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The Effects of Cigarette Smoking on Negative Priming

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Nicotine administration has been found to enhance performance on tasks of selective attention. It has been proposed that efficient attentional filtering depends on the successful inhibition of distracting information. In the work reported here, a negative priming paradigm was adopted to test whether smoking enhanced the inhibition of irrelevant information. Thirty-six minimally deprived smokers, half of whom smoked and half of whom sham smoked, completed the negative priming task. A significantly larger negative priming effect was found in participants who had smoked in comparison with those who sham smoked. These results support the hypothesis that nicotine enhances the inhibition of distracting information and thus suggest a possible mechanism by which smoking may enhance selective attention.

Substantial evidence suggests that the cholinergic neurotransmitter system plays an important role in modulating attentional processing (Everitt & Robbins, 1997). The subcortical structure, which provides the major cholinergic innervation to the neocortex, is the nucleus basalis of Meynert (nbM; Sinden, Hodges & Gray, 1995). Activity of the nbM mediates neocortical arousal (Buzsaki et al., 1988) and lesions of this system result in deficits in attention in both rats (Muir, Everitt, & Robbins, 1995) and monkeys (Voytko et al., 1994), which can be ameliorated by the administration of cholinergic agonists such as physostigmine and nicotine (Dunnett, Everitt, & Robbins, 1991; Ksir & Benson, 1983; Muir et al., 1995; Robbins, McAlonan, Muir, & Everitt, 1997; Sinden et al., 1995). Loss of cholinergic cells in the nbM during Alzheimer's disease is thought to be partly responsible for the severe impairments in attention and memory shown by Alzheimer's patients (Bartus, Dean, Beer, & Lippa, 1982; Whitehouse, Price, Clark, Coyle, & Delong, 1981).

Evidence for the role of the cholinergic system in attentional processing is also provided by studies that have examined the effects of nicotine administration on attentional performance in humans and animals. Nicotine from cigarettes has been found to improve the performance of smokers on a range of attention tasks, including, for example, measures of sustained attention (Edwards, Wesnes, Warburton, & Gale, 1985; Parrott & Winder, 1989; Revell, 1988; Warburton & Arnall, 1994; Wesnes & Warburton, 1983), choice reaction time (Bates, Mangan, Stough, & Corballis, 1995; Lyon, Ton, Leight, & Clare, 1975), and simple reaction time (Frankenhaeuser, Myrsten, Post, & Johansson, 1971; Morgan & Pickens, 1982).

Many early studies into the effects of nicotine on performance can be criticized on the grounds that they used deprived smokers (usually between 10 and 12 hr of abstinence). This raises the possibility that the effects of smoking on attention were due to nicotine reversing a deprivation-induced deficit in performance, rather than directly facilitating performance. A number of more recent studies, however, have shown nicotine-induced improvements in attentional performance for both minimally deprived smokers and nonsmokers. For example, in minimally deprived smokers, nicotine has been found to enhance perceptual speed (Stough et al., 1995), choice reaction time (Bates, Mangan, Stough, & Corballis, 1995; Frearson, Barrett, & Eysenck, 1988; Kerr, Sherwood, & Hindmarch, 1991; Pritchard, Robinson, & Guy, 1992), and short-term memory scanning (Sherwood, Kerr, & Hindmarch, 1992; West and Hack, 1991). With nonsmokers, nicotine has been found to improve vigilance (Wesnes & Warburton, 1984), choice reaction time (Kerr et al., 1991; LeHouezec et al., 1994), and tracking (Kerr et al., 1991). These results suggest that not all improvements in attention by nicotine are due to the reversal of a performance decrement caused by nicotine withdrawal. Therefore, although some studies have failed to find effects of nicotine on performance, and a number of studies can be criticized for being methodologically flawed (see Heishman, Taylor, & Henningfield, 1994, for a review), it still appears that nicotine has positive effects on attentional processing (Sherwood, 1993).

