Motivational Influences on Cigarette Smoking

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■ **Abstract**   Cigarette smoking is a leading cause of mortality and morbidity and a particularly common and intractable addictive disorder. Research shows that nicotine is a sine qua non of tobacco addiction and that it produces the hallmark effects of addictive drugs: sensitization, tolerance, physical dependence, and euphoria/elation. Research on the development of smoking reveals that although smoking prevalence has declined from a peak in the mid-1990s, close to 30% of twelfth graders still smoke. Smoking in adolescents is related to development of physical dependence, ethnicity, impulsivity, affective disorder, and peer influences. However, which of these exerts the greatest causal effects is unknown, and their influence no doubt varies across individuals and across development. Once dependence on tobacco smoking is established, evidence suggests that tobacco motivation is strongly influenced by a reduction in withdrawal symptoms, an expectation of stress reduction, and conditioned reinforcement. Nicotine motivation may also be influenced by modulation in stimulus incentive value.

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INTRODUCTION

Cigarette smoking is of great importance. First, it has great societal and clinical importance since it is the leading preventable cause of morbidity and mortality in developed countries (Peto et al. 1992). It is a major cause of cardiac disease, vascular disease, pulmonary disease, and a variety of cancers. Second, cigarette smoking is ubiquitous; about a quarter of all adult Americans smoke cigarettes, and smoking rates are higher in many other countries (CDC Prev. 1999). Finally, cigarette smoking is a prototypic addictive disorder manifesting classic features such as tolerance, withdrawal, and use despite high personal cost. Therefore, if we can discover the causal mechanisms that yield cigarette addiction, this may elucidate causal mechanisms in other addictive disorders.

The present review attempts to provide basic information on the nature of cigarette smoking and its natural history and features. However, the focus of this review is on possible motivational influences on the development and maintenance of smoking. In the first section of this chapter we describe age-related trends in smoking, factors that have been thought to influence its development or uptake, and evidence that youth smoking is associated with a true physical dependence or addiction.

In the second section, we describe the characteristics of heavy, dependent smoking, and review theories and data pertaining to its motivational basis. For instance, we review evidence that dependence on cigarettes might be due to their impact on weight regulation, their cognitive effects, their euphoriant or direct pleasurable effects, their impact on withdrawal symptoms, or their impact on affective reactions to stressors. In reviewing topics in this chapter, we attempt to evaluate the quality of evidence relevant to each topic, identify challenges or constraints that limit inference, and synthesize data with theory.

THE DEVELOPMENT OF TOBACCO SMOKING

Epidemiology of Cigarette Smoking and Tobacco Dependence Among U.S. Youth

PREVALENCE AND TRENDS OVER TIME   National epidemiological data on adolescent tobacco use have been collected using school-based surveys (e.g., Monitoring the Future or MTF, Johnston et al. 2002) and household surveys (e.g., National Household Survey on Drug Abuse or NHSDA, Koppstein 2001). The most recent data suggest that adolescent smoking is fairly common, with 27% of twelfth graders, 18% of tenth graders, and 11% of eighth graders reporting smoking in the past month (Johnston et al. 2002).

This level of smoking prevalence represents a decline from recent peaks in the mid-1990s. Since national surveillance began, there have been significant shifts over time in the prevalence of adolescent smoking. Adolescent smoking increased in the late 1960s (especially among females), peaked in the mid-1970s, and then declined in the late 1970s and 1980s (especially for African Americans; Giovino 1999, Johnston et al. 2002). New increases began in the early 1990s (for both African American and white adolescents) until the mid-1990s, and since then there has been a downturn (Johnston et al. 2002). These dynamic shifts in smoking prevalence are likely driven by complex social forces including changes in the social images of smoking, and changes in price and availability.

Compared to adolescent smoking, much less is known about the epidemiology of adolescent tobacco dependence. "Dependence" is a construct that shares much in common with everyday or prosaic notions of "addiction." Thus, in the Diagnostic and Statistical Manual (DSM) of the American Psychiatric Association (American Psychiatric Association 1994), a diagnosis of dependence requires at least three of seven criteria. These criteria include tolerance and withdrawal, but neither is necessary for the diagnosis. "Tolerance" refers to a decrease in drug effects as a function of repeated use. "Withdrawal" refers to the occurrence of a well-characterized syndrome upon discontinuation of drug use (i.e., falling levels of drug in the body). The other five DSM criteria are that (*a*) the substance is taken in larger amounts or over a longer period of time than was intended, (*b*) the user desires to cut down or control use, or was unable to do so, (*c*) a great deal of time is spent obtaining, using, or recovering from the substance, (*d*) other important activities are compromised due to substance use, and (*e*) use is continued despite knowledge of problematic consequences. Thus, the dependent individual's motivation to use a substance dominates other common motivational tendencies and normal activities.

Dependence per se can be distinguished from "physical dependence": the latter is said to occur when an organism has used sufficient drug so that withdrawal symptoms will be observed should drug use be discontinued or reduced. Thus, physical dependence is a state whose existence is inferred from the observation of withdrawal symptoms once drug use ceases.

In general, adolescent smokers are less likely to be diagnosed with tobacco dependence than are adult smokers (Colby et al. 2000a), although many adolescent smokers consider themselves addicted. Kandel & Chen (2000) used a proxy measure to approximate DSM-IV diagnoses in the NHSDA data. By these criteria, 28.5% of 12- to 17-year-old current smokers (smoking in the past month) would be considered dependent. Dependent adolescents report many of the same symptoms as do dependent adults, including craving, withdrawal, tolerance, and a desire to cut down on smoking (Colby et al. 2000a,b). Compared to adults, adolescents at the same level of self-reported intake were more likely to be diagnosed as dependent, which suggests that adolescents may be especially vulnerable to dependence or sensitive to the effects of nicotine (Kandel & Chen 2000). An important goal of research in this area is the development of valid measures of adolescent tobacco

dependence as the extant adult measures may be inappropriate for youths (Colby et al. 2000a,b).

DEMOGRAPHIC CORRELATES   Demographic correlates of smoking suggest possible influences on smoking development. Greater parental education is associated with less likelihood of smoking in offspring (Giovino 1999). In addition, girls appear more influenced by peer smoking than are boys (Hu et al. 1995, Mermelstein 1999). There are large differences in smoking as a function of race/ethnicity, with the highest smoking rates among American Indian/Alaska Native adolescents, followed by whites and then Hispanics, and the lowest rates among Asians and African Americans. These race/ethnicity differences are seen in both school-based and household surveys, so they are not due to differential school attendance (USDHHS 1998), and they are not eliminated when a bioassay is used to validate self-reported smoking (Giovino 1999).

