Electroencephalographic Asymmetry, Emotion Regulation, and Their Relationships with Depression Risk

Aliza Jacob

The Graduate Center, City University of New York

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ELECTROENCEPHALOGRAPHIC ASYMMETRY, EMOTION REGULATION, AND THEIR RELATIONSHIPS WITH DEPRESSION RISK

by

Aliza (Schwartzblatt) Jacob

A dissertation submitted to the Graduate Faculty in Psychology in partial fulfillment of the requirements for the degree of Doctor of Philosophy, The City University of New York

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Electroencephalographic Asymmetry, Emotion Regulation, and their Relationships with Depression Risk

by

Aliza Jacob

This manuscript has been read and accepted for the Graduate Faculty in Psychology in satisfaction of the dissertation requirement for the degree of Doctor of Philosophy.

April 30th, 2018
Date

Jennifer L. Stewart
Chair of Examining Committee

April 30th, 2018
Date

Richard Bodnar
Executive Officer

Supervisory Committee:

Joan C. Borod

Jennifer L. Stewart

Justin L. Storbeck

THE CITY UNIVERSITY OF NEW YORK
ABSTRACT

Electroencephalographic Asymmetry, Emotion Regulation, and their Relationships with Depression Risk

By

Aliza Jacob

Advisor: Jennifer L. Stewart

Background: Research investigating patterns of electroencephalographic (EEG) brain asymmetry aids our understanding of neural systems involved in the processing of emotion, motivation, and psychopathology. Withdrawal-motivated negative emotions characteristic of depression are associated with relative right prefrontal cortex (PFC) activity, whereas approach-motivated positive emotions are associated with relative left PFC activity. Styles of emotion regulation (ER), or modulation of the intensity and duration of emotional responses, are also associated with presence (e.g., suppression, or maladaptive ER) versus absence (e.g., cognitive reappraisal, or adaptive ER) of depression vulnerability. Most PFC asymmetry studies of emotion, depression, and/or ER rely upon EEG recorded during uncontrolled resting states that appear to be less reliable than EEG recorded during cognitive or emotional challenge tasks. To this end, the present study examines whether current depression symptoms and ER styles moderate PFC asymmetry when individuals attempt to recover from an emotional challenge and whether future depression symptoms will be predicted by PFC asymmetry and/or ER styles.

Methods: EEG asymmetry was recorded before, during and after 38 young adults experienced a state emotion manipulation induced via film clip (happy: \(n=16\); sad: \(n=22\)). Self-reported
depression symptoms and ER were collected in the same session as EEG asymmetry as well as a follow-up session one year later.

**Hypotheses:** It was predicted that participants in the sad film clip condition would demonstrate greater relative right film-clip PFC asymmetry than participants in the happy film clip condition, consistent with prior state emotion manipulations. With regard to within-subjects changes over time, it was predicted that trait sadness (depression) and use of ER strategies would be associated with PFC asymmetry during and after the film clip mood induction. For the longitudinal component, it was hypothesized that relative right PFC asymmetry and use of ER strategies would be associated with future depressive symptoms, one year after EEG recording.

**Results:** Although happy and sad groups did not differ in PFC asymmetry pre-, during- or post-clip, moderation analyses revealed that individuals with lower depression symptoms or greater use of adaptive ER strategies paired with greater leftward PFC asymmetry during the film clip resulted in greatest relative left PFC asymmetry post-clip. However, PFC asymmetry and ER styles were not associated with the onset of depressive symptoms at follow-up.

**Conclusions:** Approach-related brain activity paired with adaptive ER and lower depressive symptoms promote approach-related recovery from brief emotional states and could index a marker for resilience to stress.
ACKNOWLEDGEMENTS

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Introduction

Emotion and Prefrontal Cortex Brain Asymmetry

Emotion is arguably one of the most important constructs to study, given the extent to which it pervades our daily interactions, affects perceptions, cognitions, and experiences, and directly influences life satisfaction (Bastian, Kuppens, De Roover, & Diener, 2014). Further, emotion regulates and facilitates cognitions to guide behavior (Storbeck & Clore, 2007). Electroencephalography (EEG) prefrontal cortex (PFC) asymmetry has been an essential tool in observing lateralization of neural activity, which is important for identifying markers of risk for psychopathology (endophenotypes) as well as providing an understanding of the neural mechanisms involved in the implementation of various emotional states (Heller, Nitschke, & Miller, 1998; Tomarken, Davidson, Wheeler, & Doss, 1992). Pleasant or positive affect is associated with relatively greater left than right PFC activity, while unpleasant or negative affect is associated with greater right than left PFC activity (Heller, 1993; Heller, Nitschke, & Miller, 1998; Sutton & Davidson, 1997). Other research suggests that PFC asymmetries reflect engagement of the motivational system rather than valence (Davidson, 1992; Davidson, 1998a,b; Harmon-Jones, Gable, & Peterson, 2010). Specifically, relative left PFC asymmetry is associated with approach motivation, whereas relative right PFC asymmetry is associated with withdrawal motivation (Davidson, 1992; Davidson, 1998a,b; Harmon-Jones et al., 2010). Overall, the motivation and valence views of lateralization can be viewed as complementary rather than contradictory, wherein motivation is associated with goal-related emotion and overt actions while the valence model is related to emotional stimuli and information processing rather than overt behavior (Spielberg, Stewart, Levin, Miller, & Heller, 2008). Of note, the emotion anger is characterized by relative left PFC asymmetry due to its “approach” nature, thereby supporting
the motivational model of asymmetry rather than the valence model (Harmon-Jones, 2004; Harmon-Jones & Allen, 1998; Harmon-Jones et al., 2010; Harmon-Jones & Sigelman, 2001).

EEG can be measured through several frequency bands including delta (1-4 Hz), theta (4-8 Hz), alpha (9-12 Hz), beta (13-20 Hz), and gamma (30-80 Hz and >80 Hz). However, EEG asymmetry, specifically during emotion processing, is thought to be most robustly captured in the alpha range (Tomarken et al., 1992). Alpha power has been linked to EEG asymmetry associated with emotional states (Davidson, Ekman, Saron, Senulis, & Friesen, 1990), cognitive tasks (e.g., Davidson, Chapman, Chapman, & Henriques, 1990), and individual differences in affective predispositions (Tomarken, Davidson, & Henriques, 1990). Tomarken et al. (1992) measured theta and beta asymmetry when attempting to observe trait dimensions of emotion in healthy adults. While alpha asymmetry was associated with individual differences in affect, similar effects were not seen through asymmetry in the theta or beta bands; however, there have been some effects observed through frequency bands other than alpha including theta (e.g., Jaworska, Blier, Fusee, & Knott, 2012), beta (Pizzagalli et al., 2002), and delta (Henriques & Davidson, 1991). Despite these findings, the research with delta, theta, beta, and gamma bands remains limited and alpha asymmetry has served as the metric of choice when assessing EEG trends associated with emotion (Davidson, 2004).

Of note, alpha power reflects the inverse of cortical activity, meaning that decreased alpha power is associated with increased cortical activity, whereas increased alpha power is associated with decreased cortical activity (Davidson et al., 1990). As such, researchers generally describe relative cortical activity when discussing EEG literature and findings (Allen, Coan, & Nazarian, 2004a).

**Depression and PFC Brain Asymmetry**
Since PFC asymmetry relates to the experience of emotion, it is valuable to study this construct in individuals with mood disorders. Nearly 30% of the U.S. population will experience an episode of major depressive disorder (MDD), a condition associated with significant personal and societal burden (Birnbaum et al., 2010; Kessler, Petukhova, Sampson, Zaslavsky, & Wittchen, 2012). MDD is a disorder characterized by persistent negative mood and/or anhedonia with additional symptoms such as appetite, sleep and motor disturbance, fatigue, suicidality, indecisiveness, and impaired concentration (APA, 2013). Of note, the terms “depression” and “MDD” may be used interchangeably throughout the paper but refer to the diagnostic criteria associated with MDD.

MDD has a strong familial component and is therefore conceptualized from both biological and environmental perspectives (APA, 2013). Multiple brain regions and networks are implicated in the development of depression and depression risk including the amygdala, dorsolateral prefrontal cortex (DLPFC), anterior cingulate cortex (ACC), hypothalamic-pituitary-adrenal (HPA) axis, and the spinomedullary pathway (Herrington et al., 2010; Jaworska et al., 2012). Further, faulty neurotransmitter networks, such as the serotonergic and dopaminergic systems, are also associated with depression (Jesulola, Sharpley, Bitsika, Agnew, & Wilson, 2015).

On a hemispheric level, MDD seems to be characterized by right hemisphere hyperactivation and left hemisphere hypoactivation (Jaworska et al., 2012; Rotenberg, 2008). While right hemispheric activity is relatively greater in individuals with MDD than in non-depressed individuals, neuropsychological functions associated with the right hemisphere are impaired (e.g., dot localization versus word finding; Rotenberg, 2004; Miller, Fujiola, Chapman, & Chapman, 1995). Moreover, both emotional and cognitive processes implicated in depression
(e.g., anhedonia, negative affect, insomnia, and indecisiveness) are associated with right rather than left hemispheric function (Hecht, 2010). Additionally, MDD, depressive symptoms, and apathy are more prevalent after left than right brain damage and present in temporary anesthetization of the left hemisphere (Lee, Loring, Meader, & Brooks, 1990; Ownsworth & Oei, 1998; Palese et al., 2008).

In line with psychophysiologial theories of brain activity, MDD is associated with distinct PFC asymmetry patterns. Individuals diagnosed with MDD typically display relative right PFC asymmetry due to the withdrawal-related and negatively valenced profile of the disorder (Davidson, 1992, 1998a; Heller, 1993); this effect has been replicated in a multitude of research studies (e.g., Allen, Urry, Hitt, & Coan, 2004b; Gollan et al., 2014; Gotlib, Ranganath, & Rosenfeld, 1998; Henriques & Davidson, 1990; Jaworska et al., 2012; Kemp et al., 2010; Pizzagalli et al., 2002; Smit, Posthuma, Boomsma, & De Geus, 2007; Stewart, Coan, Towers, & Allen, 2011a; Stewart, Coan, Towers, & Allen, 2014; Vuga et al., 2006), although not without exception (Bruder et al., 1997; Carvalho et al., 2011; Debener et al., 2000; Deslandes et al., 2008; Gold, Fachner, & Erkkila, 2013). Inconsistencies in the literature may be due to methodological differences related to varying EEG reference montages and resting state characteristics (Thibodeau, Jorgensen, & Kim, 2006), as well as individual differences, including age (Carvalho et al., 2011; Deslandes et al., 2008; Vuga, Fox, Cohn, Kovacs, & George, 2008), sex (Allen, 2015; Knott, Mahoney, Kennedy, & Evans, 2001; Miller et al., 2002; Reid, Duke, & Allen, 1998; Stewart, Bismark, Towers, Coan, & Allen, 2010), disorder comorbidities (Thibodeau et al., 2006), and diagnostic discrepancies (Pizzagalli et al., 2002; Quinn, Rennie, Harris, & Kemp, 2014). For example, older adults tend to show less robust PFC asymmetry than younger adults while older children demonstrate more robust asymmetry than younger children.
(Carvalho et al., 2011; Deslandes et al., 2008; Vuga et al., 2008); women also tend to
demonstrate stronger PFC asymmetry patterns than men (Allen, 2015; Knott et al., 2001; Miller
et al., 2002; Reid et al., 1998; Stewart, et al., 2010). Further, subtypes of anxiety yield
differential PFC asymmetry profiles than MDD; therefore, it is possible that inconsistent findings
may be due to comorbid anxiety (Nusslock, Walden, & Harmon-Jones, 2015; Thibodeau et al.,
2006). Some argue that diagnostic discrepancies or different symptom profiles in MDD, such as
“psychomotor retardation” or “melancholy,” may account for differences in PFC asymmetry
(Pizzagalli et al., 2002; Quinn et al., 2014).

When recording EEG, a reference montage is required to organize EEG channels in order
to accurately measure brain activity. A number of reference montages have been used to observe
PFC asymmetry including the common vertex (Cz), average “linked” mastoids, average of all
cortical electrodes, and nose reference. When using the Cz montage, the center electrode is used
as a reference point; however, this montage tends to under- or over-estimate activity at each
target site, thereby distorting the asymmetry power measure (Allen et al., 2004a; Hagemann,
Naumann, & Thayer, 2001). Over the years, significant asymmetry effects using the Cz reference
are likely related to: 1) unique source variance arising from Cz in addition to PFC asymmetry;
and 2) systematic or random error, which contributes to inconsistent results across studies (Allen
et al., 2004a). Given these weaknesses, the Cz montage is generally discouraged (Hagemann et
al., 2001).

The linked mastoids reference, or reference montage based on the electrodes behind the
ears, is a solution to the methodological issues accompanying Cz. In this montage, two low
active electrode sites are chosen to serve as the reference lead. Because of the relatively low
active nature of these sites, there is minimal added variance, and any activity observed can be
attributed to the target (Hagemann et al., 2001). However, the linked mastoid reference alone has not been empirically supported. Instead, all electrodes are recorded to a common reference and are then re-referenced to the linked mastoid sites in order to eliminate asymmetry bias. The latter method is considered the standard recording method for EEG PFC asymmetry (Hagemann et al., 2001). Similar to linked mastoids, the nose reference is occasionally used since it serves as a relatively inactive reference electrode; however, it is still not commonly utilized (Thibodeau et al., 2006).

