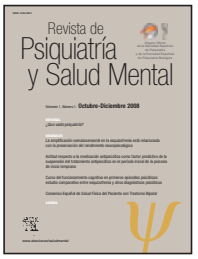
Rev Psiquiatr Salud Ment (Barc). 2008;1:3-9

Revista de Psiquiatría y Salud Mental

www.elsevier.es/saludmental



ORIGINAL ARTICLES

Somatosensory Amplif cation in Schizophrenia is Associated With Preserved Neuropsychological Function

Manuel Valdés,[a,b] Miguel Bernardo,[a,b,*] Nuria Segarra,[a] Gemma Parramón,[a] M. Teresa Plana,[a] Lorena Rami,[a] Manel Salamero,[a,b] and Nuria Bargalló[c]

[a]*Instituto Clínic de Neurociencias, IDIBAPS, Hospital Clínico Universitario, Barcelona, Spain*
[b]*Departamento de Psiquiatría y Psicobiología Clínica, Facultad de Medicina, Universidad de Barcelona, Barcelona, Spain*
[c]*Centro de Diagnóstico por la Imagen, Hospital Clínico Universitario, Barcelona, Spain*

Manuscript received August 4, 2008; accepted for publication September 16, 2008.

KEY WORDS
Alexithymia
Somatosensory amplif cation
Neuropsychological assessment
Schizophrenia

Abstract

Background: Some alexithymia features are similar to negative symptoms of schizophrenia, while somatosensory amplif cation has been def ned as an abnormal perceptual style that could be related to hallucinations and positive schizophrenic symptoms. This study analyzed whether both psychological constructs are related to the type of clinical symptoms and to the neuropsychological assessment of patients with schizophrenia.

Methods: Thirty-seven patients with schizophrenia were assessed psychologically (20 item Toronto Alexithymia Scale and Somatosensory Amplf cation Scale), clinically, and neuropsychologically and were compared with a control group of healthy subjects (n = 37).

Results: No differences were found in alexithymia or somatosensory ampli f cation between patients and controls and there was no relationship between the 2 psychological variables and the type of psychotic symptoms in the patient group. However, among patients with schizophrenia, somatosensory amplif cation was related to signif cantly better neuropsychological performance than that found in alexithymic subjects and in patients in general.

Conclusions: Alexithymia was unrelated to negative symptoms and to the neuropsychological performance of patients with schizophrenia, and as a psychological construct seems to be separate from the disease. However, in patients with an ampli f er style, neuropsychological performance was highly similar to that found in healthy controls, raising the possibility of studying somatosensory ampli f cation as a psychological variable that is potentially related to the clinical and neuropsychological outcomes of patients with schizophrenia.

*Corresponding author.
E-mail: mbernardo@clinic.ub.es (M. Bernardo).

PALABRAS CLAVE
Alexitimia.
Amplif cación somatosensorial.
Evaluación neuropsicológica.
Esquizofrenia.

La amplificación somatosensorial en la esquizofrenia está relacionada con la preservación del rendimiento neuropsicológico

Resumen
Ant ecedent es: Algunas características alexitímicas son parecidas a los síntomas negativos de la esquizofrenia, y la ampl f cación somatosensorial se ha def nido como un estilo perceptivo anormal que podría estar relacionado con las alucinaciones y los síntomas positivos de la esquizofrenia. Este estudio persigue aclarar si ambas construcciones psicológicas se relacionan con el tipo de síntomas clínicos y con el rendimiento neuropsicológico de los pacientes con esquizofrenia.
Mét odos: Un grupo de 37 pacientes afectados de esquizofrenia se evaluó desde el punto de vista psicológico (20 ítems; Toronto Alexithymia Scale y Escala de Amplif cación Somatosensorial), clínico y neuropsicológico, y se comparó con un grupo de control de sujetos sanos (n = 37).
Resul t ados: No hubo diferencias entre pacientes y controles en las puntuaciones de alexitimia y de ampli f cación somatosensorial, tampoco se encontró relación entre ambas variables psicológicas y el tipo de síntomas psicóticos en el grupo de pacientes. Sin embargo, en los pacientes con esquizofrenia las puntuaciones en ampli f cación somatosensorial se relacionaron con un rendimiento neuropsicológico mucho mejor que el presentado por los sujetos alexitímicos y los pacientes en general.
Conclusiones: La alexitimia no se relacionó con los síntomas negativos ni con el rendimiento neuropsicológico de los pacientes con esquizofrenia y pareció ser una construcción psicológica ajena a la enfermedad. En cambio, los pacientes con estilo somatosensorial ampli f cador presentaron un rendimiento neuropsicológico muy similar al de los controles sanos, lo cual permite pensar en la posibilidad de que la ampli f cación somatosensorial sea una variable psicológica potencialmente relacionada con la evolución clínica y neuropsicológica de los pacientes con esquizofrenia.

