1   **Modulating dream experience: Noninvasive brain stimulation over the**

2   **sensorimotor cortex reduces dream movement**

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Abstract

Recently, cortical correlates of specific dream contents have been reported, such as the activation of the sensorimotor cortex during dreamed hand clenching. Yet, the causal mechanisms underlying specific dream content remain largely elusive. Here, we investigated how alterations in the excitability of sensorimotor areas through transcranial direct current stimulation (tDCS) might alter dream content. Following bihemispheric tDCS or sham stimulation, participants who were awakened from REM sleep filled out a questionnaire on bodily sensations in dreams. tDCS, compared to sham stimulation, significantly decreased reports of dream movement, especially repetitive actions. Contrary to this, other types of bodily experiences, such as tactile or vestibular sensations, were not affected by tDCS, confirming the specificity of stimulation effects. In addition, tDCS reduced interhemispheric coherence in parietal areas and altered the phasic electromyography correlation between the two arms. These findings reveal that a complex reorganization of the motor network co-occurred with the reduction of dream movement, confirming spatial specificity of the stimulation site. We conclude that tDCS over the sensorimotor cortex causally interferes with dream movement during REM sleep.

Keywords

Dreaming; motor processing; sensorimotor cortex; REM sleep; transcranial direct current stimulation.

Introduction

Dreams are vivid, often emotionally intense and narratively complex experiences occurring in sleep. In our dreams, we feel immersed in alternative worlds and have the experience of interacting with other persons and objects. Often this involves the subjective experience of moving through the dream world, and movement is among the most frequently reported dream experiences, second only to visual imagery. Yet these rich subjective experiences stand in stark contrast to the outward unresponsiveness and lack of observable behaviour during sleep. This study aimed to investigate the causal mechanisms underlying dream movement and bodily experience in dreams by using tDCS over sensorimotor areas. While most existing studies of the neural underpinnings of bodily experience in dreams and dream movement are correlational, our approach allowed us to manipulate dream content and draw conclusions about its underlying causes.

Specifically, our goal was to characterize the role of sensorimotor cortex in the generation of bodily sensations in dreams. We aimed to experimentally inhibit motor and other bodily experiences as an important aspect of self-simulation in dreams through bihemispheric transcranial direct current stimulation (tDCS) during REM sleep. After awakening from REM sleep, subjective dream experience was examined through the collection of dream reports and a questionnaire specifically designed to investigate bodily experiences in dreams; neural measures were obtained through electrophysiological sleep data.

This experimental protocol was guided by theoretical and empirical considerations. Our focus on bodily experience was motivated by the centrality of self-experience and subjective presence to dreaming (Strauch and Meier 1996; Occhionero et al. 2005; Speth et al. 2013). The immersive structure of dreaming is foregrounded in simulation theories (Revonsuo et al. 2015), in which dreams are described as mental simulations characterized by the experience

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72   of a virtual world. Typically, this virtual world is centered on a virtual self and experienced

73   from an internal first-person perspective. The dream self is typically described as actively

74   engaged in dream events, and movement is reported in 75% of dreams (Strauch and Meier

75   1996; Cicogna and Bosinelli 2001). This immersive *here and now* quality is regarded as a

76   defining characteristic of dreaming. It is also striking that with few exceptions, both the

77   virtual world and the virtual self in dreams are experienced as real. Simulation views

78   advocate the idea that "being-in-a-dream" feels the same as "being-in-the-world" during

79   wakefulness. Moreover, bodily experience and movement sensations appear to be central to

80   the feeling of subjective presence both during the waking and dream state, and sensorimotor

81   interaction modulates subjective presence both in real and virtual environments (Sanchez-

82   Vives and Slater 2005).

83   Our focus on bodily experience was further guided by findings suggesting high-level activity

84   of the motor cortex during REM sleep (Hobson 1988; Maquet et al. 2000; Dang-Vu et al.

85   2005). Generally, REM sleep dreaming has been associated with relative deactivation of

86   executive networks and frontal areas, and with high levels of activity in sensory, motor, and

87   emotional networks as compared to wakefulness (Schwartz and Maquet 2002; Nir and

88   Tononi 2010; Cipolli et al. 2017). Studies focusing on the neural correlates of specific types

89   of bodily dream experiences have shown the sensorimotor cortex to be activated during hand

90   clenching in dreams (Dresler et al. 2011), and the right superior temporal sulcus, a region

91   involved in the biological motion perception, to be activated in dreams with a sense of

92   movement (Siclari et al. 2017). Furthermore, smooth pursuit eye movements during tracking

93   of a visual target are highly similar during waking perception and lucid REM sleep dreaming

94   (LaBerge et al. 2018). Taken together, these studies suggest a remarkable isomorphism of the

95   neural mechanisms underlying motor control in wakefulness and dreaming. However, the

96   correlative nature of these studies limits their potential to uncover the causal contribution of

97   specific brain regions to dream content.

98   Older studies attempted to experimentally induce different kinds of dream experience via

99   peripheral and bodily stimulation during sleep. Causal manipulations that have been shown to

100 have an effect on dream content include vestibular stimulation in rotating chairs (Hoff 1929;

101 Hoff and Pötzl 1937) or hammocks (Leslie and Ogilvie 1996); light flashes or sprays of water

102 applied to the skin (Dement and Wolpert 1958); thermal stimulation (Baldridge et al. 1965;

103 Baldridge 1966); tactile stimulation via a blood pressure cuff inflated on the leg (Nielsen

104 1993; Sauvageau et al. 1998); and olfactory stimulation (Schredl et al. 2009). The frequency

105 of stimulus incorporation in dreams is variable and dependent both on the kind of stimulus

106 and the sensory modality. Particularly high incorporation rates were achieved in studies using

107 blood pressure cuff stimulation (40-80%) (Nielsen 1993; Sauvageau et al. 1998). This method

108 of causally manipulating dream content is promising. However, because the processing of

109 external and peripheral stimuli is attenuated in REM sleep, the precise effect of sensory

110 stimulation on dream content is often nonspecific and unpredictable.

111 As a more direct method for manipulating dream content that avoids the possibly distorting

112 effect of reduced sensory processing during REM sleep, we previously suggested using tDCS

113 (Noreika et al. 2010). We argued that this method might complement previous attempts to

114 manipulate dream content through sensory and bodily stimulation in sleep. Unihemispheric

115 tDCS has been shown to facilitate motor imagery during REM sleep (Speth and Speth 2016)

116 and to modulate visual imagery during Stage 2 NREM sleep (Jakobson, Fitzgerald, et al.

117 2012a), but not during slow wave sleep (Jakobson, Fitzgerald, et al. 2012b) or REM sleep

118 (Jakobson, Conduit, et al. 2012). Furthermore, frontal tDCS increases lucidity in experienced

119 lucid dreamers (Stumbrys et al. 2013); and frontal transcranial alternating current stimulation

120 (tACS) increases dissociation, insight and control in novice lucid dreamers (Voss et al. 2014).

121   tDCS has also been reported to modulate mind wandering in wakefulness (Axelrod et al.

122   2015). This is promising, as dreaming has been proposed to be an intensified form of mind

123   wandering, based on phenomenological and neurophysiological similarities (Fox et al. 2013).

124   Here, we applied tDCS over the sensorimotor cortex, aiming to understand its causal role in

125   dream content generation. Since tDCS modulates neural processes associated with motor

126   imagery during wakefulness (Quartarone et al. 2004; Matsumoto et al. 2010; Feurra, M. et al.

127   2011), we expected a similar effect during REM sleep. However, instead of planned

128   facilitation of movement sensations in dreams with unilateral anodal tDCS (Speth and Speth

129   2016), our stimulation protocol was designed to interfere with motor processing during sleep,

130   enabling a more focused analysis of the electrophysiological mechanisms underlying dream

131   movement. Given that unilateral cathodal tDCS does not disrupt motor imagery during REM

132   sleep (Speth and Speth 2016), we adopted a bihemispheric tDCS protocol, which is known to

133   interfere with cortical and cerebellar motor networks more effectively than unilateral tDCS,

134   particularly when applied during the resting state (Lindenberg et al. 2013, 2016).

135   To investigate possible effects of bihemispheric tDCS on outward muscular activity, we

136   obtained electromyographic (EMG) measures from the arms. REM sleep is typically

137   characterized by near-complete muscle atonia (Pompeiano 1967) and a partial blockade of

138   sensory input (Hobson 1988; Wu 1993). At the same time, subtle muscular activity in the

139   form of twitching is frequent in REM sleep and may play a role in the development and

140   maintenance of motor behaviour (Blumberg 2015). A relation to dreaming seems plausible,

141   but remains incompletely understood (Windt 2018).

142   We hypothesized that if the sensorimotor cortex has a causal role in generating sensorimotor

143   dream content, bihemispheric tDCS over the sensorimotor cortex during REM sleep should

144   attenuate movement and other bodily experiences in dreams reported immediately after timed

145   awakenings in the laboratory. To test this hypothesis, we developed an empirically informed

146   questionnaire focused specifically on bodily sensations in dreams. This allowed us to probe

147   bodily experiences more systematically than the more common methods of content analysis

148   or quantitative linguistic analysis of dream reports (Speth and Speth 2016). Furthermore, we

149   hypothesized that bihemispheric tDCS during REM sleep would interfere with

150   interhemispheric motor networks as well as with spontaneous peripheral muscle activity,

151   which are possible neural pathways to the reduction of dream movement.

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Methods and Materials

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Methods outline.

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156   The study protocol consisted of a recruitment and screening session, an MRI session, and two

157   sleep sessions on non-consecutive nights (see Figure 1A). In addition, a TMS assessment of

158   motor cortical excitability took place on the evening of the first sleep session. Ten

159   participants were awakened from REM sleep two or three times per night and asked to give

160   free dream reports and to answer to the Bodily Experiences in Dreams (BED) Questionnaire,

161   which targeted the dream immediately preceding awakening (see Figure 1B). Participants

162   received sham-stimulation during REM sleep on one night and bihemispheric tDCS on the

163   other night. Bihemispheric tDCS montage included a cathode placed over the left

164   sensorimotor cortex and an anode placed over the right sensorimotor cortex (see Figure 1C).

165   In addition to standard polysomnography, central and peripheral electrophysiological data

166   were recorded using 16 EEG channels and 4 EMG channels measuring flexor and deltoid

167   muscles in both arms.

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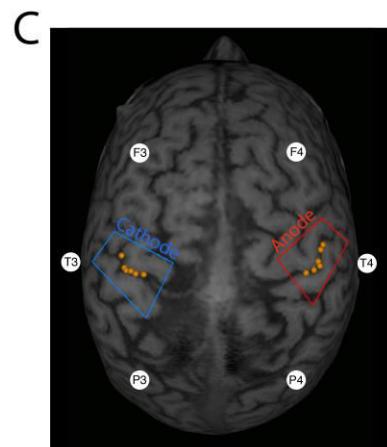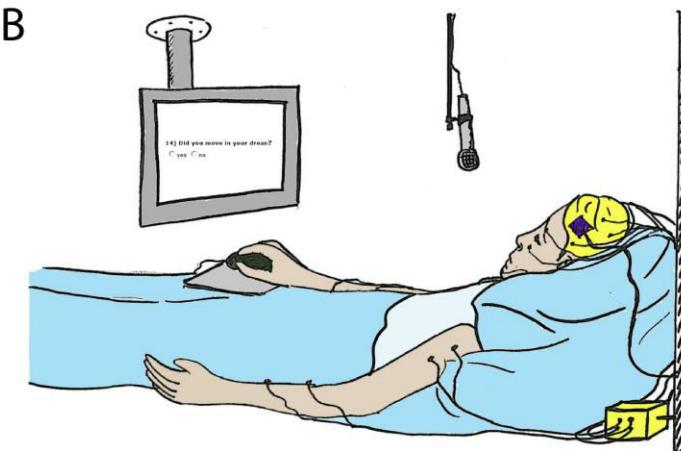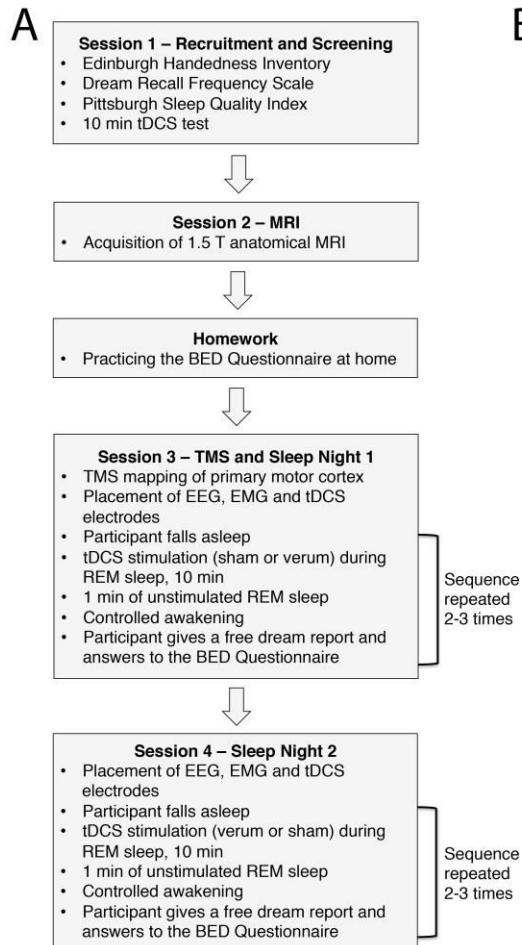
Figure 1 | Experimental design. (A) Time course of the study. **(B)** Experimental setup during sleep sessions. **(C)** Primary sensorimotor hand areas of a representative participant. Orange dots indicate stimulation sites where TMS pulses induced a subjectively experienced hand movement and/or muscle twitch (located approximately at the central sulcus between the somatosensory and somatomotor cortices). The blue box drawing over the left hemisphere represents the cathode tDCS electrode placement site, and the red box drawing over the right hemisphere represents the anode electrode placement site. White circles depict the approximate location of 6 electrodes used for the EEG inter-hemispheric coherence analysis.

