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ORIGINAL INVESTIGATION

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Smoking, processing speed and attention in a choice reaction time task

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Abstract Nineteen subjects performed a choice reaction time task in which two levels of choice (two and four stimuli), and two levels of spatial attention (narrow and wide) were manipulated under each of two smoking conditions: sham smoking (denicotinised cigarette) or regular smoking (0.8 mg nicotine cigarette). All three factors significantly affected reaction time, with the smallest reaction times being recorded to the two-choice narrow grouped stimuli recorded under the high nicotine condition. Nicotine appears to speed decision time for both complex and hard-to-attend tasks, which is compatible with a role for nicotinic receptors in systems jointly mediating attention, memory and processing speed.

Key words Information processing · Attention · Decision time · Nicotine · Smoking

Introduction

While several writers have reported null or negative effects of nicotine on some types of information processing tasks (Clarke 1994), nicotine has been shown to enhance performance on both selective attention tasks (Provost and Woodward 1991), and concentration or sustained attention, where it has been suggested that smoking acts by speeding up stimulus evaluation (Edwards et al. 1985). Provost and Woodward (1991) reported that nicotine increased the speed with which 24 non-smoking subjects, given either a 2 mg oral dose of nicotine or a placebo, learned to name the colour of incongruous color-word stimuli in the Stroop test. The test was given on three occasions and, while the 2 mg dose of nicotine had no effect on either simple color naming or word reading, it did increase the rate at which incongruous-color word naming speed increased over successive occasions. Provost and Woodward (1991) argued that, rather than altering selective attention or information processing speed, nicotine influenced the rate at which responses became automatised. This finding does not, however, exclude attentional explanations. It can be understood within conditioned attention theory (Lubow 1989) as an effect of increased latent inhibition, which facilitates learning to ignore the irrelevant color information. Similarly, increased focussed attention may have facilitated learning of the correct response strategy, while not being apparent in the RTs in the initial conflict situation.

Most recently, nicotine has been shown to enhance direct behavioral measures of processing speed, including the decision time (DT) component of a choice reaction time task (Bates et al. 1994), as well as the inspection time (Nettlebeck and Lally 1976) measure of perceptual speed (Stough et al. 1995) . The effects of nicotine on choice reaction time have been studied in a number of paradigms related to the present method, for instance by Hindmarch and his colleagues. They have demonstrated, for instance, that in regular smokers abstaining over overnight, critical flicker fusion thresholds, motor reaction time, compensatory tracking, and short-term memory RTs are all enhanced by a single dose of nicotine, with second and third doses maintaining, or, in the case of sensorimotor performance, further improving, performance (Sherwood et al. 1992). Other reports include, for instance, that of Smith et al. (1977), who showed that nicotine and caffeine both decreased the DT component of RT while caffeine, but not nicotine, also reduced the motor time component. Bates et al. (1994) also found no effect of nicotine on movement times. Both DT and IT have previously been shown to correlate with IQ test scores (Roth 1964; Jensen 1987; Nettelbeck 1987; Bates and

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Eysenck 1993), allowing the possibility that these improvements of perceptual and processing speed may index a more basic enhancement of general cognitive ability produced by enhanced nicotinic ACh receptor activity (Stough et al. 1994). It is suggested, then, that while nicotine appears to aid attention and the learning of new responses (as in the Stroop task), it may, in addition, speed information processing.

Separating the effects of nicotine on attention as opposed to memory has proven to be problematic, perhaps because of a basic connection between these processes (Warburton and Rusted 1993). Similarly, the effects of nicotine on attention and on processing speed are often confounded, making it difficult to determine whether nicotine achieves its augmenting effect via one or the other or both mechanisms. The reaction time method used by Bates et al. (1994) separately measured decision time (DT), the period of time elapsing between stimulus onset and the commencement of the physical movement, and movement time (MT), the time over which the physical response is completed. This separation of DT from MT is made possible by providing subjects with a home key which is released at the beginning of each response (Jensen 1987). Bates et al. (1994) reported a dose dependent reduction in decision time, but not movement time, suggesting that CNS effects are more important than possible influences on peripheral effectors in this paradigm. This finding, however, left unanswered the question of whether DT is reduced by enhanced information processing speed, or by alterations in attention, or by both simultaneously. In the current experiment, we examined the effect of smoking on attention and speed of information processing by modifying the standard Jensen Hick paradigm to include a separate control over the stimulus demands on spatial attention. In this new task, the number of possible choice stimuli was varied, either two or four stimuli being available, and, in addition, the spatial proximity of the stimuli was varied, with the available lights being either clustered together or separated spatially, increasing the difficulty of attending to the task and requiring subjects to concentrate harder in order to reliably detect stimulus onset. It was hoped that this method would provide information on the separate effect of nicotine on attention and processing speed.

