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RESEARCH

Motor placebo effect in obesity: how ergogenic aids can decrease fatigue and improve motor performance

Valeria Volpino[1] · Maria Elena Navarra[1] · Federica Scarpina[1,2] · Alessandro Piedimonte[1,2,5] · Massimo Scacchi[3,4] · Laura Bianchi[6] · Alessandro Mauro[1,2] · Elisa Carlino[1,2]

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Abstract

Purpose Obesity is associated with increased perceived fatigue and reduced physical activity. This study tested whether a placebo ergogenic treatment could reduce perceived exertion and enhance motor performance in individuals with obesity.

Methods Forty-four participants were randomized to a Placebo group, which received sham transcutaneous electrical stimulation paired with positive suggestions, or to a Control group. Endurance (repetitions), perceived exertion (RPE load), and the late Readiness Potential were recorded during a finger-flexion task performed to exhaustion at 60% 1-RM in two sessions separated by 30 min of rest. Outcomes (Repetitions, RPE load, and RP amplitude) were expressed as percentage change (Δ%) from baseline to test, and group differences were analysed through one-way ANOVAs.

Results Compared with Controls, the Placebo group exhibited smaller declines in repetitions and RPE load and a smaller increase in RP amplitude. Outcomes are in line with a reduced perceived fatigue.

Conclusions These findings suggest that positive expectations can alleviate perceived fatigue and reduce the cortical cost of motor preparation in obesity.

Level of evidence Randomized Experimental Trial.

Keywords Placebo · Fatigue · Motor performance · Obesity · Readiness potential

Introduction

Obesity is a clinical condition characterised by a dysregulated energy balance in lipid metabolic mechanisms, where excessive accumulation of body fat leads to structural anomalies, physiological and functional impairments, severely impacting health and the quality of life [1, 2]. It was predicted that by 2025, global obesity prevalence would reach 18% and 21% in males and females, respectively [3]. Recent evidence from university populations indicates that targeted nutritional education can measurably improve lifestyle patterns, which in turn influence perceived energy and willingness to engage in activity [4]. In parallel, narrative syntheses highlight the role of ultra-processed food consumption in obesity risk trajectories and the need for multifaceted, low-cost behavioral supports [5]. Obesity increases the risk of developing chronic disease, cardiovascular disorder, and different types of cancer, leading to a high impact on public health systems. Among a constellation of symptoms, individuals affected by obesity report higher levels of fatigue during

✉ Federica Scarpina
f.scarpina@auxologico.it

[1] Department of Neurosciences, University of Turin, Via Cherasco, 15, 10126 Turin, Italy

[2] Laboratorio di Neurobiologia, Istituto Auxologico Italiano – IRCCS Ospedale S. Giuseppe, Ospedale San GiuseppeVia Cadorna 90, 28824 Piancavallo, Oggebbio, VCO, Italy

[3] Department of Clinical Sciences and Community Health, University of Milan, Via Della Commenda, 19, 20122 Milan, Italy

[4] Istituto Auxologico Italiano, I.R.C.C.S., U.O. Medicina Generale, Ospedale San Giuseppe, Piancavallo, VCO, Italy

[5] Carlo Molo Foundation, Via della Rocca, 24, 10100 Turin, Italy

[6] Servizio di Neurofisiopatologia, IRCCS Istituto Auxologico Italiano, 28824 Piancavallo, Oggebbio, VCO, Italy

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physical exercise [6–8]. Results from the six-minute walk test (6MWT), which is a sub-maximal exercise test used to assess aerobic capacity and endurance in clinical and research settings, showed that individuals with severe obesity walked less distance and reported a higher degree of dyspnea and perceived exertion [9]. The hypothesis is that obesity is associated with a higher proportion of fast-twitch type II fatigable muscle fibres, which, coupled with the accumulation of fat within the muscle that interferes with its structure, leads to prominent fatigability [10, 11]. Moreover, less blood flow and oxygen supply reach the muscles due to reduced capillary density, compromising muscle performance [12]. In adolescents with obesity, the percentage of motor unit activations was lower for isometric and isokinetic knee extension compared with healthy weight adolescents, as well as maximal voluntary strength, leading to impairments in motor performance [13]. On the other hand, studies also report an increase in muscle strength due to increased body mass, muscle proportion, and chronic training for the weight to sustain [14, 15]. Nevertheless, endurance time at higher levels of workload was still impaired in individuals with obesity. They also experience reduced fatigue resistance when voluntarily using the quadriceps femoris muscle [15], which is particularly engaged in core daily activities, such as stair climbing and walking. Impairments in whole-body motor function are also reflected in and accompanied by difficulties in motor control, altering fine motor skills and balance [16, 17] in childhood. Furthermore, the cumulative effect of overweightness and obesity shows that their earlier onset contributes to a higher risk of mobility limitations as older adults [18]. Additional contributors to fatigability have been recognised: a lower strength-to-body-mass ratio and mechanical constraints that limit joint motion, thereby increasing the energy cost of movement [19]; an increased work of breathing due to the mechanical disadvantage of the diaphragm and altered ventilatory mechanics in obesity [20]; tendon quality alterations that link to reduced walking capacity and earlier fatigue [21]. Moreover, obesity is also associated with an increased risk of developing sleep disorders, which in turn have been shown to reduce physical activity and enhance weight gain, leading to daytime sleepiness and excessive fatigue [22]. Lower energy levels and increased perceptions of fatigue negatively impact motivation to exercise and engage in physical activity, reducing the drive needed for weight loss and adherence to weight management programmes. Consistently, evidence from Italian undergraduates indicates that lifestyle profiles and nutritional education interventions influence behavioural adherence, implying that perceived fatigue is a modifiable barrier and that positive expectations can encourage participation in activity [4].