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Participants.

Aiming to recruit 10 right-handed individuals with high dream recall frequency and good sleep quality, potential participants were screened with the Edinburgh Handedness Inventory

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182   (Oldfield 1971) and the Dream Recall Frequency (DRF) scale (Schredl 2002), which assesses

183   the frequency with which people are able to remember dreams at home. The DRF scale

184   consists of a single question "How often do you remember your dreams?" and 7 possible

185   answers: 0=never, 1=less than once a month, 2=about once a month, 3=twice or three times a

186   month, 4=about once a week, 5=several times a week, and 6=almost every morning.

187   Furthermore, potential participants filled in the Pittsburgh Sleep Quality Index (PSQI)

188   (Buysse et al. 1989). We aimed to recruit individuals whose global PSQI score did not exceed

189   4 (with 0 indicating no sleep difficulty and 21 indicating severe difficulties in sleep) and

190   whose sleep latency score indicated they typically needed less than 30 minutes to fall asleep.

191   Given that the application of tDCS may occasionally induce itching, tickling, heat sensations

192   under the electrodes, or even a temporary headache (Priori 2003), we introduced potential

193   participants to the tDCS technique before they made their final commitment to take part in

194   the study. After screening for MRI and tDCS contraindications, they were given the

195   opportunity to familiarize themselves with the tDCS procedure before spending their first

196   night in the laboratory. Participants were stimulated for 10 min with tDCS of 1 mA current

197   over the C3 and C4 electrode sites according to the 10-20 EEG system (approximately over

198   the sensorimotor cortex), which helped them decide whether they wanted to participate in the

199   actual experiment. This also helped minimize the risk that tDCS during REM sleep would

200   lead to awakening.

201   After screening 16 potential participants, we were able to recruit 10 healthy right-handed

202   university students (4 men and 6 women, mean age 26.8, range 4.4 years). The mean

203   handedness index was 0.9 (SD=0.11; range 0.73 to 1). The mean DRF score was 5.4

204   (SD=0.79, Min=4, Max=6), indicating high spontaneous dream recall. While this might

205   introduce bias towards high recallers' dreams, it is arguably the most feasible recruitment

206   strategy for a costly and time-consuming sleep laboratory study. All participants gave their

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207   written informed consent according to the Declaration of Helsinki, and the protocol of the

208   study was approved by the Ethics Committee of the Hospital District of Southwest Finland.

209   Participants were financially compensated with 40 euros per night and 10 euros per hour for

210   daytime testing.

211

212   MRI-TMS mapping of the primary sensorimotor hand area.

213   ECoG measurement of the electric field induced by tDCS in a human patient as well as

214   computational modelling of tDCS effects in healthy participants suggest that the spatial

215   focality of tDCS decreases if stimulation electrodes are misplaced by >1cm (Opitz et al.

216   2018). Thus, aiming to constrain between-participant variance of the stimulation focus below

217   1cm, the location of the hand area in the primary sensorimotor cortex in both hemispheres

218   was determined individually for each participant with the help of magnetic resonance

219   imaging (MRI) and transcranial magnetic stimulation (TMS). Anatomical brain images were

220   acquired with a 1.5 T MRI scanner Philips Intera at the Turku PET Centre. 3D models of the

221   brain were created using 3D T1-weighted MR sequence. A hospital radiologist confirmed

222   that the brain MRI was normal in all cases. Afterwards, the approximate location of primary

223   sensorimotor hand representations was visually determined from anatomical brain images

224   based on macro-anatomical landmarks (Yousry et al. 1997).

225   Based on this analysis, the location of the primary sensorimotor hand area was determined for

226   each participant in a separate TMS session, which was carried out on the evening of the first

227   experimental night at the Department of Psychology at the University of Turku. TMS pulses

228   were delivered using eXimia™ TMS stimulator with NBS navigation system (Nexstim Ltd.,

229   Helsinki, Finland), which allowed us to navigate within individual anatomical MRI with an

230   approximately 6-mm spatial resolution containing all sources of errors (Ruohonen and Karhu

231 2010). Participants sat on a reclining chair with their eyes closed and both arms supported by

232 a pillow to ensure that their arm muscles were relaxed. TMS was carried out in a single pulse

233 mode using a figure-of-eight-shaped coil that was held tangentially against the participant's

234 head. The current direction of the second phase of the biphasic pulse was oriented

235 perpendicularly to the post-central gyrus in the posterior to the anterior direction at the bank

236 between pre-central and post-central sulci (Richter et al. 2013) (see Figure 1B).

237 First, a rough location of the hand area was estimated by asking participants to report whether

238 they experienced any hand movement following a TMS pulse over the motor cortex in the

239 contralateral hemisphere. Once a reliable hotspot was found, an individual motor threshold,

240 i.e. the minimum TMS intensity required to induce the subjective experience of a hand

241 movement, was determined with the maximum likelihood threshold hunting (MLTH)

242 procedure (Awiszus, 2003). In this process, 20 pulses were delivered to the hand area with

243 different stimulus intensities, starting at 60% of maximal TMS intensity. The mean motor

244 threshold was 56.1% (SD=12.4, Min=28, Max=76.7) of maximal TMS intensity for the left

245 hemisphere, and 59.2% (SD=16.5, Min=24.8, Max=77.12) for the right hemisphere. While

246 motor thresholds did not differ systematically between the hemispheres (paired samples t test:

247 $t(9)=1.02$, $p=0.34$, Bf in favor of the null=2.2), there was a strong inter-hemispheric

248 correlation of motor thresholds (Pearson correlation: $r=0.82$, $p=0.004$).

249 Following estimation of individual motor thresholds, the most ventral and caudal points of

250 the hand representation in the primary motor cortex were estimated by delivering TMS pulses

251 with the intensity of 10% above the level of the individual motor threshold. This procedure

252 was consecutively performed for both hemispheres, yielding bilateral hand representation

253 maps that were later used to place tDCS electrodes (see Figure 2B).

254

255 **tDCS over the primary sensorimotor cortex during REM sleep.**

256 tDCS and sham-stimulation sessions were conducted in the Sleep Laboratory of the Centre

257 for Cognitive Neuroscience at the University of Turku over two non-consecutive nights with

258 each participant. Microprocessor-controlled programmable 1-channel Eldith DC-Stimulator

259 PLUS (Electro-Diagnostic & Therapeutic Systems GmbH, Ilmenau, Germany) was used as a

260 stimulation device.

261 tDCS was applied bilaterally to the hand area in order to modulate the excitability level of the

262 primary sensorimotor cortex during REM sleep. Participants were asked to avoid caffeine for

263 6 hours and alcohol and other CNS-affecting drugs for 24 hours prior to the experiment. To

264 ensure these requirements were met, participants filled out the custom-made Pre-Sleep

265 Questionnaire before each session. For each participant, the two stimulation sessions were

266 separated by at least one week in order to avoid interference effects.

267 Two 35 cm^2 sized sponge-covered rubber electrodes were soaked with water, and Ten20

268 electrode paste (Weaver and Company) was applied on both sides of the sponge. The

269 electrodes were placed bilaterally along the central sulcus posterior to the primary motor

270 hand areas, which was determined with the help of MRI-guided TMS (see Figure 1B). They

271 were supported with a comfortable bandage throughout the night. tDCS was carried out on

272 one experimental night and sham-stimulation took place on another night. Participants were

273 blind to the experimental conditions, i.e. whether the tDCS session was followed by the sham

274 session (N=5) or vice versa. An equal number of participants was assigned randomly to each

275 condition.

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277 During the tDCS night, 1mA electric current was delivered to participants' scalp two or three

278 times per night for 10 min during REM sleep, starting with the second sleep cycle. It has been

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279 reported that changes in current direction may result in qualitatively different motor effects,

280 with cathodal stimulation being more effective and largely inhibitory and anodal stimulation

281 being less effective and largely facilitatory (Nitsche et al. 2008). Furthermore, tDCS induced

282 neuroplasticity may accumulate over time (Nitsche and Paulus 2000). In order to keep the

283 stimulation effects consistent throughout the night, the electrode over the right sensorimotor

284 area was always the anode, and the electrode over the left sensorimotor area was always the

285 cathode. This procedure ensured that the asymmetric stimulation during one sleep cycle

286 would not interfere with or cancel stimulation effects during another cycle. We chose to place

287 the cathode over the dominant left hemisphere with the aim to disrupt dream movements.

288

289 During the sham-stimulation night, stimulation was conducted by switching on the DC device

290 and stimulating only for 10 sec each at the beginning and end of a 10 min period during REM

291 sleep. Stimulation that lasts only a few seconds has been shown to produce a minimal effect

292 on the brain, if any (Hummel et al. 2005). The aim of sham stimulation was to mimic the skin

293 sensation that is occasionally experienced during the onset and offset of tDCS. This

294 procedure is thought to make the two conditions subjectively indistinguishable (Gandiga et

295 al. 2006). The same procedure was repeated two or three times starting with the second sleep

296 cycle.

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298

299 **Electrophysiological recordings.**

300 To record EEG activity, 16 electrodes (Fp1, Fp2, F7, F3, Fz, F4, F8, T3, T4, T5, P3, Pz, P4,

301 T6, O1, O2) were placed on the scalp following the standard 10-20 system (Jasper 1958). C3,

302 Cz and C4 electrode locations were left empty for the placement of tDCS electrodes. To

303 record eye blinks and vertical saccades, two electrooculography (EOG) electrodes were

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304   placed below and above the left eye, while two other electrodes placed adjacent to the lateral

305   canthi of each eye were used to measure horizontal saccades. An electromyography (EMG)

306   electrode placed on the chin was used to record muscle tone, which was used for the scoring

307   of sleep stages. The reference for all these electrodes was placed on the right ear mastoid and

308   the ground electrode was placed on the temple. In addition, two bipolar EMG channels were

309   used to record muscle activity in the right and the left arm flexor digitorum profundus, which

310   were later used to analyze peripheral motor activity. Another two EMG channels recorded

311   activity of the deltoid muscles in both arms. Electrophysiological recordings were

312   continuously monitored on a computer screen and all electrodes were regularly checked

313   throughout the night to ensure that the impedance remained under 5 kΩ. All data were

314   recorded at 500 Hz sampling rate with Ag/AgCl electrodes using NeuroScan amplifier

315   SynAmps Model 5083. Given that tDCS onset induces a slow frequency artifact in the EEG

316   that may preclude online polysomnographic scoring, a 1-Hz high-pass filter was applied

317   during recording for online monitoring of sleep stages (Marshall 2004). As expected, tDCS

318   onset- and offset-induced artifacts always faded away after 5-10 sec.

319

320   **Collection of dream reports.**

321   One minute after the termination of tDCS or sham-stimulation, participants were awakened

322   from REM sleep with a standard awakening tone. They were then asked to give a verbal

323   dream report of "everything that was going through their minds before awakening", aiming to

324   facilitate dream recall. Afterwards, participants were asked if they remembered anything else

325   about their dream. To avoid a possible bias between stimulation conditions, these questions

326   were played from a pre-recorded computer audio file. Following the free dream report,

327   participants were asked to fill in the Bodily Experiences in Dreams (BED) Questionnaire.

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328   The questionnaire was designed as an internet survey programmed on www.webropol.com

329   and was projected on a screen above the bed in the sleep laboratory. Participants navigated

330   and responded to the BED Questionnaire by controlling a mouse while lying in bed.

331   Participants were stimulated and awakened two or three times per night, depending on how

332   many REM sleep periods they had. The number of awakenings was balanced across the first

333   and the second night and across the two stimulation conditions (see Table S3). White dream

334   reports (i.e. cases when a person reports the occurrence of dream experiences but cannot

335   recall any specific details) as well as sleep mentation reports (i.e. when a person reports non-

336   perceptual subjective experiences, such as thinking) were excluded from the analysis. A total

337   of 50 dreams reported during a total of 20 nights were available for analyses.

