-Järvinen, 1987; Wise, Mann, Mitchell, Hryvniak, & Hill, 1990) revealed the existence of discrete changes in the level of alexithymia contingent upon the improvement of associated somatic disorders. Patients who had suffered serious burns at least 10 months previously were more alexithymic than newly burned patients and control subjects (Fukunishi, Chishima, & Anze, 1994). The same pattern was observed in rape victims (Zeitlin, McNally, & Cassiday, 1993). Alexithymia increased in patients who had had spinal cord injuries for several years, but not in newly injured patients (Fukunishi, Koyama, & Tobimatsu, 1995).

However, there are also reports that have not observed any changes in the levels of alexithymia in response to variations in emotional or somatic distress levels (Cohen, Auld, & Brooker, 1994; Haviland, Shaw, Cummings, & MacMurray, 1988; Martínez-Sánchez, Ato, Córcoles, Huedo, & Selva, 1998; Porcelli, Leoci, Guerra, Taylor, & Bagby, 1996; Salminen, Saarijärvi, Äärelä, & Tamminen. 1994; Schmidt, Jiwany, & Treasure, 1993).

Studies on the stability of alexithymia are difficult to interpret. Lumley et al. (1996) argued that cross-sectorial studies of people with chronic diseases and alexithymia may be biased due to many factors, so that studies of people who have been raped or burned, or of long-term hemodialysis patients lack pretrauma measures, and alexithymia may have been increased in people at risk due to these negative life events; findings of a positive relationship between alexithymia and the time elapsed since the trauma may be biased, in that more alexithymic victims may be less likely to heal or recover. Moreover, reliability aspects concerning the different scales used by researchers in alexithymia levels might account for some of the longitudinal changes observed.

In a previous work (Martínez-Sánchez et al., 1998), a follow-up of 36 university students over a period of 17 weeks was carried out to evaluate several emotional variables (anxiety, depression, and somatic symptoms). The degree of alexithymia remained stable throughout this period, whereas the levels of emotional unease experienced significant changes contingent upon changes in the levels of academic stress.

The aim of the present study was to examine the changes in alexithymia levels as a consequence of variations in stress levels, in comparison with changes observed in other emotional and somatic variables. To this end, we used a complex quasi-experimental design composed of repeated measurements in two groups of participants. The recording sequence was as follows: first sequence, A-B, reduction, followed by increment of academic stress; and second sequence, B-A, increment, followed by reduction of academic stress. Our goal was to clarify the variations in alexithymia levels and, consequently, to contribute to the debate about whether alexithymia is a stable trait or a transient state that is secondary to psychological distress associated with stressful situations.

Method

Participants

Twenty undergraduate psychology students, aged between 18 and 23 years (M = 19.22, SD = 1.22) participated in this study. They were all randomly selected from students enrolled in the "Psychology of Emotion" course at the University of Murcia in 2001. The first ten students received the first recording sequence (A-B, reduction followed by increment of academic stress, evaluated between weeks 10 and 26 of the year 2001). The other ten students of the sample received the second sequence (B-A, increment followed by reduction, evaluated between weeks 5 and 14 of the year 2001). They were all given one academic credit in return for their cooperation.

We tested university students at four different times, before and after the exams. Exams can become a major source of stress in university students, especially when test scores serve as gatekeepers to future opportunities and career pathways. Exams, even if not a major source of stress, can contribute to everyday stressors or daily hassles. Research findings have indicated that these daily stressors are associated with health and adjustment problems, and that they can accumulate (Burks & Martin, 1985; Twisk, Snel, Kemper, & Van-Mechelen, 1999). Kiecolt-Glasser and Glasser (1988), for example, found that medical students showed lower levels of T-cells in the months immediately preceding their final exams than they did one month after the exams.

Materials

All participants were psychologically evaluated with the following instruments:

State-Trait Anxiety Inventory (STAI; Spielberger, Gorsuch, & Lushene, 1970). This scale was used to assess anxiety. Participants checked one out of four alternatives for each item to indicate how they "generally felt" for the Trait Anxiety Scale. Scores range from 20 to 80, with higher scores indicating higher trait anxiety. The State-Trait Anxiety Inventory is an extensively tested, documented, and validated generic measure of anxiety (see Spielberger & Rickman, 1991, for a review). Due to its longevity, ease, and longstanding track record, it has been used in over 8,000 studies. Internal consistency for the Spanish version of STAI (Spielberger, Gorsuch, & Lushene, 1988) is α = .87. Construct, concurrent, divergent, and convergent validity have been demonstrated with this questionnaire by means of correlations with different anxiety measures, such as the Taylor Manifest Anxiety Scale (TMAS; Taylor, 1953), Cattell's Trait and State Anxiety Measures (Cattell & Scheier, 1963), and the Affect Adjective Check List (AACL; Zuckerman & Lubin, 1965).