Although studies that sample multiple ethnic groups are still comparatively few and recent, there have been some suggestions that African American and Asian American adolescents report stronger antismoking socialization messages from parents, and that African American parents report feeling particularly empowered to influence their children's smoking (for reviews, see Mermelstein 1999, USDHHS 1998). Perhaps the most replicated race/ethnicity difference is that peer smoking is a relatively weak predictor of adolescent smoking for African American compared to white adolescents (Griesler & Kandel 1998, Mermelstein 1999).

Age Trends and Age-Related Smoking Trajectories

Smoking shows systematic age-related trends, with use peaking at ages 18–25. Retrospective data from the NHSDA suggest that the average age of first use of cigarettes is 15.4 (11–15 in the MTF data), with the average age of daily use being 18 (Koppstein 2001). Retrospective data from the National Comorbidity Study suggest that the onset of nicotine dependence lags at least one year after the onset of daily smoking (Breslau et al. 2001). After the mid-twenties, declines in smoking occur but these declines are modest in comparison to other forms of substance use, perhaps because cigarette smoking is highly addictive, legal, and not immediately performance-impairing (Chassin et al. 2000).

In addition to limitations associated with the retrospective nature of these data, the data are limited in that they describe a single "average" trajectory of age-related changes in smoking, which obscures substantial heterogeneity among subgroups. Recent advances in mixture modeling (Muthen & Muthen 2000) have allowed a small number of longitudinal studies to identify multiple age-related trajectories of smoking behavior (Chassin et al. 2000, Colder et al. 2001, White et al. 2002). These multiple trajectories have included an early-onset group (onset at ages 12–13) that shows a steep rise to heavy smoking; a late-onset group (onset after age 15) that smokes at more moderate levels; an experimenter group that tries smoking in adolescence but does not proceed to daily smoking and is developmentally limited

to adolescence; and a group that quits smoking. This approach of distinguishing among smoking trajectories is an important methodological advance because it has the potential to illuminate diverse etiological pathways underlying different trajectories of tobacco use.

Not only is it important to distinguish among multiple developmental trajectories into tobacco dependence but, within any single trajectory, it may be necessary to characterize smoking as a series of stages that have distinct determinants of movement across them (Mayhew et al. 2000). Even if distinct trajectories can be identified among adolescent tobacco users, it is clear that, at least in the United States, chronic tobacco use typically has pediatric origins. This raises the possibility that adolescence is a time when the individual has a heightened vulnerability to tobacco dependence or reward. Such a conjecture is supported by recent animal research showing that adolescent rats acquire nicotine self-administration behaviors much more readily than adults (Belluzzi et al. 2001, Levin et al. 2003). These findings suggest that processes involved in central nervous system development and maturation may play a critical role in the etiology of tobacco use and dependence.

INDIVIDUAL PERSONALITY AND PSYCHOPATHOLOGY   A large literature has linked adolescent tobacco use to intrapersonal characteristics such as temperament, personality, and psychopathology. Perhaps the most replicated finding is that characteristics that reflect behavioral "undercontrol," including sensation seeking and impulsivity (Masse & Tremblay 1997), rebelliousness (Burt et al. 2000), and conduct disorder (McMahon 1999), prospectively predict smoking onset. Adolescents who are "disinhibited" and "deviance prone" are more likely to engage in a variety of correlated "risky" behaviors including smoking and other substance use (Turbin et al. 2000). Less is known about the mechanisms that underlie the relations between these characteristics and tobacco use (McMahon 1999) although adolescents who are "undercontrolled" and impulsive are also more likely to affiliate with substance-using peers (Lynskey et al. 1998). They may also be less likely to consider the long-term negative consequences of smoking, and more likely to smoke as a way to attain adultlike status (Jessor & Jessor 1977).

Another understudied but important question is the relation of comorbidity to adolescent tobacco use (McMahon 1999). For example, the relation between attention deficit hyperactivity disorder (ADHD) and adolescent substance use is generally weakened or eliminated when co-occurring conduct disorder is considered (Flory & Lynam 2003). For cigarette smoking, the data are more conflicting. Although some studies find no correlations between ADHD and smoking in the absence of conduct disorder in clinical (Barkley et al. 1990, Burke et al. 2001) or community samples (Lynskey & Fergusson 1995), other studies have found unique relations between ADHD and smoking above and beyond conduct disorder (Burke et al. 2001, Disney et al. 1999). A link between ADHD and cigarette smoking raises the possibility that smoking can serve to self-medicate attentional deficits among those with ADHD (Flory & Lynam 2003). In support of this hypothesis, Molina & Pelham (2003) recently reported that it is the inattention symptoms of ADHD, as

opposed to impulsivity-hyperactivity symptoms, that are most highly associated with later cigarette smoking.

Compared to externalizing characteristics and disorders, the link between internalizing characteristics and disorders and adolescent smoking is less clear and consistent (McMahon 1999). Depressive disorders show unique relations to adolescent smoking, after controlling for other psychiatric disorders both cross-sectionally (Costello et al. 1999) and prospectively (Brown et al. 1996), and early depressive symptoms uniquely and prospectively predict smoking onset (Fleming et al. 2002). However, childhood anxiety disorders do not show those unique relations, and are associated with later smoking onset (Costello et al. 1999). Moreover, harm avoidance in kindergarten is associated with less likelihood of adolescent smoking (Masse & Tremblay 1997). Perhaps anxious children are more concerned with the negative consequences of smoking or rule breaking in general, or are less likely to spend time in peer networks that promote smoking. However, at later ages and stages of smoking, anxiety may motivate heavy smoking and raise risk for the development of nicotine dependence.

Attitudinal/cognitive risk factors   Adolescents form beliefs and attitudes about the effects of smoking before experimenting with it, and these attitudes and beliefs prospectively predict both the onset (Chassin et al. 1984, Conrad et al. 1992) and escalation (Andrews & Duncan 1998) of smoking. However, existing studies of smoking attitudes use explicit measures, which are limited by social desirability biases and people's lack of awareness of their underlying attitudes. Implicit methods for measuring attitudes have recently been developed (Greenwald et al. 1998), but are only now being applied to smoking research (Sherman et al. 2003).

Researchers have questioned whether adolescents are deterred from smoking because of their beliefs in its negative consequences, particularly when these negative consequences may not occur until years after smoking initiation. Not only do individuals generally discount the value of long-term outcomes in health decision-making (Ortendahl & Fries 2002), but adolescents have shown steeper temporal discounting rates than adults (Green et al. 1994). Moreover, many adolescent smokers do not believe there are health risks in "the first few years" (Slovic 2000), and may believe that they will stop smoking before damage is done (Arnett 2000).

There is also some debate about whether adolescents accurately perceive the risks of smoking (Romer & Jamieson 2001). This question is complex, and has produced varying answers depending on factors such as whether risk perceptions are assessed in terms of accuracy of absolute judgments or in terms of risks to people in general versus personalized risk to the self (Millstein & Halpern-Felsher 2002, Weinstein 1999).

