Emotion Regulation Failures Are Preceded by Local Increases in Sleep-like Activity

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Abstract

■ Emotion self-regulation relies both on cognitive and behavioral strategies implemented to modulate the subjective experience and/or the behavioral expression of a given emotion. Although it is known that a network encompassing fronto-cingulate and parietal brain areas is engaged during successful emotion regulation, the functional mechanisms underlying failures in emotion suppression (ES) are still unclear. In order to investigate this issue, we analyzed video and high-density EEG recordings of 20 healthy adult participants during an ES and a free expression task performed on two consecutive days. Changes in facial expression during ES, but not free expression, were preceded by local increases in sleep-like activity (1–4 Hz) in brain areas responsible for emotional suppression, including bilateral anterior insula and anterior cingulate cortex, and in right middle/inferior frontal gyrus ($p < .05$, corrected). Moreover, shorter sleep duration the night before the ES experiment correlated with the number of behavioral errors ($p = .03$) and tended to be associated with higher frontal sleep-like activity during ES failures ($p = .09$). These results indicate that local sleep-like activity may represent the cause of ES failures in humans and may offer a functional explanation for previous observations linking lack of sleep, changes in frontal activity, and emotional dysregulation. ■

INTRODUCTION

Emotions are an essential aspect of the psychological life of human beings. In fact, they greatly affect our physiological, cognitive, and behavioral responses to internal and external stimuli (Koole, 2009; Sapolsky, 2007; Cacioppo, Berntson, Larsen, Poehlmann, & Ito, 2001; Keltner & Kring, 1998). Importantly, emotions may occasionally lead to inappropriate or exaggerated reactions that could have negative consequences on our social life. Therefore, in dealing with emotions, people frequently engage in covert or overt forms of self-regulation in order to preserve a flexible and goal-oriented behavior (Kelley, Wagner, & Heatherton, 2015; Koole, 2009). In general, self-regulation involves a balance between the strength of an impulse, its related reward, and the individuals' ability to resist the impulse and to modify their behavior in accordance with relevant personal goals (Gross & Thompson, 2007; Carver & Scheier, 1998). When applied to emotions, self-regulation typically implies adjusting their type, intensity, duration, and expression (Gross, 1998). Various strategies, ranging from attention allocation to cognitive reappraisal and expressive suppression (Webb, Miles, & Sheeran, 2012; Gross, 1998), can be used to regulate an individual's reaction to emotional states. For instance, when applying expressive suppression, a response-based modulation, individuals voluntarily refrain from overtly showing their emotional state, which is kept hidden to an external observer. Previous work showed that voluntary emotion suppression (ES) during the presentation of emotion-inducing stimuli is associated with the activation of a broad fronto-parieto-insular network, including bilateral SMA, pre-SMA, anterior midcingulate cortex, anterior insula, inferior frontal gyrus, lateral orbitofrontal cortex, posterior middle frontal gyrus, dorsal temporo-parietal junction, and the left posterior middle temporal gyrus (Langner, Leiberg, Hoffstaedter, & Eickhoff, 2018; Frank et al., 2014; Kohn et al., 2014). Yet, what may cause a failure of this emotion-regulation system and the consequent generation of undesired behavioral responses still remains largely unclear.

Interestingly, sleep loss because of restriction or deprivation is known to significantly impair the ability to regulate emotional responses and affective states (Ben Simon, Vallat, Barnes, & Walker, 2020), and these changes have been suggested to depend on an altered top–down control of the medial frontal cortex on limbic structures (Yoo, Gujar, Hu, Jolesz, & Walker, 2007). However, the possible functional cause of this frontal impairment is unclear. Moreover, it remains to be determined whether a similar mechanism based on frontal impairment may also explain emotion regulation failures observed in (apparently) rested wakefulness.

Recent evidence indicates that local, temporary intrusions of sleep-like brain activity may represent a common cause of behavioral errors when involving task-related

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Journal of Cognitive Neuroscience 33:11, pp. 2342–2356
https://doi.org/10.1162/jocn_a_01753

brain regions (Nir et al., 2017; Bernardi et al., 2015). Such local sleep-like episodes are manifested with the appearance, in the EEG signal, of delta-theta waves (< 8 Hz) similar to those of actual sleep, which were shown to correspond in rats with spatially and temporally circumscribed reductions in neuronal firing (off-periods; Vyazovskiy et al., 2011). Given that they increase in number and extension as a function of time spent awake and that such changes are reverted by a night of sleep, local sleep-like episodes have been suggested to represent a signature of brain functional "fatigue" and a direct cause of behavioral impairment following sleep loss (Andrillon et al., 2019; D'Ambrosio et al., 2019). Of note, during wakefulness, the number of sleep-like episodes does not increase homogeneously over the cortical mantle. In fact, frontal areas typically display the strongest increases in low-frequency activity relative to other brain regions, suggesting a particular vulnerability to functional fatigue (Strijkstra, Beersma, Drayer, Halbesma, & Daan, 2003; Finelli, Baumann, Borbély, & Achermann, 2000).

In light of the above considerations, here, we hypothesized that local sleep-like episodes (i.e., temporary increases in delta and/or theta activity) occurring in areas of the emotion regulation network, and especially within frontal brain regions, could represent a potential cause of ES failures. In particular, we predicted that a shorter sleep time or a reduced sleep quality could lead to a higher incidence of frontal sleep-like episodes the following morning, which would in turn result in higher probability of ES failures.

METHODS

Participants

Twenty healthy adults (age range = 21–31 years, mean = 26.2 years, *SD* = 2.8 years, 11 women, all right-handed) were included in the study. All participants underwent a preliminary interview to exclude any clinical, neurological, or psychiatric conditions potentially affecting brain function and behavior. Additional exclusion criteria comprised excessive daytime sleepiness (Epworth Sleepiness Scale score > 10; Johns, 1991) and extreme chronotypes (Morningness-Eveningness Questionnaire score > 70 or < 30; Horne & Ostberg, 1976). Participants were asked to maintain a regular sleep–wake schedule for at least 1 week before each experiment. Compliance was verified by wrist-worn actigraphy (MotionWatch8, CamNTech). The study was conducted under a protocol defined in accordance with the ethical standards of the 2013 Declaration of Helsinki and approved by the local ethical committee. Written informed consent was obtained from all participants.

Experimental Procedures

Data analyzed in the present work were collected as part of a larger study aimed at investigating the neural and behavioral consequences of extended task practice (unpublished). A general overview of the whole experimental protocol is provided herein.

All participants completed a practice session and two experimental visits in which EEG activity (EGI; 64 electrodes, 500-Hz sampling rate) and behavioral data were recorded. In order to minimize interindividual differences in wake–sleep rhythms and work-related fatigue, all sessions were performed with a predefined, fixed schedule. In particular, the practice session was performed on Friday morning from 9:30 to 11:30 a.m., and the two experimental visits took place on the next Monday and Tuesday, from 8:30 a.m. to 1:00 p.m.

During the practice session, participants completed five 5-min trials of a motor-response inhibition task (Bernardi et al., 2015; Garavan, Ross, & Stein, 1999; 300 stimuli, 10% lures). Data obtained from this procedure were used to calibrate the difficulty of the same task presented during the two subsequent experimental sessions, as described in a previous work (Chuah, Venkatraman, Dinges, & Chee, 2006).

The two experimental visits required the participants to complete partially different versions of the same tasks. Each experiment began with a ~15-min long test block (baseline [BL]) including two 2-min resting-state EEG recordings with eyes open (4 min in total) and two trials of the response inhibition task. Moreover, subjective vigilance, sleepiness, mood, perceived effort, and motivation were assessed using 10-point Likert scales. This initial assessment was followed by three ~45-min long task sessions (TS1–3), each one followed by a ~15-min long test block identical to the first one (Figure 1). In one of the two experimental visits, the task sessions included three computerized tasks requiring high levels of impulse control, decision-making, and conflict resolution. The three tasks, which were always completed in the same order, included an ES task (Baumeister, Bratslavsky, Muraven, & Tice, 1998), a false response task (Bernardi et al., 2015), and a classical Stroop task (Stroop, 1935). A detailed description of the ES task, which was the focus of this study, is provided below. In the other experimental visit, a modified version of the same tasks was presented, which required no exertion of self-control (e.g., Stroop task with consistent word-ink color). Finally, before the last task block, a caffeinated or decaffeinated beverage was provided to participants. The order of the two experimental visits and the administration of caffeinated versus decaffeinated beverages were randomized across participants, so that each participant received the same type of beverage in both experimental conditions. All computerized tasks were implemented using E-Prime 2 (Psychology Software Tools).

