



A comparison of the information processing rates of non-smokers and cigarette-deprived smokers

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Summary—The performance of 88 smokers and non-smokers was assessed on reaction time, inspection time, and choice reaction time tasks. In addition personality measures and the evoked potential string length measure of brain efficiency were recorded. Intelligence was measured in a subsample of 70 subjects. Smokers deprived of nicotine for a minimum of 2 hr showed no significant differences on any of the measured variables. The implications of this finding for deprivation models of smoking-related enhancement of information processing speed are discussed.

INTRODUCTION

Smoking or, more accurately, nicotine, has been shown to enhance the memory, mood, and speeded performance of smokers. These effects are by now well established (Warburton, 1991; Colrain, Mangan, Pellett & Bates 1992; Bates, Pellett, Stough & Mangan, 1994). Debate on the effects of smoking on psychophysiological performance has shifted to focus whether or not the performance and mood enhancement effects of nicotine in smokers are due to a genuine enhancement of performance or whether they merely reflect the remission of 'withdrawal' symptoms following smoking deprivation. Quantification of the extent of any deprivation effects is central to attempts at answering the subsidiary question of whether the smoking habit is maintained by mood and performance benefits of nicotine, or whether smokers' nervous systems adapt to chronically raised nicotine levels, thus making them dependent on nicotine simply to maintain normal levels of performance. Data addressing this question are of obvious relevance to debate on the addictive power of nicotine. For this reason we assessed information processing in nicotine-deprived smokers as compared to a group of non-smoking controls.

It has previously been demonstrated that non-smokers given nicotine in tablet form show information processing enhancement effects similar to those of smokers (Wesnes, Warburton & Matz 1983). While this clearly demonstrates that the enhancing effects of smoking are not dependent on habitual use, and may, therefore, help explain some component of smoker recruitment, this is not equivalent to a demonstration that habitual smokers continue to acquire benefits from smoking and that it is these positive benefits, rather than a dependent need for nicotine, which maintain smoking behaviour.

If smoking is maintained on a cigarette-to-cigarette, or even puff-to-puff basis by the alleviation of adverse effects on performance symptomatic of nicotine withdrawal, then these deprivation effects should vary isochronously with the systemic bioavailability of nicotine.

If this is not the case, then explanations other than deprivation must be developed to explain smoking maintenance. For cigarette smoking, bioavailable nicotine levels have been shown, for pulmonary administration, to rise rapidly to peak values followed by a drop to distribution half-life within approx. 8 min (Benowitz, Porchet & Jacob 1990). Because individual differences in metabolism and temporal patterns of smoking alter the pharmacokinetics of nicotine availability, it has been recommended that smokers be deprived overnight to ensure deprivation (Wesnes & Warburton 1983). However, after just 1–2 hr post smoking, nicotine levels are approximately asymptotic. Given that the purpose of this paper is to test the hypothesis that smoking is maintained on at least a cigarette-to-cigarette basis by addiction-like deprivation effects, it is this time period of 1–2 hr in which we are most interested to show any operational retardant effects on performance.

To test the withdrawal–maintenance hypothesis, a sample of smokers and non-smokers undertook a range of information processing tasks while abstaining from nicotine for a period of at least 2 hr prior to completing the task. The tasks chosen were simple decision time (DT), the odd-man out (OMO) measure of complex choice reaction time (CRT) (Frearson & Eysenck 1986), and inspection time (IT) (Nettelbeck & Lally, 1976; Nettelbeck 1987), a measure of the speed of perceptual information accumulation. The inspection time task involves a two alternative forced choice, in which Ss are presented with one of two simple visual stimuli for a brief interval, after which a visual mask is activated to overwrite the labile iconic store and Ss are then given an opportunity to report which stimulus was presented. The presentation duration required to allow an accurate decision has been shown to correlate strongly with IQ (Bates & Eysenck, 1993a; Nettelbeck, 1987).