The average reference montage is also presumably a solution to the methodological difficulties with the Cz montage. In this case, a virtual site with averaged activity of each electrode is used as the reference point. Average reference is rarely used in asymmetry research though since it uses assumptions that are not consistently held in the alpha band (Hagemann et al., 2001).

Although it is not a reference montage per se, an additional recording method used is the current source density (CSD) transformation (Tenke & Kayser, 2012). The CSD transformation is particularly beneficial since it does not utilize references in its algorithm, it reduces the contribution of distal sources (e.g. occipital regions) in PFC asymmetry scores, and it removes ambiguous currents that may be inadvertently captured by the EEG recording (Tenke & Kayser, 2012). However, when electrodes are spaced widely apart, patches of activity may be filtered out so great care is needed when implementing this method (Smith, Reznik, Stewart, & Allen, 2017).

Stewart et al. (2010; 2011a) recorded EEG asymmetry in individuals with and without lifetime depression using four reference methodologies: average, CSD, Cz, and linked mastoids. They found that just CSD transformed EEG asymmetry differentiated individuals with and without lifetime depression when EEG was measured at rest; in contrast, Cz, linked mastoid, and
average recorded EEG at rest only differentiated depression in women but not men, suggesting that these measurements are less consistent (Stewart et al., 2010). Despite the literature, there is still no consensus on which reference montage to use when attempting to identify links between PFC asymmetry and psychopathology, given that montages do not always correlate (Thibodeau et al., 2006).

In addition to reference montage, other methodological factors such as EEG recording state may contribute to mixed results. According to the field standard, PFC asymmetries are measured during a resting state. This standard practice coincides with the dispositional model of EEG asymmetry, which states that an individual will demonstrate an emotional disposition regardless of the situation; therefore, PFC asymmetry recorded during a neutral state can be generalized to other situations (Davidson, 1998a). Although common practice, there are many methodological issues with EEG recorded during resting states. The primary concern is that it is difficult to control experimental conditions affecting resting measures of EEG (Coan, Allen, & McKnight, 2006). Although the name indicates so, resting states are not comprised of purely “resting” activity since imaging data show that individuals engage in mental behaviors during uncontrolled relaxation states (Blackhart, Kline, Donohue, LaRowe, & Joiner, 2002; Kline, Blackhart, & Joiner, 2002). Additionally, uncontrolled experimental conditions lower trait reliability measurements and increase error due to type of reference electrode used in recording (Allen et al., 2004a; Hagemann et al., 2001; Coan et al., 2006). In other words, if a reference electrode is located in an active site, then the overall results obtained may be confounded with error or additional noise from the reference electrode.

The capability model of individual differences in EEG asymmetry argues that brain asymmetry measured while the brain is processing emotional information is a superior method
for observing hemispheric differences in PFC activity (Coan et al., 2006; Stewart, Coan, Towers, & Allen, 2014). The capability model allows for the possibility that individual differences may arise from distinct situational demands, thereby increasing accuracy of measurement (Coan et al., 2006). Moreover, during emotional challenge, individual differences in brain activity are more pronounced and resistant to error due to greater reliability across reference schemes, as mentioned above (Coan et al., 2006; Stewart et al., 2014; Stewart et al., 2011a). Research has demonstrated that EEG measured during an emotional task differentiates depressed versus non-depressed individuals across four reference montages (average, linked mastoids, Cz, and CSD), while EEG asymmetry measured during an emotional task with the same participants only emerged with the CSD transformation across sexes (Stewart et al., 2010; Stewart et al., 2011a). These findings support the notion that task demands can supersede variability in EEG recording methodology.

Several additional studies have found that PFC asymmetry was associated with depression when measured during an emotional task but not when measured through rest (Beeney, Levy, Gatzke-Kopp, & Hallquist, 2014; Lin, Tsai, Peper, & Yen, 2013; Killeen & Teti, 2012). Within a healthy population, PFC asymmetry measured at rest differentiated individuals with and without dysphoria across a range of emotional conditions (unpleasant, neutral, and pleasant; Mennella, Benvenuti, Buodo, & Palomba, 2015). Overall, asymmetry measured during emotional challenges is a more robust measure of emotion processing than asymmetry measured at rest in both depressed and non-depressed individuals (Stewart et al., 2014). Because of the novelty of this technique, it is vital to study asymmetry patterns and predict variables of interest using these procedures.
The pattern of PFC asymmetry associated with depression is trait-like, meaning individuals show stable asymmetry patterns regardless of depressive status (e.g., current versus past MDD; Allen et al., 2004b; Stewart et al., 2010), depression severity (Allen et al., 2004b; Jesulola et al., 2015; Reid et al., 1998), and behavioral activation treatment – with and without treatment success (Gollan et al., 2014). However, some treatments such as mindfulness and cognitive-behavioral therapy yield PFC asymmetry changes that align with those evident in healthy controls after treatment associated with a reduction of symptoms (see Jesulola et al., 2015 for a review). Additionally, responders to certain antidepressants and deep brain stimulation treatment, measured by pre- to post- change scores, demonstrate EEG asymmetry patterns more closely resembling controls than treatment non-responders, although these differences are sometimes seen in the parietal rather than PFC regions (Bruder et al., 2008; Bruder et al., 2011; Quraan et al., 2014). PFC asymmetry is moderately stable over time in adults with depression as well as children with familial depression risk, although patterns vary with age as mentioned (Allen et al., 2004b; Carvalho et al., 2011; Deslandes et al., 2008; Vuga et al., 2006; Vuga et al., 2008).

While the PFC asymmetry trend is well studied, there is some disagreement as to what may be driving this effect – an overactivation of right frontal regions and/or an underactivation of left frontal regions. Comparing the relative differences between left and right brain activity may seem like a valid option for observing asymmetry; however, this measurement does not control for individual differences in skull size, which results in decreased power to detect reliable differences (Coan & Allen, 2004). Asymmetry difference scores are a more accurate method for measuring asymmetry since they control for individual differences in skull size and are therefore the field standard measurement (Coan & Allen, 2004). Since difference scores are
methodologically superior to measuring left versus right EEG activity, the main contributor of the PFC asymmetry effect is debated. However, researchers generally agree that the combination of a hyperactive right-sided withdrawal system and an underactive left-sided approach and reward seeking system both contribute to the asymmetry profiles and symptoms seen in depression (Jaworska et al., 2012; Rotenberg, 2008). Bruder, Stewart, and McGrath (2017) further add that reduced left PFC activity may impair downregulation of amygdala response to negative emotional information, thereby placing individuals at risk of developing depression.

As mentioned, a variety of moderators may influence asymmetry patterns such as individual differences in age, sex, and comorbidity, as well as methodological differences (e.g., resting states and reference montage). Because of differential outcomes and null findings, it has been argued that asymmetry patterns may not be conclusive in diagnosing depression but can be useful in conjunction with other measures and risk factors (Gold et al., 2013), providing further insight into the neurocircuitry involved in MDD presence and risk. Despite this assertion, Mumtaz et al. (2017) recently developed a computerized program to assist psychiatrists in diagnosing MDD in clinical populations. The researchers created what they refer to as an EEG-based computer-aided (CAD) technique that uses several EEG indexes including PFC asymmetry to distinguish individuals with depression from healthy controls. They found that PFC asymmetry significantly differentiated individuals with depression with 96.8-98.4% accuracy, 96.66% sensitivity, and 97.02-100% specificity (Mumtaz et al., 2017). This research holds great promise in the clinical utility of PFC asymmetry trends.

**Parietal Brain Asymmetry**

Posterior or parietal brain asymmetry, although not the primary focus of the current paper, is discussed in the context of depression. There is a consensus among researchers that
Parietal asymmetry reflects arousal, with relative left activity corresponding to low arousal and relative right activity corresponding to high arousal (Heller, 1993; Spielberger et al., 2008). As such, studies have demonstrated relative left posterior asymmetry in depressed individuals, ostensibly due to low arousal associated with the disorder (Bruder et al., 1997; Henriques & Davidson 1990; Kentgen et al., 2000; Reid et al., 1998; Stewart, Towers, Coan, & Allen, 2011b), although others have not (Henriques & Davidson, 1991). Henriquez and Davidson (1991) suggested that perhaps posterior asymmetry differences are only observed in less severely depressed individuals, and this reasoning may account for mixed findings; however, they acknowledged that they do not yet have the reasoning to explain such a phenomenon (Henriques & Davidson, 1991).

**Anxiety and PFC Brain Asymmetry**

It is also worth mentioning brain asymmetry patterns associated with anxiety due to high comorbidity rates with depression (APA, 2013). Due to differences in motivation and arousal, components of anxiety can be divided into two distinct categories: anxious apprehension and anxious arousal (Heller et al., 1998). Anxious arousal is characterized by panic and somatic symptoms including shortness of breath, pounding heart, dizziness, and choking sensations, while anxious apprehension is characterized by cognitive anxiety including worry, verbal rumination, and anticipatory anxiety. Anxious arousal is linked with greater relative right PFC asymmetry, similar to depression (e.g., Heller et al., 1998; Mathersul, Williams, Hopkinson, & Kemp, 2008; Nitschke, Heller, Palmieri, & Miller, 1999; Stewart, Levin-Silton, Sass, Heller, & Miller, 2008), while anxious apprehension does not significantly lateralize (e.g., Heller et al., 1998; Nitschke et al., 1999; see Nusslock et al., 2015 for a review). It had been hypothesized that anxious apprehension and depression asymmetry indices would cancel each other out due to
opposite PFC asymmetry patterns, thereby resulting in bilateral, symmetrical asymmetry (Mathersul et al., 2008). Indeed, Nusslock et al. (2018) measured PFC asymmetry differences in groups with: 1) diagnosed depression; 2) diagnosed depression and high levels of anxious apprehension; and 3) healthy controls. They found that individuals in the depression-only group demonstrated relative right PFC asymmetry while the depression and anxious apprehension group demonstrated asymmetry no different than the healthy controls; these findings suggest that anxious apprehension can mask the relationship between PFC asymmetry and depression (Nusslock et al., 2018). Differences in asymmetry profiles between depression and subtypes of anxiety cause some to suggest that inconsistent asymmetry results may be due to mixed samples (Jesulola et al., 2015). In a meta-analysis, Thibodeau et al. (2006) found that EEG asymmetry studies differentiating depressed from control groups tended to report medium effect sizes, while those reporting asymmetry findings in co-morbid depression and anxiety versus control groups tended to report small effect sizes. In addition to PFC asymmetry, there are also parietal asymmetry disparities in individuals with depression versus anxiety. Individuals with anxiety tend to show greater relative right parietal activity while individuals with depression show greater relative left parietal activity, consistent with levels of arousal associated with each disorder (Mathersul et al., 2008; Spielberg et al., 2008). Because of these differences, it is worth assessing or excluding comorbid anxiety subtypes when studying asymmetry in individuals with depression.

**State and Trait Moderators of Brain Asymmetry**

A growing literature indicates that state and trait emotion factors can interact to influence, or moderate, patterns of PFC asymmetry, and vice versa (Coan & Allen, 2004). Moderation is present when two (or more) independent variables interact to statistically predict a dependent
variable (Field, 2013), and an example of moderation follows: individuals differing in trait negative emotionality (e.g., low versus high symptoms of depression) may show diverging PFC asymmetry patterns in response to a negative stimulus (e.g., an image of an isolated, sad person) related to prior predispositions to approach versus avoid stimuli they encounter (or experience positive versus negative affect) in their respective surroundings. It could also be the case that PFC asymmetry in response to an emotional stimulus (e.g., sadness) interacts with trait negative emotion (e.g., depression) to predict recovery (e.g., relative rightward PFC asymmetry) from that emotional challenge after its termination. In sum, it may be the case that state and trait factors measured by self-report, behavior, and/or EEG may interact to influence each other and impact how individuals respond to emotional stimuli, but more research is needed to enhance our understanding of these relationships.

**PFC Asymmetry as a Predictor of Depressive Risk**

As mentioned above, asymmetry patterns are stable, trait-like constructs, independent of state depression or severity level; therefore, it would reason that these patterns are present in individuals who have not yet experienced their first depressive episode. In fact, because of the strong familial component associated with asymmetry, individual differences in asymmetry can be seen from a young age in people with family loading for depression. Infants of depressed mothers, as young as one week old, show relative right PFC asymmetry characteristic of depression (Diego et al., 2004). The same pattern is also seen in children with depressed mothers at ages ranging from three months to six years old, and this pattern distinguishes children of depressed mothers from children of non-depressed mothers (Dawson, Klinger, Panagiotides, Hill, & Spieker, 1992; Field, Fox, Pickens, & Nawrocki, 1995; Goldstein et al., 2016; Jones, Field, & Almeida, 2009; Jones, Field, Davalos, & Pickens, 1997). Others have found that relative
right PFC asymmetry during both happy and sad film clips was also associated with higher levels of depressive symptoms for children who exhibit relative right PFC asymmetry at rest (Feng et al., 2012). Physiological evidence has supported the notion that children with depression risk show diminished reactivity to positive stimuli and greater reactivity to negative stimuli (Feng et al., 2012).