Pennebaker Inventory of Limbic Languidness (PILL; Pennebaker, 1982). This instrument is a commonly used symptom checklist of 54 common physical symptoms and bodily sensations (e.g., headaches, racing heart). Participants were asked to indicate how much each item had bothered or disturbed them during the previous two weeks, on a 5-point scale ranging from *not at all* to *extremely*. The PILL has high construct validity when compared with other measures of physical symptom self-reports. Construct validation studies have shown that individuals who score highly, compared with others, on the PILL, engage in more health-related behaviors, use aspirin more often, and report more autonomic changes in the laboratory setting. The Spanish version of the PILL (Martínez-Sánchez, in press) has high internal consistency (α = .84) and test-retest reliability (α = .73). The PILL scores show high positive correlations with self-report measures, commonly used as symptom check-lists, employed by *The Diagnostic and Statistical Manual of Mental Disorders* (1987, third edition, revised, American Psychiatric Association) for its Somatization Symptoms Screening-List, and with Attanasio, Andrasik, Blanchard, and Arena's (1984) SUNYA revision of the Psychosomatic Symptom Checklists, with Derogatis's (1977) Symptom Check-List, SCL-90-R, and with the MMPI Hypochondriasis Scale. These data validate the use of the PILL as a reliable, valid and sensitive assessment measure.

Toronto Alexithymia Scale (TAS-20; Bagby, Parker, & Taylor, 1994; Bagby, Taylor, & Parker, 1994). The TAS-20 is a 20-item self-report measure of alexithymia with a three-factor structure theoretically congruent with the alexithymia construct. Factor 1: difficulty in identifying feelings and distinguishing between feelings and somatic sensations of emotional arousal; Factor 2: difficulty in describing feelings to others; and Factor 3: externally-oriented thinking. A Spanish version of TAS-20 was redesigned by Spanish psychologists fluent in both English and Spanish, using back-translation methodology (Martínez-Sánchez, 1996; Páez et al., 1999). This version showed good internal consistency (Cronbach's α = .78) and test-retest reliability, r = .71, p < .001, over a 19-week period. These results are comparable to those obtained with the English version of the scale. The TAS-20 is currently the best-validated measure of the alexithymia construct and can be recommended for both clinical and research purposes. The construct validity of the TAS-20 has been investigated in several studies (see Taylor et al., 1997, for a review). This version showed that the TAS-20 and its three factors all correlated negatively with self-report measures of closely related constructs such as need cognition, attitudes towards emotional expression, social desirability, and self–consciousness, and they correlated significantly and positively with some relevant related constructs such as trait-anxiety and somatic complaints.

Procedure

All participants completed all the instruments during the two phases of the evaluation process: A-B (reduction phase followed by increment in the first group) or B-A (increment phase followed by reduction phase in the second group). All expected changes in the dependent variables used in this study for both phases of evaluation are parallel, as the periods of evaluation had similar durations.

A-B Sequence. Phase A (reduction of stress, weeks 10 to 13, 2001): Participants began the evaluation process the day after having concluded their February mid-term exams (week 10). All of the instruments were again completed on the following three Fridays (weeks 11, 12, and 13).

Phase B (increase of stress, weeks 23 to 26, 2001): Participants filled in the instruments the day before beginning their final exams (week 23). They also completed all the instruments on the following three Fridays (weeks 24, 25, and 26).

B-A Sequence. Phase B (increase of stress, weeks 5 to 8, 2001): Participants completed the tests the day before the beginning of the final exams period (week 5); all the instruments were again completed on the following three Fridays (weeks 6, 7, and 8).

Phase A (reduction of stress, weeks 11 to 14, 2001): Participants began the evaluation process the day after having concluded their February mid-term exams (week 11); they filled in all the instruments again during the following three Fridays (weeks 12, 13, and 14).

A longitudinal framework was used on each of the five dependent variables derived from questionnaire applications (physical symptoms, trait anxiety, total alexithymia, alexithymia factors 1, 2, and 3 for the two groups or sequences (A-B and B-A). In addition to students' age and sex, the following variables, focusing on the interpretation of the results of the study, were employed as covariates:

1. The particular observed recording sequence (SEQUENCE). That is, reduction followed by increment of academic stress (A-B) versus increment followed by reduction (B-A), which was assigned to the two groups. As these observations took place during different academic courses, care must be taken when interpreting results, to avoid confounding effects.

2. The observation period (PERIOD) in each sequence was defined as an ordinal variable (ranging from 1 to 4). This proxy variable was intended to evaluate the temporal trend of the observations.