Hence, in light of previous evidence, it is mandatory to understand the peculiarities of the subjective fatigue perception in individuals with obesity, as well as finding novel tools that could assist them during weight-loss programs, increasing the engagement towards motor exercises. Interestingly, individuals who underwent laparoscopic sleeve gastrectomy reduced their weight by 29.7% after 6 months, and their scores on the Fatigue Assessment Scale (FAS) were significantly reduced for both mental and physical fatigue, indicating a link between obesity and fatigue [23].

Since perceived fatigue and physiological exertion are the integration of biochemical, psychological, and sensorimotor feedback [24] arising from peripheral body regions and cortical activations, it has been shown that they can be perturbed and externally modified. For instance, healthy individuals may report less amount of fatigue and improved physical activity after sham ergogenic aids (i.e., placebo treatment), such as inert pills, sham electrical stimulation, or coffee with no caffeine, administered along with positive expectations of performance improvement [25, 26]. Motor placebo effects were described in different sports, such as weightlifting, cycling, and running [27], lowering perceived exertion and increasing endurance, but also in a cohort of patients: individuals with Parkinson's disease decreased their perceived fatigue when exposed to a placebo [28]. Interestingly, neurophysiological changes were observed employing electroencephalography (EEG), suggesting that placebo treatments can modulate brain activity related to motor preparation and execution [28, 29]. Specifically, it has been shown that Readiness Potential (RP) amplitude increases with over-exertion [30], since it is related to the amount of voluntary force and perceived effort [30, 31], and decreases alongside a defatiguing placebo treatment, which lowers the levels of perceived fatigue.

Given the existing literature on placebo effects and fatigue, we aimed to investigate the impact of placebo treatments on fatigue perception and motor performance in individuals with obesity, whose fatigability is modified by both internal (i.e., altered neuronal recruitment) and external (i.e., lower levels of physical activity) factors. Therefore, we used a placebo-mechanism experimental study on motor performance that has already proven its effectiveness in eliciting placebo effects in clinical contexts [28]. We collected a sample of participants with obesity (BMI > 30 kg/m^2); they were divided into two groups: the Placebo group and the Control group. Participants in the Placebo group received a placebo ergogenic treatment (sham transcutaneous electrical stimulation—TENS), while the control group did not receive any treatment and served as a no-treatment group to quantify the placebo effect. Participants in the Placebo group were told that the sham TENS treatment was effective in improving motor activity and recovering from high exertion. As

behavioural measures, we recorded the number of repetitions completed in the motor exercise and the rate of perceived exertion (RPE). Moreover, we included the Readiness Potential (RP) as a neurophysiological marker for fatigue states. In line with the previous literature, we expected an increase in the number of repetitions done of the motor exercise for participants in the Placebo group compared to the Control group, as well as a lower level of RPE and a lower amplitude of the RP curve, underlying less central fatigue.

Materials and methods

Participants

The study was approved by the Ethical Committee of the I.R.C.C.S. Istituto Auxologico Italiano (Italy); it was preregistered at ClinicalTrials.gov [Identifier: NCT06299254] and it was performed in accordance with the ethical guidelines established in the Helsinki Declaration (most recently revised at Fortaleza, 2013).

In this single-centre study, individuals with obesity were consecutively recruited at their admission to the hospital (I.R.C.C.S. Istituto Auxologico Italiano, Ospedale San Giuseppe, Piancavallo, Oggebbio, VCO, Italy). The recruitment and data collection took place from March 1, 2023, to February 28, 2024 (12 months). Participants gave informed written consent before taking part in the experiment, and they were free to withdraw at any point during the experimental procedure. Participants were naïve to the rationale of the procedure used, since the study involved a placebo treatment, and they agreed that some details could be omitted during the experimental procedure, and that they would be debriefed at the end, following authorised deception norms [32]. In the Supplementary Materials (S1), we report the CONSORT (CONsolidated Standards Of Reporting Trials) chart [33, 34]. Both (cisgender) males and females were recruited. The inclusion criterion was the level of body-mass index (BMI) over 30 kg/m^2, since it is classified as obesity [35]. As exclusion criteria, we considered gastrointestinal, cardiovascular, psychiatric, and metabolic disorders, or any concurrent medical condition unrelated to obesity [35]. All participants filled out clinical questionnaires to assess the level of perceived fatigability and anxiety traits to rule out differences between groups in these domains. For details about these questionnaires, see the Supplementary Materials (S2).