A number of factors contribute to the development of this asymmetry profile consistent with depression in youth. First, maternal depression influences the fetus from the second trimester of pregnancy and onward (Diego et al., 2004). Infants whose mothers were diagnosed with “prenatal depression” show greater relative right PFC asymmetry patterns than infants of mothers with postpartum depression alone (Diego et al., 2004). Second, the environment, specifically maternal interaction style, contributes to these asymmetry patterns. In line with the motivational theory, infants of mothers with an “inhibitory” style of relating show greater relative right PFC asymmetry than infants of mothers with “intrusive” styles (Diego, Field, Jones, & Hernandez-Reif, 2006); infants and toddlers demonstrate these same patterns when engaging in “inhibitory” play themselves (Jones et al., 1996). Further, asymmetry patterns of depression in infants shift to relative left PFC asymmetry when mothers shift to an “intrusive” style of interaction post-birth while asymmetry from birth shifts relatively rightward when mothers relate in an “inhibitory” manner (Diego et al., 2006). Third, biological and genetic factors may play a role in development of asymmetry trends due to their heritability, independent of environmental factors. Children of depressed parents and grandparents with depression show greater relative right PFC activity than children of depressed parents alone (Bruder, Tenke, Warner, & Weissman, 2007). Twin studies demonstrate that PFC asymmetry characteristic of depression is more highly heritable in young adult identical twins than fraternal twins, and more
so in twins than siblings (Smit et al., 2007). Taken together, genetics, prenatal conditions, and the environment contribute to the formation of asymmetry patterns that place individuals at risk for developing depression later in life.

Because individuals demonstrate asymmetry profiles reflective of depression from a young age prior to MDD onset, researchers argue that PFC asymmetry may be a neural biomarker of lifetime risk for depression (e.g., Stewart et al., 2010). As EEG is a relatively inexpensive neuroimaging method and the measurement of PFC asymmetry takes mere minutes, early screening and intervention could be implemented to assess this risk similar to the method developed by Mumtaz et al. (2017). Researchers focus on development of a first depressive episode since: (1) individuals with remitted MDD endorse higher sub-clinical symptoms than never depressed individuals, and (2) according to the “scar” hypothesis, a depressive episode leaves a “scar” of lingering and lasting psychological and brain changes even after remission (Gotlib et al., 1998; Pössel, Lo, Fritz, & Seemann, 2008; Rohde, Lewinsohn, & Seeley, 1990).

Nusslock et al. (2011) followed a cohort of adults without lifetime psychopathology at baseline over three years, and found that relative right PFC asymmetry predicts first (major and minor) depressive episodes. Similarly, greater relative right PFC activity at baseline correlates with increase in depressive symptoms one year later in a cohort of adolescents (Pössel et al., 2008). Consistent with these findings, Stewart and Allen (under review) reported that relative right PFC asymmetry predicts increase in depressive symptoms one year later in young adult women, but not men. Similarly, greater relative right PFC asymmetry is associated with future risk of depression in a twin sample but only in women; prior MDD status was also not assessed in this sample (Smit et al., 2007). This pattern of relative right PFC asymmetry also predicts increased anxiety symptoms in never-depressed adults over one year, which authors suggested is
a precipitant of a depressive episode based on previous research and due to the high comorbidity rate between depression and anxiety disorders (Blackhart, Minnix, & Kline, 2006). Moreover, there is some predictive value in that greater relative right PFC asymmetry during an emotional film clip predicts mood decrease over the following week as well as depressive symptoms one year later (Mitchell & Pössel, 2012; Papousek et al., 2014). These results have demonstrated that asymmetry risk patterns are reflective not only of an immediate response to an emotion stimulus, but also of recovery from an emotional event.

In sum, neurobiological and environmental factors combine to yield patterns of asymmetry seen in depression (Jesulola et al., 2015). In line with the diathesis-stress model, trait-like individual differences in PFC asymmetry, developed as a result of biological factors and environment, may reflect a bias in one’s processing style that can influence depression vulnerability and therefore serve as a biomarker for depression.

**Trait Emotion Regulation as a Moderator of Brain Asymmetry**

Despite the potential importance of this neural risk marker for depression, few studies have looked at the extent to which individual difference factors, such as emotion regulation (ER), that engage the PFC, can affect PFC asymmetry patterns. Although theorists may differentially define ER, it generally involves the modulation of the intensity and duration of emotional responses in pursuit of a goal (Eisenberg & Spinrad, 2004; Gross, 1998). Certain trait or habitual use of ER strategies such as suppression, avoidance, and rumination are associated with poor outcomes and serve as risk factors for various psychopathologies, including depression, anxiety, and eating and substance-related disorders (Aldao, Nolen-Hoeksema, & Schweizer, 2010; Gross, 1998; Gross & John, 2003). Suppression is when one artificially dampens his or her emotions, thoughts, or emotional expressions. Avoidance is defined as evading psychological experiences
such as thoughts, emotions, sensations, urges or memories. Lastly, rumination as a regulatory strategy is a repetitive focus on one’s experience of emotion and its causes and consequences (Aldao et al., 2010). The negative consequences associated with these strategies include cognitive, physiological and emotional costs such as persistent negative affect, decreased habituation to the environment, increased physiological arousal, lower reported life satisfaction and self-esteem, lower emotional awareness, greater avoidance of close relationships, and impaired memory (Aldao et al., 2010; Gratz & Roemer, 2008; Gross, 1998; Gross & John, 2003).

On the other hand, common adaptive ER strategies including positive reappraisal, problem solving, and mindfulness are shown to be beneficial with minimal psychological, physiological, and cognitive costs (Aldao et al., 2010; Gross, 1998). The strategy of positive reappraisal is when one construes a situation in order to change its emotional impact (Gross & John, 2003). Of note, it is possible for individuals to negatively reappraise situations or reinterpret a situation or shift attention in a way that up-regulates the negative aspects of one’s emotional experience; in this case the strategy would be maladaptive, although the term “reappraisal” is assumed to refer to its positive counterpart. Problem-solving, another adaptive strategy, is a conscious attempt to change a situation or contain its consequences, including creating a plan or generating solutions to a problem (Aldao et al., 2010). Finally, mindfulness as a regulatory strategy includes a purposeful focus on the present moment in an accepting manner (Brown & Ryan, 2003).

ER is a system that relies on cortical centers, such as the PFC, to increase or decrease the contribution of limbic regions to affective responses and is therefore considered a higher order cognitive process (Gross, 1998; Ochsner, Bunge, Gross, & Gabrieli, 2002; Ochsner et al., 2004).
Researchers posit that disorders involving maladaptive ER styles may be a reflection of faulty neurocircuitry. Specifically, the PFC plays a major role in the ER process by integrating goal planning with other brain regions involved in monitoring, conflict resolution, memory and emotional reactivity (Davidson, Pizzagalli, Nitschke, & Putnam, 2002). Since asymmetry patterns are thought to reflect an individual’s dispositional affective style (Davidson, 1992; Davidson, 1998a,b), it would be helpful to determine whether individual differences in use of adaptive or maladaptive ER strategies are linked to hemispheric differences in brain processing. According to classic ER theories, strategies such as suppression are maladaptive because the regulator simply avoids dealing with the emotion and ignores it on a cognitive level after it is elicited (Gross, 1998). As a result, physiological costs of negative mood persist and do not yield a successful improvement in emotional or physiological states. On the other hand, adaptive strategies typically cause people to modulate and face their emotions on a deeper level, resulting in more optimal outcomes (Gross, 1998).

Adaptive and maladaptive ER is associated with PFC lateralization and relative left and right PFC asymmetry respectively. This trend is seen on both trait and state levels, either when studying individual differences in habitual regulation or when instructing people how to regulate. Trait use of reappraisal and other adaptive strategies has been associated with relative left PFC asymmetry (Blackhart et al., 2006; Choi, Sekiya, Minote, & Watanuki, 2016; Forbes et al., 2006; Kim, Cornwell, & Kim, 2012; Meyer et al., 2014; Mikolajczak, Bodarwé, Laloyaux, Hansenne, & Nelis, 2010; Tomarken & Davidson, 1994), whereas frequent use of maladaptive strategies such as suppression has been associated with relative right PFC asymmetry (Kim et al., 2012). Additionally, successful uninstructed regulation assessed during an ER task is associated with greater relative left PFC asymmetry (Goodman, Rietschel, Lo, Costanzo, & Hatfield, 2013;
Jackson et al., 2003). These findings support a relationship between trait ER and brain activity, as reflected by asymmetry patterns.

Similar effects are seen on a state level wherein individuals are instructed to regulate via certain strategies. As expected, instructed use of adaptive strategies such as reappraisal, expressive relative coping, meditation, positive refocusing, and mindfulness have been associated with relative left PFC asymmetry (Barnhofer, Chittka, Nightingale, Visser, & Crane, 2010; Choi et al., 2016; Kim et al., 2012; Master et al., 2009; Meyer et al., 2014; Moynihan et al., 2013; Wang et al., 2015; Watford & Stafford, 2015) and instructed use of maladaptive strategies have been associated with relative right PFC asymmetry (Lévesque et al., 2003; Lewis, Taubitz, Duke, Steuer, & Larson, 2015). However, other studies did not find significant effects with instructed mindfulness or maladaptive strategies (Choi et al., 2016; Keune, Bostanov, Kotchoubey, & Hautzinger, 2012). It is possible that PFC asymmetry shares a more robust relationship with adaptive rather than maladaptive ER styles. PFC asymmetry is thought to more adequately reflect the behavioral activation system rather than the behavioral inhibition system (Keune et al., 2012) and it would reason that adaptive strategies, which are thought to reflect approach mechanisms, may be more closely related to the behavioral activation system than maladaptive strategies.

Further, stimulation of the right hemisphere via high frequency repetitive transcranial magnetic stimulation and direct current stimulation results in increased rumination and difficulties regulating and inhibiting negative stimuli (Kelley, Hortensius, & Harmon-Jones, 2013; Leyman, De Raedt, Vanderhasselt, & Baeken, 2009), again implicating lateralized physiological networks in regulatory ability. Overall, habitual and experimentally manipulated
ER findings are congruent with asymmetry trends as a function of motivational and valence theories of emotion.

**ER, Depression and PFC Asymmetry**

Emotion dysregulation has been implicated as a central feature in various psychopathologies, including MDD (Aldao et al., 2010; Joormann & Stanton, 2016). ER deficits in depression are characterized by the combination of sustained negative affect and relative lack of positive affect (Joormann & Stanton, 2016). People who experience difficulty regulating their emotional responses to daily stressors may have a persistent experience of distress, which in turn can evolve into a depressive episode (Aldao et al., 2010). As such, ER can serve as a risk or protective factor to the development of depression. MDD is associated with greater use of maladaptive strategies including suppression, avoidance, rumination, catastrophizing, and self-blame, as well as a decrease in spontaneously implemented adaptive strategies, such as positive reappraisal (Aldao et al., 2010; Garnefski & Kraaij, 2006b; Joormann & Stanton, 2016). However, research has shown that there are no differences in ER effectiveness among depressed and non-depressed individuals when both groups are instructed to regulate adaptively (for a review, see Joormann & Stanton, 2016). Of note, researchers have generally found weaker associations between psychopathology and adaptive regulatory strategies, although some suggest that this may be due to the fact that adaptive strategies are implemented more variably across situations than maladaptive strategies, where the use is more consistent (Aldao & Nolen-Hoeksema, 2012).

ER deficits in depression can be conceptualized from neurological and cognitive perspectives (see Joormann & Stanton, 2016 for a review). The combination of difficulty in controlling negative affect as well as sustaining positive affect is reflected in corresponding brain
structures in individuals with MDD. Specifically, individuals with depression show: (1) reduced DLPFC activity, which is important for cognitive control; (2) increased ventrolateral PFC activity, associated with dampening of positive emotion; and (3) lack of sustained activity in the nucleus accumbens and striatal pathways associated with reward processing. Cognitively, individuals with depression show attention biases in the later stages of attentional processing, including problematic disengagement and mood recovery (e.g., Joormann, Talbot, & Gotlib, 2007; Koster, De Lissnyder, Derakshan, & De Raedt, 2011). Indeed studies have found that individuals who use maladaptive strategies and exhibit rightward PFC asymmetry are at increased risk for developing MDD (Joormann & Stanton, 2016).

Because ER asymmetry patterns are congruent with those present as a function of motivational style (i.e., approach motivation and adaptive strategies are linked to left PFC asymmetry), it would reason that motivational systems associated with each class of ER styles would promote relative left versus right PFC processing corresponding to approach and withdrawal systems respectively (Watford & Stafford, 2015). Perhaps there is an inherent approach component to adaptive strategies as well as a withdrawal component to maladaptive strategies, which in turn yield positive or negative mood states to drive this effect. According to initial research, ER is lateralized; however, few studies have examined how self-reported trait adaptive and maladaptive ER styles (e.g., cognitive reappraisal and suppression, respectively) influence PFC asymmetry when individuals attempt to recover from state emotional provocations.