338

339   **Bodily Experiences in Dreams (BED) Questionnaire.**

340   The 41-item BED Questionnaire was designed to gather detailed information about

341   kinaesthetic and other bodily experiences in dreams (see Appendix 1). The BED

342   Questionnaire consists of 5 general questions with respective sub-scales (see Table 1). Each

343   of the general questions targets a particular category of body-related experience: vestibular

344   sensations, tactile and somatosensory experiences, movement, movement alterations, and

345   body schema alterations. Each general question, if answered positively, is followed by sub-

346   scales targeting more specific instances of this category of experience. For example, if a

347   participant indicated that they had experienced movement sensations, they would then be

348   asked about the occurrence of specific types of movement sensations, such as single,

349   repetitive, and passive movements. In addition, participants were asked whether the reported

350   sensation concerned the whole body, the right or left hand, the right or left side of the face, or

351   another body part (see Appendix 1). If they answered negatively, they would skip to the next

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352   general category. Depending on whether a sub-scale asked about the intensity or the duration

353   of experience, 9 point Likert-scales for answering ranged either from "1=Low intensity" to

354   "9=High intensity" or from "1=Never" to "9=Throughout".

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356

357   **Table 1.** The BED Questionnaire: General questions and exemplary sub-scales

Five general questions (Yes/No)
5. Did you experience any tactile or somatosensory sensations in your dream?
11. Did you experience any vestibular or balance sensations in your dream?
14. Did you move in your dream (including active as well as passive movements (for instance in a vehicle) of the whole body or body parts)?
18. Were your movements (either of the whole body or of certain body parts) altered or impaired compared to wakefulness?
26. Was your dream body or were certain body parts altered compared to wakefulness?
Movement sub-scales (from 1=Never to 9=Throughout)
15. How frequently did you move in your dream (including active as well as passive movements (for instance in a vehicle) of the whole body or body parts)?
16. How frequently did you perform the following types of movements in your dream?
16.1 – single actions (e.g. placing a book on the table)
16.2 – repetitive actions (e.g. running)
16.3 – passive movements (e.g. going by car)

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361   **Word count of verbal dream reports.**

362   The length of dream reports was assessed by two blind judges (authors JW and KV), who

363   independently calculated the meaningful word count of each dream report. Murmurs,

364   repetitions of words, and any secondary reflections or comments about the dream were not

365   included in the word count. The judges initially agreed on the word count of 47 out of 50

366   dream reports (94% agreement). The judges discussed the reasons for the mismatch in the

367   remaining 3 cases and reached an agreement.

368

369   **Content analysis of movement sensations in verbal dream reports.**

370   Following the findings from the BED Questionnaire, we carried out content analysis of verbal

371   dream reports, focusing on the specific types of movement (single actions, repetitive actions,

372   passive movement) performed by the dream self. To compare the type and frequency of

373   movements reported in the BED Questionnaire to those explicitly mentioned in dream

374   reports, two blind judges (authors VN and BL) carried out a content analysis of verbal

375   reports. First, the judges scored whether each dream report contained at least one movement

376   produced by the dream self, excluding facial movements such as talking, drinking, and

377   blinking, as we reasoned that individuals do not typically consider facial musculature when

378   asked to report their movements. Movements attributed to the first-person plural "we" were

379   treated as involving movements of the dream self. Second, the judges identified individual

380   movements produced by the dream self in each dream that, in the first step, was judged to

381   contain movement. Third, they scored the type of the identified movements (single action,

382   repetitive action, passive movement). All three stages of the content analysis were first

383   carried out individually and the obtained results were then compared between the judges. In

384   the case of disagreement, the judges discussed it until an agreement was achieved.

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385   Regarding the presence or absence of movement in a given report, the judges initially agreed

386   on 45 out of 50 dream reports (90% agreement). After discussion, the judges agreed that the

387   remaining 4 dreams contained references to movements produced by the dream self, while

388   one report had no explicit references to such movement. Regarding individual movements,

389   judges initially agreed on the identification of 33 movements, and disagreed on 19

390   movements (63.5% agreement). The disagreement was caused by one judge either missing a

391   movement or treating it as part of a longer sequence of movements, e.g. treating walking

392   from A to B and from B to C as a single movement. After discussion, the judges agreed that

393   the dream reports contained a total of 48 individual movements executed by the dream self.

394   Regarding specific types of movements (single action, repetitive action, passive movement),

395   the judges initially agreed on 44 out of 48 movements (91.7% agreement). After discussion,

396   the judges agreed that the remaining 4 movements should be scored as follows: "diving" -

397   single action, "riding a bike downhill" - passive movement, "writing something" - repetitive

398   action, "made some coffee" - single action.

399

400   **EEG analysis: coherence and spectral power.**

401   To assess the electrophysiological effects of tDCS on brain functioning, we analyzed the full

402   period of 1 min of EEG signal recorded between the termination of tDCS or sham-stimulation

403   and controlled awakening. tDCS artifacts did not contaminate this EEG interval whilst sleep

404   scoring ensured that REM sleep continued up to the point of awakening. On one occasion, a

405   spontaneous awakening took place before the planned controlled awakening, and only 7 sec

406   of stimulation-free sleep EEG were available for analysis. On another occasion, a

407   spontaneous awakening took place immediately after the termination of stimulation; this

408   recording was excluded, leaving 49 EEG recordings available for analysis.

409 Continuous recordings were first high-pass (0.5 Hz) and then low-pass (45 Hz) filtered using

410 a FIR filter as implemented in EEGlab toolbox (Delorme and Makeig 2004). The data were

411 then common average referenced, and excessively noisy periods of recording were manually

412 deleted (an average of 743 ms per single recording). Detached or excessively noisy channels

413 were deselected (an average of 0.2 channels per dataset), and an independent component

414 analysis (ICA) was carried out on the remaining channels, using EEGlab toolbox (Delorme

415 and Makeig 2004). Independent components reflecting eye movements and other sources of

416 noise were manually deleted (an average of 3.3 ICs per recording), following which dropped

417 noisy EEG channels were interpolated using spherical spline interpolation. Continuous EEG

418 recordings were epoched into 2-sec segments with a 50% overlap between adjacent segments.

419 Several epochs that still contained visible artifacts were manually deleted (an average of 0.5

420 epochs per recording). Individual epochs were demeaned across the whole 2 sec interval.

421 We analyzed EEG inter-hemispheric coherence in the beta oscillation range (15-30 Hz) at the

422 electrodes adjacent to the stimulation site (F3, F4, T3, T4, P3, P4). Magnitude-squared

423 coherence was computed in the range from 2 Hz to 44 Hz with a maximum frequency

424 resolution of 2 Hz between pairs of EEG channels adjacent to the stimulation site from the

425 frontal (F3-F4), temporal (T3-T4) and parietal (P3-P4) side, using Brainstorm toolbox (Tadel

426 et al. 2011). Coherence values obtained at a single 2 sec segment level were averaged across

427 beta frequency range (15.6-29.3 Hz). Next, coherence values were averaged across each 1-

428 min pre-awakening recordings. Afterwards, individual means were averaged over several

429 awakenings for each participant according to the experimental condition, yielding 10 tDCS

430 and 10 sham-stimulation values for each electrode pair.

431 In the case of a significant difference between tDCS and sham-stimulation conditions across

432 the 1-min pre-awakening periods, coherence was computed at four separate 15 sec sub-

433 intervals preceding controlled awakening: -60 to -46 sec, -45 sec to -31 sec, -30 to -16 sec,

19

434   and -15 to -1 sec. A significant difference between tDCS and sham conditions observed

435   immediately after the termination of stimulation (-60 to -46 sec) was expected to reflect a

436   tDCS-driven modulation of EEG activity, as an effect size of neurophysiological changes

437   following motor tDCS decreases with increasing time (Nitsche and Paulus 2000). Contrary to

438   this, a significant difference between tDCS and sham-stimulation conditions at the interval

439   preceding awakening (-15 to -1 sec) with no difference at the -60 to -46 sec interval was

440   expected to reflect an unspecific modulation of EEG activity, e.g. micro-arousals caused by

441   tingling sensations could eventually trigger body movements in bed.

442   To control for a possible confound of EEG spectral power on coherence computation

443   (Bowyer, 2016), we carried out a control analysis of EEG beta power. Spectral power was

444   computed across 2 sec epochs using Hilbert transform, set from 1 Hz to 44 Hz in steps of 1

445   Hz, for the same set of 6 electrodes adjacent to the stimulation site. Power values obtained at

446   a single 2 sec segment level were averaged across beta frequency range (15-30 Hz), with

447   subsequent data averaging steps repeating coherence analysis.

448

449   **Phasic EMG analysis.**

450   We investigated the effects of tDCS on peripheral muscle tone by analyzing EMG activity

451   from the left/right arm flexor and deltoideus muscles during the 1 min interval between the

452   termination of tDCS or sham-stimulation and the controlled awakening of participants. We

453   were specifically interested whether EMG traces following tDCS and sham-stimulation

454   showed increased phasic muscle activity compared to the pre-stimulation baseline window,

455   and whether bihemispheric tDCS modulated interaction between the left/right arm EMG.

456   Since phasic EMG activity manifests during REM sleep as short-lasting muscle bursts

457   recorded by surface electrodes (Fairley et al. 2012), we split the 1-min epochs into 60 non-

20

458   overlapping 1-sec segments and carried out a binary assessment whether each segment

459   contained phasic EMG activity. Segments with phasic EMG activity were then assigned a

460   value of one, segments without phasic EMG activity a value of zero. The mean overall 60

461   binary values were then used to define the ratio of phasic EMG activity within the respective

462   epoch.

463   More specifically, since phasic EMG activity is reflected in broadband spectral power

464   changes, we used the variance of gamma band (50-250 Hz) power for the detection of short-

465   lasting bursts of muscle activity. In a first step we high-pass filtered the raw data with a 3rd

466   order butterworth filter with a cutoff-frequency at 50Hz (Suppl. Fig. 1 a-b). For the

467   subsequent time-frequency analysis, we used a single-tapered spectral analysis method

468   (Percival and Walden 2000) with a time window of 50 ms and 10-ms time steps. The relative

469   power changes were then calculated by dividing the time-resolved amplitude for each

470   frequency bin by the frequency-specific average of the whole 1 min epoch (Suppl. Fig. 1 c).

471   After splitting the epochs in 1-s segments, the variance of relative power was calculated for

472   each segment and every frequency. The variance of gamma band power was then defined as

473   the mean over all frequencies between 50 Hz and 250 Hz.

474   To assess a relative shift towards more phasic/tonic activity in response to stimulation, the

475   variance of gamma band power was calculated both for the 60 sec epochs after the

476   termination of tDCS or sham-stimulation and for a 30 sec baseline time window before tDCS

477   or sham-stimulation. The relative variance of gamma band power was then calculated by

478   dividing the variance by the averaged variance in the baseline time window (Suppl. Fig. 1 d).

479   This way, post-stimulation segments with the variance of gamma band power higher than the

480   corresponding average (median) in the stimulation-free 30 sec baseline time window received

481   a relative variance value greater than one and were defined as segments shifting towards

482   phasic EMG, while segments with a relative variance between zero and one were defined as

483   segments shifting towards tonic EMG. Finally, a proportion of phasic segments was

484   calculated across the whole 60 sec post-stimulation epoch, yielding values ranging from 0,

485   indicating a complete shift towards tonic EMG, to 1, indicating a complete shift towards

486   phasic EMG.