Existing evidence suggests that both adolescents and adults show unrealistic optimism about the personalized risks of smoking (Arnett 2000, Weinstein 1999). Thus, it is unclear whether adolescents are any more likely than are adults to underestimate the personalized risks of smoking (Quadrel et al. 1993). The one

available longitudinal study found that beliefs in the personalized risks of smoking declined during the middle school years and began to increase in the high school years (above and beyond the effects of smoking behavior; Chassin et al. 2002). Moreover, value on health as an outcome declined during the high school years and did not begin to increase until early adulthood. These data suggest that adolescence is a period of increased cognitive vulnerability to smoking, based both on decreasing perceptions of the personalized risks of smoking and decreasing values on health as an outcome.

Even if adolescents hold strong beliefs in the negative outcomes of smoking, the influence of these beliefs on behavior may be outweighed by the perceived benefits of smoking (Millstein & Halpern-Felsher 2002). One perceived benefit is that it communicates a social image of precocity and adultlike status (Jessor & Jessor 1977). In general, the social image of an adolescent smoker is an ambivalent one, with negative aspects (e.g., unhealthy, foolish) but also images of toughness, sociability, and precocity that may be particularly valued by "deviance-prone" adolescents who are at risk to smoke (Barton et al. 1982). Similarity between self-image and these smoker images have prospectively predicted smoking onset (Aloise-Young et al. 1996).

In addition to expressing a social image of toughness, sociability, and precocity, some adolescents may be influenced by their beliefs that smoking can control body weight. Weight concern, dieting, and the belief that smoking can control body weight have been shown to predict prospectively smoking initiation among adolescent girls, but not boys (Austin & Gortmaker 2001) and these beliefs are held more often by white girls than by African Americans (Klesges et al. 1997). In fact, smoking does suppress body weight (Williamson et al. 1991), which makes this attitude particularly difficult to counter.

In summary, studies of cognitive models of adolescent smoking have supported the influence of beliefs and attitudes, and have been important for antismoking interventions and public policy. However, they have been limited by a reliance on explicit measures of attitudes, they have not yet considered the implications of attitudinal ambivalence, and they have not considered the full complexity of risk perceptions (including a lack of longitudinal studies of age differences). Finally, these cognitive models may be incomplete in that they do not account for the role of affect at the time of decision-making (Loewenstein et al. 2001).

SOCIAL AND CONTEXTUAL INFLUENCES   There has been longstanding research interest in social influences on adolescent smoking (particularly parent and peer influences), which has recently been combined with a focus on macrolevel contextual variables (e.g., neighborhood effects, effects of taxation, advertising, and youth access), which are of interest because of their public policy implications.

Peer smoking is the most consistently identified predictor of adolescent smoking (Conrad et al. 1992, Derzon & Lipsey 1999). The magnitude of self-reported cross-sectional correlations between peer smoking and adolescent smoking is somewhat inflated because it reflects both the actual effects of peer influence as well as the

effects of peer selection (i.e., adolescents who smoke seeking out similar others). It also reflects adolescents' biased perceptions that their own behavior is similar to that of their friends. However, significant findings still emerge when peer smoking is tested as a prospective predictor in longitudinal designs (Chassin et al. 2000) and when friends report directly on their own smoking behavior (Urberg et al. 1997). In addition to peer cigarette smoking, affiliation with peers who engage in high levels of other problem behaviors also prospectively predicts smoking initiation (Simons-Morton 2002), as does self-identification with a high-risk social group (Sussman et al. 1994).

Formal social network analysis suggests modest peer influence effects. For example, Ennett & Bauman (1994) identified peer cliques based on patterns of friendship nominations. They found that both selection and peer influence operated to make peer cliques homogenous with regard to smoking, but also that many adolescent smokers were not members of the identified cliques. Moderator variables also suggest that the magnitude of peer influence effects on adolescent smoking varies as a function of gender (stronger effects for females; Hu et al. 1995), ethnicity (strongest effects for white, weakest effects for African Americans; Landrine et al. 1994, Urberg et al. 1997), and parent involvement (weaker effects for those with involved parents; Simons-Morton 2002).

The mechanisms underlying peer influence effects have not been clearly established. Few adolescents report experiences of direct peer "pressure" to smoke (Urberg et al. 1990). Thus, peer influence effects may operate in other ways, such as increasing perceptions that smoking is prevalent and normative (Conrad et al. 1992), communicating a positive social image of smoking, providing access and opportunities for smoking behavior, or providing a means of peer bonding.

In contrast to peer smoking, parent smoking has been less consistently related to adolescent smoking initiation (Conrad et al. 1992) and its effects have been weaker in overall magnitude (Derzon & Lipsey 1999). However, despite its overall low magnitude of effect, parent smoking is a risk factor for adolescent smoking (Derzon & Lipsey 1999). Methodological features of prior studies may have obscured the magnitude of parent smoking effects by failing to (*a*) directly measure parent smoking, (*b*) distinguish between biological and custodial parents, and (*c*) distinguish among different adolescent smoking outcomes. For example, Chassin et al. (2000) found that parent smoking was related to a particular trajectory of smoking characterized by early onset, rapid escalation to heavy levels, and persistence over time. Consistent with these findings, a recent review of twin studies concluded that heritability is stronger for tobacco dependence than for smoking initiation (Sullivan & Kendler 1999). Thus, parent smoking may be an especially powerful risk factor for serious adolescent smoking outcomes. The association between parental and adolescent smoking can reflect multiple pathways of influence, including the effects of heritable individual differences in tobacco effects, heritable personality characteristics, and modeling.

The school environment also provides an important context for smoking acquisition. Adolescents who have conduct problems in school and who have lower levels

of academic achievement are more likely to smoke, and low academic achievement and school conduct problems are reciprocally related (Bryant et al. 2000). School climate variables have also been linked to adolescent smoking, such that schools with more permissive norms about smoking, less teacher involvement, and less consistent discipline have higher rates of smoking (e.g., Johnson & Hoffman 2000, Novak & Clayton 2001). School effects may also reflect the broader influence of the neighborhoods in which schools are nested. However, findings have not been convincing in the small number of studies to examine the relation between neighborhood context and adolescent smoking.

Chaloupka (1999) reviewed studies of larger macrolevel social influences and noted several important limitations. Antitobacco social policies such as increased taxation and restrictions on youth access tend to be implemented in communities that also share antismoking norms and values, so that the effects of the policies are hard to disentangle from the effects of these broader community norms. Notwithstanding these limitations, econometric data generally show that adolescent tobacco use is price sensitive (as it is for adults). However, the magnitude of the effect may vary with age, ethnicity, and stage of smoking. For example, infrequent experimentation may be less price sensitive (Emery et al. 2001). Less clear effects have been produced by restrictions on youth access (e.g., merchant interventions, bans on vending machines), and enforcement of youth access policies has been difficult to attain. Finally, adolescent smoking is correlated with self-reported exposure to cigarette advertising (Romer & Jamieson 2002), and epidemiological trends over time show correlations between rises in adolescent smoking and times of cigarette industry advertising campaigns (Pierce & Gilpin 1995). In general, evidence suggests that tobacco advertising can increase smoking onset, and counteradvertising can delay or prevent smoking.

