



Original/Otros

## Heightened sensitivity to somatosensory stimuli in anorexia nervosa: an exploratory study with the SASTCA scale

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### Abstract

**Objectives:** To analyse the presence of heightened sensory sensitivity in patients with anorexia nervosa, which seems similar but not identical to that described in patients with unexplained somatic symptoms or body dysmorphic disorder.

**Methods:** We developed a sensory sensitivity scale in eating disorders (SASTCA), which measures the intensity of the response to specific somatosensory stimuli. The scale was completed by 48 patients with anorexia and a control group of 31 participants matched in age, sex and social and educational level. The results were compared with those obtained with the Barsky Somatosensory Amplification Scale (SSAS).

**Results:** The reliability (Cronbach's/alpha, 0.946; Guttman/split-half, 0.936) and validity (ROC, 0.933) of the SASTCA scale are indicative of its high sensitivity and specificity. The anorexia group had a significantly higher mean score on the SASTCA scale than the control group ( $p<.001$ ). Similarly, the patients with anorexia had a significantly higher mean value on the SSAS than the participants in the control group ( $p<.01$ ), although the difference was less extreme. The 2 scales correlated positively ( $r=.634$ ).

**Discussion:** These preliminary results suggest the presence in Anorexia of heightened sensory sensitivity which differs from the sensitivity of the control group. This sensitivity has a significant relationship with that described in patients with somatic complaints about health (SSD) or appearance (BDD). Could this heightened sensory sensitivity help us to explain the process of forming the distorted body self-concept (I'm fat, sick, ugly) in all these patients? Once its presence has been confirmed in other patients with anorexia, their relatives and other patients with somatic disorders this heightened sensitivity could constitute the somatic endophenotype of anorexia?

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Key words: Anorexia. Body Image. Heightened Sensory Sensitivity. Somatic Disorders. Dysmorphic Disorder.

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### ELEVADA SENSIBILIDAD A ESTÍMULOS SOMATOSENSORIALES EN ANOREXIA NERVOSA: UN ESTUDIO PRELIMINAR CON LA ESCALA SASTCA

### Resumen

**Objetivo:** destacar la presencia de una sensibilidad extrema hacia estímulos externos e internos (Amplificación Somatosensorial) en pacientes con Anorexia, similar pero no idéntica, a la descrita en pacientes con unexple somatic symptoms.

**Método:** Se ha elaborado una escala de Amplificación Somatosensorial para Trastornos de la Conducta Alimentaria, (SASTCA), que mide la intensidad de la respuesta hacia estímulos somatosensoriales específicos. La escala ha sido cumplimentada por un grupo de 48 pacientes anoréxicas y un grupo control de 31 sujetos emparejados en edad, sexo y nivel socioeducativo. Los resultados se han comparado con los obtenidos en la escala SSAS de Barsky.

**Resultados:** La Fiabilidad (Alfa de Cronbach 0,946; dos mitades de Guttman 0,936) y la Validez (ROC, 0,933), son indicativas de elevada sensibilidad y especificidad de la escala SASTCA. El grupo de pacientes presenta una media 58,73 12,38, significativamente superior al grupo control 37,81 7,47, ( $\alpha=0,001$ ). Las pacientes presentan en la escala SSAS una media 31,21 6,68 significativamente superior al grupo control 26,58 5,49 ( $\alpha=0,01$ ), aunque la diferencia es menos extremada. Ambas escalas correlacionan positivamente. 0,634

**Conclusiones:** Los resultados sugieren la presencia en AN. de una sensibilidad extrema hacia estímulos somatosensoriales. ¿Podría esta elevada sensibilidad sensorial ayudarnos a explicar el proceso de formación del auto-concepto distorsionado ("gordo, enfermo, feo") de estos pacientes? De confirmar su presencia en otras muestras de pacientes con anorexia, en sus familiares y en diferentes pacientes somatomorfos o TCA, esta elevada sensibilidad podría considerarse el endofenotipo somatomorfo del trastorno anoréxico.

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Palabras clave: Anorexia. Imagen Corporal. Elevada Sensibilidad Somatosensorial. Trastorno Somatomorfo. Trastorno Dismórfico.

## Introduction

Current studies into anorexia nervosa are attempting to determine which endophenotypes and biomarkers can cause vulnerability to the development of anorexia nervosa (AN). The cognitive, emotional and behavioural symptoms are considered to be the result of interactions among biologically determined factors, as well as neurocognitive and emotional dysfunctions expressed as abnormalities in the operation of brain neural circuits, societal and familial factors, and precipitating stressors. The importance of this understanding lies in the fact that these endophenotypes precede the onset of the disease, which helps with designing earlier treatments that are more effective and better adjusted to each patient<sup>1</sup>.