Introduction

There are tests that suggest that the abnormal establishment of neural connections in the course of neural development is associated with the clinical appearance of schizophrenia (expressed through psychotic symptoms)[1-6] and of a particular cognitive dysfunction that has been the subject of growing attention in recent years. The study of the perceptual apparatus of patients with schizophrenia has been focused on the analysis of its attributive style,[7,8] the establishment of cognitions in relation to the other patients, and on the way in which auditory hallucinations emerge from the language perception systems.[11-13] However, there are few studies dedicated to the role that other psychological variables may play, such as alexithymia (supposedly involved in emotional regulation) and somatosensory ampl f cation style, which describes a particular type of somatic stimuli and body states neuron integration.

Alexithymia has been def ned as a diff culty in identifying and describing feelings and a diff culty in distinguishing them from bodily sensations, in the context of poor imaginative activity and action oriented thinking,[14] and has been considered as a psychological risk factor for various psychosomatic disorders and some medical and psychiatric illnesses.[15,16] More recently, alexithymia has been conceptualized as a cerebral dysfunction that hinders the ability to experience conscious emotions during the autonomous activation of the emotional response.[17] The causes of this cerebral dysfunction are not yet clear, although 3 hypotheses have been proposed over the last few years: the f rst assumes the existence of a def cit in interhemispheric communication through the corpus callosum; the second suggests the presence of dysfunctions in the right cerebral hemisphere; and the third proposes the existence of dysfunctional mechanisms in the frontal cortex.[18] In some prior studies, it has been shown that patients with schizophrenia with a predominance of symptoms of def cits exhibit alexithymia scores that are signif cantly higher than the healthy controls, and alexithymia scores are positively correlated with f at affect and language impairment.[19,20]

Somatosensory amplif cation refers to the tendency to experience somatic sensations as intense, abnormal and harmful, the propensity to focus attention on infrequent bodily sensations and the disposition to react to bodily sensations with cognitions and emotional responses that intensify these sensations.[21] Therefore, it concerns, a particular style of integration of sensory stimuli that has been studied in patients with chronic pain,[22,23] and in patients affected with hypochondria and anxiety disorders.[24,25]

The study of alexithymia and somatosensory amplif cation makes sense in the context of research into the self-cognitive processes of patients with schizophrenia, and in the context of psychometric studies that attempt to identify neuropsychological correlations in the psychological vari-

ables. According to this focus, this study attempts to clarify whether alexithymia is related to the negative symptoms of schizophrenia (f at affect, poverty of speech, anhedonia, inhibition of emotional response, etc) and with poor neuropsychological function (in vocabulary and oral f uency); and, whether somatosensory amplif cation is related to hallucinations, and the positive symptoms of schizophrenia, and with less cognitive def cit. Somatosensory amplif cation has been shown to be very prevalent in patients with unexplained somatic symptoms, but there are no studies on its possible relationship to perceptual distortion and primary symptoms of schizophrenia.

