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Research Report

Light up and see: Enhancement of the visual mismatch negativity (vMMN) by nicotine

Derek J. Fisher^{a,d,*}, Terri Lynne Scott^b, Dhrasti K. Shah^{a,c,d}, Stephanie Prise^b, Mackenzie Thompson^b, Verner J. Knott^{a,b,c,d}

^aDepartment of Psychology/Institute of Neuroscience, Carleton University, Ottawa, ON, Canada

^bSchool of Psychology, University of Ottawa, Ottawa, ON, Canada

^cUniversity of Ottawa Institute of Mental Health Research, Ottawa, ON, Canada

^dRoyal Ottawa Mental Health Centre, Ottawa, ON, Canada

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ABSTRACT

Both smoking and nicotine can facilitate cognitive efficiency in humans, however the exact mechanism underlying this improvement in cognitive performance is unclear. Nicotine-related improvements in visual task performance may stem from facilitation of the identification and encoding of rare deviant stimuli at early sensory levels. Visual processes at these early levels are thought to be indexed by the visual mismatch negativity (vMMN), an event-related potential (ERP) measure of pre-conscious deviant detection. In order to contribute to our understanding of the neural mechanisms underlying nicotinic modulated cognition, the current study investigated the acute effects of nicotine on vMMN in a non-smoking sample. Twenty-seven volunteers (7 males, 20 females) were treated with nicotine gum (6 mg) in a double-blind randomized, placebo-controlled repeated measures design. ERPs (vMMN; visual N100 and P200) and motor indices of performance were extracted from an intermodal task, requiring participants to attend selectively to auditory targets presented within concurrent, non-overlapping oddball sequences of visual standard and deviant stimuli. Behavioural performance was unaffected by nicotine, however nicotine was found to enhance vMMN and P200 amplitude. The findings are discussed in relation to attentional and neurobiological theories of nicotine dependence and of cognition in general.

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1. Introduction

Recent estimates suggest that over 44.5 million American adults are dependent on cigarette smoking (Henningfield et al., 2000; Volkow, 2007). It is thought that nicotine, the main psychoactive ingredient in tobacco, is responsible for smoking reinforcement (Heishman et al., 1994; Levin et al., 2006). Nicotine's addictive potential is thought to be couched in its

rapid delivery (~8–10 s) to the brain when absorbed by alveoli in the lungs after smoking a cigarette (Heishman et al., 1994), as well as its purported mood-modulating and cognition-enhancing (Gilbert and Gilbert, 1998; Waters and Sutton, 2000) effects. The convenience, relative social acceptance (compared to other drugs of abuse) and reliability (i.e. nicotine levels in tobacco are industry regulated and consistent) of smoking also contribute to its appeal (Henningfield et al., 2000).

* Corresponding author. Department of Psychology/Institute of Neuroscience, Carleton University, Ottawa, ON, Canada.
E-mail address: dfisher@rohcg.on.ca (D.J. Fisher).

Activation of neuronal nicotinic acetylcholine receptors (nAChRs) permits modification of neuronal excitability: pre-synaptic nAChRs modulate the release of various neurotransmitters, including acetylcholine, dopamine, serotonin and norepinephrine, (Decker et al., 2000; Poltavski and Petros, 2005; Shytle et al., 2002), all of which have been implicated in the regulation of mood states and cognitive processes, including memory and attention (Decker et al., 2000; Kassel 1997; Le Houezec, 1998; Newhouse et al., 2004).

Although the positive effects that nicotine consumption exerts on the brain vastly contribute to its reinforcing and addictive properties (Waters and Sutton, 2000), avoidance of nicotine withdrawal symptoms due to abstinence makes a significant contribution to maintaining this behaviour as well (Le Houezec et al., 1994). During brief smoking abstinence, sharply declining nicotine levels are accompanied by deficits in mood, arousal and cognition (Le Houezec et al. 1994; Le Houezec, 1998; Waters and Sutton, 2000). For this reason, the use of smokers for investigations into the underlying acute reinforcing effects of nicotine, including cognitive enhancement, is problematic. Improvements in performance resulting from acute nicotine in over-night abstaining smokers may reflect relief from, or normalization of, withdrawal-associated performance decrements and not the absolute enhancing effect of nicotine *per se* (Heishman et al., 1994; Poltavski and Petros, 2005; Waters and Sutton, 2000).

