Repetitive electric brain stimulation reduces food intake in humans\textsuperscript{1–3}

Kamila Jauch-Chara, Alina Kistenmacher, Nina Herzog, Marianka Schwarz, Ulrich Schweiger, and Kerstin M Oltmanns

ABSTRACT

Background: The dorsolateral prefrontal cortex (DLPFC) plays an important role in appetite and food intake regulation.

Objective: Because previous data revealed that transcranial direct current stimulation (tDCS) of the DLPFC reduces food cravings, we hypothesized that repetitive electric stimulation of the right DLPFC would lower food intake behavior in humans.

Design: In a single-blind, code-based, placebo-controlled, counter-balanced, randomized crossover experiment, 14 healthy young men with BMI (in kg/m\textsuperscript{2}) from 20 to 25 were examined during 8 d of balanced, randomized crossover experiment, 14 healthy young men with BMI (in kg/m\textsuperscript{2}) from 20 to 25 were examined during 8 d of daily tDCS or a sham stimulation. After tDCS or sham stimulation on the first and the last day of both experimental conditions, participants consumed food ad libitum from a standardized test buffet.

Results: One week of daily anodal tDCS reduced overall caloric intake by 14\% in comparison with sham stimulation. Moreover, repetitive tDCS diminished self-reported appetite scores.

Conclusion: Our study implies that the application of anodal direct currents to the right DLPFC represents a promising option for reducing both caloric intake and appetite in humans. This trial was registered at the German Clinical Trials Register as DRKS00005811.

INTRODUCTION

Food intake behavior is regulated by complex central nervous system mechanisms with reciprocal connections between the hypothalamus, brainstem, and higher cortical regions (1). In this context, the hypothalamus as the cerebral “appetite center” plays an essential role by receiving and integrating endocrine and neuronal inputs from and signaling back to peripheral organs (2). However, particularly in humans, the initiation of a meal is influenced by intentional cognitive decisions that may even override endogenous appetite regulation (3). Therefore, food intake behavior does not exclusively originate from hypothalamic regulation but is also affected by executive control processes in the brain (4) such as the dorsolateral prefrontal cortex (DLPFC) as well as the orbitofrontal cortex and anterior-cingulate cortex. Data have shown that prefrontal and frontal cortical areas are crucial for the integration of incoming sensory signals with emotional information (5). Specifically, the activity of the DLPFC is closely linked to balanced appetite regulation (6). In this context, the diminished activation of the DLPFC has been associated with a reduced ability to control food over-consumption (7) particularly in obese individuals (8). Consequently, alterations of local activity within the prefrontal cortex may result in the modulation of food intake behavior.

To increase neuroaxial excitability in humans, anodal transcranial direct current stimulation (tDCS) of the brain represents a noninvasive investigational device that acts to excite neuronal activity (9). Generally, the anodal application of direct currents to the brain results in the depolarization of cortical neurons and an increase in spontaneous neuronal activity (10). This enhancement, in turn, is directly linked to a subsequent decrease of food craving after one single stimulation procedure (11–14). Given this background, we hypothesized that the repetitive application of anodal direct currents to the prefrontal cortical brain area would reduce food intake. To test this hypothesis, we assessed food intake behavior from a standardized ad libitum buffet in 14 normal-weight men after 1 wk of daily anodal tDCS of the right prefrontal cerebral cortex.

SUBJECTS AND METHODS

Subjects

We tested 14 healthy male volunteers aged 21–28 y (mean ± SEM age: 24.81 ± 0.58 y) with BMI (in kg/m\textsuperscript{2}) from 20 to 25 (mean ± SEM BMI: 22.65 ± 0.34). Before participation, a structured questionnaire assessed the self-reported sleep quality and typical sleep-wake cycle. Volunteers with disturbances in sleep continuity, shift workers, individuals with an average sleep duration <7 h/night, and sleep-onset latency >30 min as well as volunteers who regularly went to bed later than 0000 during the 4 wk before the experiments were not included in the study. Moreover, all volunteers completed the Three-Factor Eating Questionnaire about current dietary practices that measures the following 3 aspects of eating behavior: cognitive restraint, desinhibition, and hunger (15). Only volunteers with scores ≤6-5-4, which corresponded to low cognitive restraint, low desinhibition, and normal susceptibility to hunger scores, were included in this study. Exclusion criteria were taking any kind of medication, acute and chronic medical diseases, diabetes in first-degree family members, alcohol or drug abuse, smoking, and participation in competitive sports. In the days before the experiment, each volunteer gave written informed consent. Participants were asked to fast for 6 h before testing and go to

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bed not later than 2300 before and during the experimental testing period. The study was approved by the Ethics Committee of the University of Luebeck and carried out in accordance with the Declaration of Helsinki (2000) of the World Medical Association.

