

## ORIGINAL INVESTIGATION

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## Effects of nicotine on perceptual speed

Received: 6 June 1994 / Final version: 20 December 1994

**Abstract** Two experiments investigating the effects of nicotine on performance in the inspection time (IT) procedure are reported. Experiment 1 compared ITs in smoking (0.8 mg nicotine cigarette), sham-smoking, and no-smoking conditions. IT was significantly shorter in the smoking condition as compared to both the no-smoking or sham-smoking conditions, suggesting that nicotine enhances early information processing. This result is of particular interest because of the correlation between IT and IQ reported in previous experiments. The nicotine related decrease in IT raises the possibility that nicotine enhances at least a subset of the physiological processes underlying intellectual performance. Experiment 2 examined the persistence of this nicotine related enhancement in IT, and investigated the effects of nicotine across 480 IT trials. Results suggested that ITs derived from the last third of the 480 trials were significantly shorter in the 0.8 mg cigarette condition than in no-smoking condition. The results from these two experiments, taken together with recent work examining the effects of nicotine on the string length measure of AEP waveform complexity and Hick decision time (DT), and studies investigating cognitive functioning and cholinergic system dysfunction in dementia, suggest a role of the cholinergic system in intellectual performance.

**Key words** Nicotine · Perceptual speed · Cholinergic system · Humans

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### Introduction

Although there is substantial evidence linking enhanced information processing speed with nicotine (e.g., Wesnes and Warburton 1983, 1984a, b) it is unclear whether nicotine acts on early or late stages of information processing. In order to examine more closely the relationship between stimulus reception processes and nicotine, the present study examines the relationship between nicotine and a more specific index of information processing termed Inspection Time (IT), which is widely regarded as a measure of the early (stimulus input) stages of information processing.

### Inspection time

Visual IT is defined as the minimum amount of time required to reach near-perfect responding accuracy in a simple two-choice visual discrimination task. By contrast with reaction time experiments, responding time is not measured. Stimulus duration is controlled by manipulating the time between stimulus onset and the onset of a backward mask. In this procedure, the backward mask acts to prevent further iconic sampling of the stimulus (cf. Vickers et al. 1972). IT has been developed to circumvent many of the problems associated with strategy use in RT procedures (e.g., speed versus accuracy trade-off; cf. Vickers and Smith 1986). The procedure has been used in a number of experimental areas in psychology, but particularly in work concerned with individual differences in psychometric intelligence (IQ). Results from this research have indicated that there is a moderate negative relationship between IT and IQ (Nettelbeck 1987; Kranzler and Jensen 1989; Juhel 1991), which some authors have explained in terms of a basic mental speed factor constraining information processing and the acquisition of information from the environment (Nettelbeck and Lally 1976; Brand and Deary 1982). This information processing

factor has been considered to be biological in nature (Jensen 1987; Eysenck 1988), although the mechanism(s) responsible for such processes are as yet unknown.

Two recent studies have reported nicotine related enhancements in other information processing correlates of IQ. Bates and colleagues (1994) reported a significant decrease in Hick Decision Time (DT) in smoking conditions compared to both no-smoking and sham smoking conditions. The Hick RT paradigm has been previously employed extensively in intelligence research. Previous results from experiments employing DT and Movement Time (MT) have suggested a small but reliable negative correlation between DT and IQ (Jensen and Munro 1979; Jensen 1980, 1982, 1987; Barrett et al. 1986). Bates et al. result indicated that nicotine, possibly via its effect on central cholinergic pathways, enhances central rather than peripheral or movement time processes.