Method

Sample

A group of 40 patients with paranoid schizophrenia, between the age of 15 and 35, were selected consecutively among patients being seen at the Schizophrenia Program of the Psychiatry Department of the Hospital Clinic Barcelona through a structured interview (Structured Clinical Interview for DSM-IV Axis I Disorders-Clinical Version [SCID-I]),[26] according to the criteria of the revised text of the 4th edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV-TR).[27] Patients with a medical history of brain injury convulsions, drug use, diabetes, hypertension, and systemic illness, or with other psychiatric diagnoses on axis I were excluded. All patients signed the informed consent before participating in the study, and 3 of them did not complete the protocol. Therefore, the f nal sample was composed of 37 patients, who maintained their antipsychotic treatment for the duration of the study.

A control group of healthy subjects (n= 37), paired by sex, age, and the socioeconomic level of their parents, was selected according to the same criteria, and were assessed with the same psychological and neuropsychological protocol.

Clinical Assessment

- Structured clinical interview, according to the criteria of DSM-IV-TR (SCID-I),[28] for establishing the psychiatric diagnosis
- Brief Psychiatric Rating Scale (BPRS-18),[29] consisting of 18 items that evaluate the type and severity of the psychiatric symptoms
- Positive and Negative Symptoms Scale (PANSS),[30] validated in Spain,[31] which assesses psychopathology present in schizophrenia patients
- Edinburgh Inventory[32] for assessing cerebral laterality

Psychological Variables

- Toronto Alexithymia Scale (TAS-20),[33] validated in Spain,[34] is a self-assessment instrument with 20 items, which the subject must answer based on a scale of 1 (strongly disagree) to 5 (strongly agree) points. Results are expressed in total score (TAS total) and in the scores of 3 subscales: difficulty identifying feelings and distinguishing them from bodily sensations (TAS-1), diff culty describing feelings (TAS-2), and externally oriented thinking (TAS-3). Subjects with more than 61 points are considered alexithymic
- Somatosensory Amplif cation Scale (SSAS),[35] validated in Spain,[36] is a self-assessment instrument that evaluates the sensitivity in perceiving bodily sensations that may be disagreeable, but that are not pathological. Subjects must express their degree of agreement or disagreement with the statements of the items, according to a scale from 1 ("strongly disagree") to 5 ("strongly agree")

Neuropsychological Assessment

- Auditory Verbal Learning Test (RAVLT)[37] for evaluating the encoding of new verbal information (short-term memory), and long-term retention (repetition of a list of words over 20 minutes)
- Wechsler Adult Intelligence Scale III (WAIS-III), in its Spanish version[38,39] for assessing selective attention (numbers reversed), working memory (sequence of numbers and letters, numbers reversed), and premorbid verbal intelligence (vocabulary)
- Trail Making Test,[40] which evaluates motor attention and speed (part A) and mental f exibility (part B)
- Wisconsin Card Sorting Test,[41] which measures the formation of concepts, verif cation of hypotheses, problem solving and thought f exibility
- FAS-Test,[42] which measures verbal f uidity and can be used as a supplementary indicator of frontal function

Statistical Analysis

For comparison between groups, the Student *t* test was used and, for categorical variables, the χ^2 test was used. To study the relationship between psychological, clinical, and neuropsychological variables, the Pearson correlation was used.

Results

There were no differences between patients and controls with regard to sociodemographic variables, psychological characteristics and cerebral dominance (Table). No healthy subject was graded as alexithymic and only 2 patients (5.3%) scored above 61 points on the TAS-20. Although patients had higher scores than the controls on the TAS-3 (externally oriented thinking) ($t = -1.790$; $P = .07$), there were no signif cant differences between the 2 groups in the total scores of their TAS.

