Abstract Several studies have examined the neural correlates of mood-related emotional processing in depression, showing a greater reduction of activity in the rostral anterior cingulate cortex (rACC) in response to pleasant relative to unpleasant stimuli in depressed individuals, and the opposite pattern in healthy controls. The present study aimed at examining whether frontal theta activity—an electrophysiological measure of rACC activity—could be a reliable EEG correlate of mood-related emotional processing in individuals with dysphoria. To this end, the EEG was recorded in 27 individuals with dysphoria and 29 individuals without dysphoria during an emotional imagery task, including pleasant, neutral and unpleasant scripts. Self-reported valence, arousal and vividness, and changes in frontal theta activity were measured during the task. Frontal theta activity was more reduced from baseline to the imagery of pleasant relative to unpleasant scripts in the group with dysphoria, whereas the opposite pattern of reduction was noted in the group without dysphoria. In addition, more severe depressive symptoms were correlated with greater reduction in frontal theta activity in response to pleasant, but not neutral and unpleasant, scripts. No differences between groups in subjective ratings were noted. Consistent with the key role of rACC activity in depression-related emotional dysregulation, these findings suggest that frontal theta activity may be an EEG correlate of mood-related emotional processing in dysphoria. The current study also suggests that dysphoria is more likely to be associated with abnormal processing of pleasant rather than unpleasant stimuli.

Keywords Depression · Dysphoria · EEG · Emotion · Frontal theta activity · Mood-related emotional processing

The identification of electrophysiological measures of mood-related emotional processing in dysphoria has important implications for improving our understanding of the mechanisms that underlie abnormal affective or motivational tendencies that characterize depression. Several lines of evidence have shown that depression is characterized by mood-related biases that facilitate the processing of negatively-valenced information (e.g., Goeleven et al. 2006; Jermann et al. 2009; Sigmon and Nelson-Gray 1992; for a review, see Gotlib and Joormann 2010). By facilitating the processing of unpleasant stimuli, mood-related biases have been shown to play a critical role in the etiology, maintenance and recurrence of depression (Beck 1976; Kilford et al. 2015; Teasdale 1988).

Moreover, there is growing evidence that depressed patients are characterized by reduced processing of pleasant relative to unpleasant verbal and pictorial stimuli and/or relative to control participants (e.g., Buodo et al. 2015; Nandrin et al. 2004; Shestyuk et al. 2005). Based on such evidence, it has
been hypothesized that in healthy individuals, attention may be oriented by default toward pleasant stimuli, whereas attention to threat may take the form of a sustained background analysis of possible sources of danger in the environment (Frewen et al. 2008; Winer and Salem 2016). When a significant source of danger is identified, healthy individuals are able to rapidly set their responding to danger and to interrupt the ongoing orientation towards pleasant stimuli. In this perspective, an impaired attentional orientation toward pleasant stimuli may represent a distinct form of abnormal emotional information that characterizes sub-clinical and clinical depression, as evidenced by quantitative analyses of dot-probe studies (Frewen et al. 2008). These findings also lend support to the hypothesis that the inability to use positive and rewarding stimuli to regulate negative mood is one of the key mechanisms that underlie emotion dysregulation in depression (Brockmeyer et al. 2015; Capeceletro et al. 2013; Gotlib and Joormann 2010).

In addition to attentional biases, a substantial number of studies have reported mood-related biases in emotional responding or regulation (e.g., Cook et al. 1992; Dunn et al. 2004; Sloan et al. 2001). Such findings have been interpreted according to the negative potentiation hypothesis and the positive attenuation hypothesis. The negative potentiation hypothesis postulates that the negative mood that characterizes depression contributes to potentiate emotional responding to unpleasant stimuli. This hypothesis is consistent with studies showing that depressed individuals are characterized by greater electrodermal activity or startle reflex amplitude in response to unpleasant stimuli compared to healthy controls (e.g., Cook et al. 1992; Sigmon and Nelson-Gray 1992). Alternatively, the positive attenuation hypothesis postulates that individuals with depression show reduced emotional reactivity to pleasant stimuli relative to neutral stimuli and/or healthy controls. Specifically, there is evidence that subjective evaluation and facial reactivity in response to pleasant, but not to unpleasant or neutral, stimuli is reduced in depressed individuals compared to healthy controls (e.g., Dunn et al. 2004; Sloan et al. 2001).

More recently, research has focused on examining the neural correlates of mood-related emotional processing in depression. Some studies have shown abnormal patterns of anterior cingulate cortex (ACC) activation in depressed patients during a variety of affective tasks, such as the emotional Stroop task and the emotional go/nogo task (for a review, see Pizzagalli 2011). Specifically, a greater activation of the rostral ACC (rACC) has been consistently found in response to unpleasant stimuli in depressed patients compared to healthy controls (e.g., Dichter et al. 2009; Elliott et al. 2002; Eugène et al. 2010; Mitterschiffthaler et al. 2008). Converging evidence has also reported that depressed patients show attenuated rACC activation in response to pleasant stimuli compared to healthy controls (e.g., Elliott et al. 2002; Eugène et al. 2010). Accordingly, rACC activity has been implicated as a mediator of the mood-related processing biases typically observed in depressed patients (Eugène et al. 2010).

