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Nicotine and Smoking: A Review of Effects on Human Performance

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The purpose of this review was to examine the effects of nicotine administration and cigarette smoking on human performance to clarify the role of such effects in controlling smoking. The results of 101 studies (129 experiments) published in scientific journals from 1970–1993 were reviewed. In nonabstinent smokers and nonsmokers, nicotine enhanced finger tapping and motor responses in tests of attention; cognitive functioning was not reliably enhanced. It is unlikely that these limited performance-enhancing effects of nicotine play an important role in the initiation of cigarette smoking. In contrast, data from abstinent smokers support the conclusion that nicotine deprivation functions to maintain smoking in nicotine-dependent persons, in part, because nicotine can reverse withdrawal-induced deficits in several areas of performance.

Of the 46 million adults in the United States who smoke cigarettes (Centers for Disease Control, 1993), more than 90% are interested in quitting and approximately one in three (15–20 million) attempt cessation each year (Fiore, 1992; Pierce, Fiore, Novotny, Hatziandreu, & Davis, 1989). However, more than 90% of these cessation attempts are short lived; most individuals relapse during the first few weeks after cessation when the nicotine withdrawal syndrome is most intense (Hughes, Higgins, & Hatsukami, 1990; Kottke, Brekke, Solberg, & Hughes, 1989).

Difficulty concentrating and impaired performance are components of the nicotine withdrawal syndrome (American Psychiatric Association, 1987; U.S. Department of Health and Human Services [USDHHS], 1988), and performance has been shown to be impaired within 4 to 12 hr of smoking cessation (Gross, Jarvik, & Rosenblatt, 1993; Hatsukami, Fletcher, Morgan, Keenan, & Amble, 1989; Heimstra, Fallesen, Kinsley, & Warner, 1980; Snyder, Davis, & Henningfield, 1989). Thus, a decline in performance resulting from nicotine deprivation is generally considered a relapse factor in persons attempting to quit smoking and as a factor in the maintenance of smoking in nicotine-dependent individuals.

The purpose of this review is to examine the effects of nicotine administration and cigarette smoking on a wide range of human behavior to clarify the role of nicotine-induced performance effects as determinants of the initiation and maintenance of smoking behavior. Studies conducted with nicotine-deprived smokers are most relevant to the maintenance of smoking, whereas studies in which nonsmokers are administered nicotine may be more relevant to the initiation phase of smoking. Because effects of nicotine can differ in the maintenance and initiation phases of smoking, primarily as a function of tolerance (USDHHS, 1988), a distinction between the performance effects of nicotine under conditions of nicotine deprivation and no deprivation is emphasized throughout the review.

An overview of nicotine dependence and relevant pharmacology precedes a critical analysis of methodological issues and summary of results of

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the entire literature. Because of a lack of methodological rigor in the majority of reviewed studies, a secondary analysis follows in which we summarize and draw conclusions from studies conducted using rigorous experimental methodology (i.e., placebo control and blind procedures). Our article concludes with a behavioral pharmacological perspective, emphasizing the complex interactions that must be explored before a complete understanding of the effects of nicotine on human performance is reached.

Nicotine Dependence and Performance Effects

The offices of the U.S. Surgeon General, the World Health Organization, and other major national and international health advisory organizations have concluded that the pernicious use of tobacco products is largely due to the high dependence potential of nicotine (American Psychiatric Association, 1987; Royal Society of Canada, 1989; USDHHS, 1988, 1990; World Health Organization, 1992). Consistent with these organizations, we have defined drug dependence or drug addiction[1] as a pattern of behavior focused on the repetitive and compulsive seeking and taking of a psychoactive drug (Heishman & Henningfield, 1992). Numerous animal and human studies have demonstrated that nicotine is psychoactive and reinforces behavior leading to its self-administration. Thus, the majority of researchers in the field have concluded that nicotine is a drug of dependence and the primary controlling variable in cigarette smoking (USDHHS, 1988).

However, others have rejected the evidence that nicotine is a dependence-producing or addictive drug (Robinson & Pritchard, 1992; Warburton, 1989). Alternatively, they argue that people smoke cigarettes because smoking provides psychopharmacological benefits, such as increased mental alertness and reduced tension, not because they are dependent on nicotine (Warburton, 1990; Warburton, Revell, & Walters, 1988; Wesnes & Warburton, 1983b). However, these two views are not mutually exclusive. Drug dependence is a complex, multidetermined behavioral phenomenon involving the myriad effects (e.g., reinforcing, harmful, pleasurable, beneficial) of a psychoactive drug that are manifested in an individual interacting with the environment. Thus, a person may continue to smoke because they are nicotine dependent, because they perceive beneficial behavioral and psychological effects from smoking, because smoking has become conditioned to daily activities, because smoking strengthens belonging to a particular group, and for many other reasons. Similar conclusions have been reached for other drugs of dependence, such as cocaine, heroin, and alcohol (USDHHS, 1988). This review will focus on the performance effects of nicotine and smoking and the possible role these effects play in controlling smoking behavior.

In nicotine-dependent individuals, nicotine abstinence can produce disruptions in concentration and performance that can be reversed by cigarette smoking or other forms of nicotine delivery (USDHHS, 1988). This ability of nicotine to reverse deprivation-induced performance decrements to predeprivation levels should not be considered an absolute enhancement of performance. Rather, it reflects the restoration of normal (drug-dependent) functioning from a state of abstinence-induced disequilibrium. In our view, absolute enhancement is demonstrated only when nicotine produces a statistically significant facilitation of performance over baseline levels in nonsmokers or in nicotine-dependent persons who are not nicotine deprived. As this review indicates, few studies have been designed adequately to determine whether nicotine can function to enhance absolutely human performance.

Pharmacology of Nicotine

An understanding of the behavioral effects of a psychoactive drug requires knowledge of its pharmacological profile, which reflects the drug's chemical structure, dosage form, and route of administration. Important aspects of a drug's profile include

[1] The terms *dependence* and *addiction* are generally used synonymously. Drug dependence has become the preferred term in the scientific community, whereas drug addiction is often used in writings intended for general audiences. Thus, being dependent on nicotine is the same as being addicted to nicotine. Drug dependence should not be confused with physical dependence. The development of physical dependence on a drug may be one component of the drug dependence syndrome, but it is neither a necessary nor sufficient condition to maintain the behavioral pattern defined as drug dependence (cf. Jaffe, 1990; USDHHS, 1988).

its pharmacokinetics, pharmacodynamic effects, and ability to produce tolerance and physical dependence. The behavior being measured is influenced by all of these factors.

Dosing and Pharmacokinetics

The effects of nicotine depend critically on the dose administered and the speed with which it is delivered (Benowitz, 1990; Henningfield & Woodson, 1989; USDHHS, 1988). Cigarette smoking delivers nicotine in discrete doses that produce rapid increases in plasma nicotine concentration; arterial levels may be 10 times greater than those of venous blood (Benowitz, 1990; Henningfield, Stapleton, Benowitz, Grayson, & London, 1993). Although not essential to sustain nicotine tolerance and physical dependence, the rapid delivery of nicotine achieved through smoking appears to enhance the addictive effects of nicotine (Benowitz, 1990; Henningfield & Keenan, 1993) and could be an important determinant of the effects of smoking on performance.

Control over nicotine dosing through cigarette smoking is complicated and has rarely been achieved as investigators intended (Henningfield, 1984; USDHHS, 1988). Smokers typically extract 1–4 mg of a total of 7–9 mg of nicotine per cigarette (Benowitz, Jacob, Denaro, & Jenkins, 1991), resulting in almost no direct relationship between estimated nicotine yields of cigarettes and nicotine intake (Benowitz et al., 1983; Gori & Lynch, 1985; Russell, Jarvis, Iyer, & Feyerabend, 1980).

Nicotine delivered through cigarette smoking has an initial distribution half-life in plasma of 10–20 min, followed by an elimination half-life of 2–3 hr (Benowitz et al., 1991; Benowitz, Jacob, Jones, & Rosenberg, 1982). In daily smokers, nicotine plasma concentration reaches a peak of 10–40 ng/ml in late afternoon, declining to 5–10 ng/ml after overnight abstinence (Benowitz, Kuyt, & Jacob, 1982). Thus, daily smokers are constantly exposed to nicotine. Nicotine administered through other delivery systems (tablet, polacrilex gum, nasal spray, and injection) also has an elimination half-life of about 2 hr, but these delivery systems do not produce the rapid and large increase in arterial nicotine concentration observed with cigarettes.

These other forms of nicotine delivery offer pharmacokinetic advantages and disadvantages compared with cigarettes. Orally ingested forms of nicotine provide a convenient means of blinding the dosing procedure, but absorption is erratic and about 70% of absorbed nicotine is metabolized in its first pass through the liver (USDHHS, 1988). Nicotine polacrilex gum is absorbed effectively buccally, is conveniently administered, allows for blinding, and produces dose-related effects, but precision in dosing can be reduced by failure to control chewing technique and dietary factors (Henningfield, Radzius, Cooper, & Clayton, 1990; Nemeth-Coslett, Benowitz, Robinson, & Henningfield, 1988). Absorption of nicotine from sprays and aerosols occur through the nasal or oral mucosa; these systems allow precise delivery of measured nicotine doses under double-blind conditions (Perkins et al., 1986; Pomerleau, Flessland, Pomerleau, & Hariharan, 1992; Sutherland, Russell, Stapleton, Feyerabend, & Ferno, 1992).

Pharmacodynamics

The effects of nicotine are complex. Peripheral effects are mediated through nicotinic receptors at autonomic ganglia, sensory nerve endings, neuromuscular junctions, and adrenal medulla (Clarke, 1987; USDHHS, 1988). In the central nervous system (CNS), the psychoactive, reinforcing, and possibly cognitive effects of nicotine are mediated by several populations of nicotinic receptors that appear to underlie nicotine-induced changes in electrocortical activity and regional brain metabolism (London & Morgan, 1993). Thus, nicotine could alter cognition and performance through the CNS but effects at peripheral sites could also be involved. For example, effects of nicotine on finger tapping and arousal could be mediated through skeletal muscle and hormonal systems, respectively. To determine that an effect of nicotine is due to its effects in brain would require pretreatment with central and peripheral nicotinic antagonists. Only one experiment in this review administered nicotine after antagonist (mecamylamine) dosing (West & Jarvis, 1986, Experiment 3).

Studies have demonstrated the development of acute and chronic tolerance to some, but not all, of nicotine's effects (USDHHS, 1988). Interestingly, some tolerance is lost overnight, such that the first few cigarettes of the day produce greater subjective reports of strength and greater increases in heart rate compared with cigarettes later in the

day (Henningfield, 1984; West & Russell, 1987). Physical dependence also develops with repeated nicotine administration, as documented by the emergence of an abstinence syndrome following abrupt cessation of cigarette smoking (Hatsukami, Hughes, Pickens, & Svikis, 1984; Hughes et al., 1990) and use of smokeless tobacco products (Hatsukami, Anton, Keenan, & Callies, 1992; West & Russell, 1985).

Literature Survey Methods

Articles on the performance effects of nicotine appeared in the literature as early as the 1920s and sporadically through the 1960s (for reviews, see Heimstra, Bancroft, & DeKock, 1967; Sherwood, 1993; Wesnes & Warburton, 1983b). During the 1970s, research in this field increased and continues to be an area of considerable interest (Jarvik, 1991; West, 1993). This review encompasses research published in the journal literature from 1970 to 1993.

The literature was compiled by searching computer databases (e.g., Medline, Current Contents), references of published reviews, and bibliographies of our personal reprint files, which include more than 8,000 articles on smoking and nicotine. The 101 studies (129 experiments) in this review met two criteria. First, articles were published in English in journals;[2] book chapters, technical reports, conference proceedings, and abstracts were excluded. Second, studies involved the administration of nicotine through smoking or other delivery methods during experimental sessions and the postdosing measurement of one or more variables related to human performance. Studies that did not involve nicotine administration during at least one experimental condition were excluded because nicotine, as an independent variable, was not manipulated and recent nicotine exposure was not controlled. We defined human performance broadly to assess nicotine's effects across a wide range of behavior, from sensory and motor abilities through complex cognitive functioning. Excluded were studies assessing the effect of nicotine on physiological functioning or on behavior not related to performance and studies manipulating performance testing as a stressor to investigate the interaction with nicotine.

Methodological Problems and Limitations

There are fundamental methodological problems in the majority of reviewed studies that limit meaningful interpretation of results. A brief critique of these methodological limitations will provide an important perspective on the research findings summarized in the next section.

Small Sample Size

Studies conducted with a small number of subjects may have relatively low statistical power, and hence, the probability of concluding incorrectly that the independent variable had no effect (Type II error) is increased. However, it is important to remember that the power of a statistical test is determined not only by sample size but also by Type I error rate (alpha level) and expected effect size in the population (Rossi, 1990). To determine whether sample size was related to conclusions (correct or incorrect) of no effect of smoking or nicotine, we classified the reviewed experiments according to sample size, and the number of experiments reporting no effect on all dependent measures was noted. For experiments with n = 1–10 (18% of total), 17% reported no effect; for n = 11–20 (40%), 34% reported no effect; for n = 21–30 (14%), 39% reported no effect; for n = 31–40 (11%), 45% reported no effect; for n = 41–50 (4%), 20% reported no effect; and for n > 50 (14%), 33% reported no effect. Thus, it would appear that experiments with small sample size did not result in a disproportionate number of null outcomes.

Lack of Placebo Control

One of the hallmarks of sound experimental design is the use of a placebo control, which allows,

[2] Our original intent was to review only articles published in peer reviewed journals. However, on consulting *Uhlrich's International Periodicals Directory* (1991–92), which, according to the National Library of Medicine is the definitive source for such information, we discovered numerous instances of journals, known to be peer reviewed, listed as not being peer reviewed. Thus, we have included all relevant journal articles, a minority of which may have been published in journals that are not peer reviewed.

but does not guarantee, a study to be conducted under blind conditions. In clinical pharmacology, this means that appropriate control measures are used during drug administration to reduce the likelihood of subject, or experimenter, or both knowing the nature of the treatment. Unfortunately, the availability of an effective placebo tobacco cigarette is limited, although some studies have experimented with herbal or lettuce leaf cigarettes (Andersson & Post, 1974; Wesnes & Warburton, 1983a; West & Hack, 1991) or denicotinized cigarettes having a nicotine yield of less than 0.1 mg (Colrain, Mangan, Pellett, & Bates, 1992; Hasenfratz, Baldinger, & Bättig, 1993; Houston, Schneider, & Jarvik, 1978; Robinson, Pritchard, & Davis, 1992; Warburton, Rusted, & Fowler, 1992). As a result, 74% of experiments in this review involved subjects smoking cigarettes in at least one experimental session and not smoking in other sessions. These studies did not use a placebo smoking condition and were not conducted in a single- or double-blind manner. Without an effective placebo condition, results are difficult to interpret, and data are clearly confounded by subject and experimenter knowing whether or not nicotine was being administered.

Use of Cigarettes to Deliver Nicotine

The use of tobacco cigarettes to deliver nicotine generalizes to the environment beyond the laboratory; however, as discussed, cigarette smoking is an imprecise method of nicotine dosing. It is well known that people differ in their puffing and inhalation strategies and are able to adjust their smoking behavior to compensate for actual or perceived changes in the nicotine yield of cigarettes (Gust & Pickens, 1982; Herning, Jones, Bachman, & Mines, 1981). Thus, use of cigarettes varying in nicotine yield (e.g., Mangan & Golding, 1983; Williams, 1980) does not guarantee effective dose manipulation. Subjects smoked according to a paced or timed procedure in 28% of experiments involving cigarette smoking; however, ad lib smoking was used in the majority (70%) of studies. Zacny, Stitzer, Brown, Yingling, and Griffiths (1987) have shown that experimental control over several elements of smoking topography is necessary to deliver nicotine doses precisely with tobacco cigarettes.

In the absence of precise dosing capabilities, knowledge of plasma nicotine concentration would provide information concerning the delivered dose. However, only one study (Hindmarch, Kerr, & Sherwood, 1990) reported plasma nicotine levels, and this study administered nicotine polacrilex, which is a delivery system that can produce orderly dose-related effects (Nemeth-Coslett et al., 1988; Parrott & Craig, 1992). Future studies using tobacco cigarettes to deliver nicotine that measure plasma nicotine concentration will allow the correlation of performance changes with the delivered dose. However, for the experimental analysis of the effects of nicotine, we recommend the use of delivery systems that provide a measured dose of nicotine when administered in a standardized manner, such as polacrilex, nasal spray, or injections.

Use of Cigarette Smokers

As discussed, daily smokers are never completely nicotine free. In studies in which smokers are required to smoke, a variable dose of nicotine is delivered to a person with an unknown preexisting plasma nicotine concentration. Such imprecise dosing methodology is likely to produce inconclusive results and should be avoided in future studies.

A related issue that makes data interpretation difficult is the use of smokers who are required to be abstinent for some time before experimental sessions. Of the 113 experiments in which smokers were tested, 77 indicated the duration of tobacco deprivation, 24 did not state either deprivation duration or whether smokers were deprived, and 12 experiments tested nonabstinent smokers only. Of the experiments reporting deprivation duration, subjects were abstinent less than 4 hr in 35%, 4–8 hr in 5%, and more than 8 hr (typically overnight) in 60%. Smokers who are tobacco deprived for 4–8 hr are in early nicotine withdrawal and may be experiencing impaired concentration and performance. When nicotine-deprived subjects are administered nicotine, any observed improvement in performance may reflect a return to predeprivation levels of performance. This is not a new interpretation (Hindmarch et al., 1990; Hughes, 1991; USDHHS, 1988; West, 1990); however, it bears repeating because with rare exceptions (Parrott & Roberts, 1991; Peeke & Peeke, 1984), data are not discussed in light of this

confounding variable. To address this problem, future studies testing deprived smokers should measure and report predeprivation baseline data.

Effects of Nicotine and Smoking on Human Performance

The methods and results of 101 studies (129 experiments) are summarized according to the primary behavior necessary to perform the various tests as follows: sensory abilities (Table 1), motor abilities (Table 2), attentional abilities (Table 3), and cognitive abilities (Table 4). Such a classification scheme is somewhat arbitrary because some performance tests require several skills, and sensory abilities are necessary for all performance. However, compared with a classification based on specific performance tests, for example, the approach used here allows a broader focus on behavior, which is our intent.

In each table, studies are arranged in chronological order. An experiment was classified as double-blind only if it was so stated in the article. If an experiment appeared to be conducted in a double-blind manner but was not stated as such, it was classified as a single-blind experiment. Results are summarized in the tables as main findings that were significant according to the statistical test used by the authors of the study; nonsignificant results are indicated as "no effect" or a "trend."

In the following narrative analysis of each behavioral function, we have categorized experiments as administering nicotine, in some form, to subjects under conditions of nicotine deprivation or no deprivation (including nonsmokers who were administered nicotine). Some studies did not indicate whether smokers were abstinent before testing and are omitted from the narrative because without this basic information, data are uninterpretable. Within the two categories, the results of each experiment were classified as demonstrating enhancement, impairment, or no effect of nicotine. However, such a discrete classification was difficult because in some experiments, a combination of the three outcomes was reported.

Sensory Abilities

Table 1 summarizes the results of six studies (10 experiments) that measured critical flicker frequency (CFF) threshold. CFF is generally regarded as a measure of CNS functioning; an increase in threshold frequency indicates increased cortical and behavioral arousal (J. M. Smith & Misiak, 1976). Table 1 also includes seven studies that measured other visual and auditory abilities.

Critical Flicker Frequency

Nicotine deprivation. In 5 of the 10 experiments, subjects were tobacco abstinent (overnight in four of five experiments) when CFF was measured. All experiments reported that smoking or nicotine increased CFF threshold or enhanced sensitivity of detection (Leigh, 1982, Experiments 1, 2, and 4; Sherwood, Kerr, & Hindmarch, 1992; Waller & Levander, 1980). The magnitude of maximal increase was 3–9% of control values in studies reporting data necessary for such calculations.