One aspect of attentional performance that has been found to be enhanced by nicotine is *selective attention*, which can be defined as the process of selecting objects from the external environment that are relevant to current behavioral goals and ignoring other irrelevant objects present in the same external environment (Fox, 1995). Studies that have used the Stroop task (Stroop, 1935) as a measure of selective

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attention have shown a reduction in Stroop interference in nonsmokers when nicotine was administered orally (Hasenfratz & Battig, 1992; Provost & Woodward, 1991; Wesnes & Warburton, 1978; but see Parrott & Craig, 1992, for contrasting results). Moreover, recent studies have shown that nicotinic agonists improve monkeys' performances on the delayed matching to sample task by reducing attentional distractibility (Prendergast et al., 1998), and that nicotine nasal spray enhances short-term memory in smokers when performing the task in the presence of distracting stimuli (Grobe, Perkins, GoettlerGood, & Wilson, 1998). Finally, smoking has also been found to reduce interference in selective attention on the Garner speeded classification task (Waters, 1998). In this study, smoking also reduced error rates on a measure of Stroop interference, although it did not affect reaction time. Waters (1998) concluded that nicotine may enhance selective attention by reducing the distracting effect of irrelevant sources of information. The purpose of the present study was to further examine the effects of smoking on selective attention by investigating a potential mechanism by which nicotine may enhance attentional filtering.

An influential theory of selective attention, proposed by Tipper (1985), holds that the efficiency of attentional selection depends on the efficiency with which distracting sources of information are inhibited. Much of the evidence for the role of inhibitory processes in selective attention comes from the negative priming paradigm. This paradigm typically involves presenting participants with a target and distractor stimulus on each of two successive trials (a prime trial and a probe trial). In one condition (the ignored repetition condition) the target on the probe trial serves as the distractor on the immediately preceding prime trial, whereas in the control condition the target on the probe trial is unrelated to the prime trial stimuli. The negative priming effect is the finding that participants respond more slowly to probe targets in the ignored repetition condition compared with the control condition. Thus, participants respond more slowly to target stimuli if they ignored them on the preceding trial.

Tipper (1985) accounted for negative priming by suggesting that when participants select the target stimulus on the prime trial, interference from the distractor stimulus is reduced by suppressing the representation of the distractor. Thus, when the distractor becomes the target on the probe trial the distractor's representation is in a lowered state of availability, making participants slower to respond to targets in the ignored repetition condition in comparison to the control condition. It is thus suggested that the suppression of an interfering object, as indexed by the level of negative priming, is an adaptive process implemented to overcome interference in selective attention (Houghton & Tipper, 1994). Moreover, reductions in negative priming and increases in interference, found in people with schizophrenia (Beech, Powell, McWilliam, & Claridge, 1989; Laplante, Everett, & Thomas, 1992) and in the elderly (Hasher, Stoltzfus, Zachs, & Rympa, 1991; McDowd & Oseas-Kreger, 1991), have been interpreted as an impairment in inhibitory processes that results in an inability to screen out interfering sources of information.

One alternative measure, which has been used extensively with animals to examine the effects of drugs on inhibitory processes in selective attention, is the latent inhibition (LI) effect. *Latent inhibition* refers to the finding that the repetition of a nonreinforced stimulus during a preexposure phase retards the formation of subsequent associations to that stimulus. The LI effect has been argued to be a measure of attentional filtering (Lublow, Weiner, Schlossberg, & Baruch, 1987), with the degree to which subsequent learning is inhibited reflecting the degree to which the stimulus was ignored during the preexposure phase. As a consequence, reductions in LI found in rats when administered nicotine have been interpreted as a drug-induced disruption of attentional filtering (Gray et al., 1994). Although this finding is somewhat surprising, given the literature showing nicotine-induced enhancements in attention, it is important to note that in contrast to earlier reports (Allan et al., 1995) nicotine does not appear to disrupt LI in smokers (Thornton et al., 1996), and that smoking may in fact enhance LI (DellaCasa, Hofer, & Feldon, 1999). Moreover, other work with animals has shown that nicotine can increase LI if it is administered during the preexposure phase (Rochford, Sen, & Quiron, 1996). Rochford et al. (1996) suggested on the basis of this finding that nicotine may enhance attentional filtering.