Several studies have reported nicotine-induced performance increments in non-smokers (Heishman et al., 1994; Kassel, 1997; Le Houezec et al., 1994; Levin et al., 2006; Poltavski and Petros, 2005, 2006) and these and similar findings in smokers have been attributed to nicotine's ability to augment brain systems mediating selective attention. This position has been strongly advocated by the "stimulus-filter" hypothesis of smoking, which proposes that nicotine can narrow attention by acting as a stimulus barrier, screening distracting and irrelevant stimuli from the smokers' awareness; by implication, this concomitantly enhances the processing of target stimuli, and may facilitate task performance (Kassel, 1997). Focusing attention in this way would appear to be beneficial as there is a general limitation on how much information can be attended to and processed at one time (Posner, 1995). The revised stimulus-filter model, however, goes on to suggest that nicotine increases available processing resources, particularly those implicated in stimulus encoding, through its action on locus coeruleus noradrenergic cells (Kassel, 1997). If this is indeed the case, one might expect nicotine to enhance the processing of task-independent stimuli without any decrement in primary task performance.

Selective attention tasks assess how well target stimuli can be detected in the presence of irrelevant, non-target, potentially distracting stimuli (Kassel, 1997; Heishman et al., 1994; Warburton, 1992), and while behavioural responses to the target stimuli are recorded, many investigators purporting to provide evidence for the stimulus-filter hypothesis neglected or were unable to analyze responses elicited by the unattended, distracting stimuli. As a result, the mechanism by which nicotine influences attentional selectivity remains unaddressed and the question of whether nicotine acts to filter out irrelevant stimuli and/or increases focus on relevant stimuli remains unanswered (Kassel, 1997). To make the

distinction between these processes evident and to fully characterize nicotine's modulation of selective processing, it is essential to monitor behavioural responses to attended and non-attended stimuli in more than one stimulus category (Knott et al., 2006b).

Investigating the cortical mechanics of selective attention requires rigorous methodology that is sensitive to the time dynamics of information processing (Posner, 1995). Scalp-recorded electroencephalographic (EEG) activity has a temporal resolution in the range of milliseconds (ms) and, combined with source localization of recordings from multiple scalp sites, allows for a spatio-temporal analysis of activity of various brain regions that mediate attention (Fabiani et al., 2000; Luck and Girelli, 1998; Mangun and Hillyard, 1995). The use of multiple, time-locked, averaged EEG segments, resulting in event-related potential (ERP) waveforms, extracted during the performance of tasks requiring selective processing of stimuli averaged over multiple stimulus presentations can be useful to uncover how nicotine influences the processing of both target and distracting stimuli (Knott et al., 2006b).

The use of ERPs specifically suited to indexing various stages of information processing (e.g. sensory encoding, stimulus evaluation) may contribute to our understanding of how nicotine affects the processing of stimuli both inside and outside the focus of attention, and permits a comparison of this data (Knott et al., 2006a; Luck and Girelli, 1998; Mangun and Hillyard, 1995). One such ERP which may be well suited to enhancing our understanding of stimulus evaluation at a pre-conscious level, and how this may be affected by psychoactive substances, is the visual mismatch negativity (vMMN). The vMMN is thought to be the visual analog of the auditory MMN, a well established ERP component elicited by any irregularity in the auditory environment. However, while the auditory MMN is characterized by a fronto-central maximum in the 100–250 ms range (Näätänen, 1992; Näätänen et al., 2007), the vMMN has a more posterior orientation, with peaks occurring around occipital sites across a maximum latency range of 100–400 ms (Czigler et al., 2006). The vMMN shares several commonalities with its auditory counterpart, notably that it may be elicited by the occurrence of a repeated stimulus if the presence of that stimulus violates a rule, indicating that the vMMN is not simply elicited by change detection, but rather is elicited by sensory memory based deviance (regularity-violation) detection. Furthermore, the vMMN can be detected in response to a wide range of stimuli, including, but not limited to, deviances of color (Czigler et al., 2002; Kimura et al., 2009), motion direction (Lorenzo-Lopez et al., 2004; Amenedo et al., 2007), stimulus location (Berti and Schröger, 2004; Berti and Schröger, 2006) and line orientation (Astikainen et al., 2004) much like the auditory MMN. The vMMN (as well as other ERP components such as the N100 and P200) has been shown to be elicited by selective attention paradigms (Astikainen et al., 2008), where independent, parallel streams of stimulus sequences are presented to two separate channels (auditory and visual); participants are required to attend and detect deviants in the auditory channel while ignoring stimuli in the other (visual) channel. This methodology, analogous to the standard practice of presenting a visual task (such as a silent movie) during an auditory MMN paradigm, may even be preferable for eliciting the vMMN given that there is no

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