No deprivation. In 6 of the 10 experiments, subjects were either smokers who were not tobacco deprived before experimental sessions or were nonsmokers who were administered nicotine. Two experiments reported that smoking increased CFF threshold (Leigh, 1982, Experiments 3 and 4), whereas four experiments found nicotine had no effect on CFF (Hindmarch et al., 1990, Experiments 1 and 2; Jones, Sahakian, Levy, Warburton, & Gray, 1992 [nonpatient control groups]; Kerr, Sherwood, & Hindmarch, 1991). In the only study that directly compared tobacco-deprived and nondeprived smokers, Leigh (1982, Experiment 4) reported that predrug CFF threshold values were lower in deprived than in nondeprived smokers, suggesting a nicotine deprivation effect, and that smoking increased CFF threshold in both groups.

Other Sensory Abilities

Nicotine deprivation. Three of seven studies were conducted with smokers who were abstinent 1–12 hr. Tong, Knott, McGraw, and Leigh (1974) reported that cigarette smoking enhanced visual discrimination in a two-flash fusion test, and Tong, Booker, and Knott (1978) found that smoking produced an underestimation of velocity and time of visual stimuli in apparent motion. These results are suggestive of nicotine-induced behavioral or cortical arousal. However, Calissendorff (1977)

reported that smoking two cigarettes impaired the course of visual adaptation to the dark.

No deprivation. The only study to test nondeprived smokers included abstinent smokers and nonsmokers as control groups, both of which did not smoke (Fine & Kobrick, 1987). They reported no effect of smoking on visual contrast sensitivity and that nonsmokers exhibited superior sensitivity in certain conditions compared with smokers.

Summary of Sensory Abilities

Seven of the eight experiments conducted with abstinent smokers in Table 1 reported that cigarette smoking enhanced sensory abilities. However, none measured predeprivation performance, so a conclusion of absolute enhancement from nicotine is unclear. In contrast, two of seven experiments found that smoking or nicotine enhanced sensory abilities in nondeprived smokers or nonsmokers. Thus, when administered to abstinent smokers, nicotine may mitigate the performance deficits associated with nicotine deprivation. Unfortunately, the lack of predeprivation baseline data precludes drawing any firm conclusion about the effects of nicotine from such studies. As stated, the most unambiguous data concerning the performance effects of nicotine are found in studies testing nonabstinent smokers or nonsmokers. On this basis, the enhancing effect of nicotine on sensory abilities was not reliably observed and of limited generality.

Motor Abilities

Table 2 summarizes six studies (10 experiments) that measured finger tapping and eight studies (10 experiments) that assessed hand steadiness or tremor. These measures are generally thought to reflect purely motor abilities.

Finger Tapping

Nicotine deprivation. In 4 of the 10 experiments, smokers were abstinent for 1–12 hr before testing. All but one study reported increased finger-tapping rate in some or all subjects after smoking (O'Connor, 1986b; Roth & Bättig, 1991) or nicotine nasal spray (Perkins et al., 1990). Valeriote, Tong, and Durding (1979) found no effect of smoking on either finger- or foot-tapping rate in 4-hr abstinent smokers.

No deprivation. No study administered nicotine to nonabstinent smokers. Nonsmokers were administered nicotine through nasal spray or subcutaneous injection in seven experiments. West and Jarvis (1986) conducted five experiments and found that nicotine increased finger-tapping rate in all subjects. Perkins et al. (1990) reported increased tapping rate in 7 of 10 nonsmokers. Jones et al. (1992) reported that nicotine injections increased tapping rate in nonpatient control groups, which comprised smokers (abstinence not indicated) and nonsmokers. Across these three studies, the magnitude of the nicotine-induced increase in tapping rate was 4–6% of control values.

Hand Steadiness and Tremor

Nicotine deprivation. Smokers were abstinent for 2–12 hr in 9 of the 10 experiments; two studies did not indicate the length of deprivation. Five experiments reported that smoking or nicotine impaired performance by either decreasing hand steadiness (Frankenhaeuser, Myrsten, & Post, 1970; D. L. Smith, Tong, & Leigh, 1977) or increasing hand tremor (Shiffman et al., 1983, Experiments 1, 2, and 3). O'Connor (1986b) found an increase in hand steadiness after smoking that was not replicated in a subsequent session, and Perkins et al. (1990) reported a trend toward improvement in hand steadiness. Finally, two studies reported that the effect of smoking (Emre & de Decker, 1992) and nicotine polacrilex (Zdonczyk, Royse, & Koller, 1988) did not differ from control conditions.

No deprivation. Nonabstinent smokers were not involved in any study, but two studies administered nicotine to nonsmokers. Perkins et al. (1990) reported that nicotine nasal spray produced a trend toward impaired hand steadiness, and Zdonczyk et al. (1988) found no effect of nicotine polacrilex on finger tremor in patients with essential tremor and Parkinson's disease and control subjects.

Summary of Motor Abilities

With regard to finger tapping, in three of four experiments with abstinent smokers and in all

(text continues on page 354)

Table 1
Summary of Research on Effects of Smoking and Nicotine on Sensory Abilities

Reference	Subjects	Design	Measure	Main findings
		Critical flicker frequency (CFF)		
Waller & Levander (1980)	28 male smokers	Not blind; subjects were tobacco deprived for 2 hr; subjects either smoked a preferred-brand cigarette (M = 1.4 mg nicotine) or did not smoke in two separate counterbalanced sessions; smoking was paced at three puffs per min before each of five trials	CFF measured by forced-choice, interactive task	CFF threshold was increased during smoking session; 8 subjects showing greatest increase rated themselves as extroverted and preferred smoking in high-arousal situations.
Leigh (1982)				
Exp. 1	8 male smokers	Not blind; subjects were overnight tobacco deprived; subjects smoked ad lib six cigarettes (0.1 and 1.2 mg nicotine) per 90-min session or did not smoke in combination with alcohol or placebo in six separate counterbalanced sessions; smoking occurred before and during testing	CFF measured by detection of signal (threshold flicker) from nonsignal (steady light)	Smoking alone increased sensitivity of signal detection but had no effect on response bias. Alcohol decreased sensitivity and increased response bias. There was no smoking–alcohol interaction, but the 1.2-mg cigarette and alcohol condition suggested antagonism.
Exp. 2	8 male smokers	Same as Exp. 1, except subjects assigned to smoking (n = 4) or no smoking (n = 4) conditions; subjects either smoked ad lib four 1.2-mg nicotine cigarettes or did not smoke in two separate sessions, one with alcohol and one without; testing conducted pre- and postdrug at 20-min intervals for 100 min	CFF measured by method of limits	Smoking alone increased CFF threshold, but alcohol alone and smoking combined with alcohol decreased CFF.
Exp. 3	12 men (8 smokers, 4 nonsmokers)	Same as Exp. 2; smokers were not overnight tobacco deprived; nonsmokers were administered alcohol only	Same as Exp. 2	Smoking alone increased CFF threshold, and other conditions resulted in a decrease.
Exp. 4	8 male smokers	Not blind; subjects were either overnight tobacco deprived (n = 4) or not (n = 4); subjects either smoked ad lib four 1.2-mg cigarettes or did not smoke in combination with alcohol or placebo in four separate sessions	CFF measured as in Exp. 1 and 2	Predrug CFF threshold was lower for deprived than nondeprived smokers. Smoking increased CFF threshold, and alcohol decreased CFF in both groups. No drug interaction was observed.
Hindmarch, Kerr, & Sherwood (1990)				
Exp. 1[a]	6 female smokers	Single-blind; smokers were not tobacco deprived; nicotine polacrilex gum (0, 2, and 4 mg) was chewed for 20 min in six separate randomized sessions (each dose tested twice); test battery assessed before and 0.5, 1, 2, 3, and 4 hr after each dose	CFF measured by method of limits on three ascending and three descending scales	CFF threshold was not affected by nicotine in smokers, who were not tobacco deprived. Plasma nicotine levels averaged 7.8, 12.4, and 16.1 ng/ml for 0, 2, and 4 mg of gum, respectively.
Exp. 2[a]	5 female nonsmokers	Same as Exp. 1 except nicotine doses were 0 and 2 mg and each dose was tested three times in six separate randomized sessions	Same as Exp. 1	CFF threshold was not affected by nicotine in nonsmokers. Plasma nicotine levels averaged 1.2 and 4.9 ng/ml for 0 and 2 mg of gum, respectively.
Kerr, Sherwood, & Hindmarch (1991)[a]	10 women (5 smokers, 5 nonsmokers)	Double-blind; smokers were not tobacco deprived; nicotine polacrilex gum (0 and 2 mg) was chewed for 20 min alone and in combination with caffeine or alcohol in eight separate sessions; test battery assessed before and 0.5, 1, 2, 3, and 4 hr after each dose	CFF measured by method of limits on three ascending and three descending scales	CFF threshold was not affected by nicotine alone or in combination with other drugs; however, CFF threshold averaged over drug conditions was lower for smokers than nonsmokers.

Table 1 (*continued*)

Reference	Subjects	Design	Measure	Main findings
Jones, Sahakian, Levy, Warburton, & Gray (1992)[a]	22 patients with Alzheimer's disease; 24 normal elderly controls; 24 young adult controls; men and women smokers and nonsmokers in all groups	Single-blind; presession tobacco deprivation not indicated; single doses of nicotine (0, 0.4, 0.6, and 0.8 mg) were injected subcutaneously in ascending dose order or no injection was given in seven separate sessions; test battery assessed once after injection	CFF measured by method of limits as difference between five ascending and five descending scales	Baseline CFF threshold was higher in patients with Alzheimer's disease than either control group, which did not differ. Nicotine decreased CFF threshold difference in patients; magnitude of effect was comparable for all nicotine doses. Nicotine had no effect on CFF threshold in either control group. Men and women and smokers and nonsmokers in all groups showed no difference in CFF threshold at baseline or as a function of nicotine dose.
Sherwood, Kerr, & Hindmarch (1992)[a]	13 smokers (6 men, 7 women)	Single-blind; subjects were overnight tobacco deprived; nicotine polacrilex gum (0 and 2 mg) was chewed for 20 min at three times at 1-hr intervals in two separate counterbalanced sessions; test battery assessed before and 30 min after each of the three doses	CFF measured by method of limits on three ascending and three descending scales	First dose of nicotine increased CFF threshold over predose baseline; subsequent doses maintained elevation without causing further increases. No changes observed in placebo gum condition.
		Other sensory abilities		
Tong, Knott, McGraw, & Leigh (1974)	16 men (8 smokers, 8 nonsmokers)	Not blind; subjects were tobacco deprived for at least 12 hr; smokers either smoked ad lib two 1.3-mg nicotine cigarettes or did not smoke alone and in combination with alcohol in six separate counterbalanced sessions; nonsmokers received only alcohol; 25-min test followed drug administration	Two-flash fusion: visual discrimination of successive flashes as one or two stimuli	Smoking improved visual discrimination performance and reduced the impairing effect of alcohol. In placebo conditions, nonsmokers performed better than smokers, suggesting impairment was due to tobacco deprivation.
Calissendorff (1977)	12 smokers (gender not indicated)	Not blind; subjects were tobacco deprived for 2 hr; subjects either smoked ad lib two cigarettes (nicotine content not stated) or did not smoke in two separate sessions; testing conducted before and once after each cigarette or resting period	Dark adaptation measured by adaptometer for 25 min three times each session	After smoking two cigarettes, the course of dark adaptation was impaired compared with no-smoking condition. Effect was greatest during first 10 min of adaptation period. No changes were observed after smoking one cigarette.
Tong, Booker, & Knott (1978)	16 male smokers	Not blind; subjects were tobacco deprived 1–4 hr; subjects either smoked two 1.3-mg nicotine cigarettes via a paced procedure (one puff every 30 s) or did not smoke in two separate randomized sessions; testing conducted after 10-min smoking or resting	Velocity and time estimation of apparent motion of light stimuli	Smoking produced an underestimation in velocity and time, which was most evident at the slowest stimulus speed tested.
Dengerink, Trueblood, & Dengerink (1984)	40 men (20 smokers, 20 nonsmokers)	Not blind; presession tobacco deprivation not indicated; smokers smoked ad lib one preferred-brand cigarette for 2 min and nonsmokers rested; auditory threshold at 4 kHz was measured before and after smoking and exposure to 30- or 110-dB white noise for 5 min	Auditory threshold shift after exposure to noise	Smoking reduced the threshold-increasing effect of noise compared with nonsmokers, who showed a much greater temporary shift in auditory threshold after noise exposure.
Dengerink, Lindgren, Axelsson, & Dengerink (1987)	18 men (9 smokers, 9 nonsmokers)	Not blind; presession tobacco deprivation not indicated; smokers smoked ad lib one cigarette for 5 min and nonsmokers rested; auditory threshold at 2–8 kHz was measured before and after smoking and exposure to exercise or 105-dB white noise for 10 min	Auditory threshold shift after exposure to noise	Smoking reduced the threshold-increasing effect of noise in 3–5 kHz range compared with nonsmokers; physical exercise potentiated the effect of smoking.

(table continues)

Table 1 (*continued*)

Reference	Subjects	Design	Measure	Main findings
Fine & Kobrick (1987)	12 smokers and 16 nonsmokers (for total sample, 25 men, 3 women)	Not blind; smokers were tobacco deprived for 90 min and during testing in one session and were not deprived and smoked ad lib three preferred-brand cigarettes (one every 30 min) during testing in a second session; sessions were 24 hr apart and counterbalanced	Contrast sensitivity: ability to distinguish object from background	There was no difference in contrast sensitivity between the smoking and tobacco-deprived conditions. Nonsmokers showed superior sensitivity under certain contrast conditions compared with smokers.
Long & Franklin (1989)	4 smokers (2 light and 2 heavy, gender not indicated)	Not blind; presession tobacco deprivation not indicated; subjects smoked one cigarette (nicotine content not stated) in a paced manner (one puff every 15 s) and did not smoke in each of 12 sessions; testing conducted once immediately after smoking and not smoking	Dynamic visual acuity: ability to resolve a moving target	Smoking had no effect on dynamic visual acuity. Mean resolution thresholds decreased as a function of decreasing target velocity and increasing target duration.

Note. Exp. = Experiment.
[a]Included in section on studies using rigorous methodology

seven experiments with nonsmokers, smoking or nicotine increased tapping rate. Thus, a modest, yet reliable, increase in finger-tapping rate is an absolute enhancing effect of nicotine. In contrast, nicotine's effect on hand steadiness and tremor is less clear. In nine experiments with abstinent smokers, five found impaired performance, two reported a trend toward facilitation, and two reported no effect. In nonsmokers, nicotine produced a trend toward impaired hand steadiness in one study and had no effect on tremor in another study. Thus, the evidence for nicotine to improve hand steadiness and tremor is weak. Interestingly, nicotine-induced increases in such motor activation could be interpreted as enhanced performance or not, depending on the nature of the behavioral test.

Attentional Abilities

Table 3 summarizes the results of 63 studies that measured behaviors such as searching, scanning, and detecting visual and auditory stimuli for brief or extended periods of time. Such behaviors have been defined traditionally as attention (Kinchla, 1992; Warm, 1984). Virtually all of the performance tests conducted in the studies of Table 3 involved minimal cognitive or memorial processing and a clearly measurable psychomotor or verbal response. In all of these studies, responding was measured in some temporal form, such as reaction or response time (the vast majority), time off target, or response rate. Some studies also included a measure of response accuracy when appropriate.

On the basis of the nature of the attentional process required to perform a test, we categorized Table 3 into four sections: focused, selective, divided, and sustained attention. Each of these aspects of attentional behavior are discussed in the following subsections, which begin with a definition and end with a summary of the research results.

Focused Attention

We defined focused attention as attending to one task or aspect of information for less than 10 min; most tests actually lasted less than 5 min. Seventeen studies (19 experiments) met this crite-

rion, with the majority (12 studies) measuring simple or choice reaction time. Some studies distinguished between recognition (time from stimulus onset to movement off home key) and motor (time from home to target key) components of the reaction response. Pursuit tracking, simulated driving, digit symbol substitution, and perceptual speed were assessed in the remainder of studies.

Nicotine deprivation. In 10 of the 18 experiments, smokers were abstinent for 4–18 hr before experimental sessions. Seven experiments reported that smoking or nicotine had no effect on test behavior (Frankenhaeuser et al., 1970; Knott, 1985a; Knott & Venables, 1980; Landers, Crews, Boutcher, Skinner, & Gustafsen, 1992, Experiments 1 & 2; Petrie & Deary, 1989; Valeriote et al., 1979). One study found that extroverts were faster, but introverts were slower, in reaction time after smoking (O'Connor, 1980), and two studies reported that recognition and motor reaction times were differentially affected by smoking (D. L. Smith et al., 1977) or nicotine polacrilex (Sherwood et al., 1992).

No deprivation. Nonabstinent smokers and nonsmokers were administered nicotine in six experiments. Three reported no effect of smoking (Elgerot, 1976; Knott & Venables, 1980) or nicotine polacrilex (Hindmarch et al., 1990, Experiment 2) on reaction time and perceptual speed tasks. Two experiments found that nicotine polacrilex produced faster motor reaction times (5–9% over placebo values), but had no effect on recognition time in a choice reaction time test (Hindmarch et al., 1990, Experiment 1; Kerr et al., 1991). Spilich, June, and Renner (1992, Experiment 5) found that nonabstinent smokers who smoked before testing had more collisions in a computerized driving simulator than deprived smokers or nonsmokers.

Summary of focused attention. All 10 experiments with abstinent smokers and 6 experiments with nonabstinent smokers and nonsmokers reported that smoking and nicotine either had no effect or, in two studies, impaired test performance. Five experiments found enhanced performance in some subjects or on one aspect of the test. Thus, nicotine does not reliably enhance the ability to focus attention on a single task for a brief time or enhance the psychomotor response required in such tests.

Selective Attention

Selective attention was defined as the ability to attend to a target stimulus while simultaneously ignoring irrelevant or distracting stimuli. Twelve studies (13 experiments) assessed performance on such tests; duration of the tests was generally brief, but not always indicated. Searching for a target letter amid an array of nontarget letters was investigated in 8 experiments, and the Stroop test was used in 6 studies. The Stroop test compares the time required for subjects to name the ink color of color words that are incongruent (e.g., the word red printed in blue) versus the ink color of neutral stimuli, such as noncolor words or colored squares. Typically, the incongruent task takes more time than the neutral stimulus task because the tendency to read the color word interferes with naming its ink color; the difference in time between the two tasks is considered a measure of selective attention or distractibility (Stroop, 1935).

Nicotine deprivation. Smokers were abstinent for 1–12 hr before experimental sessions in seven studies. Smoking or nicotine enhanced letter searching (Parrott & Craig, 1992; Williams, 1980; Williams, Tata, & Miskella, 1984) and Stroop test (Hasenfratz & Bättig, 1992; Landers et al., 1992, Experiment 2) performance. The enhancement was modest (5–10% of control values); however, none of these studies measured predeprivation performance. Two studies (Parrott & Roberts, 1991; Snyder & Henningfield, 1989) avoided this methodological pitfall by assessing performance of smokers during nonabstinent and abstinent states and found that smoking and nicotine functioned to restore withdrawal-induced deficits to, but not above, nonabstinent levels. Parrott and Craig (1992) also found that smoking and polacrilex had no effect on Stroop test performance.