The prepulse inhibition (PPI) task has also been used to examine the effects of nicotine on attentional processing. When participants are presented with a sudden intense auditory stimulus, they reliably exhibit an acoustic startle response. However, when a weaker auditory stimulus precedes the sudden intense sound, participants exhibit a greatly reduced startle response, in comparison with a control group that does not receive preexposure to the initial weak stimulus (Hoffman & Ison, 1980). This is the prepulse inhibition effect, which has been suggested to be a protective mechanism that serves to screen out subsequent stimuli during the brief time required for the effective analysis of the initial stimulus (Graham, 1975). The protective role of PPI is demonstrated by the finding that prepulse stimuli reduce the distracting effect of acoustic startle stimuli on psychomotor performance in humans (Foss, Ison, Torre, & Wanseck, 1989). Thus, PPI is believed to be a measure of the processes underlying sensory gating (Swerdlow, Caine, Braff, & Greyer, 1992) and possibly attention (Acri, Morse, Popke, & Grunberg, 1994), with strong PPI reflecting efficient sensory gating (Swerdlow et al., 1992).

It is important to note that the cholinergic system appears to have a role in modulating PPI, with microinfusions of the cholinergic agonist carbachol into the caudal pontine reticular nucleus, increasing PPI in a dose-dependent manner (Fendt & Koch, 1999). Moreover, a number of studies have also found nicotine administration to increase PPI in rats (Acri, Brown, Saah, & Grunberg, 1995; Acri et al., 1994). However, the effect of nicotine on PPI may depend on the strain of rats used in such studies, with nicotine failing to enhance PPI in Long-Evans rats but tending to enhance PPI in Sprague-Dawley rats (Faraday, Rahman, Scheufele, & Grunberg, 1998). As noted by Faraday et al. (1998), this

raises the interesting possibility that the different effects of nicotine on PPI in different strains of rat may also be reflected in humans, with some individuals showing enhancements from nicotine because of their particular genotype. In accord with findings showing an increase in PPI in animals, nicotine has also been found to increase PPI in smoking participants (DellaCasa, Hofer, Weiner, & Feldon, 1998; Kumari, Checkley, & Gray, 1996). These results indicate that nicotine may enhance sensory gating through an effect on inhibitory processes that reduces the distracting effect of additional sources of sensory information.

The purpose of the present study was to further investigate the hypothesis that nicotine can enhance attentional filtering through an action on inhibitory mechanisms of selective attention, by examining the effects of smoking on levels of negative priming. Given the proposed importance of negative priming in selective attention (Tipper, 1985) and evidence indicating a nicotine-induced improvement in selective attention (Hasenfratz & Battig, 1992; Landers, Crews, Boutcher, Skinner, & Gustafsen, 1992; Provost & Woodward, 1991; Wesnes & Warburton, 1978) and attentional inhibition, it was predicted that smoking would result in an enhanced negative priming effect.

Method

Participants

Thirty-six students (16 men and 20 women) at the University of Sussex volunteered as participants. 9 women and 9 men were allocated to the smoking group and 7 men and 11 women were allocated to the sham group. The participants ranged in age from 18 to 38 years, with a mean age of 26. Each participant smoked regularly (more than 10 a day) and had smoked for more than a year. The nicotine content of the cigarettes smoked by the participants ranged from 0.7 mg to 1.7 mg, with the average being 1.2 mg. All participants abstained from smoking for 1 hr before the experimental session and received payment on completion of the experiment.