Study design

Participants were examined during the 8 d of daily tDCS or a sham stimulation. They were not allowed to take naps on the day before experiments. The study was performed in a single-blind, counterbalanced, randomized, sham-controlled, crossover design. Each volunteer was tested during 2 experimental conditions, which comprised daily tDCS for 8 d and a daily sham stimulation for the same period of time, respectively. Conditions were spaced ≥2 wk apart. The washout interval was $2.47 \pm 0.72\ wk$ on average (range: 2–4 wk).

On days of experimental testing, subjects reported to the Department of Psychiatry and Psychotherapy at 1530. Afterward, body weight was measured by using a personal floor scale with approval for medical use (MPS-M; KERN & SOHN GmbH). Thereafter, an anodal electrode was placed over the right DLPFC. The position of the electrode was defined as an area 5 cm anterior to the motor cortex target of the left first interdigital muscle, which was identified via focal transcranial magnetic stimulation before the experiment. The cathodal electrode was positioned over the left forehead (supraorbital). Both tDCS electrode sheaths (pad size $5 \times 7\ cm = 35\ cm^2$) were soaked with a standard saline solution (NaCl 0.9%) and fixed with elastic bands. A direct current stimulator plus (neuroConn GmbH) delivered 20 min of anodal stimulation (1 mA; fade in/out: 8 s). The setup was identical for the sham stimulation except without any current flow. The administration of direct currents to the brain was code based.

At 1730 on the first and last days of both experimental conditions, a standardized buffet was offered during which participants were allowed to eat ad libitum during the subsequent 40 min. Volunteers were told that the main intention of the study was to investigate effects of tDCS on mood. Therefore, participants were kept unaware of the hypothesized treatment effects on food intake behavior and were not aware that their respective food intake was quantified by weighing buffet components before and after food consumption. To prevent overeating, subjects were allowed to take any remaining food after the final food weighing. The composition of the test buffet is shown in Table 1. Furthermore, participants were asked “How is your feeling of hunger?” and were requested to rate their hunger on a 10-point rating scale [from 0 (not hungry) to 9 (very hungry)] (16). In addition, participants were asked “How is your appetite for food?” [ nonspecific appetite (ie, appetite for all types of food irrespective of the taste categories)], “How is your appetite for sweet foods?” (specific appetite for sweet foods), and “How is your appetite for

<table>
<thead>
<tr>
<th>TABLE 1 Composition of the offered free-choice test buffet</th>
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<td><strong>Food</strong></td>
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<tr>
<td>Brown bread</td>
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<tr>
<td>Whole-grain bread</td>
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<td>White bread</td>
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<td>Butter</td>
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<td>Poultry sausage</td>
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<td>Salami sausage</td>
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<td>Semihard cheese</td>
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<td>Spread cheese</td>
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<td>Cream cheese</td>
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<tr>
<td>Ham</td>
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<tr>
<td>Scrambled eggs</td>
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<tr>
<td>Iceberg lettuce</td>
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<tr>
<td>Salad dressing</td>
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<tr>
<td>Tomato/gherkin</td>
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<tr>
<td>Rissole</td>
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<tr>
<td>Mustard</td>
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<tr>
<td>Tomato sauce</td>
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<tr>
<td>Vanilla pudding</td>
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<tr>
<td>Chocolate pudding</td>
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<tr>
<td>Fruit salad</td>
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<tr>
<td>Peanuts</td>
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<tr>
<td>Marble cake</td>
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<tr>
<td>Chocolate cookies</td>
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<tr>
<td>Chips</td>
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<tr>
<td>Gummi bears</td>
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<tr>
<td>Orange juice</td>
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<tr>
<td>Apple juice</td>
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<td>Whole milk</td>
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<td><strong>Total</strong></td>
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savory foods?” (specific appetite for savory foods), respectively, and were requested to rate their perceived appetites on a 100-mm visual analog scale. Appetite and hunger ratings occurred before the tDCS or sham stimulation of the brain and before food consumption on the first and the last experimental days of both conditions.