No deprivation. Five experiments tested the effects of smoking in nonabstinent smokers or nicotine in nonsmokers. Provost and Woodward (1991) reported that polacrilex enhanced Stroop test performance in nonsmokers; however, three experiments found no effect of smoking or nicotine on letter-searching tests (Heishman, Snyder, & Henningfield, 1993; Spilich et al., 1992, Experiment 1) and the Stroop test (Wesnes & Revell, 1984, Experiment 2). Spilich et al. (1992, Experiment 2) reported that nonabstinent smokers were

(text continues on page 359)

Table 2
Summary of Research on Effects of Smoking and Nicotine on Motor Abilities

Reference	Subjects	Design	Measure	Main findings
		Finger tapping		
Valeriote, Tong, & Durding (1979)	24 male smokers	Not blind; in six separate counterbalanced sessions, subjects were either not tobacco deprived and did not smoke, tobacco deprived for 4 hr and did not smoke, or tobacco deprived for 4 hr and smoked in a paced manner (one puff per 30 s) two 1.3-mg nicotine cigarettes in combination with alcohol or placebo; testing conducted 25 min after drug administration	Finger and foot tapping; mean intertap interval of ten 10-s trials with each index finger and foot	Smoking had no effect on either finger or foot tapping. Alcohol increased intertap interval on both tests. There was no interaction between smoking and alcohol.
O'Connor (1986b)	10 smokers (gender not indicated)	Not blind; subjects were tobacco deprived for 2 hr; subjects sham smoked during testing and then smoked 0.77-mg nicotine cigarette during testing in each of two separate sessions; smoking was paced at one puff during 1-min intertrial intervals of test for total of eight puffs	Tapping rate with one finger of nonpreferred hand (mean of six 20-s trials)	Smoking increased finger-tapping rate compared with sham smoking only in 5 subjects classified as introverts. Tapping rate increased from first to second session, suggesting a practice effect.
West & Jarvis (1986)				
Exp. 1[a]	8 male nonsmokers	Single-blind; subjects received either two 2-mg doses of nasal nicotine solution (NNS) or placebo nasal solution in two separate counterbalanced sessions; tapping task performed before and 10 min after each dose	Taps per minute: time to make 300 taps on computer key with index finger of preferred hand	NNS increased finger-tapping rate in all subjects, averaging 4% over predose baseline. Placebo solution had no effect.
Exp. 2[a]	8 nonsmokers (7 men, 1 woman)	Double-blind; subjects received NNS (0.15 and 2 mg) and placebo at 2-hr intervals in single session; tapping task performed three times before and 5 min after each dose	Same as Exp. 1, except task required 200 taps	Finger-tapping rate was increased in all subjects by 2 mg NNS, but not 0.15 mg dose or placebo. Average increase was 5% over predose baseline.
Exp. 3[a]	5 nonsmokers (4 men, 1 woman)	Double-blind; subjects received 2 mg NNS before and 2 hr after 2.5 mg mecamylamine or placebo in two separate randomized sessions; tapping task performed six times before and after each NNS dose	Same as Exp. 2	Finger-tapping rate was increased in all subjects by 2 mg NNS. Mecamylamine reduced this increase compared with placebo.
Exp. 4[a]	1 male nonsmoker	Single-blind; subject received two 2-mg doses of NNS or placebo solution in two separate sessions; tapping task performed once before and 6, 9, 11, 16, 21, 26, 31, 36, 41, 46, 51, and 61 min after dose	Same as Exp. 1	Time course of rate increase from NNS showed peak at 11 min and consistent elevation over placebo to 31 min, when decline began, but remained above baseline and placebo levels at 61 min.
Exp. 5	2 male nonsmokers	Not blind; subjects received 2 mg NNS at 1-hr intervals for total of seven doses; tapping task performed twice before and after each dose	Same as Exp. 1	NNS increased finger-tapping rate after each dose in both subjects. No evidence of acute tolerance developing with later doses.
Perkins et al. (1990)[a]	20 men (10 smokers, 10 nonsmokers)	Single-blind; smokers were tobacco deprived at least 12 hr; subjects received either placebo or nicotine (15 μg/kg) nasal spray at 30-min intervals for total of four doses in two separate counterbalanced sessions; tasks performed at predrug baseline and twice after each dose	Number of key taps with index finger of preferred hand (mean of two 30-s trials)	Nicotine-induced increase in finger-tapping rate was greater in smokers than nonsmokers. Across four doses, 10 out of 10 smokers and 7 out of 10 nonsmokers showed increase after nicotine.

Table 2 (*continued*)

Reference	Subjects	Design	Measure	Main findings
Roth & Bättig (1991)	12 male smokers	Not blind; subjects were either tobacco deprived at least 1 hr and smoked ad lib one preferred-brand cigarette (smoking condition) or were deprived at least 12 hr and did not smoke (deprived condition) in two separate counterbalanced sessions; tapping task performed 3 min after smoking or rest	Mean intertap interval for 2-min tapping with index finger of preferred hand	Tapping frequency was greater in smoking condition than in deprived condition.
Jones, Sahakian, Levy, Warburton, & Gray (1992)[a]	22 patients with Alzheimer's disease; 24 normal elderly controls; 24 young adult controls; men and women, smokers and nonsmokers in all groups	Single-blind; presession tobacco deprivation not indicated; single doses of nicotine (0, 0.4, 0.6, and 0.8 mg) were injected subcutaneously in ascending dose order or no injection was given in seven separate sessions; testing conducted once after injection	Taps per minute: time to make 100 taps on space bar of computer keyboard with one finger of preferred hand	Nicotine increased finger-tapping rate in all groups. Dose-related increases seen in elderly controls and patients with Alzheimer's disease. Increase in young adults only seen with 0.6 mg nicotine. In elderly controls, nicotine produced greater increases in smokers than nonsmokers but no difference at baseline or after placebo dose.
		Hand steadiness and tremor		
Frankenhaeuser, Myrsten, & Post (1970)	9 male smokers	Not blind; presession tobacco deprivation stated but duration not indicated; subjects smoked in paced manner (two puffs per min) two cigarettes (1.3 and 2.3 mg nicotine) or did not smoke in three separate counterbalanced sessions; testing (four 20-s trials) conducted once before and three times after smoking or resting period	Number of contacts between hand-held, 1-mm stylus and side of 2.5-mm hole	Smoking impaired (increased number of contacts) hand steadiness compared with no smoking condition; effect was dose-related. Peak effect seen at 20 min postsmoking, and impairment still evident at 80-min trial.
Myrsten, Post, Frankenhaeuser, & Johansson (1972)	6 male smokers	Not blind; presession tobacco deprivation not indicated; subjects either smoked ad lib four 2.2-mg nicotine cigarettes or did not smoke during two separate counterbalanced sessions; cigarettes were smoked at 25-min intervals during 100-min reaction time test; hand steadiness assessed before and after reaction time test	Number of contacts in 20 sec between hand-held, 1-mm stylus and side of 2.5-mm hole	Smoking produced a trend toward impaired hand steadiness, whereas in the no-smoking condition, there was a trend toward improved performance compared with baseline.
D. L. Smith, Tong, & Leigh (1977)	8 male smokers	Not blind; subjects were tobacco deprived for 12 hr; subjects either smoked ad lib one cigarette (0.3 and 1.3 mg nicotine) or did not smoke in combination with caffeine (0 and 200 mg) in six separate randomized sessions; testing conducted before and twice after drug administration	Time of contact in 20 s between hand-held, 1-mm stylus and side of 2.5-mm hole	Smoking impaired hand steadiness in a dose-related manner. Caffeine alone also impaired performance, and combination of caffeine and 1.3-mg cigarette produced greatest impairment.
Shiffman et al. (1983) Exp. 1	23 smokers (11 men, 12 women)	Not blind; subjects were overnight tobacco deprived; subjects smoked two preferred-brand cigarettes (M = 1.1 mg nicotine) in paced procedure (one puff every 30 s) in single session; testing conducted before and after smoking	Hand tremor measured by current in coil induced by magnet attached to finger	Smoking produced a marked increase in hand tremor.

(table continues)

Table 2 (*continued*)

Reference	Subjects	Design	Measure	Main findings
Exp. 2	10 smokers (gender not indicated)	Same as Exp. 1	Same as Exp. 1	Power spectrum analysis showed that nicotine-induced increases in hand tremor were relatively uniform across frequency spectrum.
Exp. 3	12 smokers (6 men, 6 women)	Same as Exp. 1, except nicotine polacrilex gum (4 mg) was chewed for 20 min or one preferred-brand cigarette (1.05 mg nicotine) was smoked through paced procedure in two separate counterbalanced sessions; 8 subjects received 2 mg nicotine gum in third session	Same as Exp. 1	Nicotine polacrilex gum (4 mg) and smoking one cigarette increased hand tremor, and the effect of each treatment did not differ. The 2-mg gum did not affect tremor.
O'Connor (1986b)	10 smokers (gender not indicated)	Not blind; subjects were tobacco deprived for 2 hr; subjects sham smoked during testing and then smoked 0.77-mg nicotine cigarette during testing on each of two separate sessions; smoking was paced at one puff during 1-min intertrial intervals of test for total of eight puffs	Number of contacts and time to complete moving ring over 60.9-cm wire (mean of six 20-s trials)	Smoking decreased number of contacts compared with sham smoking but only on first session. Time to complete the test was not affected by smoking.
Zdonczyk, Royse, & Koller (1988)	10 male patients with essential tremor, 10 male patients with Parkinson's disease, 10 controls (6 men and 4 women)	Not blind; smokers in each group were tobacco deprived on day of study (testing began at 1300 hr, duration of deprivation not stated); nicotine polacrilex gum (4 mg) or regular chewing gum was chewed for 1 hr in two separate randomized sessions; finger tremor assessed before and 10, 30, and 60 min after gum	Finger tremor measured with arms outstretched, touching nose, and at rest	Nicotine had no effect on tremor in any of the patient or control groups. There were no differences between smokers and nonsmokers in each group.
Perkins et al. (1990)[a]	20 men (10 smokers, 10 nonsmokers	Single-blind; smokers were tobacco deprived at least 12 hr; subjects received either placebo or nicotine (15 μg/kg) nasal spray at 30-min intervals for total of four doses in two separate counterbalanced sessions; tasks performed at predrug baseline and twice after each dose	Time of contact between hand-held, 2-mm stylus and side of 3-mm hole (mean of two 30-s trials)	Nicotine had no significant effect on hand steadiness. However, there was a trend toward improved performance in smokers and impaired performance in nonsmokers.
Emre & de Decker (1992)	21 patients with multiple sclerosis (11 men, 10 women); 11 controls (6 men, 5 women)	Not blind; all subjects were smokers and were overnight tobacco deprived; subjects either smoked ad lib one 0.9-mg nicotine cigarette or did not smoke in two separate sessions; testing conducted before and 0, 10, 20, and 30 min after smoking or resting period	Pegboard test; needle and thread lacing; transfering beads; pricking dots on paper with needle	Scores from the four timed tests were added for a total motor performance score. At 0 and 10 min postsmoking, 16 of 21 patients showed impaired performance compared with no-smoking condition. Controls showed gradual improvement over time in smoking and no-smoking conditions, possibly because of practice.

Note. Exp. = Experiment.
[a]Included in section on studies using rigorous methodology.

slower than nonsmokers in detecting a change in a span of letters.

Summary of selective attention. Five of seven studies conducted with nicotine-deprived smokers reported that smoking or nicotine enhanced selective attention, but without predeprivation baseline data, absolute enhancement must be questioned. In two studies, smoking and nicotine reversed deprivation-induced deficits but did not enhance performance. Similarly, four of five studies with nonsmokers or nonabstinent smokers reported that nicotine had no effect or impaired test performance. Thus, in abstinent and nonabstinent smokers and nonsmokers, the evidence for nicotine to enhance selective attention is weak.

Divided Attention

The five studies (seven experiments) included in this section incorporated tests that required subjects to divide their attention between two simultaneous tasks for brief periods of time. In three studies, subjects performed a 5-min compensatory tracking task while responding to peripheral visual stimuli. The other two studies involved auditory signal detection tasks.

Nicotine deprivation. In four of the seven experiments, smokers were abstinent for 10–12 hr before testing. Sherwood et al. (1992) found that nicotine polacrilex decreased tracking errors, but had no effect on reaction time to peripheral lights. Leigh, Tong, and Campbell (1977) reported decreased error rate in a binaural signal detection test after smoking three cigarettes (Experiment 1), but no effect after one cigarette (Experiment 2). Knott (1985b) reported no effect of smoking on a test combining auditory signal detection and visual choice reaction.

No deprivation. Nonabstinent smokers and nonsmokers were administered nicotine polacrilex in three experiments. Tracking errors were reduced (4–8% of placebo values), yet reaction time was unaffected by polacrilex in two experiments (Hindmarch et al., 1990, Experiment 1; Kerr et al., 1991), and Hindmarch et al. (1990, Experiment 2) reported no effect of polacrilex on tracking or reaction time.

Summary of divided attention. The effect of nicotine on divided attention has been studied in only two experimental paradigms. Limited facilitation was reported in four of seven experiments. In tobacco-abstinent smokers, error rates were reduced by nicotine, but on only one of two tasks or after smoking three, but not one, cigarettes. Similar results were reported in nonabstinent smokers and nonsmokers, with modest nicotine-induced enhancement observed in tracking performance, but no change in reaction time.

Sustained Attention

The effect of smoking and nicotine on sustained attention has been the focus of much research; 38 studies (43 experiments) are included in this section. All of the tests used in these studies, with one exception (Jones et al., 1992), lasted at least 10 min. Although many of these tests measured aspects of focused, selective, and divided attention, we decided a separate section was appropriate because of the distinct results and processes (e.g., vigilance decrement) observed in tests of sustained attention that are not possible in tests of shorter duration (Warm, 1984). In addition to the subsections differentiating between studies testing abstinent and nonabstinent smokers or nonsmokers, two types of sustained attention testing, the rapid visual information processing (RVIP) test and vigilance tests, are discussed separately.

RVIP test. This test requires subjects to press a button when they detect three consecutive even or odd digits in a series of single digits presented visually at 600-ms intervals. In modified versions, the digit presentation rate is increased after correct responses and decreased after errors. Measures typically include rate of correct target detection (hits), errors (false alarms), digit presentation rate (modified versions), and reaction time. In the first studies reporting the effects of nicotine on the RVIP test (Wesnes & Warburton, 1983a, 1984a, 1984b), it was performed for 10 min before and for 20 min after smoking or nicotine administration. Some recent studies (e.g., Parrott & Craig, 1992) have shortened the postdrug testing to 10 min. Although frequently cited as a test that illustrates the performance-enhancing effects of smoking or nicotine (e.g., Warburton, 1992; Warburton et al., 1988), the effects of nicotine on the RVIP test are inconsistent. Of the 19 experiments that investigated the effect of smoking or nicotine in healthy subjects on the RVIP test (Table 3), 15 reported either no effect or impaired performance on some

(text continues on page 373)

Table 3
Summary of Research on Effects of Smoking and Nicotine on Attentional Abilities

Reference	Subjects	Design	Measure	Main findings
		Focused attention		
Frankenhaeuser, Myrsten, & Post (1970)	9 male smokers	Not blind; presession tobacco deprivation stated but duration not indicated; subjects smoked in paced manner (two puffs per min) two cigarettes (1.3 and 2.3 mg nicotine) or did not smoke in three separate counterbalanced sessions; testing conducted once before and three times after smoking or resting period	Choice reaction time: mean of 60 trials with 7-s intertrial interval	Smoking had no effect on reaction time. Performance improved slightly in smoking and no smoking conditions. Authors suggested effect of smoking was obscured by learning effect.
Cotten, Thomas, & Stewart (1971)	15 male smokers	Not blind; presession tobacco deprivation not indicated; subjects either smoked ad lib one 1.5-mg nicotine cigarette or did not smoke in four (each condition repeated once) separate randomized sessions; test conducted before and immediately, 5, 15, 25, 40, and 55 min after smoking or rest	Simple reaction time to light stimulus (mean of 20 trials)	Reaction time measured immediately and 5 min after smoking was slower than presmoking baseline, whereas 40- and 55-min times were faster than baseline. Reaction times during no-smoking sessions were not different from baseline.
Ashton, Savage, Telford, Thompson, & Watson (1972) Exp. 3	16 smokers (11 men, 5 women) and 17 nonsmokers (14 men, 3 women)	Not blind; presession tobacco deprivation not indicated; tracking and choice reaction time were assessed in two separate randomized sessions; smokers smoked ad lib one cigarette (nicotine content not specified) before 5-min tracking test	Pursuit rotor tracking	There was no difference between smokers and nonsmokers on the pursuit tracking test.
Elgerot (1976)	12 smokers (8 men, 4 women)	Not blind; subjects were tobacco deprived for 15 hr and during testing in one session and were not deprived and smoked ad lib (two to three preferred-brand cigarettes) during testing in a second session; sessions were 1 week apart and counterbalanced	Bourdon test of perceptual speed Proofreading of words and figures	There was no difference between the smoking and tobacco-deprived conditions for either of the tasks.
Smith, Tong, & Leigh (1977)	8 male smokers	Not blind; subjects were tobacco deprived for 12 hr; subjects either smoked ad lib one cigarette (0.3 and 1.3 mg nicotine) or did not smoke in combination with caffeine (0 and 200 mg) in six separate randomized sessions; testing conducted in three blocks of 50 trials 20 min after drug administration	Choice reaction time: mean recognition and motor time of 50 trials	Smoking produced faster recognition, but not motor, reaction times. Caffeine alone produced faster recognition and motor reaction time. The combination of caffeine and 1.3-mg cigarette produced greatest facilitation.
Valeriote, Tong, & Durding (1979)	24 male smokers	Not blind; in six separate counterbalanced sessions, subjects were either not tobacco deprived and did not smoke, tobacco deprived for 4 hr and did not smoke, or tobacco deprived for 4 hr and smoked in a paced manner (one puff per 30 s) two 1.3-mg nicotine cigarettes in combination with alcohol or placebo; testing conducted 25 min after drug administration	Pursuit rotor tracking: 1 min test with circular and triangular plates with each hand	Smoking had no effect on pursuit tracking, although alcohol decreased time on target. There was no interaction between smoking and alcohol.

Table 3 (*continued*)

Reference	Subjects	Design	Measure	Main findings
Knott & Venables (1980)	60 men (17 nonsmokers, 43 smokers)	Not blind; 26 smokers were tobacco deprived for 15–18 hr, 17 smokers were not deprived; separate groups of deprived and nondeprived smokers smoked in a paced manner (one puff every 30 s) four 1.6-mg nicotine cigarettes, received alcohol, or both; nonsmokers received alcohol or placebo; testing conducted before and once after drug administration	Simple reaction time (mean of 20 trials)	Smoking produced a nonsignificant trend toward faster reaction time in tobacco-deprived smokers compared to presmoking baseline. Smoking, alcohol, or the combination had no effect on reaction time in the other groups.
O'Connor (1980)	32 smokers (16 men, 16 women)	Not blind; subjects were tobacco deprived for 12 hr; subjects first sham smoked an unlit cigarette, then smoked in a paced manner (one puff every 4 trials) 1.45-mg nicotine cigarettes; testing conducted during sham and real smoking	Simple and choice reaction time (mean of 32 trials for each)	Subjects identified as introverts showed faster reaction time during sham smoking, whereas extroverts were faster during real smoking.
Knott (1985a)	16 female smokers	Not blind; subjects were tobacco deprived for at least 10 hr; subjects either sham smoked unlit cigarette or took four paced puffs from preferred-brand cigarette in two separate counterbalanced sessions; test performed with and without letter recall distraction at each session	Simple reaction time to light stimulus (mean of 8 trials)	Smoking had no effect on reaction time. Responding with the distractor task tended to be slower than without the distraction.
Frearson, Barrett, & Eysenck (1988)	109 smokers (70 men, 39 women)	Not blind; presession tobacco deprivation not indicated; subjects smoked ad lib one cigarette (nicotine content not specified) in two sessions; one of two reaction time tasks conducted before and once after smoking in each session	Choice reaction and odd-man-out tasks: median recognition and motor time	Smoking produced faster recognition reaction time in choice reaction task, especially evident in first 20 trials compared with last 20 trials. Motor reaction time was not reliably affected by smoking. Recognition and motor reaction time were faster after smoking in odd-man-out task.
Beh (1989)	20 female smokers and 20 female nonsmokers	Not blind; presession tobacco deprivation not indicated; subjects tested under clean and smokey air conditions in two separate counterbalanced sessions; smokers smoked ad lib one preferred-brand cigarette immediately before 5-min test	Simple reaction time: mean recognition and motor time	Recognition and motor reaction time was faster for smokers in the smokey air compared with clean air conditions. There was no difference between smokers and nonsmokers in clean air for either measure, and nonsmokers showed no difference between air conditions.
Petrie & Deary (1989)	12 smokers (6 men, 6 women)	Not blind; subjects were tobacco deprived for at least 8 hr; subjects either smoked ad lib two high-nicotine cigarettes or did not smoke in two separate counterbalanced sessions; test performed 10 min after one cigarette or rest	Digit-symbol substitution test: correct responses in 90 s	Smoking had no effect on test performance, although there was a trend toward greater number of correct responses in the smoking condition.
Hindmarch, Kerr, & Sherwood (1990)				
Exp. 1[a]	6 female smokers	Single-blind; smokers were not tobacco deprived; nicotine polacrilex gum (0, 2, and 4 mg) was chewed for 20 min in six separate randomized sessions (each dose tested twice); test battery assessed before and 0.5, 1, 2, 3, and 4 hr after each dose.	Choice reaction time: mean recognition and motor time of 20 presentations	Motor reaction time was faster with both active doses of nicotine relative to placebo but recognition time was not affected. Plasma nicotine levels averaged 7.8, 12.4, and 16.1 ng/ml for 0, 2, and 4 mg gum, respectively.
Exp. 2[a]	5 female nonsmokers	Same as Exp. 1, except nicotine doses were 0 and 2 mg and each dose was tested three times in six separate randomized sessions	Same as Exp. 1	Recognition and motor reaction times were not affected by nicotine in nonsmokers. Plasma nicotine levels averaged 1.2 and 4.9 ng/ml for 0 and 2 mg gum, respectively.

