Chapter 4
Social, Environmental, Cognitive, and Genetic Influences on the Use of Tobacco Among Youth

Introduction

This chapter addresses the important question of why young people begin to use tobacco. The immediate and long-term health consequences of use have been extensively documented over the past 50 years. Why anyone would begin to smoke or use smokeless products may therefore not seem "rational." This chapter (and Chapter 5, "The Tobacco Industry's Influences on the Use of Tobacco Among Youth") examines, within a theory-driven context, the risk factors associated with the onset and development of tobacco use over the course of adolescence and young adulthood. These particular stages of development within the life course are perhaps the only times in life when tobacco use might be appealing and even perceived as functional to individuals (Perry 1999). By definition, adolescence and young adulthood represent the social transition to adulthood, with accompanying risk-taking associated with trying and acquiring adult behaviors. Yet brain development is not complete, and there is immaturity in consequential thinking, impulsivity, and decision-making skills before adulthood. Notably, peer group influences emerge as powerful motivators of behavior change. These changes create a unique window of vulnerability for tobacco use onset in adolescence and young adulthood. As was shown in Chapter 3 ("The Epidemiology of Tobacco Use Among Young People in the United States and Worldwide"), by 26 years of age, nearly all people who are going to use tobacco have already begun, so the focus of primary prevention with young people really spans the ages of 12 to 25 years. This chapter provides important information on these developmental processes, examining large social and physical environments that support or discourage tobacco use, small social groups, cognitive and affective processes, and neurobiological and genetic factors.

The 1994 Surgeon General's report on preventing tobacco use among young people discussed psychosocial risk factors for initiating tobacco use (U.S. Department of Health and Human Services [USDHHS] 1994). That report, which described the developmental stages of tobacco use from onset to regular use, set forth several sets of factors that influence the initiation of tobacco use:

- Sociodemographic factors (socioeconomic status [SES], developmental challenges of adolescence, gender, and race/ethnicity);
- Environmental factors (acceptability and availability of tobacco products, interpersonal variables, perceived environmental variables);
- Behavioral factors (academic achievement, problem behaviors, influence of peer groups, participation in activities, and behavioral skills);
- Personal factors (knowledge of the long-term health consequences of using tobacco, functional meanings of tobacco use, subjective expected utility of tobacco use, variables related to self-esteem, and personality); and
- Current behavior relative to tobacco use (intentions to smoke and smoking status).

The chapter concluded that the following factors promote the initiation and use of tobacco products of some type:

- Relatively low SES,
- Relatively high accessibility and availability of tobacco products,
- Perceptions by adolescents that tobacco use is normative, that is, usual or acceptable behavior,
- Use of tobacco by significant others and approval of tobacco use among those persons,
- Lack of parental support,
- Low levels of academic achievement and school involvement,
- Lack of skills required to resist influences to use tobacco,
- Relatively low self-efficacy for refusal,
- Previous tobacco use and intention to use tobacco in the future,
- Relatively low self-image, and
- Belief that tobacco use is functional or serves a purpose.

The same factors were also found to predict two specific behaviors: cigarette smoking and the use of smokeless tobacco. In addition, having insufficient knowledge

about the health consequences of using smokeless tobacco was found to predict initiation of that behavior. The report noted that use of smokeless tobacco tended to be specific to males, and both parents and youth perceived the use of smokeless tobacco to be relatively safe and acceptable when compared with cigarette smoking (USDHHS 1994).

This chapter, which updates Chapter 4 of the 1994 report, is not meant to be an all-encompassing review. Instead, it focuses on highlighting information gleaned from research conducted after the 1994 report was written. Literature was collected in a theory-guided way, using the Theory of Triadic Influences, to emphasize findings deemed important by the scientific panel convened to write this chapter. To reflect the findings of researchers during the last decade and a half, the description of etiologic factors differs substantially from the earlier report. The chapter investigates the predictors of initiation and progression of tobacco use for two groups: adolescents (girls and boys aged 12–17 years) and young adults (women and men aged 18–25 years). The time from 12–25 years of age constitutes an extended developmental period in which independence in lifestyle is gradually achieved (note that in the United States, youth cannot buy tobacco legally until at least 18 years of age).

Cigarette smoking among adolescents and young adults is a multidetermined behavior, influenced by the unique and overlapping combinations of biological, psychosocial, and environmental factors. These factors can function as either risk or protective factors. Risk factors increase the probability of smoking initiation and the likelihood of continued use, characterized by increases in frequency and intensity. Conversely, protective factors decrease the probability of smoking initiation, as well as reduce the likelihood that experimental use will progress to regular use. An individual's overall risk profile is determined by the interrelations of these various risk and protective factors.

Age-related processes also play a central role in determining smoking risk. Adolescence is a sensitive developmental period, characterized by extraordinary brain changes and high levels of emotionality, impulsivity, and risk-taking. The plasticity of the adolescent brain, together with the relatively immature neurobehavioral systems necessary for self-control and affect regulation, confer a heightened vulnerability for the development of smoking behavior (Steinberg 2007). Similarly, the period following early and middle adolescence (aged 18–25 years) has particular developmental significance with regard to smoking behavior. Many risk behaviors peak during this period of life, including rates of substance use, smoking, risky driving, and unsafe sex (Arnett 2000). It is also during this time period that young people may attend college or begin to take on more conventional adult roles, such as marriage, children, and occupational responsibilities. These life transitions are often associated with concomitant decreases in risky behavior (Bachman et al. 2001; Flora and Chassin 2005) and may represent a turning point in which an individual either permanently adopts smoking behavior or rejects it in favor of a nonsmoking lifestyle.