Since alexithymia is a dimensional construct, patients and controls were divided into 2 groups (with 50 points approx. on the TAS-20), for the purpose of comparing them. There were no signif cant differences in the neuropsychological function among the controls with high scores (n = 7; average, 55.6 [2.6]) and low scores (n = 28; average, 30.5 [3.6])

Table 1. Demographic, Psychological, and Clinical Characteristics of Patients With Schizophrenia and Healthy Controls

P Schizophrenia (n	atients With = 37)	Healthy Controls (n = 37)	*P* Value
Demographics			
Age	28.68 (5.17)	28.17 (5.06)	
Sex			
Men	25 (67.5%)	24 (64.8%)	
W omen	12 (32.5%)	13 (35.2%)	
Educational level			
< 8 y	9 (24%)	10 (27%)	
8-12 y	16 (43.2%)	17 (45.9%)	
> 12 y	12 (32.5%)	10 (27%)	
Psychological			
TAS-20	39.63 (14.9)	34.30 (13.6)	
TAS-1	12.33 (7.41)	9.97 (6.79)	
TAS-2	11.28 (6.11)	10.88 (6.36)	
TAS-3	16.03 (5.83)	13.45 (6.11)	
SSAS	24.08 (6.40)	25.00 (5.81)	
Clinical			
Right-handred	33 (88.8%)	30 (83.7%)	
P resence of the illness	5.62 (2.3)		
BPRS	34.44 (8.52)		
P ANSS			
P	12.65 (5.15)		
N	18.53 (6.99)		
G	30.03 (8.14)		
Treatment[a]			
Atypical neuroleptic	[b] 1862.8 (753.2)		

[a]Measured in equivalent units of chlorpromacine/day.
[b]Risperidone: n = 16; quetiapine: n = 6; chlozapine: n = 6; aripiprazole: n = 6; quetiapine: n = 6; olanzapine: n = 5; ziprasidone: n = 2.

or among patients with high scores (n = 9; average, 58.5 [5.4]) and low scores (n = 27; average, 33.3 [2.7]) on the TAS-20.

Total alexithymia scores are not related to sociodemographic variables in either of the 2 groups, or to the psychiatric symptoms in the patient group. The TAS-1 scores are positively correlated to the somatosensory amplification points (SSAS) both in the control group ($r = 0.473$; $P = .005$) and in the patient group ($r = 0.433$; $P = .009$).

Performance on the RAVLT, the Trail Making Test, the numbers test, the WCST, and the FAS tests were significantly worse in patients with schizophrenia than in the controls, but are not related to the alexithymia scores in the latter. However, the TAS-3 (externally oriented thinking) was signif cantly correlated to form B of the Trail Making Test ($r = 0.386$; $P = .02$), ordering of letters and numbers ($r = -0.371$; $P = .008$), with the reverse numbers scores ($r = -0.434$; $P = .008$), total numbers ($r = -0.408$; $P = .01$), and vocabulary ($r = -0.322$; $P = .008$). Therefore, patients with externally oriented thinking tended to exhibit worse selective attention, worse performance in the higher functions, and less verbal intelligence. Scores on the somatosensory amplification (SSAS) were positively correlated with reversed numbers ($r = 0.322$; $P = .007$) and total numbers ($r = 0.322$; $P = .007$) in the patients, but were not related to sociodemographic variables, psychiatric symptoms, or performance on the other neuropsychological tests (RAVLT, TMT, ECST. and FAS), in which patients scored significantly less than the controls. Since somatosensory amplif cation is also considered a dimensional construct, the patients and controls were compared based upon their division into 2 groups according to a cut-off point of 23: patients with high scores on the SSAS (n = 20; average, 28.04 [4.1]) performed better on reversed numbers ($t = 2.492$; df 33; $P = .02$) and on total numbers ($t = 2.492$; df 33; $P = .01$) than the patients with low scores (n = 14; median, 18.14 [4.1]) and among the controls with high scores (n = 19; median, 29.05 [3.6]) and low scores (n = 14; median, 19.50 [2.8]) there were no signif cant differences in neuropsychological function.