Studies using high-resolution EEG and magnetoencephalographic (MEG) source modeling as well as intracranial recordings have provided evidence that the human ACC is involved in the generation of frontal theta activity (e.g., Asada et al. 1999; Nishida et al. 2004). More specifically, Pizzagalli et al. (2003) have reported that frontal theta activity is associated with cerebral metabolism in rACC by means of combined positron emission tomography (PET) and EEG measurements. Contrary to the theta activity with widespread scalp distribution, that has been linked to decreased alertness and drowsy states (Schacter 1977), theta activity with frontal midline distribution has been associated with mental effort and focused attention (e.g., Basar-Eroglu et al. 1992; Gevins et al. 1997). Consistent with the role of rACC as a key component of the “emotion circuit” (e.g., Bush et al. 2000), frontal theta activity has been also found to be implicated in emotional processing (Sammler et al. 2007) and in emotional regulation (Ertl et al. 2013). Specifically, Sammler et al. (2007) found a positive correlation between frontal theta activity and pleasantness of music excerpts in healthy participants: the lower the frontal theta activity, the lower the self-reported pleasantness during the listening of music excerpts. Consistent with these findings, decreases in frontal theta activity have been associated with reduced motivational salience of the stimulus and decreased individual emotional sensitivity and emotional involvement (Knyazev et al. 2009). In particular, Knyazev et al. (2009) reported that decreased low-frequency synchronization in response to emotional (angry, happy) vs. neutral faces was associated with reduced emotional sensitivity and emotional involvement during an explicit emotion recognition task. Emotional sensitivity was measured combining absolute self-reported ratings of angry and happy stimuli given on a scale from -100 (very hostile) to 100 (very friendly); emotional involvement was measured using a short questionnaire assessing individual attitude towards the presented faces during the task. In addition to frontal theta activity, midline posterior versus frontal (i.e., Pz-Fz) theta activity has been found to be associated with agentic extraversion (Wacker et al. 2010), and with motivational salience of emotional stimuli (Walden et al. 2015; Zhang et al. 2013).

The findings that rACC activity is implicated in depression-related emotional processing, that frontal theta activity originates from rACC, and that decreases in frontal theta activity are associated with reduced emotional involvement in healthy individuals and with anhedonia in depressed patients raise the pertinent question of whether frontal theta activity may be an EEG correlate of mood-related emotional processing in depression. However, to our knowledge, no studies have addressed this critical issue so far.

Another unresolved issue is whether mood-related processing biases are only apparent in individuals with clinically
significant depression (e.g., Elliott et al. 2002; Eugène et al. 2010), or can be reliably observed also in individuals with dysphoria, that is, individuals who report at least two or more current depressive symptoms, at least two weeks in duration, but are not formally diagnosed with major depression, minor depression or dysthymia (Judd et al. 1994, 1997). Indeed, although there is evidence indicating that mood-related processing biases also characterize individuals with sub-clinical depressive symptoms at the behavioral level (Joormann 2004; Peckham et al. 2010), it is still unclear whether changes in frontal theta activity may reflect mood-related processing biases in dysphoria at the neural level. This is surprising because mood-related emotional processing bias may serve to create the persistent negative mood and/or anhedonia that, in turn, may put a dysphoric individual at risk for a first-onset major depression compared to healthy controls (e.g., Horwath et al. 1992). Therefore, it is important to examine whether a mood-related emotional processing bias may be a psychophysiological marker associated with dysphoria, rather than being a mere correlate of clinical depression only (Pizzagalli 2011).

The present study aimed at examining whether frontal theta activity may be an EEG correlate of mood-related emotional processing in dysphoria. To this end, we examined frontal theta activity in individuals with and without dysphoria during an emotional imagery task, including pleasant, neutral and unpleasant conditions. The rationale for the use of emotional imagery paradigm is that it activates brain regions involved in the processing of emotional stimuli, it acts as an emotional amplifier, and it has been widely used with clinical populations (Holmes and Mathews 2010). Because decreases in frontal theta activity originating from rACC have been associated with reduced individual emotional involvement (Knyazev et al. 2009), it was hypothesized that dysphoric individuals would show a greater decrease in frontal theta activity from baseline to the imagery of pleasant relative to unpleasant stimuli compared to the group without dysphoria. On the basis of EEG and MEG findings showing that the rACC is the generator of frontal theta activity in the human brain (Asada et al. 1999; Nishida et al. 2004; Pizzagalli et al. 2003), frontal and frontocentral theta activity was expected to show this effect. In particular, an association between central and centroparietal theta activity and mood-related emotional processing was not hypothesized because of no known relation between posterior theta activity and rACC. In addition, we controlled whether alpha activity, which has been previously associated with depression (e.g., Davidson et al. 1999; Heller and Nitschke 1997), was implicated in the mood-related emotional processing bias in dysphoria.

Methods

Participants

In order to identify potential participants with dysphoria, 224 undergraduate students completed an online version of the Beck Depression Inventory-II (BDI-II; Beck et al. 1996; Italian version by Ghisi et al. 2006). The BDI-II is a valid and reliable self-report questionnaire that evaluates the severity of depressive symptoms in the past two weeks and is composed of 21 items. Responses are given on a four-point (0–3) Likert scale and scores range from 0 to 63, with higher scores indicating more severe depressive symptoms.