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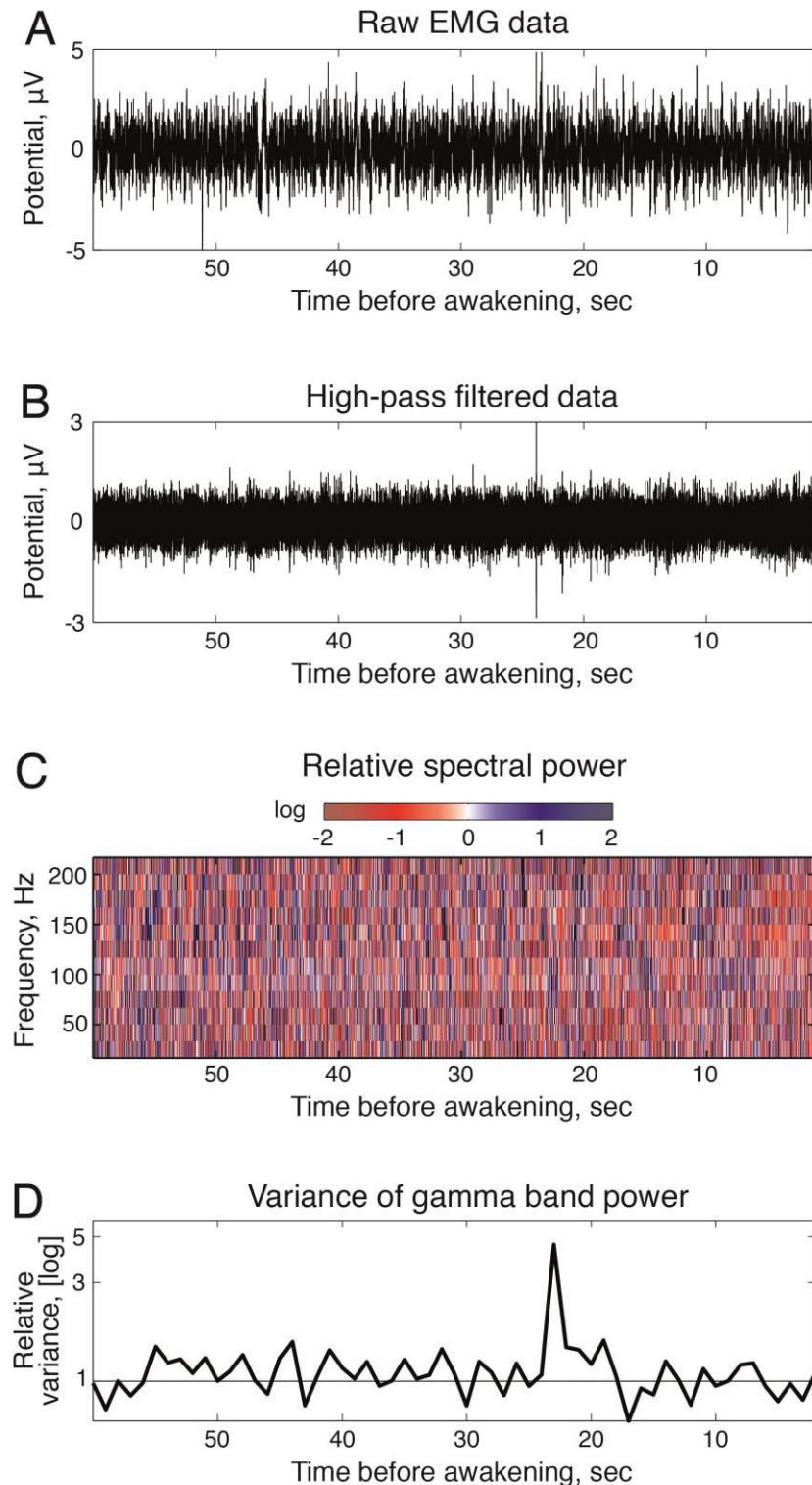
Figure S1 | Analysis of peripheral EMG activity. a) Exemplary 60 sec EMG recording of the right hand flexoris muscle between termination of tDCS and the awakening. **b)** The same EMG recording after a high-pass filter with a 50 Hz cutoff-frequency. **c)** Relative spectral power of the high-pass filtered EMG recording. **d)** Relative variance of gamma band power, i.e., divided by the average variance of gamma band power in the baseline time window.

23

494   Values greater than one (above the grey solid line) depict 1 sec segments with a shift towards

495   phasic EMG, values equal or smaller than one depict segments with a shift towards tonic

496   EMG.

497

498   **Statistical analysis.**

499   All dependent measures were averaged per individual participant separately for the sham-

500   stimulation and tDCS conditions. A Shapiro-Wilk test was used to assess the distribution

501   normality of dependent variables. Paired-samples t test and Pearson correlation were carried

502   out when distribution of given variables (or their difference in a case of t test) was normal,

503   and Wilcoxon signed-rank test (Z statistic) and Spearman rank order correlation were used in

504   the cases of non-normal distribution of one or both variables. For the paired-samples t-test,

505   Cohen's d was calculated as an effect size estimate using pooled variance. For the Wilcoxon

506   signed-rank test, r=Z/sqrt(N) was calculated as an effect size estimate. All statistical tests

507   were two-tailed. To control for multiple comparisons, Bonferroni correction was applied by

508   multiplying the obtained p value by the number of comparisons with a given set of tests.

509   Bonferroni corrected p values are denoted as p_{B-N} where N indicates the number of multiple

510   comparisons. For all control analyses, we report uncorrected p values. For the control t tests

511   where we expected null findings, we additionally report Bayes factor in favor of the null.

512   Statistical analyses were carried out with SPSS 22 and JASP 0.8.2.

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516 ## Results

517

518 **tDCS modulates dream movement.**

519 The first research question addressed whether the sensorimotor cortex is involved in the

520 generation of bodily experiences in dreams. To answer this, we compared the percentage of

521 dreams with different types of bodily experiences reported in the BED Questionnaire between

522 the tDCS and sham stimulation. Among the general dimensions of bodily experience in

523 dreams (tactile/somatosensory, vestibular/balance, movement, movement alterations, body

524 scheme alterations), we found a significant difference only for movement (see Fig 2 and

525 Table 2). Specifically, the proportion of dreams with movement was significantly lower in

526 the tDCS (M=63.1%, SEM=10.2%) compared to the sham-stimulation (M=86.6%,

527 SEM=7%) condition (paired samples t test: $t(9)=3.77$, $p_{B-5}=0.022$, $d=0.85$). That is,

528 participants were less likely to answer YES to the question "Did you move in your dream?"

529 when they were awakened 1 min after termination of bihemispheric tDCS. At the individual

530 level, 7 out of 10 participants showed this effect, whereas the remaining 3 participants had

531 equal proportions of dreams with movements between the two conditions (see Fig. 2).

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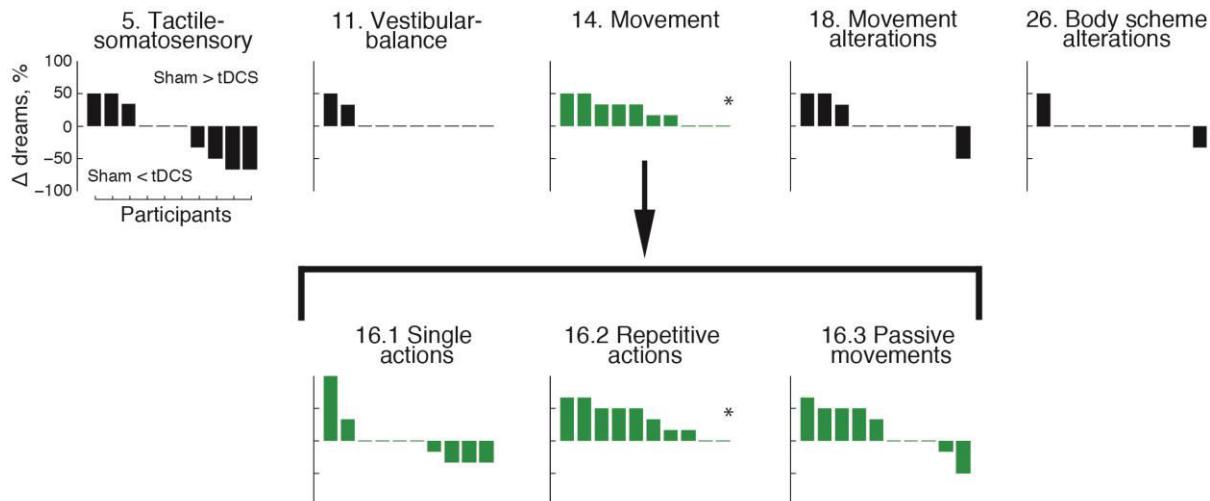
Figure 2 | tDCS effects on reported dream experiences. Changes between sham-stimulation and tDCS conditions across the five general categories of dream content (A-E) and for particular kinds of movement (F-H) per participant. Positive and negative values indicate a higher proportion of dreams with a specific experience in the sham-stimulation and tDCS condition, respectively. Individual participants are sorted in descending order beginning with the participant with the highest proportion of dreams with a specific experience in the sham-stimulation condition, compared to the tDCS condition. Participants are sorted separately for each dimension of experience. * $p_B < 0.05$.

553 **Table 2.** The BED Questionnaire: Percentage of dream reports containing specific bodily

554 experiences following sham-stimulation and tDCS during REM sleep

Bodily experiences	Sham	tDCS	Statistical test	
	M (SEM)	M (SEM)	t/Z	p_B
Five general dimensions				
5. Tactile- somatosensory	34.9 (12.3)	43.2 (12)	t(9) = 0.59	1
11. Vestibular- balance	8.3 (5.7)	0 (0)	Z = 1.34	0.9
14. Movement	86.6 (7)	63.1 (10.2)	t(9) = 3.77	0.022*
18. Movement alterations	13.3 (6.9)	5 (5)	Z = 0.76	1
26. Body scheme alterations	5 (5)	3.3 (3.3)	Z = 0.45	1
Movement sub-scales				
16.1 Single actions	53.3 (13.3)	51.7 (13.3)	Z = 0.22	1
16.2 Repetitive actions	65 (9.8)	30 (8.5)	t(9) = 4.36	0.006*
16.3 Passive movements	30 (8.5)	11.7 (7.9)	t(9) = 1.56	0.45

555 *Note.* t: paired samples t test; Z: Wilcoxon signed-rank test; * p_B< 0.05

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558   To investigate whether specific types of movement were inhibited by tDCS, we compared the

559   proportion of dreams with single actions (i.e. movements that are not repeated immediately

560   after their execution, such as placing a book on the table), repetitive actions (i.e. the same

561   movements repeated several times in a continuous sequence, such as running), and passive

562   movements (i.e. movements determined by external forces, such as traveling by car) between

563   tDCS and sham-stimulation conditions (see Table S1 for examples of movement descriptions

564   in the verbal dream reports). There were significantly less dreams with repetitive actions in

565   the tDCS condition (M=30%, SEM=8.5%) compared to the sham condition (M=65%,

566   SEM=9.8%) (paired samples t test: t(9)=4.36, d=1.21, p_{B-3}=0.006) (see Fig. 2). There were no

567   significant tDCS effects on the frequency of dreams containing single actions or passive

568   movements (see Table 2).

569   Interestingly, we found no difference in movement frequency between the stimulation

570   conditions in verbal dream reports that were content analysed by external judges (see Table

571   S2). This could be due to a considerably smaller proportion of explicitly expressed

572   movements in free reports compared to the BED Questionnaire answers. It is possible that

573   participants tended to omit movements from the spontaneous verbal reports that were given

574   before answering to explicit motor questions of the BED Questionnaire (see Supplementary

575   Results).

576   According to our questionnaire data, a majority of dream movements involved the whole

577   body (M=75.5%, SEM=7.62%) and more rarely the right hand (M=25%, SEM=8.23%) or

578   both hands (M=15.83%, SEM=7.02%); another unspecified body part was mentioned in only

579   one report. Repetitive actions typically involved the whole body (M=89.8%, SEM=6.8%),

580   with only 5.6% of repetitive movements performed by the right hand (Wilcoxon signed-rank

581   test: Z=2.71, p=0.007, effect size r=0.64). Contrary to this, the proportion of single actions

582   was comparable for the whole body (M=43.8%, SEM=12.3%) and right hand movements

583 (M=34.4%, SEM=11.5%, Wilcoxon signed-rank test: Z=0.43, p=0.67, effect size *r*=0.11). No

584 systematic body part or laterality differences were observed between the sham-stimulation

585 and tDCS conditions.

586 Importantly, the observed reduction of dream movement following tDCS was not related to

587 the overall length of dream reports, which could have been a confounding factor. To test

588 whether the reduction in dream movement was related to shorter dream reports following

589 tDCS, we compared the subjectively reported duration of dreams during the tDCS and sham-

590 stimulation conditions (BED Questionnaire - Q41, see Appendix 1). There was no difference

591 in the subjectively reported duration of dream reports between tDCS (Median=9.17 min,

592 range from 1.5 min to 97.5 min) and sham-stimulation (Median=9.67 min, range from 0.83

593 min to 40 min) conditions (Wilcoxon signed-rank test: Z=0.36, p=0.72, r=0.11). Furthermore,

594 we compared the word count of dream reports. Once again, there was no significant

595 difference between tDCS (M=76.1, SEM=16.31) and sham-stimulation (M=124.2,

596 SEM=34.68) conditions (paired samples t test: t(9)=1.69, p=0.124, d=0.56, Bf in favor of the

597 null=1.11). On four occasions, participants remembered and reported additional details of a

598 dream after completing the original dream report and questionnaire, while they were trying to

599 fall asleep again. When these secondary reports were included in the word count analysis,

600 there was still no significant difference in word count between tDCS (M=89, SEM=19.83)

601 and sham-stimulation (M=124.98, SEM=34.55) conditions (paired samples t test: t(9)=1.15,

602 p=0.281, d=0.4, Bf in favor of the null=1.91). We thus conclude that differences in the length

603 of dream reports (and in the subjectively estimated duration of dreams) were not related to

604 the observed reduction of dream movement following tDCS.

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607 **tDCS modulation of EEG activity.**

608 Given the opposing direction of bihemispheric tDCS in the current study, i.e. a cathodal

609 inhibitory effect over the left motor cortex and anodal excitatory effect over the right motor

610 cortex, we hypothesized that a reduction of repetitive whole-body actions in response to

611 tDCS was due to a decreased inter-hemispheric coordination of motor processing. To

612 investigate this hypothesis, we restricted EEG analysis to the beta frequency band, because

613 (1) transient and tonic changes in EEG beta oscillatory activity underlie cortical processing of

614 both real (Gerloff et al. 1998; Jenkinson and Brown 2011; Zaepffel et al. 2013) and imagined

615 (Neuper et al. 2005; Nam et al. 2011) movements, (2) preparation and execution of

616 movement involves inter-hemispheric functional coupling in the beta frequency range

617 (Leocani et al. 1997; Mima et al. 2000), and (3) motor impairment and successful

618 rehabilitation involve changes in the inter-hemispheric interaction in the beta frequency range

619 (Pellegrino et al. 2012; Fortuna et al. 2013). We thus expected bihemispheric tDCS to

620 destabilize motor processing by reducing inter-hemispheric coherence in the beta frequency

621 range.