Discussion

Patients with schizophrenia did not differ from the healthy controls in alexithymic characteristics (total TAS scores), but scored higher than the controls on the TAS-3 factor (externally oriented thinking), which describes a cognitive style more focused on the external details of ordinary life than on the content related to feelings, fantasy, and personal experiences susceptible to promoting emotional activation.[43,44] In patients with schizophrenia, TAS-3 scores were associated with a lower verbal capacity and poor performance of frontal functions (selective attention, working memory, and mental f exibility), so that it was the only factor of alexithymia that was related to neuropsychological function. This f nding only partially conf rms the results of a previous study with psychosomatic patients, [45] in which externally oriented thinking was signi f cantly related to a lower verbal intelligence, but not with frontal neuropsychological def cit. The presence of neuropsychological def cit in patients with schizophrenia, but not in the healthy controls, allows us to consider that frontal dysfunction associated with TAS-3 can depend on the effects of the illness more so than the presence of characteristics of alexithymia.

The total TAS scores in patients with schizophrenia were lower than those found in patients with chronic pain,[46,47] bulimia,[48] and functional dyspepsia[49] and are less than those communicated in other studies with patients with schizophrenia, using the same TAS version.[50-52] The low prevalence of characteristics of alexithymia in our sample of patients is difficult to explain but, as occurs in our study, in the research with the most chronic patients,[53,54] there are no rela-

tionships found between characteristics of alexithymia and psychotic symptoms, either positive or negative, which would suggest that alexithymia is a psychological construct independent of schizophrenia.

The scores on the somatosensory amplif cation (SSAS) in patients with schizophrenia are very similar to those found in other studies with psychiatric patients[55,56] and much less than those found in patients with chronic pain, [57] f bromyalgia,[58] and functional dyspepsia.[49] These data seem to indicate that somatosensory amplif cation is a perception style found more in patients affected by somatic illnesses than in psychiatric illnesses, although some studies have considered general amplif cation more as an index of distress and negative emotionality than as a valid index of somatic sensitivity.[59,60] These studies consider sensory amplif - cation as a "neurotic marker" due to its typical association with scores for neuroticism (N), although this association does not necessarily imply that somatosensory amplif cation is related to psychopathological manifestations. Furthermore, it must be expected that somatosensory amplif cation and neuroticism are related in some way, since neuroticism describes the tendency to respond with heightened emotional activation, and somatosensory amplif cation is a perceptual style that tends to increase the signals instigated by bodily changes. Consequently, somatosensory amplif cation and neuroticism may be functionally related in the general processing of bodily signals, but they have different relationships to psychopathological states and other psychological variables. In our study, SSAS scores were not related to psychiatric symptoms, either positive or negative, and patients with high scores on the SSAS did not show the general neuropsychological def cit found in the total group of patients. This means that patients with somatosensory amplif - cation style tend to exhibit better neuropsychological function than patients with alexithymia and psychiatric patients in general, independent from the nature of their clinical symptoms.

The relationships observed between alexithymia and the somatosensory amplif cation style are congruent with other prior studies[61-64] and with the main elements that define them as psychological constructs: SSAS scores were signif - cantly correlated to the interoceptive elements of emotional response (diff culty identifying feelings and distinguishing them from bodily sensations), but not with the most cognitive elements of alexithymia (externally oriented thinking). These results support the idea that somatosensory amplf cation is a perceptual style that can be functionally interrelated to cognitive capacity to identify and describe feelings.

In conclusion, alexithymia as a psychological construct is not more prevalent in the schizophrenic patients than in the healthy controls, and does not appear to be related to type of psychiatric symptoms or neuropsychological functioning of the patients. However, patients with schizophrenia tend to score higher on the TAS-3 (externally oriented thinking) and to exhibit lower verbal intelligence and worse performance in the frontal functions. The reason for this finding is not clear, especially if we consider that the TAS-3 has been def ned as a separate component of alexithymia, and it is the least trustworthy psychometric factor of alexithymia.[43,44]

In turn, somatosensory amplif cation is not related to psychotic symptoms either, and patients with high scores on the SSAS showed signif cantly better neuropsychological performance than alexithymia patients and, generally, all patients with schizophrenia. The f nding is interesting because somatosensory amplif cation appears to be associated with the preservation of signif cant cerebral functions and could be used as a psychological variable potentially related to clinical and neuropsychological functioning of patients with schizophrenia.

