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Effects of a brief cognitive behavioral intervention and transcranial direct current stimulation on odor sensitivity: An exploratory investigation

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Abstract

OBJECTIVE: Enhanced odor sensitivity is a phenomenon that potentially underlies conditions such as multiple chemical sensitivity (MCS). Currently, there are no treatments that have been shown to effectively decrease odor sensitivity. Given similarities of odor hypersensitivity/MCS to pain sensitization disorders such as fibromyalgia, there may be a potential for interventions that improve pain tolerance to modulate odor sensitivity.

METHODS: This exploratory study randomized 72 healthy community adult volunteers to receive one of six treatments in between two assessments of thermal pain tolerance and odor threshold. Participants were randomized to receive either cathodal, anodal, or sham transcranial direct current stimulation (tDCS) aimed at dorsolateral prefrontal cortex. Additionally, participants were provided a brief cognitive behavioral intervention (CBI) for pain consisting of task framing, cognitive restructuring, and distraction technique training, or a control intervention consisting of information about pain.

RESULTS: Persons who received brief CBI showed significantly increased odor thresholds (reduced sensitivity) over the course of intervention ($F[1, 62] = 7.29, p = .009, \eta_p^2 = .11$), whereas the control intervention was not associated with altered odor thresholds. Moreover, in those that received brief CBI, more severe anxiety associated with larger reductions in odor sensitivity ($\rho = .364, p = .035$). There was no effect of tDCS ($F[2, 62] = .11, p = .90$), nor interaction between tDCS and CBI ($F[2, 62] = .32, p = .73$).

CONCLUSIONS: Given the connection between anxiety and MCS, results suggest that CBT techniques for somatic processes may show promise in treating conditions characterized by increased sensitivity to odors (e.g. MCS).

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Keywords

Odor Sensitivity; Multiple Chemical Sensitivity; Central Sensitization; Anxiety Sensitivity; Pain; Cognitive-Behavior Therapy; Brain Stimulation

INTRODUCTION

Heightened sensory sensitivity is increasingly being investigated as an important biological factor underlying a spectrum of psychiatric conditions, including autism spectrum, anxiety, trauma-related, and obsessive-compulsive related disorders (1–4). Individuals who endorse heightened sensory sensitivity report being over-stimulated by sensory stimuli (i.e. odors, lights, sounds, touch) to a degree that is distressing and/or painful (5). Specifically, one such clinical phenomenon is multiple chemical sensitivity (MCS) (6–8). Affected individuals report the ability to detect very low concentrations of common odorants, particularly harsh chemicals and environmental pollutants (at much lower than harmful levels), and that the presence of such odorants causes intense irritation and symptoms akin to panic attacks (e.g., lightheadedness, increased heart rate, difficulty breathing, headaches, concentration difficulties) (9–11). Although little research has been conducted on the prevalence of MCS, existing data finds it common, with 11-33% of the population considering themselves “chemically sensitive” (7, 8) and 1-6% of the population being impaired by the condition (6, 12).

The diagnosis of MCS remains a controversial topic, in large part due to its unknown etiology. While numerous biological (19–24) and psychological (25–32) contributing factors have been considered, more recent work describes MCS as a disorder of central sensitization (13), a state in which sensory input becomes amplified by the central nervous system. Wherein past exposure of certain odors were benign, repeated exposures lead to a myriad of adverse reactions. Chronic pain conditions such as fibromyalgia are also believed to involve a sensitized or hyperactive central nervous system, leading to an increased gain on pain and sensory processing (14). These sensitization processes also appear to play a role at the behavioral level, as studies have linked sensitivity to negative body sensations (e.g., anxiety sensitivity) with both heightened odor sensitivity (1) and chronic pain (15). Interestingly, MCS and pain regularly co-exist, as increased sensitivity to laboratory pain is reported in MCS (16) and MCS is often comorbid with fibromyalgia and other chronic pain conditions (17, 18).

Despite its prevalence and association with pain and significant functional impairment, MCS has no well-established treatments. Several studies have reported limited benefit (e.g. increased quality of life, sense of personal control) after a behavioral intervention (mindfulness-based cognitive therapy/stress reduction) but no symptom reduction (33–35). However, case reports have indicated potential for cognitive-behavioral approaches (36–38). Indeed, cognitive-behavior therapy (CBT) has shown positive evidence for reducing sensory sensitivities across other modalities including “misophonia”, a syndrome characterized by selective sound sensitivity (39–44), and pain/chronic pain conditions (45–48). In addition, numerous studies have reported efficacy in using CBT for fibromyalgia (49–57). CBT is

believed to be effective for psychosomatic pain and other sensory sensitivities because affected individuals demonstrate poor emotional associations and maladaptive coping behaviors in response to aversive sensory experiences (58). Given that people with MCS experience negative emotional responses to common odorants (59, 60), CBT aimed to correct maladaptive thoughts and emotions, as well as provide better ways to cope could be an effective treatment.