622 As predicted, we observed a significant decrease in coherence between parietal electrodes P3-

623 P4 following tDCS compared to sham-stimulation during a 1-minute stimulation-free period

624 before awakening (Wilcoxon signed-rank test: Z=2.5, p_{B-3}=0.039, effect size *r*=0.79). No

625 inter-hemispheric tDCS effects were observed between frontal (paired samples t test:

626 t(9)=0.72, p_{B-3}=1, d=0.244) or temporal electrodes (t(9)=0.38, p_{B-3}=1, d=0.114). To control

627 for temporal specificity of the decrease of parietal coherence, we repeated the same analysis

628 in four separate time intervals following termination of stimulation: -60 to -46 sec, -45 to -31

629 sec, -30 to -16 sec, and -15 sec to -1 sec prior to awakening. A significant effect observed

630 only in the time window before awakening (i.e. -30 to -16 sec, and/or -15 sec to -1 sec) would

631 indicate a non-specific effect of experimental stimulation. Compared to sham-stimulation, a

30

632   significant decrease of parietal coherence took place in the tDCS condition throughout all

633   four sub-intervals between the offset of stimulation and the onset of awakening, confirming a

634   direct and relatively long-lasting tDCS effect on parietal coherence in the beta-frequency

635   range (see Fig 3).

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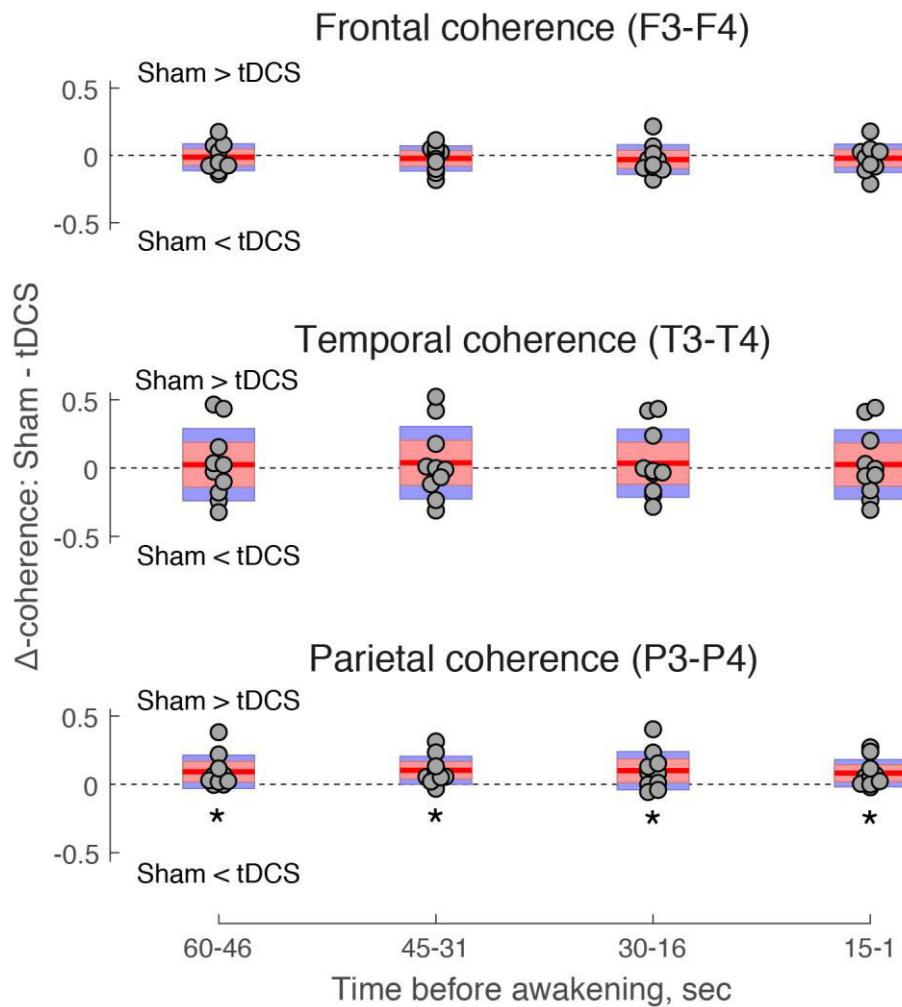
637

638   **Figure 3 | EEG coherence following tDCS during REM sleep.** Inter-hemispheric EEG

639   coherence between frontal (top), temporal (middle), and parietal (bottom) electrodes

640   surrounding the tDCS site, expressed as a difference between sham-stimulation and tDCS

641   conditions (Δ-coherence). Jittered circles represent individual participants. Red lines depict

642   the mean of Δ-coherence, pink bars represent 1 standard deviation (SD), and blue bars

643   represent 95% confidence intervals for the mean. Positive values indicate higher coherence in

644   the sham-stimulation condition, whereas negative values indicate higher coherence in the

645   tDCS condition. Δ-coherence is plotted separately in four stimulation-free time intervals

646   preceding controlled awakenings from REM sleep. In the parietal region, coherence was

647   reduced by tDCS compared to sham stimulation in -60- to 46 sec (Z=2.5, p=0.013, r=0.79), -

648   45 to -31 sec (t(9)=3.17, p=0.011, d=0.97), -30 to -16 sec (t(9)=2.27, p=0.05, d=0.88) and -15

649   to -1 sec (t(9)=2.57, p=0.03, d=0.74) time intervals. * p < 0.05.

650

651   Given that EEG coherence can be affected by spectral power differences between conditions

652   (Fein et al. 1988), we carried out a control analysis to compare beta power in the electrodes

653   adjacent to the stimulation site across a 1 min stimulation-free pre-awakening period. There

654   was a significant decrease of beta power at the left parietal site (P3) in the tDCS compared to

655   the sham-stimulation condition (paired samples t test: t(9)=2.29, p=0.048, d=0.37, Bf in favor

656   of the null=0.64), whereas tDCS did not modulate beta power in the right parietal site (P4)

657   (t(9)=0.73, p=0.48, d=0.088, Bf in favor of the null=2.93). The observed trend was

658   investigated further across four 15 sec sub-intervals. No tDCS effects were observed

659   regarding beta power in P3 electrode during time intervals immediately following motor

660   cortex stimulation, i.e. -60 to -46 sec (paired samples t test: t(9)=1.159, p=0.276, d=0.25, Bf

661   in favor of the null=1.89) and -45 to -31 sec (t(9)=1.172, p=0.271, d=0.3, Bf in favor of the

662   null=1.87). Contrary to this, beta power decreased during time intervals preceding

663   awakenings: -30 to -16 sec (paired samples t test: t(9)=2.433, p=0.038, d=0.433, Bf in favor

664   of the null=0.46) and -15 to -1 sec (t(9)=2.829, p=0.02, d=0.379, Bf in favor of the

665   null=0.28). Given that EEG beta coherence was modulated by tDCS across all four time

666   intervals, we conclude that its decrease was not due to the temporally constricted changes in

667   beta spectral power.

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672 **tDCS modulation of EMG activity.**

673 We observed a significant association in the proportion of phasic EMG activity in the flexors

674 between the left and right arms during the 1-min period of REM sleep from the offset of

675 tDCS to the controlled awakening (Pearson correlation: forearm flexors: r=-0.769, p_{B-4}=0.037;

676 deltoids: r=-738, p_{B-4}=0.06). The negative correlation between the arms likely reflects the

677 asymmetrical modality of stimulation with the cathode placed over the right sensorimotor

678 cortex and the anode over the left sensorimotor cortex. Contrary to this, there was no

679 association in the proportion of phasic EMG between forearms following sham stimulation

680 (Pearson correlation: forearm flexors: r=0.095, p_{B-4}=1; deltoids: r=0.308, p_{B-4}=1), indicating

681 that muscle activity varied independently (see Fig 4A). Regarding absolute EMG values,

682 there was no difference between phasic activity in the left as compared to the right arm in

683 either the sham-stimulation condition (paired samples t test: forearm flexors: t(9)=0.12, p_{B-4}=1, d=0.08; deltoids: t(9)=1.52, p_{B-4}=0.66, d=0.57) of following tDCS (forearm flexors:

684

685 t(9)=1.88, p_{B-4}=0.37, d=1.13; deltoids: t(9)=1.96, p_{B-4}=0.33, d=1.08).

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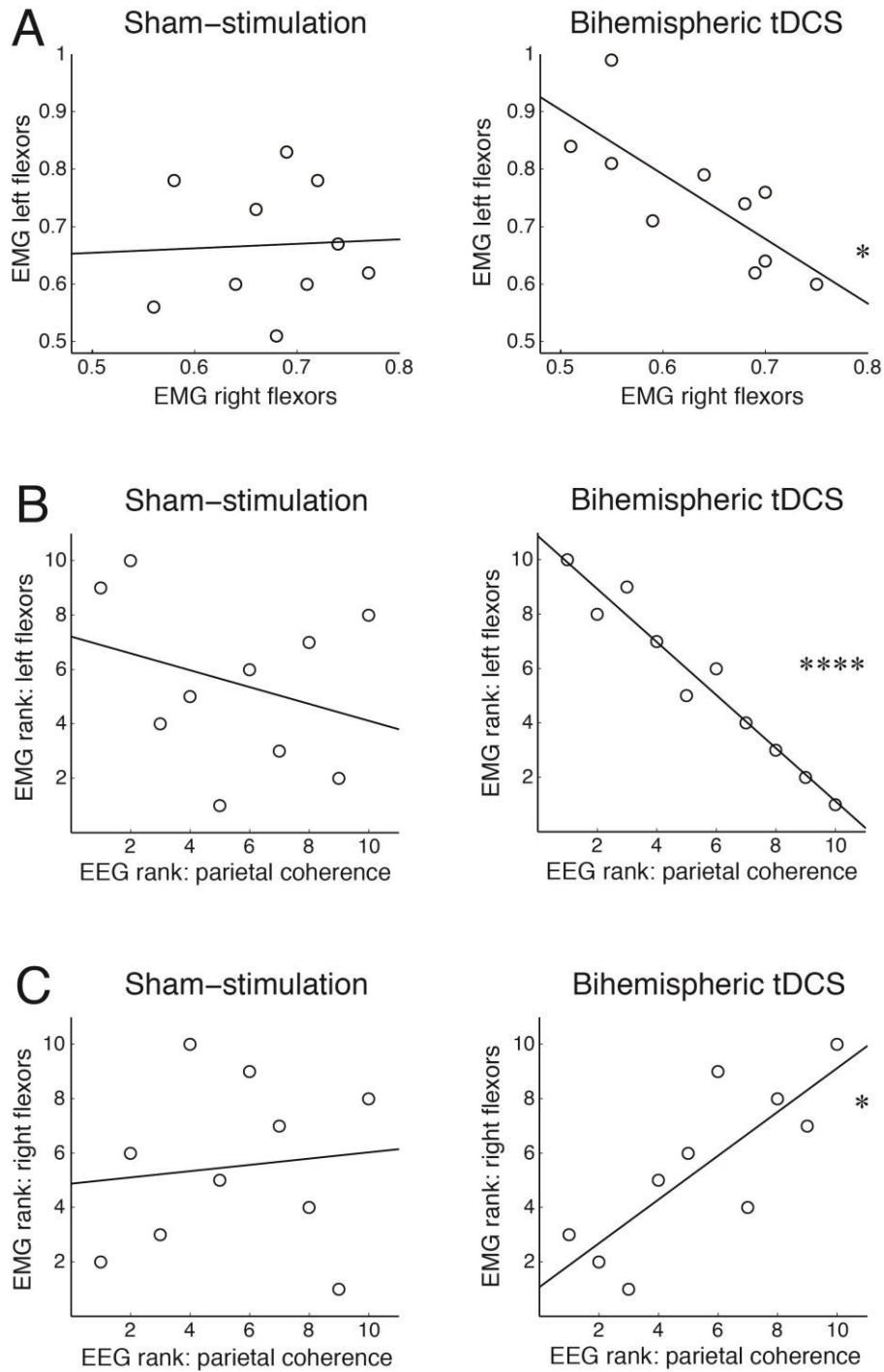
Figure 4 | Bihemispheric tDCS during REM sleep modulates phasic activity of the forearm muscles. (A) Correlation of EMG shift towards phasic activity between the left and right forearm flexor muscles in the sham-stimulation and tDCS conditions. **(B-C)** Correlation between EMG shift towards phasic activity and EEG parietal coherence in the beta frequency band, plotted separately for the left and right forearm recordings, in the sham-stimulation and tDCS conditions. Ranked data are plotted in (B) and (C) as Spearman's rank order

695   correlations were carried between EMG and EEG measures. In all plots, the least-squares

696   lines are plotted to visualize associations between variables. * $p_B < 0.05$, **** $p_B < 0.00005$.