The development of youth smoking is a dynamic process in which youth progress from early cigarette trials, to intermittent use, to regular use and dependence. Understanding the factors that either interrupt progress along this trajectory or potentiate continued use is critical to intervening with smoking behavior. Importantly, the factors that influence early trials with cigarettes may be distinct from those that influence progression and persistence. Modern conceptualizations of smoking development emphasize a social ecological perspective which considers the broader social and environmental context in which youth tobacco use occurs (Cook 2003; Wilcox 2003; Wen et al. 2009; Ennett et al. 2010). This perspective recognizes that youth and young adults do not exist in isolation. Rather, they inhabit a complex system of layered social and environmental contexts, wherein they learn, socialize, and conduct their daily activities. Theoretical models that consider these multiple levels of neurobiological, sociocontextual, and environmental influence can be labeled "integrated biopsychosocial-ecological models" (Sussman and Ames 2008). In these models, intrapersonal predictors of tobacco use are "nested" within larger social and environmental structures. For example, a person's neurobiological variables function within a set of complex cognitive-related responses and, in turn, operate within a larger context of small social groups (e.g., families, peer groups), that ultimately function within a larger socioenvironmental context (e.g., schools, neighborhoods). Large-scale environmental factors might be either social or physical (e.g., communications in the mass media, access of youth to sales of tobacco products), while environmental factors on a smaller scale could include, for example, a youth's social groups. Intrapersonal factors (e.g., cognitive processes, genetics, and brain systems and structures) could be based on biological or psychological/cognitive variables. These two kinds of predictors, environmental and intrapersonal, may affect each other. For example, a person who shows a lack of self-control related to an imbalance in neurotransmission (an intrapersonal neurobiological variable) and intends to smoke cigarettes in the future (an intrapersonal cognitive variable) would be constrained from smoking in groups of nonsmoking peers at a worksite where smoking was prohibited; here, two kinds of environmental variables would be at play: social (small groups) and physical (prohibition of smoking). Multilevel modeling techniques are commonly used

to examine how factors such as intrapersonal characteristics, families, peer groups, schools, and communities, interact together to jointly influence adolescent tobacco outcomes.

The Theory of Triadic Influence (TTI) (Flay and Petraitis 1994; Petraitis et al. 1995) classifies the elements of 14 different theories about human behavior in three substantive domains. This "meta-theory" is grounded in the major behavioral theories that have been applied to tobacco use with young people. The theories, grouped by categories (in parentheses), are as follows:

- Reasoned action, planned behavior (cognitive affective);
- Social learning, social cognitive/learning (social learning);
- Social control, social development (commitment and social attachment);
- Social ecology, self-derogation, multistage social learning, family interaction (intrapersonal); and
- Problem behavior, peer cluster, vulnerability, domain (relatively comprehensive theories).

The three substantive domains are the following:

- Social/normative,
- Cultural/environmental, and
- Intrapersonal.

These three domains have different "distances" from actual tobacco use and so can be characterized as ultimate, distal, or proximal. For example, a person is affected by her or his culture (ultimate), social and physical environments (distal), and personal perceptions of those environments (proximal) that influence subsequent tobacco use (Petraitis et al. 1995; Turner et al. 2004; Sussman and Ames 2008).

This chapter divides the etiologies of tobacco use into four categories of predictors that overlap with those used by TTI and reflect how research has been undertaken in this area: large social and physical environments, small social groups, intrapersonal cognitive processes, and intrapersonal genetic and neurobiological processes.

By considering these four categories, each of which contains sets of variables, within a broad theoretical context, the etiology of tobacco use may be more completely understood, and new options for the primary prevention or cessation of tobacco use may be suggested. (Figure 5.1 in Chapter 5 provides a visual illustration of TTI.)

Developmental Stages of Tobacco Use

As presented in the 1994 report from the Surgeon General, the uptake of tobacco use can be described as proceeding in stages from nonuse to lower to higher levels of use (USDHHS 1994). Generally, initiation is defined as having ever tried tobacco, experimental use as occasional use, and regular use as an increase in the frequency and quantity of use (USDHHS 1994; Mayhew et al. 2000). In adolescence, regular use is often marked by a pattern of monthly or weekly use and may include psychological and physical dependence on tobacco (Sussman et al. 1995). Not all experimenters become regular users, and different predictors may be important at different points along the course of a person's tobacco use, which underscores the usefulness of conceptualizing the stages of use (Leventhal and Cleary 1980; Flay et al. 1983; USDHHS 1994; Sussman et al. 1995; Mayhew et al. 2000). Social and environmental factors are likely to be more influential in low-level or early tobacco use (and thus are more appropriate targets for intervention during these stages), while intrapersonal factors tend to be strong predictors of later and higher levels of use, when addiction to nicotine is more strongly involved (Tucker et al. 2003; Sussman and Ames 2008). However, a review of 11 cross-sectional and 33 prospective studies suggested that social, environmental, and intrapersonal factors predict both the onset of adolescent smoking and subsequent increases in the frequency and quantity of use (Mayhew et al. 2000). Mayhew and colleagues (2000) found that tolerance for deviance (an intrapersonal variable) appeared uniquely related to the onset of smoking in some of the prospective studies they reviewed, although previous smoking intensity, normative beliefs, estimates of the prevalence of smoking among peers, and perceived lack of parental involvement and support appeared uniquely related to higher levels of smoking onset in other studies.

The stage model is a useful heuristic device (USDHHS 1994) and, as is true with other integrative models, helps to stimulate new research and guide efforts in prevention. In reality, however, it is a simplistic presentation of the development of smoking. In fact, substantial heterogeneity exists in the uptake and progression of smoking behavior. As newer data analytic techniques have become available (e.g., latent variable growth mixture modeling), researchers have been able to empirically identify developmental trajectories of tobacco use that more

clearly capture this heterogeneity (Chassin et al. 2000; Mayhew et al. 2000; Bernat et al. 2008). Several studies have identified three to six discrete smoking trajectories (e.g., Bernat et al. 2008). One of these trajectories typically captures about 10% of adolescents who progress rapidly to persistent, heavy cigarette smoking (Chassin et al. 2000; Colder et al. 2001; Soldz and Cui 2002; Orlando et al. 2004; Stanton et al. 2004; White et al. 2004; Karp et al. 2005; Brook et al. 2006; Bernat et al. 2008; Lessov-Schlaggar et al. 2008). Adolescents in this group may exhibit symptoms of dependence shortly after their first experimentation with cigarettes (Gervais et al. 2006; DiFranza et al. 2007), and they do not appear to go through a generic stage model (i.e., a series of stages) of the uptake of tobacco use. An important focus of research in this area is to identify factors that discriminate among trajectories, especially those factors associated with patterns of early and rapid escalation in smoking, since this group may be at greatest risk for lifelong nicotine dependence (Dierker and Mermelstein 2010). See Chapters 2 ("The Health Consequences of Tobacco Use Among Young People") and 3 for additional discussion of smoking trajectories.

Some studies have extended the stages of tobacco use to describe what a young person might experience before initiating use. For example, Pallonen and colleagues (1998) studied four stages of smoking acquisition: precontemplation (not intending to smoke in the future), contemplation (intending to smoke in the future), preparation (intending to smoke in the immediate future), and recent acquisition (experimenting with smoking). Another schema, the susceptibility model (Pierce et al. 1996, 1998), differentiates never smokers who are open to the possibility of smoking from those who are firmly committed to not smoking. In this model, "nonsusceptible" is the first stage. During this period, the adolescent has yet to consider the possibility of smoking. In the second stage (susceptible), the adolescent becomes open to the idea of smoking a cigarette in the future. The third stage (experimentation) is marked by the first puff of a cigarette. Experimentation continues with occasional smoking episodes until the adolescent has smoked 100 cigarettes. The lifetime smoking of that many cigarettes is a milestone used as a general estimate of the onset of nicotine dependence (stage four). Adolescents who reach the 100-cigarette point but discontinue smoking are classified as former smokers and, in this model, return to the nonsusceptible stage.