(table continues)

Table 3 (*continued*)

Reference	Subjects	Design	Measure	Main findings
Kerr, Sherwood, & Hindmarch (1991)[a]	10 women (5 smokers, 5 nonsmokers)	Double-blind; smokers were not tobacco deprived; nicotine polacrilex gum (0 and 2 mg) was chewed for 20 min alone and in combination with caffeine or alcohol in eight separate sessions; test battery assessed before and 0.5, 1, 2, 3, and 4 hr after each dose	Choice reaction time: mean recognition and motor time of 20 presentations	Motor reaction time was faster with nicotine and with nicotine-caffeine combination compared to placebo. Recognition reaction time was not affected by nicotine. No difference observed between smokers and nonsmokers.
Landers, Crews, Boutcher, Skinner, & Gustafsen (1992)				
Exp. 1	20 male smokeless tobacco users (10 heavy users, 10 moderate users)	Not blind; subjects were tobacco deprived for at least 10 hr; smokeless tobacco (2 g) or nothing (control) was administered in two separate counterbalanced sessions; 10 min after tobacco administration or rest, tests performed for 16 min, during which time tobacco remained in mouth	Simple and choice reaction time (mean of 42 trials for each)	Smokeless tobacco had no effect on any reaction time measure compared with control condition. Moderate users showed faster reaction time on all measures than heavy users.
Exp. 2	20 male smokeless tobacco users and 20 male nonusers	Not blind; smokeless tobacco users were tobacco deprived for at least 10 hr; users were administered no tobacco, one third of their average dose, average dose, or one and two thirds of average dose on four separate randomized sessions; nonusers received nothing; test performed 4 min after tobacco administration	Simple and choice reaction time (mean of 20 trials for each)	Smokeless tobacco had no effect on any reaction time measure. There were no differences between users and nonusers and no differences as a function of tobacco dose in users. Performance improved over the four sessions in both groups because of practice.
Spilich, June, & Renner (1992)				
Exp. 5	60 students (20 nonsmokers, 40 smokers)	Not blind; 20 smokers were tobacco deprived for 3 hr; 20 other smokers took 12 paced puffs (one puff every 25 s) from a 1.2-mg nicotine cigarette immediately before testing; deprived smokers and nonsmokers sham smoked an imaginary cigarette; 5-min practice trial preceded 3 to 4-min test trial	Simulated driving using computerized game: time to complete and number of collisions	Active smokers had more rear-end collisions than either deprived smokers or nonsmokers. There was no difference in time to complete the test between the three groups.
Sherwood, Kerr, & Hindmarch[a] (1992)	13 smokers (6 men, 7 women)	Single-blind; subjects were overnight tobacco deprived; nicotine polacrilex gum (0 and 2 mg) was chewed for 20 min at three times at 1-hr intervals in two separate counterbalanced sessions; test battery assessed before and 30 min after each of the three doses	Choice reaction time: mean recognition and motor time of 20 presentations	Motor reaction time was faster after second and third nicotine doses compared with predose baseline. Recognition reaction time was not affected by nicotine. No changes observed in placebo gum condition.
		Selective attention		
Williams (1980)	48 male smokers	Not blind; subjects were tobacco deprived for at least 12 hr; subjects smoked in paced manner (one puff every 30 s) one cigarette (0.6, 1.3, and 1.8 mg nicotine) or sham smoked in four separate counterbalanced sessions; testing conducted once before and after smoking	Letter cancellation: mean number of letters scanned in 3 min	Smoking increased number of letters scanned compared with sham smoking. Data suggest inverted-U function with the 1.3-mg cigarette producing the greatest effect.

Table 3 (*continued*)

Reference	Subjects	Design	Measure	Main findings
Suter, Buzzi, Woodson, & Bättig (1983)	28 male smokers	Single-blind; presession tobacco deprivation not indicated; subjects smoked ad lib either a 0.2-mg or 1.2-mg nicotine cigarette in two separate counterbalanced sessions; testing conducted before and after smoking	Stroop test: mean number correct and response time of 45 color–word stimuli	Smoking had no effect on Stroop test performance. No difference in smoking topography between the two cigarette conditions.
Wesnes & Revell (1984) Exp. 2[a]	12 nonsmokers (6 men, 6 women)	Double-blind; placebo, scopolamine (1.2 mg), or nicotine (1.5 mg) tablets administered three times at 1-hr intervals in four separate counterbalanced sessions; tablets held in mouth 5 min before being swallowed; Stroop test conducted 15 min after third dose	Stroop test: mean number of errors and response time of 200 color–word stimuli	Nicotine alone had no effect on Stroop test performance. Scopolamine slowed response time but did not increase errors. Nicotine combined with scopolamine reversed the impairment observed with scopolamine alone.
Williams, Tata, & Miskella (1984)	48 smokers (26 men, 22 women)	Not blind; subjects were tobacco deprived for at least 10 hr; subjects smoked either one 1.3-mg (males) or 1.5-mg (females) nicotine cigarette in paced manner (two puffs per min) in single session; testing conducted once before and after smoking	Letter cancellation: mean number of letters scanned in 3 min	Smoking increased number of letters scanned from presmoking baseline. Low-arousal and high-arousal smokers showed similar increases.
Snyder & Henningfield (1989)[a]	6 male smokers	Double-blind; subjects were tobacco deprived for 12 hr; nicotine polacrilex gum (0, 2, and 4 mg) administered in three separate counterbalanced sessions; testing conducted at baseline (stable practice) and once after 10-min gum-chewing period	Two- and six-letter search: time to search 20 letters for two or six target letters	Mean response time was slower than baseline after 0-mg nicotine, and 2- and 4-mg doses returned performance to baseline in both search tests. Nicotine had no effect on test accuracy.
Parrott & Roberts (1991)	20 smokers (12 men, 8 women)	Not blind; subjects were tobacco deprived for 12 hr before one session and for 1 hr before a second session; subjects smoked ad lib one preferred-brand cigarette at each session; test conducted once before and after smoking	Letter cancellation: 105 target letters in array of 1,350 letters	Tobacco deprivation (12 hr) decreased number of target detections and slowed response time on presmoking test. Performance after smoking returned to baseline levels. Performance in the 1-hr deprived condition did not change from pre- to postsmoking.
Provost & Woodward (1991)[a]	24 nonsmokers (12 men, 12 women)	Double-blind; subjects assigned randomly to either 0 or 2 mg nicotine polacrilex gum conditions; gum was chewed for 20 min, followed by three trials of Stroop test, each separated by 30 s	Stroop test: mean response time of 100 color–word stimuli	Nicotine produced faster response time over the three trials compared with placebo; nicotine—placebo difference was greatest (5 s) at Trial 3, but not significant. Response time was faster on Trial 3 compared with Trial 1 for both groups.
Hasenfratz & Bättig (1992)	20 female smokers	Not blind; subjects were tobacco and caffeine deprived for at least 12 hr; subjects received caffeine (0 or 250 mg) in decaffeinated coffee and either smoked ad lib two cigarettes or did not smoke in four separate counterbalanced sessions; Stroop test conducted before and after drug administration	Stroop test: response time of 110 numerical stimuli (easy and hard conditions)	Smoking produced faster response time in Stroop test, which was significant in hard (0-s response-stimulus interval), but not in easy (1-s response-stimulus interval) condition. Caffeine produced similar results, but the smoking-caffeine combination was not different from placebo.

(table continues)

Table 3 (*continued*)

Reference	Subjects	Design	Measure	Main findings
Landers, Crews, Boutcher, Skinner, & Gustafsen (1992)				
Exp. 2	20 male smokeless tobacco users and 20 male nonusers	Not blind; smokeless tobacco users were tobacco deprived for at least 10 hr; users were administered no tobacco, one third of their average dose, average dose, or one and two thirds of average dose on four separate randomized sessions; nonusers received nothing; tests performed 4 min after tobacco administration	Stroop test: mean number correct of 150 color–word stimuli	Smokeless tobacco users correctly identified more stimuli than nonusers, but the improvement was not dose related.
Parrott & Craig (1992)[a]	16 smokers (14 men, 2 women)	Double-blind (gum only); subjects were tobacco deprived for 12 hr; nicotine polacrilex gum (0, 2, and 4 mg) was chewed for 40 min or preferred-brand cigarette was smoked through paced procedure in four separate counterbalanced sessions; in gum sessions, testing occurred during the last 30 min of chewing; in smoking session, testing began after two puffs, with continued smoking (one puff per min) for 5 min and two puffs before subsequent tests	Stroop test: mean number correct and response time of 50 color–word stimuli Letter cancellation: 105 target letters in array of 1,350 letters	Stroop test: Nicotine gum and smoking had no effect on performance of this test. Letter cancellation test: Completion time was faster after smoking compared with placebo; nicotine gum had no effect. Number of correct responses was not affected by smoking or nicotine gum.
Spilich, June, & Renner (1992)				
Exp. 1	45 students (15 nonsmokers, 30 smokers)	Not blind; 15 smokers were tobacco deprived for 3 hr; 15 other smokers took 12 paced puffs (one puff every 25 s) from a 1.2-mg nicotine cigarette immediately before testing; deprived smokers and nonsmokers sham smoked an imaginary cigarette; several practice trials preceded test trials	Neisser visual search: time to search array of 96 letters for single target (mean of 48 trials)	There was no difference between the three groups on this test. Response time was faster when target letter and array letters were dissimilar than when they were similar.
Exp. 2	60 students (20 nonsmokers, 40 smokers)	Same as Exp. 1, except that 20 smokers were tobacco deprived and 20 smoked before testing	Visual attention: time to detect change of 1 letter in span of 20 identical letters (mean of 48 trials)	Nonsmokers had fastest response time, followed by active smokers, and deprived smokers showed the slowest response time.
Heishman, Snyder, & Henningfield (1993)[a]	16 nonsmokers (9 men, 7 women)	Double-blind; nicotine polacrilex gum (0, 2, and 4 mg) was administered in ascending dose order at 90-min intervals in one session; testing conducted once before and after each 15-min gum-chewing period	Two-letter search: time to search 20-letters for 2 target letters	Nicotine had no effect on percentage of correct trials and mean response time.

Table 3 (*continued*)

Reference	Subjects	Design	Measure	Main findings
		Divided attention		
Leigh, Tong, & Campbell (1977)				
Exp. 1	10 male smokers	Not blind; subjects were tobacco and alcohol deprived for at least 12 hr; subjects smoked ad lib three 1.3-mg nicotine cigarettes or did not smoke in combination with alcohol or placebo in four separate counterbalanced sessions; subjects responded to either tones or clicks after two cigarettes and to both tones and clicks after a third cigarette	Dichotic listening: detect tones in left ear, count clicks in right ear	Smoking reduced error rate when clicks and tones were attended to separately and simultaneously in the divided attention test. Alcohol increased error rate, and there was no interaction between smoking and alcohol.
Exp. 2	12 male smokers	Same as Exp. 1, except that subjects smoked one cigarette in combination with alcohol or placebo before responding to tones and clicks at 10, 35, and 60 min postdrug	Same as Exp. 1	Mean error rate for three postdrug times was increased after alcohol, but was not affected by smoking.
Knott (1985b)	16 female smokers	Not blind; subjects were tobacco deprived for at least 10 hr; subjects either took four paced puffs from preferred-brand cigarette or did not smoke in two separate counterbalanced sessions; testing conducted after smoking or resting period	Visual two-choice reaction time; auditory signal detection	Smoking had no effect on either of the performance tests.
Hindmarch, Kerr, & Sherwood (1990)				
Exp. 1[a]	6 female smokers	Single-blind; smokers were not tobacco deprived; nicotine polacrilex gum (0, 2, and 4 mg) was chewed for 20 min in six separate randomized sessions (each dose tested twice); testing conducted before and 0.5, 1, 2, 3, and 4 hr after each dose	Tracking target with cursor; reaction time to peripheral visual stimuli	Tracking error (time off target) was reduced by both active doses of nicotine compared to placebo. Reaction time to peripheral stimuli was not affected. Plasma nicotine levels averaged 7.8, 12.4, and 16.1 ng/ml for 0, 2, and 4 mg of gum, respectively.
Exp. 2[a]	5 female nonsmokers	Same as Exp. 1, except nicotine doses were 0 and 2 mg and each dose was tested three times in six separate randomized sessions	Same as Exp. 1	Tracking error and reaction time were not affected by nicotine. Plasma nicotine levels averaged 1.2 and 4.9 ng/ml for 0 and 2 mg of gum, respectively.
Kerr, Sherwood, & Hindmarch (1991)[a]	10 women (5 smokers, 5 nonsmokers)	Double-blind; smokers were not tobacco deprived; nicotine polacrilex gum (0 and 2 mg) was chewed for 20 min alone and in combination with caffeine or alcohol in eight separate sessions; testing conducted before and 0.5, 1, 2, 3, and 4 hr after each dose	Tracking target with cursor; reaction time to peripheral visual stimuli	Tracking error was reduced with nicotine and with nicotine-caffeine combination compared with placebo. Reaction time to peripheral stimuli was not affected by nicotine. No difference observed between smokers and nonsmokers.
Sherwood, Kerr, & Hindmarch (1992)[a]	13 smokers (6 men, 7 women)	Single-blind; subjects were overnight tobacco deprived; nicotine polacrilex gum (0 or 2 mg) was chewed for 20 min at three times at 1-hr intervals in two separate counterbalanced sessions; testing conducted before and 30 min after each of the three doses	Tracking target with cursor; reaction time to peripheral visual stimuli	Tracking error was reduced after all three nicotine doses compared to predose baseline. Reaction time to peripheral stimuli was not affected by nicotine, but placebo condition resulted in slowed times relative to baseline.

(table continues)

Table 3 (*continued*)

Reference	Subjects	Design	Measure	Main findings
		Sustained attention		
Frankenhaeuser, Myrsten, Post, & Johansson (1971)	12 male smokers	Not blind; subjects were tobacco deprived for 12–14 hr; subjects either smoked ad lib three 2.0-mg nicotine cigarettes or did not smoke in two separate counterbalanced sessions; cigarettes were smoked during an 80-min visual reaction time task at 20, 40, and 60 min after task began	Simple reaction time to light stimulus (mean of 160 stimuli per 10 min)	Reaction time did not change in smoking condition but became gradually slower in nonsmoking session, reaching a maximum at about 40 min.
Ashton, Savage, Telford, Thompson, & Watson (1972)				
Exp. 1	17 smokers (11 men, 6 women) and 15 nonsmokers (9 men, 6 women)	Not blind; presession tobacco deprivation not indicated; smokers smoked ad lib one 1.4-mg nicotine cigarette during first half of each of three 20-min tests and a half cigarette during 10-min rest periods between tests	Reaction time to visual signals to brake, steer, or use turn signal indicator in driving simulator	Smoking produced no clear effect on reaction time. Smokers were faster on some measures, nonsmokers were faster on other measures, and there were no group differences on other measures. Differences were observed only during first half of test when smoking occurred.
Exp. 2	35 smokers (21 men, 14 women) and 16 nonsmokers (11 men, 5 women)	Same as Exp. 1, except smokers were divided into either low-nicotine (1.0 mg) cigarette group (n = 16) or high-nicotine (2.1 mg) cigarette group (n = 19)	Same as Exp. 1	Nonsmokers were faster than smokers on the four measures showing a group difference. No difference was observed between low- and high-nicotine cigarette groups.
Exp. 3	16 smokers (11 men, 5 women) and 17 nonsmokers (14 men, 3 women)	Not blind; presession tobacco deprivation not indicated; tracking and choice reaction time were assessed in two separate randomized sessions; smokers smoked ad lib one cigarette (nicotine content not specified) during 10-min reaction time test	Choice reaction time to two and four light stimuli	There was no difference between smokers and nonsmokers on either two- or four-light reaction time test.
Myrsten, Post, Frankenhaeuser, & Johansson (1972)	6 male smokers	Not blind; presession tobacco deprivation not indicated; subjects either smoked ad lib four 2.2-mg nicotine cigarettes or did not smoke during two separate counterbalanced sessions; one cigarette was smoked during first 5–10 min of each of four 25-min testing blocks, which alternated between simple and choice reaction time; testing conducted before and continuously for 100 min during smoking	Simple reaction time to light stimulus; choice reaction time to four light stimuli; for both, 300 stimuli per 25 min	Simple reaction time was unchanged during smoking condition and was slower during no-smoking condition compared with baseline and smoking session. Choice reaction time was faster in smoking condition compared with baseline but not when compared with no smoking session, which showed trend toward slower responding.
Hartley (1973)	15 female smokers	Not blind; subjects were tobacco deprived for 1–3 hr; subjects either smoked ad lib one or two 2.0-mg nicotine cigarettes or did not smoke in three separate counterbalanced sessions; test performed for 36 min after smoking or rest	Visual signal: observing responses made on each of three lights to determine presence or absence of signal; probability of signals varied over three lights	Across the six 6-min blocks of test, more observing responses on high-probability signal light were made in the no-smoking compared with either smoking condition. Responding on medium- and low-probability signal lights did not differ for the three conditions. Frequency of responding was more closely matched to stimulus frequency in no-smoking than smoking conditions, suggesting that smoking lowered arousal during latter half of test.

Table 3 (*continued*)

Reference	Subjects	Design	Measure	Main findings
Carter (1974)	10 smokers and 10 nonsmokers (for total sample, 10 men, 10 women)	Not blind; presession tobacco deprivation not indicated; smokers smoked ad lib one preferred-brand cigarette before testing and took two puffs during each 1-min intertrial interval of 20 trials in one session; smokers did not smoke during repeat testing 1 week later	Letter-digit substitution: mean completion time per trial	Smokers took longer to complete Trials 11–20 in first session compared with nonsmokers. There was no difference between groups on Trials 1–10 in first session and on entire test in the second session.
Schori & Jones (1974)	120 students (76 men, 44 women) divided into nonsmoker, smoker, deprived smoker groups (n = 40 each)	Not blind; presession tobacco deprivation not indicated; deprived smokers were tobacco deprived during the 3.5-hr session; smokers smoked ad lib during session; test conducted in six 30-min trials, each separated by 5-min break	Visual and auditory detection tasks and mental math performed at same time	Response latency of nonsmokers was faster than smokers and deprived smokers on auditory task in high-complexity condition, but slower in low complexity. There were no other group differences on any task in terms of accuracy or response latency.
Kucek (1975)	90 men (47 smokers, 43 nonsmokers)	Not blind; presession tobacco deprivation not indicated; smokers smoked ad lib one cigarette during Min 8–12 of 15-min task; tracking task only performed for first 7 min, then math added for final 8 min	Tracking task with and without mental math	Smokers showed a trend toward greater number and duration of tracking errors during and after smoking compared to nonsmokers. There were no group differences in tracking before smoking or in math performance.
Lyon, Tong, Leigh, & Clare (1975)	16 men (8 smokers, 8 nonsmokers)	Not blind; subjects were tobacco deprived for at least 12 hr; smokers either smoked ad lib two 1.3-mg nicotine cigarettes or did not smoke alone and in combination with alcohol in six separate counterbalanced sessions; nonsmokers received only alcohol; 25-min test followed drug administration	Choice reaction time: mean recognition and motor time of 150 presentations	Recognition reaction time was faster in smokers after smoking compared with not smoking. Performance of nonsmokers did not differ from that of smokers smoking, suggesting that tobacco deprivation slowed recognition time. Motor reaction time did not differ across conditions.
Myrsten, Andersson, Frankenhaeuser, & Elgerot (1975)	16 male smokers (8 low-arousal, 8 high-arousal)	Not blind; subjects were tobacco deprived for 15 hr; subjects either smoked ad lib two 1.8-mg nicotine cigarettes or did not smoke during low- and high-arousal test conditions in four separate sessions; cigarettes were smoked during 90-min test at 30 and 60 min after test began	Low-arousal test: visual signal detection; high-arousal test: visual choice reaction and auditory signal detection	Reaction time was faster on low-arousal test for low-arousal smokers during smoking compared with no smoking session, whereas high-arousal smokers showed a trend toward impairment. In high-arousal test, reaction time was faster for high-arousal smokers after smoking compared with not smoking; performance of low-arousal smokers was not affected by smoking. The groups did not differ in test performance in the no-smoking condition.
Schori & Jones (1975)	60 students (33 men, 27 women) divided into nonsmoker, smoker, deprived smoker groups (n = 20 each)	Not blind; presession tobacco deprivation not indicated; deprived smokers were tobacco deprived during the 1-hr session; smokers smoked ad lib one cigarette before test began and allowed to smoke ad lib during breaks; test conducted in four 15-min trials, each separated by short break	Tracking and visual signal detection tasks performed at same time	There were no differences between smokers, deprived smokers, and nonsmokers in either time on target on tracking task or in correct responses and errors on signal detection task. Signal detection varied in difficulty with tracking performance; groups did not differ in level of difficulty attained.