THE MOTIVATIONAL BASIS OF CIGARETTE DEPENDENCE

Transition to Dependent Smoking

Relatively little is known about the transition from youthful experimentation with tobacco to regular, heavy use. It is known that some youthful cigarette smokers do indeed satisfy criteria for tobacco dependence but it also the case that many adolescents do not (Colby et al. 2000a,b). Some evidence suggests that as smokers become more dependent, there is a shift in the motivational basis for their tobacco use. Beginning smokers tend to rate social motives and contextual factors as influential to their smoking; heavy smokers stress the importance of control over negative moods and urges, and the fact that smoking has become "automatic" (Piper et al. 2003). Indeed, one study showed that the best predictor of continued or increased smoking among beginning smokers was reporting that smoking provided good control over negative affect (Wetter et al. 2003). As smoking becomes less linked to external cues, and more linked to internal stimuli such as affect, more

and more smokers warrant classification as nicotine dependent (Piper et al. 2003); i.e., they smoke more than they intend, smoking interferes with their lives or harms them, and so on. In the following section we review evidence on processes that may account for dependent cigarette smoking. We address whether nicotine per se is necessary for dependent smoking, whether route of delivery (i.e., smoking) is an important influence on motivation to use nicotine, and which types of nicotine effects are critical, e.g., associative effects and particular actions.

Role of Nicotine

Cigarette smoke contains thousands of constituents, leaving open the possibility that nonnicotine factors significantly influence nicotine reinforcement. Research now clearly indicates that nicotine is essential for prolonged, addictive tobacco use. Nicotine by itself yields the hallmark effects of addictive drugs. It produces tolerance and physical dependence, and acute doses produce elation and pleasure (Corrigall 1999, USDHHS 1988). In addition, pretreatment with nicotine reduces subsequent tobacco self-administration among smokers, even if the pretreatment nicotine is delivered via routes other than smoking (Perkins et al. 1996). This suggests that downward compensation in nicotine self-administration occurs because of the central effects of nicotine, not because of cues produced by the smoking ritual per se. Also, smokers will not self-administer tobacco on a chronic basis if it does not contain nicotine (Caggiula et al. 2001). Although nicotine is essential for the development and maintenance of smoking, once nicotine dependence is established, cues associated with nicotine delivery become highly influential in controlling self-administration behaviors.

PHARMACOKINETICS AND NICOTINE DELIVERY   Although cigarette smoking is highly addictive, other nicotine delivery systems are much less likely to support addictive use. For example, nicotine replacement therapies (NRTs, such as nicotine gum) only rarely sustain self-administration over the long-term (Hughes 1989). This is due, at least in part, to differences in the pharmacokinetics of cigarette smoking versus other nicotine delivery systems. Pharmacokinetics refers to how drug levels vary in body compartments or regions as a function of time.

Nicotine pharmacokinetics depends greatly upon route of administration. When a cigarette is smoked, about 80% of the inhaled nicotine is absorbed in the lungs (Armitage et al. 1975). Absorption is both efficient and extremely rapid because of the large volume of the alveolar surface area and the ready access to the extensive pulmonary capillary beds. In addition, the dissolution of nicotine in the pulmonary beds yields a greater proportion of nonionized nicotine that further promotes its rapid absorption. After absorption in the lungs, nicotine is transported to the brain via arterial blood prior to its passing through the liver or being distributed more widely in venous circulation. Thus, after smoking, nicotine levels may be some 6–10 times higher in arterial versus venous blood (Henningfield et al. 1993). A concentrated bolus of nicotine (mass of nicotine in the blood) reaches the brain

some 12–15 seconds after inhalation (Benowitz 1994), where its absorption is rapid because of the high affinity of brain tissue for nicotine (Maziere et al. 1976). Although the absorption of nicotine is rapid, so is its elimination. The terminal elimination half-life of nicotine in the body is about 2 hours, but its distributional half-life in the brain is about 10 minutes. The latter describes the time that it takes a nicotine dose to fall 50% from its peak level in the brain as the nicotine is distributed to other body compartments (Russell 1988). This pattern of rapid rises in nicotine levels in the brain, with rapid distributional tolerance, occurs against a backdrop of trough levels of nicotine that will persist or rise as long as the intercigarette interval is not much greater than 60 minutes (Russell 1988).

Nicotine taken via other routes does not produce this same dramatic sawtooth pattern of effects with respect to brain levels. When taken via buccal absorption or via other routes (even venous infusion), the nicotine bolus is less concentrated, the arrival in the brain is delayed, and there is less of a sawtooth pattern to the nicotine profile across time (Pomerleau & Pomerleau 1992, cf. Frenk & Dar 2000). These differences occur because other routes tend to yield greater initial distribution across body compartments.

Although there is general recognition that rapid onset of drug actions promotes addictive drug use (van Ree et al. 1999), it remains unclear why this is so. Researchers do not really understand which characteristics of drug pharmacodynamics are most determinant of addictiveness. Possible candidates include the concentration of drug in the bolus, the trajectory of the rise time, or the combination of a rapid onset with a rapid offset. The last property might promote high levels of withdrawal plus optimal withdrawal relief. Finally, these pharmacokinetic features might be motivationally significant merely because they reflect a high level of control over drug effects in the brain.

ASSOCIATIVE PROCESSES   Behaviors that once delivered nicotine will persist despite severe degradations in the contingency between nicotine and self-administration behaviors. Thus, repeated nicotine dosing can generate sources of reinforcement that complement, or perhaps supplant, the impact of nicotine itself. That is, self-administration behaviors become somewhat uncoupled from a contingency with nicotine.

Nicotine pretreatment can suppress self-administration, but there is only a weak relation between pretreatment dosage and the dose subsequently self-administered. Among both humans and animals with extensive histories of self-administration, very large pretreatment dosages of nicotine result in rather modest decreases in nicotine self-administration (Benowitz et al. 1998). Such imprecise downward compensation suggests that smoking behavior is reinforced by more than just attaining a particular level of nicotine in the blood.

Other evidence of uncoupling is that although denicotinized cigarettes do not sustain smoking over the long term, over brief periods they do support self-administration, produce pleasure, and reduce craving and withdrawal symptoms (Butschky et al. 1995). Also, although animals will eventually extinguish

instrumental responding once it no longer yields nicotine, they are highly resistant to such extinction. Caggiula et al. (2001) report only partial extinction after 17 days of extinction trials. Moreover, cues that were previously paired with nicotine are effective at reinstating self-administration behaviors; more effective, in fact, than nicotine itself (Caggiula et al. 2001).