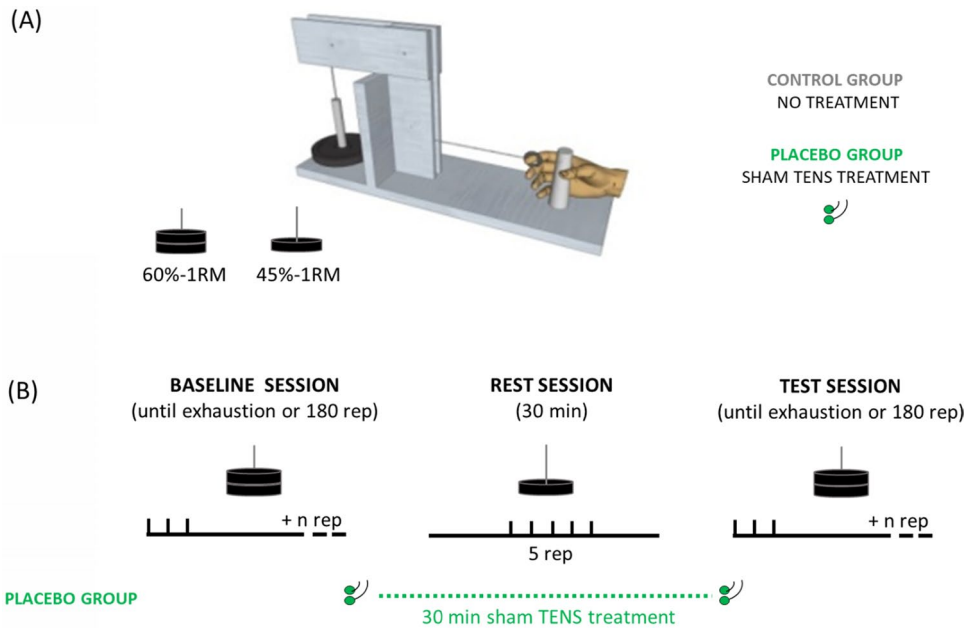

Fig. 1 Experimental setup and experimental procedure. In **A** the finger flexor device, a pulley with a load attached to one end: participants had to lift the load with their right index finger without detaching their hand from the handgrip; on the left, experimental groups. In **B** the timeline of the experiment. All participants were told that fatigue would be assessed in two sessions (baseline session and test session), with 30 min of rest between them (Rest Session). All participants were instructed to perform the motor task until exhaustion (+n rep) during the baseline and test sessions with 60% of the weight of their one (1-RM). Between sessions, participants rested for 30 min, and in the middle of the resting session they were asked to perform 5 repetitions to check for their fluctuation in the perception of fatigue. In the Placebo group this was functional to induce positive expectations on the treatment, seen that the load was reduced to the 45% of 1-RM

Experimental procedure

Participants sat on a chair with their right hand placed on a desk in front of them and were instructed to keep their eyes closed to be more focused during the task. To complete the motor task, participants used a custom-made finger flexor pulley, with a load attached to one end, already used for previous experiments on fatigue [29] (Fig. 1). The movement consisted of the flexion of the right index finger while lifting a load. Movements were self-paced (i.e., participants were free to perform the movement at their own speed), while there was an inter-movement interval of 7 s signalled by an acoustic tone. Participants were instructed to flex their right index finger to lift the weight and then to relax immediately. After each movement, participants provided a verbal rate of their perceived exertion (RPE) on a numerical rating scale ranging from 0 (indicative of the absence of fatigue) to 10 (indicative of the maximal fatigue). Movements were done until the participant reported an RPE equal to 10, having reached maximum fatigue, or if they reached the limit of 180 repetitions. Notably, participants who did not reach exhaustion within 180 repetitions were excluded, with the aim of excluding participants who did not reach adequate levels of exertion in the final data set.

Following familiarization with the experimental setup, participants determined their one-repetition maximum (1-RM), which is the maximum weight they could lift once [25]. Thus, individuals performed a single index finger flexion with 0.5 kg progressive increments, starting from 1 kg, until the weight was subjectively too heavy to be lifted. The index finger flexion was considered completed when the participant could touch the handgrip of the crafted pulley with the top of their index finger (see Fig. 1). The last successfully lifted weight represented the 1-RM. The weight to be lifted during the motor task was identified as 60% of the individual's 1-RM. After 1-RM determination, participants rested for 30 min, and EEG setup was assembled (see paragraph Electrophysiological Recordings for details). Then, participants started the experiment.

Participants performed the motor task until exhaustion, or until they reached 180 repetitions, in two different sessions: baseline session and test session. In between, participants rested for 30 min. During the resting phase, a placebo treatment was administered to the placebo group, while the control group received different instructions. Both groups, in the middle of rest, performed five repetitions of the motor task, which were functional for the placebo group to reinforce expectations on the placebo treatment [36].

Experimental groups

We conducted a single-blind study: only the participants (but not the experimenter) were unaware of which group they were assigned to. At enrollment, each participant was assigned to one of the two experimental groups (i.e., the placebo group and the control group) using a block randomization with a fixed block size of 25. The randomization sequence was computer-generated.

Placebo group. At the beginning of the rest phase, participants in the placebo group were told that a transcutaneous electrical stimulation (TENS) would be administered to expedite the recovery from high exertion. A somatosensory tactile threshold was recorded before applying the sham TENS treatment to further enhance beliefs on the effectiveness of the treatment [37]. Afterward, two sham electrodes were placed on the right dorsum of the hand connected to a stimulator (DS7 Stimulator, Digitimer Ltd), and participants were informed that the stimulator was active at a constant current below the tactile threshold, thus not perceptible. This treatment was 30 min long. After 15 min from the beginning of the sham TENS treatment, participants were asked to perform the motor task movements five times, to check how the treatment was going. The load participants lifted was surreptitiously reduced (from 60% of 1-RM to 45%), to couple the individual perception of efficacy of the sham TENS treatment to an actual physiologically reduced workload for the muscles. After five repetitions, they were instructed to continue their rest session, while they kept receiving the sham TENS treatment. Details about the verbal communication furnished to the participants are reported in the Supplementary Materials (S3). Details about the expectations checks for beliefs in treatment effectiveness are reported in the Supplementary Materials (S4).

*Control group***.** This group was used as a no-treatment group. During the rest phase, participants were invited to relax. After 15 min from the beginning of the rest phase, they were asked to lift the deceptively reduced load (45% of 1-RM) five times. Then, they continued to rest for another 15 min. Notably, no sham TENS treatment was applied, and no expectations about fatigue reduction were induced. The Control group was designed to exclude both explicit verbal expectations and implicit device-related cues (e.g., electrode application), thereby allowing us to isolate the role of expectancy induction. At the end of the entire task, all participants were informed about which group they were included (Placebo or Control) and all the experimental manipulations during the experiment.