Statistics

Data are presented as mean values ± SEMs. Statistical Package for the Social Sciences software (IBM SPSS Statistics 22) was used to conduct an ANOVA for repeated measurements that included the factors treatment (tDCS compared with sham stimulation), day (day of data collection), time (prestimulation compared with poststimulation), macronutrients (carbohydrates compared with proteins compared with fat), and the interaction effect between these factors. For pairwise comparisons, paired Student’s t test was used. P < 0.05 was considered significant. P between 0.05 and 0.100 was considered a trend.

On the basis of the existing tDCS literature, a sample size of 14 was identified adequate to detect significant treatment differences on appetite in a placebo-controlled, counterbalanced, crossover study (11–14). To choose an appropriate sample size for a valid statistical detection of a clinically meaningful difference in caloric intake, a statistical power calculation with assumptions made for this calculation derived from data of previous studies that investigated effects of brain stimulation by using intranasal insulin application on food intake behavior (see, eg, references 16–20) was done before data were collected. A medium effect size (d) > 1.09 indicated a large effect according to Cohen was considered sufficient for our study. Following this assumption, a total of ≥14 subjects was considered sufficient to detect medium-sized within-subjects effects with a probability of 1-β > 90%, which has been assumed to be adequate in experimental studies.

RESULTS

Effects of tDCS on food intake are shown in Figure 1. On day 1, total food intake did not differ between conditions (P = 0.699; Figure 1A). After 8 d tDCS, the total calorie consumption was 14.2% lower than after the same period of sham stimulation (P = 0.016). In addition, caloric intake after 8 d of tDCS was diminished compared with measures in both conditions on day 1 (tDCS on day 8 compared with tDCS on day 1: P = 0.027; tDCS on day 8 compared with sham stimulation on day 1: P = 0.022; overall ANOVA: P-treatment × day interaction = 0.032), whereas there were no differences in caloric intake within the sham condition (P = 0.724).

Macronutrient comparisons indicated that the reducing effect of 8 d of tDCS on total calorie consumption was particularly attributable to diminished food intake in the form of carbohydrates (tDCS on day 8 compared with tDCS on day 1: P = 0.048; tDCS on day 8 compared with sham stimulation on day 1: P = 0.010; tDCS on day 8 compared with sham stimulation on day 8: P = 0.001; overall ANOVA: P-treatment × day interaction = 0.011;
In contrast, carbohydrate consumption did not change within the sham condition ($P = 0.152$). Generally, tDCS had no effect on the caloric intake in forms of proteins ($P$-treatment effect = 0.651, $P$-day effect = 0.167, and $P$-treatment × day interaction = 0.767; Figure 1C) and fat ($P$-treatment effect = 0.382, $P$-day effect = 0.275, and $P$-treatment × day interaction = 0.203; Figure 1D). However, there was a significant influence of direct current stimulation on macronutrient ingestion in general ($P$-treatment × macronutrients interaction = 0.011). Additional analyses revealed no impact of tDCS on the macronutrient composition of food taken home after the buffet tests (all $P > 0.110$).

Self-rated nonspecific appetite and specified for sweet and savory foods on days 1 and 8 of experiments are shown in Figure 2. A–C. On day 1, there were no differences between conditions for any of the items (nonspecific appetite: $P = 0.734$; appetite for savory foods: $P = 0.391$; appetite for sweet foods: $P$-treatment effect = 0.653; all $P$-treatment × time interactions > 0.177, Figure 2, A–C). However, ANOVA analyses revealed a significant time effect for the items nonspecific appetite ($P = 0.017$) and appetite for savory foods ($P = 0.032$) but not for appetite for sweet foods ($P = 0.407$). Overall, after the first stimulation on day 1, ratings of nonspecific appetite were higher after tDCS than pretreatment ratings ($P = 0.038$; Figure 2A). This was also the case for ratings of appetite for savory foods ($P = 0.010$; Figure 2C). In contrast, there was no difference between scores in terms of appetite for sweet foods between prestimulation and poststimulation ratings on day 1 in the tDCS condition ($P = 0.640$; Figure 2B). In the sham condition, prestimulation and poststimulation ratings of appetite for savory and sweet foods showed no differences ($P = 0.531$ and $P = 0.241$, respectively). However, we observed a trend for an increase after the sham stimulation in terms of nonspecific appetite on day 1 ($P = 0.056$; Figure 2A).