Undergraduates who scored equal to or greater than 12 on the online version of the BDI-II (n = 59) were preliminarily selected, given that a score of 12 has been reported as the optimal cut-off score to discriminate individuals with and without clinically significant depressive symptoms in the Italian population (Ghisi et al. 2006). In order to confirm the presence of depressive symptoms and to exclude individuals with major depression, minor depression or dysthymia, each participant scoring equal to or greater than 12 on the online version of BDI-II was administered a paper-and-pencil version of the BDI-II and the mood episode module (module A) of the Structured Clinical Interview for the DSM-IV Axis I (SCID-I; First et al. 1997; Italian version by Mazzi et al. 2000) approximately one week after the initial screening. Twenty-seven participants [25 females and 2 males; age, mean (M) = 21.0, standard deviation (SD) = 1.6; BDI-II score, M = 16.3, SD = 4.4], who scored equal to or greater than 12 on both versions of the BDI-II and had at least two current depressive symptoms, at least two weeks in duration, without meeting the diagnostic criteria for major depression, minor depression or dysthymia, were assigned to the group with dysphoria. According to previous literature (see Judd et al. 1994, 1997), individuals with dysphoria were not required to endorse at least one of the key symptoms of depression (i.e., depressed mood or anhedonia). However, the majority of dysphoric patients (N = 21, 78 %) included in the current study endorsed depressed mood and/or anhedonia. In addition, dysphoric individuals had no psychiatric comorbidities or lifetime depressive episodes.

In order to ensure separation between groups with and without dysphoria, we selected 29 individuals without dysphoria [25 females and 4 males; age, M = 22.3, SD = 1.9; BDI-II score, M = 3.0, SD = 2.9] with both the online and the paper-and-pencil BDI-II scores ≤8 (i.e., the 53 percentile). The group without dysphoria had significantly lower BDI-II scores than the group with dysphoria, $F_{(1,54)} = 178.1, \ p < .001, \ \eta^2_p = .77$.

Participants who scored between 9 and 11 either on the online or the paper-and-pencil BDI-II, or had at least one depressive symptom as evaluated by the SCID-I interview...
were excluded from the present study. Participants with and without dysphoria were simultaneously recruited during the same study period. All the participants were medically healthy and free of medication.

The present study was carried out with the adequate understanding and written consent of the participants, in accordance with the Declaration of Helsinki, and was approved by the local ethics committee.

**Procedure**

Upon arrival at the laboratory, participants received general information about the experiment, and read and signed an informed consent form. After completing the paper-and-pencil version of the BDI-II, the participants were administered the SCID-I. Then, individuals were seated on a comfortable chair in front of a computer screen, and sensors were attached. After electrode placement, each participant rested for five minutes and then performed the emotional imagery task. The EEG was continuously recorded during the imagery task. Participants were instructed to stay still, and to keep their gaze on a central fixation cross during the EEG recordings in order to minimize eye movements.

**Emotional Imagery Task**

Six narratives, selected from the Affective Norms of English Text (ANET; Bradley and Lang 2007) based on standardized ratings of pleasure and arousal, were translated into Italian and categorized as pleasant (i.e., erotic scenes; narrative 4670: Pleasure, \(M = 8.15, SD = 1.28\); Arousal, \(M = 8.01, SD = 1.40\); narrative 4400: Pleasure, \(M = 8.28, SD = 1.22\); Arousal, \(M = 7.91, SD = 1.50\)), neutral (i.e., grocery shopping and getting ready to go out; narrative 2540: Pleasure, \(M = 5.54, SD = 1.19\); Arousal, \(M = 3.38, SD = 1.75\); narrative 2580: Pleasure, \(M = 5.55, SD = 1.17\); Arousal, \(M = 3.60, SD = 1.98\)), or unpleasant (i.e., a friend involved in a car accident and fear of being chased by a stranger; narrative 3310: Pleasure, \(M = 1.30, SD = 1.08\); Arousal, \(M = 8.15, SD = 1.54\); narrative 6800: Pleasure, \(M = 2.50, SD = 1.35\); Arousal, \(M = 7.50, SD = 1.65\)).

Each imagery trial consisted of three minutes of baseline and two 90-s periods of imagery in the same emotional condition. Participants were told to listen carefully while the experimenter read the first script in the first emotional (e.g., unpleasant) condition. The experimenter read the script slowly (about 20 s), in order for the participants to fully understand the script. When the experimenter completed reading the script, the participants were instructed to press a button immediately before beginning the 90-s period of imagery. Participants were instructed to stop imagining the first script after 90 s, to provide subjective ratings of valence, arousal and vividness (see below), and immediately perform a second 90-s period of active imagery in the same emotional (e.g., unpleasant) condition. Then, the same procedure (3-min baseline period and two 90-s imagery periods) was repeated for the other two emotional conditions (e.g., pleasant and neutral). The three emotional conditions were separated by five minutes, during which the participants were told to rest. The order of presentation of the three conditions and the two 90-s narratives within each emotional condition was counterbalanced across participants.

**Self-Report Measures**

After the imagery of each script, the participants were instructed to rate the subjective pleasantness and arousal experienced during the imagined scene using a computerized version of the 9-point Self-Assessment Manikin (SAM) scale, with higher scores reflecting greater pleasantness and arousal (Bradley and Lang 1994). Participants were also instructed to indicate on a 9-point Likert scale how vividly they imagined each scene, with higher scores reflecting greater vividness.

**EEG Recording and Data Analysis**

The EEG was recorded from 32 scalp positions using an elastic cap with tin electrodes (Electro-cap International, Inc.). The EEG sites were Fp1, Fpz, Fp2, F7, F3, Fz, F4, F8, FT7, FC3, FCz, FC4, FT8, T3, C3, CZ, C4, T4, TP7, CP3, CPz, CP4, TP8, P7, P3, Pz, P4, P8, O1, Oz, O2 and A2 (right mastoid), all referenced online to A1 (left mastoid). To control for eye-movements and eye-blinks, both vertical and horizontal electro-oculograms (EOGs) were recorded using a bipolar montage. The electrodes pairs were placed at the supra- and suborbit of the right eye and at the external canthi of the eyes, respectively. All electrode impedances were kept below 5 k\(\Omega\).

