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Fearless Dominance and reduced feedback-related negativity amplitudes in a time-estimation task – Further neuroscientific evidence for dual-process models of psychopathy☆

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ABSTRACT

Dual-process models of psychopathy postulate two etiologically relevant processes. Their involvement in feedback processing and its neural correlates has not been investigated so far. Multi-channel EEG was collected while healthy female volunteers performed a time-estimation task and received negative or positive feedback in form of signs or emotional faces. The affective-interpersonal factor Fearless Dominance, but not Self-Centered Impulsivity, was associated with reduced feedback-related negativity (FRN) amplitudes. This neural dissociation extends previous findings on the impact of psychopathy on feedback processing and further highlights the importance of distinguishing psychopathic traits and extending previous (neuroscientific) models of psychopathy.

1. Introduction

Psychopathy is a construct characterized by a number of deficits in adaptation and affective processing – lack of empathy, fearlessness, deficits in aversive and passive avoidance learning, and antisocial behavior among others (Cleckley, 1941; Hare, 2003; Hare & Neumann, 2008). Although primarily studied in offenders, there is a growing number of investigations in the general population, as psychopathy is not restricted to incarcerated offenders (Hall & Benning, 2006) but rather considered as a construct with a dimensional latent structure and not representing a qualitatively discrete group (Edens, Marcus, Lilienfeld, & Poythress, 2006; Marcus, John, & Edens, 2004). Moreover, this also indicates more than one causal factor in the etiology of psychopathy.

1.1. Dual-process models of psychopathy

Dual-process models (e.g., Fowles & Dindo, 2009; Patrick & Bernat, 2009) relate two potential etiological dimensions to the higher order factors of frequently applied psychometric instruments in the assessment of psychopathy in offenders, e.g. the PCL-R (*Psychopathic Checklist-Revised*; Hare, 2003) or in the general population, e.g. the PPI-R (*Psychopathy Personality Inventory-Revised*; Alpers & Eisenbarth, 2008; Lilienfeld & Andrews, 1996). The first model dimension ("Trait Fearlessness" in the model of Patrick & Bernat, 2009) focuses on emotional-interpersonal aspects and is related to an arrogant interpersonal style, lack of empathy and reduced fear reactivity. The second model dimension ("Externalizing Vulnerability", Patrick & Bernat, 2009) is associated with an impulsive, socially deviant lifestyle. In the PPI-R, they are psychometrically operationalized in form of the higher-order factors

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Fearless Dominance and Self-Centered Impulsivity, respectively. Both dimensions of psychopathic personality are thought to reflect etiologic pathways that can be already found in childhood psychopathology (Fowles & Dindo, 2009). The label "Externalizing Vulnerability" emphasizes the link to externalizing psychopathology (Patrick, Hicks, Krueger, & Lang, 2005) – one of two broad factors underlying the most common mental disorders, in particular the one associated with conduct disorder, antisocial behavior, alcohol and drug abuse among others (Krueger, 1999). However, psychopathy cannot be sufficiently described by externalizing psychopathology because the latter was unrelated to the unique variance of the emotional-interpersonal dimension of psychopathy (Patrick et al., 2005).

A dual-process perspective might allow new insights in the (neurocognitive) mechanisms underlying these pathways to psychopathic personality and the core deficits of psychopathy such as deficits in behavioral adaptation or passive avoidance learning (Newman & Kosson, 1986; Newman, Patterson, Howland, & Nichols, 1990). Dinn and Harris (2000) suggested that behavioral adaptation deficits found in ASPD (antisocial personality disorder) individuals with psychopathic traits might be related to inadequate processing of feedback information. Previous studies already reported neurocognitive dissociations between the two dimensions of psychopathy, for instance in affect recognition (Gordon, Baird, & End, 2004) or executive functions such as attention and inhibition (Carlson & Thái, 2010; Carlson, Thái, & McLarnon, 2009). The aim of our study was to investigate now feedback processing – another potentially relevant neurocognitive mechanism – from a dual-process perspective of psychopathy.

1.2. Feedback processing and psychopathy

A brain structure that has been associated with feedback processing is the dorsal anterior cingulate cortex (dACC; Holroyd & Coles, 2002; Holroyd, Pakzad-Vaezi, & Krigolson, 2008; Miltner, Braun, & Coles, 1997; Ullsperger & Von Cramon, 2003). It is an area supposed to be fundamental to response-reinforcement associations (Rushworth, Behrens, Rudebeck, & Walton, 2007), behavioral monitoring and adaptation (e.g. Holroyd & Coles, 2002), and therefore a plausible candidate for explaining behavioral adaptation deficits in psychopathy.

Electrophysiologically, external feedback after the occurrence of an error elicits a negative event-related potential (ERP) called feedback-related negativity (FRN) with a typical peak amplitude within 200–300 ms. Behaviorally, the FRN was shown to be associated with the degree of learning from negative feedback in an emotion recognition task and a probabilistic learning task (Frank, D'Lauro, & Curran, 2007; Frank, Woroch, & Curran, 2005).