On day 8, ANOVA interaction analyses revealed that tDCS decreased scores for nonspecific appetite as well as for sweet and savory foods compared those with the sham stimulation (nonspecific: $P = 0.016$; appetite for savory foods: $P = 0.006$; appetite for sweet foods: $P$-ANOVA treatment × time interaction = 0.033, Figure 2, A–C). After 1 wk of intervention, comparison between appetite ratings before the final brain stimulation revealed that volunteers rated comparable scores for all items in both experimental conditions (all $P > 0.172$; Figure 2, A–C). Ninety minutes after the stimulation period, participants showed higher scores for nonspecific appetite compared with pretreatment ratings in the sham condition ($P = 0.015$; Figure 2A). This increased nonspecific appetite after the sham stimulation was reflected in terms of savory foods ($P = 0.014$; Figure 2C) but not sweet foods ($P = 0.112$; Figure 2B). However, there was no stimulation effect within the tDCS condition (nonspecific

**FIGURE 2.** Mean (± SEM) appetite and hunger ratings after tDCS compared with sham stimulation. Values are shown of ratings for nonspecific appetite (A), appetite separately for sweet (B) as well as savory (C) foods, and feelings of hunger (D) before (no pattern) and after (coarse pattern) stimulation. Appetite and hunger ratings were analyzed by using 2- and 3-factor ANOVA including Bonferroni corrections as well as a paired Student’s t test; $P < 0.05$ was considered significant. Data showed a significant interaction effect between the factors treatment (tDCS compared with sham stimulation) and time (prestimulation compared with poststimulation) in the case of nonspecific appetite (A) and appetite for savory (C) and sweet (D) foods on day 8. The 3-factor ANOVA showed a significant interaction between the factors treatment (tDCS compared with sham stimulation), day (days of data collection), and time (prestimulation compared with poststimulation) in the case of nonspecific appetite (A) and appetite for savory foods (C). Means with different lowercase letters were significantly different at $P < 0.05$ (paired Student’s t test). Means labeled “a,b” indicated no difference to all means labeled “a” and “b,” respectively. Besides, on day 1, ratings of nonspecific appetite tended to be higher after sham stimulation than pretreatment ratings ($P > 0.05$ and < 0.1 (A)). $n = 14$. tDCS, transcranial direct current stimulation.
between conditions (tDCS: 75.14 kg; sham stimulation: 75.46 kg; 6 kg, respectively; Figure 2A and C), but had no effect on appetite for savory foods (P-ANOVA treatment × day × time interaction = 0.160; Figure 2B).

The ANOVA analysis revealed that tDCS had no influence on feelings of hunger on day 1 (P-treatment × time interaction = 0.283; P-treatment × time interaction = 0.512; Figure 2D). Generally, feelings of hunger significantly increased after the stimulation irrespective of the condition (for t test comparisons: DCS, P = 0.010; sham stimulation, P = 0.007; P-ANOVA time effect = 0.003). Moreover, there was no difference between conditions in terms of hunger on day 1 (both conditions: prestimulation, P = 0.883; poststimulation, P = 0.151).

On day 8, ANOVA analyses indicated that there was no impact of repetitive tDCS on hunger (P-treatment effect = 0.912; P-treatment × time interaction = 0.165). As on day 1, we observed that feelings of hunger increased after the sham stimulation on day 8 (P = 0.023), whereas there was a trend for an increase after tDCS (P = 0.063 as revealed by using a t test comparison (Figure 2D); P-ANOVA time effect = 0.011). This consistent increase of hunger may have been attributable to the long fasting period during experiments. Overall, there was no difference in hunger on day 8 between conditions in terms of prestimulation (P = 0.556) and poststimulation (P = 0.667) scores. The 3-factor ANOVA that comprised both conditions and time points showed that 8 d tDCS had no influence on feelings of hunger (P-ANOVA treatment × day × time interaction = 0.158).