3. The recording phase (PHASE) was defined as a dummy variable, reduction (0) versus increment (1) of academic stress in the first sequence and increment (1) versus reduction (0) of academic stress in the second one. This variable was intended to evaluate the global change between the two phases administered to each participant.

4. The interaction of period and phase (PHASE $\times$ PERIOD) was proposed to capture whether the temporal trend was similar in the first and second phases. If this interaction was significant, a different trend could be postulated for each phase; if nonsignificant, the same trend could be assumed for both phases of the study.

Thus, there would be four same-interval observational moments for each phase where marked emotional changes might take place. During the stress reduction moments, it was hypothesized that the emotional distress levels (trait anxiety and physical symptoms) would present a monotonically decreasing trend. Contrariwise, we predicted that the trend of all these variables would be reversed due to an increase of academic stress. However, the alexithymia levels would remain unaltered during all the phases of this study. Statistical analyses will allow us to confirm whether changes in stress levels, due to the presence or absence of examinations, yield concomitant changes in alexithymia levels (thus revealing its state feature) or, on the contrary, they remain similar (thus confirming that alexithymia is a stable trait).

Statistical Analyses

Statistical analysis of the data was carried out using an approach to longitudinal data based on an extension of quasi-likelihood for generalized linear models (McCullagh & Nelder, 1989), usually referred to as *generalized estimating equations* or GEE (Diggle, Lyang, & Zeger, 1994; Lipsitz, Kim, & Zhao, 1994; Zeger & Liang 1986). For comparison purposes, we also performed a mixed model ANOVA. All computations and graphical output was performed using *gee* and *nlme* packages on the Windows R platform (Ihaka & Gentleman, 1996).

With Gaussian normal data, the GEE approach is a simple extension of regression analysis for marginal models to take into account non-independence emerging from the repeated observations within each individual clustered observation. The GEE approach uses standard regression coefficient estimates with robust estimates of the standard errors, to account for the intraclass or within-cluster correlation between repeated measures. Ignoring within-cluster correlation usually leads to biased standard errors. The correction of estimated standard errors depends on the correlational structure assumed for clustered data. Zeger and Lyang (1986) proposed some common correlational structures in this regard:

1. Independence, which represents an identity matrix and assumes no correlation between repeated observations;

2. Exchangeable, which represents an association as a matrix with a constant off-diagonal element (the intraclass correlation). It is equivalent to the compound symmetry structure required, but is not commonly verified when using the univariate approach for repeated-measures ANOVA;

3. Auto-regressive, which considers association as a matrix with a decreasing correlation depending on the time lag between two repeated observations. It is a structural correlation, very common in time-series analysis;

4. Unstructured, a pattern obtained when no restrictions are imposed on the structure of association. This is the same correlation structure assumed in the multivariate approach for repeated-measures ANOVA.

In general, GEE obtains consistent estimates of marginal mean models, and robust statistical tests, even if the assumed correlation structure within observations of same individuals is mis-specified, but statistical tests will be most powerful when the working correlation matrix closely approximates to the true correlation matrix.

This procedure may be summarized, from a computational point of view, in the following steps: (a) Estimate model parameters for standard (naïve) regression coefficients assuming independence of observations; (b) remove the residuals from the model and use the model to estimate the working correlation matrix within observations of the same individual; (c) update the regression coefficients using the working correlation matrix obtained in step b; and (d) iterate until convergence.

A mixed model ANOVA was also performed, with all dependent variables taking Age and Period as covariates and Sex, Phase, and Sequence as binary factors. This approach is a repeated-measures analysis of variance with subjects as random effects. For comparison with GEE results, we assumed an independent (variance components) covariance structure.

Results

Figures 1 to 3 present smoothed conditioning plots for the sequence of observations in two phases registered in each participant. Lower quadrants correspond to the first sequence or group (A-B, reduction followed by increase of academic stress); upper quadrants correspond to second sequence or group (B-A); left quadrants correspond to the first phase; right quadrants, to the second phase.

Table 1 displays mean scores and standard deviations obtained in physical symptoms, trait anxiety, and alexithymia for the two sequences: reduction followed by increment of academic stress (A-B) versus increment followed by reduction (B-A).

For comparative purposes, GEE analysis was carried out using an independent/exchangeable correlation structure and order 1 autoregressive AR(1) correlation structure. The main results were very similar. Due to the nature of the design and observations, we focused the main interpretation on the independent/exchangeable structure.