Design

A 2 × 3 mixed factorial design was used, with group as a between-subjects factor (smoking, sham) and condition as a within-subjects factor (one-letter, control, ignored repetition). The dependent variables were the naming latencies for the target letters and accuracy of responding.

Apparatus and Materials

A Macintosh computer, attached to a voice key, was used to present the stimuli and measure naming latencies. The equipment had a time resolution of 1 ms. A set of 12 letters were used as stimuli (A, B, C, D, E, J, K, N, O, S, T, V). These letters were presented as pairs in the center of the computer monitor. Each letter was 6 mm in height and 5 mm in width. The letters were separated by 6 mm, and participants sat approximately 70 cm from the screen. Thus, the visual angle subtended by the outer edge of one letter to the outer edge of the other letter was ≈1.64 degrees. 22 lists, each of which consisted of 10 letter pairs, were prepared. For each letter pair, one of the letters was red and the other was green and they were presented on a black background. The task of the participants was to name the red letter. There were 11 ignored repetition condition lists and 11 control lists. On Trials 3 to 9 of the ignored repetition lists, the target was always the distractor of the previous trial. (The sequence for Pairs 1 to 2 and from Pairs 9 to 10 was randomly determined in order to decrease the chance of participants noticing the pattern in the ignored repetition lists.) Target letters were randomly assigned to the right or left position with the constraint that the targets could not occur in the same position more than three times in succession.

Control lists were constructed so that the target letter and target position in each of the 11 lists matched the target letter and target position in each of the ignored repetition lists. The distractor letter for each control trial was then chosen randomly from the 11 remaining letters with the constraint that no distractor letter appeared as a distractor or a target on the next trial.

Four lists consisting of only one target letter were also constructed. For these lists, the letter and its position were randomly selected with the constraint that letters were not repeated from one trial to the next and the same position could not occur more than three times in succession.

Each of the 11 ignored repetition lists and each of the 11 control lists appeared twice in the experimental session, once in the first half of the experimental and once in the second half, giving a total of 44 paired-letter list presentations. Two of the 1 letter lists occurred in the first half of the session and the other two in the second half. The order of presentation of the lists was randomly selected, for each participant, for each half of the experimental session.

Procedure

Each participant was tested individually. The participants were informed that they would be presented with two letters, one red and one green, and they were instructed to name the red letter as quickly and as accurately as they could.

Participants completed 80 practice trials. Depending on the group to which the participants had been randomly assigned, they either smoked or sham smoked prior to starting the experimental session. Each participant smoked their own brand of cigarette.

At the start of the experimental trials the instruction "Press any key to begin" was presented on the monitor. Each trial then consisted of the following events: Two white fixation points were presented for 500 ms, followed by a letter pair presented for 200 ms, which were immediately masked by asterisks of the same color as the letters they were masking. The asterisks remained on the screen until the participant responded vocally to the target letter. After the response the masks were replaced by two more location points for 500 ms and then by a second pair of letters for 200 ms. This sequence was used for all of the letter lists. (For lists consisting of 1 letter, no distractor was presented with the target.) Participant had a 1-min break between blocks of 80 letters (after completing 8 lists). Reaction times were measured from the onset of the asterisk masks to a participant's response. Reaction times were recorded on all trials, but only responses for letter Pairs 3 to 9 were used for the analysis. This gave a total of 154 responses for the control and ignored repetition conditions and 28 responses for the 1-letter condition.

Results

Extreme scores above 1500 ms and below 70 ms were removed from the analysis. Mean correct reaction times of

the sham and smoking groups for the one-letter, control, and ignored repetition conditions are presented in Figure 1 and Table 1.

A 2 × 3 [group (smoking vs. sham) by trial type (one-letter, control, ignored repetition)] mixed-model analysis of variance (ANOVA) was used to analyze the mean reaction time data. The main effect of group was not significant, $F(1, 34) = 0.44$, $p < 0.51$. There was a highly significant effect of trial type, $F(2, 68) = 105.2$, $p < 0.0001$, with participants responding faster on the one-letter condition relative to the two-letter condition, $F(1, 34) = 131$, $p < 0.0001$, and ignored repetition condition $F(1, 34) = 113$, $p < 0.0001$.