The signal was amplified with Neuroscan Synamps (El Paso, TX, USA), bandpass filtered online at 0.1-70 Hz, digitized at 500 Hz (16 bit AD converter, accuracy 0.034 µV/bit), and stored on to a Pentium II computer. The EEG signal was referenced offline to a linked mastoids montage. A regression-based correction algorithm (Scan 4.1 software) was used to correct continuous EEG data for eyeblinks. The obtained signal was segmented in epochs of 1.024 s each and EEG chunks were automatically rejected if containing artifacts greater than ±70 µV in any channel. Then, each EEG segment was visually scored for residual artifacts. For each accepted epoch, a Hamming windowing was applied and chunks were then overlapped by 50 % to minimize loss of data. A Fast Fourier Transform (FFT) method was used to derive estimates of spectral power (\(\mu V^2\)) in 1-Hz frequency bins for each electrode site. All spectral powers obtained were averaged, and power density values (\(\mu V^2/Hz\)) within the theta (4-8 Hz) band were calculated for each participant at each site. In order to ensure that the results obtained were specific for theta band, the...
power density power values within the alpha (8–13 Hz) band were also calculated.

As a first step, self-report measures (valence, arousal, and vividness), and power density values in the theta and alpha bands obtained for the two 90-s imagery periods within the same emotional condition were averaged. Power density values in the theta and alpha bands obtained during each 3-min baseline period were also averaged separately for each emotional condition. Changes in theta and alpha activity from baseline to imagery were calculated by subtracting the averaged values obtained during the 180-s baseline from those obtained during the two 90-s imagery periods for each emotional condition.

Mixed ANOVAs, with Group (with dysphoria, without dysphoria) as a between-subjects factor, and Category (pleasant, neutral, unpleasant) as within-subjects factor, were conducted on self-reported valence, arousal, and vividness.

A mixed ANOVA, with Group (with dysphoria, without dysphoria) as a between-subjects factor, and Category (pleasant, neutral, unpleasant), Area (frontal [F3, Fz, F4], frontocentral [FC3, FCz, FC4], central [C3, Cz, C4] and centroparietal [CP3, CPz, CP4]), and Laterality (left [F3, FC3, C3, CP3], midline [Fz, FCz, Cz, CPz], right [F4, FC4, C4, CP4]) as within-subjects factors, was conducted on changes in theta activity from baseline to imagery. The same statistical analysis was also conducted on changes in alpha activity from baseline to imagery, in order to ensure that the observed effects were specific for theta activity.  

Significant main effects and interactions \((p < .05)\) were followed by Fisher’s LSD post-hoc tests. Cohen’s \(d\) was calculated as a measure of the effect size.

**Results**

**Self-Report Measures**

For valence ratings, the significant main effect of Category, \(F_{(2108)} = 359.41, p < .001, \eta^2_p = .87\), showed that the pleasant condition was rated as more pleasant than the neutral \((p < .001, \text{Cohen’s} \ d = 1.55)\) and the unpleasant \((p < .001, \text{Cohen’s} \ d = 4.91)\). In turn, the unpleasant condition was rated as less pleasant than the neutral \((p < .001, \text{Cohen’s} \ d = 3.63)\).

No significant main effect for Group or interaction between Group and Category were noted \((all \ p s > .35)\).

Similarly, the ANOVA on arousal ratings revealed a significant main effect of Category, \(F_{(2108)} = 79.84, p < .001, \eta^2_p = .60\). Arousal was higher for pleasant \((p < .001, \text{Cohen’s} \ d = 1.74)\) and unpleasant \((p < .001, \text{Cohen’s} \ d = 1.90)\) than neutral conditions, whereas no significant difference in arousal between pleasant and unpleasant conditions was noted \((p = .65)\). No significant main effect for Group or interaction between Group and Category emerged \((all \ p s > .08)\).

With respect to vividness ratings, no significant main effect for Group or Category, or interaction between Group and Category was noted \((all \ p s > .06)\). Descriptive statistics of self-report measures are reported in Table 1.

**EEG Data: Changes in Theta Activity**

The mixed ANOVA on changes in theta activity yielded a significant main effect for Laterality, \(F_{(2108)} = 8.04, p < .001, \eta^2_p = .13\), showing that theta activity was larger at midline scalp sites than lateral sites \((left: p < .001, \text{Cohen’s} \ d = 0.15; right: p < .001, \text{Cohen’s} \ d = 0.11)\), whereas no significant difference between left and right sites was noted \((p = .32)\). The ANOVA also revealed a significant Group \(\times\) Category \(\times\) Area interaction, \(F_{(6324)} = 2.17, p < .05, \eta^2_p = .04\). As shown in Fig. 1, at frontal and frontocentral sites, the pattern of changes in theta activity from baseline to the imagery of pleasant and unpleasant scripts was significantly different between groups. Specifically, individuals without dysphoria were characterized by a greater decrease in theta activity from baseline to the imagery of unpleasant compared to pleasant scripts at frontal \((p < .002, \text{Cohen’s} \ d = 0.42)\) and frontocentral \((p < .02, \text{Cohen’s} \ d = 0.30)\) scalp sites. In contrast, the group with dysphoria showed the opposite pattern, that is, a greater decrease in theta activity from baseline to the imagery of pleasant relative to unpleasant scripts at frontal \((p < .001, \text{Cohen’s} \ d = 0.50)\) and frontocentral \((p < .02, \text{Cohen’s} \ d = 0.28)\) sites. Moreover, it is worth noting that the difference between groups in change in theta activity from baseline to the imagery of pleasant scripts showed a medium-to-large effect size at frontal sites \((\text{Cohen’s} \ d = 0.75)\) and a medium effect at frontocentral sites \((\text{Cohen’s} \ d = 0.57)\).

