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Review

Electrophysiological indices of biased cognitive processing of substance-related cues: A meta-analysis

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ABSTRACT

Several studies indicate that individuals with substance use disorders (SUD) exhibit biases in the cognitive processing of substance-related stimuli. These biases facilitate the detection of substance cues and have been argued to play a causal or perpetuating role in addiction. Two electrophysiological indices of cognitive processing, the P300 and Slow Potential (SP) components of the event-related potential (ERP), are associated with the deployment of attentional resources to motivationally relevant stimuli. In the present meta-analysis P300 (300–800 ms) and SP (>800 ms) amplitudes are used to investigate whether SUD persons show enhanced cognitive processing of substance cues relative to neutral cues as opposed to control participants. Results indicated the P300 and SP amplitude effect sizes were significantly larger in SUD participants than controls. This result is explained by substance users' motivated attention. Additional stratified moderator analyses revealed that both P300 and SP amplitudes were not moderated by electrode site (Fz vs. Pz), type of substance used (stimulants vs. depressants), substance use status (abstinent vs. non-abstinent), age, gender and task requirements (active vs. passive paradigms).
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1. Introduction

1.1. Cognitive processing biases in addiction

Addiction is a chronic, hard-to-treat condition characterized by cravings and frequently occurring relapse. Over the years, addiction has been associated with enhanced reactivity in response to substance-related stimuli such as the sight or smell of drugs and drug paraphernalia. This drug cue reactivity is comprised of a physiological component (e.g., skin conductance), a psychological component (e.g., self-reported urges; for a review, see Carter and Tiffany, 1999), and a cognitive component, i.e., individuals with substance use disorders (SUD) exhibit biases in the cognitive processing of substance-related stimuli. These biases, including biases in attention and memory, facilitate detection and selection of substance cues and have been argued to play a causal or perpetuating role in the physiological and psychological reactivity to substance cues. For example, it is hypothesized by Franken (2003) that SUD individuals automatically detect and orient attention toward substance-related stimuli. This increased attention for substance cues diminishes attention left for alternative cues, enhances substance-related cognitions, and causes subjective craving. The biological basis of these processing biases can be explained by the incentive-sensitization theory of addiction (Robinson and Berridge, 1993). This theory posits that repeated drug administration causes a sensitization of dopaminergic neurotransmission in the brain. Because of this sensitization drugs and drug-related stimuli acquire incentive motivational properties, which can alter the way they are perceived and processed. More specifically, substance-related stimuli will be perceived as particularly salient and reinforcing, and attention will be preferentially allocated to these stimuli (Franken, 2003; Robinson and Berridge, 1993; Ryan, 2002).

The existence of cognitive processing biases in addiction has been repeatedly confirmed in studies using various behavioral tasks (see for reviews Field and Cox, 2008; Field et al., 2009). For example, utilizing modified Stroop tasks, it has been demonstrated that SUD individuals are slower to color-name substance-related words than neutral words relative to control participants (see Cox et al., 2006). In addition, using visual probe tasks, it has been shown that substance users exhibit faster reaction times to probes replacing substance-related cues than to probes replacing neutral cues (e.g., Chanon et al., 2010; Franken et al., 2000; Mogg and Bradley, 2002). Results from these studies as well as from studies utilizing other attentional paradigms (e.g., eye movement studies, dual task procedures, flicker induced change blindness paradigms: see Field and Cox, 2008; Field et al., 2009), indicate that substance users display enhanced attentional processing of substance-related stimuli. In addition to substance-related attentional bias, substance-related memory bias has also been demonstrated. For example, Franken et al. (2003a) showed that after a picture color matching task, alcohol patients recalled more alcohol pictures, but not neutral pictures, than light drinkers. Using a more implicit measure of memory, i.e., a word-stem completion task, McCusker and Gettings (1997) found that after a modified Stroop task, word-stems primed more gambling-related words in pathological gamblers than in controls. Additional evidence comes from neuroimaging studies, in which it has been shown that presenting substance-related stimuli to SUD individuals not only increases activations in brain circuits involved in motivation and reward, such as the amygdala and the ventral striatum, but also in brain circuits that are normally involved in learning, memory and attention, such as the hippocampus, the prefrontal cortex and the anterior cingulate cortex (David et al., 2005; Hyman, 2005; Luijten et al., 2010; Robbins and Everitt, 1996).

Mounting evidence suggests that cognitive processing biases are important in the development and maintenance of addiction. Attentional bias has been associated with poor treatment outcome (Carpenter et al., 2006), relapse following treatment (Cox et al., 2002; Garland et al., 2011; Marissen et al., 2006; Waters et al., 2003) and substance consumption behavior (Cox et al., 2007; Fadardi and Cox, 2009; Field and Eastwood, 2005; Waters and Feyerabend, 2000), and both memory and attentional bias have been associated with self-reported craving (Field et al., 2009; Franken et al., 2003a). All these studies indicate that attentional bias is an important concept in addiction; at least it seems an important predictor of craving and substance use behavior. However, direct evidence for a causal role of attentional bias in substance use is lacking (e.g., Hogarth et al., 2008, 2009).

To summarize, addiction is characterized by cognitive processing biases. SUD individuals are shown to selectively attend to substance-related stimuli and memorize these at the cost of other stimuli. Associations between these biases and craving, substance use and relapse highlight the importance of further investigation in cognitive processing biases in addiction. In this meta-analysis we will focus on a relatively new method to asses cognitive processing of substance cues, i.e., the measurement of Event-Related Potentials (ERPs) using electroencephalography (EEG) techniques. ERP methodology provides a potentially more direct assessment of attentional processing than conventional behavioral (reaction time/accuracy) data relying on indirect motor-responses.

1.2. Event-related potentials as index for cognitive processing bias

ERPs are manifestations of brain activities that occur in preparation for, or in response to discrete events (Fabiani et al., 2000). They consist of several peaks and troughs that tend to co vary in response to experimental manipulations. Positive and negative deflections that have been associated with specific information-processing operations are called components (Coles and Rugg, 1995). Components are often labeled after their polarity (e.g., positive) and relative latency (e.g., 300 ms) and vary in amplitude which presumably depicts the extent to which a processing operation is engaged (Kok, 1990). In the present meta-analysis we focus on two late positive ERP components that have been consistently associated with attentional processes and have been studied most frequently in drug cue-reactivity paradigms, namely the P3 or P300 or (early) Late Positive Potential (LPP) and the Slow Positive Wave (SPW) or Slow Potential (SP) or (late/sustained) LPP. We will adopt the terms P300 and SP throughout this meta-analysis.

1.3. Late ERP components: P300 and Slow Potential (SP)

The P300 component refers to a large positive deflection of the ERP, arising about 300–800 ms after stimulus presentation, which is typically maximal at medial central and parietal electrode sites

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