Stough et al. (1995) have demonstrated an enhancement in the string length measure of Averaged Evoked Potential (AEP) waveform complexity in smoking 0.8 mg nicotine cigarettes versus no-smoking conditions. A significant relationship between string length and IQ has been reported in many studies, with increased string lengths being associated with higher IQ (e.g., Blinkhorn and Hendrickson 1982; Hendrickson 1982; Haier et al. 1983; Stough et al. 1990; Widaman et al. 1993). Although the physiological mechanism involved in this relationship is not known at this time, Hendrickson and Hendrickson (1980) have postulated that increased string lengths reflect more accurate information processing. Eysenck (1987) has discussed the relationship between accuracy and speed of information processing, and has suggested that speed is a process secondary to accuracy, so that inaccurate systems slow information processing.

The results of these two studies raise the possibility that nicotine enhances at least some of the physiological processes associated with intellectual performance. Therefore, it is of considerable interest to examine whether nicotine enhances IT, given the relationship between IQ and IT. An enhancement in IT by nicotine would further support the idea that nicotine, via its effect on central cholinergic pathways, acts to enhance the physiological processes involved in intellectual performance. This result would also provide evidence for the effects of nicotine on early information processing stages.

To date, there is only one published study examining the relationship between IT and nicotine. Petrie and Deary (1989) reported a significant difference in rapid visual information processing (RVIP) response time but no significant difference in IT under smoking and non-smoking conditions. Because IT is widely regarded as a measure of perceptual speed, their result, at first glance, suggested that smoking may improve components of information processing other than the earlier

ones involving sensory registration or stimulus sampling. However, there were a number of problems associated with the measurement of IT in this study. Firstly, as several authors have documented, the traditional mask used in this paradigm allows many subjects to use apparent motion cues to shorten their ITs, independently of their mental speed. It is possible that IT estimates in this study were not accurate in the sense that they did not reflect subjects' perceptual speed, which is commonly accepted as the process underlying IT (Brand and Deary 1982; Nettelbeck 1987). Secondly, by employing only 12 subjects, it is doubtful if their design would allow a statistically significant difference to be observed, unless ITs were to be substantially decreased in the smoking condition. Thirdly, the authors did not use a sham-smoking condition, so the effects of smoking independent of nicotine could not be controlled.

In order to examine whether IT is modified by nicotine, we conducted two experiments. In the first study, the IT procedure was administered to 35 subjects in three nicotine conditions (sham smoking, 0.8 mg nicotine cigarettes and no-smoking). All subjects were administered the full version of the Wechsler Adult Intelligence Scale-Revised (WAIS-R). In the second study, in order to examine the effects of smoking on fatigue in the IT task, 23 subjects were administered three sets of 160 IT trials (480 in total) in both smoking (0.8 mg nicotine cigarettes) and no-smoking conditions.

Consistent with the hypothesis relating cholinergic pathways with the physiological processes underlying intelligence, we hypothesised that in the smoking (0.8 mg nicotine cigarettes) condition, IT would be decreased. This result may therefore further substantiate the net of relationships between IQ, 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 and indicates that nicotine enhances early information processing stages.

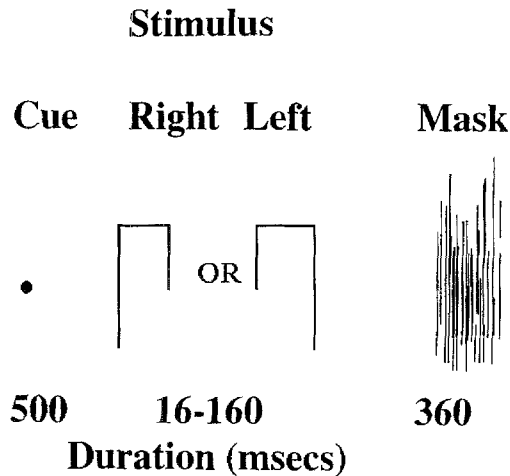
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## Study 1

### Materials and methods

#### *Subjects*

Thirty-five subjects were recruited as volunteers from an advertisement in a daily New Zealand newspaper (19 females and 16 males), with ages ranging from 18 to 35 ( $x = 24.4$ ;  $SD = 5.1$ ). Subjects were paid \$10 per hour for their participation and all were regular smokers. Subjects were requested not to smoke or drink caffeine for 2 h and to abstain from drinking alcohol for 12 h prior to the testing session. The research was approved by the Auckland University Human Subjects' Ethics Committee and all subjects provided written informed consent.