Materials and methods

Subjects

Nineteen subjects, 13 women (aged 18–34 years, mean = 22.8) and six men (aged 18–23 years mean = 19.1) were recruited from a newspaper advertisement. All subjects were habitual smokers using between 5 and 25 cigarettes/day. They were instructed not to smoke during the 2 h prior to their laboratory appointment and this was confirmed verbally upon their arrival in the laboratory. Subjects gave written informed consent to their participation and were paid NZ$10. The response box was similar to that used by Jensen and Munro (1979) and depicted in several articles (Jensen 1987).

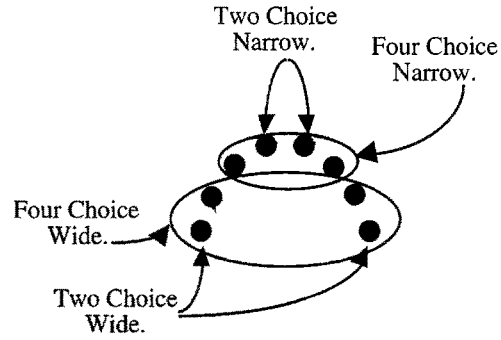

Fig. 1 The four choice stimulus configurations

Stimulus lights were green high intensity LEDs, and the response keys were 12.5 mm diameter raised press buttons. The experiment was controlled using a Macintosh II with custom Lab VIEW software (Bates 1992).

A 1-s warning tone preceded each trial, followed after an interval of 1–4 s by a stimulus light. Subjects were instructed to press the target key as soon as the target was observed. DT was the time elapsing between target onset and the release of the home key. Trials with a DT of less than 50 ms or more than 1 s, or on which a response error was made, were discarded online and additional trials given. The high degree of response compatibility and the fact that the stimulus light remained on throughout a trial ensured that few errors were made: the most errors during an entire session was three, made by two subjects during their first sessions. Most subjects made no errors.

In each condition the stimulus set consisted of either two or four lights positioned either adjacent to one another or laterally displaced by 30 degrees of visual angle (see Fig. 1). These four conditions thus varied the number of choices (two or four) and the spatial location of stimuli (narrow or wide).

Procedure

Subjects completed a smoking and a sham smoking session in balanced order on separate days. On the first session, subjects were introduced to the laboratory and briefed about the RT task in order to aid informed consent. They then completed 32 practice trials, consisting of four trials of each of the eight lights presented in random order. This was followed by 32 trials on each of the four experimental conditions under either sham or regular smoking conditions. The smoking treatment consisted of taking five puffs of either a regular 0.8 mg cigarette or a sham, denicotinised cigarette. Puffs were taken at 30-s intervals as prompted by a taped message. The sham cigarettes were denicotinised by a gas process to retain the characteristic flavours and draw of a regular cigarette, thus providing a true placebo condition. The assignment of smoking condition (smoke, sham) and of choice RT level (one bit narrow, one bit wide, two bits narrow, two bits wide) was balanced across both subjects and orders of nicotine presentation.