An added component of this trend may be a deficit in the ER process such that depressed individuals experience difficulties disengaging from negative stimuli and events (Pereira & Khan, 2016), resulting from maladaptive and unsuccessful regulation attempts. Similarly, other
researchers hypothesize weaknesses in recovery from negative mood, as individuals with relative right PFC asymmetry and suboptimal ER styles persist longer in their physiological response to negative stimuli, thereby maintaining rightward PFC asymmetry (Gatzke-Kopp, Jetha, & Segalowitz, 2014; Jackson et al., 2003). Conversely, adaptive ER is associated with persisting benefits. For instance, individuals instructed to reappraise exhibit greater leftward PFC asymmetry than those in a sham reappraisal group during a stress-inducing event; moreover, the reappraisal group continues to exhibit greater relative left PFC asymmetry upon reintroduction to the stressful stimulus, supporting the sustained benefits of adaptive regulation (Wang et al., 2015).

In sum, habitual and instructed use of adaptive ER strategies are linked to relative left PFC asymmetry while habitual and instructed use of maladaptive ER strategies are linked to relative right PFC asymmetry. Difficulties in ER are implicated in depression risk and have been explained on a neurological, cognitive, and physiological level in line with various theories of asymmetry. Given the prevalence and burden associated with depression, understanding these patterns and defining a population at risk for depression would be invaluable for early intervention and preventative care.

**The Present Study**

The current study employed a between-subjects emotion induction paradigm wherein healthy young adults were randomly assigned to either a happy or sad state mood manipulation induced via film clip. EEG was measured three times throughout the study: once during the mood induction and twice during resting states, before and after the film clip, to examine changes in patterns of brain asymmetry as a function of emotional challenge. State and trait measures of emotion experience and ER as well as individual measures of depressive symptoms
known to impact asymmetry were also measured via questionnaire. For the longitudinal component of this investigation, participants were followed one year later to complete a short clinical interview regarding MDD symptoms in the past year as well as questionnaires assessing depression symptoms. Severity level of depressive symptoms was used a dimensional measure to assess the onset of depression. Although MDD status was collected in the interview, categorical analyses would require a larger sample size and longer follow-up interval and research has demonstrated that greater depressive symptom severity, measured on a continuum, is associated with first-onset MDD (Horwath, Johnson, Klerman, & Weissman, 1992).

The present study tested five hypotheses based on findings from prior literature. It was predicted that: (1) the sad film clip would elicit greater relative right PFC asymmetry than the happy film clip, consistent with prior state emotion manipulations (e.g., Coan et al., 2006); (2) higher trait sadness (depression symptoms) indexed by the Beck Depression Inventory II (BDI-II; Beck, Steer, & Brown, 1996) would be associated with greater relative right PFC asymmetry, particularly during and after the film clip, consistent with previous depression findings during state emotion manipulations (Stewart et al., 2011a); (3) greater relative right PFC asymmetry during the film clip would predict an increase in future depressive symptoms measured one year later; (4) adaptive trait indices of ER (e.g., cognitive reappraisal) would be linked to greater relative left PFC asymmetry differences during and after the film clip, whereas maladaptive indices (e.g., suppression) would be associated with greater relative right PFC asymmetry differences at these time points, consistent with studies discussed above; and (5) use of ER strategies would be associated with an increase in depressive symptoms one year later, such that adaptive indices would be linked to decreased risk whereas maladaptive indices would be linked to increased risk for depression.
Although primary hypotheses are based on differences as a function of PFC asymmetry, exploratory analyses were computed for parietal asymmetry as well, which is thought to reflect differences as a function of arousal instead of valence/motivation (Heller, 1993; see Stewart et al., 2011b for a review). Analyses were also run with EEG change scores as a result of the film clip mood induction to explore whether “movement” in PFC asymmetry is more associated with mood and ER variables than EEG recorded at one time point alone. Given the high comorbidity between depression and anxiety as well as previous findings in the literature, the current investigation also explored whether current and future anxiety symptoms would be associated with PFC asymmetry.

**Method**

**Participants**

Participants were recruited via flyers posted in the Queens College, CUNY campus in Flushing, NY and in local community centers. An email detailing study procedures and inclusion criteria was also sent to the Queens College, CUNY, Psychology Department listserv. Participants were phone-screened by trained laboratory staff for the following inclusion criteria: (1) no history of epilepsy, migraines, head injury, other neurological disorder, or loss of consciousness > 5 minutes; (2) age range of 18-25 years old; (3) English fluency by age 9; (4) normal color vision; (5) no lifetime presence of DSM-5 (APA, 2013) mania, psychosis, attention-deficit hyperactivity disorder, substance use disorders, obsessive-compulsive disorders, posttraumatic stress disorder, eating disorders, mood and anxiety disorders; and (6) no current psychotropic medications including antidepressants. Both left- and right-handed participants were included in the study (right = 35, left = 3). All participants provided oral and written consent.
Procedures

Potential study participants were phone-screened to determine study eligibility using the inclusion criteria listed above. Eligible participants were invited to the laboratory to complete a written consent form as well as DSM-5 structured clinical interview administered by advanced clinical psychology doctoral students. The Anxiety and Related Disorders Interview Schedule (ADIS-5; Brown & Barlow, 2014) was administered to confirm absence of lifetime Axis 1 disorders. Family history of first-degree relatives with depression was gathered as well (Table 1). All members of the clinical team met to determine diagnosis consensus.

Participants who met criteria were then invited to the laboratory at a later date for an EEG recording session. Participants first completed a number of brief questionnaires reporting hours slept the previous night, and recent food, alcohol, caffeine, nicotine, marijuana and other drug consumption. They were then fitted with a 128-channel Electrical Geodesics, Inc. (EGI; Eugene, OR) EEG cap and headphones, which they wore for EEG recording. Participants were seated in a recliner in a dark room and were asked to sit quietly and focus on a fixation cross while resting baseline EEG activity was recorded for five minutes (pre-clip EEG). After baseline recording, EEG was recorded while participants were shown a three-minute happy or sad film clip (see Mood Induction below) on a 21” monitor, corresponding to the condition to which they were randomly assigned at the start of the study (film clip EEG). Next, participants completed two blocks of an inhibition task (results not presented here) followed by another five-minute eyes open resting baseline after the film clip was presented (post-clip EEG). Lastly, experimenters removed the EEG cap, participants completed measures to assess their current mood state as a result of the induction after watching the film clip as well as trait ER and depression.
questionnaires and were orally debriefed. Participants were compensated $10 for the clinical interview and $30 for the EEG session.

Participants were contacted one year following the EEG session to complete questionnaires and a DSM-5 structured clinical interview addressing MDD symptoms within the past year, including a repeat administration of the BDI-II; the interview was administered by advanced clinical psychology doctoral students. The follow-up duration of one year has been used in previous research (Blackhart et al., 2006; Mitchell & Pössel, 2012; Pössel et al., 2008) to capture a change in depressive symptoms. The session was conducted in the lab or remotely and participants were compensated $30 for participation. 35 (92%) of the 38 participants completed the follow-up session.

**Mood Induction**

Participants were randomly assigned to one of two emotion induction groups and then were induced into happy (n = 16) or sad (n = 22) mood states via commercial film clips according to standardized procedures used in previous research (Rottenberg, Ray, & Gross, 2007; Storbeck, 2012; Storbeck & Watson, 2014). The “sad” clip was a three-minute excerpt from “The Champ” movie, and the three-minute “happy” clip was taken from a Seinfeld stand-up comedy routine.

**Questionnaires**

After the film clip, participants completed a post-film questionnaire and momentary mood scale as a check for the mood manipulation, and the Positive and Negative Affect Scale (PANAS; Watson, Clark, & Tellegen, 1988) to assess current mood. After the EEG session was completed, participants completed the BDI-II, the Positive and Negative Affect Scale, Extended (PANAS-X; Watson & Clark, 1994), the Penn State Worry Questionnaire (PSWQ; Meyer,
Miller, Metzger, & Borkovec, 1990), the Mood and Anxiety Symptom Questionnaire (MASQ; Watson et al., 1995), the Emotion Regulation Questionnaire (ERQ; Gross & John, 2003) and the Cognitive Emotion Regulation Questionnaire Short Form (CERQ-short; Garnefski & Kraaij, 2006a). During the follow-up session, participants completed additional measures as well as repeat administrations of the MASQ and PSWQ and two repeat administrations of the BDI-II. For the first BDI-II administered, participants were instructed to recall the most impairing month that they experienced over the previous year and to answer the items referring to that time period (worst BDI-II at follow-up). During the second BDI-II, participants were instructed to complete the questionnaire as it is traditionally administered, endorsing current symptoms that were present over the two weeks prior to administration (current BDI-II at follow-up). Of the two follow-up BDI-II questionnaires, the worst BDI-II, which captures an increase in depressive symptoms at any time-point over the prior year, better fit with the primary goal of the follow-up aims. Furthermore, more participants completed the worst BDI-II over the past year than follow-up current BDI-II (n=35 versus n=31, respectively). For these reasons, the worst BDI-II was used as the follow-up depressive symptom index in all analyses over the current BDI-II at follow-up.

The ERQ (Gross & John, 2003) is a 10-item self-report measure focusing on two well-defined and commonly used ER strategies: expressive suppression and cognitive reappraisal. Expressive suppression is the artificial dampening of one’s emotional expression (e.g. modulating facial expression to hide sadness) while cognitive reappraisal refers to the reconstruction of a situation in order to change its emotional impact (e.g. reframing a negative situation in a positive light). These strategies originate from two distinct points in the ER process. Cognitive reappraisal is known as an antecedent-focused strategy, performed earlier in the regulatory process, since it alters the trajectory of an emotional experience while expressive
suppression, a response-focused strategy, occurs once the emotion is elicited. Expressive suppression is related to greater experience and expression of negative emotion, whereas cognitive reappraisal is related to greater experience of positive emotion and less experience of negative emotion. The relationship between reappraisal and suppression is independent, meaning that individuals who reappraise frequently are no more or less likely to suppress than those who reappraise less. Higher scores on each subscale indicate greater levels of agreement with each respective strategy (Gross & John, 2003).

The CERQ (Garnefski & Kraaij, 2006a) assesses nine different cognitive strategies of ER. The CERQ short form, used in the current study, is an 18-item questionnaire with two items per subscale, reduced from the 36-item original with four items per subscale. Higher scores indicate greater use of each strategy. Subscales include self-blame, blaming others, acceptance, refocus on planning, positive refocusing, rumination or focus on thought, positive reappraisal, putting into perspective, and catastrophizing. The categories cluster into two categories: a theoretically more “adaptive” category termed “positive-focused cognitive ER” (positive refocusing, positive reappraisal, putting into perspective, refocus on planning, assessment) and a “less adaptive” group or “negative-focused cognitive ER” (rumination, self-blame, blaming others, catastrophizing). After controlling for the influence of other strategies, analyses by the authors show a significant positive relationship between rumination, self-blame and catastrophizing and depressive symptoms. Conversely, a negative relationship is observed between depression and positive refocusing and positive reappraisal (Garnefski & Kraaij, 2006a).

The BDI-II is a method of assessing depressing symptoms via self-report. The 21-items each correspond to different symptoms of depression such as sadness, feelings of worthlessness,
loss of pleasure, irritability, fatigue, and changes in sleep and appetite, with greater scores indicating higher depression severity (Beck et al., 1996).

Two questionnaires were used to evaluate symptoms of anxiety: the MASQ and the PSWQ. The MASQ is based on a “tripartite” model with two distinct categories of depression and anxiety symptoms and one category of overlapping symptoms. The subscale of interest is referred to as the Anxious Arousal scale (AA), which is comprised of 18-items that capture hyperarousal, a component distinctly attributed to anxiety (Watson et al., 1995). The PSWQ is a 16-item questionnaire that measures the trait of worry, which is regarded as a dominant feature of generalized anxiety (Meyer et al., 1990).

**EEG Data Collection and Reduction**

EEG data were collected using a 128-channel Hydrocel Geodesic Sensor Net (see Figure 1) connected to a NET 300 amplifier and Net Station acquisition system supplied by Electrical Geodesics Incorporated (Eugene, OR). Impedances were maintained under 50K Ohms and data were digitized continuously at 250 Hz. Although data were acquired with an online Cz reference, they were subsequently re-referenced offline to averaged (“linked”) mastoids (LM; channels 57 and 100 illustrated in Figure 1).

