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Abstract.

Body ownership (i.e., the conscious belief of owning a body) and *sense of agency* (i.e., being the agent of one's own movements) are part of a pre-reflective experience of *bodily self*, which grounds on low-level complex sensory-motor processes. While previous literature had already investigated body ownership in obesity, sense of agency was never explored.

Here, we exploited the sensory attenuation effect (i.e., an implicit marker of the sense of agency; SA effect) to investigate whether the sense of agency was altered in a sample of eighteen individuals affected by obesity as compared with eighteen healthy-weight individuals. In our experiment, participants were asked to rate the perceived intensity of *self-generated* and *other-generated* tactile stimuli.

Healthy-weight individuals showed a significantly greater SA effect than participants affected by obesity. Indeed, while healthy-weight participants perceived self-generated stimuli as significantly less intense as compared to externally generated ones, this difference between stimuli was not reported by affected participants.

Our results relative to the SA effect pinpointed an altered sense of agency in obesity. We discussed this finding within the motor control framework with reference to obesity. We encouraged future research to further explore such effect and its role in shaping the clinical features of obesity.

Keywords: obesity; sensory attenuation; sense of agency; body awareness.

Introduction

Body ownership (i.e., the conscious attribution of ownership towards one’s own body (Blanke et al., 2015) and *sense of agency* (i.e., the experience of being the agent of one’s own actions (Haggard & Chambon, 2012) are part of a pre-reflective experience of embodied experience generated in low-level complex sensory–motor processes (i.e., *bodily self*; Tsakiris et al., 2007). Specifically, sense of agency describes “*the experience of controlling one’s own actions, and, through them, events in the outside world*” (Haggard and Chambon, 2012); in other words, when we perceive a cause-effect relationship between our actions and external events. Body ownership and sense of agency seem to be strictly related each other to generate a full and efficient experience of bodily self: indeed, it was reported that sense of body ownership is necessary to experience sense of agency, as well as sense of agency contributes to a coherent and integrated sense of body ownership (Tsakiris et al., 2007).

Scarce and not conclusive evidence addressing body ownership had been reported (Tagini et al., 2020a; 2021; Scarpina et al., 2019a) in obesity. A recent work by Tagini and colleagues (2020a) compared the susceptibility of individuals affected by obesity to the well-known Rubber Hand Illusion (Botvinik & Cohen, 1998, i.e., an experimental procedure leading individuals to feel as if a rubber hand was part of their own body) with a sample of healthy-weight individuals. Their findings indicated that affected participants showed an illusory experience that was in line with the expect effect (Botvinik & Cohen, 1998) at the explicit level (i.e., the questionnaire about the subjective experience of the illusion), but a reduced effect of the illusion at the implicit level (i.e., the proprioceptive drift). This alteration in terms of recalibration of hand position after the illusion had been hypothesized as linked to the aberrant multisensory integration process subserving body representation registered in obesity (Tagini et al., 2020b; Scarpina et al., 2016, 2019b). Yet, to the best of

our knowledge, no previous study investigated possible alteration of sense of agency in this clinical population.

To provide the first evidence about sense of agency in obesity, we exploited the *sensory attenuation* effect (SA effect; Blakemore et al., 1998; 2000) in a sample of affected individuals compared with healthy-weight controls. SA is traditionally described as an implicit marker of the sense of agency (Haggard & Chambon, 2012, p. 197). It is well established that the intensity of self-generated tactile stimuli is subjectively perceived as less intense in comparison with stimuli delivered at the same level of intensity and generated by someone' else (Blakemore et al., 1998; 2000). The SA effect of incoming sensory input is explained by the comparator model of the motor control system (Blakemore et al., 2000; Frith et al., 2000). When the motor program of voluntary actions is sent to the muscles, an efferent copy of the commands is used by an internal cognitive model to predict their sensory consequences. A correct prediction, grounded on the match between the expected and the actual feedback, induces the attenuation of the sensory consequence for self-generated actions (i.e., *self-monitoring mechanism*) (Blackmore et al., 2000). Thus, this mechanism might support individuals to determine whether a sensory event is caused by them or not (Moore & Fletcher, 2012). In line with the previous literature about the SA effect; (Blakemore et al., 1998; 2000), in our experiment individuals affected by obesity were asked to judge the perceived intensity of tactile stimuli which could be either self- or other-generated (i.e., produced by the experimenter). We compared their judgments with that provided by the control sample, which consisted of individuals with a healthy weight. We expected to observe the SA phenomenon in controls, according with the previous literature (Blakemore et al., 1998; 2000). About the individuals with obesity, it should be underlined that compelling evidence showed that when the sense of body ownership is altered, the sense of agency is affected as well (see e.g., Kilteni & Ehrsson, 2017; Pyasik et al, 2021). The