Electrophysiological recordings

An electroencephalogram (EEG) setup (Galileo; EBNeuro, Firenze, Italy) was assembled to record the RP in an acoustically and electromagnetically attenuated chamber during the baseline session and the test session. EEG recordings were acquired through 19 scalp locations following the 10–20 international system, with linked common ears

reference. Impedance was < 5 KΩ in each active lead. Data were collected and digitized at a sampling rate of 512 Hz. The finger flexor device was connected with EEG; thus, the movement onset (i.e., load lifting) was directly inscribed in the EEG continuous data recordings. EEG continuous data were pre-processed and analysed using Matlab (Mathworks Inc., Natick, MA, USA), via EEGLab toolbox [38]. Trials were divided by sessions (baseline and test) and segmented into the number of epochs corresponding to the actual number of finger flexions performed by each participant in one session (e.g., 35 finger flexions, 35 epochs). Epochs were long 2100 ms (from 2000 ms before the movement to 100 ms after). Epochs belonging to the same session were averaged together, time-locked to the onset of the movement. The number of epochs considered for Group and Session followed closely participants' number of repetitions (control group–baseline–mean epochs: 65,68; control group–test–mean epochs: 33, 77; placebo group–baseline–mean epochs: 63,36; placebo group–test–mean epochs: 45, 46). Each epoch was baseline-corrected using a pre-movement interval from 2000 to 1500 ms as a reference. EEG epochs were low-pass filtered at a 5 Hz passband edge with EEGLAB's Basic FIR filter (new) (pop_eegfiltnew), i.e., a linear-phase, zero-phase (non-causal) FIR windowed-sinc filter with a hamming window, implemented via FFT convolution. The transition bandwidth followed the EEGLAB default (automatic), and the corresponding − 6 dB cutoff lies at 5 Hz plus half the transition width. This choice was made to attenuate alpha (8–12 Hz) activity expected in eyes-closed recordings [39]. Independent component analysis was run (ICA), and artefactual ICs were removed [40]. Thus, a mean of 3.8 ± 1.9 ICs was removed in all sessions. For each participant, we obtained two averaged RPs: one corresponding to the mean of trials of the Baseline session, and one corresponding to the test session. Electrodes over the motor cortex area (M1) and the supplementary motor area (SMA) were considered [41–43]. In particular, the analysis has been carried out considering C3 and Cz electrodes, since participants used the right hand to perform the task. For the electrophysiological statistical analysis, the mean amplitude of the RP curve was used as a dependent variable. The late component of the potential was investigated (from 500 ms before the movement to the actual onset, 0 ms) as it was shown to be the most informative, compared to its early component [29].

Experimental outcomes

For all participants, two behavioural and one electrophysiological outcomes were considered for the analyses:

(1) *Repetitions:* the number of index finger-flexion repetitions (i.e., the number of times the participants performed one repetition of the motor task until the maximal level of reported fatigue was reached) performed in each session
(2) *RPE load:* the weighted level of perceived fatigue, for each subject and session. Mean values of reported RPE were computed for each session and weighted by the associated number of repetitions. The RPE load was calculated as the mean of the reported exertion for that session (RPE) times the number of repetitions of the session (*NRS*Repetitions.*) [44].
(3) *RP amplitude:* the amplitude of the curve of the Readiness Potential for each subject and session.

Statistical analysis

All statistical analyses were conducted using Jasp software (version 0.18.3.0, JASP Team, 2024). Differences in the descriptive data were tested using an independent sample *t* test.

The level of significance was set at $p < 0.05$ for all the analyses.

Sample size. G-Power 3.1 was used to a priori identify the sample size [45]. We planned to perform a mixed ANOVA with the within-subjects factors of *Condition* (baseline vs. test) and the between-subjects factor of *Group* (placebo vs control). We chose a meaningful yet moderate effect size of 0.25, as done by other studies on the placebo effect [46–48], indicating our expectation of a significant statistical effect while recognising that it would not account for all the variance. Thus, assuming a medium effect size ($f = 0.25$) and an $\alpha = 0.05$, an overall sample size of $N = 34$ (17 for each group) is necessary to achieve a power ($1 - \beta$) of 0.80. We increased the number of participants to prevent potential dropouts due to technical issues.

Results

Demographic information and psychological questionnaires

Fifty-one participants (age in years $M = 36.05$; SD = 11.54; range = 18–56; education in years $M = 12.86$; SD = 3.07; range = 8–18; BMI as kg/m^2 M = 46.26; SD = 7.16; range = 34.83–66.85). Six participants were excluded, since they did not reach exhaustion within 180 repetitions, while one participant was excluded due to technical issues. Therefore, 44 participants with obesity were included in the final data set used for the analyses (See the CONSORT-style flow in the Supplementary Materials: S1).

The two groups were comparable in terms of their demographic and clinical characteristics (Table 1). Furthermore,

Table 1 Demographical information and psychological tests. We report the mean score (M), the standard deviation in parentheses, and the range in brackets, relative to the whole sample, as well as for the two experimental groups. We report the statistical results; in bold, significant differences if any

	Overall–mean (SD) [range]	Control–mean (SD) [range]	Placebo–mean (SD) [range]	Statistical difference
	$N=44$	$N=22$	$N=22$	
	$m=23$; $f=21$	$m=11$; $f=11$	$m=12$; $f=10$	
Age in years	36.36 (11.84) [18-56]	35.68 (10.94) [19-53]	37.04 (12.90) [18-56]	$t=0.37$; $p=0.707$; $d'=0.11$
Education in years	13.02 (3.01) [8-18]	12.18 (3.08) [8-18]	13.86 (2.76) [8-18]	$t=1.90$; $p=0.064$; $d'=0.57$
BMI	46.34 (7.28) [34.83–66.85]	47.43 (8.11) [34.83–66.85]	45.26 (6.34) [38.20–65.05]	$t=0.98$; $p=0.329$; $d'=0.29$
Chalder fatigue scale				
Total score	10.04 (4.55) [0–24]	10.04 (4.78) [0–24]	10.04 (4.43) [0–18]	$t=0.00$; $p>0.999$; $d'<0.0001$
Physical fatigue	6.09 (4.55) [0–24]	6.31 (3.52) [0–20]	5.86 (3.04) [0–12]	$t=0.42$; $p=0.674$; $d'=0.12$
Psychological fatigue	3.95 (1.76) [0–10]	3.72 (1.48) [0–6]	4.18 (2.01) [0–10]	$t=0.85$; $p=0.399$; $d'=0.25$
State anxiety (STAI Y-1)				
State anxiety	29.11 (5.77) [20–41]	28.95 (5.70) [22–40]	29.27 (5.98) [20–41]	$t=0.18$; $p=0.857$; $d'=0.05$
Trait anxiety	43.90 (9.86) [32–66]	41.27 (8.49) [32–62]	46.54 (10.60) [33–66]	$t=1.81$; $p=0.076$; $d'=0.54$
1-RM in kg	4.39 (1.31) [2.2–8.3]	4.5 (1.36) [2.5–8.3]	4.28 (1.28) [2.2–6.5]	$t=0.54$; $p=0.587$; $d'=0.17$
60% of 1-RM in kg	2.63 (0.78) [1.32–5]	2.7 (0.81) [1.5–5]	2.57 (0.77) [1.32–3.9]	

df = 42; *m* = males; *f* = females

they reported comparable scores in terms of perceived fatigue and expressions of anxiety symptoms [49–51].