After acquisition, data were reduced from 128 to 38 channels for the present analysis (see 32 frontal and parietal channels highlighted in green in Figure 1 as well as two LM channels and electrooculogram (EOG) channels 125-128). Trained research assistants visually inspected and manually removed continuous data segments with movement and muscle artifacts using EEGLab (Delorme & Makeig, 2004) run on the MATLAB R2016a platform (Mathworks, Natick, MA). Data were then filtered from 0.1-30Hz and upsampled to 1000Hz to implement further data reduction using custom MATLAB scripts identical to those used in Stewart, Bismark, Towers,
Coan, and Allen (2010). A blink rejection algorithm rejected data segments where ocular activity exceeded +/- 75 microvolts in the vertical ocular channel (126 in Figure 1), and an artifact rejection algorithm rejected segments with large fast deviations in amplitude in any channel (e.g., direct current shifts and spikes) that may have eluded human inspection. Data were epoched into 2.048 second epochs, overlapping by 1.5 seconds. This overlapping compensates for the minimal weight applied to the end of the epoch by the use of the Hamming window function. Following windowing, a Fast Fourier Transform (FFT) was applied to all artifact-free epochs. The power spectra from all artifact-free epochs were averaged to provide a summary spectrum for each condition (pre-clip, film clip, and post-clip). Total alpha power (8-13 Hz) was then extracted from the spectrum for each condition and each of the 32 EEG channels (EOG and LM channels were omitted).

Alpha power was log-transformed for each channel and condition due to skew. Eight channels each of log-transformed data were averaged to obtain left frontal (22, 23, 24, 26, 27, 28, 33, 34), right frontal (2, 3, 9, 116, 117, 122, 123, 124), left parietal (51, 52, 53, 58, 59, 60, 65, 66) and right parietal (84, 85, 86, 90, 91, 92, 96, 97) composites per condition (see Figure 1 for channel locations). Frontal and parietal asymmetry scores for each condition were then calculated by subtracting the natural log transformed average scores (i.e., ln[Right] – ln[Left]). Higher values on this index putatively reflect relatively greater left activity (i.e., greater right than left alpha; cf. Allen et al., 2004a).

**Statistical Analysis**

**Demographic information and questionnaires.** Independent sample t-tests compared age, ERQ cognitive reappraisal, ERQ suppression, CERQ adaptive strategies, CERQ maladaptive strategies, BDI-II baseline, worst BDI-II at follow-up, current BDI-II at follow-up,
MASQ AA baseline, MASQ AA follow-up, PSWQ baseline, and PSWQ follow-up scores between groups (happy, sad). Homogeneity of variance was assessed by Levene’s test. A chi-square test was computed to evaluate sex differences between groups. Cohen’s $d$ is reported as an index of effect size.

Mood manipulation check. Independent sample t-tests were compared between groups (happy, sad) to determine successful mood manipulation via film clips. Self-reported mood ratings corresponding to the predicted mood states (e.g., happy, cheerful, sad, unhappy) were dependent variables. Levene’s test was computed to assess the homogeneity of variance assumption between groups. If the mood induction was successful: (1) the happy group should endorse positive emotions such as “happy” and “pleasant” more than the sad group; and (2) the sad group should endorse negative emotions such as “sad” more than the happy group. Cohen’s $d$ is reported as an index of effect size.

EEG data quality check. A mixed analysis of variance (ANOVA) was conducted to determine whether the number of usable epochs (dependent variable) differed after artifact removal as a function of group (between-subjects variable: happy, sad) and time (within-subjects variable: pre-clip, clip, post-clip). Moreover, epochs per subject were compared to an acceptable cutoff for reliable epochs as established by the asymmetry literature (>100; Towers & Allen, 2009). Levene’s test assessed homogeneity of variance between groups, whereas corrections for sphericity were applied as appropriate. Partial eta-squared was computed as a measure of effect size.

Aim 1. State PFC asymmetry: Group analysis. To examine our first hypothesis that the sad group would exhibit greater rightward PFC activity than the happy group during and after the film clip, a mixed ANOVA was conducted with group (happy, sad) as the between-subjects
factor and condition as the within-subjects factor (pre-clip, clip, post-clip), with PFC asymmetry score averaged across 8 channel pairs as the dependent variable. The group by condition interaction was the effect of interest. Levene’s test assessed homogeneity of variance between groups, whereas corrections for sphericity were applied as appropriate. Partial eta-squared was computed as a measure of effect size.

Exploratory Analyses. State parietal asymmetry: Group analysis. An analogous exploratory ANOVA was computed with parietal EEG asymmetry score averaged across 8 channel pairs as the dependent variable.

Aim 2. Film clip PFC asymmetry: Depression scores. To examine our second hypothesis that trait sadness (depressive symptoms) would be associated with greater relative right PFC activity during the film clip (regardless of happy or sad group status), a Pearson’s correlation was computed between z-scored baseline BDI-II scores and z-scored clip PFC asymmetry. Furthermore, to examine whether depression would moderate relationships between PFC asymmetry during and after the film clip in favor of relative right PFC activity, regressions were computed across groups (n=38) to examine whether individual differences in self-reported trait depressive symptoms (measured via BDI-II), PFC asymmetry during the film clip, and their interaction would predict post clip PFC asymmetry. After the total BDI-II z-score is entered (predictor 1), z-scored PFC asymmetry during the film clip (predictor 2) and their interaction (predictor 1 x predictor 2) were included together to predict the z-scored PFC asymmetry score post-clip (dependent variable).

Aim 3. Film clip PFC asymmetry: Depression Risk. To examine our third hypothesis testing whether clip PFC asymmetry predicting an increase in depressive symptoms at follow-up across groups (n=38), a regression was computed with z-scored PFC clip asymmetry, z-scored
baseline BDI-II score, and their interaction predicting z-scored worst BDI-II score at follow-up.

**Aim 4. Film clip PFC asymmetry: ER scores.** To investigate our fourth hypothesis that trait ER styles would be associated with PFC asymmetry during the film clip, Pearson correlations were computed between z-scored clip PFC asymmetry scores and four z-scored ER indices: (1) ERQ cognitive reappraisal; (2) ERQ suppression; (3) CERQ adaptive strategies; and (4) CERQ maladaptive strategies. We examined whether ER styles would interact with film clip PFC asymmetry to predict emotional recovery via post-clip PFC asymmetry (with reappraisal and other approach-related styles such as planning linked to relative left PFC activity, and low use of suppression and other withdrawal-related styles linked to relative left PFC activity). To this end, four regressions were computed, wherein each z-scored trait ER subscale was entered (predictor 1), followed by the z-scored PFC asymmetry score during the film clip (predictor 2) and their interaction (predictor 1 x predictor 2) all in the same step to predict the z-scored PFC asymmetry score post-clip (dependent variable). Of note, regression analyses were computed for both ERQ subscales (cognitive reappraisal and suppression), whereas the nine CERQ subscales were split into two categories: adaptive (positive refocusing, positive reappraisal, putting into perspective, refocus on planning, assessment) and maladaptive (rumination, self-blame, blaming others, catastrophizing) ER based on previous research (Garnefski & Kraaij, 2006a), reducing the number of statistical tests computed to minimize type I error inflation.

**Exploratory analyses. EEG Change Score.** Difference scores were computed between PFC asymmetry pre- to post- and clip-to post- and were each correlated with worst BDI-II score at follow-up and ER indices, as it was hypothesized that EEG change, or an individual’s ability to recover from a brief emotional event, would predict greater depressive symptoms or be associated with certain ER styles more so than a static EEG score within a respective time block.
Aim 5. ER scores, Depression Risk, Trait PFC Asymmetry. To investigate our fifth hypothesis that baseline ER strategies would be associated with an increase in depressive symptoms at follow-up, a regression analysis was conducted, examining if z-scored BDI-II at baseline, z-scored ER subscale, and their interaction predicted z-scored worst BDI-II at follow-up.

Exploratory Analyses. Anxiety. To explore the role of anxiety in PFC asymmetry, correlations were run between anxiety measures (e.g., MASQ baseline, MASQ at follow-up, PSWQ baseline, PSWQ at follow-up), depressive measures (e.g., BDI-II baseline, current BDI-II at follow-up, and worst BDI-II at follow-up), and pre-, clip-, and post- PFC and parietal asymmetry. Due to an error, the PWSQ at baseline was not administered to all participants. As a result, descriptive statistics are reported but the measure was not used in analyses. Several moderations were then conducted examining if: 1) z-scored MASQ at baseline, z-scored PFC asymmetry score during the film clip, and their interaction predicted z-scored MASQ at follow-up; 2) z-scored MASQ at baseline, z-scored PFC asymmetry score during the film clip, and their interaction predicted z-scored PFC asymmetry score post-clip; 3) z-scored BDI-II at baseline, z-scored PFC asymmetry score during the film clip, and their interaction predicted z-scored MASQ at follow-up; 4) z-scored PSWQ at follow-up, z-scored PFC asymmetry score during the film clip, and their interaction predicted z-scored PFC asymmetry score post-clip; and 5) z-scored BDI-II at baseline, z-scored PFC asymmetry score during the film clip, and their interaction predicted z-scored PSWQ at follow-up.

Results

Demographic and Questionnaires
Table 1 displays demographic, trait and state emotion questionnaire responses, baseline/follow-up depressive symptoms, number of usable EEG epochs, and PFC/parietal asymmetry scores per condition as a function of group membership. Baseline BDI-II, current BDI-II at follow-up, and worst BDI-II at follow-up were not normally distributed; to approximate normality, log transformed data was used in place of the raw scores for the respective questionnaires in all analyses. Groups did not differ on any questionnaire. Of note, some participants did not complete all scales, contributing to variance in sample size across measures (Table 1).¹

**Mood Manipulation Check**

Analysis of PANAS state emotion items indicated that the state emotion induction was successful, wherein the happy group exhibited greater happiness than the sad group, who in turn endorsed higher unhappiness and sadness than the happy group (see Table 1 for descriptive and inferential statistics).

**EEG Data Quality Check**

Groups did not differ on number of usable epochs across or between conditions; however, participants had a lower number of usable epochs during the film clip than pre- or post-clip. One participant did not have the number of epochs determined usable according to the literature (>100; Towers & Allen, 2009) in one of the three time conditions (post=97); however, data from this participant was used in the present analysis to maintain a sizable sample. All other data were

¹ Given that EEG asymmetry effects are typically more robust in women (Stewart, et al., 2010), correlations were run amongst women who participated in the study (n=21) to examine associations between depression, anxiety, ER, and PFC and parietal asymmetry scores before, during, and after the film clip mood induction. Significant correlations emerged between several anxiety and depression measures in women only (results not presented here); however, there were no significant associations between mood, anxiety and ER with PFC or parietal asymmetry.
determined to be within proper bounds for analysis and interpretation (see Table 1 for descriptive and inferential statistics).

**Aim 1. State PFC asymmetry: Group analysis.**

Figure 2 illustrates that PFC asymmetry did not differ as a function of group, condition, or their interaction (see Table 1 for descriptive and inferential statistics).

**Exploratory Analyses. State parietal asymmetry: Group analysis.**

Similarly, Figure 3 illustrates that parietal asymmetry did not differ as a function of group, condition, or their interaction (see Table 1 for descriptive and inferential statistics).

**Aim 2. Film clip PFC asymmetry: Depression scores.**

No significant correlation emerged between baseline BDI-II scores and clip PFC asymmetry across groups, $r(37)=0.06, p=0.75$. Within the moderation analysis, there was a significant main effect wherein greater left PFC asymmetry during the film clip predicted greater left PFC asymmetry post-clip. Furthermore, there was a significant interaction wherein lower baseline BDI-II scores (less depressive symptoms) and greater relative left PFC asymmetry during the clip predicted greater relative left PFC asymmetry post-clip, as illustrated in Table 2 and Figure 4.

**Aim 3. Film clip PFC asymmetry: Depression Risk.**

A significant correlation emerged between current BDI-II at follow-up and worst BDI-II at follow-up, $r(35)=0.73, p<0.001$. Overall, 63% ($n=22$) of participants experienced an increase in depressive symptoms from baseline BDI-II to worst BDI-II at follow-up, 31% ($n=11$) experienced a decrease in symptoms, and 6% ($n=2$) did not experience a change in symptoms.

Table 3 illustrates that although higher baseline BDI-II scores were associated with higher worst BDI-II scores at follow-up, neither baseline PFC clip asymmetry nor its interaction
with baseline BDI-II predicted follow-up worst BDI-II score (Figure 5).

**Aim 4. Film clip PFC asymmetry: ER scores.**

No significant correlations emerged between trait ER indices and PFC asymmetry during the film clip (Table 4a). Table 4b includes regression model results across groups, whereas Figures 6a-d illustrate moderation effects. Across all regression models, greater left PFC asymmetry during the film-clip predicted greater left PFC asymmetry post-clip. There was a significant main effect such that higher ERQ cognitive reappraisal scores predicted greater left PFC asymmetry post-clip. Furthermore, there was a significant interaction wherein greater ERQ cognitive reappraisal scores and greater left PFC asymmetry during the film clip predicted greater left PFC asymmetry post-clip, as illustrated in Figure 6a. Figure 6c illustrates a similar interaction effect for CERQ adaptive, wherein higher CERQ adaptive scores and greater left PFC asymmetry during the film-clip predicted greater left PFC asymmetry post-clip. Of note, there was a significant correlation between z-scored ERQ cognitive reappraisal and z-scored CERQ adaptive, $r(35)=0.46$, $p<0.01$, suggesting that similar ER factors contributed to this significant effect. Regression analyses for each individual group (happy and sad) showed similar results (findings not presented here). Moderation analyses with ERQ suppression and CERQ maladaptive ER as predictors were not significant (Figures 6b, 6d).