The most important contribution has emerged from the study of neurocognitive functioning. Cognitive rigidity (difficulty in set shifting) in patients meets the requirements of an endophenotype: it is present in the acute stage, it remains after eating recovery and it appears in relatives without the disorder. The executive dysfunction can be found in other mental diseases (obsessive-compulsive disorder, bipolar disorder and schizophrenia)<sup>2</sup> and has been associated with a specific cerebral dysfunction. Neuroimaging studies of patients with cognitive inflexibility (obsessive-compulsive disorder, anorexia, and autism spectrum disorder) show a low activation of the ventral cortico-striato-thalamo-cortical pathway during the execution of set-shifting tasks. The other neurocognitive deficiency observed in anorexia, the difficulty in overall visuospatial processing (weak central coherence) and an exceptional processing of details is also a characteristic of patients with autism spectrum disorder and is therefore also considered a nonspecific endophenotype for the development of anorexia nervosa<sup>3,4</sup>. The nonspecificity of the two endophenotypes has not prevented the development of new therapeutic interventions such as cognitive remediation therapy, which supplement current treatments and boost their positive result, reducing the risk of chronicity in this disease<sup>5</sup>.

Another important therapeutic contribution resulted from the study of the difficulty in recognising emotions (alexithymia) and self-regulating them, as observed in patients with eating disorders<sup>6</sup>. Pathological eating behaviours appear to act as an avoidance of negative emotions or as method of regulating them. The course of the disorder (recovery vs. chronicity) has been associated with the severity of the emotional difficulties<sup>7,8</sup>. These results have encouraged new causality models for this disease, with their subsequent therapeutic implications, and the search for the potential underlying emotional endophenotype<sup>9,10</sup>. In the clinical area, therapists are urged to incorporate emotional treatment techniques for their patients to increase the perception of emotions and to teach how to properly regulate them. Current research attempts to define the emotional endophenotype of ED based on the study of

heightened sensitivity to punishment and the response to reinforcement<sup>11,12</sup>.

Our research seeks to contribute to the search for potential endophenotypes in anorexia and focuses on body image distortion<sup>13,14</sup>. Our hypothesis is that patients with anorexia (and possibly patients with other ED) have a heightened sensory sensitivity that could be involved in the formation of the patients' distorted body self-concept ("I'm fat"), which precedes the onset of pathological eating behaviours. The decision to focus the study on this possible heightened body sensitivity is supported by several results from clinical observation and research on anxiety, perceptual bias and distortion in patients who experience SSD or BDD<sup>15,16,17,18</sup>. Thus, clinical observation has allowed us to observe certain similarities between the somatic, cognitive and emotional function of patients with SSD and BDD and that of patients with anorexia, which might shed some light on the understanding of the formation of body image distortion<sup>19,20</sup>. At the start of the disease, it is not uncommon to observe some adolescent patients visiting the doctor with gastrointestinal complaints, which result in numerous medical tests before being diagnosed with an eating disorder. Too often, these complaints are interpreted as a manipulation by the patient to justify rejecting food, without pausing to consider that perhaps the majority of these patients really were experiencing discomfort that we were unable to explain. Similarly, during the process of recovery, many patients go through a period of health concerns which can occasionally lead to agoraphobia. Moreover, the perceptive and cognitive function described in patients with unexplained somatic complaints about health or body shape (amplifying somatic style)<sup>21</sup>, appears to be present in patients with anorexia nervosa. This function is characterised by considerable concern and anxiety about the body, heightened sensitivity to somato/sensory stimuli, cognitive bias (focusing of attention) towards subtle physical perceptions, catastrophic interpretations of physical perception as a possible sign of disease (SSD) or deformity (BDD), avoidance behaviour, memory biases and increased expectations of symptom onset<sup>22,23</sup>. Authors have studied these characteristics and have associated them with individuals who have undergone stressful body experiences and who have a limited awareness of their emotional states<sup>24</sup>.

The similarity to patients with anorexia nervosa is somewhat surprising. These patients report undergoing stressful experiences with their body image before developing the disorder. The most common experiences are those involving teasing by school-mates and the most traumatic are related to sexual abuse. These patients demonstrate a limited awareness of their emotional states. They manifest continuous anxiety concerning the possibility of becoming fat. They interpret certain physical sensations in an alarmist manner, mistaking them for sign of fatness. One of the most

common misinterpretations is when patients attribute fatness to the feeling of discomfort produced by per- tinacious constipation experienced during the peak of anorexia and during its relapse. Patients present a cog- nitive bias towards stimuli associated with fatness and caloric food, which is processed as a matter of pri- ority<sup>25,26,27</sup>. This bias is present in their emotional me- mory<sup>28,29</sup>. Their pathological behaviour is considered fatness avoidance behaviour.

