



Research report

Early onset cigarette smokers exhibit greater P300 reactivity to smoking-related stimuli and report greater craving

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ARTICLE INFO

Article history:

Received 29 August 2017

Received in revised form 9 January 2018

Accepted 24 February 2018

Available online 7 March 2018

Keywords:

Smoking

Early onset

P300

Event-related potentials

Cue reactivity

EEG

ABSTRACT

Adolescence is a period during which a number of critical neuromaturation processes occur and the vulnerability for developing nicotine dependence is extremely high. Thus, early-onset (EO; age < 16 years old), relative to late-onset (LO; age ≥ 16 years old), tobacco smoking may be uniquely deleterious for developmentally immature systems that regulate neural signaling reactivity. This study investigated how age of tobacco smoking onset affects neurophysiological measures of smoking cue reactivity and reported craving in adult smokers. EO smokers (EOS; n = 8; 4 females), LO smokers (LOS; n = 10; 5 females), and healthy non-smokers (HNS; n = 10; 5 females) participated in an event-related potential (ERP) cue reactivity study with tactile and image stimuli. Participants handled neutral objects during one interval and smoking-related objects during a second interval. After each interval, they viewed smoking-related, neutral, or arousing images using an oddball paradigm. P300 ERPs and craving for tobacco were recorded during each session. P300 amplitudes were significantly higher in central midline (Cz) channel to smoking, but not neutral or arousing, images after handling smoking objects. Specifically, Cz P300 smoking amplitudes were significantly greater in EOS, relative to LOS and HNS, and associated with greater craving at baseline. There were no other group differences in mood or craving. EOS exhibited greater P300 reactivity to smoking-related stimuli, relative to LOS, suggesting a more sensitized neural response. EO smoking during early neuromaturation may alter neurophysiological signaling involved in responding to smoking-related stimuli, which could impact the outcome of smoking cessation interventions.

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1. Introduction

Tobacco smoking among adolescents remains a persistent threat to public health (Ng et al., 2014). Nationwide population data shows that, among adolescents aged 12–17, approximately 850,000 are recently initiated cigarette smokers and approximately 1.2 million recently reported current cigarette smoking (SAMHSA, 2015). Previous reports have shown that nearly 80% of adult chronic smokers began smoking prior to age 18 (CDC, 2014), and commensurate with these data, individuals who initiate smoking during adolescence are more likely to have greater difficulty quitting as an adult and to become lifetime smokers (Taioli and Wynder, 1991; Sussman, 2002).

Adolescence represents a vulnerable period for smoking initiation and dependence that also temporally coincides with crucial brain maturation processes. Cortical synaptic pruning and white matter myelination refinements persist throughout adolescence and early adulthood, and plateau at approximately ages 24–25 (Gogtay et al., 2004; Tamnes et al., 2010). These developmentally critical processes are particularly important for greater neural signaling efficiency, normative neurotransmitter release, and strengthening of neural connections (Rakic et al., 1994; Dwyer et al., 2009). The lasting effects of nicotine on the adult brain are determined in part by the neurodevelopmental stage, such as early or late adolescence, at which nicotine exposure is initiated (Trauth et al., 1999). More specifically, the neurobiological impact of early-onset smoking, defined as smoking initiated prior to age 16, may be especially deleterious because maturational changes in active synaptic pruning and rearrangement (Huttenlocher, 1979, 1990) and white matter myelination (Paus et al., 1999) begin to accelerate during early adolescence. Age 16 also has been used

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consistently in substance abuse research as a dividing mark for early- vs. late-onset drug use (Ehrenreich et al., 1999; Slade et al., 2008; Norberg et al., 2009; Becker et al., 2010; Gruber et al., 2012a,b; Sagar et al., 2015; Dahlgren et al., 2016).

Persistent exposure to nicotine via tobacco smoking throughout adolescent neurodevelopment can damage newly maturing synaptic signaling pathways and alter patterns of neurotransmitter release, increasing sensitivity to reward-related neural activation and susceptibility to developing nicotine dependence (Abreu-Villaca et al., 2003a; Dwyer et al., 2009). To this end, preclinical findings have shown distinct differences in nicotine self-administration behaviors between adult rats treated with nicotine during either periadolescence or postadolescence (Adriani et al., 2003). Periadolescent rats treated with nicotine between postnatal days 34–43 (an age range comparable to ‘early onset’ or early adolescence (Arrant et al. 2013)), compared to postadolescent rats treated with nicotine between postnatal days 60–69 (an age range comparable to ‘late onset’ or late adolescence/early adulthood), exhibited greater motivation and higher rates of responding to self-administer nicotine when tested as adults (approximately postnatal day 105) (Adriani et al., 2003). Thus, periadolescent (e.g. ‘early onset’) exposure to nicotine can produce distinct sensitized responses to the salient appetitive properties of the drug that correspond to greater self-administration behavior and facilitate the transition to long-term drug dependence in adulthood (Morgan et al., 2006).