Emotion Suppression/Expression Task

In one experimental visit, participants completed three instances (one for each task session) of an ES task in which they were explicitly requested to suppress their facial

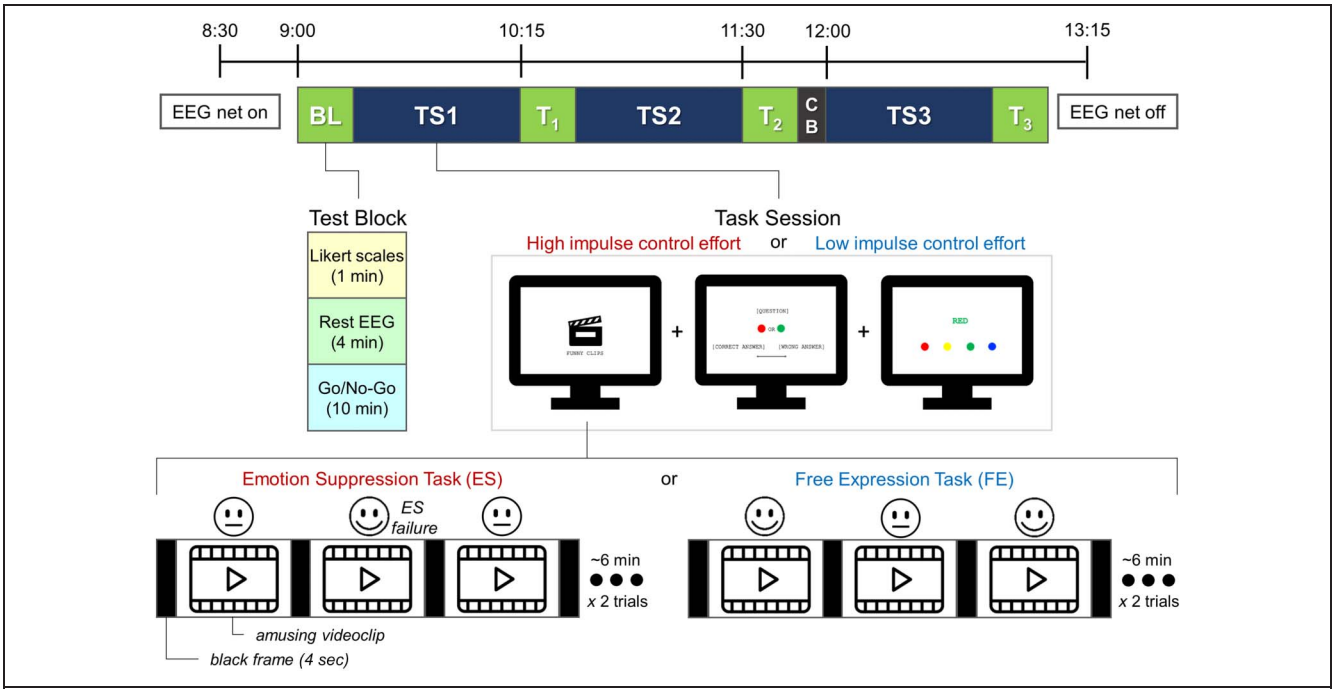

Figure 1. Experimental paradigm. The BL assessment and each test block (T_1–T_3) included Likert scales, resting-state EEG recordings, and the completion of two trials of a response inhibition task (go/no-go). In one of the two experimental visits (high impulse-control effort condition), participants completed an ES task, a false response task, and a Stroop task. During the other experimental visit (low impulse-control effort condition), participants completed a modified version of the same tasks requiring no exertion of self-control. The bottom panel of the figure includes a schematic representation of the ES task and the FE task used as a control condition. CB = coffee break (see main text).

reactions while watching amusing video clips ("ES" condition). In the task sessions of the other experimental visit, volunteers watched similar video clips, but they were left free to express their emotional responses ("free expression" [FE] condition). For each task session, the video clips were presented in two separate trials (henceforth indicated with the letters A and B appended to the name of the task session; e.g., TS1-A and TS1-B), during which they were alternated with 4 sec of black frames, for an average total duration of 5.9 ± 0.3 min per trial. The participants' faces were video-recorded using a camera positioned above the PC monitor and synchronized with the software used to record the EEG signals (NetStation 5.4, EGI).

In order to present different contents to participants during each task session, a total of 261 clips depicting people and/or animals in amusing situations were downloaded from the Internet. An initial validation procedure was performed in an independent sample of 12 participants (age range = 23–36 years, mean = 29.6 years, *SD* = 4 years, 8 women) to ensure a balanced distribution of the emotional contents across sessions. Specifically, the volunteers were asked to rate the set of amusing clips and an additional set of 20 clips showing simple actions/activities with no obvious emotional content. The clips were presented in random order. After each video, the participants answered the question *"How difficult would it be for you to keep a neutral expression while watching this clip?"* using a rating scale ranging from 1 (*Not difficult at all*) to 5 (*Very difficult*). As expected, the 261 amusing clips were associated in each participant with significantly higher ratings with respect to the 20 neutral clips ($p < .001$). For each participant, the ratings given to the amusing video clips were rescaled by computing the ratio with respect to the average rating of the 20 neutral clips, which were considered as an individual baseline. Then, the group-average ratios were computed for each clip, and a randomization procedure was used to assign the clips to each task trial, thus ensuring a similar distribution of ratings and a similar total duration of the video stimuli (Table 1).

Table 1. Video Ratings

	ES	*FE*		
	Mean ± SD	*Mean ± SD*	*z Score*	*p Value*
TS1-A	1.85 ± 0.23	1.90 ± 0.20	0.393	0.705
TS1-B	1.94 ± 0.29	1.87 ± 0.25	0.608	0.551
TS2-A	1.88 ± 0.27	1.97 ± 0.17	0.718	0.488
TS2-B	1.88 ± 0.25	1.82 ± 0.28	0.464	0.661
TS3-A	1.92 ± 0.16	1.91 ± 0.24	0.083	0.946
TS3-B	1.88 ± 0.18	1.87 ± 0.26	0.103	0.924

Mean and standard deviation (*SD*) of video ratings (ratio with respect to mean score of neutral videos) for each task trial, presented during the ES condition and the FE condition. No statistically significant differences were found across experimental conditions.

Scoring of Video Recordings

In order to identify and quantify the occurrence of facial expression changes in each task trial, video recordings of each participant were visually inspected and scored by one of the authors (D. B.). The scoring procedure was performed in two steps. First, the operator watched each video using a custom-made program written in Psychtoolbox v3.0.1 (Kleiner et al., 2007) for MATLAB (v9.7; The MathWorks Inc.). Each time a change in facial expression was identified, the scorer pressed a button and the corresponding time point (in milliseconds) was stored. In the second step, the same operator used a custom-made MATLAB program to re-inspect, frame-by-frame, the time period around the tagged time point (±2 sec) and to select the video frame that immediately preceded the change in facial expression. In this step, each event was also accurately re-evaluated and all cases in which a clear change in facial expression was not confirmed were marked for rejection. For this whole procedure, the scorer remained blind to the experimental condition of each video. Finally, tagged events that occurred after 1 sec from the end of a clip and before the beginning of the subsequent clip (i.e., during interstimulus black frames) were also automatically discarded.

EMG Data Analysis

A facial EMG signal was derived from two EEG electrodes located below the two eyes, on the cheeks, approximately above the zygomatic muscles. The continuous signal of these two channels was referenced on homolateral, preauricular electrodes, and band-pass filtered between 30 and 200 Hz (zero-phase Hamming-windowed finite impulse response filter; cutoff frequencies 26.25–203.75 Hz, −6 dB). A notch filter at 50 Hz was also applied (cutoff frequencies 46–54 Hz, −6 dB).

Variations in facial EMG activity were evaluated to confirm the expected association between tagged events and changes in facial expression. Moreover, given that the estimation of expressive changes based on visual inspection of the video clips may be inaccurate with respect to the actual beginning of muscular activity, the EMG signal was also inspected to determine the specific onset of each tagged event (Fiacconi & Owen, 2015). In particular, for the EMG inspection procedure, the root mean square of the filtered signals was computed using a moving-window approach (1-sec length; 1 time point steps). Then, 8-sec-long data segments, including 4 sec before and 4 sec after the manually tagged onset of changes in facial expression, were extracted. Each individual event was visually inspected using a custom-made MATLAB function, and the onset of the increase in EMG activity was marked. Cases for which a clear increase in EMG activity were not identified were excluded from further analyses. Finally, for all the retained episodes, 8-sec-long data segments centered on the EMG activation onset were extracted and the total signal power (30–200 Hz) was computed in 2-sec epochs using the Welch's method (*pwelch* MATLAB function; 8 Hamming windows with 50% overlap). The mean power in the 4 sec after onset time were compared to the mean power in the 4-sec epoch preceding the same time point at group level (i.e., after within-subject averaging across episodes). The ratios between the two data segments (post/pre) were also compared across experimental conditions (ES, FE) to investigate potential differences.