We have recently reported smoking enhancement effects of nicotine in two independent Ss populations for both simple and choice DT (Bates *et al.* 1994) and IT (Stough, Mangan & Bates, submitted). We have previously reported data from the present S population indicating significant correlations between IQ and each of IT ($r = -0.624$), OMO decision time ($r = -0.365$), and CRT DT ($r = -0.28$). IT and OMO DT correlated 0.364 (Bates & Eysenck, 1993a). In addition, we have reported a relationship between evoked potential (EP) string length, a measure of metabolic efficiency relating to intelligence (Bates & Eysenck 1993b; Bates, Stough, Mangan & Pellett, submitted), and each of IT, simple DT, and choice DT (Bates & Eysenck, 1993b). These previous findings suggest that if withdrawal effects do underlie smoking maintenance, a clear

response depression effect should be visible on at least some of these measures in this large and well matched sample of non-smokers and deprived smokers.

METHOD

Subjects

Ss were 63 women (\bar{x} age = 37 yr; SD 10.1) and 25 men (\bar{x} = 33.6 yr; SD 11.525) recruited as volunteers from the local government unemployment bureau and from within the Institute of Psychiatry, London. Of these Ss 55 were smokers (\bar{x} age 38.7 yr, SD 10.6) and 33 were non-smokers (\bar{x} age 31.5 yr, SD 9). All Ss completed the EPQ-R (Eysenck, Eysenck & Barrett, 1985) as well as the odd-man out (OMO) (Frearson & Eysenck, 1986), choice reaction time (CRT) (Jensen & Munro, 1979) and IT (Nettelbeck & Lally, 1976) tasks. A total of 70 Ss also completed the Multidimensional Aptitude Battery (MAB) IQ test (Jackson, 1984). Smoking Ss were required to abstain from smoking for the 2 h prior to the commencement of testing and this condition was verified verbally.

Apparatus

The CRT and OMO paradigms were both administered on a reaction-time box functionally identical to that described by Jensen and Munro (1979). This box consists of a home button around which eight lights are arranged in a semi-circle, each with a response key beneath it.

Inspection time was measured using a custom stimulus presentation unit. Stimuli were formed by lighting various segments of an inverted U 150 mm high with a 40 mm wide top bar formed from rectangular LEDs (light emitting diodes). A fixation light was centred between the arms 105 mm below the top bar. Each bar had four LED segments of equal length. A short line consisted of two lit segments, lighting three segments made the long line, and all four segments were lit on each side to form the mask.

String length was recorded simultaneously with the IT task. During the IT task, EEG was recorded from 19 standard 10–20 sites referenced to linked ears. The supra-orbital sites Fp1 and Fp2 served as eye-blink markers. All 19 channels were filtered through Butterworth 3rd order analogue bandpass filters (0.8–300 Hz) and digitised synchronously with 12 bit accuracy in the range $\pm 60\mu\text{V}$ ($\pm 300\mu\text{V}$ for the Fp sites) at 1024 Hz, beginning immediately prior to the IT session and continuing uninterrupted for the duration of the task. An ElectrocapTM was used to position and secure the Sn electrodes, and impedances were reduced to below $5\text{k}\Omega$ by gentle abrasion. Trials were screened for artifact, defined as EEG amplitude $> 30\mu\text{V}$ in either Fp1 or Fp2 and then a linear phase lowpass finite impulse response filter set at 45 Hz was used to remove high frequency and mains-line noise. At least 75 trials were available for averaging for each S. Also, because the raw amplitude of the EP is associated with personality variables such as extraversion (Haier, Robinson, Braden & Williams, 1984; Bates *et al.*, submitted) we normalized the EPs to a mean of zero and a standard deviation of one to measure complexity independently of response magnitude. String length was determined using EP data evoked during the 300 msec following stimulus onset, and was computed as

$$\text{String} = \sqrt{\sum_{j=1}^n (x_j - x_{j-1})^2}$$

where string is expressed as $\mu\text{V}/\text{msec}$, x_j indexes the EP array, and n is the number of samples in the EP. The square root was taken in order to return the actual length of the string rather than its square (Haier, Robinson, Braden & Williams, 1983).

The psychometric tests [EPQ-R (Eysenck *et al.*, 1985), MAB (Jackson, 1984)] were administered in accordance with the directions outlined in the respective manuals.