Of particular relevance to the present hypothesis was the significant interaction between group and trial type, $F(2, 68) = 3.50$, $p < .0356$. This effect was analyzed further with partial interactions. The interaction between group and performance on the control and ignored repetition conditions was significant, $F(1, 34) = 9.86$, $p < .0035$. Additional analysis showed that the smoking group showed a significant slowing for ignored repetition compared with control trials, $F(1, 17) = 8.07$, $p < .0113$. In contrast, no such slowing was apparent for the sham group $F(1, 17) = 1.9$, $p < .19$. Thus, the smoking group demonstrated the negative priming effect, believed to be due to the suppression of unselected information on the previous trial, whereas the sham group did not.

There was an additional interaction between group and performance on the one-letter and control condition, $F(1, 34) = 5.74$, $p < .022$. This was due to the sham group exhibiting a greater slowing on the control condition (45-ms increase), $F(1, 17) = 99.85$, $p < 0.0001$, in comparison to the smoking group (29-ms increase), $F(1, 17) = 39.75$, $p < 0.0001$.

A further 2 × 2 × 2 group (smoking vs. sham) trial type (control vs. ignored repetition) session (first half vs. second half) mixed-model ANOVA was conducted on the first and second halves of the experimental session for the control and ignored repetition trials. This was conducted in order to examine whether there were any changes in the negative priming effect for each group from the first half (Lists 1–11) to the second half (Lists 12–22) of the experimental session.

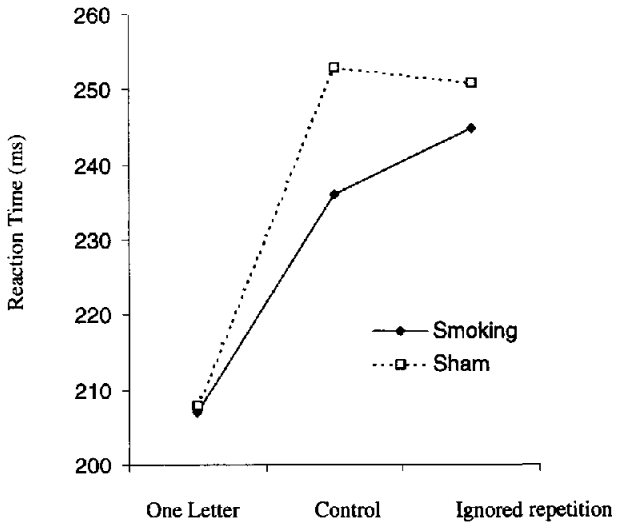

Figure 1. The effects of smoking on levels of negative priming.

Table 1
Mean Reaction Times for Each Condition

Condition	Smoking group		Sham Group	
	M	*SE*	*M*	*SE*
Ignored repetition	245	8	251	7
Control	236	10	253	9
Single letter	207	10	208	8

The results of this analysis for the main effects of group, trial type, and the interaction between group and trial type have already been reported in the previous analyses. In addition, the analysis showed a highly significant main effect of session, $F(1, 34) = 30.75$, $p < 0.0001$, which reflected a reduction in reaction time for the second half of the session ($M = 231$ ms) relative to the first half of the session (261 ms). Clearly, this was a practice effect, with participants becoming faster as the task progressed. However, the effect of session did not interact with group, $F(1, 34) = 0.05$, $p < 0.8$, with both the smoking and sham groups responding more quickly on the second half of the session (smoking, first half = 255 ms, second half = 227 ms; sham, first half = 267 ms, second half = 236 ms).