The original model of susceptibility, as proposed by Pierce and colleagues (1996), allowed adolescents who had already tried smoking to be classified as nonsusceptible if they expressed a firm commitment not to smoke in the future. Other models (Unger et al. 1997; Filice et al. 2003; Gritz et al. 2003; Sun et al. 2005) used the susceptibility concept to refer primarily to never smokers (who were classified as nonsusceptible), although youth who had already smoked were automatically classified as susceptible. Gilpin and colleagues (2001) added more intermediate stages to this model for adolescents, including puffers (have puffed on a cigarette but have not smoked a whole one), noncurrent experimenters (have smoked between 1 and 100 cigarettes but have not smoked during the past month), and noncurrent established smokers (have smoked more than 100 cigarettes but have not smoked during the past month). Other classifications and measures have been proposed (e.g., Kremers et al. 2001; Prokhorov et al. 2002) in attempts to predict which youth are more likely to become regular smokers as adults. The discussion later in this chapter will focus on specific variables within different levels of influence pertaining to adolescents and young adults but will not specifically incorporate the concept of stages, as these variables have generally not been examined relative to staging.

Considering Different Types of Tobacco Use

Most studies on the etiology of tobacco use have focused on cigarette smoking. Where available, information will be presented in this chapter on smokeless tobacco products (chewing tobacco and snuff), cigars, pipes, and other types of smoked tobacco (e.g., narghile [water pipe] smoking). Despite some differences in the social images associated with different types of tobacco products (e.g., smokeless tobacco is more strongly associated with playing sports, such as baseball, than is cigarette smoking) (Sussman et al. 1989), one could assume that the effects of the different predictors are reasonably similar across different types of tobacco products (e.g., risk taking is associated with use of both cigarettes and smokeless tobacco) (Sussman et al. 1989; Gilpin and Pierce 2003). This chapter examines the four levels of predictive factors of tobacco use and their associations with the onset of these different types of tobacco use and increased levels of use among youth.

Large Social and Physical Environments

The large social and physical environments include influences outside the individual, family, and immediate peer group that may either promote or restrict the use of tobacco. In general, these are more distal influences, including demographic factors that in some way affect a person's subjective perception about the acceptability of smoking, her or his beliefs about the social image that smoking conveys to others, and the availability of tobacco and places to smoke (Petraitis et al. 1995). Examples of these influences are described in detail below.

Large Social Environment

The large social environment defines the norms within a society about whether, when, and for whom smoking is acceptable. Social norms about smoking have changed substantially since the Surgeon General's report of 1964 (U.S. Department of Health, Education and Welfare 1964); in that year, 50% of the U.S. adult male population smoked (Garfinkel 1997), and smoking was becoming increasingly prevalent among women and youth (Cummings et al. 2002). Smoking was also acceptable in nearly all locations, such as worksites, movie theaters, hospitals, and airplanes (Americans for Nonsmokers' Rights 2005). Cigarettes were advertised in many different kinds of media and their use was tied to glamour, wealth, sex appeal, popularity, power, and good health (USDHHS 1994). Now, in the early twenty-first century, however, Americans, especially the better educated and more affluent, are much less likely to smoke (Morgan et al. 2007; Stuber et al. 2008) than in the middle of the twentieth century. According to the Centers for Disease Control and Prevention (CDC 2011), in 2010, 19.3% of adults in the United States were current smokers: 21.5% of men and 17.3% of women.

Religious and Cultural Influences

Religion

Religious doctrines can create social norms that constrain smoking behavior. However, the texts of most of the world's major religions were written before tobacco use became prevalent worldwide. Religious scholars have interpreted the texts and have issued official statements about whether tobacco use is consistent with the doctrines that have emanated from these texts (Simpson 2005). Christianity, Judaism, Buddhism, Hinduism, and Baha'i, for example, do not specifically forbid smoking but indicate that the practice is inconsistent with the teaching and writings of these varied religions, including not deliberately harming one's body, and religious leaders often suggest avoiding intoxicating and addictive substances that can impair judgment (World Health Organization [WHO] 1999). The Mormon religion forbids smoking and refuses smokers entry into the temple (Church of Jesus Christ of Latter-Day Saints 2006). In response to increasing evidence about the physical, social, and cultural effects of tobacco use, Islamic leaders have forbidden tobacco use in several countries (WHO, Eastern Mediterranean Regional Office 2001). In some religions, abstaining from tobacco use is viewed as a sign of the strength essential for religious piety (Bradby 2007).

In contrast, American Indian religions have used tobacco for healing and ceremonies but, in general, do not condone everyday smoking outside of spiritual contexts (Pego et al. 1995). A challenge for tobacco control in American Indian communities is to acknowledge that the sacred use of tobacco is culturally important while preventing recreational use and nicotine dependence. In some instances, tobacco control organizations have partnered with American Indian tribes to develop health education messages that distinguish the sacred use of tobacco from the habitual use of commercial tobacco products (American Indian Tobacco Education Network 2000). For other groups, religious beliefs and practices can create opportunities for smoking cessation during specific occasions such as Ramadan or Lent (Afifi 1997).

Across religious traditions, smoking tends to be less prevalent among those more likely to participate in religious activities. This association has been documented among Jews in Israel (Shmueli and Tamir 2007), Christians in the United States (Nasim et al. 2006; Mann et al. 2007; Turner-Musa and Lipscomb 2007), and adolescents who belong to various religious groups in the United States (Scott et al. 2006; Rostosky et al. 2007). Participation in religious or faith-based activities also appears to exert a uniquely protective effect against smoking escalation among adolescents who have already experimented with cigarettes (Choi et al. 2002; Van den Bree et al. 2004; Metzger et al. 2011). Some studies have distinguished between private religiosity (e.g., frequency of prayer, importance of religion) and public religiosity (e.g., frequency of attendance at religious services, frequency of youth group attendance) (Nonnemaker et al. 2003, 2006).