At central and centroparietal sites, the groups with and without dysphoria were characterized by a greater decrease in theta activity from baseline to the imagery of pleasant \((all \ p s < .10, \text{all} \ \text{Cohen’s} \ d s > 0.18)\) and unpleasant \((all \ p s < .05, \text{all} \ \text{Cohen’s} \ d s > 0.22)\) vs. neutral scripts, whereas changes in theta activity did not differ between pleasant and unpleasant conditions \((all \ p s > .26)\). Figure 2 depicts maps of change in theta activity from baseline to each emotional condition in the group with vs. without dysphoria.

In order to test whether the interaction between the severity of depressive symptoms and the emotional category predicts change in frontal and frontocentral theta activity, we computed a linear model \((\text{ANCOVA})\) with Category (pleasant, neutral, unpleasant), and Area (frontal, frontocentral) as within-subjects factors, and BDI-II score (continuous variable treated
as covariate) as the predictor of change in theta activity from baseline to imagery. We found a significant Category × BDI-II score interaction, $F_{(2108)} = 3.38$, $p < .05$, $\eta^2_p = .06$, and a Category × Area × BDI-II score interaction, $F_{(2108)} = 3.48$, $p < .04$, $\eta^2_p = .06$. A post-hoc analysis for this interaction was carried out, by calculating the single correlation between changes in frontal and frontocentral theta activity of each of the three emotional categories and BDI-II scores. An inverse correlation was noted between changes in theta activity at anterior scalp sites during the imagery of pleasant, but not neutral and unpleasant, scripts and BDI-II scores (see Table 2). Specifically, the more severe the depressive symptoms, the greater the decrease in theta activity at frontal than frontocentral scalp sites during the pleasant imagery (Fig. 3).

The mixed ANOVA on changes in alpha activity revealed a significant Area × Laterality interaction, $F_{(6324)} = 8.41$, $p < .001$, $\eta^2_p = .13$, showing a greater decrease in left alpha activity from baseline to imagery at central and centroparietal relative to frontal and frontocentral scalp sites (all $p$s < .001). Similarly, the ANOVA showed a greater decrease in right alpha activity from baseline to imagery at central relative to frontal, frontocentral, and centroparietal scalp sites (all $p$s < .04). In addition, a greater decrease in left relative to right and midline alpha activity from baseline to imagery at central and centroparietal scalp sites was observed (all $p$s < .006). Of note, the ANOVA did not reveal any significant main effect of Group, Category, Area and/or Laterality, or any other interaction involving Group, Category, Area and/or Laterality (all $p$s > .06).

Descriptive statistics of theta and alpha power density at each scalp site during the baseline period and the imagery of each emotional condition are reported for the two groups in Tables 3 and 4.

### Discussion

The present study was designed to investigate whether frontal theta activity may be an EEG correlate of mood-related emotional processing in dysphoria. In line with our hypothesis, and with the limited literature data available, the current study showed a greater decrease in frontal and frontocentral theta activity from baseline to the imagery of pleasant relative to unpleasant stimuli in dysphoric individuals, and the opposite pattern in healthy controls. This novel finding adds to the literature on the neural basis of mood-related processing biases in depression by showing that frontal and frontocentral theta activity could be an EEG correlate of mood-related emotional processing that characterizes dysphoric individuals (Joormann 2004; Peckham et al. 2010). The finding that frontal theta activity reflects mood-related emotional processing is consistent with previous studies showing that the sources of this cortical rhythm are to be located within the rACC (e.g., Nishida et al. 2004; Pizzagalli et al. 2003), and that rACC is
involved in depression-related emotional dysregulation (e.g., Elliott et al. 2002; Eugène et al. 2010). Indeed, rACC activity has been consistently found to be increased in response to unpleasant stimuli and diminished in response to pleasant stimuli in depressed patients, whereas the opposite pattern has been observed in healthy controls (e.g., Elliott et al. 2002; Eugène et al. 2010).

We found that frontal theta activity consistently decreased from baseline to the active imagery condition. This result is consistent with previous evidence suggesting that decreases in theta activity are likely to reflect an active involvement in the emotional imagery task (Sebastiani et al. 2003). This finding is also in line with those of previous studies showing that rACC is a key region within the default mode network, which is referred to as a task-negative network (Broyd et al. 2009; Fox et al. 2005). While a task-positive network, including the dorsolateral prefrontal cortex, the dorsal ACC (dACC), the intraparietal sulcus and middle temporal area, becomes activated during externally-driven tasks requiring attentional and cognitive resources (Corbetta and Shulman 2002; Sonuga-Barke and Castellanos 2007), the default network becomes less activated during demanding cognitive tasks (Pizzagalli 2011). This might explain why frontal theta activity, an EEG correlate of rACC activity, showed a task-induced reduction in the present study. However, given the paucity of studies examining changes in theta activity during an emotional imagery task, this hypothesis needs to be further tested.