In addition, the two-way interaction between trial type and session did not reach significance, $F(1, 34) = 1.90$, $p < 0.17$. Thus, there was no effect of session on levels of negative priming. Finally, the three-way interaction among session, group, and trial type was not significant, $F(1, 34) = 0.12$ $p < 0.72$. Therefore, the effect of smoking on levels of negative priming did not vary significantly from the first half of the session to the second half of the session.

Percentage of errors for each condition are presented in Table 2. The error rates in task performance were negligible (less than 1%) and were not analyzed.

Discussion

In support of the experimental predictions, a significant interaction between smoking status and negative priming was obtained, with smoking resulting in a significant negative priming effect in contrast to an absence of negative priming in the sham group. This result indicates that smoking can increase the level of suppression of distracting information and also suggests a mechanism by which smoking may reduce interference from irrelevant sources of information.

Table 2
Mean Percentage Error Rates for Each Condition

Condition	Smoking group	Sham group
Ignored repetition	0.6	0.5
Control	0.4	0.3
Single letter	0.1	0.1

The lack of a negative priming effect for the sham group was unexpected and it conflicts with previous work that has demonstrated significant negative priming effects using this task (Tipper & Cranston, 1985). One potential explanation for this effect is that the sham group, but not the smoking group, may have recognized the critical relationship between the target and distractor across the ignored repetition trials. This would have enabled the sham participants to prepare for the arrival of the stimulus ignored on the previous trial and eliminated the negative priming effect. If this were the case, however, then there should have been evidence of this across the first and second halves of the experiment, with the sham group showing a reduction in negative priming in the second half relative to the first half. As there was no evidence of this in the analysis of the negative priming effect for each half of the experimental session, it appears unlikely that the lack of negative priming in the sham participants was due to their recognizing the relationship between the trials in the ignored repetition condition. Furthermore, as noted by Hasher, Zacks, Stoltzfus, Kane, & Connelly (1996), this procedure tends to produce small negative priming effects of approximately 10 ms, and so it is possible that with an alternative task, which results in higher levels of negative priming, that the sham group would have shown a significant negative priming effect. Finally, it is also possible that the lack of negative priming in the sham group was caused by the minimal smoking deprivation impairing selective attention and thus reducing negative priming, with smoking reinstating the negative priming effect in the smoking group.

There was no difference between the performance of the sham and smoking groups on the one-letter condition, which suggests that there were no intrinsic differences between the groups in terms of motivation or information processing speed. However, the sham group showed a larger increase in reaction time than the smoking group for the control condition relative to the one-letter condition. One possible interpretation of this effect is that it was due to the smoking group overcoming the interfering effects of the distracting letter more effectively than the sham group, perhaps by applying more suppression to the distractor. This would support the idea that smoking increases negative priming and thus enables participants to inhibit distracting sources of information more effectively. Unfortunately, however, no direct measure of interference was used in the present experiment. A true measure of distractor interference would be obtained by comparing responses to a condition where a target and distractor have the same identity (response compatible) with a condition where they have different identities (response incompatible). Interference would be measured as any increase in response latency in the response incompatible condition (Fox, 1994). Without a direct measure of distractor interference, it is therefore not possible to determine whether this effect was due to the smoking group overcoming interference more than the sham group. For example, it could be the case that the smoking group simply segregated and encoded the target more quickly when there were two letters, rather than overcame response interference from distractor more effectively. The issue of whether smoking both increases negative priming and reduces interference can be addressed in future work.

The enhanced negative priming effect in the smoking group, relative to the sham group, supports the hypothesis that the cholinergic system has a role in maintaining selective attention and that nicotine may modulate attentional filtering by increasing the suppression of distracting sources of information. Consistent with this view are studies that have shown a nicotine-induced enhancement in PPI (Kumari et al., 1996), a reduction in distractability in monkeys after administration of a nicotinic agonist (Prendergast et al., 1998), and increases in distractability after either cholinergic lesions (Muir et al., 1995) or blockade of the cholinergic system by scopolamine (Jones & Higgins, 1995).