With so many similarities, it seems plausible that they would also have heightened sensory sensitivity towards internal and external body stimuli. If heigh- tened sensitivity is confirmed to be present in patients with anorexia, could this help us explain the process of forming the distorted body image in all these patients?

## Methodology

### Objectives

The aim of this study was to highlight the presen- ce of heightened sensory sensitivity in patients with anorexia, which seems similar but not identical to that described in patients with other unexplained soma- tic disorders. This heightened sensory sensitivity is a potential vulnerability factor for the development of anorexia?

### Hypothesis

Patients diagnosed with Anorexia Nervosa have sig- nificantly greater heightened sensory sensitivity than control participants without eating disorders.

### Design

Analytical, cross-sectional, case-control study.

### Participants

We selected 48 patients diagnosed with anorexia nervosa (restrictive and purging) or eating disorder not otherwise specified (EDNOS) with anorexic profile. The patients met the criteria according to the Diag- nostic and Statistical Manual of Mental Disorders, 4th Edition, Text Revision (DSM-IV-TR) (42) and were referred to the Unit for Eating Disorders (UED) of Hospital La Paz of Madrid. We excluded patients with other ED diagnoses (bulimia nervosa, EDNOS with bulimic profile and binge eating disorder) and patients with physical or mental disease (diabetes, drug and al- cohol consumption) associated with ED. To obtain a control group of similar age, sex and social and edu- cational level, we asked each patient to ask someone

in their community (classmate, workmate or neigh- bour) to participate in the study. This control group was composed of 31 participants who met the same inclusion and exclusion criteria as the selected cases but who did not present any diagnostic criterion for ED according to the DSM-IV. The control group had no family relationship to the patients.

All participants were informed about the study, and the team invited them to participate in the study to contribute to the understanding of the disorder and to obtain some type of positive conclusion for impro- ving treatment. Once they were informed about the study and gave their consent, the participants signed the informed consent approved by the hospital ethics committee. The data were recorded starting the third week of admission to the UED within the general as- sessment.

### Instruments

**SASTCA Scale:** To assess the presence of heigh- tened sensory sensitivity in anorexia, we developed a somatosensory scale for patients with eating disorders, the SASTCA scale. The 3 first letters (SAS) refer to the Barsky SSAS scale and attempt to recognise the author's pioneering work in the description of heigh- tened sensitivity of patients with hypochondria. The remaining 3 letters (TCA) stand for eating disorders. The scale was constructed based on the observations collected in the clinical setting. The first version of the questionnaire was designed with 26 questions, which was reduced to 18 after eliminating redundant ques- tions. The items included bodily sensations and reac- tions to stimuli from the various body senses (smell, pressure, texture, sound, vision). These items were estimated on a 5-point scale. The possible scores ranged from 18 to 90. (Appendix A).

**SSAS Scale:** In order to compare the specificity of the heightened sensory sensitivity of patients with ano- rexia, we used the Barsky somatosensory amplification scale (SSAS) used in patients with hypochondria<sup>30</sup>. We used the Spanish version<sup>31</sup>. This scale consists of 10 items that include questions about benign bodily sen- sations that are bothersome but do not constitute typi- cal symptoms of disease. These items were estimated on a 5-point scale. The possible scores ranged from 10 to 55.

### Data analysis

The data analysis was performed using the SPSS 12. 0 program. All hypothesis tests were considered bilate- ral, and values were considered statistically significant when  $p < .05$ . Before assessing the potential differen- ces in somatosensory sensitivity between the patient group and the control group, we studied the psycho- metric characteristics of the SASTCA scale: reliabili-

ty, validity, sensitivity, specificity and positive and negative predictive value. After determining the scale's psychometric characteristics, we compared the results of the 2 groups (patients and controls) in both tests. The scale's reliability was studied using the statistical Cronbach's alpha and the Guttman split-half test. The scale's validity was studied using the calculation of the area under the receiver operating characteristic (ROC) curve, along with its 95% confidence interval. Based on the ROC curve, we tested various cut-off points, selecting the one with the greatest performance for the measures of sensitivity, specificity and predictive values. The chosen cut-off point was 49. These values were compared with the predictive performance of the SSAS (sensitivity, specificity and positive and negative predictive value) for anorexia. The chosen cut-off point (27) was that recommended for Spanish samples. The quantitative correlation between the 2 questionnaires was analysed using the Pearson correlation coefficient.