These studies found that both domains were protective but that private religiosity was more protective against smoking onset while public religiosity was more important for smoking escalation. By encouraging the bonding of adolescents to conventional social institutions and norms, religious involvement may discourage young people from affiliating with irreligious peers, who might introduce them to smoking. Furthermore, adolescents in observant families may be relatively more likely to be monitored closely by their parents, have more adult role models, and be more apt to participate in conventional community activities (Whooley et al. 2002; Bartkowski and Xu 2007).

Race, Ethnicity, and Culture

Chapter 3 of this report describes the epidemiology of smoking across racial and ethnic groups. This section focuses on people's subjective reactions to their racial, ethnic, and cultural identity, including perceived discrimination, the development of ethnic identity, and ethnic pride, in the context of tobacco use.

Research has identified multiple pathways through which race, ethnicity, and culture may influence youth smoking. Among other factors, patterns of youth smoking across racial and ethnic groups have been linked to processes of acculturation, racial/ethnic discrimination, ethnic identity, and cultural norms. Across several immigrant groups in the United States, tobacco use among adolescents increases as the groups acculturate to U.S. ways of living (Epstein et al. 1998; Chen et al. 1999a,b; Unger et al. 2000; Kaplan et al. 2001; Bethel and Schenker 2005; Weiss and Garbanati 2006; Choi et al. 2008). Compared with adolescents who are more oriented toward their families' culture of origin, adolescents who speak English, embrace the individualistic culture of the United States, and prefer U.S. media and customs are more likely to use tobacco (Lara et al. 2005). Among several ethnic minority groups, perceptions of discrimination are associated with an increased risk of smoking (Landrine and Klonoff 2000; Harris et al. 2006; Borrell et al. 2007; Chae et al. 2008; Horton and Loukas in press), perhaps because people are attempting to reduce the resulting emotional stress through self-medication. Conversely, ethnic pride was found to protect against smoking among African American youth (Wills et al. 2007), and a strong ethnic identity was found to be associated with a lower risk of tobacco use among youth in several ethnic/racial minority groups, including African Americans and Hispanics (Brook et al. 2007).

A person's subjective experiences of cultural identity and corresponding place in society may also be associated with whether and how often they use tobacco. Cultural norms against youth smoking within the African American community are thought to contribute to lower rates of youth smoking in this subgroup (Mermelstein 1999; Ellickson et al. 2004; Skinner et al. 2009; Oredein and Foulds 2011). For example, Clark and colleagues (1999) found that antitobacco socialization practices were more common in African American families than in White families. Relative to White households, African American households were more likely to set clear ground rules about smoking and to have had discussions with their children about these rules. Furthermore, Xue and colleagues (2007) found that African American youth living in predominantly African American neighborhoods were less likely to smoke than those living in predominantly White neighborhoods, suggesting that cultural norms in the African American community may operate to constrain youth smoking. Unfortunately, the adolescent advantage seen among African American youth with regard to smoking behavior is not carried into adulthood (Gardiner 2001).

As noted in Chapter 3, African Americans are more likely to smoke menthol cigarette brands than other major subgroups. The reasons probably include several factors (Allen and Unger 2007; Tobacco Products Scientific Advisory Committee 2011). First, the tobacco industry has advertised menthol cigarettes directly to African Americans by associating them with attractive or popular African American role models, including jazz and rap musicians (Gardiner 2004). Second, some African Americans may associate the taste and smell of menthol with folk remedies (e.g., menthol rubs and treatments for sore throat) that are popular in the southern United States. This association between menthol and folk medicine may cause some African Americans to believe, erroneously, that menthol cigarettes are less harmful than nonmenthol cigarettes (Castro 2004). Tobacco advertising perpetuates this belief by labeling menthol cigarettes as "cool" and "smooth." Third, because smoking menthol cigarettes has become normative among African Americans, some members of this minority group may smoke mentholated brands simply because their parents or older siblings smoked them or because they are readily available at home and from friends. The result perpetuates the stereotype that menthol cigarette brands are for African Americans, even among those who are several generations removed from the culture in which menthol was used medicinally.

Among youth in the United States, American Indians and Alaska Natives have the highest prevalence of tobacco use among all racial/ethnic subgroups (see Chapter 3), with usage rates comparable to those of adult American Indians and Alaska Natives (Hodge 2001). Traditionally,

American Indians have used tobacco in ceremonial practices to protect and heal sick individuals and, as a ceremonial tool, it is important in ritualistic exchanges used for social and peaceful purposes. This population also uses tobacco as an educational tool and often links it with storytelling (Hodge 2001). Strong social norms within these communities may support tobacco use, which can be even more of a problem when these communities, or individuals within them, are relocated to urban environments (Hodge and Nandy 2011). However, unlike the studies noted above for other populations, ethnic pride may not protect against tobacco use in this subgroup (LeMaster et al. 2002; Yu et al. 2005). It is also important to note that in epidemiologic and etiologic studies of tobacco use among American Indian and Alaska Native youth, "recreational" use of tobacco is typically not separated from ceremonial use in the design of the research study. Rather, the outcome variable in these studies is simply current (i.e., in the past 30 days) use of tobacco products (LeMaster et al. 2002; Yu et al. 2005; Osilla et al. 2007; Beebe et al. 2008; Yu 2011), and may overlook important differences in the etiology of tobacco use for these groups.

Cultural norms influence smoking in numerous other cultures as well. For example, in China, cigarettes are typically offered to guests as gifts, and refusing cigarettes is viewed as impolite. In that country, men and adolescent boys smoke together after meals as a way of cementing social bonds (Pan 2004; Chen et al. 2006; Grenard et al. 2006; Weiss et al. 2006). In addition, Westernization of developing countries, such as India, has been associated with more tobacco use (Stigler et al. 2010).

Gender

As discussed in Chapter 3, among adolescents there are only small differences by gender in the prevalence of cigarette smoking. In contrast, far larger differences are seen for two other forms of tobacco use. Boys are 4 to 10 times as likely as girls to have used smokeless tobacco in the past month (depending on age), and they are twice as likely as girls to have smoked cigars in the past month (Substance Abuse and Mental Health Services Administration [SAMHSA] 2009; Eaton et al. 2010; Johnston et al. 2011a). Differences between the genders in the prevalence of tobacco use also exist worldwide, but the magnitude of the disparities varies across countries (Warren et al. 2008). According to the 2000–2007 Global Youth Tobacco Surveys (GYTS), the prevalence of cigarette smoking was significantly higher among boys (than girls) in Africa, the Eastern Mediterranean, Southeast Asia, and the Western Pacific but not in the Americas and Europe. Among 151 GYTS sites, 87 showed no differences in cigarette smoking between the genders, 59 demonstrated a higher prevalence among boys, and 5 revealed a higher prevalence among girls. Boys were significantly more likely than girls to report using other tobacco products—pipes, water pipes, smokeless tobacco, and bidis—in the Americas, Europe, and Southeast Asia, but differences between the genders in the use of other tobacco products were not significant in other regions.