(table continues)

Table 3 (*continued*)

Reference	Subjects	Design	Measure	Main findings
Tong, Leigh, Campbell, & Smith (1977)	120 students (60 men, 60 women) divided into nonsmoker, smoker, deprived smoker groups (n = 40 each)	Not blind; smokers were tobacco deprived for 3 hr; smoking group smoked ad lib two 1.3-mg nicotine cigarettes 5 min apart during instructions and test practice; test conducted for 60 min after smoking or no smoking	Auditory signal detection: signal of three consecutive, unequal digits presented at one digit per second	Across all five 12-min blocks of test, nonsmokers correctly identified greater percentage of signals than smoking or deprived smoking groups, which did not differ. Performance of nonsmokers declined over session, deprived smokers remained stable, and smoking group improved.
Taylor & Blezard (1979)	56 men (30 smokers, 26 nonsmokers)	Not blind; smokers were not tobacco deprived and smoked ad lib one preferred-brand cigarette before 50-min test	Visual signal detection: signal of three consecutive even or odd digits; interdigit interval of 700 ms	Smoking had no effect on accuracy (hits minus false positives) during first 12 min of test. However, percentage change from first to last 12-min epoch showed performance of nonsmokers improving more than that of smokers. Authors interpreted poorer performance of smokers to be the result of nicotine deprivation during testing.
Heimstra, Fallesen, Kinsley, & Warner (1980)				
Exp. 1	16 female smokers	Not blind; presession tobacco deprivation not indicated; subjects assigned randomly to either smoking (n = 8) or no-smoking (n = 8) conditions; no-smoking group smoked one cigarette before, but did not smoke during 6-hr test; smoking group smoked ad lib during test	Multiple measures: tracking, reaction time, vigilance, mental arithmetic	Tracking performance of deprived smokers declined over course of test, whereas that of smokers was unchanged. No other group differences were observed.
Exp. 2	16 male smokers	Same as Exp. 1	Same as Exp. 1	Tracking performance declined for both groups over the course of the test; however, performance was poorer for deprived smokers. No other group differences were observed.
Tong, Henderson, & Chipperfield (1980)	32 male smokers	Not blind; subjects were tobacco deprived for 12 hr; subjects assigned randomly to either paced smoking of two 1.3-mg nicotine cigarettes before testing and three puffs in each of four intervals during testing or not smoking; each condition tested with and without alcohol in two separate sessions; testing conducted for 60 min after smoking or rest	Auditory signal detection: signal of three consecutive, unequal digits presented at one digit per second	Smoking tended to prevent decline in performance over course of 60-min test observed in no-smoking condition; however, smoking had no effect on mean number of correct signal detections or errors. There was no interaction between smoking and alcohol.
Mangan (1982)	24 male smokers	Not blind; subjects were tobacco deprived for 2 hr; subjects assigned randomly to either low-nicotine (0.7 mg) cigarette group (n = 12) or moderate-nicotine (1.3 mg) cigarette group (n = 12); subjects either smoked ad lib one cigarette or did not smoke in two separate counterbalanced sessions; 30-min test conducted after 10-min smoking period	Auditory signal detection: 30 signals (5 dB greater) in 600 stimuli presented at 3-s interval	Performance over course of 30-min test declined in all groups. Number of correct signal detections was greatest in low-nicotine condition, and false positive rate was lowest for moderate-nicotine group.

Table 3 (*continued*)

Reference	Subjects	Design	Measure	Main findings
Morgan & Pickens (1982)	12 smokers (3 men, 9 women)	Not blind; presession tobacco deprivation not indicated; subjects either smoked ad lib one preferred brand cigarette (M = 0.7 mg nicotine), smoked ad lib one research cigarette (1.2 mg nicotine), or smoked one research cigarette in paced manner in three counterbalanced sessions on separate days; task performed before (50 trials) and after (110 trials) smoking cigarette	Choice reaction time: mean recognition (time to leave start button) and motor (time to press correct button) times	Recognition reaction time was slower after smoking preferred-brand cigarette than after smoking both research cigarettes, which did not differ. Motor reaction time showed similar pattern, but did not differ between the three smoking conditions because of large variability in data.
Wesnes & Warburton (1983a)				
Exp. 1	24 male smokers	Single-blind; subjects were overnight tobacco deprived; subjects smoked ad lib one cigarette (0.28, 0.71, and 1.65 mg nicotine) in three separate counterbalanced sessions; task performed for 10 min after smoking (baseline) and for 20 min after smoking	Rapid visual information processing (RVIP): detection of three consecutive even or odd digits; interdigit interval of 600 ms	Probability of correct target detection (hit) was greatest and reaction time to respond correctly was fastest after 1.65-mg nicotine cigarette than either lower nicotine cigarette and reflected improvements over baseline. After both lower nicotine cigarettes, hits decreased from baseline during second 10 min of task. Number of errors (false alarms) was not affected.
Exp. 2[a]	12 smokers (6 men, 6 women)	Not blind; subjects were overnight tobacco deprived; subjects either smoked ad lib one cigarette (0, 0.6, and 1.84 mg nicotine) or did not smoke in four separate counterbalanced sessions; task performed for 10 min before smoking (baseline) and for 20 min after smoking or resting period	Same as Exp. 1	Performance (hits and reaction time) was better after both active cigarettes compared with 0-mg cigarette and no smoking. Increase in hits over baseline was not dose-related, and reaction time did not change from baseline for active cigarettes. Performance declined in placebo cigarette and no-smoking conditions.
Wesnes, Warburton, & Matz (1983)[a]	36 men and women (12 light and 12 heavy smokers, 12 nonsmokers)	Double-blind; smokers were tobacco deprived for 12 hr; nicotine tablets (0, 1, and 2 mg) were administered at 20, 40, and 60 min after start of task in three separate counterbalanced sessions; tablets held in mouth 5 min before being swallowed; signal detection task performed for 80 min, first 20 min taken as predrug baseline	Mackworth clock test: hits and false alarms to signal of pause in sweep of clock hand	Signal detection theory analysis indicated a decline from baseline in stimulus sensitivity during the task for all conditions. This decline was greatest with placebo, and was reduced by nicotine after the second and third tablets. Response bias was not affected by nicotine. No differences observed between light and heavy smokers and nonsmokers.
Wesnes & Revell (1984)				
Exp. 1[a]	12 nonsmokers (6 men, 6 women)	Double-blind; placebo, scopolamine (1.2 mg), or nicotine (1.5 mg) tablets administered in four separate counterbalanced sessions; tablets held in mouth 5 min before being swallowed; task performed for 10 min predrug and for 20 min postdrug	RVIP (see Wesnes & Warburton, 1983a)	Data from 6 subjects were available for analysis. Nicotine had no effect on correct responses, reaction time, or errors compared with placebo. Scopolamine decreased correct responding over time, but the scopolamine–nicotine combination had no effect.
Exp. 2[a]	12 nonsmokers (6 men, 6 women)	Double-blind; placebo, scopolamine (1.2 mg), or nicotine (1.5 mg) tablets administered three times at 1-hr intervals in four separate counterbalanced sessions; tablets held in mouth 5 min before being swallowed; task performed for 10 min predrug and for 15 min after second and third drug administration	Same as Exp. 1	Nicotine had no effect on correct responses, reaction time, or errors compared with placebo. Scopolamine decreased correct responding, and nicotine, combined with scopolamine, reversed this impairment.

(table continues)

Table 3 (*continued*)

Reference	Subjects	Design	Measure	Main findings
Wesnes & Warburton (1984a)	25 male smokers	Not blind; subjects were overnight tobacco deprived; subjects either smoked ad lib one cigarette (0.9, 1.3, 1.5, and 1.7 mg nicotine) or did not smoke in five separate counterbalanced sessions; task performed for 10 min before (baseline) and for 20 min after smoking or resting period	RVIP (see Wesnes & Warburton, 1983a)	Correct responding was increased and reaction time was faster after smoking than not smoking. Increase in hits over baseline observed only during first 10 min after smoking. Performance declined from baseline in no-smoking condition.
Wesnes & Warburton (1984b) Exp. 2[a]	12 male nonsmokers	Double-blind; nicotine tablets (0, 0.5, 1.0, and 1.5 mg) administered in four separate counterbalanced sessions; tablets held in mouth 5 min before being swallowed; task performed for 10 min predrug and for 20 min postdrug	RVIP (see Wesnes & Warburton, 1983a)	Data from 9 subjects were available for analysis. Correct responding decreased in all dose conditions, except 1.5 mg nicotine, which was not different from baseline. The only change in reaction time was slower responding in the placebo condition.
Edwards, Wesnes, Warburton, & Gale (1985)	19 male smokers	Not blind; subjects were tobacco deprived for 12 hr; subjects either smoked ad lib one cigarette (0.9 and 1.5 mg nicotine) or did not smoke in three separate counterbalanced sessions; task performed for 10 min before (baseline) and for 20 min after 10-min smoking or resting period	RVIP (see Wesnes & Warburton, 1983a)	Correct responding was increased by smoking in the first 10 min of task compared with no smoking; accuracy declined in all conditions in second 10 min. Reaction time was faster after the 1.5-mg cigarette compared with the other conditions, which showed little change.
O'Connor (1986a)	16 smokers (9 men, 7 women)	Not blind; presession tobacco deprivation not indicated; subjects sham smoked during testing and then smoked ad lib 1.45-mg nicotine cigarettes during testing in each of four separate sessions; smoking allowed during 10-s intertrial intervals; testing involved standard signal detection paradigm and conditions manipulating subject expectancy and response bias	Visual signal detection: green light set at threshold; 128 trials per condition	Smoking produced an increase in sensitivity compared with sham smoking in extroverts only in standard condition. Introverts showed lower sensitivity during smoking in standard condition compared with expectancy and bias manipulations. Smoking increased response bias in introverts in conditions manipulating bias (motivation); extroverts showed no change in bias across conditions.
Michel, Nil, Buzzi, Woodson, & Bättig (1987)	21 smokers (12 men, 9 women)	Not blind; subjects were tobacco deprived for 5 hr; subjects smoked ad lib one preferred-brand cigarette in single session; 30-min task performed before and after 20-min smoking period	RVIP (modified version)	Interdigit interval began at 600 ms, increased with correct responding and decreased with errors by 40 ms; interval was dependent measure. Low CO absorbers performed better than medium and high CO absorbers. Practice and fatigue effects evident.
Michel, Hasenfratz, Nil, & Bättig (1988)[a]	20 female smokers	Single-blind; subjects were tobacco deprived for at least 2 hr; subjects assigned randomly to either 0 or 4 mg nicotine polacrilex gum conditions; 20-min task performed twice before and once after 15-min gum-chewing period	RVIP (see Michel et al., 1987)	Nicotine had no effect on inter-digit interval or reaction time. Performance declined from first to second 10 min of each of the three task trials.
Nil, Woodson, Michel, & Bättig (1988)	34 smokers (6 men, 28 women) and 9 female nonsmokers	Not blind; smokers were tobacco deprived for 5 hr; 24 smokers preselected as high (n = 12) or low (n = 12) CO absorbers; 10 smokers did not smoke during session; 30-min test performed before and after 10-min smoking or resting period; amount and nature of smoking not stated	RVIP (see Michel et al., 1987)	Performance declined during both test periods for all groups, except low CO absorbers during first test. During postsmoking test, performance decline was greater for both no-smoking groups compared with both smoking groups. No difference between high and low CO absorbers.

Table 3 (*continued*)

Reference	Subjects	Design	Measure	Main findings
Revell (1988)	30 smokers (15 men, 15 women)	Not blind; smokers were overnight tobacco deprived; subjects either smoked one cigarette (1.2 and 1.5 mg nicotine), sham smoked an unlit cigarette, or did not smoke in four separate counterbalanced sessions; cigarettes smoked one puff per minute during Min 6–15 of 20-min task	RVIP (see Wesnes & Warburton, 1983a)	Smoking increased correct responding and resulted in faster reaction time than both no-smoking conditions. Performance improvements were seen after only two puffs and generally persisted throughout rest of task. No difference between two cigarettes or between two control conditions.
Hasenfratz, Michel, Nil, & Bättig (1989)	24 female smokers	Not blind; subjects were tobacco deprived for 10 hr; subjects assigned randomly to either smoking (n = 12) or no-smoking (n = 12) groups; 30-min test performed before and after smoking ad lib one preferred-brand cigarette or resting in two separate sessions	RVIP (see Michel et al., 1987)	Performance declined during pre- and postsmoking testing periods for smoking and no-smoking groups. Smoking increased number of digits presented per minute from pre- to postsmoking test. Reaction time was not affected by smoking.
Hasenfratz, Pfiffner, Pellaud, & Bättig (1989)				
Exp. 2	22 male smokers	Not blind; subjects were not tobacco deprived; subjects either smoked ad lib one preferred-brand cigarette or did not smoke in two separate counterbalanced sessions; 20-min test conducted once 10 min after smoking or resting	RVIP (modified version)	Interdigit interval began at 666 ms, increased with correct responding and decreased with errors by 33 ms. Smoking had no effect on stimulus presentation rate (digits per minute) or reaction time compared with no-smoking condition.
Keenan, Hatsukami, & Anton (1989)	27 male smokeless tobacco users and 10 male nonusers	Not blind; users were not tobacco deprived at first session; users randomly assigned to deprived (n = 15) or nondeprived (n = 12) groups for 24 hr before Session 2; tobacco chewed for 20 min at Session 1 (all users) and Session 2 (nondeprived group only); 23-min test conducted once after tobacco chewing	Choice reaction time: respond to signal at 12:00, but not to signal at 6:00 position	There was no difference in reaction time or number of errors between users and nonusers at Session 1. Nondeprived users and nonusers showed comparable decreases in reaction time at Session 2, whereas deprived users were slower compared with Session 1.
Michel & Bättig (1989)	20 female smokers	Not blind; subjects were tobacco deprived for at least 2 hr; subjects smoked ad lib one preferred-brand cigarette (M = 0.9 mg nicotine) or sham smoked unlit cigarette and received alcohol or placebo in four separate counterbalanced sessions; 20-min test conducted before and after drug administration	RVIP (see Michel et al., 1987)	During first 10 min of postdrug test, performance (stimulus presentation rate and reaction time) was improved equally for smoking and sham smoking over predrug test. Performance declined during second 10 min to greater extent for sham than actual smoking.
Parrott & Winder (1989)[a]	16 male smokers	Double-blind (gum only); subjects were tobacco deprived for 3–6 hr; nicotine polacrilex gum (0, 2, and 4 mg) was chewed for 21 min or preferred-brand cigarette was smoked through paced procedure in four separate counterbalanced sessions; in gum sessions, testing occurred during the last 12 min of chewing; in smoking session, testing began after two puffs, with continued smoking (one puff per minute) for 5 min	RVIP (see Wesnes & Warburton, 1983a)	During second 5 min of test, smoking and 4-mg nicotine gum increased correct responding comparably over placebo condition, which showed a decline in performance. Reaction time and errors were not significantly affected.

(table continues)

Table 3 (*continued*)

Reference	Subjects	Design	Measure	Main findings
Petrie & Deary (1989)	12 smokers (6 men, 6 women)	Not blind; subjects were tobacco deprived for at least 8 hr; subjects either smoked ad lib two high-nicotine cigarettes or did not smoke in two separate counterbalanced sessions; 10-min visual information-processing test performed after first cigarette or rest and 15–20-min inspection time test performed after second cigarette or rest	RVIP (see Wesnes & Warburton, 1983a). Inspection time test: stimulus exposure time at 85% accuracy in discrminating longer of two lines	RVIP: Smoking produced faster reaction time during first, but not second, 5 min of test compared with no-smoking condition. Smoking had no effect on number of correct responses or errors. Inspection time test: Smoking had no effect on this 15- 20-min test, which estimates transfer rate of visual information to short-term memory.
Hasenfratz, Jaquet, Aeschbach, & Bättig (1991)				
Exp. 1	12 male smokers	Not blind; subjects were tobacco deprived on day of study, but duration not stated; subjects smoked ad lib one preferred-brand cigarette or did not smoke and received either caffeine or placebo in four separate sessions; 20-min test conducted once before and after 20-min drug administration period	RVIP (see Hasenfratz, Pfiffner et al., 1989)	Smoking increased stimulus presentation rate from baseline, whereas no increase was seen in the no-smoking condition. Reaction time was not affected, but smoking produced a trend toward faster responding during the second 10 min of the test.
Jones, Sahakian, Levy, Warburton, & Gray (1992)[a]	22 patients with Alzheimer's disease; 24 normal elderly controls; 24 young adult controls; men and women, smokers and nonsmokers in all groups	Single-blind; presession tobacco deprivation not indicated; single doses of nicotine (0, 0.4, 0.6, and 0.8 mg) were injected subcutaneously in ascending dose order or no injection was given in seven separate sessions; test battery assessed once after injection	RVIP (modified version)	Task required detection of only ascending even or odd sequences (e.g., 2, 4, 6), was graded in difficulty for different subject groups, and lasted for 7 min. Signal detection theory analysis indicated an overall effect of nicotine to increase stimulus sensitivity, but this was evident only in patients with Alzheimer's disease. Response bias and reaction time were not affected.
Parrott & Craig (1992)[a]	16 smokers (14 men, 2 women)	Double-blind (gum only); subjects were tobacco deprived for 12 hr; nicotine polacrilex gum (0, 2, and 4 mg) was chewed for 40 min or preferred-brand cigarette was smoked through paced procedure in four separate counterbalanced sessions; in gum sessions, testing occurred during the second 10 min of chewing; in smoking session, testing began after two puffs, with continued smoking (one puff per minute) for 5 min and two puffs before subsequent tests	RVIP (see Wesnes & Warburton, 1983a). Width of attention; mean number correct and response time to indicate position of 360 visual stimuli	RVIP: Correct responding was increased over placebo by smoking during first 5 min of test and by 2-mg nicotine gum during second 5 min of test. Reaction time and errors were not affected. Width of attention test: Nicotine gum and smoking had no effect on performance of this 15-min test.
Pritchard, Robinson, & Guy (1992)	24 smokers (13 men, 11 women)	Not blind; subjects were not tobacco deprived; subjects either smoked ad lib one 0.6-mg nicotine cigarette at two times or did not smoke on two separate counterbalanced sessions; 20-min task performed twice, once after each cigarette or rest period	Continuous performance test: left response to single digits 1–9, right response to target digit 0	Reaction time to target and nontarget digits was faster (17 ms) in smoking compared with nonsmoking session. Low CO absorbers were faster on nontarget digits than high CO absorbers, especially in nonsmoking session. Number of incorrect responses was not affected by smoking.
Morris & Gale (1993)				
Exp. 3	18 male smokers	Not blind; subjects were tobacco deprived for 1 hr; subjects either smoked ad lib preferred-brand cigarettes or did not smoke in two separate sessions; smoking occurred during last 20 min of 40-min test	Continuous performance test: detection of four consecutive odd digits	Smoking had no effect on reaction time or number of errors of omission and commission.