**Fig. 1** Inspection time procedure.

#### Apparatus

A Macintosh LC with a frame rate of 16.6 ms (60 Hz) was used to present the stimuli to subjects.

#### Procedure

Subjects attended three IT sessions in which they were administered the IT procedure (Fig. 1) under sham smoking, no-smoking or 0.8 mg nicotine smoking conditions. In addition, each subjects participated in one session in which the full version of the WAIS-R was administered.

**IT session.** The order of the three IT sessions was balanced. In the smoking sessions, subjects were given a cigarette to smoke. Smoking was paced, with five puffs being taken 30 s apart. In the no-smoking condition, subjects were given a 3-min break. The IT procedure involved the administration of 180 trials, 16 each at the following stimulus durations; 16.7, 33.3, 50.0, 66.7, 83.3, 100.0, 116.7, 133.3, 150.0 and 166.7 ms. In addition, all subjects completed 16 practice trials at 200 and 300 ms. The IT procedure is similar to that described by Nettelbeck (1987). Briefly, a cue was presented for 500 ms prior to each stimulus which consisted of two parallel vertical lines 24 mm and 34 mm long separated by 10 mm. Subjects were required to respond by pressing a left button if the shorter line was on the left side and a right button if the shorter line was on the right side. The shorter line had an equal probability of occurring on the left or right side. The two lines were joined by a horizontal bar across the top of each line. Following the stimulus, a mask was presented for 500 ms. The inter-stimulus interval was varied by each subject, as the next trial would only proceed after the subject made a response. Subjects were instructed to respond as accurately as possible. Following a recent study in which the effects of apparent motion were examined in the IT procedure (Stough, Bates, Mangan, Colrain and Pellett, in press), the lines mask, which is illustrated in Fig. 1, was employed. The total correct was recorded and later used to estimate IT at 75% responding accuracy (using probit analysis, in which the data are fitted to the inverse of the cumulative standard normal distribution function).

**WAIS-R IQ test.** In order to check if IT and IQ were correlated in the present sample, the full version of the WAIS-R was administered to all subjects according to the instructions outlined in the manual (Wechsler 1981).

## Results and discussion

Table 1 displays means and SDs for ITs derived from sham-smoking, no-smoking and 0.8 mg nicotine cigarette conditions. ITs derived from the nicotine condition were significantly shorter than those derived from either the sham smoking ( $t_{1,34} = 2.0$ ,  $P < 0.05$ ) or no-smoking ( $t_{1,34} = 2.2$ ,  $P < 0.05$ ) conditions. ITs were not significantly different in the sham-smoking or no-smoking conditions ( $t_{1,34} = 0.46$ , NS). This result indicates a significant nicotine related enhancement for IT, and suggests that nicotine may enhance early information processing stages. This result does not support the Petrie and Deary (1989) study in which no difference was reported between smoking and no-smoking conditions. The discrepancy between the Petrie and Deary (1989) study and the present study may be attributable to the different methodologies employed in the two studies. The present study employed a mask in which the apparent motion effect strategy is largely eliminated. The traditional lines mask employed in nearly all previous IT-IQ studies allows substantial apparent motion use (Mackenzie and Cumming 1986; Chaiken and Young 1993). Although apparent motion use in the IT procedure has not been reliably shown to invalidate or relate to the IT-IQ correlation it is possible that such a confounding variable could mask the effect of nicotine on IT.