## Results

The sample consisted of 79 participants, of whom 48 (60.75%) were patients and 31 (39.25%) were controls; 88.6% were women and 11.4% were men. The ED group consisted of 89.59% women and 10.41% men. The control group was composed of 86.097% women and 12.903% men. The proportion of men to women in the patient and control groups was not significantly different ( $p > .05$ ). The mean ages of the patient and control groups were mean 29.26, (SD 7.8) years and mean 27.88 (SD 8.12) years, respectively. The difference in age between the groups was not significant ( $p > .05$ ). Body mass index (BMI): At the time of the study, the patient group diagnosed with Anorexia Nervosa or EDNOS of type AN had a mean BMI of 17.2 (range 11.2–20.4), although they had much lower values over the course of their disorder. Duration of the disorder: the patient group has a mean

duration of 7.19 (SD 5.73) years for the disorder. Previous treatments: A total of 62.66% of the patients had undergone prior treatment at other healthcare centres. Of these, 27.08% had undergone 3 or more treatments. Comorbidity: A total of 56.25% of the patients were diagnosed with other associated psychiatric diseases (e.g., obsessive compulsive disorder, anxiety and personality disorder). Attempted suicide: Some 22.91% of the patients had attempted suicide over the course of their disorder. Self-harm: A total of 45.83% has inflicted self-harm at some point during the course of their disorder.

### 1) Psychometric Characteristics of the SASTCA and SSAS scales

#### A) SASTCA Scale

Reliability: The scale's Cronbach alpha value was 0.946, which indicated that the scale's reliability was very high and within the recommended criteria for clinical research ( $>0.8$ ). Guttman split-half test: The reliability between the two halves of the scale was very high (0.936). This result indicates considerable consistency between the items of the scale, which according to the results measure the same trait. The correlation between forms was 0.880. Only 0.12 was due to possible measurement error (table I). Validity: Diagnostic performance (ROC curve): The scale's overall diagnostic performance was 0.915 (ROC), with a 95% confidence interval in a range from 0.837 to 0.994 (table I) (Fig. 1). These values indicate that the scale's predictive capacity is very high in terms of the diagnosis of AN. Predictive performance of the SASTCA scale for the cut-off of 49: With this cut-off, the SASTCA scale achieved a sensitivity index of 81.25%. A specificity of 93.5%. A positive predictive value of 95.1%. A negative predictive value of 76.3% (table II).

**Table I**  
*Reliability and Validity (SASTCA Scale)*

Reliability (SASTCA Scale)				
Alfa Cronbach				,946
Guttman split-half test				,936
Correlation between forms				,880
Validity: Area under the Curve ROC (SASTCA Scale).				
Test Result Variable(s)				
Area	SD Error <sup>a</sup>	Asymptotic Sig. <sup>b</sup>	Asymptotic 95% Confidence Interval	
			Lower Bound	Upper Bound
,915	,040	,000	,837	,994

<sup>a</sup>Under the nonparametric assumption. <sup>b</sup>Null hypothesis: true area = 0.5.

**Table II**  
Predictive Performance for the cut-off of 49 (SASTCA scale)

SASTCA	AN Group		Control Group		Total Group
	N	%	N	%	N
Value Positive ( $\geq 49$ p)	39	–	2	–	41
<sup>a</sup> Sensitivity index	39/48	81,25	–	–	–
<sup>c</sup> Positive predictive value	39/41	95,12	–	–	–
Value Negative ( $< 49$ p)	9	–	29	–	38
<sup>b</sup> Specificity	–	–	29/31	93,54	–
<sup>d</sup> Negative predictive value	–	–	29/38	76,31	–
Total Group	48		31		79
Mean (SD)	58.72 (12.4)		37.80 (7.5)		F 47,248
					p <.001

<sup>a</sup>Sensitivity index (AN Group Positive/AN Group). <sup>b</sup>Specificity (Control Group Negative/Control Group Total). <sup>c</sup>Positive predictive value (AN Group Positive/ Positive Total Group). <sup>d</sup>Negative predictive value (Negative Control Group/Negative Total Group).

**Table III**  
Validity: Area Under the Curve ROC (SSAS Scale)

Test Result Variable				
Area	SD Error <sup>a</sup>	Asymptotic Sig. <sup>b</sup>	Asymptotic 95% Confidence Interval	
			Lower Bound	Upper Bound
,712	,068	,008	,578	,845

<sup>a</sup>Under the nonparametric assumption. <sup>b</sup>Null hypothesis: true area = 0.5.