Results and discussion

Median DTs were calculated for each subject at each condition. A three way repeated measures ANOVA with factors of nicotine level, bits of choice, and spatial distance was then computed. As was to be expected, DT was linearly related to bits of choice [$P(F = 10.63, df = 1) < 0.004$]. The effect of nicotine on DT was also significant [$P(F = 4.44, df = 1) < 0.048$], replicating the

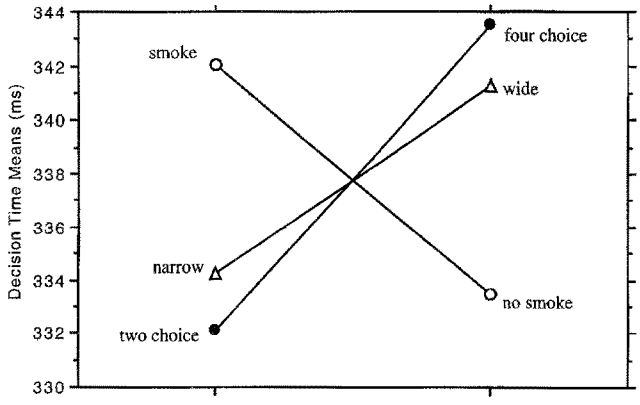

Fig. 2 Main effects of spatial distance bits of choice and smoking

Bates et al. (1994) finding, as was the effect of spatial location [$P(F = 6.09,\ df = 1) < 0.023$]. These main effects are graphed in Fig. 2. None of the interaction effects reached significance, although some suggestion of non-additivity was apparent, with the greatest smoke-sham difference being recorded in high information, wide attention condition.

These results indicate that, as expected, increases in stimulus information content and in the spatial displacement of the stimuli increase DTs. By contrast, nicotine reduces DT in all conditions. From Fig. 2, it would appear that a medium dose of nictotine affects DT approximately as much as either of the two stimulus manipulations. Notably, the effect of nicotine is present in all combinations of the information-content and attentional demand manipulations.

The present demonstration that smoking counteracts the adverse effects of increased attentional demands on DT is consistent with the previously demonstrated role of acetylcholine in selective attention (see Callaway et al. 1992 for a recent review). The result also has implications for conventional DT research, suggesting that the traditional Jensen (1987) method confounds attentional power with information level, as has previously been suggested (Longstreth 1984; Bors et al. 1993).

While additional studies including a range of nicotine doses are required to specify the dose dependency of the nicotine effect on attention and DT reported here, the DT results at least are consistent with previous reports that nicotine decreases DT (Bates et al. 1994) while brief withdrawal from smoking does not raise DT in smokers (Bates and Eysenck 1994). Together, these results suggest that the speed of information processing may be at least partially underpinned by nicotinic receptor functioning, an assertion also supported by clinical studies of Alzheimer's disease (Jones et al. 1992).

The finding that nicotine affects both speed and attention is thought provoking. Plausible neural mechanisms based on fast transmission-speed cholinergic pathways can explain the effects of nicotine on both information processing and attention, the latter possibly mediated by regulation of dopamine receptors by ACh systems (Zhou et al. 1993). There is also evidence that nicotine may directly enhance sensory gating, thus helping subjects maintain attentional set (Adler et al. 1992). A recent study compatible with this speed-attention-memory nexus reports that anticholinergics impair both memory, and attention-shifting as measured by Wisconsin Card Sorting Test performance (Vanspaendonck et al. 1993). This test is believed to index frontal lobe functioning and to be a marker of schizophrenic thought disorder. Again, card sort performance is aided by neuroleptics, perhaps by an upregulation of NMDA receptor sensitivity (Gray et al. 1992). This latter connection would also help explain some of the effects of nicotine on memory as aspartate receptors are believed to underlie long term potentiation.

The present finding that nicotine increases information processing speed by a constant amount irrespective of stimulus complexity, and also enhances attending under conditions requiring a high focus of attention, is compatible with concurrent choice RT and evoked potential (EP) recordings (Knott 1986) which have shown that smoking causes a decrease in DT and also increases the electrophysiological differentiation between responses to attended and distracter stimuli as reflected in increased amplitudes of N1 the evoked potential component, a marker of selective attention (Näätänen and Picton 1987). The recorded improvement in attention is consistent with the finding that memory is enhanced by nicotine if we postulate that memory and attention operate via a common, limited capacity executive utilising nicotinic ACh receptors (Warburton and Rusted 1993).

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