previous literature on body ownership in obesity was still controversial; thus, we could only speculate on a specific directionality of such an effect, in other words if individuals affected by obesity would show a SA perception that was in line with the controls. On the other hand, considering the absence of the illusory body ownership experience at the implicit level in obesity (Tagini et al., 2020a), we may predict that the SA effect, being an implicit marker of the sense of agency, may be altered in our sample. Nevertheless, we may be argued the directionality of SA effect, when altered, has been observed as different across different clinical conditions, such as less perceived intensity for self-generated stimuli in individuals with Borderline Personality Disorder as reported by Colle and colleagues (2020) as well as no perceived difference between self-produced and externally produced stimuli when compared in individuals with auditory hallucinations (Blakemore et al., 2000). Thus, an altered SA effect in obesity might be represent by similar level of perceived intensity between self- and other-generated stimuli or less perceived intensity for self-generated stimuli in comparison with the other-generated ones.

Materials and Methods

Participants. The experimental procedure was approved by the Ethic Committee of the Istituto Auxologico Italiano (21C727_2017) and of University of Turin (3167, 1/02/2016). Participants were naïve to the experimental procedure; they gave written informed consent before taking part in the study. Only right-handed individuals participated in this experiment (Oldfield, 1971).

Eighteen participants affected by obesity were recruited during the first week of a diagnostic recovery in the IRCCS Istituto Auxologico Italiano, Ospedale San Giuseppe (Piancavallo, Italy). In line with previous studies (Scarpina et al., 2016; 2019ab; Tagini et al., 2020ab,2021), only participants with a body mass index (i.e., BMI) over the value of 30 (i.e.,

indicating obesity, World Health Organization, 2000) were included. We excluded individuals with any neurological symptoms/signs, especially of neuropathic pain, according to the clinical assessment. Also, through a structured interview (American Psychology Association, 2013), we excluded individuals with psychiatric diseases, personality disorders, or eating disorders. Finally, we excluded individuals who have done any rehabilitative procedures.

We also included eighteen individuals with a body mass index below the value of 25 as controls, while all the previous criteria were followed. They were recruited outside the clinical institute.

The target of our experiment was the hand. However, in obesity, its physical dimension is typically enlarged due to the presence of adipose tissue. Thus, in this work, we collected the anthropometric measures of our participants' left hand using a standard caliper. Specifically, we measured the hand width (i.e., the distance between the knuckles of the index and little fingers) and the hand thickness, as the distance between the dorsum and the palm at the level of the knuckles of the middle finger. Considering that individuals affected by obesity generally report a negative body image, exhibiting greater body dissatisfaction compared to healthy weight individuals, (Schwartz and Brownell, 2004), in this experiment we required our participants to fill out the Body Uneasiness Test (Cuzzolaro et al., 2006). This questionnaire is a widely used clinical instrument to assess body uneasiness in obesity; moreover, it has been validated in patients with obesity and has good internal consistency (Cronbach's α coefficient >0.7) (Marano et al., 2007). Details are reported in Table 1.

[Table 1 around here]

SA paradigm. We applied a previously employed experimental protocol (Colle et al. 2020; Fossataro et al., 2020). Participants laid their hands palm down on a desk and gazed a button placed in between them and the experimenter seated in front of them, so that they could observed both their hands and the experimenter’s (see Figure 1).