The causes of uncoupling of nicotine receipt and instrumental behavior are unknown. However, Caggiula et al. (2001) suggest that nicotine may be particularly potent at conferring conditioned reinforcement properties on associated cues. Therefore, organisms may engage in self-administration behaviors because the self-administration behaviors themselves have become reinforcing. Other environmental or social cues might also become rewarding through being paired with nicotine. Of course, the research reviewed above does not reveal the nature of the associative effects that spur self-administration. For instance, nicotine-paired cues could elicit pleasurable/euphoric effects that serve as incentives for further self-administration. Or instead, nicotine could elicit aversive withdrawal effects that set the stage for negative reinforcement.

The evidence reviewed above yields two conclusions: (*a*) Nicotine is essential to the development and long-term maintenance of tobacco self-administration, but (*b*) after extensive self-administration experience, cues associated with nicotine receipt can, by themselves, powerfully affect self-administration behaviors. Thus, although the dependent smoker may smoke to quell or achieve internal states (e.g., to reduce negative moods), external cues powerfully influence self-administration behaviors.

It is clear that pharmacodynamics and associative effects are critically important to the motivation to use nicotine. We now turn to the question of which particular nicotine actions are motivationally significant, and how best to conceptualize nicotine motivational processes.

Motivational Processes

TOLERANCE   As was previously noted, the DSM criteria for diagnosing dependence include the existence of tolerance and physical dependence (the occurrence of a withdrawal syndrome upon discontinuation/reduction of drug use). In the case of tolerance, researchers have reported tolerance to nicotine's tachycardic, euphoriant, and akinetic actions (e.g., Perkins et al. 1994, 2001a). Research also indicates that tolerance is heterogeneous with respect to time course. For example, some tolerance phenomena persist after years of abstinence (Perkins et al. 2001a). Other types are short-lived, such that abstinence of a few hours may restore some cigarette effects (Benowitz 1998). Therefore, even at very high smoking rates, each cigarette may continue to produce pharmacologic effects (e.g., Porchet et al. 1988).

There is some evidence that tolerance plays an important early role in permitting the development of dependent smoking. That is, the youthful smoker must become inured to the toxic and irritating effects of smoking (e.g., nausea) in order for him

or her to escalate use to levels that will produce physical dependence (Leventhal & Cleary 1980). However, there is scant evidence that tolerance is an important index of the degree or severity of tobacco dependence among heavy smokers. For instance, Perkins et al. (2002) found little evidence linking tolerance magnitude to withdrawal severity or relapse likelihood among dependent smokers. This suggests that tolerance may be a "low hurdle" in the development of dependence; all dependent smokers are somewhat tolerant to nicotine effects, but tolerance and dependence may not be produced by the same processes. Consequently, tolerance is an insensitive index of dependence.

PHYSICAL DEPENDENCE   Research shows clearly that chronic nicotine exposure results in physical dependence to nicotine; i.e., the tendency to display well-characterized signs of the nicotine withdrawal syndrome. For instance, Malin (2001) has shown that rats exposed to continuous infusion for 7 days of 3- or 9-mg/kg/day nicotine hydrogen tartate (1.05 or 3.15 mg/kg/day expressed as the base), displayed the following abstinence signs: teeth chattering, chewing, gasping, writhing, head shakes, body shakes, tremors, ptosis, and assorted other signs such as spontaneous ejaculation and licking. These signs peaked at about 18–22 hours postcessation. The evidence that these reflect nicotine withdrawal per se is compelling. These signs are temporally tied to drug abstinence; they are linearly related to dose, and they can be precipitated by a competitive nicotine antagonist such as dihydro-∃-erythroidine (e.g., Epping-Jordan et al. 1998, Malin et al. 1998).

One feature of the nicotine withdrawal syndrome may have unique motivational significance. Nicotine withdrawal results in an increase in the threshold required for rewarding brain stimulation (Epping-Jordan et al. 1998). This is seen in the withdrawal syndromes of other drugs and this may account for the anhedonia that tends to accompany withdrawal (Baker et al. 2003). Thus, withdrawal may be aversive not only because negative affect is a principal manifestation, but also because withdrawal decreases the rewarding properties of nonpharmacologic incentives (e.g., social stimuli).

The human nicotine withdrawal syndrome does not comprise the dramatic physical signs that are observed in nicotine research with rats. This may reflect the relatively higher doses given animals (Malin 2001) as well as species differences. However, the nicotine withdrawal syndrome in humans does manifest with a reliable core set of symptoms and signs including dysphoria, anxiety, inability to concentrate, increased appetite, weight gain, sleep disruption, and others (Hughes et al. 1991). Moreover, recent work suggests that the nicotine withdrawal syndrome may persist for months following nicotine abstinence (Piasecki et al. 1998, 2000).

Both animal and human research suggests that the withdrawal syndrome is aversive. For instance, Suzuki et al. (1996) determined that rats would avoid a chamber associated with the nicotine abstinence syndrome precipitated by mecamylamine, a nicotine antagonist. Not only is the syndrome aversive, but nicotine quickly and efficiently alleviates its aversive components. For instance, nicotine administered

to withdrawn smokers consistently reduces their self-reports of sadness, anxiety, and anger/irritability (Jorenby et al. 1996, Zinser et al. 1992), and the affective components of withdrawal appear to be most influential in motivating further nicotine self-administration (e.g., Piasecki et al. 2000). Relief of withdrawal-induced negative affect by nicotine has also been reported in animals (Cheeta et al. 2001). These observations, plus the fact that smokers often smoke more, or relapse, in the context of negative affect (Baker et al. 2003), suggest that negative reinforcement through the relief of withdrawal symptoms may play a critical role in sustaining smoking among dependent smokers.

APPETITIVE/EUPHORIANT EFFECTS AND POSITIVE REINFORCEMENT   Like other psychomotor stimulants, nicotine produces subjective sensations that are characterized as a "rush," "elation," or "buzz." These effects are not dependent upon smoking as a delivery system, or upon associative elicitation, since they can follow venous infusion. However, consistent with the material on pharmacokinetics, such subjective effects are more likely to occur with delivery systems that promote rapid rise times of nicotine in the brain.

In research conducted by Garrett & Griffiths (2001), caffeine, cocaine, and nicotine were infused into volunteers who had a history of use of these drugs. Nicotine elicited dose-dependent increases in ratings of "drug effect," "good effect," "like drug," and "high." Moreover, the highest dose of nicotine (3.0 mg/70 kg) was never identified as a placebo, and instead was identified as a stimulant by all subjects. Seven of the nine subjects identified it as cocaine.