EEG Data Analysis

Continuous EEG recordings performed during the ES/FE task and in the resting-state condition were band-pass filtered between 0.5 and 45 Hz (Kaiser-windowed finite impulse response filter; −0.01 dB passband gain, −40 dB stopband gain, 0.49 Hz rolloff). All EEG traces were visually inspected to identify and mark bad channels containing clear artifactual activity. In addition, resting-state (but not task-related) data were divided into nonoverlapping 4-sec-long epochs, and epochs containing strong artifacts were rejected upon visual inspection. Then, an independent component analysis was performed in EEGLAB (Delorme & Makeig, 2004) to remove signal components reflecting ocular, muscular, and electrocardiograph artifacts. Rejected bad channels were subsequently interpolated using spherical splines.

After preprocessing, all task-related EEG traces were rereferenced to average reference and 4-sec-long data epochs immediately preceding the onset ($t = 0$ sec) of changes in facial expression were extracted. Finally, for each participant, the signal power in delta (1–4 Hz) and theta (4–8 Hz) frequency bands was computed for each epoch (1-sec Hamming windows, 50% overlap) and averaged across episodes of interest. Paired comparisons between experimental conditions were then performed at group level. Because local, sleep-like episodes are expected to precede, rather than follow, behavioral errors, we also performed a control analysis on 4-sec epochs that followed the onset of facial expression changes, expecting no significant differences between experimental conditions.

Detection and Analysis of Individual EEG Waves

In order to investigate whether possible differences in low-frequency activity reflected the occurrence of actual low-frequency waves similar to those of sleep, we used an automated algorithm to detect and analyze individual negative half-waves, as in previous work (Andrillon, Burns, MacKay, Windt, & Tsuchiya, 2021; Hung et al., 2013). Based on power-based results (see below), we focused on delta waves (< 4 Hz).

The average-referenced EEG signal of all electrodes was down-sampled to 128 Hz and band-pass filtered using a type-2 Chebyshev filter (25-dB attenuation in the stopband [0.1, 10 Hz]; 3-dB attenuation in the pass-band [0.5, 4 Hz]). Then, the algorithm identified all negative

half-waves with duration between 0.125 and 1.0 sec (from first [positive-to-negative] to second [negative-to-positive] zero-cross). For each wave, we extracted the negative peak amplitude and its position in time. Then, a minimum amplitude threshold was defined for each channel as the 25th percentile of the distribution of all negative amplitudes of slow waves detected during the FE condition. This approach allowed to exclude small signal deflections potentially reflecting artifacts or oscillations with no physiological significance. Finally, we computed the density (waves per second) and the mean amplitude (μV) of slow waves detected in the 4 sec that preceded changes in facial expression and compared the obtained values across experimental conditions, as described above.

Analysis of Task-related EEG Activity

Specific analyses were planned to determine whether potential differences between experimental conditions could be explained by differences in task-related demands (i.e., ES vs. FE) rather than emotion regulation failures per se.

In a first analysis, signal power estimates computed in the 4 sec preceding changes in facial expression were divided by task-related power values to account for potential overall (non-time-locked) differences between ES and FE. In particular, task-related brain activity values were estimated for each electrode by computing the median signal power across 2-sec-long nonoverlapping epochs covering each task EEG recording. The obtained ratios were then compared across ES and FE.

A second analysis was performed on data of the ES condition only. Specifically, for each participant, we calculated the Spearman's correlation coefficients between mean power values computed across nonoverlapping 2-sec epochs recorded during the presentation of each video clip and the corresponding ratings expressing the effort needed to suppress emotional responses. All video clips that were associated with a change in facial expression were excluded from this computation. A one-sample test based on *z*-scored transformed correlation values (averaged across trials TS1-A and TS1-B) was performed to identify potential group-level effects.

Source Modeling of EEG Data

The broadband signals of EEG epochs corresponding to the 4 sec immediately preceding the onset of changes in facial expression were source modeled using *Brainstorm* (Tadel, Baillet, Mosher, Pantazis, & Leahy, 2011). Specifically, the conductive head volume was modeled using a three-layer symmetric boundary element method (OpenMEEG BEM; Gramfort, Papadopoulo, Olivi, & Clerc, 2010; Kybic et al., 2005) and the default ICBM152 anatomical template. A standard set of electrode positions was used to construct the forward model. The source space was constrained to the cerebral cortex, which was modeled as a 3-D grid of 15,002 vertices. The inverse matrix was computed using the standardized low-resolution brain electromagnetic tomography constraint with a regularization parameter equal to 10^{-2} λ. Finally, the signal power was computed for each vertex in source space using the Welch's method (1-sec Hamming windows, 50% overlap).

Analysis of Actigraphic Data

The actigraphic data of the nights that preceded the two experimental visits were analyzed using the MotionWare Sleep Analysis software (CamNTech). Two parameters, corresponding to assumed sleep time (total elapsed time between sleep onset and morning awakening) and actual sleep time (time from sleep onset to morning awakening minus all epochs categorized as wake), were compared between ES and FE to exclude potential systematic differences in sleep quantity and quality across experimental conditions. We then tested the possible correlation of actual sleep time, an index reflecting the amount of obtained "good quality" sleep, with the number of ES failures in the ES condition. Upon request by an anonymous reviewer, the same correlation was tested for two additional parameters, corresponding to sleep efficiency (percentage of actual sleep time with respect to time in bed) and sleep fragmentation index (the percentage of movement time and of periods of immobility < 1 min relative to sleep time).

Statistics

All analyses of behavioral and EEG/EMG data collected during the ES/FE task were performed on the first task session (TS1) in order to avoid potential confounding effects related to extended practice with partially different tasks in ES and FE experiments. However, additional control analyses were also performed using all available task sessions (TS1–3; Figure 7), with and without the application of a data-normalization procedure. Specifically, the band-limited signal power of each task-session was normalized by expressing it as a ratio with respect to the signal power of the preceding resting-state recordings (median across 2-sec nonoverlapping epochs).

Comparisons of EEG signal power across experimental conditions were performed using paired *t* tests and a permutation-based suprathreshold cluster-mass correction (Nichols & Holmes, 2002). This method is similar to a cluster-size correction but also allows for the identification of relatively small but strongly significant electrode clusters. In brief, each contrast was repeated after shuffling the labels of the two experimental conditions and all clusters of significant electrodes were identified ($p_{unc.} < .05$). Then, we computed the sum of test statistics across electrodes belonging to the same cluster and the maximum obtained value was saved in a frequency table. A minimum cluster-mass threshold corresponding to the 95th percentile of the resulting distribution was

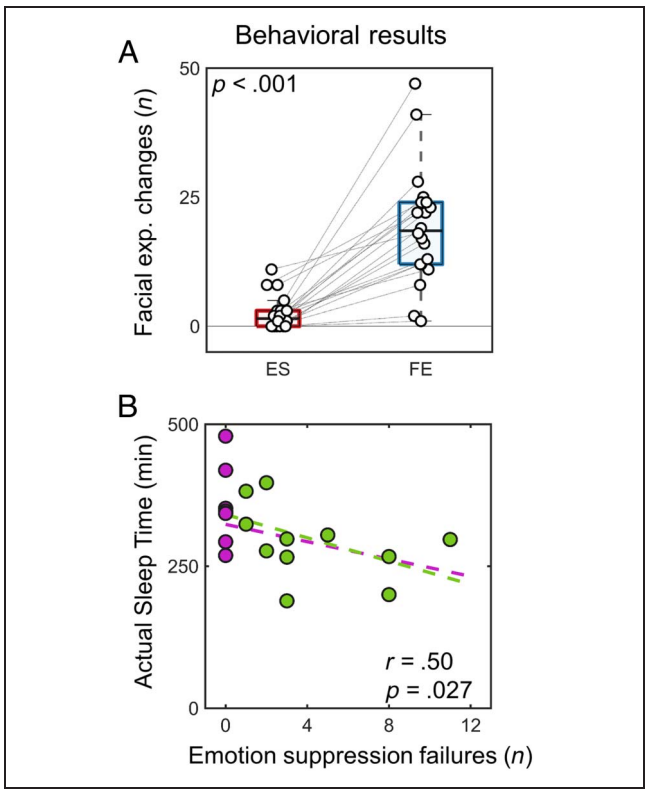

Figure 2. (A) Changes in facial expression across the two experimental conditions (TS1, $n = 20$; $p < .001$; Nperm = 10,000). In boxplots, the horizontal black line indicates the median, whereas the bottom and top edges of the box indicate the 25th and 75th percentiles. The whiskers extend to the most extreme data points within ±2.7 σ. (B) Relationship (Spearman's rho) between the number of ES failures and actual sleep time in the night preceding the experimental session ($n = 19$; $p = .0270$; Nperm = 10,000). This analysis was performed on 19 participants because of missing actigraphic data in one participant. The participants who showed no changes in facial expression during TS1 are marked with red color. The remaining participants are displayed using green color. Dashed lines represent the least-square fit for the distributions including all participants (purple) or only participants who had at least one facial expression change (green).