**Exploratory Analyses. EEG Change Score.**

A significant correlation emerged between ERQ cognitive reappraisal and the pre- to post- difference score, $r(37)=-0.41$, $p<0.05$, and between ERQ cognitive reappraisal and the clip- to post- difference score, $r(37)=-0.46$, $p<0.01$ (Table 6). In other words, individuals who

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2 This moderation was run with only women in the sample and did not yield significant results.
frequently use ERQ cognitive reappraisal demonstrated a significant leftward shift in their PFC asymmetry from pre- to post- and from clip- to post-film clip EEG recordings.

**Aim 5. ER scores, Depression Risk, Trait PFC Asymmetry.**

A significant main effect emerged wherein individuals who frequently use adaptive CERQ strategies demonstrated greater follow-up worst BDI-II scores (Table 5). This effect was still significant even when BDI-II at baseline was accounted for ($p<0.05$). However, results from the regression analyses were not significant (Figure 7). Given that this result was contrary to general field findings, a correlation scatterplot was generated with z-scored CERQ adaptive and z-scored worst BDI-II at follow-up. There were two outliers that emerged in the data and once the data of the two participants was removed, the regression results were no longer significant.\(^3\)

**Exploratory Analyses. Anxiety.**

Significant associations emerged between MASQ baseline and BDI-II baseline, $r(35)=0.38$, $p<0.05$, MASQ Anxious Arousal at follow-up and current BDI-II at follow-up, $r(35)=0.41$, $p<0.05$, and PSWQ at follow-up and BDI-II worst at follow-up, $r(35)=0.58$, $p<0.01$. Correlations between baseline and follow-up anxiety scales and PFC and parietal asymmetry scores were not significant. Regression analyses were not significant (results not presented here).

**Discussion**

This study sought to investigate five hypotheses relating to EEG asymmetry, depressive symptoms, trait/state emotion, and ER. First, it was predicted that the sad group would exhibit greater right PFC asymmetry during and after the film clip than the happy group. This hypothesis was not supported. No differences between happy or sad groups emerged for PFC asymmetry, likely due to lack of statistical power to detect small-to-medium effect sizes within the context of

\(^3\) This moderation was run with only women in the sample and did not yield significant results.
this between-subjects design. When manipulating state emotions (anger, joy, sadness, fear, and
disgust) within-subjects (a more powerful design than between-subjects manipulations) in a
sample of 31 healthy individuals, Coan and Allen (2003) detected only small-to-medium effect
sizes for correlations between PFC asymmetry and anger, joy, and fear; moreover, they did not
find significant results with regard to sadness or disgust, independent of reference scheme
(average and linked mastoids). In contrast, Stewart et al. (2011a) demonstrated medium-to-large
effect sizes for PFC asymmetry differentiating depressed and non-depressed groups during a
state-manipulation task across reference schemes (average, linked mastoids, Cz, and CSD). In a
meta-analysis of EEG asymmetry across reference montages, Thibodeau et al. (2006) similarly
found moderate effect sizes (Cohen’s $d = 0.54$) for PFC asymmetry differences between
depressed and healthy control groups. Results suggest that PFC asymmetry during mood
manipulations may more strongly index differences between healthy individuals and those
vulnerable to psychopathology rather than differences in valenced mood states in healthy
individuals alone.

Second, it was predicted that higher trait sadness, or greater depressive symptoms in
healthy young adults would be associated with greater relative right PFC asymmetry, particularly
during the film clip. While there was no direct relationship between depressive symptoms and
PFC asymmetry during the clip, trait sadness proved to be a moderator for the relationship
between PFC asymmetry during and after the clip. Given that depressive symptoms differentially
impacted PFC asymmetry, a non-significant direct relationship between these factors would be
expected. Specifically, lower depressive symptoms and greater left PFC asymmetry during an
emotional mood induction predicted greater post-clip recovery. Similar to the literature
demonstrating that relative right PFC asymmetry is associated with depression, leftward PFC
asymmetry paired with lower depressive symptoms seems to be a marker for resilience to stress and helps individuals recover from emotional events on a neural level.

Third, it was predicted that greater relative right PFC asymmetry during the film clip would predict an increase in the worst depressive symptoms experienced over the following year. While higher baseline depressive symptoms were associated with higher depressive symptoms from the worst month over the past year, PFC asymmetry was not associated with an increase in depressive symptoms over time\(^4\). In line with these results, Blackhart et al. (2006) did not find an association between PFC asymmetry and depressive symptoms one year after EEG recording. However, they found that relative right PFC asymmetry predicted anxiety symptoms at follow-up, suggesting that anxiety symptoms may be a precursor to depressive symptoms given the high co-morbidity rates. Although the current investigation included anxiety symptom analyses, PFC asymmetry did not predict future anxiety, nor was anxiety related to PFC asymmetry during or after the film clip mood induction. Other researchers have found that relative right PFC asymmetry is indeed a risk marker for future depression, but only in women (Stewart & Allen, under review; Smit et al., 2007). The current investigation included additional analyses with only women and no significant effects were seen, likely due to power limitations. Moreover, it has been shown that the percentage of females who participate in EEG asymmetry studies is not related to the magnitude of effects (Thibodeau et al., 2006).

Fourth, it was predicted that trait ER styles would be linked to PFC asymmetry during the film clip, with approach-related styles being associated with leftward asymmetry and withdrawal-related styles being related to rightward asymmetry. Hypotheses were partially supported in that higher ERQ cognitive reappraisal scores predicted greater left PFC asymmetry

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\(^4\) Sample size, sex breakdown, reference montage, and significant effects are listed in Table 7 for all studies predicting first episode depression through PFC asymmetry.
post-clip. Moreover, greater use of ERQ cognitive reappraisal and CERQ adaptive ER strategies moderated the relationship between film clip and post-clip PFC asymmetry, wherein individuals with higher reappraisal/adaptive scores in conjunction with greater left PFC asymmetry during the film clip had the highest left PFC asymmetry post-clip. With regard to EEG change over time, individuals who generally engage in cognitive reappraisal experienced a significant leftward shift in their PFC asymmetry from watching the clip to after the clip, and those individuals even experienced the shift from prior to watching the film clip to after the clip presentation. Meyer et al. (2014) demonstrated that PFC asymmetry was moderated by the extent to which individuals were able to engage in cognitive ER, specifically cognitive reappraisal, after a negative film clip. They found that individuals who were primed with information to reappraise as well as those who viewed a film clip that allowed for reappraisal demonstrated a greater shift towards relative left PFC asymmetry after the mood induction, in two separate samples (Meyer et al., 2014). Of note, the current study observed these effects across groups, in both the happy and sad film clip conditions. Overall, adaptive strategies included in the CERQ and cognitive reappraisal are related and thought to reflect approach-related strategies with which to manage emotional experiences.

No relationships were found between maladaptive, withdrawal-related ER strategies (e.g., suppression) and PFC asymmetry. As mentioned above, other studies that reference the link between ER technique and motivational style (Choi et al., 2016; Keune et al., 2012) did not find significant results with respect to maladaptive strategies and relative right PFC asymmetry. It is possible that adaptive ER strategies are more associated with approach motivation or the behavioral activation system, which results in more robust PFC asymmetry than withdrawal motivation, or behavioral inhibition. Therefore, adaptive strategies may share a stronger
relationship with PFC asymmetry than maladaptive strategies, accounting for the current findings.

It is also worth noting that while adaptive and maladaptive strategies generally correspond to approach and withdrawal processes respectively, additional cognitive components may confound this laterality. For instance, imaging research has demonstrated that deactivation of left hemisphere language regions (e.g., Broca’s and Wernicke’s areas) was associated with mindfulness, an adaptive ER strategy (Farb et al., 2010). As such, neural correlates observed during mindfulness training suggest that additional cognitive processes such as metacognition or the awareness of one’s thought processes, and viewing emotions in a detached, visceral manner may associated with greater activation of the right rather than the left hemisphere even though it is considered to be an adaptive ER strategy. Indeed, researchers have found mixed effects with regard to asymmetry and mindfulness (Keune et al., 2012).

Another example arises with the adaptive ER strategy of reappraisal. Individuals are able to reappraise situations either through reinterpretation of the stimulus or situation (e.g., telling oneself that a sick person in the hospital will recover soon) or distancing him or herself from the stimulus or situation (e.g., telling oneself that the film clip they watched is not real); Ochsner and Gross (2008) have argued that these two aspects of cognitive reappraisal are associated with different brain systems and hemispheres. It is hypothesized that reappraisal through reinterpretation may employ left-lateralized language and verbal working memory systems – in order to construct a new “story” – while distancing may employ right hemisphere and PFC systems involved in attentional control and evaluation of self-relevance (Ochsner & Gross, 2008). Although cognitive reappraisal, as operationalized in the current investigation, was associated with relative left PFC asymmetry, it would be possible for an adaptive strategy to be
linked to relative right PFC asymmetry. Given these factors, it is important to consider additional cognitive processes when conceptualizing ER within the context of EEG asymmetry.

Fifth, it was hypothesized that use of adaptive ER strategies would be linked to decreased depression risk whereas maladaptive indices would be linked to increased risk. Similar to the results discussed in aim 3, no relationships emerged between ER strategies and future depressive symptoms after one year. Several limitations highlighted below may have contributed to the lack of significant results.

**Limitations & Future Directions**

A number of limitations exist in the current study, including the study sample size. Future studies should attempt to increase the number of participants in order to obtain greater power. While comparable study included a similar sample size (Nusslock et al., 2011, n=40, 17 females, age M=20.32, SD=1.25), participants were followed over three years with diagnostic interviews occurring every four months. The longer participants are followed, the greater the likelihood is to observe the onset of a first depressive episode. In fact, the median age of onset for depression is 25 years old (Kessler et al., 2012) and given the age range used in the current study, a longer follow-up period would likely have captured more depressive episodes or an increase in depressive symptoms amongst more participants. In Nusslock et al.’s (2011) sample, 13 of the 40 participants developed a depressive episode (n=3 major depressive episode, n=10 minor depressive episode), while in the current study, only three of the 38 participants developed a minor depressive episode and none developed a major depressive episode. Although there was a considerable range of scores on the worst BDI-II at follow-up, it is possible that with a small

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5Eight months into data collection, the EGI amplifier was recalled to UC San Diego indefinitely; despite aiming for n = 60 controls and n = 60 depressed individuals, study recruitment was terminated given these circumstances.
sample size and limited age range of participants, a longer follow-up period would have been necessary to capture the onset of a first depressive episode.

Second, the difference in length between the film clips (3 minutes) and resting states (5 minutes) may have contributed to the significant difference in epochs between groups. Future researchers may consider using a film clip the same length as the resting states in order to have consistent epochs across conditions. In adults, shorter EEG resting periods (within a range of 1 minute to 8 minutes) have been associated with larger effects (Thibodeau et al., 2006). However, this is not always the case. One study that measured EEG during 8 minutes of a mood induction and 8 minutes of resting EEG obtained significant results (Coan et al., 2003). Moreover, Stewart et al. (2010, 2011b) used an 8-minute resting recording with a 4-minute emotional task induction and obtained significant results. Papousek et al. (2014) used a 10-minute film clip as their method of mood induction and recorded EEG asymmetry during the last 5 minutes of the clip. They found that relative right PFC asymmetry during the clip predicted negative affect over the following week (Papousek et al., 2014). Despite these findings, Towers and Allen (2009) argue that sufficient reliability of PFC asymmetry can be obtained with 1 to 3 minutes of artifact-free recorded data (100 epochs). Therefore, shorter and equal time resting periods can be considered in future research.

As discussed above, there has been much discussion regarding EEG methodology and its impact on PFC asymmetry results. The current study used a linked mastoid reference that was re-referenced from Cz-referenced collected data. Blackhart et al. (2006) have stated that this linked mastoids re-referencing method is the most optimal for capturing robust asymmetry patterns relative to other methods and as such, it has historically been considered the standard montage of choice for these analyses (Hagemann et al., 2001). Despite this assertion, Blackhart et al. (2006)
similarly did not find an association between PFC asymmetry and future depression (n=28, 23 females). Another study observing a large twin sample (n=760) used the re-referenced linked mastoids reference montage and found a significant link between depression and relative right PFC asymmetry; however, prior depressive status was not assessed (Smit et al., 2007). Of the other comparable studies, Nusslock et al. (2011) observed significant effects using a Cz reference grounded at Fz while Pössel et al. (2008) and Mitchell and Pössel (2012) found effects using a nose reference. Stewart and Allen (under review) assessed depression risk using multiple reference schemes including the current source density (CSD) transformation (Tenke & Kayser, 2012), re-referenced linked mastoids, and average montages, reporting that average-referenced, offline linked mastoids-referenced, and CSD-transformed resting PFC asymmetry metrics were all linked to increased depression symptoms in women (n=38) during the worst period they experienced in the following year (Stewart & Allen, under review). Future research should consider using other reference montages, specifically CSD, in addition to a re-referenced linked mastoid reference to compare and contrast effect sizes of these methods.