Although it is highly likely that nicotine's direct appetitive effects are important to nicotine motivation and addiction, the extent and nature of their influence is unclear. Evidence supporting a role for nicotine's appetitive effects includes the fact that smokers reliably report positive reinforcement as a reason for smoking (e.g., for "pleasurable relaxation," and "stimulation"; Ikard et al. 1969), and they hold expectancies that smoking will enhance positive affect (Copeland et al. 1995). Additionally, a significant proportion of smoking relapse episodes occur when individuals are in positive affect states (Brandon et al. 1990, Shiffman et al. 1996) and positive affect predicts urges to smoke during the course of ongoing smoking (Zinser et al. 1992). Finally, in the laboratory, positive affect imagery elicits stronger urges to smoke than does neutral imagery (Tiffany & Drobes 1990).

Numerous observations, however, suggest that positive reinforcement is not the major motivational influence on dependent smoking. For example, although positive affect imagery does elicit smoking urges, it is less effective than negative affect imagery (Tiffany & Drobes 1990). Also, expectations of negative reinforcement from smoking (e.g., relief of negative affect) predict relapse likelihood, but positive reinforcement expectancies do not. Thus, the latter may have less motivational significance (Copeland et al. 1995, Wetter et al. 1994). Further, relapse is about half as likely to occur during positive affect states than during negative affect states (Brandon et al. 1990, Shiffman et al. 1996). Finally, humans rapidly

acquire acute tolerance to nicotine's appetitive actions, so that in the inveterate smoker relatively few cigarettes produce such effects (e.g., Perkins et al. 1994).

SENSITIZATION OF INCENTIVE EFFECTS   According to the Incentive Sensitization Theory (IST; Robinson & Berridge 1993), addictive drugs exert dopaminergic actions in critical mesotelencephalic brain sites. These dopamine actions are thought to imbue drug and drug cues with potent incentive value such that drug use is seductively attractive, even if the organism actually experiences little drug reward. Earlier theories (Wise 1988) held that dopamine determined how rewarding a drug would be. However, based upon a careful review of relevant research, Robinson & Berridge (1993, 2001) concluded that dopamine does not primarily influence drug reward, but instead, it influences expectation or anticipation of reward.

IST explicitly separates the subjective evaluation of drug effects ("liking") from the incentive-motivational effects ("wanting"). According to IST, these two processes are associated with different neuronal systems, and therefore, they are affected differently by drug exposure. Repeated drug use increases the incentive properties of drug cues because of sensitization of mesotelencephalic dopamine systems, but it also simultaneously produces tolerance to drug reward. This could explain why smokers smoke compulsively but often report that they derive scant pleasure from it.

Most support for IST has come from research on addictive drugs other than nicotine (Robinson & Berridge 2001). However, research has suggested striking similarities in motivational influences across different types of addictive drugs (Robinson & Berridge 2001). Therefore, to the extent that IST accounts for other examples of drug dependence, incentive sensitization may also play a role in nicotine dependence. In addition, the tenets of IST are generally consistent with findings from tobacco research (e.g., Carter & Tiffany 1999). For instance, there is clear evidence that nicotine sensitizes the core of the nucleus accumbens so that repeated administration of the same dose produces increased dopamine release (Balfour 2003). In addition, a measure of positive affect and approach motivation (cerebral asymmetry) showed increases when smokers were exposed to smoking cues, but not after they were actually allowed to smoke (Zinser et al. 1999). This suggests that there is more "reward" in the anticipation of smoking than in smoking per se. Finally, there is mounting evidence that nicotine is a potent modulator of the incentive value of cues of reward. Thus, nicotine has the capacity to enhance the incentive value or salience of cues for nonpharmacologic rewards such as a visual reinforcer (Donny et al. 2003).

IST is consistent with many features of dependent smoking. However, more research is needed to establish its relevance. IST is predicated on the notion that addicts derive little pleasure from drug use. This assumption of the model has been little studied in smokers. Little is known regarding the proportion of cigarettes that yield pleasure, the immediacy of the pleasure, and its magnitude—or how cigarettes compare on these dimensions with other reinforcers available to the smoker. In addition, to the extent that nicotine modulates incentive value, researchers should

explore how smokers are affected by the increased incentive value of nonpharma-cologic appetitive stimuli once they quit smoking. Does smoking make the whole world "brighter" and more appealing and does quitting make the world more dull and uninteresting? Finally, how do withdrawal and other aversive states affect the incentive value of smoking cues? Is the appeal or salience of such cues largely a function of nicotine deprivation (Baker et al. 2003)? And, if deprivation enhances both incentive salience and nicotine reward, a negative reinforcement model might provide a better explanation for dependence motivation than does IST per se.

NEGATIVE REINFORCEMENT THROUGH REDUCTION OF STRESS AND WITHDRAWAL
Negative affect exerts potent motivational effects on smoking. For instance, smok-ers regularly cite affective control as a principal motive for their tobacco use (e.g., Copeland et al. 1995, Ikard et al. 1969). Numerous studies have found that stress manipulations increase cravings for cigarettes (Payne et al. 1991, Perkins & Grobe 1992) as well as amount and intensity of smoking (e.g., Dobbs et al. 1981, Payne et al. 1991). In addition, expectancies about negative affect reduction are associ-ated with magnitude of nicotine dependence, severity of withdrawal symptoms, and treatment outcome (Copeland et al. 1995, Wetter et al. 1994).

Additional support for the link between smoking and affect is found in population-based studies examining the covariation of smoking with clinical syn-dromes characterized by high negative affectivity, particularly clinical depression. National epidemiological studies in the United States and Australia have found a high degree of comorbidity between depression and smoking (Anda et al. 1990, Degenhardt & Hall 2001). Individuals with a history of major depression are ap-proximately twice as likely as others to be smokers, and smokers are more likely than others to have affective disorders. Such correlational findings do not address the direction of causality, and it is likely that causality between smoking and de-pression is bidirectional, and may reflect a common genetic risk (Kendler et al. 1993).

Finally, postcessation relapse to smoking tends to be precipitated by situations characterized by stress and negative affect (e.g., Brandon et al. 1990). Shiffman et al. (1996) sampled affect and smoking in real-time using palm-top computers. Initial relapse episodes were compared with nonrelapse "temptation" episodes, and with random sampling during nonsmoking. Negative affect differed across the three assessment situations, with the greatest negative affect associated with the relapse episodes, followed by the temptation episodes. Moreover, level of postcessation negative affect is an excellent marker of relapse vulnerability (Kenford et al. 2002; Piasecki et al. 2003a,b).

These strong links between negative affect and measures of smoking motivation and dependence suggest that negative reinforcement plays a critical role in smoking motivation; i.e., smokers smoke to reduce distress (Baker et al. 2003). What is not known is what accounts for this relation. As noted previously, there is compelling evidence that nicotine ameliorates elements of the tobacco withdrawal syndrome including negative affect (Baker et al. 2003, Cheeta et al. 2001). This certainly

could account for a relation between negative affect and the motivation to smoke. Moreover, this account is consistent with the fact that negative affect is highly associated with measures of smoking motivation only among abstaining smokers (Sayette et al. 2003, Shiffman et al. 2002, Zinser et al. 1992).