applied to correct for multiple comparisons. For comparisons performed at scalp level, the analyses were restricted to 51 "internal" electrodes in order to minimize the possible impact of residual artifactual activity in channels located near the eyes or on the temporal/neck muscles (Hung et al., 2013). Moreover, permutation tests were used to assess the statistical significance of comparisons regarding behavioral and subjective variables and of correlations computed using the Spearman's correlation coefficient. Specifically, for each comparison between experimental conditions, a null distribution was generated by repeatedly shuffling the condition labels and recomputing the test statistics. For correlations, values of one of the two variables were randomly shuffled and correlation coefficients were recomputed at each iteration of the permutation procedure. Of note, for all cases in which the number of possible data recombinations was greater than 10,000, this value was used to approximate the null distributions. In all other cases, the exact number of possible data recombinations (Nperm) was used. Finally, given that different numbers of data segments were entered in the analyses for ES and FE, and that this could affect the estimation of mean values per condition, bootstrap tests were also performed to verify the reliability of obtained results. In particular, for each of 10,000 bootstrap iterations (Nboot), only one data segment from ES and one data segment from FE were used and compared. The 95% confidence intervals (CIs [2.5th, 97.5th]) of the obtained bootstrap distribution were estimated to evaluate test significance. All statistical analyses were performed in MATLAB. Effect sizes were reported using Hedge's g (g) for paired comparisons and the Spearman's correlation coefficient (r) for correlations.

RESULTS

Behavioral Results

During the first task session (TS1), all participants showed a lower number of facial expression changes in ES (2.5 ± 3.2) relative to FE (19.3 ± 11.3) condition ($p < .001$, $|z| = 3.791$, $g = 1.97$; Figure 2A). Importantly, we found a negative correlation between actual sleep time in the night preceding the ES experiment and the absolute number of ES failures ($p = .027$, $r = -.50$; Figure 2B). Consistent but nonsignificant correlations were also obtained using

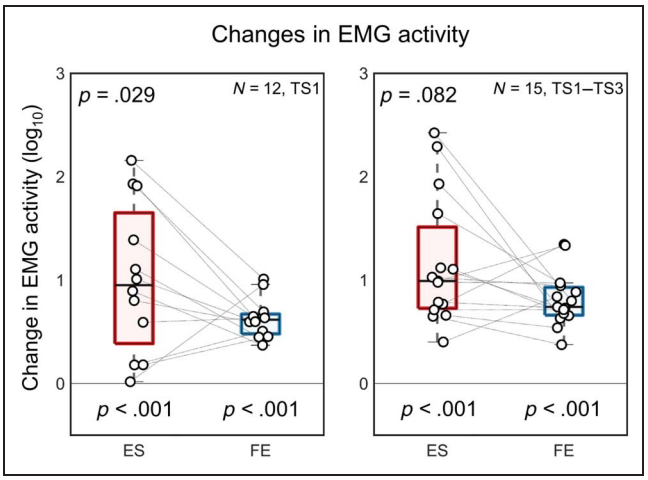

Figure 3. Variations of EMG activity associated with changes in facial expression. Signal power changes (30–200 Hz) were computed as the ratio between EMG activity in the 4 sec after the onset of facial expression changes and EMG activity in the 4 sec that preceded this event. Values were log-transformed for display purposes. Here, values greater than 0 indicate an increase. The panel on the left shows changes in EMG activity in the 12 participants who had at least one facial expression change in the first task session (TS1; Nperm = 4,096), whereas the panel on the right shows changes in EMG activity in the 15 participants who had at least one facial expression change across all the three task sessions (TS1–3; Nperm = 10,000). In boxplots, the horizontal black line indicates the median, whereas the bottom and top edges of the box indicate the 25th and 75th percentiles. The whiskers extend to the most extreme data points within ±2.7 σ.

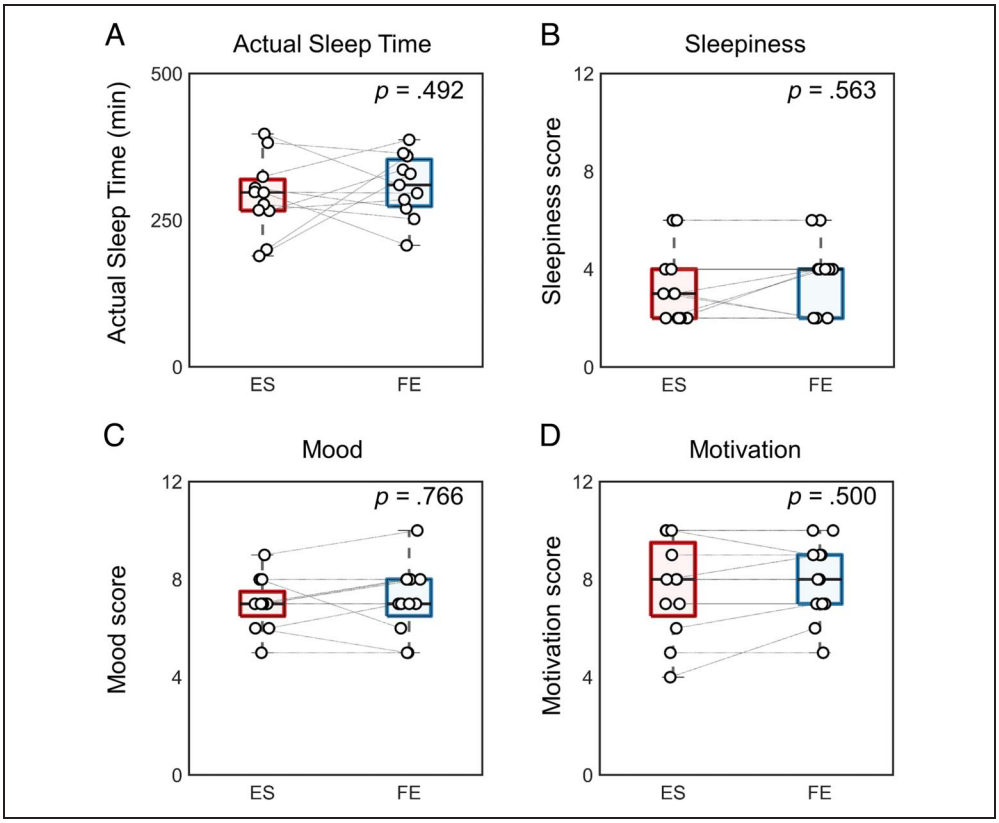

Figure 4. (A) Total minutes of sleep in the night that preceded each experimental session (n = 11). (B–D) Subjective scores (1–10) for sleepiness, mood, and motivation in the two experimental conditions (n = 12; Nperm = 4,096). No systematic differences were found between any of the tested parameters. In boxplots, the horizontal black line indicates the median, whereas the bottom and top edges of the box indicate the 25th and 75th percentiles. The whiskers extend to the most extreme data points within ±2.7 σ.