Another approach at influencing sensory processing may be to modulate emotion-related neural activity in the limbic cortex. Studies have found that minimally invasive brain stimulation targeting the dorsolateral prefrontal cortex (DLPFC) improves acute pain tolerance as well as chronic pain (61–68). Activation of DLPFC is thought to improve cognitive control over pain by modulating downstream limbic activity and emotional aspects of pain (63, 69). A previous study (from which the data for the current study were derived) found that cathodal transcranial direct current stimulation (tDCS) aimed at DLPFC, when combined with a brief cognitive behavioral intervention (CBI), was associated with a significant increase in pain tolerance in healthy adults as compared to these interventions by themselves, anodal tDCS, sham tDCS, or pain education (70). However, a lack of data exist regarding the effects of brain stimulation on olfactory sensitivity and MCS. While clinical trials for transcranial pulsed electromagnetic fields, a less intense form of brain stimulation than tDCS that does not target specific neural regions, are currently being conducted for MCS (71), available published data has focused on brain stimulation methods to improve smell acuity in persons with smell loss (72–75).

Due to the associations between MSC and pain, including neuroimaging evidence that olfactory sensitization utilizes the same brain networks responsible for pain processing (76) and the significant role that emotion plays in both pain and olfaction (77), it stands to reason that the therapeutic approaches that alter pain sensitivity may be similarly effective at modulating odor sensitivity. Thus, a subset of the healthy adults who took part in the randomized controlled trial of a brief cognitive behavioral intervention (CBI) and tDCS for acute pain tolerance (70) were recruited for this proof of concept study. The purpose of this exploratory study was to create hypotheses for future, large-scale randomized trials. However, due to our prior findings for pain, we made preliminary hypotheses that both CBI and tDCS would be associated with reduced odor sensitivity (higher threshold), and that their combination would produce the greatest effect. Also, given the purported limbic-emotional moderating effects of both CBT and tDCS, as well as evidence showing an association between odor sensitivity and stress and anxiety (1, 78), we explored the relationship between anxiety sensitivity and odor thresholds at baseline and follow-up, including whether anxiety sensitivity predicted a post-intervention change in odor detection.

METHODS

Participants

A sample of seventy-two healthy adults were recruited over a 4-month time period as part of a pilot trial of brief CBI and tDCS for acute pain tolerance (70). The sample was primarily middle-aged ($M = 30.86$, $SD = 11.47$, Age Range = 21–66), female (76.4%), and white (87.5%). Exclusionary criteria consisted of factors that would interfere with tDCS or the

validity of the pain study: (a) pregnancy, (b) history of chronic pain conditions, (c) seizures or family history of seizure disorders, (d) active suicidality, (e) implanted metal devices (e.g., pacemakers, metal plates, wires), (f) history of brain surgery or loss of consciousness for > 15 minutes, (g) prescribed medication associated with lowered seizure threshold, and (h) latex allergy. Additional exclusionary criteria for participating in the olfactory component of the study included current smokers or any problem with sense of smell (e.g. current/recent upper respiratory infection, chronic rhinosinusitis, polyps, etc.). All participants signed a written informed consent approved by the institutional review board at the Medical University of South Carolina.

Measures

Odor sensitivity was determined via the Smell Threshold Test™ (STT™ [79]). The STT™ required the systematic presentation of a set of squeeze bottles containing a serial dilution of phenyl-ethyl alcohol (PEA), a “rose-like” scent. In a single staircase method with forced choice regarding which bottle smelled stronger, a bottle containing a given concentration of PEA was presented under the nose in rapid succession with an odorless bottle. Propylene glycol served as the odorless control. Subsequent presentation of a higher or lower concentration of odorant was dependent on a correct or incorrect response from the previous trial. This method was repeated until 7 reversals (up and down the staircase) were made. Odor detection threshold score was determined by the average of the last 4 reversals. The STT™ is based upon over three decades of research, with norms based on hundreds of subjects, and 75%, 95%, and 99% confidence intervals provided for each decade of age (80, 81).