Reinforcement Learning Theory (RLT; Baker & Holroyd, 2009, 2011; Holroyd & Coles, 2002) suggests that reward-prediction error signals are transmitted via the mesencephalic dopamine system to the dACC eliciting the FRN. As the FRN is sensitive to the unpredictability of the outcome, its amplitude becomes smaller in the course of learning the specific action-outcome association, enabling a switch from external (i.e. via external feedback information) to internal error monitoring (i.e. comparing actual and intended behavior) indexed by a functional related component called error-related negativity (ERN), peaking earlier than the FRN, about 100 ms after erroneous response (Falkenstein, Hohnsbein, Hoormann, & Blanke, 1991; Gehring, Gross, Coles, Meyer, & Donchin, 1993; Holroyd & Coles, 2002). This is called backward propagation after learning (Holroyd & Coles, 2002). In particular, the rostral cingulate zone anterior (RCZa), which is part of the dACC, is sensitive to both forms of error monitoring and also reflects these learning-dependent dynamics (Mars et al., 2005). However, van der Veen, Röde, Mies, van der Lugt, & Smits, (2011) proposed rather an involvement of the RCZ in remedial action than a signaling function as stated in the RLT.

Another ERP repeatedly investigated during feedback processing is the P3(b) component, peaking between 200 and 600 ms at posterior electrode sites (Yeung & Sanfey, 2004). This classical P3 component seems to index the task relevance of a stimulus (Coles, Smid, Scheffers, & Otten, 1995) and resource allocation (Israel, Chesney, Wickens, & Donchin, 1980; Kahneman, 1973). One influential theory links the classical P3 with context-updating of working memory, i.e. revisions of mental representations by stimuli classified as new after comparison with previous stimuli (Donchin & Coles, 1988, 1998; Polich, 2007).

These ERPs in error monitoring have been associated with several personality traits in previous studies, for instance with trait anxiety or anxiety disorders (Gu, Ge, Jiang, & Luo, 2010; Hajcak, McDonald, & Simons, 2003; Ladouceur, Dahl, Birmaher, Axelson, & Ryan, 2006) and the Behavioral Inhibition System (BIS; Boksem, Tops, Wester, Meijman, & Lorist, 2006; De Pascalis, Varriale, & D'Antuono, 2010). The question arises if these electrophysiological components are also linked to psychopathy, in particular the FRN, consistent with the suggestion of Dinn and Harris (2000) of impaired feedback processing underlying the behavioral adaptation deficits found.

The majority of studies related to psychopathy investigated internal error monitoring (i.e. ERN; Brazil et al., 2009, 2011; Munro et al., 2007; von Borries et al., 2010) with inconsistent results. As far as feedback processing is concerned, two studies reported no FRN amplitude modulation related to psychopathy in a probabilistic gambling task (von Borries et al., 2010) and in a visual Go/No Go task (Varlamov, Khalifa, Liddle, Duggan, & Howard, 2010). With regard to the P3 component, but unrelated to feedback processing, PPI-R Self-Centered Impulsivity was associated with reduced frontal P3 amplitudes in an oddball task (Carlson et al., 2009), whereas PPI-R Fearless Dominance was associated with increased P3 amplitudes in a continuous performance task (Carlson & Thái, 2010). A meta-analysis of Gao and Raine (2009) showed inconsistent, task-dependent effects on the P3 for psychopathy.

1.3. The present study

Importantly, none of the studies investigating error monitoring focused on specific psychopathic traits in a multi-dimensional fashion, as also discussed in Pfabigan, Alexopoulus, Bauer, Lamm, and Sailer (2011). This creates two potential problems for investigating associations between psychopathy and feedback processing. First, psychopathic traits might be differentially related to error monitoring. Working with a unitary construct (i.e. total scores instead of specific psychopathic traits/higher-order factors) could obscure potential associations with both, the FRN and ERN. Second, categorical grouping of dimensional data (i.e. splitting subjects into low- and high-scoring groups) leads to a loss of information about individual differences (MacCallum et al., 2002). From a dual-process perspective, each individual is located on two functionally interrelated dimensions rather than belonging to qualitatively discrete groups of psychopaths and non-psychopaths. To overcome these shortcomings we investigated the potentially differential associations between dimensional psychopathic traits and feedback processing.

Therefore, we used the PPI-R (Alpers & Eisenbarth, 2008), which is applicable also in the low and moderate range of psychopathy, enabling us to investigate potential etiological processes across a broader dimensional range in an undergraduate/graduate sample at the University of Vienna. Moreover, we investigated a female only sample to control for any gender differences that might occur and to enhance our knowledge about this less-studied population. Participants performed a modified time-estimation task (Miltner