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Frontal alpha asymmetry neurofeedback for the reduction of negative affect and anxiety

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ABSTRACT

Frontal alpha asymmetry has been proposed to underlie the balance between approach and withdrawal motivation associated to each individual's affective style. Neurofeedback of EEG frontal alpha asymmetry represents a promising tool to reduce negative affect, although its specific effects on left/right frontal activity and approach/withdrawal motivation are still unclear. The present study employed a neurofeedback training to increase frontal alpha asymmetry (right - left), in order to evaluate discrete changes in alpha power at left and right sites, as well as in positive and negative affect, anxiety and depression. Thirty-two right-handed females were randomly assigned to receive either the neurofeedback on frontal alpha asymmetry, or an active control training (N = 16 in each group). The asymmetry group showed an increase in alpha asymmetry driven by higher alpha at the right site ($p < 0.001$), as well as a coherent reduction in both negative affect and anxiety symptoms ($ps < 0.05$), from pre-to post-training. No training-specific modulation emerged for positive affect and depressive symptoms. These findings provide a strong rationale for the use of frontal alpha asymmetry neurofeedback for the reduction of negative affect and anxiety in clinical settings.

1. Introduction

Several research lines converge in indicating the existence of two fundamental motivational systems in the mammalian brain (Davidson, 1992; Dickinson & Dearing, 1979; Gray & McNaughton, 1982; Konorski, 1967; Lang & Bradley, 2013; Lang, 2010; Lang, Bradley, & Cuthbert, 1997): 1) an *appetitive/approach* system, that drives approach behaviors toward rewarding stimuli, and 2) a *defensive/withdrawal* system, associated with avoidance from aversive stimuli. The balance between appetitive and defensive dispositions has been referred to as the *affective style* of the individual (Davidson, 1998a, 1998b, 2004; Davidson, Jackson, & Kalin, 2000), which prompts responses to emotional stimuli, dispositional mood and vulnerability to psychopathology. On one hand, the motivational systems are embedded in evolutionary ancient brain structures which subserve similar functions in humans and in other mammals. On the other hand, humans further developed control over primary motivational impulses, mostly subtended by the prefrontal cortex (Damasio & Carvalho, 2013; Damasio et al., 2000; Ochsner & Gross, 2005).

In this sense, the dorsolateral prefrontal cortex acts as a moderator of the primary motivational and emotional responses, through its anatomical and functional connections with core limbic structures such as the amygdala, the basal ganglia, the anterior cingulate and the orbitofrontal cortex (Spielberg et al., 2012). Furthermore, an hemispherical specialization for the prefrontal cortex in motivation and affect has been reported: prevalent activity in the right compared to the left prefrontal areas has been related to withdrawal behaviors and to the experience of negative emotions, while the opposite pattern (i.e., greater left vs. right activity) accompanies approach behaviors and positive affect (Davidson, 1988, 1998b; Harmon-Jones, Gable, & Peterson, 2010; Papousek, Reiser, Weber, Freudenthaler, & Schulter, 2012; Papousek et al., 2014). Accordingly, it has been advocated that 1) the left prefrontal cortex has a role in organizing limited resources toward goal-oriented behaviors, sustaining approach and positive affect; 2) the right prefrontal cortex mediates vigilance for threat and sensitivity to punishment, thus promoting avoidance and withdrawal (Gray & McNaughton, 1982; Sutton & Davidson, 1997). Therefore, the differential activity between the right and left prefrontal lobes is commonly considered a measure of affective style (Coan & Allen, 2004; Davidson, 1992).

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Since electroencephalographic (EEG) alpha power is an inverse index of cortical activity (Cook, O'Hara, Uijtdehaage, Mandelkern, & Leuchter, 1998; Davidson, Chapman, Chapman, & Henriques, 1990), the asymmetry in frontal alpha power is thought to reflect the balance between the activity of the right and left prefrontal lobes (Allen, Coan, & Nazarian, 2004). Accordingly, reduced alpha at right compared to left frontal sites (i.e., greater right-sided activity) has been associated with withdrawal motivation (Sutton & Davidson, 1997), negative affect (Jacobs & Snyder, 1996; Schaffer, Davidson, & Saron, 1983; Tomarken, Davidson, Wheeler, & Doss, 1992), as well as reports of more intense negative emotions after unpleasant film viewing (Papousek et al., 2014; Wheeler, Davidson, & Tomarken, 1993). On the other hand, reduced alpha at left compared to right frontal sites (i.e., greater left-sided activity) has been related to approach motivation (Harmon-Jones & Allen, 1998, 1997; Sutton & Davidson, 1997), trait positive affect (Tomarken et al., 1992), rates of pleasantness after positive film viewing (Wheeler et al., 1993), reward responsiveness (De Pascalis, Varriale, & D'Antuono, 2010), dispositional optimism (De Pascalis, Cozzuto, Caprara, & Alessandri, 2013), greater emotional flexibility (Papousek et al., 2012) and better emotional regulation (Jackson et al., 2003).