Pain testing involved the application of a thermal stimulus to the left volar forearm that increased in temperature from room temperature (32° C) at a rate of 0.25° C per second until participants could no longer tolerate the pain (see Powers et al. [70]). Participants were able to stop the thermode at peak tolerance using a handheld button, and the thermode was programmed to stop heating at 52° C to avoid the possibility of tissue damage.

Anxiety sensitivity, or the fear of experiencing anxiety and its related cognitive, physiological, and social consequences, was measured with the 18-item Anxiety Sensitivity Index-3 (ASI; 82). Using a 5-point rating scale that ranged from 0 (very little) to 4 (very much), items were rated according to how much the respondent agreed to each statement. The cognitive concerns items related to fear of the mental consequences of anxiety such as worry of “going crazy” or being “mentally ill”. The physical concerns items related to fear of anxiety-related physiological arousal including worry of a “heart attack” or “choking to death”. And finally, the social concerns items related to fear of the social aspects of anxiety such as worry of being evaluated negatively by others for blushing, sweating, or fainting. Total ASI scores were calculated by the sum of all items and could range from 0 to 72, with higher scores indicating greater anxiety sensitivity. The psychometric properties of the ASI are adequate to good on indices of reliability and validity (82, 83). In the current sample, internal consistency was good as evidenced by a Cronbach’s alpha of .88.

Procedure

The brief CBI (developed by Powers et al. [70]) was delivered via a 3-minute audio recording consisting of some key components of CBT, delivered to participants via audio recording on headphones. The first component of the intervention involved cognitive framing, which included information about the nature of the thermal pain tolerance task, information about the minimal risk of thermal stimulation to do harm, and information about the participant's capacity to control/stop the stimulation at any time. The second component consisted of cognitive restructuring, which included information and examples of negative or pain-catastrophizing thoughts, how to recognize negative thoughts, and how to change negative thoughts into positive or self-supportive thoughts regarding pain tolerance. Finally, participants were given a distraction technique, which involved encouragement to engage in tasks like slowly counting backwards from 10 to distract from the pain and to challenge impulses to stop the painful stimulation early. In contrast, those who received the control condition listened to a different 3-minute audio recording through headphones describing pain physiology (e.g., function and purpose of pain, information about transduction, transmission and modulation), gate theory of pain (e.g., information about spinal and brain stem processes involved in limiting pain signals reaching the level of perception), and central pain processing (e.g., sensory, affective, and cognitive dimensions of pain experience, information about pain processing in the reticular system, somatosensory cortex and the limbic system). The audio recordings were balanced for length and number of words, were written at an eighth grade comprehension level, and the same person's voice was used for both recordings. Experimenters were blind to which recording the participants received. A manipulation check was performed to ensure validity of the interventions, which revealed that participants were engaged and learned material unique to their condition (see Powers et al. [70]).

The tDCS session was 20-minutes long and conducted with the Phoresor-II Auto (Model PM850, Iomed, Salt Lake City, Utah, USA; this product is not labeled for the use under discussion) using 2.0mA current. We used 4×4 cm sponge electrodes soaked in sterile saline. The first electrode was placed over the left DLPFC (F3 from the EEG 10-20 system) located via the Beam F3 measurement system (84). In order to isolate the effects of directional current flow on the DLPFC, and to minimize confounding effects of potential distal brain changes associated with placement of the reference electrode over cortical areas that might impact pain perception, sensory processing, and/or cognitive processing (85), the second electrode was attached to the right shoulder. Sham tDCS consisted of turning the device on for 30 seconds to temporarily mimic tingling and skin sensations of verum tDCS and then ramped-down to 0mA for the remainder of the 20-minute session (86, 87). An attached laptop computer conducted custom-developed software, which interfaced with an ONTRAK ADU218 device that randomized participants, keeping both the participant and device operator masked to condition. The system delivered anodal, cathodal, or sham stimulation. The current density and total charge delivered according to the above parameters is consistent with those used safely in other studies (88). Participants were given vitamin-E cream to apply to the scalp following tDCS to reduce possible skin irritation. At the end of the study, participants were asked to guess whether they received active or sham tDCS. Guesses were no better than chance for all groups (see Powers et al. [70]).