[Figure 1 around here]

Tactile stimuli were delivered randomly on two sites of the lateral dorsal section of the left-hand by means of a constant current electrical stimulator in two experimental conditions: “*self-generated*”, wherein subjects were instructed to press the button with the right-hand to generate the stimulation; “*other-generated*”, wherein participants observed the experimenter pressing the button to generate the stimulation. Note that the participants could always see the experimenter throughout the task. The intensity of stimulation in the self-generated and in the other-generated conditions was fixed across trials for each participants (see details in Electrical Stimulation section). To check for any false alarms, catch trials (i.e., trials in which no tactile stimulation was delivered; in other words, the intensity of stimulation was set at 0 mA) were randomly included.

After each electrical stimulation, the experimenter asked participants to verbally report the perceived intensity on a seven-point Likert scale, ranging from “no stimulus” to “very intense stimulus”. The experimenter recorded the participants’ answer. No time limit was imposed for answering.

The experiment consisted of two blocks of 48 trials (20 trials self-generated; 20 trials other-generated; 4 catch trials self-generated; 4 catch trials other-generated). The trial order was randomized among participants.

Electrical Stimulation. Transcutaneous electrical stimuli were delivered on the left-hand dorsum through three surface cup electrodes attached to a constant-voltage peripheral nerve stimulator (D185, Digitimer). The stimulus delivery was concomitant with the button press and its duration was 200 μ s. The stimulation intensity was adjusted according to the individual sensory threshold level (i.e., the stimulation intensity wherein participants were able to detect stimuli in the 50% of trials). During the experiment the stimulation intensity was set slightly above the threshold according to the formula $x*2.5+4$ mA, where x represents the stimulation intensity at threshold (i.e., the electric stimulation intensity which causes participants to report 5 stimuli out of 10). Stimuli were randomly delivered to two different stimulation sites to minimize habituation, as shown in Figure 1.

Data analysis. Firstly, to verify the presence of the SA effect in each group independently, Wilcoxon matched-pairs tests were performed between the rating relative to the self-generated stimuli and other-generated stimuli. Secondly, to compare the SA effect between groups, we computed a *SA index* by subtracting the mean ratings of the self-generated condition from the mean ratings provided in the other-generated condition (*SA index* = other-generated ratings minus self-generated ratings). Therefore, an index with positive values indicates the presence of the SA effect (self-generated stimuli are perceived as less intense in comparison with other-generated stimuli); an index with negative values, the opposite effect (other-generated stimuli perceived as less intense as compared to self-generated stimuli). The between-group differences in the SA indices were analyzed through an independent sample t-test. In the case of significant difference in the between-subjects analysis, we compared the stimulation threshold between groups through an independent sample t-test to exclude that the results was due to the differences in the stimulation intensities. For each contrast of interest we reported the p value and the corresponding effect

size. Finally, we explored the relationship between the SA index and the anthropometric measures (i.e., body mass index, hand width, and hand thickness), as well as the scores reported at the Body Uneasiness Test (Cuzzolaro et al., 2006) through the Spearman correlation, independently for the two groups.

Sample size. We estimated *a priori* the sample size for our study (Faul et al., 2007).. A total sample of 36 participants with two equal sized groups of $n = 18$ was necessary to achieve a power ($1 - \beta$) of 0.95, considering the use of a two-tailed test, an alpha of 0.05, and the effect size as 1.44 (as observed in unpublished data collected in a different pathological sample employing the very same paradigm).

Results

In all the catch trials, all participants rated the stimulation as zero; thus, we can exclude false alarms. Wilcoxon tests showed that healthy-weight participants rated the self-generated stimuli ($M=4.46$; $SD=1.09$) as less intense compared to other-generated stimuli ($M=4.74$; $SD=0.94$) [$w=91$; $p=0.04$; $dz = 0.56$], in line with the SA effect (Figure 2A)

[Figure 2 around here]