Procedure

The tasks have been described in detail elsewhere (Bates & Eysenck, 1993a). Briefly, the IT task was administered using a staircase procedure (Wetherill & Levitt, 1965) with termination after nine consecutive correct trials in the third phase of the procedure. Stimuli consisted of inverted 'U' shapes with either the left or right descender being longer in length than its mate. The stimuli were masked by extending both 'legs' of the stimulus downwards. Each trial was preceded by a warning tone presented through head phones and a small warning light appearing on the stimulus box. After a brief delay, a left- or right-side short stimulus was presented and then masked after an interval varying from 500 msec downwards.

Both the OMO and CRT were administered at a response box with eight lights arranged in a semicircle above a home key with response keys arranged concentrically beneath the stimuli. Each trial consisted of a warning tone (1000 Hz and 70 dB SPL for 54 msec), followed, after a random interval of between 1–4 sec, by a choice stimulus. Following a practice session lasting until Ss announced that they felt confident with the procedure (typically 1.5 min and never more than 11 trials), Ss completed either 20 or 30 error-free trials on the OMO and CRT procedures. The first 29 Ss completed 20 trials, and the remaining 59 Ss 30. The additional trials, given to some Ss to explore reliability parameters, did not significantly affect either the mean or the variance of RT scores.

For CRT the S responded to a single light on each trial but was uncertain as to which of the eight possible lights would be presented. In the case of the OMO, three of the eight stimulus lights were lit in a pattern arranged so that two lights were closer to each other than the third 'odd man out' stimulus. The task was to determine as quickly as possible which light was the odd man out and to depress the appropriate response key.

In all cases the time for the S to lift his/her finger from the home button (decision time) and the subsequent time to hit a target key (movement time) were recorded separately under computer control. Response accuracy was also logged.

RESULTS

Only psychoticism ($t = 2.19$, $P = 0.03$), OMO DT ($t = -2.009$, $P = 0.0476$) and string length at T4 ($t = -2.14$, $P < 0.05$) differed significantly in smokers and non-smokers under these non-smoking conditions. For each of OMO DT and string length at T4, the differences favoured smokers. Psychoticism was raised by approximately one point in smokers, perhaps indicating

that one must be a non-conformist to continue smoking in the '90s. None of the 18 additional EEG channels, three personality factors, or numerous decision time and intelligence scales showed any significant effects of smoking group. Neither psychoticism, OMO, or T4 string length were significant when corrected for the number of tests made in this analysis.

DISCUSSION

These results indicate that smokers do not show sub-normal levels of performance on speeded tasks when deprived of nicotine. Nor do they appear to show altered levels of intelligence or personality, excepting, perhaps, P. Rather, given the reported enhancement effects of nicotine on DT (Bates *et al.*, 1994) and IT (Stough *et al.*, submitted), they merely revert to normal levels of functioning. We suggest, therefore, that smoking maintenance may be explained by the enhancement effects of nicotine rather than by withdrawal-induced behavioural depression. Certainly the frequencies of smoking behaviour correlates well enough with situational demands and plasma half-lives to suggest that this may be the case. It appears, then, that smokers are similar to non-smokers in their non-smoking performance. The possibility remains that smoking may have effects on factors not measured here, such as alterations in perceived control over mood (Eiser, Morgan & Gammage, 1987). Prior to taking up the smoking habit, smokers-to-be tend to have raised E and N scores (Cherry & Kiernan, 1976; Sieber & Angst, 1990) and the lack of differences on these variables in the present sample may indicate that chronic smoking 'normalizes' these traits, though of course we have no evidence for such an effect within this experiment. The absence of such differences within the present moderate-size sample may be due to sampling error or it may reflect a long term change in personality for these Ss.

In all these results lend support to the psychological tool (Warburton & Wesnes, 1979) and arousal modulation (Mangan & Golding, 1978) models of smoking maintenance, and tend to detract from the pharmacological or addiction model (Schachter, 1978). Addictive or pharmacological models do not appear able to explain, at least, the short term, cigarette-to-cigarette maintenance of the smoking habit. Research into the effects of longer periods of withdrawal, over days or weeks, may illuminate secondary physiological effects, but these effects, if they exist, do not appear to mediate either the performance enhancement effects of smoking or smoking maintenance. It seems more likely, given the rapid onset of performance enhancement effects, that these effects of nicotine lead smokers to maintain an adaptive smoking routine to provide acute control over arousal and information processing (Warburton & Wesnes, 1979).

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