Behavioral outcomes

The results from the planned 2 × 2 repeated measures ANOVA (session: baseline vs. test × group: placebo vs. control) for the Repetitions and the RPE Load were significant for the main effect of Session (Supplementary Material S5). These results could be inflated by the marked inter-individual variability observed in Baseline performances (repetitions: $M=64.5$, SD = 33.3, range 19–148). Thus, we changed our statistical approach, expressing the behavioural outcomes (Repetitions and RPE load) as percentage change (Δ%) between the baseline and test sessions, computed as

$$\Delta\% = \frac{\text{Test} - \text{Baseline}}{\text{Baseline}} \times 100$$

This approach allowed us to express improvement/decline relative to each participant's initial capacity, weighting each individual by their own baseline (high vs. low starters) and making differences comparable that would otherwise be dominated by starting level. In addition, since the task was conducted within the same experimental session for reasons of participants' availability and practicality, a general effect of fatigue was expected at the test session for both groups. The use of Δ%, therefore, permits analyses to rule out this general fatigue effect, enabling a comparison between two equally fatigued groups while highlighting the phenomenon of interest: the placebo group, although fatigued, was less fatigued than the control group. These Δ% values were used as dependent variables in one-way ANOVAs with group (placebo vs. control) as a between-subjects factor. To further corroborate our findings, we performed two ANCOVAs with test values as dependent variables, Baseline as a covariate, and Group as the between-subjects factor (Supplementary Material S5).

Raw mean data for Repetitions and RPE load are reported in the Supplementary Material (Table S1). In Fig. 2, we showed the raw data trajectory (A) and the percentage data (B) relative to the number of repetitions and the RPE load. Table 2 outlines the descriptive repetitions and RPE load absolute changes values on which the statistics have been carried out, with relative confidence intervals.

For the Repetitions and RPE Load Δ%, the Control group showed a higher motor decline in comparison with the Placebo group [F (1, 42) = 4.77, $p=0.035$, $\eta_p^2=0.10$]. Similarly, regarding the RPE load, the Control group reported a significantly higher decline in the perception of fatigue

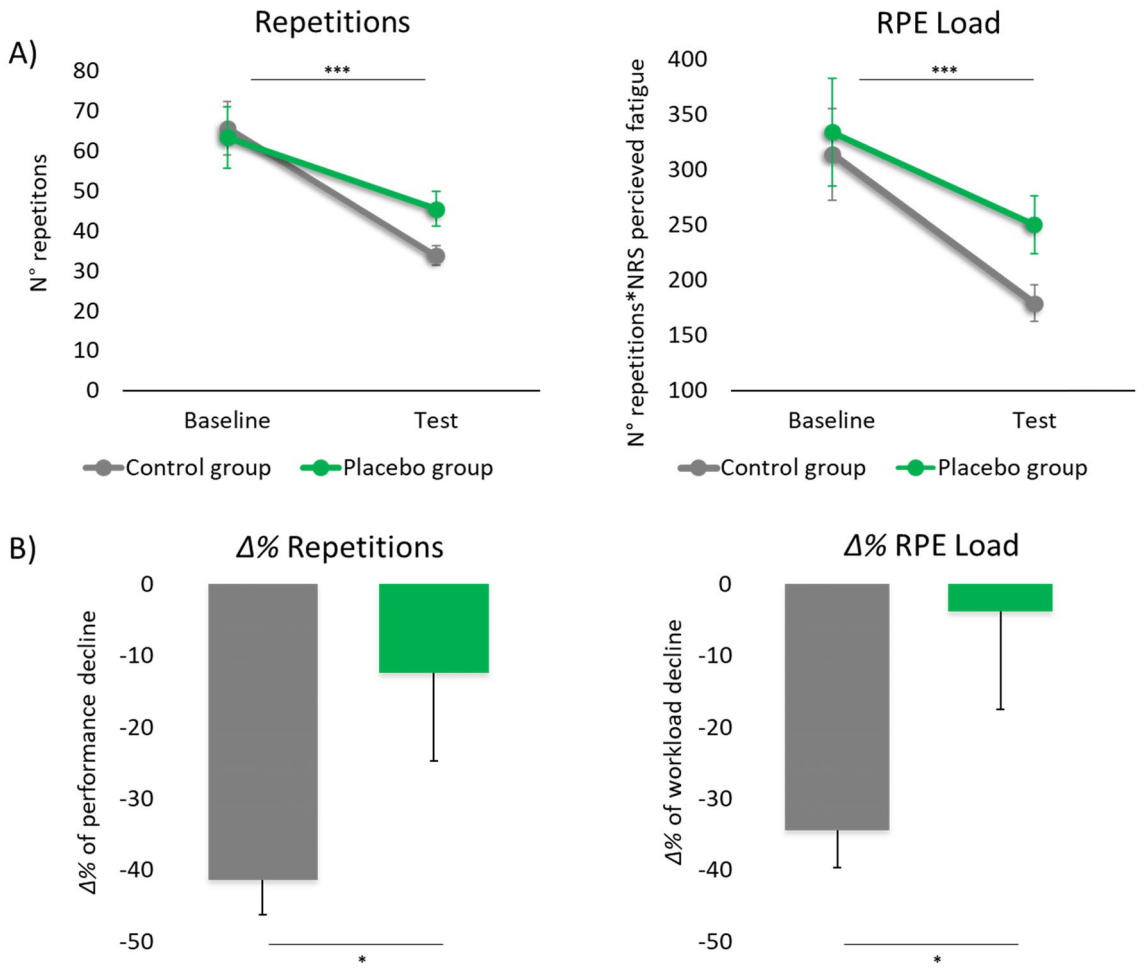

Fig. 2 Behavioral outcomes. **A** Repetitions and RPE Load represented with raw data; **B** repetitions and RPE load represented as percentage changes (Δ%). In **A**, **B** green lines and bars identify the Placebo group, grey lines and bars the Control group. Error bars represent the standard error from the mean (SEM)—*$p < 0.05$; **$p < 0.01$; ***$p < 0.001$