Qualitative and quantitative studies conducted in several cultural groups—including Indonesian adolescent boys (Ng et al. 2007), Korean American men (Kim et al. 2005), and Vietnamese young adults (Morrow et al. 2002)—indicate that smoking is viewed as a sign of manhood while being seen as inappropriate for females. In Europe, a study of three generations of women residing in Scotland found that those born in the 1950s associated smoking with femininity, but women born in the 1930s and 1970s did not (Hunt et al. 2004). This suggests generational fluctuations in gender-related norms, but other interpretations are possible.

As immigrant groups acculturate to the United States, gender-related differences in smoking prevalence for these groups may begin to diminish, often because increases are observed among females but not among males. For example, some research has found that acculturation is associated with an increase in smoking among Hispanic girls but not among boys (Epstein et al. 1998), and increases have been observed among Asian American girls but not among boys (Weiss and Garbanati 2006; Choi et al. 2008; Zhang and Wang 2008).

Socioeconomic Status

The SES of youth is derived from such measures as parental income or occupation, parental education, and access to resources. Population-based studies typically use indicators of SES (e.g., education or income) or self-reported measures (e.g., perceived social class or wealth relative to others), or both, to measure SES. Some studies also use measures of neighborhood- or school-level SES as the basis for individual SES.

Numerous studies worldwide have assessed the association between SES and smoking among adults and youth. Low SES has been associated with a high prevalence of smoking in population-based studies in France (Baumann et al. 2007), Germany (Haustein 2006), India (Neufeld et al. 2005; Thankappan and Thresia 2007; Mathur et al. 2008), and the United States (Flint and Novotny 1997). Moreover, even after controlling for individual-level sociodemographic factors, several studies found that the prevalence of smoking was highest in low-income neighborhoods in the the Czech Republic (Dragano et al. 2007), Germany (Dragano et al. 2007), New Zealand (Barnett 2000), the United Kingdom (Kleinschmidt et al. 1995;

Shohaimi et al. 2003), and United States (Cubbin et al. 2001; Tseng et al. 2001; Chuang et al. 2005a,b; Datta et al. 2006; Stimpson et al. 2007).

A growing body of evidence suggests that social and organizational characteristics of disadvantaged neighborhoods may contribute independently to higher rates of smoking, above and beyond the aggregate demographic profile of the community's residents. The "area effect" of smoking has been documented in both national and international studies (Kleinschmidt et al. 1995; Reijneveld 1998; Duncan et al. 1999). However, few studies have directly examined area effects in relation to youth and young adults. Some studies have found that low SES at the neighborhood level (based on income level by U.S./Canada Census block group) or school level was associated with an increased risk of adolescent smoking (Scarinci et al. 2002; Scragg et al. 2002; Matheson et al. 2011). Another study (Lee and Cubbin 2002) found that individual-level, but not neighborhood-level, SES was inversely associated with the prevalence of adolescent smoking. In contrast, one study (Chuang et al. 2005b) found that adolescents in low-SES neighborhoods had a low prevalence of smoking because they received more parental monitoring. Neighborhood characteristics such as social capital (i.e., community cohesion, civic engagement, social ties) have also been examined in relation to smoking. Evans and Kutcher (2010) examined the role of social capital in buffering the effects of neighborhood deprivation on youth smoking outcomes. They found that youth living in low-income communities with high levels of social capital had no excess risk of smoking compared to their more affluent counterparts. Conversely, Matheson and colleagues (2011) found that the effect of neighborhood-level deprivation on youth smoking risk was more pronounced among youth with a strong sense of community belonging, suggesting that in some cases community norms in disadvantaged neighborhoods may function to promote smoking behavior.

The association between SES and adolescent smoking may be moderated by racial, ethnic, and cultural factors. For example, in the National Longitudinal Study of Adolescent Health, neighborhood poverty was a risk factor for smoking among White, but not Black, adolescents (Nowlin and Colder 2007). In a similar analysis of data from the National Longitudinal Study of Adolescent Health, Goodman and Huang (2002) found that low SES was a risk factor for smoking among White adolescents but that high SES was a risk factor for smoking among non-White adolescents. In a study of adolescents in Ontario, Canada (Georgiades et al. 2006), low SES at the family level was a risk factor for smoking among adolescents, but this association was limited to native-born Canadians.

Although one assumes that low SES increases smoking rates, the relationship may actually be bidirectional, with early smoking leading to the attainment of low SES. For example, in a longitudinal study in Finland (Paavola et al. 2004), parents' SES was not a risk factor for adolescents' smoking behavior at age 13 years, but early smoking was a risk factor for adolescents' own low SES in the future (at ages 21 and 28 years). Early smoking also appeared to predict educational attainment later in life. For example, persons who smoked by age 13 years showed lower educational attainment by the age of 28 years.

Several studies have associated adolescents' access to spending money with their risk of smoking (e.g., Darling et al. 2006; Wong et al. 2007). A study of adolescents in Ontario, Canada (Wong et al. 2007), found that compared with students who had less than $10 in spending money per week, students with more than $20 per week were significantly more likely to be experimental smokers, students with more than $30 per week were significantly more likely to be current smokers, and students with more than $60 per week smoked significantly more cigarettes per day. In a New Zealand study (Scragg et al. 2002), students in low-SES schools reported receiving more spending money than students in high-SES schools, and their possession of spending money was a risk factor for smoking. In addition, adolescents who held jobs while going to school were found to have an increased risk of smoking (Wu et al. 2003), possibly because they had money to buy cigarettes or were influenced by their coworkers to smoke, or both.

Economic stress within a family may also be a risk factor for smoking. Unger and colleagues (2004) found that job loss by a parent predicted subsequent smoking among adolescents during a 1-year period. Other studies have found that unemployment (Haustein 2006) and self-reported financial stress in the household (Siahpush et al. 2003) were risk factors for smoking among adult family members, which might then affect children in the household. Employment status also represents a key risk factor for smoking among young adults. Young adults who are unemployed are more likely to be current, daily, and heavy smokers (Novo et al. 2000; Merline et al. 2004; Lawrence et al. 2007).