It has been suggested that the cholinergic system modulates attention by enhancing the signal-to-noise ratio of synaptic processing at certain cortical regions, with optimal acetylcholine transmission producing optimal signal-trace detectability (Drachman & Sahakian, 1980). According to this account, the synaptic coding of relevant signals has to be discriminated from the coding of irrelevant signals and background neuronal activity. It can be argued that an enhancement of the signal-to-noise ratio by acetylcholine could involve, in addition to the enhancement of the signal, the inhibition or attenuation of the noise (Everitt & Robbins, 1997). In support of this, evidence suggests that activation of the cholinergic system can enhance the intensity of strong afferent stimuli (Edeline, Hars, Maho, & Hennevin, 1994; Krnjevic, Pumain, & Renaud, 1971) and that it may do this, in part, by attenuating cortical responses to weaker stimuli (Metherate & Ashe, 1995). Thus, it is possible that stimulation of the cholinergic system by nicotine results in an amplification of cortical responses to attended stimuli, which is partly caused by the inhibition of cortical responses to unattended stimuli. This proposal parallels Houghton & Tipper's (1996) account of negative priming, which suggested that suppression of the distractor stimulus may be implemented in the cortex by the inhibition of those neural regions responsible for processing distracting information.

Researchers have also proposed that the cholinergic system may modulate selective attention through an action on the thalamus (Steckler & Sahgal, 1995). Much evidence supports this proposal. First, of the brain regions that receive a strong cholinergic projection from the nbM and that are involved in attention (e.g., the prefrontal cortex, thalamus, and parietal cortex), it is the thalamus that has been most strongly implicated in attentional filtering (Laberge, 1995). Second, the pulvinar nucleus of the thalamus, which may have a role in negative priming (Houghton & Tipper, 1994), attentional filtering (Laberge, 1995), and covert orienting (Posner & Peterson, 1990; Robinson & Peterson, 1992), has a relatively high concentration of nicotinic receptors (Rubboli et al., 1994). Finally, results from functional brain imaging studies suggest that cholinergic blockade by scopolamine may impair attention by affecting thalamic functioning (Cohen, Gross, Semple, Nordahl, & Sunderland, 1994). Thus, it is feasible that the effects of smoking on attentional

inhibition obtained in this study may have been due to the actions of nicotine on the thalamus.

It can be noted, however, that some theorists have proposed noninhibitory accounts of negative priming (Lowe, 1985; Neill, Valdes, Terry, & Gorfein, 1992), and thus an increase in negative priming may not necessarily reflect an increase in distractor inhibition. Although this is an important consideration, it can also be emphasized that, regardless of the exact cause of negative priming, increases in negative priming are associated with efficient selection, whereas reductions in negative priming are often found in participant populations with deficits in attentional processing (Fox, 1995; Neill et al., 1992). Thus, work indicates that negative priming has adaptive consequences, reflecting efficient information processing (Fox, 1995).

Finally, it may be the case that the high incidence of smoking in schizophrenics, who exhibit deficits in attentional filtering and reduced levels of negative priming (Beech, Powell, McWilliam, & Claridge, 1989) and PPI (Braff, Grillon, & Geyer, 1992), is partly due to the schizophrenics who smoke as a form of self-medication to reduce their attentional impairments (Goff, Henderson, & Amico, 1992; McEvoy & Lindgren, 1996; Stevens, Kern, Mahnir, & Freedman, 1998). That is, schizophrenics may smoke excessively because smoking enhances levels of negative priming and thus reduces attentional deficits.

To our knowledge, this study is the first demonstration that cigarette smoking results in a larger negative priming effect in smokers relative to smokers who sham smoke. This result is concordant with other work indicating that the cholinergic system may enhance attentional filtering through an effect on inhibitory mechanisms in attention. As first suggested by Warburton (1972, p. 457), it appears that activation of the cholinergic system is "involved in the facilitation of stimulus inhibition and thus selection of relevant stimuli."

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