Electroencephalographic (EEG) biofeedback (*neurofeedback*) has been proposed as a tool to modulate the hemispherical asymmetry in prefrontal activity, in order to regulate affect (Rosenfeld, Cha, Blair, & Gotlib, 1995). More in general, biofeedback is a bio-behavioral technique which aims at modifying physiological activity and, in turn, improving health and/or performance. In line with the biofeedback principles, neurofeedback relies on the assumption that, by providing real-time information on brain's activity, individuals can extend their conscious control and learn how to regulate their own brain activity (Thibault, Lifshitz, & Raz, 2016). Operant conditioning is a possible mechanism of action, since the positive feedback can be conceived as a reinforcement for the desired bio-behavioral pattern (Miller & DiCara, 1967). As a preliminary step, neurofeedback studies tested whether or not it was possible to modulate frontal alpha asymmetry, as well as the effects of this modulation on reported affect. Through five sessions of frontal alpha asymmetry neurofeedback using rewarding and non-rewarding tones, Allen, Harmon-Jones, and Cavender (2001) trained two groups of healthy participants either to increase or decrease their alpha asymmetry score, computed by subtracting left (F3) from right (F4) alpha power; therefore higher scores corresponded to reduced right compared to left frontal activity. Only participants trained to decrease alpha asymmetry succeeded and neither training modified current affect, as measured by the Positive and Negative Affect Scale, state version (PANAS; Watson, Clark, & Tellegen, 1988). Recently, one comprehensive study on a sample of 60 individuals replicated Allen and colleagues' results, providing a visual feedback and adding a random-feedback control group (Quaedflieg et al., 2015); accordingly, this study showed that after 6 sessions participants were able to increase left compared to right alpha at frontal sites, but not vice versa. Further studies supported the efficacy of neurofeedback for the modulation of frontal alpha asymmetry in both directions (i.e., increasing or decreasing the asymmetry score) after three sessions of acoustic neurofeedback, with a similar design as the one from Allen and colleagues (Harmon-Jones, Harmon-Jones, Fearn, Sigelman, & Johnson, 2008), or even after one session of visual feedback (Peeters, Ronner, Bodar, van Os, & Lousberg, 2014). Again, neither positive nor negative affect were influenced by the training.

Although there is evidence for neurofeedback to be effective in modulating frontal alpha asymmetry, previous studies did not address whether observed modifications were mostly driven by changes in alpha power at right, left, or both frontal sites. From a clinical perspective, this is of particular relevance, given that similarly altered patterns of frontal alpha asymmetry have been found in different conditions characterized by affect dysregulation, such as anxiety and depression (Beaton et al., 2008; Mennella, Messerotti Benvenuti, Buodo, & Palomba, 2015; Moscovitch et al., 2011; Stewart, Coan, Towers, & Allen, 2011, 2014). In particular, even though both anxiety and depressive symptoms have been associated with reduced frontal alpha asymmetry (right - left), in anxiety this is subtended by a dominance in withdrawal motivation and negative affect (increased right compared to left frontal activity); on the other hand, in depression the asymmetry dysregulation has been related to a reduction in approach motivation and positive affect (reduced left compared to right frontal activity) (Davidson, 1998a; Shankman & Klein, 2003). For this reason, it seems crucial establishing the specific effect of frontal alpha asymmetry neurofeedback on left and right activity, in order to provide a strong rationale for its clinical application.

The present study evaluated the effectiveness of frontal alpha asymmetry neurofeedback in *increasing* the alpha asymmetry index (F4 - F3). Furthermore, it was tested whether variations in asymmetry are subtended by relative changes in left (F3) and/or right (F4) frontal alpha power. Finally, the effects of the training on affect were assessed using a comprehensive battery, including the PANAS, for the evaluation of both positive and negative affect, as well as measures of anxiety and depressive symptoms.

It was hypothesized that 1) the frontal alpha asymmetry training would be effective in increasing alpha asymmetry (F4 - F3); 2) in case higher alpha asymmetry was subtended by an increase in alpha power at the right site (less dominant right-sided activity) this would be associated with a decrease in negative affect and anxiety scores; 3) if higher alpha asymmetry was driven by decreased alpha power at the left site (more prominent left-sided activity), higher positive affect scores and a reduction in depressive symptoms were expected.

2. Methods

2.1. Participants

Thirty-two healthy and free from medication undergraduate students (M age = 23.1, SD = 1.2) from the University of Padova were enrolled. The present study included only right-handed females, since asymmetrical alpha activity is influenced by handedness (Davidson, 1988), and previous studies have primarily examined female participants (Allen et al., 2001; Harmon-Jones et al., 2008; Peeters, Ronner, et al., 2014). Exclusion criteria were: previous head injury, chronic mental or neurological diseases and treatment with medications known to influence EEG, such as tranquilizers or antidepressants. Participants were randomly assigned to receive a biofeedback training designed either to increase frontal alpha asymmetry (i.e., F4 - F3; *asymmetry group*; N = 16), or to increase mid-frontal (Fz) alpha activity (*active control*; N = 16). Neurofeedback aimed at increasing alpha activity has been previously employed in order to reduce stress and anxious symptoms (Brown, 1970; Hammond, 2005; Hardt & Kamiya, 1978), due to the positive association of alpha power with states of relaxation and low arousal. Usually, this training is carried out at posterior sites, since the alpha rhythm is predominant in the occipital and parietal regions. On the contrary, in the present study the training targeted the mid-frontal site, to serve as a specific control condition for the frontal alpha asymmetry neurofeedback.

The groups were comparable with respect to sociodemographic variables (Table 1). Participants were told that at the end of the seven sessions they would receive a monetary payment proportional to their performance during training sessions (total payment