Instead, participants affected by obesity did not report any difference between the two experimental conditions (self-generated stimuli $M=4.52$; $SD=1.79$; other-generated stimuli ($M=4.29$; $SD =1.77$) [$w=-40$; $p=0.36$; $dz=0.41$] (Figure 2B). Crucially, a significant difference emerged between groups [$U=92.50$; $p=0.02z$; $dz =0.92$] in the SA index; indeed, the SA index was significantly higher in healthy-weight participants ($M=0.27$, $SD=0.49$) as compared to participants with obesity ($M=-0.22$; $SD=0.58$) (Figure 2C). Note that we

employed a non-parametric analyses since the distribution of residuals was not normal (Shapiro Wilk test <0.05). Importantly, the sensory threshold was not different [$t_{(34)}=0.48$; $p=0.63$; $d=0.15$] between healthy-weight participants ($M=33.94$; $SD=14.39$) and participants with obesity ($M=32.06$; $SD=8.24$) (Figure 2D).

To verify the consistency of SA effect across different studies, an *a-posteriori* independent sample *t*-test was performed to compare the SA index registered in our experiment to the same outcome reported in a previous work in which the same setting was used (Colle et al., 2020). Crucially, the SA index registered in our study on the healthy participants was comparable to what registered in the previous study by Colle and colleagues (2020) ($N=20$; $M=0.20$; $SD=0.34$) [$t(32)=0.5$, $p=0.3$; Cohen's $d=0.16$].

[Figure 2 around here]

As reported in Table 2, a significant negative correlation emerged between SA index and both the hand thickness and Body Mass Index (see Figure 2E). Whereas, no significant relationship emerged between the SA index and the hand with, as well as with the scores reported at the Body Uneasiness Test (Cuzzolaro et al., 2006).

[Table 2 around here]

Discussion

In the present study, we explored a possible alteration of the sense of agency in individuals with obesity by applying the SA paradigm (Blakemore et al., 1998; 2000), wherein the perceived intensity of self- and other-generated stimuli is compared.

We confirmed the SA effect in our healthy-weight participants, since they rated the intensity of self-generated stimuli as significantly less intense in comparison with other-generated stimuli, in agreement with the previous evidence (Blakemore et al., 1998; 2000; Colle et al., 2020). Instead, our participants affected by obesity did not show such a difference. Moreover, we reported that our controls showed a higher SA effect, as suggested by the positive SA index, in comparison with the participants with obesity, exhibiting a negative SA index. Since we did not observe any difference in the tactile threshold between our samples, in line with other recent evidence (Tagini et al., 2021), our results were unlikely to be explained by the intensity of the peripheral stimulation.

SA is traditionally described as an implicit marker of the sense of agency, meaning “the experience of controlling one’s own actions, and, through them, events in the outside world” (Haggard & Chambon, 2012, p. 197). The perception of a cause-effect relationship between our actions and external events allows us to recognize ourselves as agents of those actions (Moore & Fletcher, 2012). It has been suggested that the preparation and initiation of actions (i.e., *motor intentionality*) and the prediction of their consequences on the environment seem to hold a crucial role in promoting the sense of agency. When the motor program of voluntary actions is sent to the muscles, an efferent copy of the commands is used by an internal cognitive model to predict their sensory consequences. A correct prediction, grounded on the match between expectations and actual feedbacks, determines the attenuation of the sensory consequence for self-generated actions (i.e., *self-monitoring mechanism*) (Blackmore et al., 1998; 2000). According to the *comparator model* (Blakemore et al., 2000; Frith et al., 2000), the SA effect might represent a tool for assessing maladaptive efference copy generation (Ford et al., 2010; Hughes et al., 2013). Thus, an alteration of this mechanism might point out difficulties in forming or accessing representations of the actual or predicted sensory consequences of action (Hughes et al., 2013). However, our results,