More importantly, the finding that frontal theta activity was less reduced in response to unpleasant relative to pleasant stimuli in dysphoric vs. nondysphoric individuals is consistent with studies showing a reduced rACC deactivation in response to unpleasant relative to pleasant stimuli in depressed patients (for a review, see Pizzagalli 2011). Because rACC has been involved in regulating the motivational and emotional significance of events (Bush et al. 2000; Simoes-Franklin et al. 2010), the reduced rACC deactivation in response to unpleasant relative to pleasant stimuli has been suggested to reflect increased motivational significance of negative vs. positive events in depression (Pizzagalli 2011). Accordingly, the decreased task-induced reduction in frontal theta activity in response to unpleasant relative to pleasant stimuli may suggest that dysphoric individuals are more sensitive to negative vs. positive events compared to nondysphoric individuals.

Notably, we found a greater decrease in frontal and frontocentral theta activity from baseline to the imagery of pleasant scripts in the group with dysphoria relative to the group without dysphoria. In line with this finding, the severity of depressive symptoms was associated with decrease in frontal and frontocentral theta activity during the imagery of pleasant, but not neutral or unpleasant, stimuli. This is consistent
with previous findings by Eugène et al. (2010), reporting an inverse correlation between the severity of depressive symptoms, as measured with BDI-II, and rACC activation for pleasant, but not neutral and unpleasant, stimuli. These results suggest that dysphoric individuals are less sensitive to pleasant relative to unpleasant stimuli rather than the opposite pattern – that is, more sensitive to unpleasant relative to pleasant stimuli. Accordingly, a growing number of studies have recently suggested that the inability to process positive and rewarding stimuli is one of the key mechanisms that underlie emotion dysregulation in depression (Gotlib and Joormann 2010; Joormann 2004; Peckham et al. 2010).

The suggestion that decreases in frontal theta activity may reflect reduced motivational salience of pleasant stimuli in dysphoria is also supported by the evidence that frontal theta activity is associated with emotional processing of pleasant stimuli in healthy individuals. Specifically, decreases in frontal theta activity during the processing of pleasant stimuli have been associated with reduced emotional involvement in healthy individuals (Knyazev et al. 2009). Similarly, Sammler et al. (2007) reported that lower increases in frontal theta activity during the listening of pleasant music excerpts were associated with lower pleasantness ratings. Likewise, reduced frontal and frontocentral theta activity has been found to be associated with less positive “blissful” experience during meditation (Aftanas and Golocheikine 2001).

In line with evidence showing that greater resting rACC activity is associated with better treatment response in depressed patients (e.g., Mayberg et al. 1997; Ritchey et al. 2011), greater frontal theta activity within the rACC has been found to predict treatment outcome in depression (Pizzagalli et al. 2001). Because these studies examined rACC activity or frontal theta activity in resting state condition, while the current study focused on frontal theta activity during an imagery task, it is unclear whether the present findings may be associated with clinical outcome in dysphoria. Therefore, future research is warranted to examine whether changes in frontal theta activity during an emotional imagery task predicts the course of depressive symptoms in dysphoric individuals.

Alpha activity was not implicated in mood-related emotional processing in participants with dysphoria. Although this is in line with the lack of evidence for an association between

### Table 3

Power density in theta band (μV²/Hz) during the emotional imagery task in the group without and with dysphoria