A preliminary analysis revealed no significant differences due to sequence (group). We therefore discarded this variable for subsequent analysis. Table 2 displays a summary of

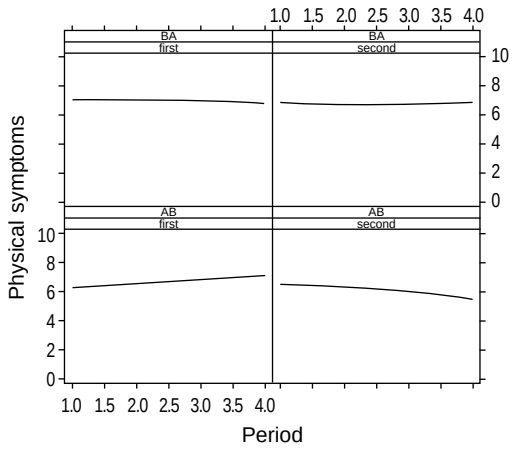

Figure 1. Smoothed conditioning plot of physical symptoms for all four periods observation. Each quadrant corresponds to a combination of sequence (A-B: reduction followed by increase; B-A: increase followed by reduction of academic stress) and observation phase (first or second).

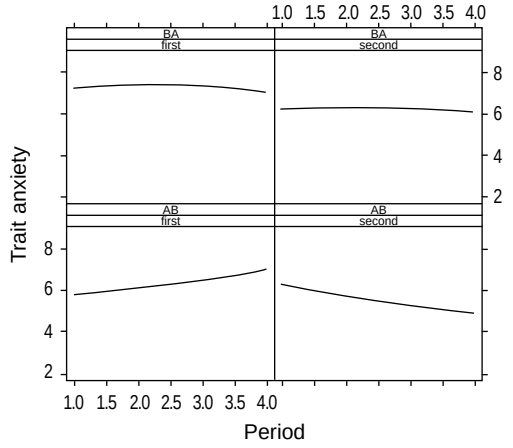

Figure 2. Smoothed conditioning plot of trait anxiety for all four observation periods. Each quadrant corresponds to a combination of sequence (A-B: reduction followed by increase; B-A: increase followed by reduction of academic stress) and observation phase (first or second).

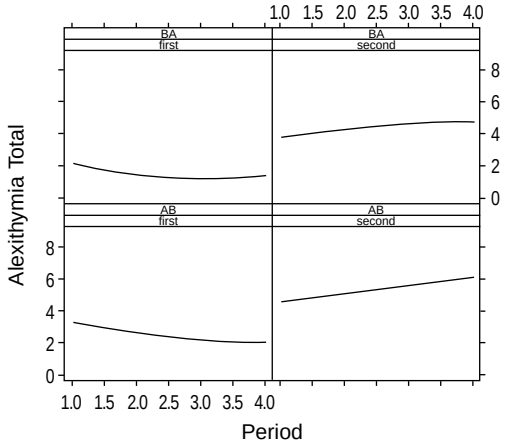

Figure 3. Smoothed conditioning plot of alexithymia total for all four observation periods. Each quadrant corresponds to a combination of sequence (A-B: reduction followed by increase; B-A: increase followed by reduction of academic stress) and observation phase (first or second).

Table 1
Mean Scores and Standard Deviations (in Brackets) Obtained in Physical Symptoms (PILL), Trait Anxiety (STAI), and Alexithymia (TAS-TOT) for the Two Groups or Sequences (A-B and B-A)

	First sequence (A-B)		Second sequence (B-A)	
Variable	Reduction of stress	Increase of stress	Increase of stress	Reduction of stres
PILL	25.30 (9.43)	29.30 (10.62)	28.20 (11.34)	22.90 (10.30)
STAI	38.72 (8.75)	47.11 (9.11)	49.76 (8.22)	40.25 (7.37)
TAS-TOT	50.57 (12.07)	51.90 (10.71)	52.04 (9.56)	49.34 (10.92)
TAS-F1	18.97 (5.89)	19.72 (6.96)	19.32 (6.69)	18.87 (6.38)
TAS-F2	13.80 (4.31)	14.10 (3.96)	14.28 (4.01)	13.36 (4.16)
TAS-F3	17.80 (4.76)	18.08 (4.78)	18.44 (4.78)	17.11 (4.21)
TAS-F3	17.80 (4.76)	18.08 (4.78)	18.44 (4.78)	17.11 (4.21)

Note. PILL = Pennebaker Inventory of Limbic Languidness; STAI = Stait-Trait Anxiety Inventory; TAS-TOT = Toronto Alexithymia Scale; TAS-F1 = Factor 1 (Difficulty in identifying feelings and distinguishing between feelings and somatic sensations); TAS-F2 = Factor 2 (Difficulty in describing feelings); TAS-F3 = Factor 3 (Externally-oriented thinking).