697

698   Next, we investigated whether peripheral EMG activity is associated with EEG parietal

699   coherence in the beta frequency band, which decreased in response to tDCS during REM

700   sleep (see Fig 4B-C). In the tDCS condition, EEG coherence was significantly associated

701   with the proportion of phasic activity in the left flexor muscles (Spearman rank order

702   correlation: rho=-0.976, p_{B-8}=0.00001), and the right flexor muscles (rho=0.806, p_{B-8}=0.039).

703   Interestingly, higher parietal coherence was associated with a larger proportion of phasic

704   activity in the right forearm muscles and a lower proportion of phasic activity in the left

705   forearm muscles, once again likely reflecting differential effects of anodal vs. cathodal

706   stimulation. No association was observed between parietal EEG coherence and the proportion

707   of phasic activity in flexor muscles in the sham stimulation condition (lowest p_{B-8}=1).

708   Likewise, there was no association between parietal EEG coherence and deltoid EMG,

709   neither during sham-stimulation (lowest p_{B-8}=1) nor tDCS conditions (lowest p_{B-8}=0.72),

710   indicating a site specific interaction between EEG and EMG measures.

711

Discussion

713

714   The foremost aim of our study was to investigate the role of the sensorimotor cortex in

715   generating bodily sensations in REM sleep dreams by modulating the excitability of the

716   sensorimotor cortex with tDCS. We found that compared to sham stimulation, bihemispheric

717   tDCS over the sensorimotor cortex reduced the frequency specifically of repetitive actions of

718   the dream self in preceding REM sleep dreams, providing causal evidence that the

719   sensorimotor cortex is involved in the generation of dream movement. Furthermore, tDCS

720   interfered with inter-hemispheric EEG coherence and peripheral EMG activity, pointing to a

721   change in both the central and peripheral motor systems in response to bihemispheric tDCS

722   during REM sleep.

723

724   **Frequency of bodily sensations and movement in dreams.**

725   To systematically assess and directly interfere with bodily sensations in dreams, we

726   developed a questionnaire designed to capture various dimensions of bodily experiences in

727   dreams (see Table 2 and Appendix 1 for the complete questionnaire). Interestingly,

728   independently of tDCS, our data suggest that while dream movements were very common,

729   other bodily sensations such as somatosensory sensations, vestibular sensations or body

730   schema alterations were rather rare. This overall pattern of frequent dream movement

731   coupled with rare reports of other bodily sensations has been found in previous studies

732   (Hobson 1988; Schwartz 2000; Windt 2018). Our study extends the previous work based on

733   spontaneous dream reports by showing that when different types of bodily experiences are

734   specifically investigated through use of a questionnaire, movements and tactile sensations

735   remain the predominant dimensions of bodily experience in dreams. Thus, content analysis-

736   and questionnaire-based studies provide converging evidence for the important role of

737   sensorimotor phenomena in dreams.

738   The predominance of dream movement in our data also seems to be in line with a recent

739   suggestion that kinesthesia is central to the generation of dream experience, at least during

740   sleep onset (Nielsen 2017). At the same time, in our study, 36.9 % of dream reports following

741   tDCS contained no movements. It therefore seems that specifically self-movements are not

742   strictly necessary to sustain REM sleep dreaming. Moreover, the decrease of dream

36

743   movement did not reduce the length of dream reports in our sample. Whether these dreams

744   still involved e.g. observed movement is an open question.

745   **Electrophysiological effects of bihemispheric tDCS.**

746   Bihemispheric tDCS over the sensorimotor cortex, as compared to sham stimulation,

747   specifically altered repetitive actions in dreams. Repetitive actions are typically dependent on

748   implicit memory of learnt motor sequences (e.g., walking), the automatic processing of which

749   does not require explicit awareness and monitoring of movements. Such learnt, automatic

750   movements, as compared to more controlled and deliberate movements, are also associated

751   with a smaller increase of activity in brain areas related to motor processing (Wu and Hallett

752   2005). Thus, arguably, a relatively modest tDCS interference with cortical processing might

753   have down-regulated motor cortex activity involved in the processing of automatic

754   movements, reducing it to the baseline resting level and simultaneously inhibiting the

755   occurrence of repetitive actions in dreams. Contrary to this, the relatively stronger cortical

756   activation underlying single controlled actions might not have been reduced sufficiently by

757   tDCS interference to significantly alter dream content. This would explain why our results

758   showed a specific decrease in repetitive actions, while the frequency of single actions in

759   dreams remained relatively high during tDCS and did not significantly differ from sham

760   stimulation. Alternatively, bihemispheric stimulation might have interfered with the temporal

761   coordination of dream movement, prohibiting long sequences of repetitive actions, but

762   sparing temporally restricted single actions. Indeed, dream imagery is notoriously unstable

763   and prone to change in discontinuous jumps (Revonsuo and Salmivalli 1995). Such

764   possibilities should be more directly assessed in future studies, e.g. using motor imagery tasks

765   during wakefulness that would allow for a more stringent control of movement complexity.

766   We found that bihemispheric tDCS interfered with neural processing in the beta frequency

767   band, classically linked to motor processing (Leocani et al. 1997; Gerloff et al. 1998; Mima et

768   al. 2000; Neuper et al. 2005; Jenkinson and Brown 2011; Nam et al. 2011; Pellegrino et al.

769   2012; Fortuna et al. 2013; Zaepffel et al. 2013; Khanna and Carmena 2015). In our setup,

770   bihemispheric tDCS reduced inter-hemispheric coherence of parietal beta oscillations.

771   Arguably, the differential montage of tDCS electrodes, i.e. the excitatory anode over the right

772   sensorimotor cortex and the inhibitory cathode over the left sensorimotor cortex, disrupted

773   inter-hemispheric coordination of motor commands, reducing the rate of repetitive actions

774   associated with whole body movements in dreams. A differential effect of bihemispheric

775   tDCS was also observed in the phasic EMG activity of the arm muscles. While phasic EMG

776   varied independently between the arms during sham stimulation, a strong negative correlation

777   was observed following tDCS, i.e. it suppressed phasic muscle activity in one arm while

778   increasing it in the other arm.

779   We expected that such destabilizing and hemisphere-specific effects of tDCS would also

780   cause unilateral distortions of bodily sensations in dreams, i.e. stronger effects on one side of

781   the dream body. However, the observed reduction of dream movement in dreams was

782   independent of the laterality of stimulation. That is, the decrease of inter-hemispheric EEG

783   coherence and the emergence of phasic EMG anticorrelation between arms did not translate

784   into unilateral effects on the dream body. We can only speculate on the rather surprising lack

785   of side-specific effects, and further studies will be important to understand underlying

786   mechanisms. To detect effects on other modalities (e.g. body image distortion, vestibular

787   sensations), a larger group of participants might be necessary. Moreover, the absence of

788   modulatory effects of tDCS on somatosensory experiences, which were reported quite

789   frequently by our participants, could be related to the placement of the tDCS electrodes that

790   was specifically determined by the location of the hand area in the primary motor cortex.

38

791

Implications for consciousness studies.

Our study suggests a methodology for identifying, via causal manipulation, the neural correlates of specific types of dream experience. Thus, beyond dream and sleep research, our findings also have more general implications for consciousness research. First, they add another piece of evidence that the neural correlates of specific dream content match the neural correlates of corresponding cognitive and behavioural functions during wakefulness (Siclari et al. 2017). Going beyond mere correlation, our results provide *causal* evidence that the motor cortex is involved in the generation of movement sensations in dreams.

Our results also shed light on the phenomenological profile of self-representation in dreams. In simulation theories, the subjective sense of presence, or the experience of a self in a world, is central to dreaming. While this highlights the importance of self-simulation, the precise pattern of self-experience in dreams, as compared to wakefulness, raises questions (Windt 2015). One possibility is that bodily experience in dreams replicates waking experience; another is that dreams are characterized by a comparative overrepresentation of movement and an underrepresentation of other types of bodily experience (e.g. tactile, thermal, or pain sensations). Our finding that tDCS selectively altered dream movement, taken together with the comparatively low frequency of other types of bodily experience in dreams, is consistent with the second possibility. Future studies could aim to further investigate this question by gathering reports of bodily experience in both dreams and wakefulness, enabling a more direct comparison.

A related question concerns the relation between bodily experiences in dreams and the sleeping physical body. It is commonly thought that dream experience, including bodily experience, is completely independent of outward muscular activity and stimulation of the

39

815   physical body. However, there are empirical and theoretical reasons for thinking that varying

816   degrees of concordance between dream experience and the physical body exist, on both the

817   levels of sensory input and motor output (Windt et al. 2016; Windt 2018). Lesion studies in

818   cats have shown that pontine lesions, which eliminate REM-sleep related muscular atonia,

819   induce organized motor behavior, such as searching and attacking, during REM sleep

820   (Henley and Morrison 1974; Sastre and Jouvet 1979), possibly indicating dream behaviours.

821   Further examples include (illusory) own-body perception, such as when stimulation to the

822   sleeping body is incorporated in dreams (Nielsen 1993; Sauvageau et al. 1998), and dream

823   enactment behaviors in humans, in which outward muscular activity corresponds to

824   movement sensations in dreams. REM sleep behavior disorder, in which seemingly goal-

825   directed behaviors during REM sleep (such as attacking one's sleeping partner, attempting to

826   run, etc.) match subjective dream reports, is an extreme example (Schenck et al. 1986; Valli

827   et al. 2012; Howell and Schenck. 2015). But REM sleep is also accompanied by subtler

828   muscular activity in the form of twitching (Blumberg and Plumeau 2016). Its concordance

829   with dream experience seems plausible but has not been systematically investigated.

830   In our study, bihemispheric tDCS during REM sleep modulated not only dream movement

831   but also outward muscular activity in the arms. Due to the absence of movement reports in

832   several participants, we could not reliably relate individual variance in subjective movement

833   reports to electrophysiological measures. However, our findings are consistent with the

834   possibility that changes in dream movement are related to changes in outward muscular

835   activity during REM sleep. A promising avenue for future research could be to investigate the

836   relevance of bihemispheric tDCS for several movement-related sleep disorders. REM sleep

837   behaviour disorder would be a good place to start because of the match between dream

838   movements and outward physical activity. Other disorders that could benefit from the

839   inhibition of motor activity include sleep walking and restless leg syndrome. Here, however,

840   the association with dream experience is less clear and should be investigated more directly.

841

842   **Limitations and outlook.**

843   Despite these promising results, the current study has several limitations. First, the effects of

844   tDCS on mental states have been repetitively challenged by replicability difficulties

845   (Tremblay et al. 2014; Horvath et al. 2015a, 2015b) and should thus be treated with caution.

846   Nevertheless, given that motor cortex tDCS during wakefulness provides the most reliable

847   effects (Horvath et al. 2015b; Buch et al. 2017), we expect the same to hold during REM

848   sleep. Second, due to the very complicated and time-intensive protocol of the study, we could

849   only recruit a rather small number of participants. Thus, larger samples and replication

850   studies will be needed in future (Minarik et al. 2016). Furthermore, and again due to the

851   complexity of the setup, we did not include a control stimulation site nor did we switch the

852   side of the bihemispheric stimulation (to left anodal, right cathodal stimulation), which would

853   be especially interesting to disentangle hemisphere-specific effects. Future studies with a

854   larger sample of participants should also explore whether bihemispheric tDCS during REM

855   sleep interferes with a wider range of EEG frequencies involved in motor processing,

856   including alpha and gamma bands as well as broadband responses (Ball et al. 2008; Babiloni

857   et al. 2016).

858

859   **Conclusions.**

860   To conclude, this study provided, in a controlled setup, evidence that stimulation over the

861   sensorimotor cortex modulates dream content in healthy participants during REM sleep. This

862   has important implications for various research fields, including consciousness research, and

863 sleep and dream research. Future studies will have to pinpoint more specifically which neural

864 mechanisms underlie the inhibition of repetitive movements of the dream self and whether

865 the observed subjective and neurophysiological effects are sufficiently long-lasting to warrant

866 clinical studies in, for example, parasomnia patients.

867

Acknowledgements

874

Author Contributions.

876 V.N., J.M.W., A.A.K., T.B. and B.L. conceived the study and designed the experiments.

877 V.N., T.S. and R.P. conducted experiments. V.N., T.B., B.L. analyzed and interpreted EEG

878 data. M.K. and T.B. analyzed and interpreted EMG data. V.N., J.M.W., K.V. and B.L.

879 analyzed and interpreted dream data. V.N., J.M.W., M.K., K.V., T.S., R.P., A.R., T.B., and

880 B.L. prepared the manuscript.

881

Declaration of Interests

883 The authors declare no competing interests.

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References

Axelrod V, Rees G, Lavidor M, Bar M. 2015. Increasing propensity to mind-wander with transcranial direct current stimulation. Proc Natl Acad Sci. 112:3314–3319.