| Group       | Channel | Pleasant Baseline | Imagery | Neutral Baseline | Imagery | Unpleasant Baseline | Imagery |
|-------------|---------|-------------------|---------|------------------|---------|---------------------|---------|
| Without dysphoria | F3      | 1.84 (0.79)       | 1.83 (0.87) | 1.80 (0.67) | 1.75 (0.73) | 1.85 (1.00)       | 1.77 (0.91) |
|             | Fz      | 2.31 (0.94)       | 2.28 (1.03) | 2.25 (0.85) | 2.22 (0.87) | 2.31 (1.18)       | 2.19 (1.01) |
|             | F4      | 1.9 (0.79)        | 1.90 (0.89) | 1.86 (0.72) | 1.87 (0.79) | 1.95 (1.04)       | 1.83 (0.87) |
|             | FC3     | 1.85 (0.75)       | 1.85 (0.80) | 1.79 (0.64) | 1.76 (0.67) | 1.83 (0.84)       | 1.76 (0.78) |
|             | FCz     | 2.68 (1.04)       | 2.63 (1.07) | 2.59 (0.91) | 2.55 (0.90) | 2.63 (1.15)       | 2.52 (1.02) |
|             | FC4     | 1.82 (0.75)       | 1.81 (0.81) | 1.76 (0.63) | 1.78 (0.70) | 1.82 (0.87)       | 1.72 (0.73) |
|             | C3      | 1.71 (0.75)       | 1.67 (0.76) | 1.65 (0.64) | 1.61 (0.64) | 1.68 (0.80)       | 1.60 (0.72) |
|             | Cz      | 2.60 (1.13)       | 2.50 (1.08) | 2.51 (1.03) | 2.46 (0.96) | 2.53 (1.13)       | 2.41 (1.04) |
|             | C4      | 1.70 (0.79)       | 1.63 (0.77) | 1.61 (0.61) | 1.64 (0.71) | 1.67 (0.80)       | 1.56 (0.69) |
|             | CP3     | 1.78 (0.90)       | 1.72 (0.85) | 1.73 (0.76) | 1.71 (0.77) | 1.75 (0.93)       | 1.65 (0.87) |
|             | CPz     | 2.45 (1.30)       | 2.36 (1.17) | 2.38 (1.17) | 2.33 (1.05) | 2.38 (1.26)       | 2.26 (1.18) |
|             | CP4     | 1.73 (0.89)       | 1.64 (0.83) | 1.66 (0.71) | 1.66 (0.80) | 1.69 (0.86)       | 1.59 (0.79) |
| With dysphoria | F3      | 1.88 (0.7)        | 1.70 (0.59) | 1.78 (0.65) | 1.68 (0.62) | 1.74 (0.57)       | 1.69 (0.62) |
|             | Fz      | 2.41 (1.01)       | 2.14 (0.79) | 2.29 (0.90) | 2.14 (0.84) | 2.23 (0.79)       | 2.14 (0.86) |
|             | F4      | 1.90 (0.64)       | 1.72 (0.57) | 1.83 (0.65) | 1.72 (0.59) | 1.79 (0.54)       | 1.71 (0.59) |
|             | FC3     | 1.85 (0.63)       | 1.71 (0.55) | 1.77 (0.60) | 1.69 (0.59) | 1.74 (0.53)       | 1.69 (0.58) |
|             | FCz     | 2.82 (1.17)       | 2.56 (0.96) | 2.66 (0.98) | 2.55 (1.03) | 2.69 (0.98)       | 2.53 (0.96) |
|             | FC4     | 1.82 (0.58)       | 1.66 (0.64) | 1.75 (0.60) | 1.65 (0.56) | 1.76 (0.54)       | 1.65 (0.54) |
|             | C3      | 1.67 (0.57)       | 1.52 (0.48) | 1.60 (0.55) | 1.54 (0.52) | 1.61 (0.50)       | 1.51 (0.49) |
|             | Cz      | 2.69 (1.16)       | 2.45 (0.97) | 2.52 (0.89) | 2.47 (1.13) | 2.60 (0.94)       | 2.38 (0.84) |
|             | C4      | 1.66 (0.54)       | 1.50 (0.47) | 1.60 (0.51) | 1.51 (0.51) | 1.64 (0.52)       | 1.50 (0.47) |
|             | CP3     | 1.68 (0.59)       | 1.54 (0.48) | 1.63 (0.58) | 1.54 (0.50) | 1.64 (0.53)       | 1.52 (0.47) |
|             | CPz     | 2.36 (0.96)       | 2.16 (0.81) | 2.24 (0.78) | 2.14 (0.84) | 2.26 (0.76)       | 2.07 (0.69) |
|             | CP4     | 1.64 (0.54)       | 1.47 (0.48) | 1.55 (0.50) | 1.45 (0.49) | 1.59 (0.53)       | 1.44 (0.46) |

**Note.** Data are M (SD)
alpha activity and rACC (Larson et al. 1998; Lindgren et al. 1999), the present finding is at odds with evidence that individuals with depressed mood often display a stable pattern of low cortical activity (i.e., high alpha power) in the left relative to the right hemisphere (e.g., Gotlib et al. 1998). The fact that the emotional imagery task did not induce a sufficient activation of approach- and/or withdrawal-related motivational system may explain why the present study failed to confirm a link between dysphoria and frontal alpha asymmetry.

Dysphoric and nondysphoric individuals did not differ on self-report measures of valence and arousal. This null finding diverges from those obtained by other studies (e.g., Rottenberg et al. 2002, 2005), which reported differences between clinically depressed individuals and healthy controls with respect to self-report measures of emotional experience. An explanation for these discrepant findings may lie in the methodology used to evaluate emotional experience. The majority of previous studies reporting a significant difference between groups used a list of discrete emotions to evaluate subjective emotional experience (e.g., Rottenberg et al. 2002, 2005). By contrast, studies, like ours, using the SAM 9-point scale to assess self-reported valence and arousal for each script were less likely to report differences between groups at the subjective level (Allen et al. 1999; Dichter et al. 2004; but see also Sloan et al. 1997). Alternatively, it is possible that mild to moderate depressive symptoms typically reported in dysphoria may not be sufficient to elicit differences in subjective ratings of emotional experience (Mneimne et al. 2008; Sloan and Sandt 2010).

The current findings should be interpreted in light of some limitations. First, the definition of dysphoria did not require participants to endorse at least one of the key symptoms of depression (i.e., depressed mood or anhedonia). This method for sample selection may have resulted in a heterogeneous group that may not be characterized by mood-related difficulties. It should be noted, however, that the vast majority of dysphoric patients (N = 21, 78%) included in the current study endorsed depressed mood and/or anhedonia. Second, we did not include behavioral measures, such as measures of attention or inhibitory control, which would have provided a more complete picture of the influence of mood-related emotional processing in dysphoria. Lastly, it is not clear whether

Table 4  Power density in alpha band (μV²/Hz) during the emotional imagery task in the group without and with dysphoria