Note. Exp. = Experiment.
[a]Included in section on studies using rigorous methodology.

measure. Nicotine or smoking improved accuracy, reaction time, or both in 10 experiments; however, all were conducted with smokers who were tobacco deprived for 3–12 hr before sessions and none included a nonabstinent baseline assessment. Thus, these studies reporting facilitation may have simply documented reversal of withdrawal-induced deficits to predeprivation performance levels.

Vigilance tests. Tests of sustained attention lasting at least 30 min are considered tests of vigilance (Jerison, 1970; Warm, 1984). The typical decline in performance over time in such tests is referred to as the vigilance decrement. There were 15 studies that involved vigilance testing, and a clear interpretation of the data was possible in 12 studies. All involved cigarette smoking, except Wesnes, Warburton, and Matz (1983), who administered nicotine tablets orally. The tests used were varied and measured divided attention (Heimstra et al., 1980; Myrsten, Andersson, Frankenhaeuser, & Elgerot, 1975), reaction time (Frankenhaeuser, Myrsten, Post, & Johansson, 1971; Myrsten, Post, Frankenhaeuser, & Johansson, 1972), signal detection (Mangan, 1982; Morris & Gale, 1993, Experiment 3; Taylor & Blezard, 1979; Tong, Henderson, & Chipperfield, 1980; Tong, Leigh, Campbell, & Smith, 1977; Wesnes et al., 1983), and RVIP (Hasenfratz, Michel, Nil, & Bättig, 1989; Nil, Woodson, Michel, & Bättig, 1988). Of the 12 studies, 8 found that smoking or nicotine prevented the vigilance decrement. However, only 4 of the 12 studies reported that nicotine enhanced performance over baseline or control group levels. In 3 of these 4 studies, subjects were abstinent for 3–15 hr before sessions, and the 4th study did not indicate abstinence instructions; none of the studies measured performance when subjects were not tobacco deprived.

Nicotine deprivation. In 23 of the 43 experiments testing sustained attention, smokers were abstinent for 1–14 hr before testing. Thirteen experiments reported that smoking or nicotine enhanced some aspect of performance; however, none measured baseline performance before enforced abstinence, so again, absolute improvement must be questioned. Seventeen experiments found no effect of nicotine and 3 experiments reported impaired performance. Thirteen other experiments did not indicate whether subjects were tobacco deprived.

No deprivation. Nonabstinent smokers, nonabstinent smokeless tobacco users, and nonsmokers either smoked or were administered nicotine in 9 of the 43 experiments. In 8 experiments, 5 of which used the RVIP test, performance was not affected by smoking or nicotine (Hasenfratz, Pfiffner, Pellaud, & Bättig, 1989, Experiment 2; Jones et al., 1992; Keenan, Hatsukami, & Anton, 1989; Taylor & Blezard, 1979; Wesnes & Revell, 1984, Experiments 1 & 2; Wesnes & Warburton, 1984b, Experiment 2; Wesnes et al., 1983). One study reported that smoking enhanced reaction time in a simple continuous performance test (Pritchard, Robinson, & Guy, 1992).

Summary of sustained attention. In studies with abstinent smokers, the evidence that smoking or nicotine facilitates sustained attention is mixed, with 13 of 23 experiments indicating enhancement (5–15% of control values), but 20 of 23 experiments reporting no effect or impairment on some aspect of test performance. There is stronger evidence that smoking or nicotine can prevent the performance decrement observed in vigilance tests. In eight of nine studies with nonabstinent tobacco users and nonsmokers, smoking or nicotine had no effect on sustained attentional abilities.

Cognitive Abilities

Table 4 summarizes the results of 35 studies (43 experiments), and is divided into three sections: learning, memory, and other cognitive abilities. Although learning and memory have been studied for decades in psychology as distinct, yet obviously related, cognitive processes, the vast majority of nicotine research has focused on memory compared with learning. Relatively few studies have investigated other cognitive abilities such as problem solving and reasoning skills.

Learning

Traditional rote, serial, or paired-associate learning paradigms were used in six of seven experiments. Dependent measures included number of errors or correct responses over a fixed number of trials or number of trials required to reach a set criterion of errorless trials.

Nicotine deprivation. Three studies used a paired-associate paradigm and tested smokers who

(text continues on page 381)

Table 4
Summary of Research on Effects of Smoking and Nicotine on Cognitive Abilities

Reference	Subjects	Design	Measure	Main findings
		Learning		
Andersson & Post (1974)[a]	20 male smokers	Single-blind; appears that subjects were overnight tobacco deprived; subjects smoked ad lib two cigarettes (0 or 2.1 mg nicotine) in each of two separate counterbalanced sessions; 10 anticipatory learning trials conducted before Cigarette 1 and after Cigarettes 1 and 2	Rote learning of 30 nonsense syllables (visual)	After Cigarette 1, number of correct responses was greater in placebo than in nicotine cigarette condition, but after Cigarette 2, scores did not differ. Gain in learning from first to second postsmoking period was greater in smoking condition.
Carter (1974)	10 smokers and 10 nonsmokers (for total sample, 10 men, 10 women)	Not blind; presession tobacco deprivation not indicated; smokers smoked ad lib one preferred-brand cigarette before testing and took two puffs after each test trial in one session; smokers did not smoke during repeat testing 1 week later	Serial learning (12 nonsense syllables to criterion of one errorless trial)	There was no difference between smokers and nonsmokers in trials to criterion summed over both sessions.
Andersson (1975)	10 male smokers	Not blind; subjects were tobacco deprived for at least 10 hr; subjects either smoked ad lib one 2.1-mg nicotine cigarette or did not smoke in two separate counterbalanced sessions; 10 anticipatory learning trials conducted before and after smoking or resting; final test 45 min after last postsmoking trial	Rote learning of 25 nonsense syllables (visual)	Number of correct responses per trial in the five trials after smoking or resting was greater for no-smoking than smoking condition. At 45-min test, smoking group showed trend toward more correct responses.
Mangan (1983) Exp. 1	16 male smokers	Not blind; subjects were tobacco deprived for 1 hr; subjects either smoked ad lib one cigarette (0.7 and 1.3 mg nicotine) or did not smoke in six separate randomized sessions; word pairs learned to criterion immediately after smoking or resting; retested after 30-min rest period	Paired-associate learning (10 word pairs to criterion of two successive, errorless trials)	Nicotine had no effect on mean number of errors during acquisition trials to criterion. On retest 30 min later, nicotine reduced number of errors compared with not smoking in dose-related manner.
Mangan & Golding (1983)	69 men (54 smokers, 15 nonsmokers)	Not blind; smokers were tobacco deprived for 1 hr; subjects assigned to 1 of 5 groups: nonsmokers, smokers not smoking, and smoking ad lib one 0.8-, 1.3-, or 2.0-mg nicotine cigarette immediately after test acquisition; retested to criterion 30 min, 1 day, 1 week, and 1 month after acquisition	Paired-associate learning (10 word pairs to criterion of one errorless trial)	Trials to criterion and errors on Trial 2 of initial acquisition showed no differences between groups. At 30 min retest, nonsmokers had fewer trials to criterion and errors than other groups. Similar results at 1 day and 1 week, except 0.8-mg cigarette group improved to nonsmoker performance level.
Colrain, Mangan, Pellett, & Bates (1992)[a]	39 smokers (gender not indicated)	Not blind; subjects were tobacco deprived for 2 hr; subjects either smoked ad lib one cigarette (0.07, 0.6, and 1.1 mg nicotine) or did not smoke in four counterbalanced weekly sessions; smoking or resting occurred after word pairs learned to criterion; retested to criterion 1 week after acquisition	Paired-associate learning (10 word pairs to criterion of two errorless trials)	Total number of errors to reach criterion at 1-week recall was reduced in 0.6-mg nicotine cigarette condition compared with 0.07-mg cigarette and no smoking, which did not differ. Errors in 1.1-mg cigarette condition were intermediate.
Roth, Lutiger, Hasenfratz, Bättig, & Knye (1992)	20 female smokers	Not blind; subjects were either not tobacco deprived and smoked at four times (smoking condition) or were deprived for 11 hr and did not smoke (deprived condition) in two separate counterbalanced sessions; amount and nature of smoking not stated; testing conducted after smoking or resting	Maze learning and retention; word recognition from repeated subliminal presentations	There was no difference between the smoking and deprived conditions in either maze learning and retention tested 1 hr after learning or in word recognition test. In both tests, smokers who smoked first cigarettes within 1 hr of waking performed better in smoking than deprived condition. "Later" smokers performed better during deprivation.

Table 4 (*continued*)

Reference	Subjects	Design	Measure	Main findings
		Memory		
Andersson & Hockey (1977)	50 female smokers	Not blind; subjects were tobacco deprived the morning of testing (3–4 hr); subjects equally assigned to either smoking or no-smoking conditions; subjects smoked ad lib one 2.3-mg nicotine cigarette or rested before word-list presentation and recall	Immediate serial recall of two 8-word lists (visual)	Number of words recalled did not differ between smoking and no-smoking groups. Recall of incidental cue of word position (one of four corners of screen) was greater in no-smoking than smoking group.
Houston, Schneider, & Jarvik (1978)[a]	23 smokers (11 men, 12 women)	Single-blind; subjects were tobacco deprived for 3 hr; subjects randomly assigned to 1.5-mg nicotine cigarette (n = 11) or nicotine-free cigarette (n = 12) condition; paced smoking (12 puffs at 25-s intervals) followed initial list presentation and recall and preceded three presentation-recall trials; recall tested 2 days later after nicotine or nicotine-free cigarette smoked by half of subjects in each group	Immediate and delayed (2 days) free recall of 75-word list (auditory)	Number of correctly recalled words in each of the three postsmoking trials was greater in nicotine-free cigarette condition than nicotine condition in immediate and delayed recall tests.
Gonzales & Harris (1980)	10 smokers (5 men, 5 women)	Not blind; subjects were tobacco deprived for at least 6.5 hr; subjects randomly assigned to 1.5-mg nicotine cigarette or no-smoking condition; paced smoking (12 puffs at 25-s intervals) followed recall of 40-word list and preceded immediate and delayed recall of 60-word list (20 new and 40 original words)	Immediate and delayed (45 min) free recall of 40- and 60-word lists (visual)	Smoking and no-smoking groups did not differ in number of total words and new words recalled either immediately or at 45-min test. Smoking group recalled fewer of 40 original words than no-smoking group at immediate and delayed recall tests.
Williams (1980)	48 male smokers	Not blind; subjects were tobacco deprived for at least 12 hr; subjects smoked in paced manner (1 puff every 30 s) one cigarette (0.6, 1.3, and 1.8 mg nicotine) or sham smoked in four separate counterbalanced sessions; testing conducted once before and after smoking	Immediate serial recall of nine-digit spans	Number of serial position errors over six digit sequences increased as a function of nicotine content of smoked cigarettes.
Peters & McGee (1982) Exp. 2	56 smokers (gender not indicated)	Single-blind; subjects were overnight tobacco deprived; 14 subjects each assigned to one of four conditions: ad lib smoking of high-nicotine (1.4 mg) cigarette on Days 1 and 2, low-nicotine (0.2 mg) cigarette on Days 1 and 2, high nicotine on Day 1 and low nicotine on Day 2, or low nicotine on Day 1 and high nicotine on Day 2; immediate recall on Day 1; details of Day 2 testing not stated	Immediate free recall and delayed (24 hr) recognition of 15-word list (visual)	Nicotine had no effect on Day 1 immediate recall. Day 2 recall and recognition testing indicated asymmetrical state-dependent learning with high–high and low–low groups performing best, and high–low scoring lower than low–high group.
Mangan (1983) Exp. 2	8 male smokers	Not blind; subjects were tobacco deprived for 1 hr; smoked ad lib one cigarette (0.7 and 1.3 mg nicotine) or did not smoke in three separate randomized sessions; eight sets of word lists tested immediately after smoking or rest	Immediate free recall of 20-word list (visual)	Nicotine had no overall effect on number of words recalled; however, both cigarette conditions increased primacy effect (first four words of lists) compared with not smoking.

(table continues)

Table 4 (*continued*)

Reference	Subjects	Design	Measure	Main findings
Peeke & Peeke (1984)				
Exp. 1	18 male smokers	Not blind; subjects were tobacco deprived for 2 hr; subjects smoked ad lib one preferred-brand cigarette either before or after word list or did not smoke in three separate counterbalanced sessions; recall tested immediately, 10, and 45 min after word list presentation	Immediate and delayed free recall of 50-word lists (auditory)	Pretrial smoking increased number of words recalled at 10 and 45 min compared with not smoking (average increase of three words), but had no effect on immediate recall. Posttrial smoking had no effect on immediate recall and marginally increased delayed recall.
Exp. 2	76 light (n = 26), moderate (n = 25), and heavy (n = 25) smokers (37 men, 39 women)	Not blind; subjects were tobacco deprived for 2 hr; equal number of smoker type and gender assigned to five conditions: smoking ad lib one 1.4-mg nicotine cigarette before, 1, 5, or 30 min after list presentation or not smoking; recall tested at 24 hr	Delayed free recall and recognition of 20-word list (auditory)	Pretrial smoking increased number of words recalled and decreased number of errors compared with posttrial smoking conditions. Effect seen only in light and moderate smokers. Posttrial smoking had no effect on recall. Recognition test was not affected.
Exp. 3	10 light and 10 heavy smokers (5 men, 5 women in each group)	Not blind; subjects were tobacco deprived for 2 hr; subjects either smoked ad lib one cigarette (0.4 and 1.4 mg nicotine) before word list or did not smoke in three separate randomized sessions; recall tested immediately, 10, and 45 min after word list	Immediate and delayed free recall of 50-word lists (auditory)	High-dose nicotine cigarette increased number of words recalled in immediate and both delayed recall tests compared with low-dose and no-smoking conditions. No differences seen between light and heavy smokers.
Exp. 4	Same subjects as Exp. 2	Not blind; subjects were tobacco deprived for 2.5 hr; equal number of smoker type and gender assigned to three conditions: smoking ad lib one cigarette (0.4 or 1.4 mg nicotine) before word list or not smoking; level of stimulus encoding varied across words and subjects; subjects not aware of subsequent recall test.	Immediate free recall and recognition of 72-word list (visual)	Smoking condition had no effect on either recall or recognition scores. However, recall and recognition improved as depth of stimulus encoding increased from a structural to an acoustic to a semantic level of processing.
Kunzendorf & Wigner (1985)	32 smokers (gender not indicated)	Not blind; presession tobacco deprivation not indicated; subjects randomly assigned to one of four conditions: smoking during study of article and during test, no smoking at each time, smoking during study only, or smoking during test only; amount and nature of smoking not stated	Questions about 550-word article answered after 10-min delay	Number of correct answers was greatest in groups who either smoked or did not smoke during both studying and testing. State-dependent learning demonstrated.
Dunne, MacDonald, & Hartley (1986)[a]	16 female nonsmokers	Double-blind; nicotine polacrilex gum (0 and 4 mg) administered in two separate randomized sessions; recall and recognition testing lasted for 50 min after 30-min gum-chewing period	Immediate and delayed recall and recognition of answers to problems	Nicotine decreased number of words and numbers recalled (immediately and delayed) and recognized compared with placebo.
Jubis (1986)	90 male smokers	Not blind; subjects were tobacco and alcohol deprived for 1 hr; subjects randomly assigned to one of six groups (n = 15 each): smoking ad lib two 1.3-mg nicotine cigarettes or not smoking crossed with alcohol (placebo, low, high doses); list presentation and recall conducted twice after drug administration	Immediate free recall of 20-word list (visual)	Smoking alone had no effect on number of words recalled after first or second list presentation compared to no-smoking group. Smoking also had no effect on incidental (first presentation) or intentional (second presentation) recall of word color.

Table 4 (*continued*)

Reference	Subjects	Design	Measure	Main findings
Warburton, Wesnes, Shergold, & James (1986)				
Exp. 1	40 smokers (20 men, 20 women)	Not blind; subjects were tobacco deprived for at least 10 hr; 10 subjects assigned to one of four conditions: smoking ad lib one 1.3-mg nicotine cigarette before learning and recognition, no smoking at each time, smoking before learning only, or smoking before recognition only	Delayed (1 hr) recognition of 15 Chinese ideograms (visual)	Mean number of correctly recognized ideograms reported in text is discrepant with that shown in Figure 1 for group who smoked before learning but not before recognition. Figure 1 shows smoking–smoking group scoring best, no-smoking–smoking group scoring worst, and other groups intermediate. State-dependent learning demonstrated.
Exp. 2	40 smokers (20 men, 20 women)	Double-blind; subjects were tobacco deprived for at least 10 hr; 10 subjects assigned to one of four conditions similar to Exp. 1, except nicotine (dose not indicated) and placebo tablets replaced smoking and not smoking	Immediate and delayed (1 hr) free recall of 48-word list (auditory)	Nicotine increased number of words recalled compared with placebo in immediate recall test. Delayed recall data similar to that in Exp. 1. State-dependent learning again demonstrated.
Newhouse et al. (1988)[a]	6 patients with Alzheimer's disease (all nonsmokers; 2 men, 4 women)	Single-blind; nicotine (0.125, 0.25, and 0.5 μg/kg/min) and saline were intravenously infused over 60 min in ascending dose order in four separate sessions; testing conducted 0.5 hr before and 0.5, 1, 4, 8, and 24 hr after infusion began	Delayed free recall of eight-word list. Category retrieval	The 0.25-μg dose of nicotine produced fewer recall errors at 1-hr test and a trend toward more words recalled at 8-hr test compared with placebo. Nicotine had no effect on category retrieval test.
Parrott & Winder (1989)[a]	16 male smokers	Double-blind (gum only); subjects were tobacco deprived for 3–6 hr; nicotine polacrilex gum (0, 2, and 4 mg) was chewed for 21 min or preferred-brand cigarette was smoked through paced procedure in four separate counterbalanced sessions; in gum sessions, testing occurred during the last 12 min of chewing; in smoking session, testing began after two puffs, with continued smoking (one puff per min) for 5 min	Immediate and delayed free recall of 16-word lists	Smoking and nicotine had no effect on either number of words correctly recalled or on intrusion errors.
Snyder & Henningfield (1989)[a]	6 male smokers	Double-blind; subjects were tobacco deprived for 12 hr; nicotine polacrilex gum (0, 2, and 4 mg) administered in three separate counterbalanced sessions; testing conducted at baseline (stable practice) and once after 10-min gum-chewing period	Digit recall test: mean of correct trials and response time per trial	Mean response time was slower than baseline after 0-mg nicotine, and 2- and 4-mg doses returned performance to baseline. Nicotine produced dose-related trend toward increased accuracy.
Hindmarch, Kerr, & Sherwood (1990)				
Exp. 1[a]	6 female smokers	Single-blind; smokers were not tobacco deprived; nicotine polacrilex gum (0, 2, and 4 mg) was chewed for 20 min in six separate randomized sessions (each dose tested twice); testing conducted before and 0.5, 1, 2, 3, and 4 hr after each dose	Sternberg test: reaction time to probe of four digits (mean of 24 trials)	Nicotine had no effect on reaction time in test of short-term memory scanning ability. Plasma nicotine levels averaged 7.8, 12.4, and 16.1 ng/ml for 0, 2, and 4 mg gum, respectively.