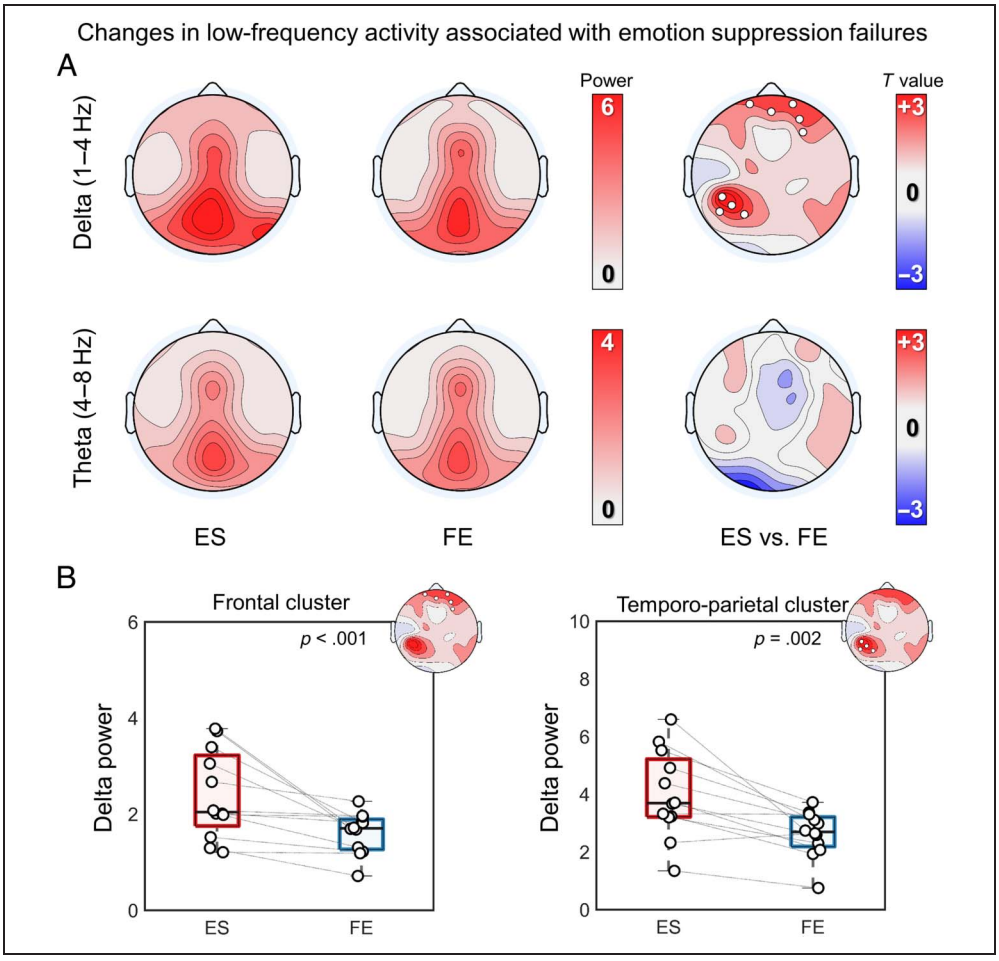

Figure 5. Low-frequency EEG activity associated with ES failures. (A) Topographic plots on the left show absolute values of delta (top) and theta (bottom) power for the two experimental conditions (ES and FE) in the 4 sec that preceded changes in facial expression. Topographic plots on the right show the statistical comparison between experimental conditions for the two frequency bands. White dots mark $p < .05$, cluster-mass correction (TS1, n = 12; 9 significant electrodes; Nperm = 4,096). (B) The plots show the data of individual participants for the two significant clusters reported in A. Mean values per participant were computed using 4.1 ± 3.2 episodes, range 1–11, for ES, and 22.9 ± 11.4 episodes, range 11–47, for FE. In boxplots, the horizontal black line indicates the median, whereas the bottom and top edges of the box indicate the 25th and 75th percentiles. The whiskers extend to the most extreme data points within ±2.7 σ.

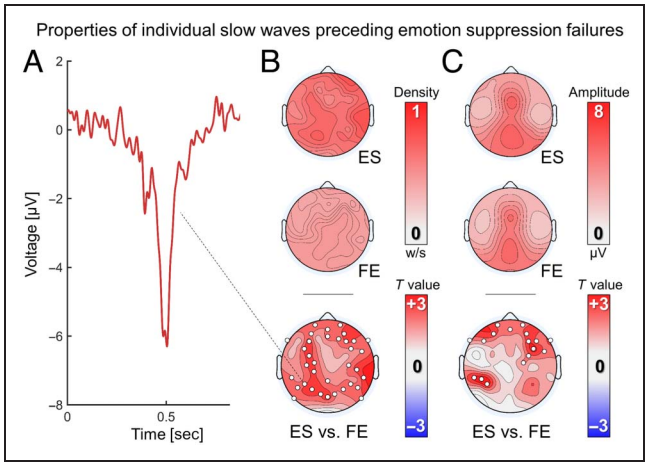

Figure 6. Density and amplitude of slow waves preceding ES failures. (A) Average waveform of the slow waves detected in a left parietal electrode within the 4 sec that preceded changes in facial expression during the ES task. (B–C) Topographic plots showing average slow-wave density and amplitude during FE (first row) and ES (second row), and the comparisons between experimental conditions obtained for the same parameters (third row). White dots mark $p < .05$, cluster-mass correction (TS1, $n = 12$; Nperm = 4,096).

relative measures of sleep quality, such as sleep efficiency ($p = .340$, $r = -.23$) and sleep fragmentation index ($p = .323$, $r = .23$). Of note, self-reported mood showed no relationship with emotion regulation failures ($p = .317$, $r = -.23$).

Twelve participants (mean age = 26.7 ± 3.1, 8 men) had at least one ES failure in the ES condition and were thus included in further analyses. Specifically, these participants had, on average, 4.1 ± 3.2 facial expression changes in the ES condition, a value still significantly lower than the one observed in the FE condition (22.9 ± 11.4; $p < .001$, $|z| = 2.980$, $g = 2.14$). In the same participants, the analysis of variations in EMG activity (30–200 Hz) of the zygomatic muscle confirmed that marked facial expression changes were associated with significant activity increases in both experimental conditions (ES: $p < .001$, $|z| = 1.682$, $g = 0.72$; FE: $p < .001$, $|z| = 2.581$, $g = 1.06$; Figure 3). Relative EMG changes tended to be stronger in ES ($p = .029$, $|z| = 1.842$, $g = 0.79$), although this effect was not confirmed in the 15 participants (mean age = 26.6 ± 2.8, 9 men) who had at least one ES failure across all three task sessions (TS1–3; $p = .082$, $|z| = 1.672$, $g = 0.63$). A visual re-inspection of video recordings in ES revealed a relative variability in the manifestation of suppression failures, with some participants showing "explosive" losses of control, and others only presenting minimal changes in facial expression. Instead, reactions tended to be more homogeneous across participants during FE.

Sleep Quality, Vigilance, and Mood

Several control analyses were performed to exclude potential systematic differences between experimental

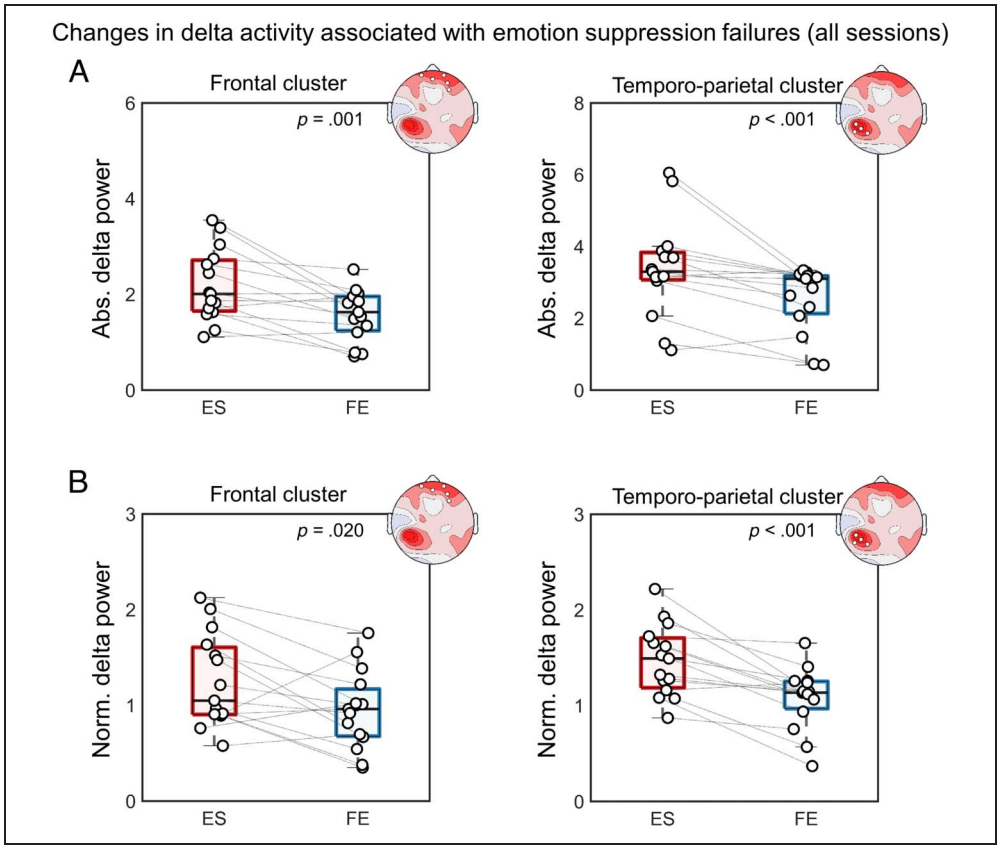

Figure 7. (A) Absolute delta EEG activity associated with ES failures in the 15 participants that presented at least one ES failure across the three task sessions of ES (TS1–3; Nperm = 10,000). The plots show the data of individual participants for two regions of interest corresponding to the frontal (left plot) and the left temporo-parietal (right plot) clusters identified in the analysis based on the 12 participants who had at least one ES failure in TS1. Mean values per participant were computed using 9.1 ± 7.9 episodes, range 1–26, for ES, and 60.0 ± 27.0 episodes, range 19–122, for FE. (B) Same as (A), but normalized delta EEG activity is shown. Specifically, delta power was normalized by computing the ratio with respect to median delta power of the resting-state recordings obtained before the beginning of the corresponding task session.