Table 2 Raw percentage changes values

	Δ% repetitions		Δ% readiness potential		Δ% RPE load	
	Control	Placebo	Control	Placebo	Control	Placebo
Mean	− 41.41	− 12.47	92.75	1.92	− 34.39	− 3.81
95% CI mean upper	− 31.20	13.12	166.02	27.07	− 23.49	24.81
95% CI mean lower	− 51.63	− 38.06	19.47	− 23.23	− 45.29	− 32.43

Values on which percentage changes analysis were conducted

in comparison with the Placebo group [F (1, 42) = 4.31, $p = 0.044$, $\eta_p^2 = 0.09$]. To summarize, the Control group reported a higher difference in terms of number of movements (repetitions) and perception of fatigue (RPE load) between the baseline and the test session, while this difference was lower for the placebo group, who received the placebo treatment between sessions.

In detail, we observed a significantly enhanced endurance (i.e., number of repetitions of the motor exercise) and lower levels of perceived fatigue (i.e., RPE load) when the physical activity was performed after receiving the placebo treatment. Thus, our evidence may suggest that placebo treatments positively influence the subjective experience of effort and discomfort in the case of fatigability in motor exercises in obesity, as registered in other clinical conditions [52].

Electrophysiological recordings

The results from the planned 2 × 2 Repeated Measures ANOVA (session: baseline vs. test × group: placebo vs. control) for the RP were significant for the interaction effect (Supplementary Material S5). To ensure analyses consistency, as for behavioral data, here we reported the statistical analyses in which we used the RP percentage change (Δ%)—computed as the difference between test and baseline, normalized by the absolute value of the Baseline—as main outcome. Crucially, baseline RP amplitudes could be either negative (as expected for cortical negativities) or occasionally positive; normalizing by the absolute baseline ensured that Δ% consistently represented the relative change in magnitude. As done before, we also performed an ANCOVA with test values as dependent variables, Baseline as a covariate, and Group as the between-subjects factor (Supplementary Material S5).

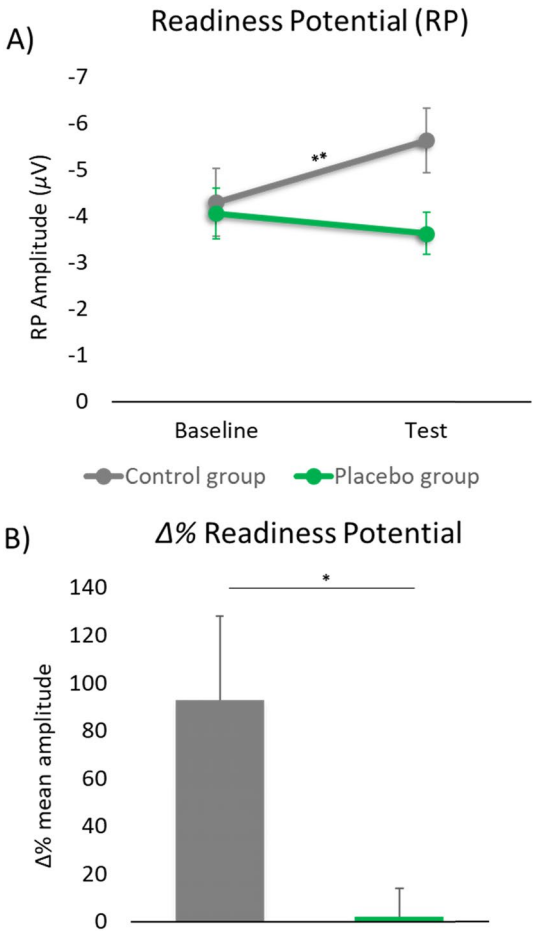

Fig. 3 Electrophysiological outcomes. **A** RP represented with raw data; **B** RP represented as percentage changes (Δ%). In **A**, **B**, green lines and bars identify the Placebo group, grey lines and bars the Control group. Error bars represent the standard error from the mean (SEM)—*$p < 0.05$; **$p < 0.01$; ***$p < 0.001$

Raw mean data for RP are reported in the Supplementary Material (Table S1). Table 2 outlines the descriptive RP absolute changes values on which the statistics have been carried out, with relative confidence intervals. In Fig. 3, we showed the raw data trajectory (A) and the percentage data (B) relative to the RP.

For the RP Δ%, we observed a significant increase in RP amplitude in the Control group compared with the Placebo group [$F(1, 42) = 5.94$, $p = 0.019$, $\eta_p^2 = 0.12$] (Fig. 3A) between the baseline session and the test session. Electrophysiological results mimic behavioral ones, and as reported in previous studies [28, 29], we confirmed the effect of the placebo treatment in modulating brain activity related to motor preparation and execution. Specifically, we observed a significant increase in RP amplitude in the Control group due to fatigue, but not in the Placebo group, where levels of RP amplitude remained unaltered when physical motor activation was performed after receiving the placebo treatment. This evidence shows a lower level of cortical activity associated with the motor task, which may be interpreted as lower perceived effort and enhanced performance. Figure 4 displays the RPs potentials and their topographies.