(table continues)

Table 4 (*continued*)

Reference	Subjects	Design	Measure	Main findings
Exp. 2[a]	5 female nonsmokers	Same as Exp. 1, except nicotine doses were 0 and 2 mg and each dose was tested three times in six separate randomized sessions	Same as Exp. 1	Nicotine had no effect on reaction time in test of short-term memory scanning ability. Plasma nicotine levels averaged 1.2 and 4.9 ng/ml for 0 and 2 mg gum, respectively.
Kerr, Sherwood, & Hindmarch (1991)[a]	10 women (5 smokers, 5 nonsmokers)	Double-blind; smokers were not tobacco deprived; nicotine polacrilex gum (0 and 2 mg) was chewed for 20 min alone and in combination with caffeine or alcohol in eight separate sessions; testing conducted before and 0.5, 1, 2, 3, and 4 hr after each dose	Sternberg test: reaction time to probe of four digits (mean of 24 trials)	Reaction time was faster after nicotine alone, but slower after nicotine–alcohol combination compared with placebo in test of short-term memory scanning ability. No difference observed between smokers and nonsmokers.
Rusted & Eaton-Williams (1991)[a]	20 male smokers	Double-blind; subjects were tobacco deprived for 9–10 hr; nicotine (0, 0.75, and 1.5 mg) tablets administered alone or in combination with scopolamine (1.2 mg orally) in five separate counterbalanced sessions; tablets held in mouth 5 min; testing conducted before and 10 min after nicotine	Immediate free recall of 10- and 30-word lists on four trials	Mean number of words recalled in 10- and 30-word list conditions did not differ between nicotine and placebo conditions. Recall improved over the four trials in all conditions. Scopolamine and scopolamine–nicotine combination impaired recall compared with placebo.
West & Hack (1991)[a]	15 regular smokers (7 men, 8 women) and 14 occasional smokers (5 men, 9 women)	Single-blind; for Session 1, subjects were not tobacco deprived; for Session 2, subjects were tobacco deprived for 24 hr; at each session, subjects smoked ad lib two cigarettes (0 and 1.5 mg nicotine) in counterbalanced order; testing conducted before and after cigarettes	Sternberg test: reaction time to probes of 2- and 5-digit spans	Reaction time was faster for five-digit span condition after 1.5-mg nicotine cigarette compared with placebo cigarette in both groups and in both sessions. No difference between Sessions 1 and 2 or between groups. Nicotine had no effect on two-digit span.
Jones, Sahakian, Levy, Warburton, & Gray (1992)[a]	22 patients with Alzheimer's disease; 24 normal elderly controls; 24 young adult controls; men and women, smokers and nonsmokers in all groups	Single-blind; presession tobacco deprivation not indicated; single doses of nicotine (0, 0.4, 0.6, and 0.8 mg) were injected subcutaneously in ascending dose order or no injection was given in seven separate sessions; testing conducted once after injection	Delayed matching to location-order test (visual memory). Digit span forward test (auditory memory)	Only patients with Alzheimer's disease showed attentional and memory deficits on delayed matching test. Nicotine reduced these attentional errors as a function of dose but had no effect on memory deficits. Nicotine also had no effect on digit span forward test in any group.
Rusted & Warburton (1992)	20 smokers (13 men, 7 women)	Not blind; subjects were tobacco deprived for at least 1 hr; subjects either smoked ad lib one preferred-brand cigarette or sham smoked an unlit cigarette and engaged in a distractor task or not in four separate randomized sessions; smoking and distraction occurred during 10 min after list presentation and before recall	Delayed free recall of 24-word list presented 10 min earlier	Smoking had no effect on number of words recalled, and fewer words were recalled with distraction than no distraction. With no distraction, subjects in smoking condition recalled an average of 1.2 words more than in sham-smoking condition.
Sherwood, Kerr, & Hindmarch (1992)[a]	13 smokers (6 men, 7 women)	Single-blind; subjects were overnight tobacco deprived; nicotine polacrilex gum (0 or 2 mg) was chewed for 20 min at three times at 1-hr intervals in two separate counterbalanced sessions; testing conducted before and 30 min after each of the three doses	Sternberg test: reaction time to probe of four digits (mean of 24 trials)	Reaction time was faster after all three nicotine doses compared with predose baseline in test of short-term memory scanning ability. No changes observed in placebo gum condition.

Table 4 (*continued*)

Reference	Subjects	Design	Measure	Main findings
Spilich, June, & Renner (1992)				
Exp. 3	63 students (21 nonsmokers, 42 smokers)	Not blind; 21 smokers were tobacco deprived for 3 hr; 21 other smokers took 12 paced puffs (one puff every 25 s) from a 1.2-mg nicotine cigarette immediately before testing; deprived smokers and nonsmokers sham smoked an imaginary cigarette; several practice trials preceded test trials	Sternberg test: reaction time to probe of one to six digits (mean of 84 trials)	Nonsmokers had faster reaction time than either active or deprived smokers, who did not differ. Nonsmokers also made fewer errors than smokers, and deprived smokers made fewer errors than active smokers. Reaction time increased as a function of digit span length for all groups.
Exp. 4	60 students (20 nonsmokers, 40 smokers)	Same as Exp. 3, except that 20 smokers were tobacco deprived and 20 smoked before reading aloud a story and subsequent oral free recall of story	Recall of propositions of a story	Nonsmokers recalled more propositions than smokers, and deprived smokers recalled more than active smokers. Nonsmokers recalled more central than trivial themes, whereas active and deprived smokers recalled central and trivial themes about equally.
Warburton, Rusted, & Fowler (1992)				
Exp. 1[a]	20 smokers, gender not indicated (Figure 1 indicates $n = 26$)	Double-blind; subjects were tobacco deprived for at least 10 hr; tobacco cigarettes (0.1 and 0.6 mg nicotine) were smoked in two separate randomized sessions; subjects took one puff before a 10-s rehearsal of each of eight sets of four words	Immediate free recall of 32-word list	Difference scores between low- and high-nicotine cigarette conditions showed 15 subjects recalled more words (M = 3.3) after nicotine, 7 recalled more (M = 2.0) after placebo, and 4 showed no difference.
Exp. 2[a]	20 smokers, gender not indicated	Same as Exp. 1, except subjects engaged in 10-min distractor test after rehearsal of last set of four words	Delayed free recall of 32-word list after 10-min distractor test	Difference scores between low- and high-nicotine cigarette conditions showed 14 subjects recalled more words (M = 3.4) after nicotine, 5 recalled more (M = 2.6) after placebo, and 1 showed no difference.
Warburton, Rusted, & Müller (1992)[a]	20 smokers, gender not indicated	Double-blind; subjects were tobacco deprived for at least 10 hr; nicotine (0 and 1.5 mg) tablets were administered in two separate randomized sessions; 10 min after nicotine, subjects rehearsed each of eights sets of four words for 10 s	Delayed free recall of 32-word list after 10-min distractor test	Difference scores between nicotine and placebo conditions showed 14 subjects recalled more words (M = 3.5) after nicotine, 3 recalled more (M = 2.3) after placebo, and 3 showed no difference.
Heishman, Snyder, & Henningfield (1993)[a]	16 nonsmokers (9 men, 7 women)	Double-blind; nicotine polacrilex gum (0, 2, and 4 mg) was administered in ascending dose order at 90-min intervals in one session; testing conducted once before and after each 15-min gum-chewing period	Digit recall test: mean of correct trials and response time per trial	Nicotine had no effect on either percent of trials in which subject correctly determined digit in eight-digit span missing from nine-digit span presented 3 s earlier or on mean response time per trial.
		Other cognitive abilities		
Elgerot (1976)	12 smokers (8 men, 4 women)	Not blind; subjects were tobacco deprived for 15 hr and during testing in one session and were not deprived and smoked ad lib (two to three preferred-brand cigarettes) during testing in a second session; sessions were 1 week apart and counterbalanced	Raven Progressive Matrices; letter reasoning; mental arithmetic	In all three tests, subjects correctly solved more items during the tobacco-deprived condition compared with the smoking condition.

(table continues)

Table 4 (*continued*)

Reference	Subjects	Design	Measure	Main findings
Dunne, MacDonald & Hartley (1986)[a]	16 female nonsmokers	Double-blind; nicotine polacrilex gum (0 and 4 mg) administered in two separate randomized sessions; word and number problem-solving testing lasted for 50 min after 30-min gum-chewing period	Word and number completion problems	Nicotine had no effect on generation of correct answers to word and number problems, whether easy or difficult.
Snyder & Henningfield (1989)[a]	6 male smokers	Double-blind; subjects were tobacco deprived for 12 hr; nicotine polacrilex gum (0, 2, and 4 mg) administered in three separate counterbalanced sessions; testing conducted at baseline (stable practice) and once after 10-min gum-chewing period	Logical reasoning test; serial addition–subtraction	Mean response time was slower than baseline after 0-mg nicotine for both tests. In logical reasoning, 2- and 4-mg doses returned performance to baseline but produced faster responding than baseline in math test. Nicotine had no effect on test accuracy.
Pritchard (1991)	15 smokers (7 men, 8 women) and 15 nonsmokers (8 men, 7 women)	Not blind; smokers were overnight tobacco deprived; smokers smoked ad lib one 1.1-mg nicotine cigarette; testing conducted twice before and twice after smoking or resting (nonsmokers)	Mental arithmetic: counting forward and backward by three's for 30 s	In nonsmokers, the number of counts made increased over the four trials. Post hoc differentiation of smokers into light and deep inhalers indicated no change after smoking for deep inhalers and a slight increase for light inhalers.
Landers, Crews, Boutcher, Skinner, & Gustafsen (1992)				
Exp. 2	20 male smokeless tobacco users and 20 male nonusers	Not blind; smokeless tobacco users were tobacco deprived for at least 10 hr; users were administered no tobacco, one third of their average dose, average dose, or one and two thirds of average dose on four separate randomized sessions; nonusers received nothing; tests performed 4 min after tobacco administration	Mental arithmetic: multiply or divide two-digit numbers	Smokeless tobacco users correctly answered more problems than nonusers, but the improvement was not dose related.
Heishman, Snyder, & Henningfield (1993)[a]	16 nonsmokers (9 men, 7 women)	Double-blind; nicotine polacrilex gum (0, 2, and 4 mg) administered in ascending dose order at 90-min intervals in one session; testing conducted once before and after each 15-min gum-chewing period	Logical reasoning test; serial addition–subtraction	Nicotine had no effect on percentage of correct trials and mean response time on logical reasoning test and on mental arithmetic problems requiring addition or subtraction of single digits.
Morris & Gale (1993)				
Exp. 2	14 male smokers	Not blind; subjects were tobacco deprived for 1 hr; subjects either smoked ad lib preferred-brand cigarettes or did not smoke in two separate sessions; smoking occurred during 7-min test (14 problems)	Raven Progressive Matrices	Smoking had no effect on number of correct responses.

Note. Exp = Experiment.
[a]Included in section on studies using rigorous methodology.

were minimally abstinent (1–2 hr) before experimental sessions. Mangan (1983, Experiment 1) reported that smoking had no effect on acquisition of word pairs immediately after smoking but produced fewer errors 30 min later. Acquisition testing preceded smoking in two studies, which reported conflicting results. Mangan and Golding (1983) found that nonsmokers made fewer errors than smoking groups when retested at 30 min, 1 day, and 1 week. In contrast, Colrain et al. (1992) reported smoking reduced errors 1 week after initial acquisition. Smokers were abstinent for 8–10 hr in two studies assessing rote learning of nonsense syllables; both reported impaired responding immediately after smoking (Andersson, 1975; Andersson & Post, 1974).

No deprivation. Roth, Lutiger, Hasenfratz, Bättig, and Knye (1992) compared nonabstinent smokers who smoked at testing and 11-hr abstinent smokers who did not smoke at testing and found no difference between the groups on maze learning and recall 1 hr later.

Summary of learning. There is little evidence that smoking and nicotine enhance learning abilities. Only one of seven studies reported an unambiguous improvement from smoking (Colrain et al., 1992). It is difficult to generalize from results on the basis of only three experimental models to learning on a broader scale. Clearly, more research is needed in which other paradigms are used to clarify nicotine's effect, if any, on the initial acquisition of information.

Memory

Memorial functions were measured in 25 studies (32 experiments). The most common method of assessing memory was to present visually or auditorily a list of words or numbers to subjects and require immediate or delayed recall of all or part of the stimulus information. This paradigm was used in 23 experiments, yielding a measure of response accuracy (number of correct responses or errors) and, in some studies, response time. Six experiments used a test of information retrieval from short-term memory, in which the primary measure was the time to respond to whether a probe digit was present or absent from an original series of digits (Sternberg, 1966).

Nicotine deprivation. Smokers were abstinent for 1–24 hr before testing in 22 of the 32 experiments. Length of deprivation did not consistently affect results. Thirteen experiments reported that smoking or nicotine had no effect on some subjects or aspect of memory, 9 found enhancement, and 7 reported impairment. Firm conclusions are elusive when differential effects of nicotine on various measures of memory are found in one third of the experiments. It is clear, however, that nicotine does not enhance memory under a broad range of experimental conditions.

No deprivation. Ten experiments included nonabstinent smokers and nonsmokers. Two studies were conducted with nonsmokers diagnosed with Alzheimer's disease. Newhouse et al. (1988) reported that a moderate dose of intravenous nicotine improved delayed recall of a word list but had no effect on retrieval of word categories. Jones et al. (1992) found that subcutaneous injections of nicotine reduced attentional, but not memorial, errors in a delayed matching test. Of the nine experiments testing healthy subjects, four reported no effect of nicotine (Heishman et al., 1993; Hindmarch et al., 1990, Experiments 1 and 2; Jones et al., 1992), three found impairment (Dunne, MacDonald, & Hartley, 1986; Spilich et al., 1992, Experiments 3 and 4), one reported enhancement (Kerr et al., 1991), and one found enhanced reaction time to a longer, but not shorter, digit span condition of the Sternberg test (West & Hack, 1991).

Summary of memory. There is no consensus on the effects of smoking and nicotine on memory. Under conditions of nicotine deprivation and no deprivation, a roughly equal number of experiments indicated that smoking and nicotine either had no effect, enhanced, or impaired memorial functions. Nearly one third of the 32 experiments reported varying effects as a function of the dependent measures or individual subject differences.

Other Cognitive Abilities

Nicotine deprivation. Abstinent tobacco users were tested in four studies. Landers et al. (1992, Experiment 2) reported that smokeless tobacco enhanced mental arithmetic ability in 10-hr abstinent users, but the effect was not dose-related. In contrast, Pritchard (1991) found no effect of smoking on mental arithmetic in overnight-deprived smokers, and Morris and Gale (1993, Experiment 2) reported no effect of smoking on the Raven

Progressive Matrices test in 1-hr abstinent smokers. In the only study that measured baseline performance before enforced abstinence, Snyder and Henningfield (1989) reported that nicotine polacrilex enhanced response time above baseline in an arithmetic test but had no effect on accuracy. Snyder and Henningfield also reported no effect of polacrilex on either speed or accuracy in a test of logical reasoning.

No deprivation. In two studies, polacrilex was administered to nonsmokers and results showed no effect on problem-solving skills (Dunne et al., 1986) or on tests of mental arithmetic and logical reasoning (Heishman et al., 1993). Elgerot (1976) found that nonabstinent smokers who smoked during testing solved fewer items on the Raven Progressive Matrices, reasoning, and mental arithmetic tests compared with when they were abstinent.

Summary of other cognitive abilities. In tobacco-abstinent smokers, two studies reported that smokeless tobacco and nicotine polacrilex enhanced mathematical abilities. However, three studies indicated that smoking and nicotine had no effect on mathematical and reasoning abilities. Similarly, three studies with nonabstinent smokers or nonsmokers reported no effect or impaired performance on tests of mathematical, reasoning, and problem-solving abilities. Thus, there is little evidence for smoking and nicotine to enhance these cognitive abilities.

Summary of Effects of Nicotine and Smoking on Performance

Nicotine Deprivation

As discussed, results of studies conducted with nicotine-deprived smokers are difficult to interpret. Without predeprivation baseline data, which few studies reported, it is difficult to conclude whether nicotine functioned to reverse deprivation-induced deficits or to enhance performance beyond that observed in the nonabstinent state. Most, but not all, studies in the areas of sensory abilities, finger tapping, selective attention, and sustained attention found a facilitating effect of nicotine and smoking in abstinent smokers. In contrast, half or fewer of the studies reported positive effects in tests of hand steadiness, focused and divided attention, learning, memory, and other cognitive abilities. Thus, in abstinent smokers, nicotine and smoking may at least reverse deprivation-induced deficits in certain abilities, but such beneficial effects have not been observed consistently across a range of performance measures.

No Deprivation

Studies conducted with nonabstinent smokers or nonsmokers provide a stronger test of the hypothesis that nicotine and smoking absolutely enhance performance. Such studies indicated that nicotine reliably enhanced finger-tapping rate and produced modest, but limited, improvement in a test of divided attention. In all other areas of performance, the evidence for an enhancing effect of nicotine was weak or nonexistent.

Studies Using Rigorous Experimental Methodology

Because of the preponderance of methodological shortcomings in this literature, a secondary analysis was conducted of studies that, in our opinion, used rigorous experimental methodology. Results of these studies would presumably form a strong basis for conclusions concerning the effects of smoking and nicotine on performance. The primary selection criterion was use of a placebo control, and a secondary criterion was use of single- or double-blind procedures. Twenty-five studies (31 experiments) met these criteria and are identified in Tables 1–4. All experiments were placebo-controlled, and all but two administered nicotine under blind conditions. Other positive design features were frequently used. For example, nonsmokers or nonabstinent smokers were tested in 18 experiments and nicotine was administered in a form other than smoking (i.e., nasal solution or spray, polacrilex gum, or tablets) in 24 experiments. The seven experiments in which subjects smoked used nicotine-free or denicotinized cigarettes as placebos.

Sensory Abilities

Nicotine Deprivation

Sherwood et al. (1992) administered nicotine polacrilex (0 and 2 mg) to overnight-deprived

smokers three times at 1-hr intervals, measuring CFF after each administration. They found that CFF threshold was increased over predose baseline after the first 2-mg dose, but no further increase after the second and third doses was observed. This suggests that the initial dose reversed deprivation-induced deficits, and subsequent doses maintained normal functioning. Baseline CFF was not measured in a nonabstinent state.

No Deprivation

Nicotine was administered through polacrilex or subcutaneous injection to nonabstinent smokers and nonsmokers in four experiments, and all reported no effect of nicotine on CFF (Hindmarch et al., 1990, Experiments 1 & 2; Jones et al., 1992; Kerr et al., 1991). This lack of effect of nicotine in the absence of nicotine deprivation is consistent with the data of Sherwood et al. (1992), further suggesting that nicotine functions to reverse withdrawal-induced deficits but does not produce absolute enhancement in tests of CFF threshold.

Motor Abilities

Nicotine Deprivation

Perkins et al. (1990) administered placebo and nicotine (15 μg/kg) nasal spray to smokers who were tobacco deprived for at least 12 hr. They reported that nicotine reliably increased finger-tapping rate in all subjects and produced a nonsignificant trend toward improved hand steadiness.

No Deprivation

In six experiments, nonsmokers were administered nicotine nasal solution or spray (Perkins et al., 1990; West & Jarvis, 1986, Experiments 1, 2, 3, & 4) or subcutaneous injection (Jones et al., 1992). Finger-tapping rate was increased by nicotine in all experiments. Perkins et al. also reported that nicotine nasal spray produced a nonsignificant trend toward impairment in nonsmokers on a test of hand steadiness.

Attentional Abilities

Focused Attention

Nicotine deprivation. Sherwood et al. (1992) administered polacrilex (0 and 2 mg) to overnight-deprived smokers three times at 1-hr intervals and measured recognition and motor reaction time after each administration. They reported that recognition time was not affected by nicotine, but motor reaction time was increasingly faster after the second and third doses compared with prenicotine baseline. However, it should be noted that the mean predose baseline assessment for the nicotine session was 29 ms slower than that for the placebo session, such that motor time after the third nicotine dose was only 5 ms faster than the predose baseline score in the placebo session. Post hoc comparisons between nicotine and placebo sessions were not reported.

No deprivation. The Hindmarch group (Hindmarch et al., 1990; Kerr et al., 1991) examined the effect of nicotine polacrilex on choice reaction time, again differentiating between recognition and motor components. Testing nonsmokers, Hindmarch et al. (1990, Experiment 1) reported no effect of 2-mg polacrilex on test performance. However, in nonabstinent smokers (Hindmarch et al., 1990, Experiment 2) and a group of nonabstinent smokers and nonsmokers (Kerr et al., 1991), polacrilex produced faster motor reaction time but did not affect recognition time. Between-session baseline differences were not large in these two studies.