Investigating the effects of early or late onset adolescent smoking on maintenance of smoking behaviors in adulthood, which also could affect and interfere with smoking cessation attempts, will help define the pathophysiology of nicotine dependence. Elevated reactivity to smoking-related cues (i.e. ‘smoking cue reactivity’), such as smoking-related images or cigarette smoke, is hypothesized to play a critical role in the maintenance of nicotine dependence and in increasing relapse vulnerability if a quit attempt is made (Niaura et al., 1988; Rohsenow et al., 1990; Janes et al., 2010). Exposure to smoking-related objects, (e.g., a cigarette box, cigarettes, or an ashtray), relative to neutral objects (e.g. a pen or pad of paper similarly sized to a cigarette box), is also an important determinant of smoking craving and reactivity (LaRowe et al., 2007). Additionally, behavioral measures of anxiety, withdrawal, depressive feelings, impulsivity, and craving reported by smokers are also known to influence cigarette smoking behaviors and cue reactivity (e.g. Doran et al., 2009; Weinberger et al., 2012). Overall, examining if early onset adult smokers exhibit greater smoking cue reactivity to smoking-related images and objects, relative to late onset adult smokers, who initiated smoking at or after 16 years old, will give some indication of functional differences between the two groups in smoking cue sensitivity. Furthermore, investigating relationships between elevated cue reactivity and mood and craving measures will reveal if different aspects of subjective affect or craving reported by EOS and LOS specifically influence smoking cue reactivity.

One approach for modeling smoking cue reactivity in the laboratory is through the use of electroencephalography (EEG) and event-related potentials (ERPs). EEG directly measures continuous neuronal electrical activity within the cortical surface of the brain. A methodological strength of EEG is millisecond-range high temporal resolution, which facilitates synchronization of cortical electrical activity with specific experimental events, such as responding elicited by repeated image stimulus presentations (Hajcak et al., 2010). The EEG signal encompasses positive and negative voltage fluctuations over time that, when time-locked to a discrete event or defined stimulus, are called ERPs (Hajcak et al., 2010; Wallois et al., 2012). ERPs are commonly defined by the polarity and approximate latency of the voltage deflection in the EEG signal. Thus, the ‘P300’ ERP is a positive waveform that peaks

approximately 250–550 ms following stimulus presentation (Kemper et al., 2012). Notably, P300 amplitude increases following presentation of an infrequent target or task-relevant stimulus, suggesting an increase in the degree of cortical information processing and attentional resource allocation that is taking place (Kok, 1990; Polich and Criado, 2006). Other factors that influence P300 amplitude and latency include attentional resource redirection and incentive saliency of novel stimuli (Spencer et al., 2001; Polich and Criado, 2006; Mantini et al., 2009). The P300 reflects automatic motivational attention processing and allocation to salient stimuli (Kujawa et al., 2013) whereas a longer latency ERP, the late positive potential (LPP), is considered to be sensitive to sustained elaborative processing of emotionally salient stimuli (Weinberg and Hajcak, 2011). Our *a priori* intent is to measure automatic reactivity related to the motivational salience and relevance of smoking-related cues, which are properties known to support drug-seeking and drug-taking behaviors (Robinson and Berridge, 1993), thus our focus is on the P300 component.

Drug cue reactivity oddball paradigms are commonly used to study the Associations between brain electrical reactions and drug-related image stimuli presentations. Three-stimulus oddball paradigms, in particular, utilize a sequence of stimuli presentations in which rare infrequent ‘target’ stimuli and rare infrequent non-target stimuli are randomly inserted in a series of frequently presented neutral stimuli (Katayama and Polich, 1998). Studies indicate that drug-dependent patients will redirect attentional resources to highly salient target or distractor drug-related images, which increases corresponding P300 amplitudes (Littel and Franken, 2012; Robinson et al., 2016). Relative to frequent neutral picture cues and healthy non-drug-using adults, P300 amplitudes in drug-dependent adults increased in response to target heroin- (Lubman et al., 2007), cocaine- (Penetar et al., 2012), smoking- (Littel and Franken, 2011; Evans et al., 2013), and marijuana-related (Nickerson et al., 2011; Henry et al., 2014) picture cues. Furthermore, each of these three-stimulus oddball P300 studies included target non-drug picture cues, which did not elicit significant P300 reactivity comparable to the specificity of elevated P300 drug cue reactivity. Less is known, however, about P300 smoking cue reactivity measured by a three-stimulus oddball paradigm during acute nicotine deprivation. Changes in P300 amplitude and latency to smoking-related cues have not been characterized in early and late onset adult smokers but could elucidate novel patterns of neural reactivity that are elicited by the incentive value of tactile and visual cues and influenced by nicotine exposure during early or late stages of brain maturation.

Previous smoking cue reactivity studies have revealed that P300 amplitudes measured from midline Fz, Cz and Pz locations in cigarette smokers were larger in response to smoking-related than neutral cues (Warren and McDonough, 1999; McDonough and Warren, 2001; Littel and Franken, 2007, 2011; Luijten et al., 2016). Centro-parietal channels typically reflect a strong P300 response elicited by rare target stimuli (Brown et al., 2015), therefore the primary focus of the present study was on Cz and Pz reactivity. Given the strong centro-parietal topography of the P300 response, Fz P300 amplitudes were assessed as a secondary analysis. Findings of Fz reactivity to smoking-related imagery are sometimes inconsistent, with some observing higher P300 amplitudes (e.g., Warren and McDonough, 1999; Littel and Franken, 2007) and others observing less pronounced reactivity (Engelmann et al., 2011). P300 ERP were used to explore whether early and late onset smokers exhibit differences in smoking cue reactivity and if reactivity was altered by handling neutral or smoking-related objects. It was hypothesized that early onset smokers would exhibit greater P300 amplitudes in Cz and Pz electrode sites in response to viewing smoking-related images after handling smoking-related objects and would further report greater craving prior to and during the

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