Babiloni C, Del Percio C, Vecchio F, Sebastiano F, Di Gennaro G, Quarato PP, Morace R, Pavone L, Soricelli A, Noce G, Esposito V, Rossini PM, Gallese V, Mirabella G. 2016. Alpha, beta and gamma electrocorticographic rhythms in somatosensory, motor, premotor and prefrontal cortical areas differ in movement execution and observation in humans. Clin Neurophysiol. 127:641–654.

Baldridge BJ. 1966. Physical concomitants of dreaming and the effect of stimulation on dreamsitle. Ohio State Med J. 62:1272–1275.

Baldridge BJ, Whitman R, Kramer M. 1965. The concurrence of fine muscle activity and rapid eye movements during sleep. Psychosom Med. 27:19–26.

Ball T, Demandt E, Mutschler I, Neitzel E, Mehring C, Vogt K, Aertsen A, Schulze-Bonhage A. 2008. Movement related activity in the high gamma range of the human EEG. Neuroimage. 41:302–310.

Blumberg MS. 2015. Developing Sensorimotor Systems in Our Sleep. Curr Dir Psychol Sci. 24:32–37.

Blumberg MS, Plumeau AM. 2016. A new view of "dream enactment" in REM sleep behavior disorder. Sleep Med Rev. 30:34–42.

Buch ER, Santarnecchi E, Antal A, Born J, Celnik PA, Classen J, Gerloff C, Hallett M, Hummel FC, Nitsche MA, Pascual-Leone A, Paulus WJ, Reis J, Robertson EM, Rothwell JC, Sandrini M, Schambra HM, Wassermann EM, Ziemann U, Cohen LG. 2017. Effects of tDCS on motor learning and memory formation: A consensus and critical position paper. Clin Neurophysiol. 128:589–603.

43

910   Buysse DJ, Reynolds CF, Monk TH, Berman SR, Kupfer DJ. 1989. The Pittsburgh sleep
911   quality index: A new instrument for psychiatric practice and research. Psychiatry Res.
912   28:193–213.

913   Cicogna P, Bosinelli M. 2001. Consciousness during dreams. Conscious Cogn. 10:26–41.

914   Cipolli C, Ferrara M, De Gennaro L, Plazzi G. 2017. Beyond the neuropsychology of
915   dreaming: Insights into the neural basis of dreaming with new techniques of sleep
916   recording and analysis. Sleep Med Rev. 35:8–20.

917   Dang-Vu TT, Desseilles M, Albouy G, Darsaud A, Gais S, Rauchs G, Schabus M, Sterpenich
918   V, Vandewalle G, Schwartz S, Maquet P. 2005. Dreaming: A neuroimaging view.
919   Schweizer Arch fur Neurol und Psychiatr. 156:415–425.

920   Delorme A, Makeig S. 2004. EEGLAB: An open source toolbox for analysis of single-trial
921   EEG dynamics including independent component analysis. J Neurosci Methods. 134:9–
922   21.

923   Dement W, Wolpert EA. 1958. The relation of eye movements, body motility, and external
924   stimuli to dream content. J Exp Psychol. 55:543–553.

925   Dresler M, Koch SP, Wehrle R, Spoormaker VI, Holsboer F, Steiger A, Sämann PG, Obrig
926   H, Czisch M. 2011. Dreamed movement elicits activation in the sensorimotor cortex.
927   Curr Biol. 21:1833–1837.

928   Fairley JA, Georgoulas G, Mehta NA, Gray AG, Bliwise DL. 2012. Computer detection
929   approaches for the identification of phasic electromyographic (EMG) activity during
930   human sleep. Biomed Signal Process Control. 7:606–615.

931   Fein G, Raz J, Brown FF, Merrin EL. 1988. Common reference coherence data are
932   confounded by power and phase effects. Electroencephalogr Clin Neurophysiol. 69:581–
933   584.

934   Feurra, M., Bianco G, Polizzotto NR, Innocenti I, Rossi A, Rossi S. 2011. Cortico-cortical
935   connectivity between right parietal and bilateral primary motor cortices during imagined
936   and observed actions: A combined TMS/tDCS study. Front Neural Circuits. 5:10.

937   Fortuna M, Teixeira S, Machado S, Velasques B, Bittencourt J, Peressutti C, Budde H, Cagy
938   M, Nardi AE, Piedade R, Ribeiro P, Arias-Carrión O. 2013. Cortical reorganization after
939   hand immobilization: The beta qEEG spectral coherence evidences. PLoS One.
940   8:e79912.

941   Fox KCR, Nijeboer S, Solomonova E, Domhoff GW, Christoff K. 2013. Dreaming as mind
942   wandering: evidence from functional neuroimaging and first-person content reports.
943   Front Hum Neurosci. 7:412.

944   Gandiga PC, Hummel FC, Cohen LG. 2006. Transcranial DC stimulation (tDCS): A tool for
945   double-blind sham-controlled clinical studies in brain stimulation. Clin Neurophysiol.
946   117:845–850.

947   Gerloff C, Richard J, Hadley J, Schulman AE, Honda M, Hallett M. 1998. Functional
948   coupling and regional activation of human cortical motor areas during simple, internally
949   paced and externally paced finger movements. Brain. 121:1513–1531.

950   Henley K, Morrison AR. 1974. A reevaluation of the effects of lesions of the pontine
951   tegmentum and locus coeruleus on phenomena of paradoxical sleep in the cat. Acta
952   Neurobiol Exp (Wars). 34:215–232.

953   Hobson JA. 1988. The dreaming brain. New York: Basic Books.

954   Hoff H. 1929. Zusammenhang von Vestibulärfunktion, Schlafstellung und Traumleben. Eur
955   Neurol. 71:366–372.

956   Hoff H, Pötzl O. 1937. Über die labyrinthären Beziehungen von Flugsensationen und
957   Flugträumen. Eur Neurol. 97:193–211.

958   Horvath JC, Forte JD, Carter O. 2015a. Quantitative review finds no evidence of cognitive
959   effects in healthy populations from single-session transcranial direct current stimulation
960   (tDCS). Brain Stimul. 8:535–550.

961   Horvath JC, Forte JD, Carter O. 2015b. Evidence that transcranial direct current stimulation
962   (tDCS) generates little-to-no reliable neurophysiologic effect beyond MEP amplitude
963   modulation in healthy human subjects: A systematic review. Neuropsychologia. 66:213–
964   236.

965   Howell MJ, Schenck. CH. 2015. REM sleep behavior disorder. In: Videnovic A,, Högl B,
966   editors. Disorders of Sleep and Circadian Rhythms in Parkinson's Disease. Vienna:
967   Springer. p. 131–144.

968   Hummel F, Celnik P, Giraux P, Floel A, Wu WH, Gerloff C, Cohen LG. 2005. Effects of
969   non-invasive cortical stimulation on skilled motor function in chronic stroke. Brain.
970   128:490–499.

971   Jakobson AJ, Conduit RD, Fitzgerald PB. 2012. Investigation of visual dream reports after
972   transcranial direct current stimulation (tDCS) during REM sleep. Int J Dream Res. 5:87–
973   93.

974   Jakobson AJ, Fitzgerald PB, Conduit R. 2012a. Induction of visual dream reports after
975   transcranial direct current stimulation (tDCs) during Stage 2 sleep. J Sleep Res. 21:369–
976   379.

977   Jakobson AJ, Fitzgerald PB, Conduit R. 2012b. Investigation of dream reports after
978   transcranial direct current stimulation (tDCs) during slow wave sleep (SWS). Sleep Biol
979   Rhythms. 10:169–178.

980   Jasper HH. 1958. The ten-twenty electrode system of the International Federation.
981   Electroencephalogr Clin Neurophysiol. 10:371–375.

46

982   Jenkinson N, Brown P. 2011. New insights into the relationship between dopamine, beta
983   oscillations and motor function. Trends Neurosci. 34:611–618.

984   Khanna P, Carmena JM. 2015. Neural oscillations: Beta band activity across motor networks.
985   Curr Opin Neurobiol. 32:60–67.

986   LaBerge S, Baird B, Zimbardo PG. 2018. Smooth tracking of visual targets distinguishes
987   lucid REM sleep dreaming and waking perception from imagination. Nat Commun.
988   9:3298.

989   Leocani L, Toro C, Manganotti P, Zhuang P, Hallett M. 1997. Event-related coherence and
990   event-related desynchronization/synchronization in the 10 Hz and 20 Hz EEG during
991   self- paced movements. Electroencephalogr Clin Neurophysiol - Evoked Potentials.
992   104:199–206.

993   Leslie K, Ogilvie R. 1996. Vestibular dreams: The effect of rocking on dream mentation.
994   Dreaming. 6:1–16.

995   Lindenberg R, Nachtigall L, Meinzer M, Sieg MM, Floel A. 2013. Differential Effects of
996   Dual and Unihemispheric Motor Cortex Stimulation in Older Adults. J Neurosci.
997   33:9176–9183.

998   Lindenberg R, Sieg MM, Meinzer M, Nachtigall L, Flöel A. 2016. Neural correlates of
999   unihemispheric and bihemispheric motor cortex stimulation in healthy young adults.
1000   Neuroimage. 140:141–149.

1001 Maquet P, Laureys S, Peigneux P, Fuchs S, Petiau C, Phillips C, Aerts J, Del Fiore G,
1002   Degueldre C, Meulemans T, Luxen A, Franck G, Van Der Linden M, Smith C,
1003   Cleeremans A. 2000. Experience-dependent changes in changes in cerebral activation
1004   during human REM sleep. Nat Neurosci. 3:831–836.

1005 Marshall L. 2004. Transcranial Direct Current Stimulation during Sleep Improves Declarative

47

1006   Memory. J Neurosci. 24:9985–9992.

1007 Matsumoto J, Fujiwara T, Takahashi O, Liu M, Kimura A, Ushiba J. 2010. Modulation of mu
1008   rhythm desynchronization during motor imagery by transcranial direct current
1009   stimulation. J Neuroeng Rehabil. 7:27.

1010 Mima T, Matsuoka T, Hallett M. 2000. Functional coupling of human right and left cortical
1011   motor areas demonstrated with partial coherence analysis. Neurosci Lett. 287:93–96.

1012 Minarik T, Berger B, Althaus L, Bader V, Biebl B, Brotzeller F, Fusban T, Hegemann J,
1013   Jesteadt L, Kalweit L, Leitner M, Linke F, Nabielska N, Reiter T, Schmitt D, Spraetz A,
1014   Sauseng P. 2016. The importance of sample size for reproducibility of tDCS effects.
1015   Front Hum Neurosci. 10:453.

1016 Nam CS, Jeon Y, Kim YJ, Lee I, Park K. 2011. Movement imagery-related lateralization of
1017   event-related (de)synchronization (ERD/ERS): Motor-imagery duration effects. Clin
1018   Neurophysiol. 122:567–577.

1019 Neuper C, Scherer R, Reiner M, Pfurtscheller G. 2005. Imagery of motor actions: Differential
1020   effects of kinesthetic and visual-motor mode of imagery in single-trial EEG. Cogn Brain
1021   Res. 25:668–677.

1022 Nielsen TA. 1993. Changes in the Kinesthetic Content of Dreams Following Somatosensory
1023   Stimulatipn of Leg Muscles During REM Sleep. Dreaming. 3:99–113.

1024 Nielsen TA. 2017. Microdream neurophenomenology. Neurosci Conscious. 3:nix001.

1025 Nir Y, Tononi G. 2010. Dreaming and the brain: from phenomenology to neurophysiology.
1026   Trends Cogn Sci. 14:88–100.

1027 Nitsche MA, Cohen LG, Wassermann EM, Priori A, Lang N, Antal A, Paulus W, Hummel F,
1028   Boggio PS, Fregni F, Pascual-Leone A. 2008. Transcranial direct current stimulation:

1029   State of the art 2008. Brain Stimul. 1:206–223.

1030   Nitsche M, Paulus W. 2000. Excitability changes induced in the human motor cortex by weak
1031   transcranial direct current stimulation. J Physiol. 527:633–639.

1032   Noreika V, Windt JM, Lenggenhager B, Karim AA. 2010. New perspectives for the study of
1033   lucid dreaming: From brain stimulation to philosophical theories of self-consciousness.
1034   Int J Dream Res. 3:36–45.

1035   Occhionero M, Cicogna P, Natale V, Esposito MJ, Bosinelli M. 2005. Representation of self
1036   in SWS and REM dreams. Sleep Hypn. 7:77–83.

1037   Oldfield RC. 1971. The assessment and analysis of handedness: The Edinburgh inventory.
1038   Neuropsychologia. 9:97–113.

1039   Opitz A, Yeagle E, Thielscher A, Schroeder C, Mehta AD, Milham MP. 2018. On the
1040   importance of precise electrode placement for targeted transcranial electric stimulation.
1041   Neuroimage. 181:560–567.