## B) SSAS Scale

Diagnostic Performance of the SSAS scale. The ROC curve of SSAS scale achieved an area of 0.712 with a 95% confidence interval in a range from 0.578 to 0.

845, (table III), (Fig. 2). Predictive performance of the SSAS scale for a cut-off point of 27. With this cut-off, the scale achieved a sensitivity index of 79.2%, a specificity of 48.4%, a positive predictive value of 70.4% and a negative predictive value of 60.0% (table IV).

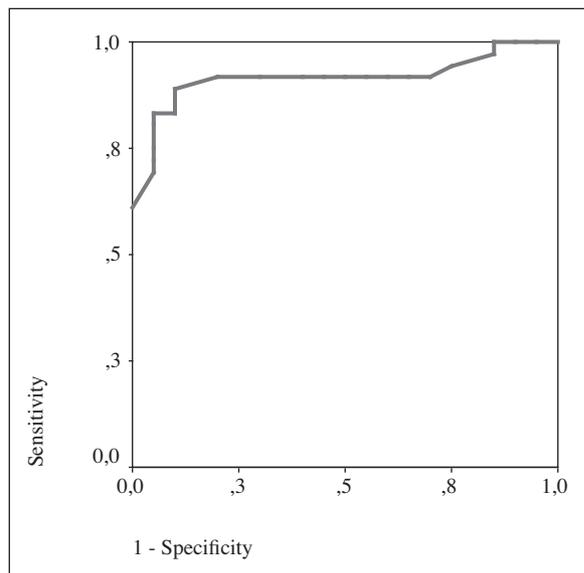


Fig. 1.—Validity: Area under the Curve \*ROC (SASTCA Scale). \*Receiver Operating Characteristic (ROC).

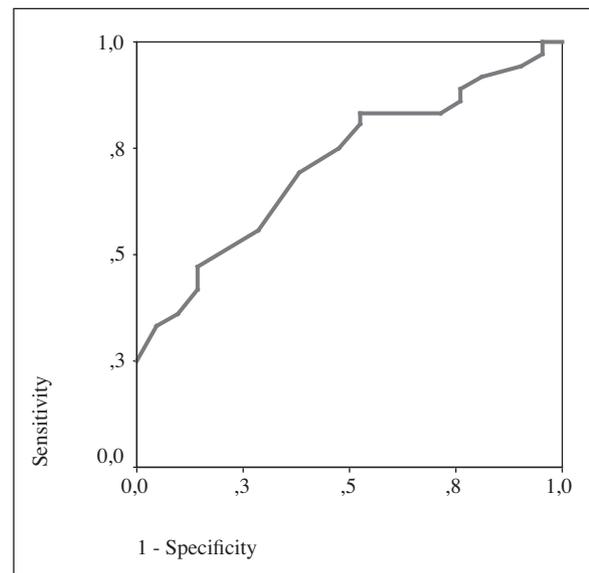


Fig. 2.—Validity: Area under the Curve \*ROC (SSAS Scale). \*Receiver Operating Characteristic (ROC).

**Table IV**  
*Predictive Performance for the cut-off of 27 (SSAS scale)*

SASTCA	AN Group		Control Group		Total Group
	N	%	N	%	N
Value Positive ( $\geq 49$ p)	38	–	16	–	54
<sup>a</sup> Sensitivity index	38/48	79,2	–	–	–
<sup>c</sup> Positive predictive value	38/54	70,4	–	–	–
Value Negative ( $< 49$ p)	10	–	15	–	25
<sup>b</sup> Specificity	–	–	15/31	48,4	–
<sup>d</sup> Negative predictive value	–	–	15/25	60,0	–
Total Group	48		31		79
Mean (SD)	31.19 (6.7)		26.57 (5.5)		F 7,202 p .010

<sup>a</sup>Sensitivity index (AN Group Positive/AN Group). <sup>b</sup>Specificity (Control Group Negative/Control Group Total). <sup>c</sup>Positive predictive value (AN Group Positive/ Positive Total Group). <sup>d</sup>Negative predictive value (Negative Control Group/Negative Total Group).

### C) Relationship between the two scales.

The 2 scales correlated with each other with a correlation coefficient of  $r = .634$ , which was statistically significant ( $p < .001$ )

#### 2) Differences between the groups in the 2 scales.

The patients with Anorexia Nervosa and EDNOS achieved a mean on SASTCA scale of 58.72 (SD 12.4), which was much higher than the mean of 37.80 (SD 7.5) achieved by the control group. The difference was statistically significant ( $p < .001$ ) and occurs to a lesser extent in the case of the SSAS scale. The patient group achieved a mean of 31.19 (SD 6.7), higher than the mean of 26.57 (SD 5.5) from the control group ( $p < .001$ ), (table II, table IV).