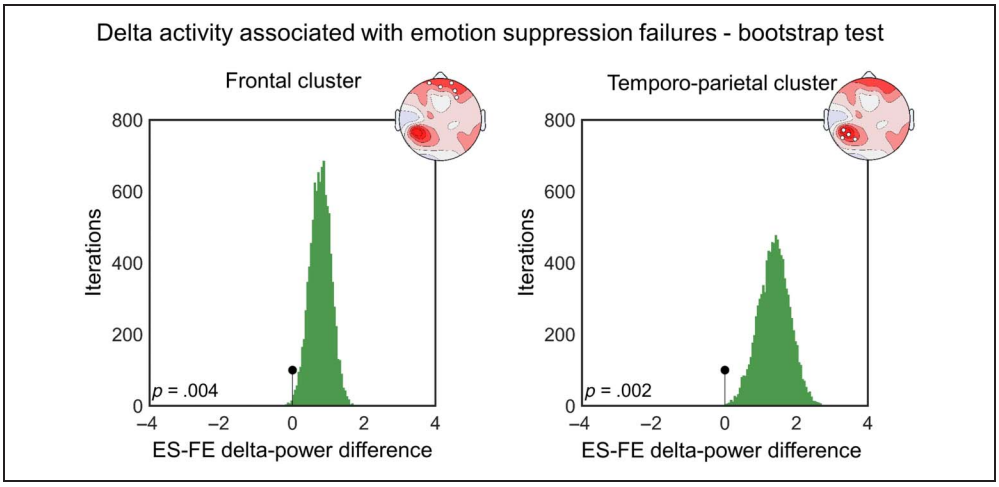

Figure 8. Results of the bootstrap tests investigating differences in delta power between ES and FE in the frontal (left histogram) and the left-parietal (right histogram) clusters. For each iteration of the bootstrap test (Nboot = 10,000) and for each participant, delta activity was computed within the cluster of interest (average) in the 4 sec preceding one randomly selected change in facial expression for both ES and FE. Then, the difference between experimental conditions was computed. This analysis was performed for the 12 participants who had at least one ES failure during TS1.

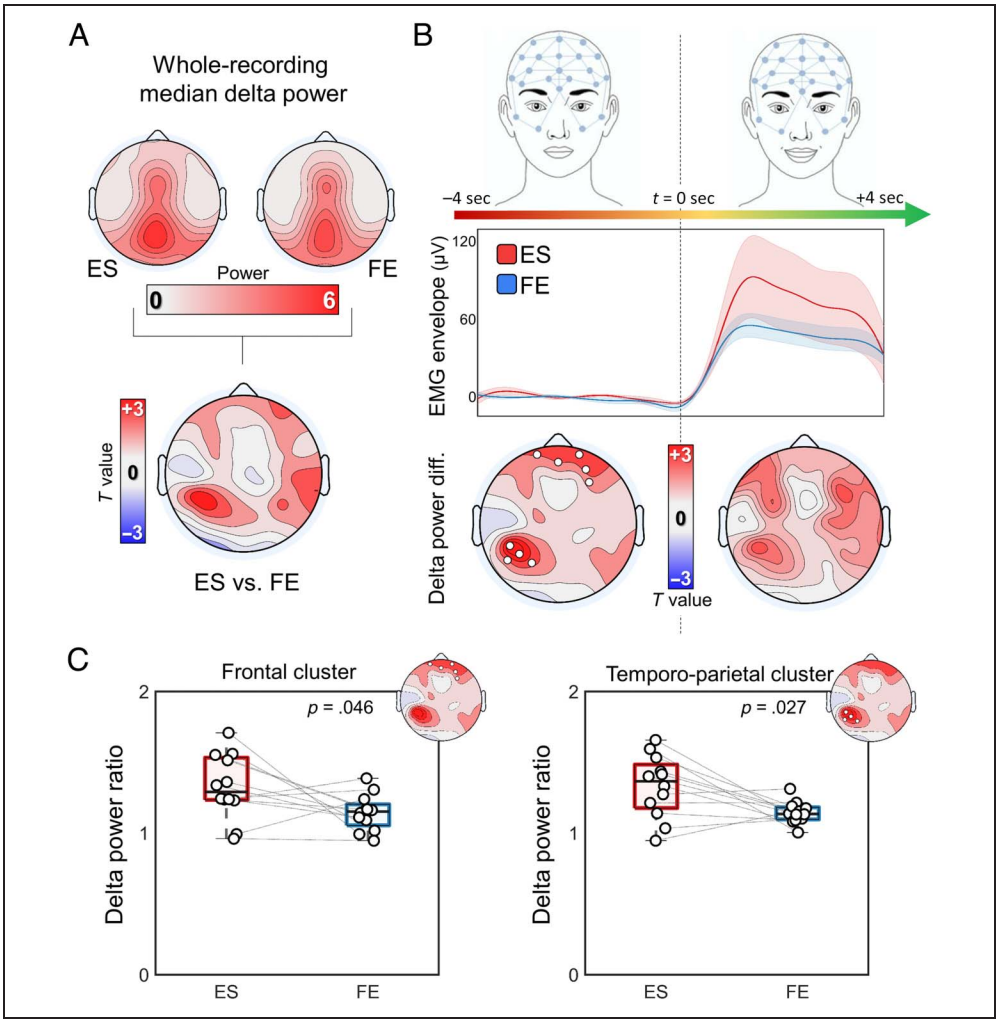

Figure 9. (A) Median task-related delta power computed for the entire EEG recordings. The topographic plot at the bottom shows the contrast between delta activity of ES and FE (TS1, $n = 12$; all $p > .05$, cluster-mass correction; Nperm = 4,096). (B) Contrasts between experimental conditions in the 4 sec before and in the 4 sec after change in facial expression ($t = 0$ sec). The central plot shows the variation of EMG activity (envelope of rectified filtered EMG signal) in the same time windows. Values were normalized before averaging by subtracting the mean EMG activity computed in the 4 sec before the onset of changes in facial expression ($t = 0$ sec). Shaded areas correspond to the standard error of the mean. For topographic plots, white dots mark $p < .05$, cluster-mass correction. (C) Delta-power ratios computed between signal power in the 4 sec preceding changes in facial expression and median task-related signal power. The plots show the data of individual participants for the frontal and left temporo-parietal clusters shown in Figure 5. In boxplots, the horizontal black line indicates the median, whereas the bottom and top edges of the box indicate the 25th and 75th percentiles. The whiskers extend to the most extreme data points within ±2.7 σ.

conditions in the 12 examined participants. First, we investigated possible differences in time spent in bed and in actual sleep time between the nights that respectively preceded ES and FE experiments (of note, this analysis was performed on 11 participants because of missing actigraphic data from one volunteer; age 28 years, male). We found no evidence of systematic differences (assumed sleep time: ES = 373.7 ± 50.0 min, FE = 395.7 ± 62.3 min; $p = .341$, $|z| = 1.004$; actual sleep time: ES = 291.1 ± 64.0 min, FE = 308.6 ± 53.5 min; $p = .492$, $|z| = 0.720$; Figure 4A). Similarly, we found no systematic differences in sleepiness (ES = 3.3 ± 1.5, FE = 3.5 ± 1.5; $p = .563$, $|z| = 0.904$; Figure 4B), mood (ES = 7.0 ± 1.0, FE = 7.2 ± 1.4; $p = .766$, $|z| = 0.632$; Figure 4C), and motivation (ES = 7.7 ± 2.0, FE = 7.9 ± 1.6; $p = .500$, $|z| = 1.134$; Figure 4D). Similar results were obtained when considering 19 participants for actual sleep time (missing data in one participant) and all 20 participants for the Likert scales (*data not shown*).

Delta and Theta EEG Activity

We next evaluated whether increases in low-frequency (delta/theta) activity preceded the occurrence of ES failures (Figure 5). To this aim, we compared the signal power computed in the 4 sec preceding changes in facial expression across ES and FE conditions. No significant differences were found for theta activity (4–8 Hz; $p_{cor.} > .338$). Instead, the analysis revealed significant delta activity (1–4 Hz) increases within the frontal and left temporo-parietal areas in ES relative to FE ($p_{cor.} < .05$; $g > 0.53$). Such differences corresponded with significant increments in the density and amplitude of individual slow waves (< 4 Hz; Figure 6) in ES. This result was confirmed in the sample of 15 participants who had at least one ES failure across all three task sessions (Figure 7; $p < .05$), as well as using a bootstrap test including only one data epoch per condition for each participant (Figure 8; $p < .05$). Of note, we found no significant differences between the two experimental conditions when comparing delta activity in the 4 sec after the onset of facial expression changes (Figure 9B; $p_{cor.} > .126$).

Given that changes in low-frequency activity may represent a signature of brain functional fatigue caused by insufficient sleep, we then explored the possible relationship between delta activity and actual sleep time the night preceding the ES experiment. We found that median delta power in frontal electrodes tended to be negatively correlated with sleep time ($p = .087$, $r = -.55$), so that shorter sleep duration tended to be associated with higher levels of failure-related delta activity. A similar relationship was not found for left temporo-parietal electrodes ($p = .500$, $r = .23$).