Educational and Academic Achievement

Among children and adolescents, low academic achievement is associated with smoking. Several studies have found that middle and high school students who smoked had lower grades than those who did not smoke (Dewey 1999; Sutherland and Shepherd 2001; Diego et al. 2003; Scal et al. 2003; Cox et al. 2007; Forrester et al. 2007; Tucker et al. 2008). In one study, this association

appeared to be bidirectional, with poor grades preceding the onset of smoking and smoking preceding poor grades (Tucker et al. 2008). Youth who experience difficulties in school may also feel less connected to their school than do their high-achieving peers, putting them at greater risk for smoking. Connectedness with school (e.g., commitment to school, good relationships with teachers, and a feeling of belonging in school) (Libbey 2004) has been consistently associated with a reduced risk of smoking in the literature (Battistich and Hom 1997; Dornbusch et al. 2001; Scal et al. 2003; Dierker et al. 2004; Rasmussen et al. 2005; Bond et al. 2007).

Among young adults, college students have a lower prevalence of smoking than their peers who do not attend college. For example, in the 2003 Tobacco Use Supplement to the Current Population Survey (Green et al. 2007), current smoking prevalence among 18- to 24-year-olds who were enrolled in college or had college degrees was 14%, compared with 30% among those who did not attend college. In addition, those who did not go to college initiated smoking at younger ages and were less likely to have made quit attempts. According to the 2010 Monitoring the Future study (Johnston et al. 2011b), only 3.9% of college students reported smoking one-half pack or more of cigarettes per day, compared with 15.0% of their peers not in college. The disparity in smoking rates between college students and those not in college appears to precede actual college attendance. In their report, Johnston and colleagues (2011a) also found that the prevalence of smoking one-half pack of cigarettes or more per day was three times as high among high school seniors who were not planning to attend college (12%) as it was among seniors planning to attend college (3.1%). Table 4.1 demonstrates a strong relationship between educational attainment and smoking, with 57.0% of school dropouts aged 16–19 years estimated to be current smokers versus an estimate of 18.6% for those who remained in school (data are from 2006–2010).

School Environment

Youth spend approximately one-third of their time in the school environment (Hofferth and Sandberg 2001). The school setting is frequently used to educate youth about the risks of tobacco use and to implement antitobacco policies. See Chapter 6 ("Efforts to Prevent and Reduce Tobacco Use Among Young People") for a comprehensive discussion of school-based prevention programming. The current discussion is limited to features of the school environment that either promote or protect against youth smoking behavior. One such feature is the tolerance of smoking activity among students or teachers anywhere on the school grounds (Sussman et al. 1995; Ennett et al. 1997; Poulson et al. 2002). Youth who witness adolescents or adults smoking in public (e.g., school) are more likely to perceive smoking as a socially acceptable behavior (Alesci et al. 2003). In this regard, perceptions of prevalent tobacco use on school grounds may promote social norms that encourage smoking uptake and persistence. Studies comparing schools with high versus low smoking rates have found that attending a school with a relatively high smoking rate increases susceptibility to smoking among nonsmoking students (Leatherdale et al. 2006) and increases the odds of ever smoking and current smoking (Ennett et al. 1997; Leatherdale and Manske 2005; Leatherdale et al. 2005). School-based antitobacco policies provide school officials with a mechanism to create a tobacco-free school environment and reduce perceived acceptability of smoking (USDHHS 1994). A growing body of evidence suggests that school smoking restrictions can curb youth smoking behavior, both on and off school premises, when strictly enforced (Evans-Whipp et al. 2004). Studies have shown that consistent enforcement of school tobacco policies results in fewer observations of smoking on school grounds, as well as lower rates of ever smoking and current smoking (Wakefield et al. 2000; Griesbach et al. 2002; Piontek et al. 2008; Adams et al. 2009; Lipperman-Kreda et al. 2009; Lovato et al. 2010). Importantly, Leatherdale and colleagues (2005; Leatherdale and Manske 2005) noted that social influences (e.g., peer smoking, parental smoking) and school factors (e.g., school smoking prevalence) make independent contributions to youth smoking behavior and thus recommend that interventions target both at-risk schools and at-risk students.

Schools are regulated by laws and policies at national, state, district, and school levels. Thus, a district may have more stringent or specific policies than the state in which it resides. Further, individual schools may implement policies beyond those required by the state or district. CDC's School Health Policies and Programs Study, which collects data on school policies from all states and representative samples of school districts and schools every 6 years, shows that the majority of states (90.2%) and districts (99.4%) prohibited cigarette smoking by students in school buildings in 2006 (Jones et al. 2007). However, fewer prohibited cigarette smoking by faculty and staff in school buildings (74.5% of states and 94.3% of districts). Further, the prevalence of restrictions on smoking in other settings and smokeless tobacco use was lower. Only 38.0% of states and 55.4% of sampled districts prohibited all tobacco use during any school-related activity. Similarly, 63.6% of schools (elementary, middle, and high schools) prohibited all tobacco use during school-related activities in 2006.

Table 4.1 Prevalence of smoking in previous month among adolescents aged 16–19 years who have not completed 12th grade, by enrollment status in school; National Survey on Drug Use and Health (NSDUH) 2006–2010; United States

	Enrolled in school but have not completed 12th grade % (95% CI)	Not currently enrolled in school and have not completed 12th grade % (95% CI)
Overall	18.6 (18.1–19.1)	57.0 (54.9–59.2)
Gender		
Male	19.6 (18.9–20.4)	60.1 (57.2–62.9)
Female	17.4 (16.7–18.1)	52.6 (49.4–55.7)
Age (in years)		
16	14.1 (13.5–14.8)	46.0 (37.7–54.5)
17	18.9 (18.1–19.7)	52.3 (47.9–56.7)
18	26.1 (24.7–27.6)	58.9 (55.2–62.4)
19	38.4 (34.1–42.9)	58.5 (55.3–61.6)
Race/ethnicity		
White	22.0 (21.4–22.7)	71.1 (68.5–73.6)
Male	22.5 (21.6–23.5)	72.1 (68.5–75.4)
Female	21.5 (20.6–22.4)	69.8 (66.1–73.3)
Black or African American	11.1 (10.1–12.2)	48.3 (43.2–53.4)
Male	13.5 (12.0–15.2)	53.9 (46.9–60.8)
Female	8.5 (7.3–9.9)	38.8 (31.5–46.6)
Hispanic or Latino	15.1 (14.0–16.3)	38.2 (34.3–42.2)
Male	17.2 (15.5–19.0)	44.9 (39.7–50.3)
Female	12.8 (11.3–14.5)	27.1 (21.7–33.2)
Other[a]	15.1 (13.4–17.1)	65.2 (55.1–74.1)
Male	15.4 (13.1–18.0)	NR
Female	14.8 (12.4–17.6)	NR
Last grade completed		
9th or lower	17.1 (16.1–18.2)	52.3 (48.6–55.9)
10th	16.4 (15.7–17.2)	57.2 (53.5–60.9)
11th	21.2 (20.4–22.1)	61.5 (58.1–64.9)

Source: 2006–2010 NSDUH: Substance Abuse and Mental Health Services Administration (unpublished data).
Note: **CI** = confidence interval; **NR** = low precision, no estimate reported.
[a]Includes Asians, American Indians or Alaska Natives, Native Hawaiians or Other Pacific Islanders, and persons of two or more races/ethnicities.