**Body weight**

On day one, the body weight of the volunteers did not differ between conditions (tDCS: 75.14 ± 1.71 kg; sham stimulation: 75.46 ± 1.74 kg; P = 0.110). After 1 wk of intervention, comparison between participants’ body weights revealed similar values (tDCS: 75.29 ± 1.86 kg; sham stimulation 75.50 ± 1.85 kg; P = 0.117). Overall, ANOVA analyses indicated that there was no impact of repetitive tDCS on body weight (P-treatment × day interaction = 0.530).

**Side effects**

Local sensations that occurred at the anode or cathode site during tDCS were skin redness (n = 9), tingling (n = 4), itching (n = 7), and feelings of skin burning (n = 2). Sensations during the sham stimulation were skin redness at the anode or cathode site (n = 8), tingling (n = 1), and itching (n = 8). All sensations were transient and ranged from mild to moderate. Additional analyses revealed that participants were not able to differentiate between the 2 study conditions (ie, 6 participants considered tDCS as real and the sham stimulation as a sham stimulation, 5 volunteers considered tDCS as a sham condition and vice versa, sham stimulation as tDCS, 2 participants identified both conditions as tDCS, and one volunteer perceived both conditions as sham stimulation [OR (sham stimulation/real stimulation): 1.333; 95% CI: 0.301, 5.912; OR (for cohort treatment = no): 1.167; 95% CI: 0.524, 2.597; OR (for cohort treatment = yes): 0.875; 95% CI: 0.438, 1.750; Pearson’s chi-square: P = 0.705; likelihood ratio: P = 0.705; Fisher’s exact test (2-sided): P = 1.000].

**DISCUSSION**

The results showed that 8 d of sham-controlled anodal prefrontal tDCS reduced food consumption in humans. This outcome is in line with previous data that indicated that a single application of tDCS enhanced the self-reported ability to resist food incentives (12) and acutely reduces snack ingestion (11). Moreover, our data showed that 1 wk of repetitive tDCS distinctly decreased total calorie consumption. Differential analyses revealed that this effect was mainly attributable to reduced carbohydrate intake, suggesting that anodal tDCS not only generally dampens appetite but, moreover, influences the preference for specific nutrients.

However, the physiologic mechanisms that underlie these effects could not be derived from our human experimental approach. Notwithstanding, there has been evidence that alterations in regional brain activity, specifically within the DLPFC, may add to this effect (11). The DLPFC, as one part of the prefrontal cortex, is involved in the processing of reward perception, motivation, and decision making (21). This brain area, moreover, has previously been associated with food reward and satiety (22) and is at least responsible for the forecast of future consequences and inhibitory control over inappropriate impulsive behavior because of an instantaneous feelings of reward (23). Consequently, it can be speculated that anodal tDCS activates neuronal networks within the DLPFC and, therefore, inhibits neuroaxial signaling between the orbitofrontal and anterior cingulate cortex that are both associated with cognitive control over food intake behavior, which, in turn, leads to reduced calorie consumption. Alternatively, the suppressing effect of tDCS on food consumption may be a consequence of the stimulation of mesolimbic dopaminergic pathways, which have previously been described after the activation of the DLPFC through repetitive transcranial magnetic stimulation (24). In this context, it is known that the dopaminergic system modulates appetitive motivational processes associated with food intake (25), wherein food deprivation increases rewarding properties of food (26). In our study, participants were asked to fast for 6 h before testing (ie, they were food-deprived for 7.5 h when food was presented). Therefore, it seems reasonable to assume that tDCS enhanced cerebral dopamine secretion, and the observed reduction in food intake was attributable to an accelerated achievement of satisfactory reward perception and, therefore, overall reduced appetite. In contrast, recent studies showed that the cerebral energy status plays a crucial role in food intake regulation because the brain’s high-energy phosphate content is a predictor for subsequent food consumption (16) (ie, high-energy phosphate amounts relate to low caloric intake and vice versa). Therefore, an alternative explanation for the reduced food consumption in the current study could have been that tDCS enhanced brain energy amounts and, therefore, reduced the amount of subsequent free-choice food consumption. This reasoning has been supported by recent data that showed a boosting effect of tDCS on the cerebral high-energy phosphate content after an initial decrease (27).
Compatible with our findings at the behavioral level, our data showed that, compared with the sham stimulation, the daily application of tDCS over 8 d reduced sensations of appetite by exerting a preventive effect on the physiologic, time-related increase in self-reported appetite scores, whereas subjective feelings of hunger remained unaffected. These findings confirm and extend previous observations of tDCS diminishing the desire to eat (13) as well as cravings for sweet foods (12, 14) and carbohydrates accompanied by the enhancement of self-reported ability to resist food incentives shortly after the stimulation (12). The absence of an effect on feelings of hunger, in turn, is in line with the previous observation that stimulation of the DLPFC by repetitive transcranial magnetic stimulation reduced cue-induced food craving independent of hunger perception (28). In contrast, our data did not reveal any influence of tDCS on appetite perception on the first experimental day, which seemed to contradict the aforementioned results. This discrepancy may be explained by differences in the electrode position as well as the intensity of the applied direct currents. In our study, we administered currents of 1 mA to minimize potential side effects, whereas double currents were used in previous studies (12–14, 29). In addition, although an electrode montage with the anode over the left DLPFC and the cathode over the contralateral DLPFC occurred in previous studies, we positioned the anode over the right DLPFC and the cathode over the left supraorbital area. Hence, it may be assumed that the lower current intensity over the right DLPFC may have resulted in delayed responses. Nonetheless, all of these findings point to a boosting influence of tDCS on cognitive control over the brain’s appetite network.