The results are also consistent with the Bates et al. (1994) and the Stough et al. (1995) studies in which the authors reported a significant decrease in Hick DT and increase in the string length measure of AEP waveform complexity in nicotine conditions. The present results, taken together with the results of these latter two experiments, suggest a role for nicotine, via its effect on cholinergic system activity, in the physiological processes underlying intellectual performance. As several studies have reported that IT and IQ are moderately negatively correlated, shorter ITs in nicotine conditions suggests that the physiological processes responsible for IQ performance may be pharmacologically enhanced. Although IT and IQ are normally correlated, it remains to be seen whether a negative correlation is found in this sample and whether the correlations still exist after the administration of nicotine. Means, SDs and range of scores were for Verbal IQ (VIQ),  $x = 107.6$ ,  $SD = 12.8$ , range 83–138; for Performance IQ (PIQ),  $X = 108.3$ ,  $SD = 11.5$ , range 81–137; and for Full-scale IQ (FSIQ),  $X = 109.2$ ,  $SD = 12.6$ , range 86–140. Thus the sample displayed slight restriction in range of IQ scores which may

**Table 1** Mean and SD for ITs derived under three conditions

IT condition	Mean	SD
Nicotine (0.8 mg)	62.7	21.2
Sham-smoking	73.4	38.4
No-smoking	78.8	50.0

**Table 2** Pearson correlations between VIO, PIO, FSIO and ITs derived under smoking, no-smoking and sham-smoking conditions

IT	VIO	PIO	FSIO
Nicotine (0.8 mg)	-0.35*	-0.40**	-0.39**
Sham-smoking	-0.50**	-0.63**	-0.63**
No-smoking	-0.50**	-0.46**	-0.55**

\* $P < 0.05$ , \*\* $P < 0.01$

underestimate the correlation between IQ variables and IT. Table 2 presents Pearson correlations between VIQ, PIQ and FSIQ from the WAIS-R and ITs derived from the three smoking conditions employed in this study.

All Pearson correlations between IT and IQ variables in the three conditions were significant. The magnitude of the correlations, despite small restriction in range, exceed the estimations of the Nettelbeck (1987) and Kranzler and Jensen (1989) meta-analyses in which the corrected correlations between these two variables was estimated to be approximately  $-0.5$ . IT and VIQ correlations were not significantly different to IT and PIQ correlations, which does not support the findings of a recent study linking IT with WAIS-R PIQ (Deary 1993). The magnitude of the correlations was not significantly different under the three conditions. The magnitude of the correlations suggests that IT may be an important index of psychometric intelligence, and provides further evidence for the validity of IT in studies of intelligence. The fact that IT and IQ were significantly related in this study, and that nicotine significantly decreased IT is suggestive of a role of nicotine in intellectual performance. Further research should investigate the effects of nicotine on intelligence test performance.

The second study examined the persistence of this nicotine enhancement of IT across 480 IT trials.

## Study 2

### Materials and methods

#### Subjects

Twenty-three subjects were recruited as volunteers from an advertisement in a daily New Zealand newspaper (17 females and 6 males), with ages ranging from 18 to 37 ( $x = 25.9$ ;  $SD = 5.5$ ). Subjects were paid \$ 10 per hour for their participation and all were regular smokers. Subjects were requested not to smoke or drink caffeine for 2 h and to abstain from drinking alcohol for 12 h prior to the testing session.

#### Apparatus

A Macintosh LC with a frame rate of 16.6 ms (60 Hz) was used to present the stimuli to subjects.

### Procedure

Two smoking conditions were administered to all subjects: smoking (0.8 mg nicotine cigarette) and non-smoking. The IT procedure is identical to that described in the first experiment except that the IT procedure (160 trials to ten different stimulus durations) was administered three times, so that all subjects completed 480 trials across ten stimulus durations per condition. ITs were derived employing probit analysis for the first, second and third group of 160 trials.

## Results and discussion

Table 3 presents means and SDs for the three IT estimates derived under smoking and no-smoking conditions. Significant differences were obtained between IT derived from smoking compared to no-smoking from the first 160 trials ( $t_{1,22} = 2.8$ ,  $P < 0.01$ ), second 160 trials ( $t_{1,22} = 2.3$ ,  $P < 0.05$ ) and third 160 trials ( $t_{1,22} = 3.5$ ,  $P < 0.001$ ).