Finally, our future research will attempt to replicate these f ndings with a sample of patients large enough so that the recommended cut-off point for selecting subjects with alexithymia may be used (61 instead of 50) and it will be directed towards identifying possible structural and neurochemical substrates of somatosensory amplification in patients with schizophrenia.

Acknowledgments

This study has been f nanced by FIS N2003 F 5030494.

References

1. Weinberger DR. Implications of normal brain development for the pathogenesis of schizophrenia. Arch Gen Psychiatry. 1987; 44:660-9.
2. McGlashan TH, Hoffman RE. Schizophrenia as a disorder of developmentally reduced synaptic connectivity. Arch Gen Psychiatry. 2000;57:637-48.
3. McGlashan TH, Hoffman RE. Schizophrenia as a disorder of developmentally reduced synaptic connectivity. Arch Gen Psychiatry. 2000;57:637-48.
4. Crow TJ, Ball J, Bloom SR, Brown R, Bruton CJ, Colter N, et al. Schizophrenia as an anomaly of development of cerebral asymmetry. A postmortem study and a proposal concerning the genetic basis of the disease. Arch Gen Psychiatry . 1989;46: 1145-50.
5. Murray RM, Lewis SW . Is schizophrenia a neurodevelopmental disorder? Br Med J (Clin Res Ed). 1987;295(6600):681-2.
6. McGlashan TH, Hoffman RE. Schizophrenia as a disorder of developmentally reduced synaptic connectivity. Arch Gen Psychiatry. 2000;57:637-48.
7. Kinderman P, Bentall RP . Causal attributions in paranoia and depression: internal, personal, and situational attributions for negative events. J Abnorm Psychol. 1997;106:341-5.
8. Blackwood NJ, Howard RJ, Ffytche DH, Simmons A, Bentall RP, Murray RM. Imaging attentional and attributional bias: an fMRI approach to the paranoid delusion. Psychol Med. 2000;30: 873-83.
9. Frith CD, Corcoran R. Exploring 'theory of mind' in people with schizophrenia. Psychol Med. 1996;26:521-30.