Another methodological consideration is whether to use pairs of EEG sites rather than the average of multiple sites when measuring asymmetry. More specifically, researchers traditionally report PFC asymmetry results from two to four channel pairs (e.g., F2F1, F4F3, F6F5, and F8F7) in order to obtain more precise effects (e.g., Blackhart et al., 2006; Nusslock et al., 2011; Stewart & Allen, under review). In fact, Thibodeau et al. (2006) found that studies who used data for mid-frontal (F4F3) scalp sites reported larger effect sizes than studies who used composite frontal data, whose mean weighted effect was not significantly different from zero. The current study employed an average of four electrode pairs in order to minimize pairwise comparisons,
thereby reducing type 1 error. Despite this reasoning, a paired sites approach may be more sensitive in capturing PFC activity and can be considered in future research.

Fourth, the current study did not exclude left-handedness in order to retain a sizable sample and as such, three left handed participants were included in analysis. Although similar studies only included right-handed participants (e.g., Blackhart et al., 2006; Nusslock et al., 2011; Pössel et al., 2008; Stewart & Allen, under review), Smit et al. (2007) found that although left-handed individuals tended to show relatively greater right PFC asymmetry than right-handed individuals, the difference was not significant. As a result of this and additional analyses, they conclude that handedness does not appear to be a confound in PFC asymmetry (Smit et al., 2007).

Fifth, the post-resting EEG condition occurred after an inhibition task (the task presentation was identical for both happy and sad groups; results not discussed here); since inhibition generally taps into right hemispheric activity, perhaps this may have impacted post-clip analyses (Sutton & Davidson, 1997). However, findings demonstrate that, despite the potential influence of the inhibition task, individuals endorsing the lowest depressive symptoms and greater use of trait adaptive ER strategies (thought to index approach-related systems and resilience to stress) still show the greatest left frontal asymmetry twenty minutes after the film clip.

Lastly, the current study was unable to consider demographic determinants such as sex or cultural factors as variables of interest given the small sample size. As mentioned, EEG asymmetry differs as a function of sex, wherein relative right PFC asymmetry patterns are typically more robust in women than in men (e.g., Allen, 2015; Stewart et al., 2010). Analyses
were computed using only women in the sample and significant effects did not emerge; however, a greater sample size would be needed to entertain the possibility of observing these trends.

Unlike sex, EEG asymmetry studies have yet to investigate whether these patterns differ as a function of ethnicity or cultural values. Within the ER literature, it has been well documented that individual differences contribute to mixed effects. Various cultures reinforce emotional responding and problem solving in different ways and this in turn influences which ER strategies are employed as well as the consequences associated with implementation of those strategies (Butler, Lee, & Gross, 2007). For instance, ethnic minorities in the U.S. including Asian, Black, and Latino individuals, as well as Americans with self-reported “Asian values” reported higher use of suppression than European Americans and Americans with “Western values” (Butler et al., 2007; Gross & John, 2003). In Asian cultures, primary collectivist values such as interdependence and relationship harmony tend to encourage suppression even in prosocial, positive situations (i.e., hiding joy when winning against an opponent), while the goal of suppression for European or Western Americans is typically for the purpose of self-protection (Butler et al., 2007). Therefore, researchers have found that the use of suppression is associated with less negative social and emotional consequences for Americans holding Asian values more so than Americans holding European and Western values (Butler et al., 2007). Given that our sample was largely comprised of Asian participants and other ethnic minority groups (see Table 8 for a demographic breakdown of the sample), it is possible that effects with suppression were mitigated. As such, it would be valuable to incorporate individual differences in diversity such as culture, ethnicity, and sex as variables of interest in future studies.

Overall, future studies should consider including some or all of the above noted methodological changes including using a larger sample cohort, longer follow-up period,
multiple reference montage recordings, equal time blocks across resting and emotion induction conditions, and consideration of individual differences. Given the significant relationship between ER and PFC asymmetry, it may also be worthwhile to use an experimental design to explore whether individuals’ spontaneous and/or instructed ER approach during an emotional task would be associated with PFC asymmetry.

Conclusions

Overall, these findings highlight the importance of trait sadness and adaptive ER strategies in neural recovery from emotional states which further our understanding of the neural networks implicated in emotion processing seen through asymmetry. Further, the findings could have meaningful impact in depression risk, prevention, and treatment. Emotion dysregulation has been implicated as a central feature in various types of psychopathology, including depression (Aldao et al., 2010; Joormann & Stanton, 2016). ER deficits in depression are characterized by the combination of sustained negative affect and relative lack of positive affect (Joormann & Stanton, 2016). People who experience difficulty regulating their emotional responses to daily stressors may have a persistent experience of distress, which in turn can transition into psychological disorder (Aldao et al., 2010). Moreover, the pattern of relative left PFC asymmetry combined with left PFC asymmetry during an emotional task may serve as a protective factor for recovery from brief emotional states. Studying the relationship between trait sadness, ER, and recovery from a mood induction could shed light on the potential role of these construct in the development of mood pathology and resilience to stress.
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Table 1. Demographics, state emotion, emotion regulation, depressive symptoms, usable electroencephalographic (EEG) epochs, prefrontal cortex (PFC) and parietal asymmetry scores over time (PRE, CLIP, POST) as a function of group membership.

<table>
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<th>Happy (n=16)</th>
<th>Sad (n=22)</th>
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</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>M</td>
<td>SD</td>
<td>M</td>
</tr>
<tr>
<td>Sex</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sex</td>
<td>7 females</td>
<td>16 females</td>
<td>χ² (1)=3.26, p=0.07</td>
</tr>
<tr>
<td>Age</td>
<td>21</td>
<td>1.77</td>
<td>21.00</td>
</tr>
<tr>
<td>PANAS Happy</td>
<td>3.27¹</td>
<td>1.10</td>
<td>1.68</td>
</tr>
<tr>
<td>PANAS Sad</td>
<td>1.13¹</td>
<td>0.52</td>
<td>3.33²</td>
</tr>
<tr>
<td>PANAS Unhappy</td>
<td>1.20¹</td>
<td>0.56</td>
<td>2.41</td>
</tr>
<tr>
<td>PANAS Cheerful</td>
<td>3.40¹</td>
<td>1.24</td>
<td>1.45</td>
</tr>
<tr>
<td>ERQ Suppression</td>
<td>16.47¹</td>
<td>4.72</td>
<td>14.45</td>
</tr>
<tr>
<td>ERQ Cognitive Reappraisal</td>
<td>29.40¹</td>
<td>6.53</td>
<td>32.32</td>
</tr>
<tr>
<td>CERQ Adaptive</td>
<td>36.36³</td>
<td>6.13</td>
<td>37.90²</td>
</tr>
<tr>
<td>CERQ Maladaptive</td>
<td>21.71³</td>
<td>5.27</td>
<td>19.52²</td>
</tr>
<tr>
<td>BDI-II Baseline</td>
<td>7.00¹</td>
<td>9.20</td>
<td>6.14</td>
</tr>
<tr>
<td></td>
<td>(log=1.59)</td>
<td>(log=1.03)</td>
<td>(log=1.59)</td>
</tr>
<tr>
<td>----------------------</td>
<td>------------</td>
<td>------------</td>
<td>------------</td>
</tr>
<tr>
<td>BDI-II Follow-up</td>
<td>11.08^4</td>
<td>15.46</td>
<td>4.74^5</td>
</tr>
<tr>
<td>Current</td>
<td>(log=1.66)</td>
<td>(log=1.41)</td>
<td>(log=1.44)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BDI-II Follow-up Worst</td>
<td>12.43^3</td>
<td>14.52</td>
<td>9.43^2</td>
</tr>
<tr>
<td></td>
<td>(log=1.91)</td>
<td>(log=1.35)</td>
<td>(log=1.87)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MASQ Anxious Arousal</td>
<td>25.93^1</td>
<td>8.85</td>
<td>22.05</td>
</tr>
<tr>
<td>Baseline</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MASQ Anxious Arousal</td>
<td>22.21^3</td>
<td>5.96</td>
<td>23.24^2</td>
</tr>
<tr>
<td>Follow-up</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PSWQ Baseline</td>
<td>48.00^6</td>
<td>11.49</td>
<td>51.71^7</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PSWQ Follow-up</td>
<td>47.29^3</td>
<td>16.16</td>
<td>48.10^2</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>EEG Epochs PRE</td>
<td>401.06</td>
<td>121.20</td>
<td>424.62^2</td>
</tr>
<tr>
<td></td>
<td>Group: F(1, 35)= 0.01, p=0.93, η²= 0.00</td>
<td></td>
<td></td>
</tr>
<tr>
<td>EEG Epochs CLIP</td>
<td>222.13</td>
<td>73.53</td>
<td>248.9^2</td>
</tr>
<tr>
<td></td>
<td>Condition: F(1.77, 61.99)= 97.7, p&lt;0.01, η²= 0.74</td>
<td></td>
<td></td>
</tr>
<tr>
<td>EEG Epochs POST</td>
<td>431.06</td>
<td>100.66</td>
<td>388.67^2</td>
</tr>
<tr>
<td></td>
<td>Group x Condition: F(1.77, 61.99)= 3.61, p=0.04, η²= 0.09</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>PRE</td>
<td>CLIP</td>
<td>POST</td>
</tr>
<tr>
<td>--------------------------</td>
<td>------</td>
<td>------</td>
<td>-------</td>
</tr>
<tr>
<td>PFC Asymmetry</td>
<td>0.02</td>
<td>0.04</td>
<td>0.02</td>
</tr>
<tr>
<td></td>
<td>0.17</td>
<td>0.17</td>
<td>0.17</td>
</tr>
<tr>
<td></td>
<td>0.03</td>
<td>0.10</td>
<td>0.09</td>
</tr>
<tr>
<td></td>
<td>0.33</td>
<td>0.31</td>
<td>0.32</td>
</tr>
<tr>
<td>Parietal Asymmetry</td>
<td>-0.08</td>
<td>-0.11</td>
<td>-0.11</td>
</tr>
<tr>
<td></td>
<td>0.23</td>
<td>0.20</td>
<td>0.38</td>
</tr>
<tr>
<td></td>
<td>0.02²</td>
<td>-0.13²</td>
<td>0.05²</td>
</tr>
<tr>
<td></td>
<td>0.51</td>
<td>0.54</td>
<td>0.62</td>
</tr>
<tr>
<td>Family history count of</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Depression (first-degree</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>relative)</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

n = 4
n = 5

Note: ¹ n=15. ² n=21. ³ n=14. ⁴ n=12. ⁵ n=19. ⁶ n=10. ⁷ n=7. Assumptions of sphericity were met for all repeated measures ANOVAs except Epoch clip and post; Huynh-Feldt corrections were reported for all sphericity assumptions violated. The homogeneity of variance assumption was met for all scales except independent t-tests involving PANAS Cheerful, Sad, and Unhappy, BDI-II Follow-up Current, MASQ Baseline, and Parietal asymmetry Pre and Clip; a correction was used for unequal variances. A log transformation was computed for BDI-II Baseline, Follow-up Worst, and Follow-up Current to approximate normality. Raw and log transformed data
were presented. T-tests were computed using log transformed data. Groups did not differ on any questionnaire ERQ = Emotion Regulation Questionnaire. CERQ = Cognitive Emotion Regulation Questionnaire. CERQ Adaptive subscales include positive refocusing, positive reappraisal, putting into perspective, refocus on planning, and assessment. CERQ Maladaptive subscales include rumination, self-blame, blaming others, and catastrophizing. BDI-II = Beck Depression Inventory-II.
Table 2. Moderation analyses with depression scores, prefrontal cortex (PFC) asymmetry, and their interaction predicting post-clip PFC asymmetry.

<table>
<thead>
<tr>
<th>Predictor</th>
<th>$\beta$</th>
<th>$t$</th>
<th>$p$</th>
<th>$R^2$ Model</th>
<th>ANOVA Model</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baseline BDI-II</td>
<td>-0.18</td>
<td>-1.82</td>
<td>0.08</td>
<td>0.72</td>
<td>$F(3,33)=27.61, p&lt;0.001$</td>
</tr>
<tr>
<td>Clip PFC Asymmetry</td>
<td>0.75</td>
<td>7.58</td>
<td>&lt;0.001</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Interaction</td>
<td>-0.22</td>
<td>-2.10</td>
<td>0.04</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Note. BDI-II = Beck Depression Inventory
Table 3. Moderation analyses with baseline depressive symptoms, prefrontal cortex (PFC) asymmetry, and their interaction predicting worst BDI-II at follow-up.

<table>
<thead>
<tr>
<th>Predictor</th>
<th>$\beta$</th>
<th>$t$</th>
<th>$p$</th>
<th>$R^2$ Model</th>
<th>ANOVA Model</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baseline BDI-II</td>
<td>0.60</td>
<td>3.97</td>
<td>&lt;0.001</td>
<td>0.42</td>
<td>$F(3,29)=7.00,$</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>$p&lt;0.01$</td>
</tr>
<tr>
<td>Clip PFC Asymmetry</td>
<td>-0.06</td>
<td>-0.40</td>
<td>0.70</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Interaction</td>
<td>-0.11</td>
<td>-0.69</td>
<td>0.50</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Note. BDI-II = Beck Depression Inventory
Table 4a. Correlations between emotion regulation indices and prefrontal cortex (PFC) asymmetry during the film clip within and across groups.