## Discussion

The preliminary results from this study support the possible presence of heightened sensory sensitivity in patients with anorexia, which differs from the sensitivity of the control group. This heightened sensitivity has been related to the individual's susceptibility to developing unexplained somatic complaints about health (SSD) or appearance (BDD)<sup>32,33,34</sup>. This sensitivity has been related to the individual's susceptibility to developing the somatic symptoms disorder<sup>19</sup>. To measure this construct, we developed the SASTCA scale. Both the reliability and the validity of the SASTCA scale indicate that it can be a useful instrument for assessing the amplified somatosensory sensitivity of patients with anorexia. The scale's reliability measured by the statistical value Cronbach's alpha indicates that the scale is highly reliable and is within the recommended criteria for clinical research. The Guttman split-halves

revealed considerable consistency between the items on the scale, which according to the results measure the same trait. In terms of the scale's validity. The ROC values showed that the overall diagnostic performance of this scale (i. e. , its predictive capacity) is extraordinarily high for the diagnosis of AN.

After several attempted cut-off points, we selected the cut-off of 49 because it offered the scale's best predictive value. With this cut-off, the SASTCA scale had excellent diagnostic performance, which was particularly reflected in the extremely high specificity. The scale not only correctly identified patients with anorexia but also detected the absence of disease in the healthy participants. This fact makes the scale a good tool for confirming the diagnosis, given its high positive predictive value. After comparing the performance of the patient group and the control group on the SASTCA scale, the data indicate that the patients with Anorexia Nervosa or EDNOS have a much higher sensitivity than the control group. The difference was statistically significant.

When comparing the results from the SASTCA scale with those from the SSAS scale, we can see that the 2 scales are related despite their significant differences. The SSAS scale has a high sensitivity index and correctly identified patients with anorexia. However, its specificity is notably lower. The SSAS scale detects numerous false positives because it cannot properly discriminate the absence of disease in healthy patients. These values reveal that the 2 scales are related and share or describe aspects of the same "dimension" but are not identical.

The notable predictive capacity and precision of the SASTCA, reflected in the ROC curve, suggest that its sensitivity could be maximised if other cut-off points are chosen and provide the SASTCA with the capacity to screen for anorexia. Given that the heightened sensory sensitivity measured by the SASTCA detects the patient group with anorexia and discriminates those

from the participants in the control group, and given that the scale does not include any pathological eating behaviour, it is feasible to think that this heightened sensitivity could precede the onset of pathological eating behaviours. In this case, the highly sensory sensitivity would constitute a vulnerability factor for the formation of a distorted body image for patients with anorexia.

Is it possible that heightened sensory sensitivity, common to other somatic disorders, is part of a somatic vulnerability (Sensory-Processing Sensitivity)<sup>35</sup> which would explain the formation of body self-concept distortion in all these patients? This possibility is supported by several arguments. Firstly, the limited awareness of emotional states is also present in patients with anorexia<sup>36</sup>. Studies of the emotional characteristics of patients with ED indicate that these patients have greater difficulty identifying and describing emotions<sup>37,38</sup> and less appropriate methods (avoidance, escape, denial) for regulating the emotion than the control groups of women without ED<sup>39</sup>. Difficulties recognizing emotional states are also associated with the level of severity. Secondly, there appears to be a parallel in the process of development of various perceptive distortions (disease, fatness, ugliness) in the above-mentioned patients groups<sup>40,41</sup>. Given that the most detailed description in the scientific literature is that reported for patients with unexplained health complaints, we focused the comparison of the development of the self-concept "I am sick" with the development of the self-concept "I'm fat" in patients with anorexia. The correlation between the 2 processes is somewhat striking.

The start of the process is related to stressful experiences associated with the body. Patients with health concerns report having undergone negative experiences prior to the disorder, related or not to the disease itself and they exhibit considerable anxiety about their health due to the uncertainty of medical interventions. In addition, heightened sensitivity to certain external and internal body stimuli has been demonstrated in these patients<sup>42,43</sup>. The interaction between a lower perceptive threshold and this fearful uncertainty would produce a heightened perception of potential sources of danger<sup>44,45</sup>. As a result of this interaction, these patients react with considerable concern and alarm when faced with any bodily sensation. Their anxiety leads to focusing their attention on these sensations, which they monitor excessively and continuously, assessing the sensations as abnormal regardless of how subtle they are. The alarmist interpretation of these sensations leads them to believe that they are sick. The distorted beliefs remain relatively silent until a critical event, internal or external, mobilises them. In supposedly threatening situations, the dysfunctional beliefs activate launching alarmist thoughts about the bodily sensations ("I have cancer"), triggering strong anxiety about their health and leading to relief behaviours (visiting the emergency department<sup>46,47</sup>. These relief

behaviours (negative reinforcement) contribute to the maintenance of the distorted self-concept.