In addition to school characteristics, increasing attention is being paid to the role of contextual factors within the school neighborhood. Density of tobacco outlets in proximity to schools has been investigated as a possible risk factor for youth smoking. Henriksen and colleagues (2008) found that the prevalence of smoking was 3.2 percentage points higher among students in schools with the highest density of surrounding tobacco retailers compared with students in schools without any tobacco retail outlets. Chan and Leatherdale (2011) found that the number of tobacco retailers surrounding a school increased students' susceptibility to future smoking. Leatherdale and Strath (2007) found a positive association between the density of tobacco retailers surrounding

a school and the likelihood that underage minors would purchase their own cigarettes. Between-school variability in smoking prevalence has also been associated with exposure to tobacco industry promotional and advertising activities in school neighborhoods. Tobacco retail outlets located near schools with higher smoking prevalence had significantly lower cigarettes prices, fewer government-sponsored health warnings, and more in-store tobacco promotions, relative to those located near schools with lower smoking prevalence (Lovato et al. 2007).

Extracurricular and Organized Activities

Adolescents' normative development often includes participation in a wide range of organized group activities (e.g., athletics, school clubs, extracurricular) (Dye and Johnson 2006; Mahoney et al. 2006). Empirical studies have been conducted to examine the effects of different organized activities on adolescents' involvement in substance use, including cigarette smoking. Overall, participation in organized group activities appears to be protective against youth tobacco use (Elder et al. 2000). In particular, team sports involvement has been linked to lower levels of adolescent cigarette smoking (Page et al. 1998; Melnick et al. 2001), with consistent sports involvement (involvement over consecutive years) having a greater influence on smoking behavior than does intermittent participation (Rodriguez and McGovern 2004). In one of the only prospective studies of activity involvement using multiple waves of data (baseline, 15 months, and 24 months), Metzger and colleagues (2011) examined the relation between involvement in organized activities, problem peer associations, and smoking escalation among a sample of experimenting smokers. Participation in team sports directly reduced smoking behavior among current users for boys but not for girls. Among girls, participation in school clubs indirectly reduced smoking behavior via reduced exposure to problem peers.

Large Physical Environment

The large physical environment, or built environment, involves features of public and private spaces that may make tobacco use more or less tolerated or enjoyable. Features of the environment that promote smoking include the tolerance of this activity in public spaces; proximity to entertainment, recreation, and social interaction; and locations that are relatively unlikely to be monitored by adults. In contrast, two of the major goals of antismoking policies (beyond the protection of nonsmokers from exposure to secondhand smoke) are to establish antismoking social norms and to discourage smoking by forcing smokers to refrain from smoking in indoor public places, including indoor workplaces and public housing (Epstein et al. 1999; Levy and Friend 2001; Winickoff et al. 2010). Thus, increases in smoke-free indoor-air policies have logically helped to recast smoking as an activity that can be performed only in specific areas that are typically segregated from entertainment and business locations (Gilpin et al. 2004). Restrictions may create perceptions of social disapproval among both adults and youth, and structuring the physical environment to make it inconvenient for youth to smoke may influence them to not take up tobacco use (Alamar and Glantz 2006).

Another important aspect of the physical environment is the relative accessibility of tobacco products. Strict enforcement of policies that ban retail sales of cigarettes to minors, sales of cigarettes using vending machines, and other means by which youth can gain access to tobacco in the commercial setting can limit their opportunities to obtain these products (Jason et al. 1996, 2008; Rigotti et al. 1997; Stead and Lancaster 2000). The influence of tobacco industry practices is considered in great detail in Chapter 5. Here, tobacco advertising is considered only briefly. The Master Settlement Agreement from 1998 severely restricted cigarette and smokeless tobacco advertising in several venues, including billboards and print media, that have substantial youth readership (Ruel et al. 2004), but tobacco advertising is still ubiquitous in many other venues, such as convenience stores, grocery stores, and bars, and in magazines (Pierce 2007; National Cancer Institute [NCI] 2008). In addition to signs that advertise specific cigarette brands, tobacco advertising can appear on functional items that are distributed to store owners, such as trash cans or change trays near cash registers, napkins and decorations in bars, and logos on race cars or sports uniforms (CDC 2008). (While still subject to a legal challenge, the FDA rule prohibits the distribution of cigarette or smokeless tobacco branded functional items, and it prohibits brand name sponsorship of athletic events or teams [*Federal Register* 1996; 2010]). Thus, even after the Master Settlement Agreement, opportunities for exposure to tobacco brand names and images are widespread. Numerous studies have found that youth who recall more exposure to tobacco advertising are more likely to experiment with smoking or to hold favorable attitudes toward it (DiFranza et al. 2006). Furthermore, a meta-analysis of 51 studies (Wellman et al. 2006) found that exposure to protobacco marketing and media significantly increased the odds among youth of holding positive attitudes toward tobacco use (odds ratio [OR] = 1.51; 95% confidence interval [CI], 1.08–2.13) as well as the odds of initiating tobacco use (OR = 2.23; 95% CI, 1.79–2.77).

Youth are also exposed to tobacco imagery through product placements in movies, television shows, and video

games. Exposure to fictional characters who smoke can create an exaggerated social norm about the prevalence and acceptability of smoking (Sargent et al. 2000). Indeed, longitudinal studies have found that adolescents whose favorite movie stars smoke on screen or who are exposed to a large number of movies portraying smokers are at a high risk of smoking initiation (Sargent et al. 2000; Distefan et al. 2004). For example, among 10- to 14-year-old adolescents, those in the highest quartile of exposure to smoking in movies were 2.6 times as likely to initiate smoking as were those in the lowest quartile (Sargent et al. 2005). Tobacco is also promoted to youth on the Internet through social media and online tobacco retailers and the informal Web sites and chat rooms that glamorize the smoking lifestyle and culture (Ribisl et al. 2003).