Thus, overall, these results demonstrate that the tested placebo treatment not only influenced the level of endurance and the subjective reports of fatigue, but it also had a measurable impact on neurophysiological markers of motor preparation and execution.

Discussion

In this research, we successfully tested a motor placebo effect on fatigue perception, motor performance, and electrophysiological responses in individuals with obesity. This study extends the amount of evidence about the beneficial effect of placebo treatment on motor performance in healthy individuals [29] and in clinical condition [28], to include obesity. Our results have crucial clinical implications. According to the 2000 American Heart Association Science Advisory [53], resistance training and aerobic training should be integrated in rehabilitative treatments for weight control. However, the psychological reactions to increased muscle pain and fatigue experienced during motor activities may significantly impact the level of participation in regular physical activity [54, 55], especially in individuals with higher fatigue perception as obesity. In this research, we provided evidence about the efficacy of a psychological intervention focused on a positive expectation on motor endurance and perception of physical fatigue, together with a distinguishable biological marker (i.e., RP). For example, during resistance training, the clinicians should take into account the complex interaction between mental and physical fatigue, and eventually propose a psychological

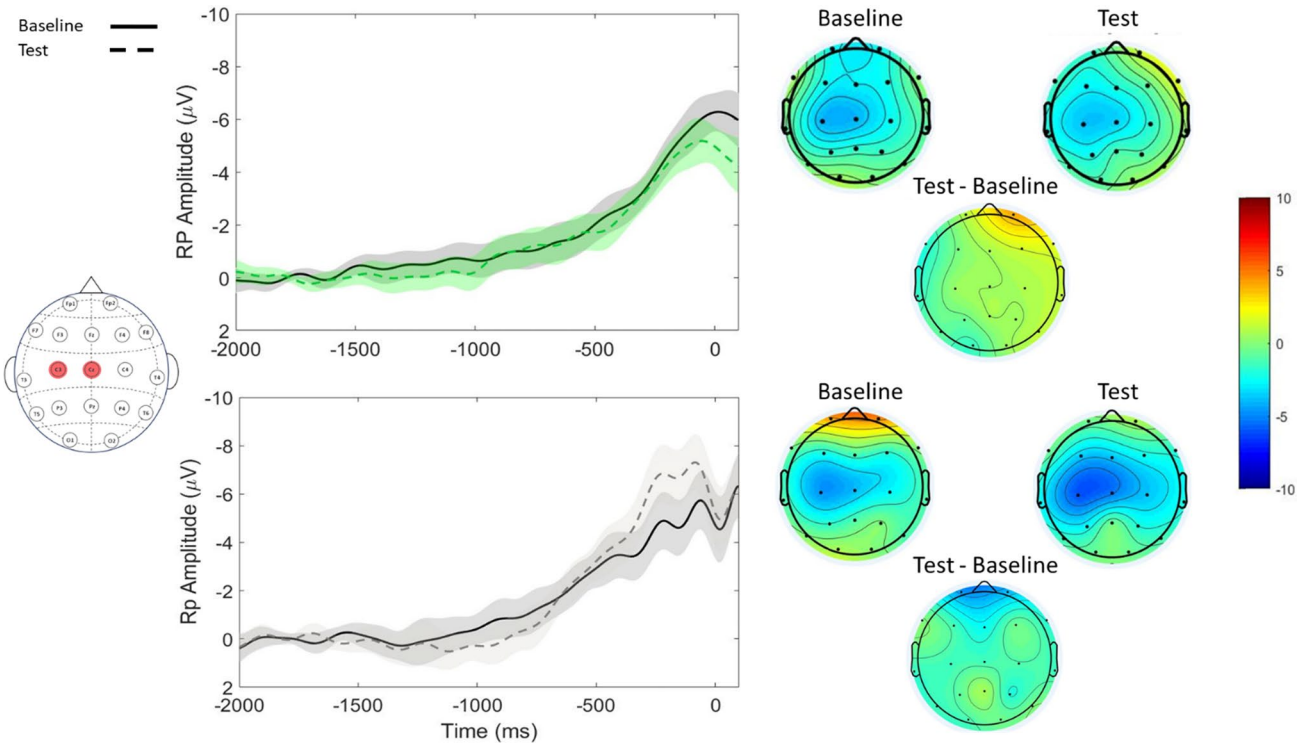

Fig. 4 Electrophysiological potentials. On the left, readiness potential for the placebo group (green) and the control group (grey), on the scalp, the selected electrodes for the analysis. On the right, Readiness Potentials topographies

intervention that may support the individuals mentally during the physical exercise. The psychological intervention, even during physical activities, would be of crucial importance in obesity. Because of social stigma [56], people affected by obesity would experience a lower level of trust and a lower level of self-efficacy about their own performance during motor exercises. The positive outcomes of placebo treatment in the motor domain could help the individual to experience the body in action during high-intensity activities, and, in turn, shape the feedforward inferences on a future approach to an exercise. In other words, placebos may be useful in modeling emotional states, motivations, and beliefs, which have been proven to modulate motor performances [57, 58]. The psychological mechanisms underlying placebo effects, such as increased motivation and altered perception of effort, could be harnessed in therapeutic settings to complement conventional treatments. Furthermore, while many of the fatigue-related challenges in individuals with obesity stem from structural and physiological factors, such as a lower strength-to-body-mass ratio, altered ventilatory mechanics, increased tendon stiffness, and elevated energy cost of basic movements [19–21], our findings suggest that central, expectation-driven mechanisms can partially override or mitigate these limitations. The placebo intervention modulated the central processing of effort and motor planning. This supports the idea that fatigue perception is not solely a reflection of peripheral deficits but is also shaped by cortical representations of effort and expected outcomes. The late RP window likely computes from SMA/pre-SMA within the cortico-striato-thalamo-cortical (CSTC) loops, where expected effort costs are integrated with action selection. A smaller late RP amplitude is consistent with a lower central effort signal (reduced predicted cost), at motor preparation [59–61]. The placebo-driven expectations are likely integrated with other processes, such as action anticipation and timing, shifting down the CSTC threshold for effort coding, treating the upcoming movement as less costly, needing less preparatory drive. Therefore, by cognitive appraisal and motivation, placebo-induced expectations may reduce the perceived cost of movement, leading to a central governor more prepared to act and less cognitively taxed, effectively improving performance even in the presence of physiological constraints. By leveraging these placebo effects, healthcare providers could develop innovative, low-cost strategies to improve the quality of life and physical functioning in obesity, as in other clinical conditions characterized by chronic fatigue (i.e., fibromyalgia, chronic fatigue syndrome, and multiple sclerosis).