cannot be highlighted what cognitive stage (forming the representation of action, accessing to this representation, or the prediction of sensory consequence of action) would be altered in obesity, leading to an altered SA effect. Nevertheless, sensory-physiological input plays a role too in shaping sense of agency (Saito et al., 2015): the somatosensory and motor systems communicate via a network of extensive connections between the sensory and motor cortices (e.g., Anasuma et al., 1968; Strick & Preston, 1978; Stepniewska et al., 1993; Mao et al., 2011; Tamè et al., 2015), but also by motor cortex cells responding directly to sensory stimuli (e.g., Albe-Fessard & Liebeskind, 1966; Fromm et al., 1984) and sensory cortex cells controlling motor behaviour (e.g., Matyas et al., 2010). In this vein, some preliminary evidence about altered activity in motor cortex in individuals affected by obesity can be traced in the literature (Sui et al., 2020; Wang et al., 2002). Nevertheless, our participants affected by obesity do not show an alteration of tactile perception as compared to healthy-weight controls. Thus, it is reasonable to hypothesize some alteration at the higher (i.e., cognitive) level of processing; specifically, we would suggest that the alteration of the sense of agency in obesity may be explained at the light of the tight link between sense of agency and body ownership, which both jointly contribute to a coherent representation of the bodily self (Tsakiris et al., 2007; Kilteni & Ehrsson, 2017; Pyasik et al., 2021). In this vein, the results from the present experiment, suggesting the alteration of the sense of agency in obesity, may be read in conjunction with previous, even though rare, evidence about the characteristics of body ownership in this clinical condition (Tagini et al., 2020a; Scarpina et al., 2019). Nevertheless, to have conclusive remarks, both components should be investigated in the same sample. Furthermore, since previous evidence pinpointed that individuals affected by obesity show altered temporal sensitivity (Scarpina et al., 2019; 2019b; Tagini et al., 2020b), future studies could investigate whether the alteration of the SA effect observed here is linked to this impairment, by including different temporal delays between the self- and

other-generated action and the stimulus delivery. The alteration of temporal sensitivity may be particularly interesting in the context of sensory-motor interactions, since it has been reported that the timing between tactile stimuli as well as the timing between tactile and motor events changes the motor response (e.g., Bays et al., 2005; Tamè et al., 2015). The physiological counterparts of such temporal effects between action and its sensory consequences could benefit of neurophysiological techniques.

It would be interesting to better explore in future studies if an alteration of the predicting system might play a role in eating behaviors. Indeed, individuals with obesity affected by eating disorders generally experience loss of control or of being not in charge of own feeding behavior (Goossens et al., 2007). In this work, we enrolled, only participants with no diagnosis of eating disorder; in other words, these individuals reported no loss of control of their own feeding behaviour in the clinical assessment. Given that the sensory attenuation is an implicit marker of sense of agency and thus of the participants' control over their own actions, the altered sensory attenuation in our sample without altered eating behaviors may suggest the presence of a subclinical loss of control. Thus, one may speculate that the SA paradigm, being specifically developed to implicitly measure the sense of agency (Moore and Fletcher, 2012), may represent a potential tool to unveil subclinical loss of control over feeding behavior, which is as a critical factor in obesity (Mobs et al., 2010). Nevertheless, we cannot exclude the role played by top-down factors, such as the emotional valence (Takahata et al., 2012) or beliefs about the sensations (Synofzik et al., 2013), in shaping sense of agency in our participants affected by obesity.

This study is pivotal; future investigation needs to further explore SA in obesity, overcoming the limitations of the present work. The first one regards the sample size, that should be enlarged. Nevertheless, our sample size is in line with previous studies in which the SA mechanism was investigated (Blackmore et al., 2006; Colle et al., 2020). Moreover, in the

enrollment, we adopted tight inclusion/exclusion criteria to exclude the confounding effects of comorbidity with other pathologies. Thus, despite the acknowledged limitations, the very novel results included in the present manuscript would be generated future research in the field.

Summarizing, even though we cannot specify at which cognitive or neurophysiological level the alteration occurs, our data suggested that sense of agency might be differently shaped in obesity. However, what would be the effect of an altered sense of agency? This mechanism refers to the feeling of being an individual who is capable of taking action in and on his/her environment, and who can plan and carry out intentional actions (Colle et al., 2020). However, if after-effects observed in the environment cannot be correctly attribute to own-self rather than to other-self, this might diminish the perception to be in charge of their own actions. In this vein, sense of agency seems to provide a key link between perception and higher psychological and cognitive processes, such as volition, motivation, and responsibility (Beck et al., 2017), as suggested by studies relative to different clinical conditions characterized by altered volition (Frith, 2005; Geurts et al., 2012; Oren et al., 2016), but also self-esteem (Greenberg et al., 1982) and social behavior (Pfister et al., 2014). With this study, we would encourage future research in which sense of agency and its relationship with individual' psychological functioning are explored in the context of obesity.