Table 2
Robust t-Test Assuming Independence/Exchangeable Correlation Structures

Variables	PILL	STAI	TAS-TOT	TAS-F1	TAS-F2	TAS-F3
AGE	–2.528	–0.474	0.845	0.623	0.748	–1.167
SEX	4.937****	0.095	–0.008	–0.100	1.354	1.499
PHASE	–2.269*	–2.993**	0.919	–1.435	–0.666	1.132
PERIOD	–1.472	–3.556***	–0.565	–1.895	–0.810	1.061
PHASE × PERIOD	–4.064****	4.556****	–0.543	1.438	0.664	–1.206

** p < .05. ** p < .005. *** p < .0005. ****p = .0000*

Note. PILL = Pennebaker Inventory of Limbic Languidness; STAI = Stait-Trait Anxiety Inventory; TAS-TOT = Toronto Alexithymia Scale; TAS-F1 = Factor 1 (Difficulty in identifying feelings and distinguishing between feelings and somatic sensations); TAS-F2 = Factor 2 (Difficulty in describing feelings); TAS-F3 = Factor 3 (Externally-oriented thinking).

Table 3
Type 3 F-Tests of Fixed Effects from a Mixed Model ANOVA

Variables	PILL	STAI	TAS-TOT	TAS-F1	TAS-F2	TAS-F3
AGE	4.12*	0.12	0.00	0.26	0.29	0.84
SEX	11.01**	0.01	0.55	0.01	1.34	0.75
PHASE	2.37	4.58*	0.06	3.23	1.14	2.16
PERIOD	0.01	0.46	0.00	0.03	0.07	0.29
PHASE × PERIOD	10.46**	16.15****	0.53	2.84	0.77	1.77

** p < .05. ** p < .005. *** p < .0005. ****p = .0000.* All tests assume 1 and 136 degrees of freedom.

Note. PILL = Pennebaker Inventory of Limbic Languidness; STAI = Stait-Trait Anxiety Inventory; TAS-TOT = Toronto Alexithymia Scale; TAS-F1 = Factor 1 (Difficulty in identifying feelings and distinguishing between feelings and somatic sensations); TAS-F2 = Factor 2 (Difficulty in describing feelings); TAS-F3 = Factor 3 (Externally-oriented thinking).

results of the effects of covariates (rows) on the dependent variables (columns), assuming an independent correlation structure, that is, assuming no relationship between repeated measures taken on each participant. All dependent variables related to alexithymia revealed nonsignificant results in all covariates, suggesting a longitudinal pattern composed of a flat Period and no change between Phases. The rest of the dependent variables had a different pattern. Physical symptoms had a significant effect on Age, p = .0115; Sex, p =.0000; Phase, p =.0233; and on the interaction Period × Phase, p =.0000, suggesting a model with a null general trend but with significant individual trends, depending on

Age and Sex for each Phase, and also significant changes between Phases. Trait anxiety had a significant effect on Phase, $p = .0028$, Period, $p = .0004$, and on the interaction Phase × Period, $p = .0000$, suggesting a different model as a function of trend and phase of data recording.

Similar results were obtained using a mixed model ANOVA. A preliminary analysis also revealed no significant effects due to sequence, and hence was discarded from the final analysis. Table 3 summarizes type 3 F-tests of fixed effects of mixed model. All dependent variables related to alexithymia revealed nonsignificant results in all factors and covariates. Physical symptoms and Trait anxiety showed similar patterns to those found with the GEE approach.

Discussion

The results of this study confirm that the degree of alexithymia is not influenced by the state effects of the level of academic stress. The absence of effects for Period and Phase × Period interactions suggests that alexithymia scores remain unchanged. All covariates related to alexithymia had nonsignificant effects on all dependent variables, suggesting a longitudinal pattern composed by a flat period and no change between phases.

The finding of the stability of alexithymic features is consistent with the results of several reports (Cohen et al., 1994; Haviland et al., 1988; Keller, Carroll, Nich, & Rounsaville, 1995; Martínez-Sánchez et al., 1998; Pinard, Negrete, Annable, & Audet, 1996; Porcelli et al., 1996; Posse, 2002; Salminen et al., 1994; Schmidt et al., 1993; Todarello et al., 1997).

Schmidt et al. (1993) used the Toronto Alexithymia Scale to investigate alexithymia in a group of female patients with eating disorder, before and after a 10-week interval of drug treatment. Although there was a significant improvement in their eating pathology after 10 weeks of treatment, there was no significant change in mean TAS scores. In another study that carried out a longitudinal follow-up of alexithymia levels in a group of psychiatric out-patients who showed clinical improvement, Salminen et al. (1994) reported that their alexithymia levels did not reveal meaningful changes over a year, despite the fact that other clinical evolution indexes varied significantly.