What is not clear is whether nicotine also alleviates the negative affect that arises from stressors. If nicotine reduces negative affect arising from environmental stressors, this would certainly increase the opportunity of smokers to attain negative reinforcement from smoking.

STRESS REDUCTION   A recent authoritative review (Kassel et al. 2003) reveals conflicting evidence as to whether nicotine reduces stress-induced negative affect (SINA) in smokers with little nicotine deprivation (<1.5 h). In theory, smoking-induced reductions in negative affect in these smokers should not be due to withdrawal relief (given the brief deprivation period). Several laboratory studies have reported reductions in SINA (typically anxiety; e.g., Jarvik et al. 1989, Juliano & Brandon 2002, Perkins & Grobe et al. 1992). However, caution is warranted. Even over a brief 1.5-h abstinence period, withdrawal relief may have colored these findings, given the short distributional half-life of nicotine. In addition, nicotine's ability to reduce SINA may depend upon contextual factors. Kassel & Unrod (2000) reported that smoking reduces anxiety in the laboratory setting only when administered concomitantly with a distraction. Nicotine may constrain cognitive workspace such that information about stressors is not processed in the context of distraction. Thus, as has similarly been reported with alcohol (e.g., Steele & Josephs 1988, Curtin et al. 1998) stress reduction may occur via a cognitive mechanism. Finally, there is evidence that smoking is more likely to reduce anxiety before or after a stressful event, rather than affecting reactions during the event (Gilbert 1995, Gilbert et al. 1989, Kassel et al. 2003).

Field research suggests that nicotine can reduce negative affects. For instance, using real-time data collection methods, Jamner and his colleagues (Delfino et al. 2001) found that smoking was followed by decreased anger in both men and women, and decreased sadness in men. However, such reductions in negative affect cannot be attributed to stress reduction per se, as they might instead reflect withdrawal relief.

In contrast to studies suggesting smoking-induced reductions in SINA, others report negative evidence. In one study, smoking did not reduce anticipatory anxiety prior to white noise or a vigilance task stressor (Jarvik et al. 1989). Smoking did reduce anxiety in anticipation of cold pressor and anagram tasks, but posttask anxiety ratings were not, in fact, lower among individuals who smoked. Herbert et al. (2001) reported no reduction in anticipatory anxiety with or without a distraction task. Recently, Britt et al. (2001) showed that when smokers were exposed to a social stressor, smoking reduced the level of withdrawal symptoms, but not anxiety per se. In general, it appears that there are about as many human studies showing little or no relief of SINA as there are studies showing such relief (Kassel et al. 2003).

The studies reviewed above all face significant interpretive challenges. For instance, as noted earlier, it is difficult to disambiguate the stress-reduction versus the withdrawal-reduction effects of nicotine. One strategy used to avoid the prospect of withdrawal coloring results is to examine the effects of nicotine in non-addicted subjects. However, nicotine can produce aversive effects that may mask stress-reducing actions in nicotine-naïve subjects (Foulds et al. 1997). In addition, smokers and nonsmokers tend to differ on the basis of constitutional factors (e.g., personality and psychiatric variables; Breslau 1995), and these differences might influence the degree of stress-reduction obtained from nicotine.

The use of animal models reduces the threat of some of these interpretive challenges. Animal research has shown that nicotine can produce anxiolytic (anxiety reducing) as well as anxiogenic (anxiety causing) effects, with the nature of the effect being determined by dose, the type of anxiety test, the animal's prior experience with nicotine, and the delay between the dose and test (Irvine et al. 2001). Nevertheless, there exists both behavioral (e.g., social interaction under stress; File et al. 1998) and neuropharmacological (e.g., mesoprefrontal dopaminergic response to acute inescapable footshock stress; George et al. 2000) evidence for nicotine-induced anxiolysis. However, animals quickly develop tolerance to the ameliorating effects of nicotine on SINA, but not to withdrawal-induced anxiolysis (Irvine et al. 2001, Szyndler et al. 2001). Thus, the animal data suggest that for chronic users the most available and reliable source of negative affect reduction is withdrawal relief. This conclusion is consistent with much of the human research and with recent theorizing (Baker et al. 2003, Parrott 1999).

EXPECTATIONS OF STRESS REDUCTION   Regardless of whether smoking actually reduces SINA, smokers expect it to do so (Brandon et al. 1999, Copeland et al. 1995, Wetter et al. 1994), and this may influence smoking motivation. For example, a recent study using the "balanced placebo" design (Juliano & Brandon 2002) showed that smokers' experience of smoking-induced anxiety relief depended upon their expectations of such an effect.

It is unknown why smokers have strong expectations that smoking will alleviate SINA. Smoking may indeed alleviate SINA reliably, and research has simply failed to index this sensitively. On other hand, it may be that nicotine's ability to ameliorate withdrawal distress may overshadow, motivationally, its evanescent or inconsistent impact on stress reactivity. Smokers experience some level of withdrawal throughout much of the day, even if smoking ad libitum (see Baker et al. 2003). It is possible that this withdrawal-induced negative affect adds to SINA, and that it is difficult for smokers to distinguish between withdrawal relief and stress relief.

In sum, the bulk of the evidence strongly supports negative reinforcement motives in the maintenance of smoking behavior. Not only is nicotine highly effective in alleviating withdrawal-induced negative affect, but a wide range of research paradigms suggests that expectations of stress relief contribute to the maintenance

of adult smoking. What is unclear is the extent to which nicotine is actually effective in reducing affective reactions to external stressors. It may be that smokers assume that smoking ameliorates stress-induced negative affect because it is so effective at reducing withdrawal-induced negative affect (Baker et al. 2003, Parrott 1999).

Acceptance of any conclusions about nicotine's anxiolytic- or stress-reducing effects must be tempered by appreciation of the challenges to research synthesis in this area. Studies differ in terms of stressors, nicotine dose, withdrawal-stress latencies, and measures of affect or stress response. In addition, research in this area is subject to the problems that plague any research on affective phenomena, such as desynchrony across response systems (e.g., Lang 1968) and the fact that affective responses may be unavailable to conscious awareness.

ADDITIONAL MOTIVATIONAL INFLUENCES

Weight regulation   Nicotine delivery via cigarette smoking can certainly produce other effects that influence smoking motivation. For instance, nicotine can reduce body weight, apparently by adjusting body weight set point (Cabanac & Frankham 2002, Schwid et al. 1992). Thus, on average, smokers weigh less than nonsmokers (Williamson et al. 1991), and once people stop smoking they tend to gain weight (Perkins 1993). It is certainly possible that some smokers, particularly women, smoke for weight control motives. Although this is a plausible hypothesis, it is unclear how common this motive is, and the extent to which it is a determinant of smoking maintenance or relapse back to smoking.