If we change the concept of ill to fat, the process fits almost entirely with what we observe in anorexia nervosa. The patients with anorexia reported having undergone stressful experiences with the body during childhood and adolescence (premature or late maturity, negative verbal comments and mocking of their body, sexual abuse), which lead them to reject their body. They were exposed to other iatrogenic factors such as a gaining reinforcements (admiration for their thin appearance) at the start of weight loss, which they fear losing. In addition to these personal experiences, there is the unattainable body ideal promoted by society, which for these patients is another highly stressful element. The combination of several or all of these elements causes them considerable anxiety when faced with the possibility of being fat. Extreme anxiety, together with the heightened sensory sensitivity would produce an increased perception of potential sources of danger<sup>45</sup>. Once the sensation is distorted, the all-or-nothing cognitive processing leads to alarmist interpretations of these sensations<sup>48</sup>. As a result of the interaction between fearful uncertainty, heightened sensitivity, and cognitive distortion, these patients react with considerable concern and alarm when faced with certain bodily sensations. From that moment on, the belief that one is fat transforms into the defining cognitive schema of their body self-concept<sup>49</sup>. Their entire behaviour is directed towards avoid fatness<sup>50</sup>. The possibility of not achieving this goal creates so much anxiety that their attention is focused on monitoring these sensations in an exaggerated and continuous manner. Unfortunately for the patient, the threat is constant because the external stimuli (e. g. , news or information on thinness, fatness and related issues) are constantly present in the environment. The bothersome internal sensations that they erroneously interpret as hunger or fatness (e. g. , anxiety, tension, constipation) are hard to escape, and the possibility of gaining control over their body weight is impossible.

There are numerous research studies that have demonstrated the relationship between fatness anxiety and bias in ED. Patients process the threatening words and images of fatness and high-calorie food and actively ignore the words and images that connote a thin physique<sup>51</sup>. They also better remember the issues related to food and weight than other nonthreatening issues<sup>52</sup>. The bias is kept in their mind, which also stores the memory of bodily sensations that warn them of danger. In supposedly threatening situations (e. g. , greater than planned food intake, comparison of their body with other actual or photographed thin bodies), the dysfunctional beliefs activate triggering alarmist thoughts and body images that lead to relief behaviours (e. g. , restricting food intake, exercising excessively, vomiting, performing stomach crunches). These relief behaviours, as with those performed by patients with SSD who go to multiple medical consultations, act as

negative reinforcement, which contributes to the maintenance of the distorted self-concept.

The similarities between the 2 processes do not end here. The mistaken body perceptions of the 2 patient groups are not reduced by the verbal feedback provided by their close friends and practitioners who treat them<sup>53</sup>. The difficulties in changing their distorted view are similar and correspond to that of patients with cerebral disconnections, such as anosognosia and split brains<sup>54</sup>. Just as patients with anosognosia deny their paralysis and patients with SSD are not convinced by either the medical tests or the information provided by their doctors, the information received by patients with anorexia on their thinness and risks (e. g. , confronted with the child-sized clothing they wear, data on their malnutrition and osteoporosis) does not change their “perceptive judgment. ” On the contrary, they create reasons and justifications that support their opinion and continue with their self-deception. Through attentional bias towards the stimuli that supposedly indicate danger, patients ignore reality thereby ratifying their mental integrity while validating their distorted self-concept (“I’m fat”), which allows them to justify their negative eating behaviours.

Finally, there appears to be a similarity in their neurocognitive processing<sup>55</sup>, and the participation of structures involved in the brain circuits that govern body image, both in terms of multisensory integration and the experience of personification or belonging<sup>56</sup>. A recent review of the cognitive–affective neuroscience of somatisation disorder suggested that the dysfunctional brain regions are the dorsolateral prefrontal, insular, rostral anterior cingulate, premotor and parietal cortices, which are similar to the structures affected in AN<sup>57,58,59,60</sup>. Elaborating further on the similarities between patients with anorexia and patients with somatic disorders, if we change the concepts of ill and fat for misshapen and ugly, the processes of forming distorted self-concepts in patients with BDD are dramatically similar.