While the study presents promising findings, some limitations should be addressed in future research. Gender was not included in the analyses; however, gender-related differences in obesity were reported [62, 63] with a possible

effect on fatigue perception and on the effectiveness of the placebo treatment. Analysiswise, the BMI could be considered as a factor, identifying different obesity categories. Future studies should also investigate the underlying neural mechanisms of placebo effects in greater detail, potentially using advanced neuroimaging techniques to complement the neurophysiological data. On top of this, given the nature of our motor task and the portability of new neuroimaging techniques, such as functional near-infrared spectroscopy (fNIRS), future studies should focus on designing more ecological experimental paradigms, engaging larger body districts (i.e., leg or arm movements) and the whole body (i.e., walking or running).

In conclusion, our evidence may suggest that an ergogenic placebo treatment significantly reduces perceived fatigue and enhances motor performance, together with a modulation of the brain activity related to motor preparation and execution, in obesity. Our evidence may be useful to shape future weight-loss programs, in which psychological interventions grounded on motor placebos may support the increase of the individual's levels of physical activity. Beyond the finger-flexion task employed here, the same expectancy-based mechanisms could be integrated into whole-body resistance training programs, such as leg press, cycling, or treadmill protocols. Given that placebo interventions have already proved their efficacy on healthy populations in leg press and endurance exercise, it is reasonable expected these effects also in clinical populations, as the finger-flexion exercise. Hence, in clinical contexts for lifestyle and behavioral intervention programs, pre-exercise placebo rituals or positive suggestion interventions, where the exercise load for that specific session is deceptively reduced, may enhance endurance and reduce perceived exertion, facilitating adherence to high-effort sessions. By embedding such low-cost, psychologically informed strategies within multidisciplinary rehabilitation and exercise programs, clinicians may promote sustained engagement and improve both physical and psychological outcomes in individuals with obesity.

Strengths and limits

A key strength of this study lies in its experimental design, which includes both behavioral and neurophysiological outcomes, allowing for a comprehensive assessment of placebo effects on motor fatigue in obesity. All participants completed the procedure without adverse events (such as abnormal pain due to experimental procedure, abnormal fatigue, injury, fainting, and headache) or withdrawals, suggesting the feasibility and tolerability of the experimental protocol and its applicability in clinical contexts. Furthermore, the inclusion of a Control group strengthens the internal validity by isolating the effect of the placebo treatment from spontaneous recovery or learning. It is important to note that while a sham device in the control group could have more closely matched the Placebo group in terms of contextual ritual, we deliberately avoided electrode application in the Control group to isolate the effect of expectancy induction. This design choice enabled a rigorous comparison under matched motor conditions, with the only difference being the presence of placebo-related expectations. This study presents some limitations that should be acknowledged. First, we describe a single-blinded study, where participants did not know if they were in the experimental group or the control group, while the researchers were not blinded to the participants' assignment to the groups, increasing the risk of experimental bias. However, we underline that this design recalls the clinical contexts in which double-blinded interventions are not generally performed. Nevertheless, in future research, a double-blind design should be favoured. Our sample was not stratified by obesity class, which restricts the generalizability of the findings and calls for larger and more heterogeneous cohorts in future research. We also suggest collecting a larger set of clinical information, including sleep quality, medication use, and the time of day in which the experiment is performed: all these components may impact both fatigue and RP.

The motor task involved only a single joint and lacked functional complexity, limiting its ecological validity and applicability to whole-body physical activity. In addition, because participants who did not reach exhaustion were excluded, the results pertain only to conditions eliciting fatigue; further investigations should, therefore, adopt tasks that induce more consistent fatigue states.

What is already known about this subject?

Placebo treatments can enhance motor performance and reduce perceived fatigue in healthy individuals and clinical populations, such as those with Parkinson's disease. Research has provided neurophysiological evidence (i.e., readiness potential) that reflects changes in motor effort under a placebo. However, no previous study had tested these effects in obesity, a clinical population characterised by higher fatigue perception and decreased motor activity.

What does this study add?

This study shows that individuals with obesity can respond to placebo treatments, improve endurance, and reduce their perceived fatigue, with corresponding changes in brain activity. This opens new possibilities for enhancing physical activity engagement during treatment.

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Authors contribution Conception or design of the work was performed by E.C., F.S., V.V., M.E.N. Acquisition, analysis, or interpretation of data for the work was performed by V.V., M.E.N., F.S., A.P., E.C., M.S., L.B. Drafting or revising was performed by V.V., M.E.N., F.S., E.C. Supervision and Funding was provided by F.S., A.M., E.C, A.P. All authors have approved the final version of the manuscript. All authors agree to be accountable for all aspects of the work. All persons designated as authors qualify for authorship, and all those who qualify for authorship are listed above.

Data availability The data sets generated during and/or analysed during the current study are available in Zenodo repository at (https://doi.org/10.5281/zenodo.12732213).

Declarations

Ethics approval This study was performed in line with the principles of the Declaration of Helsinki. Approval was granted by the Ethics Committee of the I.R.C.C.S. Istituto Auxologico Italiano (approval number: 2023_01_31_02).

Consent to participate Written informed consent was obtained by all individual participants included in the study.

Competing interests The authors declare no competing interests.

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