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Smoking and Raven IQ

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Abstract. Nicotine has recently been shown to enhance measures of information processing speed including the decision time (DT) component of simple and choice reaction time and the string length measure of evoked potential waveform complexity. Both (DT and string length) have been previously demonstrated to correlate with performance on standard intelligence tests (IQ). We therefore hypothesised that nicotine is acting to improve intellectual performance on the elementary information processing correlates of IQ. In the current experiment we tested this hypothesis using the Raven Advanced Progressive Matrices (APM) test. APM scores were significantly higher in the smoking session compared to the non-smoking session, suggesting that nicotine acts to enhance physiological processes underlying performance on intellectual tasks.

Key words: Intelligence – APM – Nicotine – Smoking Cholinergic system

Introduction

Over the last decade substantial interest has been recorded in elementary cognitive information processing correlates of intelligence tests (Blinkhorn and Hendrickson 1982; Haier et al. 1983; Eysenck and Barrett 1985; Nettelbeck 1987; Kranzler and Jensen 1989; Juhel 1991; Matarazzo 1992; Widaman et al. 1993). Correlations between IT, DT and the string length measure of Averaged Evoked Potential (AEP) waveform complexity and IQ test performance have lead some authors to suggest that intelligence may be a quantity that is elementary in nature and which resides in physiological processes of the central nervous system (CNS; Brand and Deary 1982; Eysenck 1987, 1988; Jensen 1982, 1987). Central to this endeavour has been the notion of an information processing-intelligence factor which by limiting the rate of information processing constrains the development of knowledge. For instance, Jensen (1982, pp 98–99) has suggested that:

> Individuals with greater speed of information processing acquire more cognitively integrated knowledge and skill per unit of time that they interact with the environment. Seemingly small individual differences in speed of information processes accounting to only a few milliseconds per bit of information, when multiplied by months or years of interaction with the environment can account in part for the relatively large differences observed between individuals in vocabulary, general information and the other developed cognitive skills assessed by IQ tests.

However, the specific physiological processes involved in this relationship have not been identified. One recent approach is to use nicotine which is a mimetic of the neurotransmitter acetylcholine at nicotinic-cholinergic receptor sites. Nicotine has previously been shown to improve information processing (e.g., Wesnes and Warburton 1983, 1984a, b) and to reduce fatigue on vigilance tests (e.g., Mangan and Golding 1978), and it has been suggested that these effects are due to alterations in the activity of CNS nicotinic acetylcholine receptors. Mangan and Golding, in their arousal modulation model of smoking, proposed that some subjects smoked to modify their level of CNS arousal. Changes in arousal level (which have for many years been known to affect performance on cognitive tasks), has been hypothesised by Warburton (1981) to reflect alterations in cholinergic activity which are in turn assumed to influence electrocortical arousal. Thus, nicotine via its effect on CNS nicotinic-cholinergic receptor sites may alter electrocortical arousal which may account for individual differences in performance on cognitive and information processing tasks. In order to examine whether such a relationship may exist for tasks requiring intelligence, recent experiments have examined the relationship between nicotine and RT and the string length measure of AEP waveform complexity. Bates, Pellett, Stough and

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Mangan (1994) provided evidence that the DT component of simple and choice reaction time (RT) was significantly shortened (enhanced) in nicotine conditions compared to no-smoking and sham-smoking conditions. The DT process employed was the same as that described by Jensen and colleagues in many studies in which a negative relationship between DT and IQ has been reported (Jensen 1980, 1982, 1987; Jensen and Munro 1979; Vernon 1983). In addition, a nicotine related enhancement for the string length measure of AEP waveform complexity (Stough et al. 1994) has also been reported, suggesting that nicotine enhances performance on these information processing correlates of IQ. These experiments are suggestive of a role of nicotine (possibly via its effect on central cholinergic pathways) in enhanced intellectual performance.

There is also other evidence for a role of the cholinergic system in intellectual functioning in studies examining information processing and memory in subjects with impaired central cholinergic pathways in clinical studies studying dementia (Kopelman 1987; Broks et al. 1988). Thus, the arousal model of smoking, studies in which cholinergic system dysfunction are associated with degraded cognition, and recent studies examining the effects of nicotine on RT and the string length measure of AEP waveform complexity are all indicative of a role of the cholinergic system in intellectual performance. In order to test this hypothesis, we administered the APM in smoking and no-smoking conditions. Consistent with the hypothesis relating cholinergic pathways with intelligence, we hypothesised that in the smoking (0.8 mg cigarettes) condition Raven raw test scores would be significantly greater than in the no-smoking condition. This result may therefore further substantiate the net of relationships between intelligence, DT, string length, IT, and central nicotinic ACh receptor systems. Positive findings would support the idea that nicotine enhances the activity of at least a subset of the neurophysiological processes which underly intellectual functioning.

Materials and methods

Subjects

Sixteen subjects, ten women and six men (with ages ranging from 18 to 32 years, X=18.5) participated. All subjects were regular smokers and were instructed not to smoke during the 2 h prior to their participation in the experiment. The research was approved by the Auckland University Human Subjects' Ethics Committee and all subjects provided written informed consent.

Procedure

Each subject completed the even and odd numbered items of the APM (Yates and Forbes 1967) in two sessions, one smoking and one no-smoking. Test half and smoking conditions were assigned in a balanced order. In each of the two experimental sessions, subjects were given 20 min to complete the assigned half of the APM. Prior to beginning the smoking session subjects took six puffs of a medium (0.8 mg) nicotine cigarette every 20 s. After 10 min, subjects were prompted to take two additional self paced puffs. The test timer was not interrupted during this period. Because a split half version of the test was administered APM raw scores were used in the analysis.

Results and discussion

A paired samples *t*-test revealed a significant effect of nicotine on test score performance ($t_{1,15}$=1.9, P<0.05), suggesting that smoking significantly improved APM scores. Mean APM difference between smoking and no-smoking conditions was 0.875 points, which is approximately equal to an increase of six Raven IQ points.

These results are consistent with the hypothesis that nicotine acts centrally to improve neural processes underpinning intellectual performance. The magnitude of this effect is surprisingly large but consistent with the results of recent experiments investigating the effects of nicotine on elementary information processing correlates of IQ (Bates et al. 1994; Stough et al. 1994). The present result raises the possibility of modelling intelligence at the level of neurotransmitter systems, including ACh, as well as the future prospect of developing nootropic drugs which may selectively enhance the fast, nicotinic, acetylcholinergic receptor systems. The current results are also consistent with studies examining information processing and memory in subjects with impaired central cholinergic pathways in clinical studies using patients with dementia (Kopelman 1987; Broks et al. 1988). The arousal model of smoking in which nicotine acts to modulate central nervous system arousal level, studies in which cholinergic system dysfunction are associated with degraded cognition and the present result are all indicative of a role of the cholinergic system in processes associated with intellectual performance.

Future studies should attempt replication of this result by administering additional levels of smoking, no-smoking and sham smoking conditions in a larger sample of subjects. Although the Raven tests are generally regarded as a relatively pure measure of general intelligence, other intelligence tests should be used in future studies to more adequately address whether this nicotine related effect is specific to non-verbal intelligence or may generalise to verbal intelligence measures.

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