At first glance, the finding that self-reported appetite and hunger scores increased from prestimulation to poststimulation query during the sham stimulation condition mostly on day 8, whereas there was no effect in this regard in the verum condition, which was surprising. However, this finding was not as surprising as it appeared when we took into account that participants were food deprived for 6 h before and 7.5 h after the stimulation session. Therefore, the observed increase in appetite and hunger scores can easily be explained by physiologically elevated food cravings because of a long fasting period in the sham condition. Notwithstanding, this explainable drive for food consumption after so many hours of fasting was completely suppressed by tDCS, which represented a stimulation effect per se.

There were some limitations of our study that must be addressed. One limitation was the lack of food intake control before experiments. However, the waiving of food intake control via questionnaires was necessary to draw attention away from food intake behavior. Therefore, to confirm our results, they require further testing in larger studies that include female volunteers, follow-up testing, and the examination of food intake behavior before experiments.

From a clinical point of view, our finding of a tDCS-induced decrease in consumed calories by >14% as well as a self-reported lower appetite makes tDCS a particularly interesting research target with respect to obesity. There has been evidence that this disease is linked to structural reductions within the prefrontal cortex and ventral striatum (30, 31). A more-recent study showed regional reductions of gray-matter volume within the DLPFC in obese individuals (32). On the basis of the knowledge that brain morphometry correlates with brain activation (33), it appears plausible that such structural lowering is accompanied by disinhibited appetite regulation. In addition, data comprising different BMI categories, from underweight to obese, have shown an inverse relation between the cerebral energy content and BMI (34), thereby implying that the pathogenesis of obesity is associated with a reduced high-energy phosphate status of the brain. In this context, both tDCS-related activation of the DLPFC and its enhancing effects on cerebral energy may rebound to body-weight reduction in overweight and obesity. However, it must be pointed out that the current results were obtained in healthy, young, normal-weight men and, therefore, cannot be directly generalized to individuals with obesity. Hence, it will be of utmost importance to explore whether similar effects of tDCS on food intake behavior and appetite exist in obese humans.

In conclusion, our study suggests a boosting influence of tDCS on cognitive control over the brain’s appetite network that results in reduced self-reported appetite scores and a diminished total calorie consumption and affects the preference for specific foods in healthy, young, normal-weight men and, however, cannot be directly generalized to individuals with obesity. The translation of these results into the treatment of obesity is still a long way out and needs additional research.

The authors’ responsibilities were as follows—KJ-C: is the guarantor of this work and, as such, had full access to all data in the study and took responsibility for the integrity and accuracy of the data analysis; KMO: received funding; KJ-C and KMO: conceived and designed the study; KJ-C, AK, MS, and NH: collected data and performed data analyses; KJ-C, US, and KMO: interpreted data and wrote the manuscript; and all authors: edited and approved the final manuscript. None of the authors had a conflict of interest.

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