After providing consent, participants completed the ASI-3, followed by a baseline assessment of their odor threshold via the STT, and then baseline pain testing. Participants were then randomized to receive either cathodal, anodal, or sham tDCS, and either CBI or a control intervention. Thus, participants were randomized to one of six conditions (groups) in a 3×2×2 design (cathodal/anodal/sham tDCS x CBI/control x time). This design could be seen as too complex for traditional treatment outcome research (89), but the purpose of the current exploratory study was to generate preliminary data for future hypothesis-driven clinical trials. The number of participants assigned to each condition, and their demographic characteristics, are shown in Table 1. After receiving their experimental intervention, participants completed follow-up pain and STT™ testing. The experiment took approximately 1 hour and participants were compensated \$50 for time and effort.

Statistical Analyses

Because the current data were collected as an exploratory study within an ongoing investigation, effect size and, in turn, sample size was determined by the main study hypotheses, namely the effects of CBI and tDCS on pain tolerance. Thus, we conducted a post-hoc power analysis to determine whether the current sample was powered adequately to detect significant group differences in odor threshold, i.e. detect medium effect sizes (Cohen's $f = .25$) given the current sample size, number of groups and measurements, and alpha level at .05. Data were also checked to ensure they met the assumptions of parametric tests. Independent-samples t -tests were used to rule out whether demographic variables influenced baseline or follow-up odor threshold scores, while factorial ANOVA determined any baseline differences in odor threshold across groups. However, even in the case where no relationships exist between demographic variables and odor sensitivity, they could still influence change over time and were thus included as covariates in the primary analytic model. In addition, a repeated-measures analysis (versus ANCOVA) was chosen to control for dependencies between measures within subjects (90) and to ensure that the analysis reflected differences in the change in odor sensitivity, rather than differences in post-intervention odor sensitivity per se (91). Thus, a 3 (sham, anodal or cathodal tDCS) x 2 (CBI or control) x time (pre- and post-intervention) repeated measures ANCOVA (age and sex as covariates) was used to examine the effects of the interventions on the change in odor threshold score. To better clarify the nature of any intervention by time interactions, group differences in odor threshold using percent change from baseline were explored with univariate ANOVA and post-hoc tests (uncorrected). And finally, associations between anxiety sensitivity and odor threshold at baseline, follow-up, and in the percent change from baseline were examined using correlation with a two-tailed test of significance. Critical alpha level was set at .05.

RESULTS

The post-hoc power analysis revealed that we achieved adequate power ($\beta = .90$). Thus, we determined that the current dataset was suitable to test the proposed hypotheses. Kolmogorov-Smirnov and Shapiro-Wilks tests revealed that odor threshold data at baseline and post-treatment, as well as ASI scores, were not normally distributed. While the odor threshold data were only non-normal in some conditions, the ASI data were consistently

positively skewed and platykurtic. Given that ANOVA is typically robust to violations of normality (92) and that the assumption of sphericity was not violated ($\chi^2 = .00, p > .999$), the data were deemed suitable for parametric testing in the main analysis.

In the overall sample of 72 adults, mean odor threshold was $-4.69 (\pm 1.24)$ at baseline and $-4.56 (\pm 1.06)$ at follow-up. There were two participants (one from the Anodal-Control group and the other from the Cathodal-CBI group) whom had baseline odor threshold scores of -2 , indicative of a significant olfactory deficit, and thus removed from subsequent analyses. Sex had no impact on odor threshold at baseline ($M_{\text{male}} = -4.89, SD_{\text{male}} = 1.29; M_{\text{female}} = -4.73, SD_{\text{female}} = 1.14; p = .63$), at post intervention follow-up ($M_{\text{male}} = -4.70, SD_{\text{male}} = 0.92; M_{\text{female}} = -4.56, SD_{\text{female}} = 1.10; p = .63$), or in percent change from baseline ($M_{\text{male}} = 0.35, SD_{\text{male}} = 21.28; M_{\text{female}} = 1.20, SD_{\text{female}} = 22.91; p = .89$). There were also no associations between age and odor threshold at baseline, follow-up, or in the percent change from baseline (all p -values $> .22$). Table 2 shows pre- and post-intervention odor thresholds for each group. There were no differences between the six groups on odor detection threshold at baseline ($F[5, 64] = .45, p = .81$), or post-intervention ($F[5, 64] = .35, p = .88$).