Further support for the viewpoint that alexithymia is not a reaction to emotional distress was provided by another 1-year follow-up study of psychiatric outpatients whose level of alexithymia remained consistent, while their psychological distress decreased significantly during the follow-up period (Cohen et al., 1994). This finding is consistent with a study of patients with alcohol dependence who showed a significant decrease in depression scores after successful treatment, whereas there was no concomitant decrease in their alexithymia scores (Haviland et al., 1988). Similar results were obtained by Porcelli et al. (1996) in a longitudinal study of a group of 104 medical outpatients suffering from inflammatory bowel disease. Whereas their anxiety and depression scores were influenced over time by changes in the degree of activity of their disease, there was no significant change in their mean alexithymia scores.

Similar results were also obtained in a recent work by Luminet, Bagby, and Taylor (2001) to study both absolute and relative stability of alexithymia in a sample of patients who entered a treatment program for major depression and who received antidepressant medication for 14 weeks. Their results indicate that, although alexithymia scores may change (indicating absolute changes) in the context of a marked reduction in depression, there is nevertheless strong evidence for the relative stability of alexithymia as revealed by hierarchical regression analyses. According to these authors, further indications for stability were provided by the magnitude of absolute change in alexithymia scores, which was small, in comparison to the magnitude of absolute change in depression severity.

In another recent study of a longitudinal one-year follow-up of alexithymia levels in a group of patients with major depression, Saarijärvi, Salminen, and Toikka (2001) reported that patients' levels of depression and distress were significantly lower at follow-up than at baseline, whereas the alexithymia scores did not change significantly during follow-up. Honkalampi et al., (2001), in a study of the prevalence of alexithymia and its relationship with depression during a one-year follow-up, also report that the mean values of the TAS-20 and its subfactors remained unchanged between the study phases; however, when using the original cut-off points, the authors found that some of the participants were classified in a different TAS-20 category at follow-up than at baseline. These authors state that, whereas alexithymia appears to be a stable trait, on the basis of the similarity of the mean TAS-20 scores in separate study phases, alexithymic features also appear to be state-dependent and strongly related to depressive symptoms. As Honkalampi, Hintikka, Saarinen, Lehtonen, and Viinamaeki (2000) assert, alexithymia does not appear to be a stable personality trait among depressed patients.

Our results are not in agreement with the findings of some other studies (i.e., Ahrens & Deffner, 1986) which suggest that, rather than being considered a personality trait, alexithymia should be regarded as a label for a set of coping behaviors that only occurs in specific situations.

The results of this study should be considered conservatively, concerning the generalization of the results, as sample sizes were small ($N = 20$). Future research in this area should be conducted using larger samples.

Lastly, our results are consistent with the consideration of alexithymia as an extreme point of a continuous personality dimension (Taylor et al., 1997) that has a normal distribution in the general population (Taylor & Bagby, 2000), rather than just a consequence of psychological distress or functional somatic symptoms.

References

Ahrens, S., & Deffner, G. (1986). Empirical study of alexithymia: Methodology and results. *American Journal of Psychotherapy, 40*, 430-447

American Psychiatric Association (9187). *Diagnostic and statistical manual of mental disorders* (3rd ed., revised). Washington, DC: Author.

Attanasio, V., Andrasik, F., Blanchard, E.B., & Arena, J.G. (1984). Psychometric properties of the SUNYA Revision of the Psychosomatic Symptom Checklist. *Journal of Behavior Medicine, 7*, 247-257.

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

Berenbaum, H., & Prince, J.D. (1994). Alexithymia and the interpretation of emotion-relevant information. *Cognition and Emotion, 8*, 231-244.

Burks, N., & Martin, B. (1985). Everyday problems and life change events: Ongoing versus acute sources of stress. *Journal of Human Stress, 11*, 27-35.

Cattell, R.B., & Scheier, I.H. (1963). *Handbook for the IPAT Anxiety Scale (2nd ed.)*. Champaign, IL: Institute for Personality and Ability Testing.

Cohen, K.R., Auld, F., & Brooker, H. (1994). Is alexithymia related to psychosomatic disorder and somatizing? *Journal of Psychosomatic Research, 38*, 119-127.

De Groot J.M., Rodin, G., & Olmsted, M.P. (1995). Alexithymia, depression, and treatment outcome in bulimia nervosa. *Comprehensive Psychiatry 36*, 53–60.

Derogatis, L.R. (1977). *SCL-90. Administration, scoring and procedures manual. Clinical psychometric research.* Baltimore, MD: Johns Hopkins University School of Medicine.

Diggle, P.J., Lyang, K.Y., & Zeger, S.L. (1994). *Analysis of longitudinal data*. Oxford, UK: Oxford University Press.