| Group          | Channel | Pleasant Baseline | Pleasant Imagery | Neutral Baseline | Neutral Imagery | Unpleasant Baseline | Unpleasant Imagery |
|----------------|---------|-------------------|------------------|------------------|-----------------|---------------------|--------------------|
| Without dysphoria | F3      | 2.51 (2.91)       | 2.43 (2.74)      | 2.38 (2.18)      | 2.32 (2.17)     | 2.42 (2.83)         | 2.43 (2.76)        |
|                | Fz      | 2.96 (3.44)       | 2.85 (3.14)      | 2.82 (2.61)      | 2.77 (2.57)     | 2.85 (3.25)         | 2.85 (3.20)        |
|                | F4      | 2.53 (2.86)       | 2.48 (2.84)      | 2.41 (2.14)      | 2.39 (2.21)     | 2.49 (2.86)         | 2.47 (2.83)        |
|                | FC3     | 2.47 (2.48)       | 2.38 (2.36)      | 2.40 (2.05)      | 2.31 (1.88)     | 2.43 (2.55)         | 2.38 (2.40)        |
|                | FCz     | 3.28 (3.48)       | 3.17 (3.19)      | 3.19 (2.73)      | 3.13 (2.65)     | 3.21 (3.39)         | 3.20 (3.29)        |
|                | FC4     | 2.48 (2.53)       | 2.40 (2.57)      | 2.35 (1.91)      | 2.34 (1.94)     | 2.44 (2.61)         | 2.39 (2.47)        |
|                | C3      | 2.87 (2.74)       | 2.70 (2.59)      | 2.81 (2.35)      | 2.70 (2.19)     | 2.83 (2.88)         | 2.63 (2.54)        |
|                | Cz      | 3.67 (3.84)       | 3.56 (3.65)      | 3.60 (3.22)      | 3.63 (3.22)     | 3.63 (3.85)         | 3.60 (3.71)        |
|                | C4      | 2.98 (2.81)       | 2.81 (2.84)      | 2.81 (2.22)      | 2.81 (2.28)     | 2.92 (2.94)         | 2.77 (2.59)        |
|                | CP3     | 3.93 (3.92)       | 3.69 (3.65)      | 3.87 (3.24)      | 3.83 (3.41)     | 3.86 (3.88)         | 3.62 (3.57)        |
|                | CPz     | 5.08 (6.05)       | 4.96 (5.77)      | 4.95 (5.06)      | 5.12 (5.27)     | 5.00 (5.84)         | 4.92 (5.53)        |
|                | CP4     | 4.06 (3.87)       | 3.92 (3.85)      | 3.90 (3.22)      | 4.06 (3.54)     | 3.93 (3.80)         | 3.84 (3.56)        |
| With dysphoria | F3      | 1.64 (1.06)       | 1.50 (1.02)      | 1.60 (0.97)      | 1.48 (1.00)     | 1.53 (0.94)         | 1.51 (0.97)        |
|                | Fz      | 1.96 (1.35)       | 1.76 (1.21)      | 1.88 (1.19)      | 1.75 (1.22)     | 1.81 (1.18)         | 1.78 (1.20)        |
|                | F4      | 1.68 (1.15)       | 1.51 (1.02)      | 1.62 (1.00)      | 1.50 (1.03)     | 1.55 (0.99)         | 1.51 (1.00)        |
|                | FC3     | 1.67 (1.04)       | 1.52 (0.98)      | 1.67 (1.00)      | 1.52 (1.00)     | 1.58 (0.95)         | 1.53 (0.96)        |
|                | FCz     | 2.27 (1.57)       | 2.04 (1.41)      | 2.16 (1.35)      | 2.02 (1.38)     | 2.12 (1.38)         | 2.06 (1.42)        |
|                | FC4     | 1.71 (1.22)       | 1.53 (1.04)      | 1.64 (1.04)      | 1.53 (1.05)     | 1.59 (1.01)         | 1.52 (1.02)        |
|                | C3      | 1.92 (1.31)       | 1.68 (1.18)      | 1.99 (1.37)      | 1.77 (1.38)     | 1.88 (1.27)         | 1.68 (1.26)        |
|                | Cz      | 2.49 (1.86)       | 2.26 (1.72)      | 2.37 (1.63)      | 2.25 (1.70)     | 2.33 (1.56)         | 2.24 (1.79)        |
|                | C4      | 1.99 (1.57)       | 1.76 (1.33)      | 1.88 (1.29)      | 1.77 (1.37)     | 1.82 (1.15)         | 1.70 (1.28)        |
|                | CP3     | 2.54 (2.23)       | 2.25 (2.14)      | 2.56 (2.13)      | 2.34 (2.31)     | 2.42 (1.39)         | 2.16 (2.09)        |
|                | CPz     | 3.16 (3.08)       | 2.96 (3.24)      | 2.97 (2.70)      | 2.88 (2.81)     | 2.90 (2.28)         | 2.76 (2.79)        |
|                | CP4     | 2.57 (2.42)       | 2.39 (2.41)      | 2.38 (2.04)      | 2.33 (2.23)     | 2.29 (1.65)         | 2.23 (2.09)        |

Note. Data are M (SD)
the findings reported herein are specific for dysphoric patients or may be generalized to patients with clinically significant depression. Clearly, future research should address these issues by investigating whether frontal theta activity may reflect mood-related emotional processing in tasks requiring behavioral responses, such as the emotional go/nogo task, and/or in patients with major depression.

In summary, the present study showed that individuals with dysphoria are characterized by greater decrease of frontal and frontocentral theta activity in response to pleasant relative to unpleasant stimuli, with healthy controls showing the reversed pattern. The present study suggests that frontal and frontocentral theta activity may be an EEG correlate of mood-related biases in emotional processing in dysphoria. More specifically, these findings suggest that decreases in frontal and frontocentral theta activity observed in dysphoric individuals are likely to reflect a reduced motivational salience of pleasant stimuli, which, in turn, has been implicated as a key feature of depression. The current study may contribute to a better understanding of the neural correlates of emotional dysregulation that characterize dysphoria and depression.

Compliance with Ethical Standards

Role of the Funding Source None.

Conflicts of Interest Simone Messerotti Benvenuti, Rocco Mennella, Giulia Buodo, and Daniela Palomba declare that they have no conflict of interest.

Experiment Participants All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

Informed Consent Informed consent was obtained from all individual participants included in the study.

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