10. Doody GA, Gotz M, Johnstone EC, Frith CD, Owens DG. Theory of mind and psychoses. Psychol Med. 1998;28:397-405.
11. Hoffman RE. New methods for studying hallucinated 'voices' in schizophrenia. Acta Psychiatr Scand Suppl. 1999;395:89-94.
12. Weiss AP, Heckers S. Neuroimaging of hallucinations: a review of the literature. Psychiatry Res. 1999;92:61-74.
13. Stephane M, Barton S, Boutros NN. Auditory verbal hallucinations and dysfunction of the neuronal substrates of speech. Schizophr Res. 2001;50:61-78.
14. Nemiah JCSPE. Affect and fantasy in patients with psychosomatic disorders. In: Hill OW, editor. Modern Trends in Psychosomatic Medicine. New York: Appleton-Century-Crofts; 2007. p. 430-9.
15. Larsen JK, Brand N, Bermond B, Hijman R. Cognitive and emotional characteristics of alexithymia: a review of neurobiological studies. J Psychosom Res. 2003;54:533-41.
16. Taylor GJ, Bagby RM, Parker JDA. Disorders of affect regulation: alexithymia in medical and psychiatric diseases. Cambridge: Cambridge University Press; 1997.
17. Lane RD, Sechrest L, Riedel R, Shapiro DE, Kaszniak AW. Pervasive emotion recognition def cit common to alexithymia and the repressive coping style. Psychosom Med. 2000;62:492-501.
18. Larsen JK, Brand N, Bermond B, Hijman R. Cognitive and emotional characteristics of alexithymia: a review of neurobiological studies. J Psychosom Res. 2003;54:533-41.
19. Nkam I, Langlois-Thery S, Dollfus S, Petit M. [Alexithymia in negative symptom and non-negative symptom schizophrenia]. Encephale. 1997;23:358-63.
20. Cedro A, Kokoszka A, Popiel A, Narkiewicz-Jodko W. Alexithymia in schizophrenia: an exploratory study. Psychological Reports. 2001;89:95-8.
21. Barsky AJ, Goodson JD, Lane RS, Cleary PD. The amplif cation of somatic symptoms. Psychosom Med. 1988;50:510-9.
22. Kosturek A, Gregory RJ, Sousou AJ, Trief P. Alexithymia and somatic amplif cation in chronic pain. Psychosomatics. 1998;39: 399-404.
23. Ak I, Sayar K, Yontem T. Alexithymia, somatosensory amplif cation and counter-dependency in patients with chronic pain. Pain Clinic. 2007;16:43-51.
24. Barsky AJ, Barnett MC, Cleary PD. Hypochondriasis and panic disorder. Boundary and overlap. Arch Gen Psychiatry. 1994;51: 918-25.
25. Martinez MP, Belloch A, Botella C. Somatosensory Amplif cation in hypochondriasis and panic disorder. Clin Psychol Psychother. 1999;6:46-53.
26. First MB, Spitzer RL, Gibbon M, Williams JBW. Structured Clinical Interview for DSM-IV Axis I Disorders-Clinical Version. Washington: Am Psychiat Press; 1997.
27. American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders. 4th edition text Rised (DSM-IV-TR). Washington: American Psychiatric Association; 2000.
28. First MB, Spitzer RL, Gibbon M, Williams JBW. Structured Clinical Interview for DSM-IV Axis I Disorders-Clinical Version. Washington: Am Psychiat Press; 2007.
29. Overall JE, Gorham DR. The Brief Psychiatric Rating Scale. Psychol Rev. 2007;10:799-812.
30. Kay SR, Fiszbein A, Opler LA. The positive and negative syndrome scale (PANSS) for schizophrenia. Schizophr Bull. 1987;13: 261-76.
31. Peralta V, Cuesta MJ. Psychometric properties of the positive and negative syndrome scale (PANSS) in schizophrenia. Psychiatry Res. 1994;53:31-40.
32. Old f eld RC. The assessment and analysis of handedness: the Edinburgh inventory. Neuropsychologia. 1971;9:97-113.
33. Taylor GJ, Ryan D, Bagby RM.Toward the development of a new self-report alexithymia scale. Psychother Psychosom. 1985;44: 191-9.
34. Rodrigo G, Lusiardo M, Normey L. Alexithymia — Reliability and Validity of the Spanish Version of the Toronto Alexithymia Scale. Psychother Psychosom. 1989;51:162-8.
35. Barsky AJ, Wyshak G, Klerman GL. The somatosensory amplif cation scale and its relationship to hypochondriasis. J Psychiatr Res. 1990;24:323-34.
36. Martinez MP, Belloch A, Botella C. Somatosensory Amplif cation in hypochondriasis and panic disorder. Clin Psychol Psychother. 1999;6:46-53.