<table>
<thead>
<tr>
<th></th>
<th>Clip PFC Asymmetry Score</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>All (n=38)</td>
</tr>
<tr>
<td>ERQ Cognitive Reappraisal</td>
<td>0.01</td>
</tr>
<tr>
<td></td>
<td>(n=37)</td>
</tr>
<tr>
<td>ERQ Suppression</td>
<td>0.07</td>
</tr>
<tr>
<td></td>
<td>(n=37)</td>
</tr>
<tr>
<td>CERQ Adaptive</td>
<td>-0.02</td>
</tr>
<tr>
<td></td>
<td>(n=35)</td>
</tr>
<tr>
<td>CERQ Maladaptive</td>
<td>0.03</td>
</tr>
<tr>
<td></td>
<td>(n=35)</td>
</tr>
</tbody>
</table>

Note. ERQ = Emotion Regulation Questionnaire. CERQ = Cognitive Emotion Regulation Questionnaire. No correlation results were significant (p<0.05).
Table 4b. Moderation analyses with emotion regulation, prefrontal cortex (PFC) asymmetry, and their interaction predicting post-clip PFC asymmetry.

<table>
<thead>
<tr>
<th>Moderation Analysis</th>
<th>Predictor</th>
<th>β</th>
<th>t</th>
<th>p</th>
<th>R² Model</th>
<th>ANOVA Model</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>ERQ Cognitive Reappraisal</td>
<td>0.32</td>
<td>3.80</td>
<td>0.001</td>
<td>0.78</td>
<td>F(3,33)=38.75, p&lt;0.001</td>
</tr>
<tr>
<td></td>
<td>Clip PFC Asymmetry</td>
<td>0.77</td>
<td>9.11</td>
<td>&lt;0.001</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Interaction</td>
<td>0.21</td>
<td>2.43</td>
<td>0.02</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>ERQ Suppression</td>
<td>0.05</td>
<td>0.52</td>
<td>0.61</td>
<td>0.70</td>
<td>F(3,33)=25.34, p&lt;0.001</td>
</tr>
<tr>
<td></td>
<td>Clip PFC Asymmetry</td>
<td>0.81</td>
<td>8.41</td>
<td>&lt;0.001</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Interaction</td>
<td>-0.17</td>
<td>-1.74</td>
<td>0.09</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>CERQ Adaptive</td>
<td>0.13</td>
<td>1.40</td>
<td>0.17</td>
<td>0.76</td>
<td>F(3,31)=32.09, p&lt;0.001</td>
</tr>
<tr>
<td></td>
<td>Clip PFC Asymmetry</td>
<td>0.71</td>
<td>7.11</td>
<td>&lt;0.001</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Interaction</td>
<td>0.25</td>
<td>2.51</td>
<td>&lt;0.05</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>CERQ Maladaptive</td>
<td>-0.05</td>
<td>-0.52</td>
<td>0.61</td>
<td>0.75</td>
<td>F(3,31)=30.40, p&lt;0.001</td>
</tr>
<tr>
<td></td>
<td>Clip PFC Asymmetry</td>
<td>0.81</td>
<td>8.89</td>
<td>&lt;0.001</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Interaction</td>
<td>-0.26</td>
<td>-2.70</td>
<td>0.11</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Note. ERQ = Emotion Regulation Questionnaire. CERQ = Cognitive Emotion Regulation Questionnaire
Table 5. Moderation analyses with baseline BDI-II, emotion regulation, and their interaction predicting worst BDI-II at follow-up.

<table>
<thead>
<tr>
<th>Moderation Analysis</th>
<th>Predictor</th>
<th>β</th>
<th>t</th>
<th>p</th>
<th>R² Model</th>
<th>ANOVA Model</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>ERQ Cognitive Reappraisal</td>
<td>0.04</td>
<td>0.28</td>
<td>0.79</td>
<td>0.42</td>
<td>F(3,29)=7.00, p&lt;0.01</td>
</tr>
<tr>
<td></td>
<td>Baseline BDI-II</td>
<td>0.66</td>
<td>4.38</td>
<td>&lt;0.001</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Interaction</td>
<td>0.08</td>
<td>0.56</td>
<td>0.58</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>ERQ Suppression</td>
<td>-0.15</td>
<td>-1.08</td>
<td>0.29</td>
<td>0.48</td>
<td>F(3,29)=9.07, p&lt;0.001</td>
</tr>
<tr>
<td></td>
<td>Baseline BDI-II</td>
<td>0.71</td>
<td>5.07</td>
<td>&lt;0.001</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Interaction</td>
<td>0.26</td>
<td>1.89</td>
<td>0.07</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>CERQ Adaptive</td>
<td>0.31</td>
<td>2.08</td>
<td>0.05</td>
<td>0.49</td>
<td>F(3,28)=8.81, p&lt;0.001</td>
</tr>
<tr>
<td></td>
<td>Baseline BDI-II</td>
<td>0.72</td>
<td>4.82</td>
<td>&lt;0.001</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Interaction</td>
<td>0.01</td>
<td>0.04</td>
<td>0.97</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>CERQ Maladaptive</td>
<td>0.31</td>
<td>1.83</td>
<td>0.08</td>
<td>0.47</td>
<td>F(3,28)=8.24, p&lt;0.001</td>
</tr>
<tr>
<td></td>
<td>Baseline BDI-II</td>
<td>0.56</td>
<td>3.89</td>
<td>&lt;0.01</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Interaction</td>
<td>-0.06</td>
<td>-0.39</td>
<td>0.70</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Note. ERQ = Emotion Regulation Questionnaire. CERQ = Cognitive Emotion Regulation Questionnaire. BDI-II = Beck Depression Inventory.
Table 6. Correlations between prefrontal cortex (PFC) asymmetry difference scores, Worst BDI-II at follow-up and emotion regulation indices.

<table>
<thead>
<tr>
<th></th>
<th>Worst BDI-II</th>
<th>ERQ Suppression</th>
<th>ERQ Cognitive Reappraisal</th>
<th>CERQ Adaptive</th>
<th>CERQ Maladaptive</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pre- to Post- PFC</td>
<td>0.05</td>
<td>0.02</td>
<td>-0.41*</td>
<td>0.05</td>
<td>0.20</td>
</tr>
<tr>
<td>Asymmetry Difference Score</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Clip- to Post- PFC</td>
<td>&lt;0.001</td>
<td>-0.11</td>
<td>-0.46**</td>
<td>-0.26</td>
<td>-0.03</td>
</tr>
<tr>
<td>Asymmetry Difference Score</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Note. BDI-II = Beck Depression Inventory.

**correlation is significant at the 0.01 level

*correlation is significant at the 0.05 level
Table 7.

<table>
<thead>
<tr>
<th>Article</th>
<th>Sample Size (sex)</th>
<th>Age Mean (SD)</th>
<th>Reference Montage</th>
<th>Follow-up Duration</th>
<th>Asymmetry Effects with Depression</th>
<th>Asymmetry Effects with Anxiety</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blackhart et al. (2006)</td>
<td>n=28 (23 females)</td>
<td>18.78 (1.02)</td>
<td>Linked mastoid</td>
<td>One year</td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td>Mitchell &amp; Pössel (2012)</td>
<td>n=41 (41 males)</td>
<td>13.93 (0.61)</td>
<td>Nose reference</td>
<td>One year</td>
<td>Yes</td>
<td>Not assessed</td>
</tr>
<tr>
<td>Nusslock et al. (2011)</td>
<td>n=40 (23 males)</td>
<td>20.32 (1.25)</td>
<td>Cz (grounded at Fz)</td>
<td>Three years</td>
<td>Yes</td>
<td>Not assessed</td>
</tr>
<tr>
<td>Pössel et al. (2008)</td>
<td>n=80 (45 males)</td>
<td>13.92 (0.57)</td>
<td>Nose reference</td>
<td>One year</td>
<td>Yes</td>
<td>Not assessed</td>
</tr>
<tr>
<td>Stewart &amp; Allen (under review)</td>
<td>n=54 (38 females)</td>
<td>18.78 (0.77)</td>
<td>CSD</td>
<td>One year</td>
<td>Yes</td>
<td>Not assessed</td>
</tr>
</tbody>
</table>
Table 8. Demographic descriptive statistics of the sample.

<table>
<thead>
<tr>
<th>Race/Ethnicity</th>
<th>Frequency (n=38)</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Asian</td>
<td>12</td>
<td>32</td>
</tr>
<tr>
<td>African American/Black</td>
<td>7</td>
<td>18</td>
</tr>
<tr>
<td>Latino/a</td>
<td>6</td>
<td>16</td>
</tr>
<tr>
<td>Mixed/Other</td>
<td>2</td>
<td>5</td>
</tr>
<tr>
<td>Pacific Islander</td>
<td>1</td>
<td>3</td>
</tr>
<tr>
<td>White</td>
<td>10</td>
<td>26</td>
</tr>
</tbody>
</table>
**Figure Captions**

Figure 1. Frontal electrodes highlighted in grey used for computation of asymmetry scores used in statistical analysis.

Figure 2. PFC asymmetry as a function of group (sad, happy) and time (pre-clip, film clip, post-clip). Positive scores indicate greater left than right PFC activity whereas negative scores indicate greater right than left PFC activity. Error bars reflect ± 1 standard error.

Figure 3. Parietal asymmetry as a function of group (sad, happy) and time (pre-clip, film clip, post-clip). Positive scores indicate greater left than right parietal activity whereas negative scores indicate greater right than left parietal activity. Error bars reflect ± 1 standard error.

Figure 4. Baseline Beck Depression Inventory-II (BDI-II), PFC film clip asymmetry, and their interaction predicting PFC post-clip asymmetry. Trait depression moderated the effect of film clip asymmetry on post-clip asymmetry, such that participants with lower depression scores and greater left PFC asymmetry during the clip also exhibited greater left PFC asymmetry post-clip.

Figure 5. Baseline BDI-II, PFC film clip asymmetry, and their interaction predicting worst BDI-II at follow-up. Although higher baseline BDI-II scores were linked to higher worst BDI-II scores at follow-up, a significant PFC asymmetry moderation effect did not emerge.

Figure 6a. Emotion Regulation Questionnaire (ERQ) cognitive reappraisal, PFC film clip asymmetry, and their interaction predicting PFC post-clip asymmetry. ERQ cognitive reappraisal moderated the effect of film clip asymmetry on post-clip asymmetry, such that participants with higher reappraisal scores and greater left PFC asymmetry during the clip also had greater left PFC asymmetry post-clip.
Figure 6b. Although greater clip PFC asymmetry was associated with greater post-clip PFC asymmetry, a significant moderation effect did not emerge for Emotion Regulation Questionnaire (ERQ) expressive suppression.

Figure 6c. CERQ adaptive subscales, PFC film clip asymmetry, and their interaction predicting PFC post-clip asymmetry. CERQ adaptive subscales moderated the effect of film clip asymmetry on post-clip asymmetry, such that participants with higher adaptive ER scores and greater left PFC asymmetry during the clip also had greater left PFC asymmetry post-clip.

Figure 6d. Although greater clip PFC asymmetry was associated with greater post-clip PFC asymmetry, a significant moderation effect did not emerge for the CERQ maladaptive subscales.

Figure 7a. ERQ cognitive reappraisal, baseline BDI-II, and their interaction predicting worst BDI-II at follow-up. Although higher baseline BDI-II scores were associated with higher worst BDI-II scores at follow-up, ERQ cognitive reappraisal did not moderate this relationship. Figure 7b. Although higher baseline BDI-II scores were associated with higher worst BDI-II scores at follow-up, ERQ suppression did not moderate this relationship.

Figure 7c. Although higher baseline BDI-II scores were associated with higher worst BDI-II scores at follow-up, CERQ adaptive subscales did not moderate this relationship.

Figure 7d. Although higher baseline BDI-II scores were associated with higher worst BDI-II scores at follow-up, CERQ maladaptive subscales did not moderate this relationship.

Figure 8a. There was a significant correlation between the Clip-to-Post EEG change score and ERQ cognitive reappraisal.

Figure 8b. There was a significant correlation between the Pre-to-Post EEG change score and ERQ cognitive reappraisal.
Figure 1.
Figure 2.

![Graph showing PFC asymmetry for SAD (n=22) and HAPPY (n=16) groups pre, film, and post.](image-url)
Figure 3.
Figure 4.
Figure 5.
Figure 6.

Figure 6a.

Figure 6b.

Figure 6c.

Figure 6d.
Figure 7.

Figure 7a. [Graphs showing the relationship between worst BDI at follow-up and baseline BDI for low and high ERQ reappraisal.

Figure 7b. [Graphs showing the relationship between worst BDI at follow-up and baseline BDI for low and high ERQ suppression.

Figure 7c. [Graphs showing the relationship between worst BDI at follow-up and baseline BDI for low and high CERQ adaptive.

Figure 7d. [Graphs showing the relationship between worst BDI at follow-up and baseline BDI for low and high CERQ maladaptive.]
Figure 8.

Figure 8a.

Figure 8b.