A repeated measures ANOVA was not significant for ITs derived under smoking ( $F_{1,22} = 0.40$ , NS) or no-smoking conditions ( $F_{1,22} = 1.0$ , NS), indicating that the three IT estimates did not differ significantly across the 480 trials in either smoking or no-smoking conditions. This suggests that there is no significant fatigue or learning effect in the IT procedure, at least up until 480 trials. Although there was a trend towards a fatigue effect, particularly in the no-smoking condition, this was not significant. These results support the findings of the first experiment and indicate that nicotine enhances IT. The fact that even for the third IT estimate there was a significant nicotine effect suggests that nicotine from a single cigarette continues to decrease IT for at least 30 min after delivery and beyond its reported half-life.

## General discussion

The two experiments presented here have provided evidence for a nicotine related enhancement in a measure of perceptual speed. This result is interesting for two reasons. Firstly, previous studies examining the effects of nicotine on information processing have generally employed tasks which are gross measures of reaction and/or decision time. Although these tasks have been useful in assessing the pharmacological effects of nicotine on human performance, it is unclear whether nicotine acts on early information processing stages in

**Table 3** Mean and SDs for ITs derived under smoking and no-smoking conditions

IT	Smoking	No-smoking
IT <sub>(1-160)</sub>	X = 49.0 SD = 20.0	X = 61.8 SD = 20.5
IT <sub>(161-320)</sub>	X = 53.3 SD = 10.5	X = 62.8 SD = 18.8
IT <sub>(321-480)</sub>	X = 53.7 SD = 18.3	X = 69.6 SD = 17.5

addition to central processes. Results from the present two studies suggest that nicotine acts to enhance stimulus sampling. It is unclear whether previous studies reporting RT decrements following nicotine administration can be explained in terms of early stimulus sampling processes as total RT subsumes IT processes. This possibility will await further research. Secondly, the two experiments reported here add further weight to the hypothesis that nicotine enhances processes associated with intellectual performance. Recently, Matarazzo (1992) has reviewed emerging research on the relationship between "physiological" measures of human performance and the cognitively more complex psychometric measures of intelligence. In his view, substantial evidence has accumulated for a significant relationship between RT, IT and the string length measure of AEP waveform complexity, all of which reflect, to some degree, speed of information processing. Although the relationship between these measures is far from clearly defined there is substantial evidence of their association with psychometric intelligence (e.g., Jensen 1980, 1982, 1987; Eysenck and Barrett 1985; Nettelbeck 1987; Lehl and Fischer 1990; Caryl 1991). Apart from the present results, which link IT, IQ and nicotine, Bates et al. (1994), and Stough et al. (1995), have provided evidence that RT and the string length measure of AEP waveform complexity are enhanced in nicotine compared to no-smoking or sham-smoking conditions. Future research may wish directly to examine the possibility that nicotine enhances IQ test performance, although as discussed by Stough et al., this pursuit may be best confined to measures of fluid rather than crystallized IQ.

It is well known that nicotine may enhance other cognitive processes (e.g., attention and memory) and it may well be that the results of this experiment are explicable in terms of these same processes. The results are also consistent with the arousal modulation model proposed by Mangan and Golding (1978) in which it is proposed that subjects smoke to modulate their arousal level. Changes in arousal level affect performance on information processing tasks, and the theory postulates a close relationship between modulation of arousal through smoking and optimal performance in subjects. This modulation in arousal has been hypothesised by Warburton (1981) to reflect alterations in cholinergic activity which are assumed in turn to influence electrocortical arousal. 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, studies in which cholinergic system dysfunction are associated with degraded cognition, and the present results are all indicative of a role of the cholinergic system in processes associated with intellectual performance.

**Acknowledgement** The authors gratefully acknowledge the support given by Philip Morris Research and Development USA.

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