Higher Frequency Bands

Additional analyses were performed to verify whether the observed association between changes in delta activity and ES failures were frequency-band specific. In particular, we investigated possible differences between ES and FE and found no significant electrode clusters in the frequency ranges of alpha (8–12 Hz; $p_{unc.} > .05$), sigma (12–16 Hz; $p_{unc.} > .05$), and beta (18–30 Hz; $p_{cor.} > .08$) activities (8 Hamming windows, 50% overlap; Figure 10).

Possible Confounding Effects of Task Demands

Differences in delta power between experimental conditions could have emerged because of differences in task demands. Additional analyses were thus performed to investigate this possibility.

First, we compared the median delta power of the entire recordings of ES and FE to identify potential differences in overall task-related brain activity. This analysis revealed no significant differences between the two experimental conditions ($p_{cor.} > .053$; Figure 9A). In line with this, differences between ES and FE remained significant in the frontal and the left temporo-parietal clusters when comparing delta-power ratios computed with respect to median task-related signal power ($p < .05$; Figure 9C).

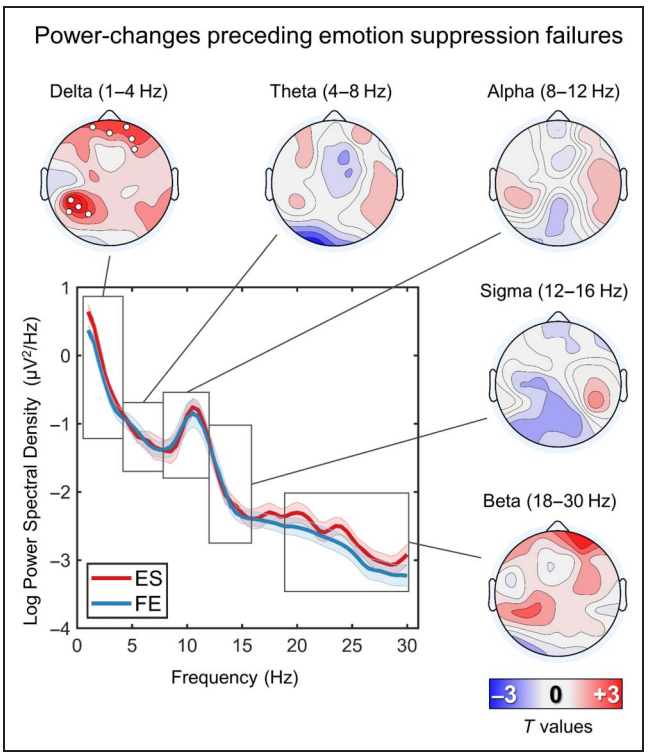

Figure 10. Differences in brain activity preceding the onset of changes in facial expression in the two experimental conditions (ES and FE). The central plot shows the power spectral density (PSD) computed across the electrodes presenting a significant difference in delta activity between ES and FE (white dots). Additional comparisons were performed for other typical frequency bands, including theta, alpha, sigma, and beta. For these bands, signal power was computed within 2-sec-long epochs using the Welch's method (8 Hamming windows, 50% overlap). White dots mark $p < .05$, cluster-mass correction (TS1, $n = 12$; Nperm = 4,096).

Next, we evaluated whether delta power was correlated with the effort required to suppress emotional responses in the EF conditions, as determined based on the scores provided by an independent sample of participants (Table 1). No significant correlations were found between delta power and effort scores ($p_{cor.} > .268$; Figure 11). Together, these results indicate that differences in delta power did not simply reflect specific task-related demands of the ES condition.

Source Modeling Analysis of Delta Activity

In order to identify the actual sources of changes in delta activity observed in the ES condition, the same analysis shown in Figure 5 was repeated after source reconstruction of the EEG signals (standardized low-resolution brain electromagnetic tomography; but very similar results were obtained using dynamical Statistical Parametric Mapping). The obtained results are shown in Figure 12 ($p_{cor.} < .05$, $g > 1.26$; cluster-forming threshold set to uncorrected $p_{unc.} < .001$). In particular, we found significant clusters in the bilateral anterior cingulate and medial frontal cortex, the anterior insula, the right precuneus, and the right motor/premotor cortex, including the middle and inferior frontal gyri.

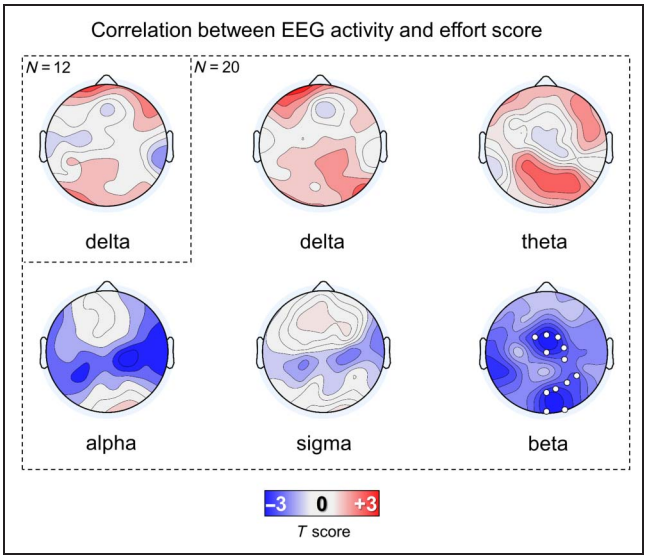

Figure 11. Correlation between delta power and ES effort. For each participant, we calculated the Spearman's correlation coefficients between mean signal power values during the presentation of each video clip and the corresponding ratings expressing the effort needed to suppress emotional responses. A one-sample test based on *z*-transformed correlation values was performed to identify potential group-level effects. The top–left topographic plot shows the results obtained for delta activity in the 12 participants who had at least one ES failure in TS1 (Nperm = 4,096; mean included clips per participant = 20.0, range 14–22). Remaining topographic plots have been obtained using all 20 participants (Nperm = 10,000; mean included clips = 20.8, range 14–22). White dots mark $p < .05$, cluster-mass correction. No significant correlations were found for delta power, either using data from 12 (all $p_{cor.} > .569$) or 20 participants (all $p_{cor.} > .195$). A significant (negative) correlation was instead found in the beta band (12 significant electrodes; $p_{cor.} < .05$, $g > 0.66$), so that higher suppression effort was associated with lower levels of beta activity in centro-frontal and occipital electrodes. All analyses were performed using data collected in TS1.

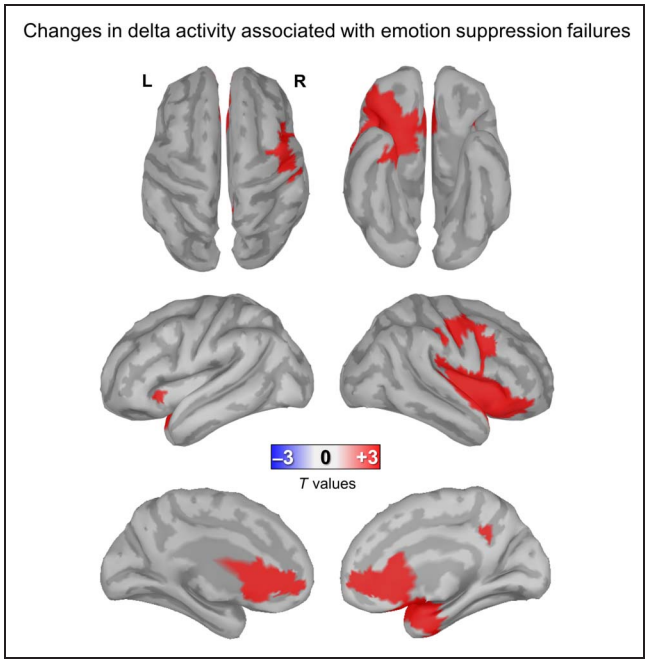

Figure 12. Delta EEG activity associated with ES failures. Significant differences ($p < .05$, cluster-mass correction; TS1, $n = 12$; 1,420 significant vertices; Nperm = 4,096) in delta activity between ES and FE in the 4 sec that preceded the onset of changes in facial expression.

DISCUSSION

Previous evidence indicates that frontal, parietal, and limbic brain regions are activated during successful ES (Langner et al., 2018; Dörfel et al., 2014; Frank et al., 2014; Kohn et al., 2014). However, the neural correlates of ES failures were unknown. Here, we demonstrated that self-control failures in an ES task are preceded by increases in low-frequency, sleep-like activity over frontal, insular, and parietal regions. In addition, a positive correlation was found between actual sleep time the night before practice with the ES task and the absolute number of self-control failures, so that shorter sleep duration was associated with a poorer behavioral performance. These results indicate that intrusions of sleep-like activity in the brain network responsible for emotion regulation affect the efficacy of ES in healthy individuals. In this light, the occurrence of local, sleep-like episodes may offer a possible neurophysiological mechanism for previous observations regarding the effects of sleep loss on emotion regulation.