1042   Pellegrino G, Tomasevic L, Tombini M, Assenza G, Bravi M, Sterzi S, Giacobbe V, Zollo L,
1043   Guglielmelli E, Cavallo G, Vernieri F, Tecchio F. 2012. Inter-hemispheric coupling
1044   changes associate with motor improvements after robotic stroke rehabilitation. Restor
1045   Neurol Neurosci. 30:497–510.

1046   Percival D, Walden AT. 2000. Wavelet Methods for Time Series Analysis (Cambridge Series
1047   in Statistical and Probabilistic Mathematics). Cambridge: Cambridge University Press.

1048   Pompeiano O. 1967. Sensory inhibition during motor activity in sleep. In: Yahr MD,, Purpura
1049   DP, editors. Neurophysiological basis of normal and abnormal motor activities. New
1050   York: Raven Press. p. 323–375.

1051   Priori A. 2003. Brain polarization in humans: A reappraisal of an old tool for prolonged non-

1052   invasive modulation of brain excitability. Clin Neurophysiol. 114:589–595.

1053   Quartarone A, Morgante F, Bagnato S, Rizzo V, Sant'Angelo A, Aiello E, Reggio E,
1054   Battaglia F, Messina C, Girlanda P. 2004. Long lasting effects of transcranial direct
1055   current stimulation on motor imagery. Neuroreport. 15:1287–1291.

1056   Revonsuo A, Salmivalli C. 1995. A content analysis of bizarre elements in dreams.
1057   Dreaming. 5:169–187.

1058   Revonsuo A, Tuominen J, Valli K. 2015. The Avatars in the Machine - Dreaming as a
1059   Simulation of Social Reality. In: Metzinger T,, Windt JM, editors. Open MIND.
1060   Frankfurt am Main: MIND Group. p. 32.

1061   Richter L, Neumann G, Oung S, Schweikard A, Trillenberg P. 2013. Optimal Coil
1062   Orientation for Transcranial Magnetic Stimulation. PLoS One. 8:e60358.

1063   Ruohonen J, Karhu J. 2010. Navigated transcranial magnetic stimulation. Neurophysiol Clin
1064   Neurophysiol. 40:7–17.

1065   Sanchez-Vives M V., Slater M. 2005. From presence to consciousness through virtual reality.
1066   Nat Rev Neurosci. 6:332–339.

1067   Sastre JP, Jouvet M. 1979. Oneiric behavior in cats. Physiol Behav. 9:293–308.

1068   Sauvageau A, Nielsen TA, Montplaisir J. 1998. Effects of somatosensory stimulation on
1069   dream content in gymnasts and control participants: Evidence of vestibulomotor
1070   adaptation in REM sleep. Dreaming. 8:125–134.

1071   Schenck CH, Bundlie SR, Ettinger MG, Mahowald MW. 1986. Chronic behavioral disorders
1072   of human REM sleep: A new category of parasomnia. Sleep. 9:293–308.

1073   Schredl M. 2002. Dream recall frequency and openness to experience: A negative finding.

50

1074   Pers Individ Dif. 33:1285–1289.

1075   Schredl M, Atanasova D, Hörmann K, Maurer JT, Hummel T, Stuck BA. 2009. Information
1076   processing during sleep: The effect of olfactory stimuli on dream content and dream
1077   emotions. J Sleep Res. 18:285–290.

1078   Schwartz S. 2000. A Historical Loop of One Hundred Years: Similarities between 19th
1079   Century and Contemporary Dream Research. Dreaming. 10:55–66.

1080   Schwartz S, Maquet P. 2002. Sleep imaging and the neuropsychological assessment of
1081   dreams. Trends Cogn Sci. 6:23–30.

1082   Siclari F, Baird B, Perogamvros L, Bernardi G, LaRocque JJ, Riedner B, Boly M, Postle BR,
1083   Tononi G. 2017. The neural correlates of dreaming. Nat Neurosci. 20:872–878.

1084   Sikka P, Feilhauer D, Valli KJ, Revonsuo A. 2017. How you measure is what you get:
1085   Differences in self- and external ratings of emotional experiences in home dreams. Am J
1086   Psychol. 130:367–384.

1087   Sikka P, Valli K, Virta T, Revonsuo A. 2014. I know how you felt last night, or do I? Self-
1088   and external ratings of emotions in REM sleep dreams. Conscious Cogn. 25:51–66.

1089   Speth J, Frenzel C, Voss U. 2013. A differentiating empirical linguistic analysis of dreamer
1090   activity in reports of EEG-controlled REM-dreams and hypnagogic hallucinations.
1091   Conscious Cogn. 22:1013–1021.

1092   Speth J, Speth C. 2016. Motor imagery in REM sleep is increased by transcranial direct
1093   current stimulation of the left motor cortex (C3). Neuropsychologia. 86:57–65.

1094   Strauch I, Meier B. 1996. In search of dreams. Results of experimental dream research.
1095   Albany, New York: SUNY Press.

1096   Stumbrys T, Erlacher D, Schredl M. 2013. Testing the involvement of the prefrontal cortex in
1097   lucid dreaming: A tDCS study. Conscious Cogn. 22:1214–1222.

1098   Tadel F, Baillet S, Mosher JC, Pantazis D, Leahy RM. 2011. Brainstorm: A user-friendly
1099   application for MEG/EEG analysis. Comput Intell Neurosci. 879716:1–13.

1100   Tremblay S, Lepage JF, Latulipe-Loiselle A, Fregni F, Pascual-Leone A, Théoret H. 2014.
1101   The uncertain outcome of prefrontal tDCS. Brain Stimul. 7:773–783.

1102   Valli K, Frauscher B, Gschliesser V, Wolf E, Falkenstetter T, Schönwald S V., Ehrmann L,
1103   Zangerl A, Marti I, Boesch SM, Revonsuo A, Poewe W, Högl B. 2012. Can observers
1104   link dream content to behaviours in rapid eye movement sleep behaviour disorder? A
1105   cross-sectional experimental pilot study. J Sleep Res. 21:21–29.

1106   Voss U, Holzmann R, Hobson A, Paulus W, Koppehele-Gossel J, Klimke A, Nitsche MA.
1107   2014. Induction of self awareness in dreams through frontal low current stimulation of
1108   gamma activity. Nat Neurosci. 17:810–812.

1109   Windt JM. 2015. Dreaming: A conceptual framework for philosophy of mind and empirical
1110   research. Cambridge, MA: MIT Press.

1111   Windt JM. 2018. Predictive brains, dreaming selves, sleeping bodies: how the analysis of
1112   dream movement can inform a theory of self- and world-simulation in dreams. Synthese.
1113   195:2577–2625.

1114   Windt JM, Nielsen T, Thompson E. 2016. Does Consciousness Disappear in Dreamless
1115   Sleep? Trends Cogn Sci. 20:871–882.

1116   Wu MF. 1993. Sensory processing and sensation during sleep. In: Carskadon MA, editor.
1117   Encyclopedia of Sleep and Dreaming. New York: Macmillan. p. 533–535.

1118   Wu T, Hallett M. 2005. A functional MRI study of automatic movements in patients with

1119   Parkinson's disease. Brain. 128:2250–2259.

1120   Yousry TA, Schmid UD, Alkadhi H, Schmidt D, Peraud A, Buettner A, Winkler P. 1997.
1121   Localization of the motor hand area to a knob on the precentral gyrus. A new landmark.
1122   Brain. 120:141–157.

1123   Zaepffel M, Trachel R, Kilavik BE, Brochier T. 2013. Modulations of EEG Beta Power
1124   during Planning and Execution of Grasping Movements. PLoS One. 8:e60060.

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1139 **SUPPLEMENTARY MATERIALS**

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1141 **Content analysis of movement sensations in verbal dream reports: Results.**

1142 Movements were reported in 49.8% (SEM=10) of dreams following sham-stimulation and

1143 54.9% (SEM=10.9) of dreams following tDCS. Repetitive actions were the most common

1144 type of movement, followed by single actions, with passive movements being the least

1145 common (see Tables S1 and S2), replicating the pattern observed in the BED Questionnaire

1146 data. However, there were no significant differences between the sham-stimulation and tDCS

1147 conditions (see Table S2), in contrast to the effects observed in the questionnaire data (see

1148 Table 1). The discrepancy could be due to a considerably smaller proportion of explicitly

1149 expressed movements in free dream reports compared to the BED Questionnaire answers, i.e.

1150 participants tended to omit movements from the spontaneous verbal reports unless asked

1151 about them explicitly.

1152 The difference between questionnaire results and dream report analyses has also been found

1153 for emotions. The frequency of emotions increases 10-fold if participants are asked to report

1154 emotions on a line-by-line basis, as compared to free dream reports (Merritt et al. 1994).

1155 When participants are asked to rate the kinds of emotions experienced in their dreams, they

1156 specifically report more positive emotions than when their dream reports are analyzed by

1157 independent judges (Sikka et al. 2014, 2017). This discrepancy raises important

1158 methodological issues that to date have not been fully resolved, and both methods likely have

1159 weaknesses and suffer from different kinds of bias (Sikka et al. 2017). One reason for the

1160 discrepancy, however, could be that free dream reports lack the focus to allow independent

1161 judges to pick up on specific aspects of dream phenomenology, such as emotions or

1162 movements. By contrast, when participants' focus is directed to these aspects, such as

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1163   through use of questionnaires, this leads to more precise reporting. In our data, similar

1164   proportions of different types of movements between external ratings and questionnaire

1165   responses, together with the fact that movements are reported more frequently in the

1166   questionnaire data, makes us lean towards this interpretation. There are also likely differences

1167   in what is reported: in free dream reports, individual movements need to be described in some

1168   detail for them to be rated by external judges. By contrast, in the questionnaire, participants

1169   rate the occurrence and frequency of specific movement types over the entire dream. Again,

1170   this may lead to a more comprehensive picture, but also bears the danger of overgeneralizing.

1171   Nevertheless, the proportion of repetitive actions correlated strongly between the free dream

1172   reports and the BED Questionnaire answers in the sham-stimulation condition (Spearman

1173   rank order correlation: rho=0.81, p_{B-8}=0.033), indicating a strong convergence between these

1174   two types of measurement. Interestingly, this association did not hold in the tDCS condition

1175   (rho=-0.19, p_{B-8}=1). No other correlations were significant.

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1191 **Supplementary tables**

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1193 **Table S1.** Examples of different types of movement reported in verbal dream reports

Movement types	*Sham*	*tDCS*
Single actions	"I […] took away the wires" (P4, N2, A2)	"I was diving" (P1, N1, A1)
	"I was putting together some board" (P10, N2, A2)	"I was […] to take a pose" (P5, N2, A1)
	"We [...] sat down" (P10, N2, A2)	"I painted a sunset and there was a ship" (P6, N2, A3)
	"I hugged her" (P10, N2, A3)	"I jumped there to the movie" (P7, N1, A1)
Repetitive actions	"I remember rubbing quite hard […] my leg" (P1, N2, A1)	"I was swimming in a pool" (P1, N1, A1)
	"I was walking there" (P3, N2, A2)	"I […] was writing something" (P3, N1, A1)
	"we are running away from him" (P4, N2, A1)	"we […] went to the bathroom" (P5, N2, A2)
	"I had been sleepwalking" (P4, N2, A2)	"I was cleaning a table" (P5, N2, A2)

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	"I was digging the vegetable garden" (P6, N1, A3)	"I was climbing upstairs" (P7, N2, A2)
	"I was coming out from some room" (P10, N2, A2)	"I was scratching [our cat]" (P8, N2, A3)
Passive movements	"we were coming from Lappeenranta with a train" (P4, N2, A1)	"our father was driving me and my brother […] with a car" (P6, N2, A1)
	"they somehow forced to put my hand to fist" (P7, N1, A1)	"he took my hand and pulled me to the middle" (P6, N2, A1)

1194 *Note.* P – participant (1-10), N – night (1-2), A – awakening (1-3).

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1207 **Table S2.** Dream content analysis: Percentage of dream reports containing movement

1208 sensations following sham-stimulation and tDCS during REM sleep

	Sham	*tDCS*	*Statistical test*	
	M (SEM)	*M (SEM)*	*t/Z*	*p*
Movement				
	49.8 (10)	54.9 (10.9)	t(9) = 0.31	0.77
Movement sub-scales				
Single actions	21.6 (10.5)	24.9 (8.6)	t(9) = 0.19	0.85
Repetitive actions	38.2 (12.7)	43.2 (9.4)	Z = 0.54	0.59
Passive movements	16.6 (7)	8.3 (5.7)	Z = 0.76	0.45

1209 *Note.* t: paired samples t test; Z: Wilcoxon signed-rank test. Uncorrected p values.

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1219 **Table S3.** Balance of awakenings between the first and the second night and between the

1220 sham-stimulation and tDCS conditions

Participant	1st night, N	2nd night, N	Sham, N	tDCS, N
1	2	3	3	2
2	2	3	2	3
3	2	2	2	2
4	3	2	2	3
5	3	2	3	2
6	3	3	3	3
7	3	3	3	3
8	3	3	3	3
9	2	3	3	2
10	3	2	2	3
Total:	25	25	25	25

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