Information processing   Smokers report that smoking enhances their cognitive processing (Warburton & Walters 1995), and that abstinence from smoking produces an inability to concentrate (Hughes et al. 1991). In fact, laboratory research supports these assertions. Domino & Kishimoto (2002) showed that nicotine withdrawal disrupted the processing of meaningful stimuli as reflected by P3 event-related potentials, and this effect was reversed by smoking. When withdrawn smokers smoked, it did not produce any frank or net improvements in attentional processing; it only neutralized the deleterious impact of withdrawal. It is clear that smoking restores the information-processing deficits produced by withdrawal. However, the evidence is mixed as to whether and how it enhances information processing in nonwithdrawn individuals.

Despite difficulties in interpreting research on nicotine's impact on cognitive processes (Heishman et al. 1994, Park et al. 2000), some conclusions may be drawn about its effects in nonwithdrawn individuals. First, there is a fairly consistent body of evidence that nicotine enhances alerting (Domino & Kashimoto 2002, Mancuso et al. 2001), i.e., maintaining a state of vigilance such that one is sensitive to stimulus changes (Fan et al. 2002). However, research in nonwithdrawn humans suggests that nicotine does not consistently enhance higher-level attentional processes such as orienting (selection of information from sensory input) or executive control (resolution of conflict regarding response options; e.g.,

Atchley et al. 2002, Griesar et al. 2002, Heishman & Henningfield 2000, Park et al. 2000). [However, nicotine may enhance orienting in schizophrenics (Kumari et al. 2001), and, consequently, contribute to elevated smoking rates in this population.] Moreover, research with humans provides little evidence that nicotine enhances other cognitive faculties such as memory (Heishman et al. 1994). Although some animal research suggests that nicotine may enhance selective attention (Hahn & Stolerman 2002, Hahn et al. 2002), this effect may not occur in humans due to species differences (Park et al. 2000).

In sum, nicotine may directly enhance alertness, and it may produce broader cognitive effects via the alleviation of withdrawal. It is possible that these effects motivate the uptake of smoking or its maintenance. However, if it were discovered that smokers do indeed continue smoking in order to escape or avoid withdrawal-induced cognitive deficits, this would constitute just one particular mechanism via which negative reinforcement affects the maintenance of smoking. At present, there is insufficient information to determine the extent to which smokers maintain smoking or relapse in pursuit of cognitive enhancement.

Although the motivational impact of cognitive enhancement is unknown, cognitive measures are elucidating some of the information-processing substrata of smoking motivation and dependence. Cognitive assays such as the Stroop paradigm and the dot-probe task are revealing that smokers' attention to smoking stimuli is enhanced by withdrawal (Waters & Feyerabend 2000), and enhanced by information that the opportunity to smoke is imminent (Wertz & Sayette 2001). Such cognitive measures may not only offer new methods for assessing the strength of nicotine dependence, but may also suggest stages in motivational processing where tobacco approach behavior may be interrupted effectively.

Motivation: Further Complexities

The above review did not do justice to the complexity of the motivational influences on tobacco smoking. For example, any single smoking episode may be a function of multiple motives that change as function of context and other factors such as cost and the availability of other reinforcers (Carroll & Campbell 2000, Gilbert 1995, Vuchinich & Tucker 1998). Moreover, many smoking episodes may be difficult to attribute to particular motivational influences because smoking may be reflexively primed by subtle or indistinct instigators (Tiffany 1990). In addition, individual differences such as gender may moderate the influence of smoking motivational processes (Gilbert 1995, Perkins et al. 2001b).

CONCLUSIONS

The motivation to smoke cigarettes is multifaceted. No single, monolithic motive accounts for cigarette smoking across individuals and development. The complexity of smoking motivation is reflected by the range of factors associated with the initiation and maintenance of smoking. These factors include stress, genotype,

peer and parental relations, personality/temperament, expectancies, and presence of affective symptomatology (Gilbert 1995).

Not only do multiple factors affect cigarette smoking, but their relative impacts appear to vary across the development of addictive smoking. For instance, although peer influences and impression management motives strongly influence the behavior of the neophyte smoker, control of withdrawal symptoms appears to be relatively more influential for heavy-smoking adults.

Our literature review showed that youthful initiation and use is related to a broad array of factors. Such smoking appears to be influenced by family environment, ethnicity, personality, economics, comorbidity, attitudes and beliefs, social networks, and genetics. Although research has now documented the broad range of factors that may contribute to youthful uptake, what is missing is a clear understanding of the relative impacts of these factors, how they "work" together, and which factors are most influential in the progression to tobacco dependence. Moreover, as noted previously, there is evidence that adolescence represents a sensitive period for the development of tobacco use and dependence. It is not known, at present, which factors, in particular, mediate this heightened vulnerability. Research that examines distinct trajectories of smoking progression may elucidate critical risk factors and determinants of dependence. Such research may also be useful for identifying genotypes associated with a vulnerability to dependence.

Despite the heterogeneity in smoking motives, some factors stand out as being influential across the ontogeny of smoking. Multiple sources of evidence point to strong links between negative affect and smoking. For example, preexisting depressive symptoms may set the stage for smoking initiation, and smoking may then further exacerbate depression. Moreover, negative affect appears to be a principal instigator of relapse among smokers trying to abstain.

Although a great deal has been learned about cigarette smoking, many unanswered questions remain. We still do not know enough about nicotine's effects on information processing. For instance, we don't understand why studies yield discrepant results regarding nicotine's attentional effects, and we know little about the motivational significance of nicotine's perceived effects on alertness or attention. Also, we don't yet understand when and how nicotine influences affective and other reactions to stressors. In addition, while we appreciate the strong links between negative affect and smoking motivation, we do not understand the motivational basis of this relation. Does negative affect set the stage for negative reinforcement, or does it merely inflate the incentive value of smoking cues? Similarly, we do not know why negative affective symptoms or certain personality traits create vulnerability to addictive smoking. Is it because they encourage smoking initiation along with a host of other risky behaviors, or do they affect the reinforcing value of nicotine? Along those lines, it remains unclear why the development of addictive smoking is so tightly linked to adolescence. Does central nervous system maturation constitute an optimal neuropharmacologic environment for the acquisition of strong smoking motives?

Certainly, research will ultimately provide answers to these questions, and these questions might be most profitably approached via research that is highly integrative and transdisciplinary. The influences on smoking are diverse, and these influences involve multiple cognitive, biologic, behavioral, and social systems. Relations among these influences may be reciprocal, and each may play a different role in the development of smoking. Such complexity argues for transdisciplinary research efforts that foster a comprehensive and integrated perspective on the development and maintenance of cigarette smoking.

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