Neural Correlates of ES Failures

Previous work showed that a neural network encompassing frontal, cingulate, and parietal regions is recruited

during ES tasks (Langner et al., 2018). However, to the best of our knowledge, no study investigated the neurophysiological events underlying failures in ES. Here, we showed that increases in delta (1–4 Hz) activity over frontal and parietal areas encompassing the brain network involved in impulse control and emotion regulation precede the occurrence of ES failures. Of note, although delta activity is considered as a typical hallmark of sleep, a growing body of evidence indicates that temporary regional increases in low-frequency, sleep-like activity may often occur also during wakefulness. Indeed, seminal work in rats demonstrated that locally synchronized neuronal *off-periods* similar to those underlying the generation of sleep slow waves (0.5–4 Hz) occur more frequently as a function of time spent awake (Vyazovskiy et al., 2011). Studies in humans also revealed that such increases are of greater magnitude in brain areas that are more intensively "used" during the waking period (Bernardi et al., 2019; Quercia, Zappasodi, Committeri, & Ferrara, 2018; Hung et al., 2013). Interestingly, the occurrence of sleep-like events in brain areas related to the execution of specific activities has been shown to potentially determine temporary behavioral impairments in a variety of different tasks, including impulse control, visuo-motor coordination, and stimulus categorization (Nir et al., 2017; Bernardi et al., 2015). Based on these observations, it was suggested that local sleep-like episodes may represent the neurophysiological correlate of "functional fatigue" and sleep need (Andrillon et al., 2019). Our present results extend the previous literature by demonstrating that local, sleep-like episodes occurring in brain areas involved in emotion regulation precede—and thus, represent a potential cause of—failures in ES. Interestingly, our results also showed, for the first time, that sleep-like episodes related to behavioral errors do not occur only after sleep deprivation or extended task practice but may also be observed in apparently well-rested individuals, during the first hours of the morning. This finding has important implications for our understanding of the actual influence of local sleep regulation on human behavior.

Poor Sleep Quality and ES Failures

We found that emotion regulation failures—measured as the inability to voluntarily suppress changes in facial expression—are more common in individuals who had shorter sleep duration. Moreover, shorter sleep tended to be associated with higher delta activity over frontal regions in those who failed at suppressing their facial expressions. These results are consistent with evidence indicating the existence of a tight interplay between sleep and emotion regulation. Indeed, both acute and chronic sleep loss determine alterations of mood and emotional reactivity, as well as increased stress, anxiety, and depression (Beattie, Kyle, Espie, & Biello, 2015; Mauss, Troy, & LeBourgeois, 2013; Minkel et al., 2012; Anderson & Platten, 2011; Zohar, Tzischinsky, Epstein, & Lavie, 2005). The sleep-deficient individual may often display greater emotional reactivity to stimulus salience independently from valence, biased cognitive evaluation, and flawed behavioral expression (Ben Simon et al., 2015; Gujar, McDonald, Nishida, & Walker, 2011). In addition, affective/mood disorders and sleep disturbances are often found associated, thus implying a potential role of inadequate sleep in the development or worsening of these clinical conditions (Goldstein & Walker, 2014; Benca, Obermeyer, Thisted, & Gillin, 1992). In line with this, good sleep quality has been associated with enhanced emotional well-being and is commonly considered as a protective factor for humans' emotional functioning (Palmer & Alfano, 2017; Watling, Pawlik, Scott, Booth, & Short, 2017; Gruber & Cassoff, 2014; van der Helm et al., 2011).

Neuroimaging studies demonstrated that one night of sleep deprivation impairs the medial prefrontal cortex (mPFC) top–down regulation on limbic brain areas, resulting in an "executive dysfunction" (Ben Simon, Rossi, Harvey, & Walker, 2020; Gruber & Cassoff, 2014; Gujar, Yoo, Hu, & Walker, 2011; Yoo et al., 2007). Notably, even a single night of slight, 1- to 2-hr sleep curtailment has been shown to robustly impair mPFC activity and its related limbic functional connectivity, as well as to determine increased emotional distress in healthy adult individuals (Killgore, 2013). Interestingly, the occurrence of local sleep-like episodes in the mPFC could offer a functional explanation for alterations in top–down emotional regulation observed after sleep loss. Nevertheless, previous work suggested that total sleep deprivation may lead to a reduction, rather than an increase, in facial expressiveness (Minkel, Htaik, Banks, & Dinges, 2011). These different findings could be reconciled by considering the methodological differences between the present and previous work. First, in our study, but not in the work by Minkel et al., participants were specifically required to suppress their emotional responses. Because local sleep-like episodes affect performance in the task at hand, an effect on emotional regulation may become evident only if participants explicitly attempt to suppress their emotional responses. Second, previous work focused on the effects of complete sleep deprivation, whereas here participants had a normal sleep opportunity the night before each experimental visit. Indeed, the behavioral effects of local sleep-like episodes may change based on their numerousness and spatial extent, as well as according to which brain areas are affected. All these factors have been suggested to change with time spent awake and with the types of performed activities (Fattinger, Kurth, Ringli, Jenni, & Huber, 2017; Bernardi et al., 2015; Hung et al., 2013).

Limitations

Although the original sample included 20 participants, some of the analyses have been performed on a reduced sample of 12 participants. This is because ES failures are relatively uncommon in healthy young and rested

individuals. Importantly, though, the same analyses repeated after including additional experimental sessions and participants (n = 15; Figure 6) confirmed the results obtained in the reduced sample. In future investigations, sleep restriction or deprivation paradigms could be used to increase the incidence of ES failures and thus obtain a greater statistical power. In addition, here, we only used emotional stimuli with a positive valence, thus limiting the possibility to generalize our results to other emotional stimuli, such as negative and/or aversive stimuli. However, it should be noted that the expressive suppression strategy has been shown to rely on similar brain substrates for both negative and positive emotional stimuli (e.g., Morawetz, Bode, Derntl, & Heekeren, 2017; Paul, Simon, Kniesche, Kathmann, & Endrass, 2013; Korb, Grandjean, Samson, Delplanque, & Scherer, 2012; Dennis & Hajcak, 2009; Hajcak & Nieuwenhuis, 2006).

Conclusions

Our study shows that ES failures are associated with temporary, sleep-like episodes occurring in key brain areas involved in emotion regulation. Given previous observations indicating that the incidence of local sleep-like episodes increases with time spent awake, the same functional mechanism may contribute to emotional dysfunctions commonly observed following sleep restriction or deprivation. The present results may have broad and important implications. In particular, future studies should investigate whether specific individual factors may favor a faster build-up of or a greater vulnerability to local sleep-like episodes in specific brain areas or networks. Moreover, it will be important to clarify whether alterations in the local regulation of sleep need may be responsible for alterations of emotional regulation observed in psychopathological conditions.

Acknowledgments

This work was supported by intramural funds from the IMT School for Advanced Studies Lucca. The authors wish to thank Andrea Leo, Monica Betta, and Giacomo Handjaras, for helpful discussion during the planning of the main research project, Francesca Setti for help with data acquisition, and Demetrio Grollero for assistance during data analysis.

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Author Contributions

Giulia Avvenuti: Conceptualization; Formal analysis; Investigation; Visualization; Writing—Original draft; Writing—Review & editing. Davide Bertelloni: Formal analysis; Writing—Review & editing. Giada Lettieri: Methodology; Writing—Review & editing. Emiliano Ricciardi: Resources; Supervision; Writing—Review & editing. Luca Cecchetti: Methodology; Writing—Review & editing. Pietro Pietrini: Conceptualization; Resources; Supervision; Writing—Review & editing. Giulio Bernardi: Conceptualization; Formal analysis; Methodology; Supervision; Visualization; Writing—Original draft; Writing—Review & editing.

Diversity in Citation Practices

A retrospective analysis of the citations in every article published in this journal from 2010 to 2020 has revealed a persistent pattern of gender imbalance: Although the proportions of authorship teams (categorized by estimated gender identification of first author/last author) publishing in the *Journal of Cognitive Neuroscience (JoCN)* during this period were M(an)/M = .408, W(oman)/M = .335, M/W = .108, and W/W = .149, the comparable proportions for the articles that these authorship teams cited were M/M = .579, W/M = .243, M/W = .102, and W/W = .076 (Fulvio et al., *JoCN*, 33:1, pp. 3–7). Consequently, *JoCN* encourages all authors to consider gender balance explicitly when selecting which articles to cite and gives them the opportunity to report their article's gender citation balance. The authors of this article report its proportions of citations by gender category to be as follows: M/M = .597, W/M = .21, M/W = .08, and W/W = .113.

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