Results of the RM ANCOVA revealed that the CBI, but not the tDCS, intervention was associated with a change in odor threshold. Specifically, there was a significant interaction effect of CBI and time ($F[1, 62] = 7.20, p = .009, \eta_p^2 = .10$), demonstrating that odor threshold scores were significantly increased (odor sensitivity was reduced) in participants who underwent CBI (i.e. cognitive framing, cognitive restructuring, and behavioral techniques), but unchanged in participants who received the control condition (i.e. information on pain theory, physiology, and processing). Subsequent analysis of the percent change in odor threshold showed a trend-level effect of group ($F[5, 64] = 1.98, p = .09, \eta_p^2 = .13$), and significant differences between the Anodal-CBI and both the Sham-Control ($p = .02$) and Anodal-Control ($p = .03$) groups. Moreover, there were trend-level differences between the Sham-Control and both the Sham-CBI ($p = .09$) and Cathodal-CBI ($p = .06$) groups, as well as between the Cathodal-CBI and the Anodal-Control groups ($p = .09$) (see Figure 1). In contrast to the significant interaction of CBI and time, there was no interaction between tDCS and time ($F[2, 62] = .11, p = .90$), nor any interaction between tDCS, CBI, and time ($F[2, 62] = .32, p = .73$).

Because ASI scores were highly non-normal in distribution, Spearman's rank-order correlation coefficients were used. Results showed no significant associations between AS and odor threshold at baseline or post-intervention in the overall sample (all p -values $> .22$). However, severity of AS related to a greater reduction in odor sensitivity in those that received CBI ($\rho = .364, p = .035$), but was unrelated to changes in odor sensitivity in those that received the control intervention ($\rho = -.093, p = .59$) (see Figure 2).

DISCUSSION

Results of the current study indicate that brief CBI for pain tolerance, but not tDCS aimed at DLPFC, associated with reduced odor sensitivity in healthy adults. In addition, more severe anxiety sensitivity associated with greater decreases in odor sensitivity in persons who

received CBI. These results suggest that at least some features of CBT for somatic issues may influence olfactory sensitivity, particularly in persons with high anxiety sensitivity.

Indeed, anxiety sensitivity is related to the fear of aversive bodily sensations (93). Studies have shown that persons high in anxiety sensitivity are less tolerant of pain (15, 94), more perceptive of anxiety-related sensations (95), and more sensitive to threat (96), including threat-related odors (1). Yet, because PEA is a somewhat neutral odor, it is unlikely that CBI influenced participants' perceptual thresholds via their cognitive appraisals of its threat-relevance. A more likely explanation, based in part upon the highly overlapping limbic circuitry that sub-serves both emotional and olfactory processes (97, 98), is that CBI was associated with dampening of participants' overall limbic activity and thus gain on olfactory processing, particularly in those with higher baseline anxiety sensitivity. We know from our previous work that anxiety sensitivity associates with odor hypersensitivity (1), and from the work of other investigators that high rates of anxiety and psychological distress exist within MCS (25–28, 59, 99, 100). Thus, the findings from the present study lend additional support for the notion that odor hypersensitivity/MCS is linked to affective psychopathology, while also suggesting that CBT may be an effective treatment for odor hypersensitivity, especially in anxious populations like MCS.

Modulation of DLPFC activity via tDCS, while effective for pain tolerance (70), was not associated with a change in odor sensitivity. Though preliminary, this perhaps indicates that neither activation nor deactivation of DLPFC influences the detection of small-to-modest differences in odor concentration. It may also indicate that prefrontal modulation of odor and pain appear to be sub-served via distinct neural circuitry. Indeed, while studies have shown that damage to prefrontal cortex (i.e., alcoholic Korsakoff's disease) impairs odor detection (101), other work indicates that the involvement of the frontal cortices in olfaction is mostly constrained to the orbitofrontal cortex (OFC) (102–104). Yet this seemingly disparate finding for prefrontal modulation of pain and odors does not rule out the possibility that separate DLPFC and OFC pathways impinge upon, or regulate, a final common pathway underlying the core features of chronic pain, odor hypersensitivity (i.e. MCS), and other disorders of central sensitization.