Selective Attention

Nicotine deprivation. Snyder and Henningfield (1989) tested smokers before enforced abstinence and administered polacrilex (0, 2, and 4 mg) after 12 hr of tobacco deprivation. They found that deprivation (0-mg condition) produced slowed responding on a letter-searching test and that 2 and 4 mg of polacrilex returned performance to predeprivation baseline. Parrott and Craig (1992) reported that polacrilex (2 and 4 mg) had no effect on Stroop and letter cancellation tests in 12-hr deprived smokers.

No deprivation. Three studies tested the effects of nicotine in nonsmokers. Using the Stroop test, Wesnes and Revell (1984, Experiment 2) found no

effect of 1.5-mg nicotine tablets, whereas Provost and Woodward (1991) reported faster response time after 2 mg of polacrilex. Heishman et al. (1993) found no effect of polacrilex (2 and 4 mg) on letter-searching response time or accuracy.

Divided Attention

Nicotine deprivation. Using a test that required subjects to perform a central tracking task and respond to peripheral visual stimuli, Sherwood et al. (1992) found that 2 mg of polacrilex decreased tracking errors but had no effect on reaction time to the peripheral lights in overnight deprived smokers. Tracking errors were more reduced after the third nicotine dose compared with the first dose and placebo responding was unchanged, suggesting an absolute enhancement of performance.

No deprivation. Using the same divided attention paradigm with nonabstinent smokers and nonsmokers, Hindmarch et al. (1990, Experiment 1) and Kerr et al. (1991) also found that polacrilex decreased errors on the tracking task but had no effect on reaction time to peripheral stimuli. Testing nonsmokers only, Hindmarch et al. (1990, Experiment 2) reported no effect of 2 mg of polacrilex on either tracking or reaction time performance.

Sustained Attention

Nicotine deprivation. The RVIP test was used in four experiments. Wesnes and Warburton (1983a, Experiment 2) reported that test accuracy and reaction time were improved in overnight deprived smokers after smoking active cigarettes compared with a nicotine-free cigarette. In smokers who were tobacco deprived for 3–12 hr, two studies (Parrott & Craig, 1992; Parrott & Winder, 1989) found that correct responding on the RVIP test was improved by polacrilex (2 and 4 mg) relative to placebo, but reaction time was not affected. Michel, Hasenfratz, Nil, and Bättig (1988) tested the effect of polacrilex (0 or 4 mg) in 2-hr abstinent smokers and found that RVIP test accuracy and reaction time were not affected by nicotine. Lastly, Wesnes et al. (1983) administered nicotine tablets (0, 1, and 2 mg) to 12-hr deprived smokers three times during an 80-min vigilance test. Signal detection declined from baseline in all dose conditions, but the decline was less with active nicotine compared with placebo.

No deprivation. Nonsmokers were administered nicotine tablets or injections in five experiments. Four experiments found that nicotine had no effect on the RVIP test compared with placebo conditions (Jones et al., 1992; Wesnes & Revell, 1984, Experiments 1 & 2; Wesnes & Warburton, 1984b, Experiment 2). Nonsmokers were also administered nicotine tablets in the Wesnes et al. (1983) study, in which vigilance performance declined less after nicotine than placebo. There was no difference between abstinent smokers and nonsmokers, suggesting that nicotine functioned to reverse a vigilance decrement and potential withdrawal-induced deficits in the 12-hr abstinent smokers.

Cognitive Abilities

Learning

Two studies investigated the effect of smoking in nicotine-deprived smokers. Using a verbal rote-learning paradigm, Andersson and Post (1974) presented a list of nonsense syllables 30 times to overnight-deprived smokers. One cigarette (active or nicotine-free) was smoked after the 10th and 20th trial. Following the first cigarette, anticipatory responding was improved in the placebo compared with the nicotine condition; after the second cigarette, there was no difference between conditions. Because of the decreased scores after the first nicotine cigarette, the gain in learning from Trials 11–20 to Trials 21–30 was greater for the nicotine condition compared with placebo. In a study by Colrain et al. (1992), smokers who were 2-hr abstinent learned a list of word pairs to criterion, then rested or smoked one cigarette with a nicotine yield of 0.07 (denicotinized), 0.6, or 1.1 mg nicotine. When retested 1 week later, number of errors to criterion was reduced by nicotine in a dose-related manner.

Memory

Nicotine deprivation. Abstinent smokers were tested in nine experiments. In each of three experiments conducted with 10-hr abstinent smokers (Warburton, Rusted, & Fowler, 1992, Experiments 1 & 2; Warburton, Rusted, & Müller, 1992),

individual differences were evident, such that most subjects recalled more words after nicotine tablets or cigarettes, yet some subjects' recall improved after placebo tablets or denicotinized cigarettes and some showed no difference between conditions. Reaction time on the Sternberg (1966) memory test was faster after smoking (Sherwood et al., 1992) and polacrilex (West & Hack, 1991) compared with placebo conditions. In contrast to these positive effects of nicotine on memory, three studies reported no effect of nicotine tablets or polacrilex on tests of immediate and delayed recall (Parrott & Winder, 1989; Rusted & Eaton-Williams, 1991; Snyder & Henningfield, 1989). Finally, Houston et al. (1978) reported that immediate and delayed recall of 3-hr abstinent smokers was impaired after smoking a nicotine cigarette compared with a nicotine-free cigarette.

No deprivation. The eight well-designed experiments were discussed in the preceding narrative of the entire literature. Briefly, two experiments reported that nicotine improved some aspects of memorial functioning in patients with Alzheimer's disease (Jones et al., 1992; Newhouse et al., 1988), two found enhanced reaction time on the Sternberg test (Kerr et al., 1991; West & Hack, 1991), four reported no effect of nicotine on tests of immediate and delayed recall (Heishman et al., 1993; Hindmarch et al., 1990, Experiments 1 & 2; Jones et al., 1992), and one found that polacrilex impaired immediate and delayed recall and recognition memory (Dunne et al., 1986).

Other Cognitive Abilities

Nicotine deprivation. Testing 12-hr abstinent smokers, Snyder and Henningfield (1989) reported that polacrilex (2 and 4 mg) enhanced response time above predeprivation baseline in an arithmetic test but had no effect on accuracy. Polacrilex also had no effect on either speed or accuracy in a test of logical reasoning.

No deprivation. Two studies conducted with nonsmokers reported no effect of nicotine on several cognitive tests. Dunne et al. (1986) found that 4 mg of polacrilex did not affect the ability to generate correct answers to word and number problems. Heishman et al. (1993) reported that polacrilex (2 and 4 mg) had no effect on response time and accuracy on tests of logical reasoning and mental arithmetic.

Summary of Studies Using Rigorous Methodology

Nicotine Deprivation

The issue of distinguishing between nicotine producing reversal of deprivation-induced performance deficits and absolute enhancement remains a methodological problem in placebo-controlled studies conducted with abstinent smokers. An additional problem in drawing conclusions from studies in this category is that only one rigorous study was identified in each of six areas of performance (sensory; motor; focused, selective, and divided attention; and other cognitive abilities). About half of the studies assessing sustained attention and memory reported a positive effect of nicotine; however, the effects were limited to some subjects or one aspect of test performance.

No Deprivation

The strongest conclusions concerning the effects of nicotine and smoking on human performance can be drawn from studies in this category. These studies indicated that nicotine absolutely enhanced finger-tapping rate and motor responding in tests of focused and divided attention. On the basis of more limited evidence, nicotine produced faster motor responses in the Sternberg (1966) test of memory and reversed the vigilance decrement in a sustained attention test. However, no studies reported absolute enhancement of sensory abilities, hand steadiness, selective and sustained attention, learning, and other cognitive abilities. Thus, on the basis of placebo-controlled studies conducted with nonabstinent smokers or nonsmokers, nicotine and smoking reliably enhanced only motor abilities, including motor responses to visual stimuli in brief tests of attention.

A Behavioral Pharmacological Analysis: Modulating Variables

That nicotine's effects on performance differ under conditions of nicotine deprivation versus no deprivation should not be surprising when one considers the behavioral pharmacological principle that the effect of a drug in an individual is an interactive function of organismic and environmental conditions (Thompson & Schuster, 1968). Thus,

the mechanisms by which a drug alters performance are varied and complex (Heishman & Henningfield, 1991). Some of the numerous interacting variables that determine the ultimate effect of a drug include: (a) drug dose and route of administration; (b) physiological and psychological state of the individual, including degree of previous experience with the drug; (c) reinforcement contingencies maintaining behavior, and (d) nature and demands of the performance test. The effects of nicotine on performance will be discussed in terms of these variables.

Nicotine Dose

A basic principle in pharmacology is that magnitude of effect varies with drug dose. However, the dose-response function of nicotine has not been adequately characterized for measures of human performance. Because of the difficulties associated with effective dose control during cigarette smoking, we examined only placebo-controlled studies in which at least two active doses of nicotine were administered in a form other than smoking.

Of the 11 studies that met these criteria, 8 involved the administration of two active nicotine doses (Heishman et al., 1993; Hindmarch et al., 1990, Experiment 1; Parrott & Craig, 1992; Parrott & Winder, 1989; Rusted & Eaton-Williams, 1991; Snyder & Henningfield, 1989; Wesnes et al., 1983; West & Jarvis, 1986, Experiment 2), and 3 studies administered three active doses (Jones et al., 1992; Newhouse et al., 1988; Wesnes & Warburton, 1984b, Experiment 2). Most studies measured several aspects of performance, and all categories of performance were represented. Seven studies reported no effect of nicotine dose on some or all performance tests (Heishman et al., 1993; Hindmarch et al., 1990, Experiment 1; Jones et al., 1992; Parrott & Craig, 1992; Parrott & Winder, 1989; Rusted & Eaton-Williams, 1991; Wesnes & Warburton, 1984b, Experiment 2). Three studies reported nicotine effects that were comparable in magnitude for all active doses (Hindmarch et al., 1990, Experiment 1; Jones et al., 1992; Wesnes et al., 1983). However, dose-related effects were observed in 7 studies. In 6 of these studies, dose-response curves were essentially linear for various measures across all performance categories (Hindmarch et al., 1990, Experiment 1; Jones et al., 1992; Newhouse et al., 1988; Parrott & Winder, 1989; Snyder & Henningfield, 1989; West & Jarvis, 1986, Experiment 2). In two studies, dose effects were curvilinear; optimal performance was observed at mid-range nicotine doses in tests of sustained attention (Parrott & Craig, 1992) and finger tapping (Jones et al., 1992).

That only 11 studies administered placebo and multiple doses of nicotine in a form other than tobacco cigarettes and that only 1 study (Hindmarch et al., 1990) measured plasma nicotine concentration suggest that more research is needed before definitive answers are reached concerning the relation between delivered dose of nicotine and magnitude of performance effect.

Physiological and Psychological State

The most frequently manipulated state variable among the reviewed studies was tobacco deprivation. This variable was not investigated systematically in most studies, rather, subjects were required to abstain from smoking for some period of time before experimental sessions, and verification of abstinence was based only on a verbal response from subjects. In general, this review has shown that facilitating effects of nicotine were more frequently reported in studies with abstinent smokers than in those testing nonabstinent smokers or nonsmokers. These salutary effects indicate nicotine's ability to reverse deprivation-induced deficits in concentration and task performance.

Another state-related issue is the possibility that nonsmokers respond differently to the effects of nicotine than smokers. Numerous studies tested smokers who smoked and nonsmokers who did not smoke (e.g., Mangan & Golding, 1983; Spilich et al., 1992); however, nicotine was administered to smokers and nonsmokers and their performance compared under identical test conditions in only six studies. All of these studies administered nicotine to nonsmokers in a form other than tobacco cigarettes for ethical and scientific reasons. Two studies reported differential responding (Hindmarch et al., 1990; Perkins et al., 1990), whereas four found no differences (Jones et al., 1992; Kerr et al., 1991; Wesnes et al., 1983; Zdonczyk et al., 1988). Further research comparing performance effects of nicotine in smokers and nonsmokers may elucidate important behavioral processes underlying individual differences in the etiology of tobacco use and dependence.

Reinforcement Contingencies

The field of behavioral pharmacology has used many of the principles of operant psychology in analyzing experimentally the interactions of drugs and behavior. One of the most important determinants of behavior are the reinforcement contingencies maintaining that behavior (Thompson & Schuster, 1968). The arrangement specifying the relation between the behavior (response) of interest and reinforcement is referred to as a schedule of reinforcement. Because an individual's performance can vary due to many factors, it would seem that studying test performance under explicitly defined reinforcement (e.g., monetary) contingency conditions would be important to an analysis of a drug's effect on performance. However, only three studies (Perkins et al., 1990; Pritchard et al., 1992; Schori & Jones, 1975) used some form of reinforcement contingency to maintain optimal performance, and none compared behavior under varying reinforcement schedules or under contingent versus noncontingent conditions. With so few studies using an explicit schedule of reinforcement, any conclusion concerning the effectiveness to maintain stable performance would be premature. Future studies should compare performance with and without reinforcement contingencies and incorporate such schedules of reinforcement whenever possible.

Nature and Demands of the Performance Test

Studies have shown that performance tests are differentially sensitive to the effects of drugs. For example, ethanol and several benzodiazepines impaired performance to a greater extent and for a longer time on difficult versions of a tracking task and Digit Symbol Substitution Test compared with easier versions of the tests (Linnoila et al., 1990; Nikaido, Ellinwood, Heatherly, & Gupta, 1990). Using the repeated acquisition of behavioral chains test, which allows a separate analysis of learning and performance, Higgins and colleagues (Bickel, Hughes, & Higgins, 1990; Higgins et al., 1992; Higgins, Woodward, & Henningfield, 1989) reported that ethanol, atropine, and benzodiazepines impaired acquisition of novel responses without adversely affecting repeated performance of the same response. These studies suggested that difficult versions of a test or aspects of tests requiring greater cognitive processing (learning vs. performance) were more sensitive to the impairing effects of various drugs.

The effects of smoking or nicotine have been assessed in several studies that varied test difficulty. In 15 studies (16 experiments), test difficulty was directly manipulated or allowed to vary according to accuracy of performance (Dunne et al., 1986; Hasenfratz & Bättig, 1992; Hasenfratz, Jaquet, Aeschbach, & Bättig, 1991; Hasenfratz, Michel, et al., 1989; Hasenfratz, Pfiffner, et al., 1989; Knott, 1985a; Kucek, 1975; Michel & Bättig, 1989; Michel et al., 1988; Nil et al., 1988; Rusted & Warburton, 1992; Schori & Jones, 1974, 1975; Spilich et al., 1992, Experiments 1 & 3; West & Hack, 1991). In 6 of the 9 experiments in which level of difficulty was manipulated, smoking or nicotine condition did not interact with test difficulty (Dunne et al., 1986; Knott, 1985a; Kucek, 1975; Rusted & Warburton, 1992; Spilich et al., 1992, Experiments 1 & 3). In 2 of the other 3 studies, smoking produced faster reaction time in difficult compared with easy test versions (Hasenfratz & Bättig, 1992; West & Hack, 1991), whereas 1 study found slower responding after smoking in the less complex test (Schori & Jones, 1974). Of 6 studies using a modified version of the RVIP test, which increased in difficulty as performance accuracy increased, 2 reported that smoking enhanced performance (Hasenfratz et al., 1991; Hasenfratz, Michel, et al., 1989), 1 showed smoking produced less of a decline in performance than not smoking (Nil et al., 1988), and three reported no effect (Hasenfratz, Pfiffner, et al., 1989; Michel & Bättig, 1989; Michel et al., 1988). Schori & Jones (1975) found that smoking had no effect on level of difficulty attained on a divided attention test. Thus, although nicotine effects can vary according to task difficulty, the majority of studies did not indicate that difficult tests were more sensitive than easier tests to the effects of smoking or nicotine.

Comparing across and within studies in Tables 1–4, there is no evidence that tests demanding cognitive abilities are more sensitive to the effects of nicotine compared with those measuring motor or attentional behavior. For example, of the 13 studies assessing cognitive and motor or attentional abilities in the same study (Carter, 1974; Elgerot, 1976; Heishman et al., 1993; Hindmarch et al., 1990; Jones et al., 1992; Kerr et al., 1991;

Landers et al., 1992; Morris & Gale, 1993; Parrott & Winder, 1989; Sherwood et al., 1992; Snyder & Henningfield, 1989; Spilich et al., 1992; Williams, 1980), none reported that smoking or nicotine altered performance on the cognitive test and not on the motor or attentional test. Additionally, there is little evidence that nicotine differentially affects cognitive versus motor abilities when both are required in the same test. For example, the RVIP test involves some degree of cognitive processing to detect a target stimulus, as do all attentional tests, and a timed motor response. Of the 19 experiments using the RVIP test in healthy subjects (Table 3, Sustained attention), only 5 reported that smoking or nicotine affected accuracy and not reaction time.

Concluding Comments

The purpose of this review was to examine the effects of nicotine administration and cigarette smoking on human performance to clarify the role of nicotine-induced effects as determinants of the initiation and maintenance phases of smoking behavior. The distinction between studies conducted with nonabstinent smokers or nonsmokers and those conducted with abstinent smokers can provide some insight.

In nonabstinent smokers and nonsmokers, nicotine produced absolute enhancement of finger-tapping rate and motor responses in brief tests of attention. All other aspects of performance, including cognitive functioning, were not reliably enhanced. These enhancing effects do not appear to be of sufficient generality or magnitude to explain why people find cigarette smoking highly reinforcing during the initiation phase of tobacco dependence.

In contrast, studies with abstinent smokers reported a greater frequency of nicotine enhancement. Although critical predeprivation baseline data was missing from the majority of such studies, these positive effects indicated that nicotine can reverse deprivation-induced deficits in sensory abilities, finger tapping, and selective and sustained attention. These data support the conclusion that nicotine deprivation is a condition that controls the maintenance of cigarette smoking in nicotine-dependent persons and that one mechanism of this behavioral control is that nicotine functions to restore deprivation-induced deficits in performance (USDHHS, 1988).

The methodological problems inherent in much of this literature limit the extent to which firm conclusions can be drawn. Placebo control conditions were used in only 24% of experiments and single- or double-blind testing conditions were maintained in 26% of experiments. An imprecise method of nicotine dosing, ad lib cigarette smoking, was used in 70% of studies, and smokers who were tobacco deprived for more than 4 hr were tested in at least 65% of experiments. Studies in which an unknown dose of nicotine is administered to individuals in withdrawal who know they are smoking contribute little to our understanding of the performance effects of nicotine.

Although it appears that studies with small sample sizes did not result in a disproportionate number of null outcomes, it should be noted that our emphasis was on the overall tests of significance and not on the effect size of the independent variable and the power of the statistical test to detect that effect. Determining the average statistical power of a research domain can provide insight into a pattern of significant results. For example, if power is around .50, some studies will report significance and others will not (Rossi, 1990). Such inconsistency is clearly evident in the nicotine and performance literature and may be due to marginal statistical power. It seems appropriate to further the critical analysis of this literature by conducting a meta-analysis, in which effect size is calculated for each study and close attention is paid to study characteristics (moderating variables) that may be associated with effect size. Such a research synthesis provides a systematic means for determining the presence or absence of an effect, the magnitude and directionality of the effect, and the specific conditions under which the effect is likely to occur (Cooper & Hedges, 1994). We plan to conduct a research synthesis on this literature to enhance the discussion of research issues in the present review.

To delineate more clearly the conditions under which nicotine functions to enhance or impair performance will require more research on the interactive influences of nicotine dose and route of administration, state of the individual, reinforcement contingencies maintaining performance, and the nature of the performance test. Specifically, future research should attempt to (a) use nicotine

delivery systems other than tobacco cigarettes to control dosage more effectively; (b) measure nicotine plasma concentrations, especially if cigarettes are used, to verify delivered dose; (c) compare the performance of smokers in nicotine-deprived and nondeprived states; (d) test nonsmokers to examine nicotine effects in the absence of chronic tolerance and dependence; (e) manipulate reinforcement contingencies under which subjects perform to examine potential interactive motivational effects with nicotine; and (f) use a variety of tests that span the range of human abilities to further our understanding of the effects of nicotine on human performance.

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