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Data availability. The datasets generated during and analyzed during the current study are not publicly available due to ethical reasons, but are available from the corresponding author on reasonable request.

Author Contributions: F.S., C.F., and F.G. conceived the experiment; F.B. performed the data collection on the experimental subjects; C.F. and A.R.S. collected performed the data collection on the controls; F.S., C.F. and A.R.S. performed the statistical analyses; F.S., C.F. and A.R.S. wrote the first draft of the paper; F.G. contributed to the last version of the paper; M.S. and A.M. supervised the data collection on the experimental subjects. All authors have approved the final article.

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Figure Captions

Figure 1. Experimental setting (right panels). Participants sat in front of each other and gazed at the button between them. In the *self-generated* condition, the participant pressed the button triggering the stimulus-delivery on their own hand; in the *other-generated* condition, the experimenter pressed the button triggering the stimulus-delivery on the participant's hand. Three electrodes were attached on the participant's left hand (left panel): two of them with a positive polarity, which represented the two stimulation sites (green dots), and the other one with negative polarity (red dot). In half of the trials, one of the two electrodes with the positive polarity was employed, whereas in the remaining half the other positive electrode was employed. The electrode with the negative polarity was never exchanged.

Figure 2. Mean (bars), standard error (vertical lines) and each subject's mean (dots) relative to the subjective ratings (y-axis) in self-generated (dotted bar) vs other-generated (empty bar) conditions are shown for healthy-weight participants (in blue) in **A)** and for participants with obesity (in red) in **B)**. **C)** The sensory attenuation index is shown for the two groups. **D)** The sensory threshold (mA) is shown for the two groups. **E)** Correlation analyses between SA index and the anthropometric measures of hand thickness and Body Mass Index (BMI). * denotes a significant difference ($p < 0.05$).

Figure 1. Experimental setting (right panels). Participants sat in front of each other and gazed at the button between them. In the *self-generated* condition, the participant pressed the button triggering the stimulus-delivery on their own hand; in the *other-generated* condition, the experimenter pressed the button triggering the stimulus-delivery on the participant’s hand. Three electrodes were attached on the participant’s left hand (left panel): two of them with a positive polarity, which represented the two stimulation sites (green dots), and the other one with negative polarity (red dot). In half of the trials, one of the two electrodes with the positive polarity was employed, whereas in the remaining half the other positive electrode was employed. The electrode with the negative polarity was never exchanged.