Freyberger, H. (1977). Supportive psychotherapeutic techniques in primary and secondary alexithymia. *Psychotherapy and Psychosomatics, 28*, 337-342.

Fukunishi, I., Chishima, Y., & Anze, M. (1994). Post-traumatic stress disorder and alexithymia in burn patients. *Psychological Report, 75*, 1371-1376.

Fukunishi, I., Koyama I., & Tobimatsu, H. (1995). Psychological acceptance and alexithymia in spinal cord injury patients. *Psychological Report, 76*, 475-481.

Haviland, M.G., Shaw, D.G., Cummings, M.A., & MacMurray, J.P. (1988). Alexithymia subscales and relationship to depression. *Psychotherapy and Psychosomatics, 50*, 164-170.

Hendryx, M.S., Haviland, M.G., & Shaw, D.G. (1991). Dimensions of alexithymia and their relationships to anxiety and depression. *Journal of Personality Assessment, 56*, 227-237.

Hendryx, M.S., Haviland, M.G., Shaw, D.G., & Henry J. (1994). Alexithymia in women and men hospitalized for psychoactive substance dependence. *Comprehensive Psychiatry, 35*, 124-128.

Honkalampi, K., Hintikka, J., Saarinen, P., Lehtonen, J., & Viinamaeki, H. (2000). Is alexithymia a permanent feature in depressed patients? Results from a 6-month follow-up study?. *Psychotherapy and Psychosomatics, 69*, 303-308.

Honkalampi, K., Koivumaa-Honkanen, H., Tanskanen, A., Hintikka, J., Lehtonen, J., & Viinamaeki, H. (2001). Why do alexithymic features appear to be stable? *Psychotherapy and Psychosomatics, 70*, 247-253.

Horton, P.C., Gewirtz, H., & Kreutter, K.J. (1992). Alexithymia-state and trait. *Psychotherapy and Psychosomatics, 58*, 91-96.

Ihaka, R., & Gentleman, R. (1996). R: A language for data analysis in graphics. *Journal of Computational and Graphical Statistics, 5*, 299-314.

Infrasca, R. (1997). Alexithymia, neurovegetative arousal and neuroticism. *Psychotherapy and Psychosomatics, 66*, 76-280.

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

Keller, D.D., Carroll, K.M., Nich, C., & Rounsaville, B.J. (1995). Alexithymia in cocaine abusers: Response to psychotherapy and pharmacotherapy. *American Journal of Addictions, 4*, 243-244.

Keltikangas-Järvinen, L. (1987). Concept of alexithymia II. The consistency of alexithymia. *Psychotherapy and Psychosomatics, 47*, 337-342.

Kiecolt-Glasser, J., & Glasser, R. (1988). Behavioral influences on immune function: Evidence for the interplay between stress and health. In T.M. Field (Ed.), *Stress and coping across development* (pp. 189-206). Hillsdale, NJ: Erlbaum.

Lipsitz, S.R., Kim, K., & Zhao, L. (1994). Analysis of repeated categorical data using generalized estimated equations. *Statistics in Medicine, 13*, 1149-1163.

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

Luminet, O., Bagby, R.M., Wagner, H., Taylor, G.J., & Parker, J.D.A. (1999). Relation between alexithymia and the Five-Factor Model of Personality: A facet-level analysis. *Journal of Personality Assessment, 73*, 345-359.

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

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

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

Martínez-Sánchez, F. (1996). Adaptación española de la Escala de Alexitimia de Toronto (TAS-20). *Clínica y Salud, 7*, 19-32.

Martínez-Sánchez, F. (in press). Adaptación española de la Inventario de Síntomas de Pennebaker, PILL. *Revista Española de Motivación y Emoción*

Martínez-Sánchez, F., Ato, M., Córcoles, E., Huedo, T., & Selva, J. (1998), Stability in the alexithymia levels: A longitudinal analysis of temporary series on various emotional answers. *Personality and Individual Differences, 24*, 767-772.

Martínez-Sánchez, F., & Marín, J. (1997). Influencia del nivel de alexitimia en el procesamiento de estímulos emocionales en una tarea Stroop. *Psicothema, 9*, 519-527.

McCullagh, P., & Nelder, J.A. (1989). *Generalized lineal models*. New York: Chapman & Hall.

Páez, D., Martínez-Sánchez, F., Velasco, C., Mayordomo, S., Fernández, I., & Blanco, A. (1999). Validez psicométrica de la Escala de Alexitimia de Toronto: un estudio transcultural. *Boletín de Psicología, 63*, 55-76.

Pandey, R., & Mandal, M.K. (1996). Eysenckian personality dimensions and alexithymia: Examining the overlap terms of perceived autonomic arousal. *Personality and Individual Differences, 20*, 499-504.