Once we have proposed the parallelism of the processes of forming distorted self-concept (disease, deformity, fatness) and observed the presence of heightened sensory sensitivity in patients who exhibit them, numerous still unanswered questions arise. The first of course refers to the replication of data from this study in other groups with anorexia. If the results were positive, we should ask ourselves whether this construct proposed in the SASTCA also occurs in other eating patients at various points in their development of the disorders and in their first-degree relatives.

If the results were positive, could the heightened sensory sensitivity constitute a broad-spectrum trait (sensory processing sensitivity?) that includes patients with anorexia, patients with somatic symptom disorder, patients with body dysmorphic disorder? Such a trait would be associated with greater sensitivity and responsiveness to the environment and social stimuli<sup>61</sup>. In the setting of anorexia nervosa, it would be reflected

in an avoidant attitude that resembles the “Highly Sensitive Person”, with more inhibited behaviour in the face of novel, highly stimulating situations<sup>62</sup>.

We also must ask about the specificity of the distortion. Why does body image distortion focus on disease, deformity and fatness? Our study does not provide data supporting any type of accurate explanation for this question. However, we have once again returned to the hypothesis associated with the rest of the patients who show heightened body sensitivity to understand what explanation they give to this dilemma<sup>63</sup>. It has been suggested that the heightened sensitivity of patients with SSD could be the result of cerebral sensitisation caused by chronic exposure to stressful experiences concerning their body. In other words, it is the result of a learning process that, through repeated exposure to stimuli, produces progressive heightened sensitivity of the response (physiological, cognitive, emotional and behavioural) to the prominence of somatosensory stimuli<sup>64</sup>. The specificity of the stressors in each patient group would indicate the specificity of the disorders. However, considering the items on the SASTCA, our hypothesis is that a number of aspects of the construct (e. g. , “I feel choked when clothing rubs or pinches me”, “I notice right away if my stomach growls”) could be the result of a process of cerebral sensitisation. Other aspects (e. g. , tendency to bruising, skin redness and scratches), however, seem to belong to genetically determined factors (sensory processing sensitivity?), that affect the perceptive threshold of body stimuli.

Lastly, the importance of this study lies in the value it provides in understanding another neurobiological characteristic of AN that can facilitate the early detection of vulnerable individuals and, as a result, establish pre-emptive intervention. Although a large number of studies and investigations are needed to reach a similar conclusion, the results seem sufficiently suggestive to interest other research groups in the topic and for them to provide their ideas and conclusions.

## Conclusions

The preliminary results from this study indicate the presence of heightened sensitivity to internal and external body stimuli in patients with anorexia, which differs from the sensitivity of the control group. The presence of a lower perceptive threshold when faced with specific body stimuli, when interacting with the extreme body anxiety resulting from social pressure to be thin, makes individuals who have this lower threshold more vulnerable to experiencing the eating disorder than individuals who, under the same social pressure, do not present somatosensory amplification.

This heightened sensitivity has a significant relationship with heightened sensitivity described in patients with SSD and BDD and could contribute to the formation of a distorted body self-concept in these pa-

tient groups. This sensitivity, which precedes the onset of pathological eating behaviours, could be the expression of a somatic endophenotype, whose understanding could provide information on an underlying biomarker in this patient group?

The importance of this study lies in the value it provides in understanding another neurobiological characteristic of AN. As has occurred with the knowledge provided by other endophenotypes (difficulties in set shifting, weak central coherence and heightened sensitivity to punishment), this vulnerability trait can facilitate the early detection of the disorder and, as a result, establish pre-emptive intervention.

The SASTCA scale, which was developed to highlight this heightened sensitivity, presents sufficient internal consistency and optimal validity, which enables it to discriminate between patients with Anorexia Nervosa and healthy control participants. Further work is needed to establish validity and reliability in a wider population, and particularly in those at risk of eating disorders in the general population.

### Limitations of the study

The majority of the content of this article is suggestions based on a small group of patients, and, as such, it does not attempt to draw conclusions but rather suggest a complementary viewpoint on the genesis of anorexia. All patients included in this study are adults with numerous severity criteria, which could constitute a bias in the interpretation of the results. Given that the vulnerability of first-degree relatives is being studied and that the similarities and differences with other somatic disorders were not compared, the proposition that this vulnerability could constitute a somatic endophenotype is more a desideratum than a certainty. Similarly, our data do not allow us to determine whether this vulnerability is genetically determined or whether brain sensitisation occurred at some point in the patients' life.

The SASTCA scale contains the reference to cold sensitivity, although sensitivity to cold is a symptom of anorexia. This sensitivity to cold could be a vulnerability factor and an effect of malnutrition. Future research should be done to verify this.

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