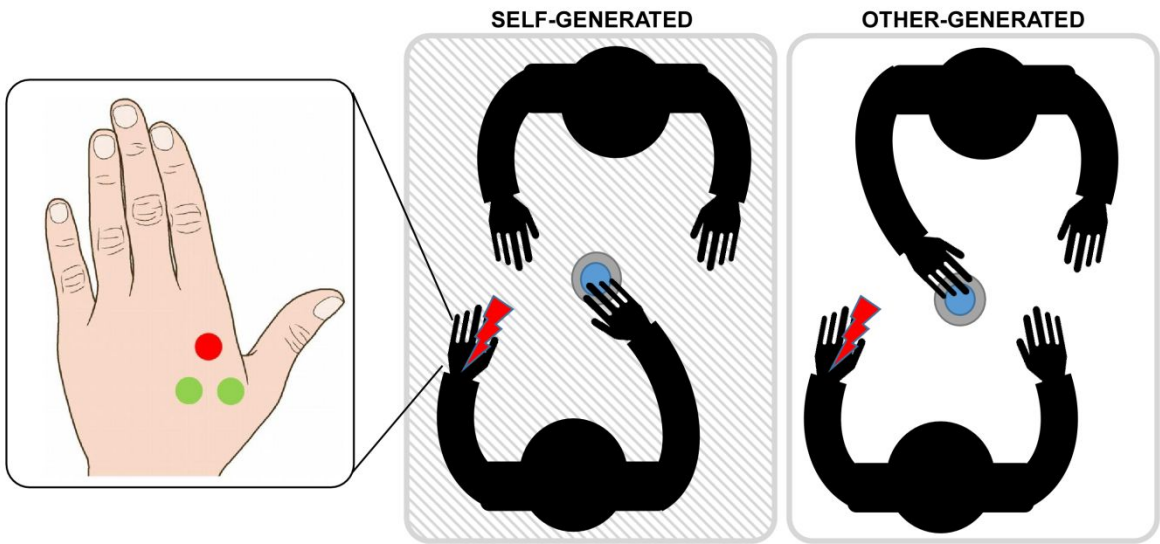


Figure 2. Mean (bars), standard error (vertical lines) and each subject's mean (dots) relative to the subjective ratings (y-axis) in self-generated (dotted bar) vs other-generated (empty bar) conditions are shown for healthy-weight participants (in blue) in **A)** and for participants with obesity (in red) in **B)**. **C)** The sensory attenuation index is shown for the two groups. **D)** The sensory threshold (mA) is shown for the two groups. **E)** Correlation analyses between SA index and the anthropometric measures of hand thickness and Body Mass Index (BMI). * denotes a significant difference ($p < 0.05$).