Papciak, A.S., Feuerstein, M., & Spiegel, J.A. (1985). Stress reactivity in alexithymia: Decoupling of physiological and cognitive responses. *Journal of Human Stress, 11*, 135-142.

Parker, J.D., Taylor, G.J., & Bagby, R.M. (1993). Alexithymia and processing of emotional stimuli: An experimental study. *New Trends in Experimental and Clinical Psychiatry, IX*, 9-14.

Pennebaker, J.W. (1982). *The psychology of physical symptoms*. New York: Springer-Verlag.

Pinard, L., Negrete, J.C., Annable, L., & Audet, N. (1996). Alexithymia in substance abusers: Persistence and correlates of variance. *American Journal of Addictions, 5*, 32-39.

Porcelli, P., Leoci, C., Guerra, V., Taylor, G.J., & Bagby, R.M. (1996). A longitudinal study of alexithymia and psychological distress in inflammatory bowel disease. *Journal of Psychosomatic Research, 41*, 569-573.

Posse, M. (2002). *Alexithymia: Background and consequences*. Unpublished doctoral dissertation. Karolinska Institutet, Sweden.

Saarijärvi, S., Salminen, J.K., & Toikka, T.B. (2001). Alexithymia and depression: A 1-year follow-up study in outpatients with major depression. *Journal of Psychosomatic Research, 51*, 729-733.

Salminen, J.K., Saarijärvi, S., Äärelä, E., & Tamminen, T. (1994). Alexithymia – state or trait? One-year follow-up study of general hospital psychiatric consultation out-patients. *Journal of Psychosomatic Research, 38*, 681-685.

Schmidt, U., Jiwany, A., & Treasure, J. (1993). A controlled study of alexithymia in eating disorders. *Comprehensive Psychiatry, 34*, 54-58.

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

Spielberger, C.D., Gorsuch, R.L., & Lushene, R.E. (1988). *Cuestionario de ansiedad estado-rasgo* (3rd ed.). Madrid: Publicaciones de Psicología Aplicada, TEA.

Spielberger, C.H., & Rickman, R.L. (1991). *Anxiety: Psychobiological and clinical perspectives*. Washington, DC: Hemisphere/Taylor & Francis.

Spielberger, C.S., Gorsuch, R.L., & Lushene, R.E. (1970). Manual for the State-Trait Anxiety Inventory. Palo Alto, CA: Consulting Psychologists Press.

Suslow, T. (1998). Alexithymia and automatic affective processing. *European Journal of Personality, 12*, 433-443.

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

Taylor, G.J., & Bagby, R.M. (2000). An overview of the alexithymia construct. In R. Bar-On & J.D.A. Parker (Eds.), *The handbook of emotional intelligence* (pp. 40-67). San Francisco, CA: Jossey-Bass.

Taylor, G.J., Bagby, R.M., & Parker J.D. (1991). The alexithymia construct. A potential paradigm for psychosomatic medicine. *Psychosomatics, 32*, 153-164.

Taylor, G.J., Bagby, R.M., & Parker, J.D. (1997). *Disorders of affect regulation*. Cambridge, UK: Cambridge University Press.

Taylor, J.A. (1953). A personality scale of manifest anxiety. *Journal of Abnormal and Social Psychology, 48*, 285-290.

Todarello, O., Casamassima, A., Daniele, S., Marinaccio, M., Fancinllo, F., Valentino, L., Tedesco, N., Wiesel, S., Simone, G., & Marinaccio, L. (1997). Alexithymia, immunity and cervical intraepithelial neoplasia: Replication. *Psychotherapy and Psychosomatics, 66*, 208-213.

Twisk, J., Snel, J., Kemper, H., & Van-Mechelen, W. (1999). Changes in daily hassles and life events and the relationship with coronary heart disease risk factors. *Journal of Psychosomatic Research, 46*, 229-240.

Wise, T.N., Mann, L.S., Mitchell, J.D., Hryvniak, M., & Hill, B. (1990). Secondary alexithymia: An empirical validation. *Comprehensive Psychiatry, 31*, 284-288.

Zeger, S.L., & Liang, K.Y. (1986). Longitudinal data analysis for discrete and continuous outcomes. *Biometrics, 42*, 121-130.

Zeitlin, S.B., McNally, R.J., & Cassiday, H.L. (1993). Alexithymia in victims of sexual assault: An effect of repeated traumatization? *American Journal of Psychiatry, 150*, 661-663.

Zuckerman, M., & Lubin, B. (1965). *Manual for the Multiple Affect Adjective Check List*. San Diego, CA: Educational and Industrial Testing Service.

Received June 6, 2002
Revision received November 21, 2002
Accepted December 12, 2002