37. Rey A. L'Examen Clinique en Psychologie. Paris: Press University; 2007.
38. Wechsler D. Escala de inteligencia de Wechsler para adultos-WAIS III, Spanish version. Madrid: TEA Ediciones; 1999.
39. Wechsler D. Escala de inteligencia de Wechsler para adultos-WAIS III, Spanish version. Madrid: TEA Ediciones; 2007.
40. TMT, Army Individual Test Batery. Manual of Directions and Scoring. Washington: War Department, Adjuntant General's Off ce, 1944.
41. WCST, Heaton R. A Manual for the Wisconsin Card Sorting Test. Odessa: Psychological Assessment Resources; 1981.
42. Borkowsk JG, Benton AL, Spreen O. Word Fluency and Brain Damage. Neuropsychologia. 1967;5:135.
43. Kooiman CG, Spinhoven P, Trijsburg RW. The assessment of alexithymia: a critical review of the literature and a psychometric study of the Toronto Alexithymia Scale-20. J Psychosom Res. 2002;53:1083-90.
44. Taylor GJ, Bagby M, Luminet O. Assessment of alexithymia: self-report and observer-rated measures. En: Parker JDA, Bar-On R, editors. The Handbook of Emotional Intelligence. San Francisco: J. Bass; 2000. p. 301-19.
45. Valdes M, Jodar I, Ojuel J, Sureda B. [Alexithymia and verbal intelligence: a psychometric study]. Actas Esp Psiquiatr. 2001; 29:338-42.
46. Kosturek A, Gregory RJ, Sousou AJ, Trief P. Alexithymia and somatic amplif cation in chronic pain. Psychosomatics. 1998;39: 399-404.
47. Ak I, Sayar K, Yontem T. Alexithymia, somatosensory amplif cation and counter-dependency in patients with chronic pain. Pain Clinic. 2007;16:43-51.
48. Sureda B, Valdes M, Jodar T, de Pablo J. Alexithymia, type A behaviour and bulimia nervosa. European Eating Disorders Review. 1999;7:286-92.
49. Jones MP, Schettler A, Olden K, Crowell MD. Alexithymia and somatosensory amplif cation in functional dyspepsia. Psychosomatics. 2004;45:508-16.
50. Maggini C, Raballo A. Alexithymia and schizophrenic psychopathology. Acta Biomed. 2004;75:40-9.
51. Nkam I, Langlois-Thery A, Popiel A, Narkiewicz-Jodko W. Alexithymia in schizophrenia: an exploratory study. Psychol Rep. 2007;89:95-8.
52. Todarello O, Porcelli P, Grilletti F, Bellomo A. Is alexithymia related to negative symptoms of schizophrenia? A preliminary longitudinal study. Psychopathology. 2005;38:310-4.
53. Maggini C, Raballo A. Alexithymia and schizophrenic psychopathology. Acta Biomed. 2004;75:40-9.
54. Todarello O, Porcelli P, Grilletti F, Bellomo A. Is alexithymia related to negative symptoms of schizophrenia? A preliminary longitudinal study. Psychopathology. 2005;38:310-4.
55. Martinez MP, Belloch A, Botella C. Somatosensory Amplif cation in hypochondriasis and panic disorder. Clin Psychol Psychother. 1999;6:46-53.
56. Wise TN, Mann LS. The relationship between somatosensory amplif cation, alexithymia, and neuroticism. J Psychosom Res. 1994;38:515-21.
57. Ak I, Sayar K, Yontem T. Alexithymia, somatosensory amplif cation and counter-dependency in patients with chronic pain. Pain Clinic. 2007;16:43-51.

58. Sayar K, Barsky AJ, Gulec H. Does somatosensory amplif cation decrease with antidepressant treatment? Psychosomatics. 2005; 46:340-4.
59. Wise TN, Mann LS. The relationship between somatosensory amplif cation, alexithymia, and neuroticism. J Psychosom Res. 1994;38:515-21.
60. Aronson KR, Barrett LF, Quigley KS. Feeling your body or feeling badly: evidence for the limited validity of the Somatosensory Amplif cation Scale as an index of somatic sensitivity J Psychosom Res. 2001;51:387-94.
61. Ak I, Sayar K, Yontem T. Alexithymia, somatosensory amplif cation and counter-dependency in patients with chronic pain. Pain Clinic. 2004;16:43-51.
62. K osturek A, Gregory RJ, Sousou AJ, Trief P. Alexithymia and somatic amplif cation in chronic pain. Psychosomatics. 1998;39:399-404.
63. Jones MP, Schettler A, Olden K, Crowell MD. Alexithymia and somatosensory amplif cation in functional dyspepsia. Psychosomatics. 2004;45:508-16.
64. Wise TN, Mann LS. The relationship between somatosensory amplif cation, alexithymia, and neuroticism. J Psychosom Res. 1994;38:515-21