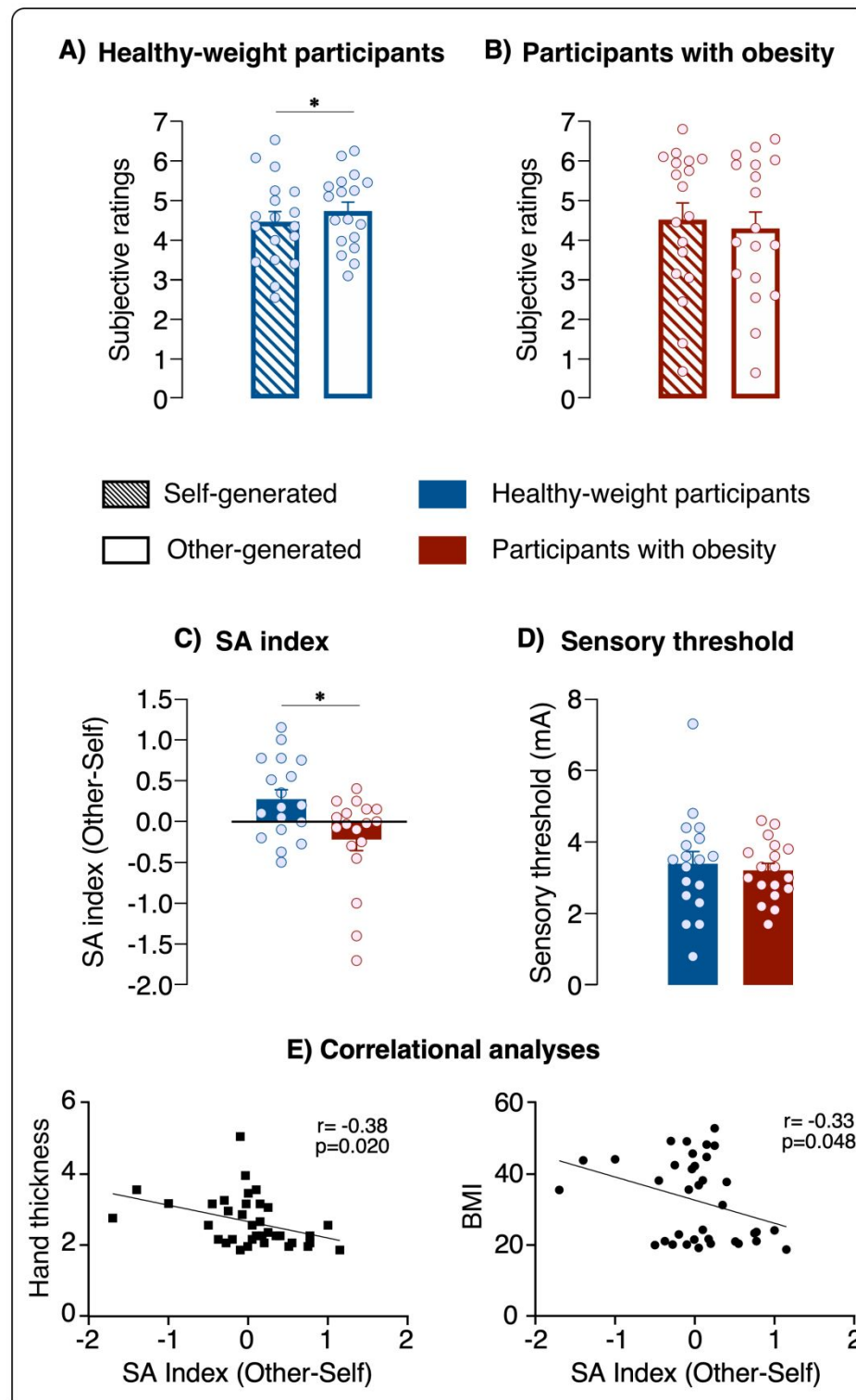


Table 1. Participants’ demographical characteristics, anthropometric measures, and scores reported at the Body Uneasiness Test.

	Participants affected by obesity (N=18)	Healthy-weight participants (N=18)	Statistical results
Age in <i>years</i>	37.61 ± 11.24	31.78 ± 9.87	$t_{(34)}=1.66$; $p=0.11$; $d' = 0.55$
Education in <i>years</i>	12.89 ± 2.22	14.5 ± 3.2	$t_{(34)}=1.75$; $p=0.09$; $d' = 0.54$
Hand width in cm	8.82 ± 1.22	7.9 ± 0.55	$t_{(34)} = 2.73$; $p=0.01$; $d' = 0.97$
Hand thickness in cm	3.11 ± 0.66	2.07 ± 0.21	$t_{(34)}= 5.86$; $p<0.001$; $d' = 2.1$
Body Mass Index	42.99 ± 5.25	21.89 ± 2.86	$t_{(34)}=14.99$; $p<0.001$; $d' = 4.99$
Body Uneasiness Test			
Global Score Index	1.69±1.17	0.56±0.44	$t_{(34)}=3.6$; $p =0.001$; $d' = 1.27$
Weight phobia	1.97±1.33	0.81±0.64	$t_{(34)}=3.06$; $p=0.004$; $d' = 1.11$
Body image concerns	2.5±1.38	0.53±0.43	$t_{(34)}=5.21$; $p<0.001$; $d' = 1.11$
Avoidance	1.21±1.27	0.2±0.31	$t_{(34)}=3.21$; $p=0.003$; $d' = 1.92$
Compulsive self-monitoring	1.03±0.92	0.69±0.59	$t_{(34)}=1.24$; $p=0.22$; $d' = 0.44$
Depersonalization	1.18±1.32	0.26±0.48	$t_{(34)}=2.69$; $p=0.01$; $d' = 0.92$
Positive Symptom Total	14.17±14.55	9±5.63	$t_{(34)}=1.41$; $p=0.17$; $d' = 0.46$
Positive Symptom Distress Index	2.5±1.12	1.46±0.55	$t_{(34)}3.46$; $p=0.001$; $d' = 1.17$

In bold, significant difference.

Table 2. The relationship between the SA index and *i)* the anthropometric measures and *ii)* the scores at the Body Uneasiness Test.

Correlation results		
		SA index
anthropometric measures	Hand width (cm)	r= -0.06 p=0.69
	Hand thickness (cm)	r= -0.38 p=0.02
	Body Mass Index	r= -0.33 p=0.04
Body Uneasiness Test	Global Severity Index	r= -0.09 p=0.56
	Weight phobia	r=-0.08 p=0.61
	Body image concerns	r=-0.15 p=0.36
	Avoidance	r=0.02 p=0.88
	Compulsive self-monitoring	r=-0.09 p=0.6
	Depersonalization	r=-0.07 p=0.66
	Positive Symptom Total	r=-0.03 p=0.84
	Positive Symptom Distress Index	r= -0.08 p=0.78