While the present study was not specifically designed to determine the neural substrates of pain versus odor sensitivity, future studies should address this topic and also whether tDCS and/or CBI impact odor processing of more intense and/or noxious odors (e.g. sniff volume could replace detection as the measure of olfactory function). Several limitations to the current study should also be addressed in future investigations. First, sample size in the current study was relatively small, given that groups consisted of between 11 and 14 participants. While it would be ideal to conduct clinical trials with cell sizes of at least 30, the current pilot study was well powered to detect medium effect sizes. Nevertheless, there still could have been meaningful effects that our study was underpowered to detect. On the other hand, the fact that we tested multiple hypotheses increased the risk of Type I error. Given the exploratory nature of this study, future analyses would need to be well powered and more stringent by correcting for multiple comparisons. In addition, because the current sample was composed of healthy participants without clinically significant odor hypersensitivity, it is unclear whether CBT for somatic processes and/or tDCS aimed at

different neural circuits would be clinically robust in MCS. Along the same lines, the CBI in the current study was very brief, and while successful for reducing odor sensitivity acutely, may not have long-term therapeutic effects. It is also likely that the CBI was too brief to be deemed a treatment, and its effects on odor sensitivity may be due to other effects such as distraction. Despite these limitations however, this exploratory study lends support for a more rigorous examination of CBT as a behavioral intervention for individuals suffering from pathological odor hypersensitivity.

While future research may eventually elucidate mechanisms of change, we can conclude that cognitive-behavioral techniques for somatic processes have some initial promise for treating issues related to heightened odor sensitivity, a core feature of MCS. Thus the clinical implication of these findings is that clinicians could implement CBT techniques in an attempt to modulate odor sensitivity in persons with MCS. For example, clinicians could educate their patients that problematic odor sensitivity can be managed, as well as draw upon common CBT techniques to discourage maladaptive thoughts and problematic avoidance. The present findings may also have implications beyond MCS, extending a range of somatic issues (i.e. perceptual sensitivities) that are found across multiple forms of psychopathology including autism spectrum disorders (3), Tourette's disorder (2), obsessive-compulsive disorder (105), trichotillomania (106, 107), attention-deficit/hyperactivity disorder (108–110), posttraumatic stress disorder (111–113), panic disorder (115), generalized anxiety disorder (115), and social phobia (115).

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Abbreviations:

MCS	Multiple Chemical Sensitivity
CBT	Cognitive Behavior Therapy
CBI	cognitive behavioral intervention
DLPFC	Dorsolateral Prefrontal Cortex
tDCS	transcranial direct current stimulation
PEA	brief, phenyl-ethyl alcohol
STTTM	Smell Threshold Test TM
ASI-3	Anxiety Sensitivity Index-3
AS	anxiety sensitivity

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Figure 1.

Mean (SEM) percent change from baseline in the detection (threshold score) of phenyl ethyl alcohol (PEA), a neutral, rose-like, odor. Participants were randomized to either anodal, cathodal, or sham transcranial direct current stimulation (tDCS) aimed at the dorsolateral prefrontal cortex (DLPFC), plus a brief cognitive behavioral intervention (CBI) or a control condition. Participants who underwent CBI, regardless of tDCS condition, demonstrated a significant (or trend-level) difference in the percent change in odor sensitivity compared to participants in the pure control condition (Sham-Control). * = $p < .10$ and ** = $p < .05$

Table 1.

Group demographic characteristics

Group	Sham-Control (<i>n</i> = 12)	Sham-CBI (<i>n</i> = 11)	Anodal-Control (<i>n</i> = 14)	Anodal-CBI (<i>n</i> = 13)	Cathodal-Control (<i>n</i> = 11)	Cathodal-CBI (<i>n</i> = 11)	<i>p</i>-value
Age: <i>M</i> (<i>SD</i>)	31.33 (12.07)	26.18 (4.85)	31.79 (12.12)	31.00 (14.36)	33.82 (13.53)	30.73 (9.65)	.76
Sex (% female)	8 (66.7%)	7 (63.6%)	11 (78.6%)	12 (92.3%)	10 (90.9%)	7 (63.6%)	.34
Race (% white)	12 (100%)	10 (90.9%)	11 (78.6%)	11 (84.6%)	10 (90.9%)	9 (81.8%)	.76

CBI = brief cognitive behavioral intervention

Table 2.

Mean (SD) odor threshold scores before and after intervention across groups.

Group	Odor Threshold	
	Baseline	Post-Intervention
Sham-CBI	−4.92 (1.78)	−4.40 (1.18)
Anodal-CBI	−4.95 (0.90)	−4.34 (1.12)
Cathodal-CBI	−5.06 (0.43)	−4.65 (0.80)
Sham-Control	−4.48 (1.23)	−4.74 (1.08)
Anodal-Control	−4.58 (1.35)	−4.78 (1.14)
Cathodal-Control	−4.64 (0.96)	−4.65 (1.09)

CBI = brief cognitive behavioral intervention