Research on the effects of tobacco advertising on smoking behavior is methodologically challenging, although recent approaches have provided more valid and reliable data than were available in earlier years. Still, survey measures of exposure to tobacco advertising may be inaccurate. Their validity requires the respondent to see an ad, recognize it as a tobacco ad, encode the image in memory, and retrieve the image from memory when prompted by a survey question (Unger et al. 2001). Moreover, tobacco advertising may affect tobacco-related attitudes and behaviors without the respondent's conscious awareness or recall. To avoid this problem, some studies have assessed attitudes about tobacco after having placed, and randomly assigned, study participants in artificial laboratory settings to view either tobacco advertisements or neutral stimuli (e.g., Shadel et al. 2008). These studies have internal validity but lack external validity. Another approach is to use time-series data to examine the effects of bans on tobacco advertising on the subsequent prevalence of smoking. A review of 24 such studies (Quentin et al. 2007) concluded that, overall, bans on tobacco advertising produce modest decreases in tobacco consumption, even though the changes found by the authors were not statistically significant for all of the studies. More information about the effects of tobacco advertising, promotional activities, and bans on advertising is presented in Chapters 5 and 6.

Summary

The large social environment incorporates numerous macrolevel social processes that affect tobacco use by influencing social norms relating to gender role, religion, and culture as well as norms for specific segments of the population, such as those with low SES or modest educational attainment. For most of the twentieth century, tobacco use was more socially acceptable for men than for women in the United States. In recent decades, however, such differences between the genders have greatly narrowed, although in most ethnic groups, boys and young men are still more likely than girls and young women to use certain forms of tobacco (smokeless tobacco, cigars, and pipes).

In general, religious participation protects against tobacco use. Some religions have specific prohibitions against tobacco use, while others encourage certain social behaviors to prevent youth from experimenting with substance use and rebellious actions. American Indians use tobacco as a sacred substance, but many tribes attempt to maintain a distinction between the sacred use of traditional homegrown tobacco and the use of commercially produced tobacco.

Other chapters in this report present detailed information about variations in tobacco use among different racial/ethnic groups. The present chapter points out the consistent finding that racial/ethnic pride and a strong ethnic identity generally protect against tobacco use, but perceptions of racial/ethnic discrimination are a risk factor for such use. Additional research is needed to understand the psychological and cognitive mechanisms through which perceptions of racial/ethnic identity influence decisions about tobacco use.

The differences in tobacco use between the genders are more pronounced in many other countries than they are in the United States (Warren et al. 2008). Immigrants from such countries bring their norms for gender roles with them when they move to the United States, and thus, many immigrant groups show a higher prevalence of smoking among males than among females. As immigrants acculturate, these gender-based differences narrow, generally because tobacco use among females often increases. Therefore, immigrant girls and young women who acculturate to the United States represent a higher-risk group for tobacco use.

Mainstream U.S. culture has increasingly embraced an antitobacco norm. As a result, only about one in five American adults now use tobacco, but use is far more common among those of low SES or low educational achievement. Among adolescents, poor school achievement is associated with both low SES and tobacco use. However, the association between educational achievement and tobacco use may be bidirectional, or another variable, such as risk taking, may influence educational attainment while also being tied to smoking. Furthermore, neighborhood-level risk factors may contribute to the probability of youth smoking, in excess of the risk conferred by individual-level influences. The large physical environment contains features that facilitate or impede

tobacco use, including the availability of comfortable and convenient places to smoke, the availability of or access to tobacco products, and cues from the media to use tobacco. In general, the available evidence suggests that (1) nonsmoking policies create antismoking social norms and decrease smoking behavior, and (2) exposure to protobacco media messages, particularly in movies or advertising, increases perceptions of the acceptability of smoking and thus increases smoking behavior. More details about the effects of changes in the larger social and physical environments are provided in Chapter 6.

Small Social Groups

The family and peer groups are the two most important small social groups in the development of young people and their use of tobacco. This section focuses on the influence of these social groups on youth and, when research is available, on young adults.

Homogeneity of Tobacco Use Among Adolescents and Friends

Multiple cross-sectional and longitudinal studies have shown that peer factors—in particular, friends' smoking behavior and adolescents' perceptions of their friends' smoking behavior—are associated with adolescents' own smoking (Conrad et al. 1992; USDHHS 1994; Jackson 1997; Tyas and Pederson 1998; Alesci et al. 2003; Kobus 2003; Ali and Dwyer 2009; McVicar 2011; Villanti et al. 2011). The similarity, or homogeneity, of smoking patterns for adolescents and their friends has led many researchers to infer that peers influence adolescent smoking (Bauman and Ennett 1996; Kobus 2003; Arnett 2007). The mechanism of influence most often postulated is social learning (Bandura 1977b; Petraitis et al. 1995), whereby adolescents learn about tobacco use by observing peers who use tobacco and are reinforced for using tobacco by perceiving apparent advantages, such as gaining acceptance by peers or establishing a particular social identity. Other mechanisms of transmission from peers are direct pressure to smoke and offers of cigarettes and other tobacco products. However, direct peer pressure is infrequently documented as a risk factor for smoking (Urberg et al. 1990; Sussman et al. 1993; Hoving et al. 2007). Adolescents are more likely, however, to obtain cigarettes from peers than from adults or through commercial transactions (Harrison et al. 2000; Forster et al. 2003; White et al. 2005; Robinson et al. 2006a), and youth who reported receiving offers of cigarettes from friends were more likely to initiate smoking and progress to experimentation (Flay et al. 1998).

Cross-sectional studies cannot reveal whether youth are influenced to smoke by their friends or whether they choose friends on the basis of their smoking status (Bauman and Ennet 1996; Kobus 2003; Arnett 2007). Longitudinal studies, however, demonstrate that having friends who smoke is a consistent predictor of tobacco use: youth who report having more friends who smoke (than friends who do not smoke) are more likely to have initiated or to subsequently initiate smoking (Flay et al. 1994; Jackson et al. 1998; Scal et al. 2003) or to progress to higher levels of smoking (Wang 2001; Dierker et al. 2004; Audrain-McGovern et al. 2006a–c). Also, perceptions of friends' smoking predict developmental trajectories of smoking (Chassin et al. 2000; Audrain-McGovern et al. 2004; Abroms et al. 2005), and according to both cross-sectional (Boyle et al. 1997) and longitudinal (Tomar and Giovino 1998) studies, youth who perceive that their peers use smokeless tobacco are at increased risk of using that product.

Two studies (Killen et al. 1997; Urberg et al. 1997) found that having friends who smoke influences the initiation of smoking among both adolescent boys and girls, and two other studies (Hu et al. 1995; Flay et al. 1998) found the effects of friends' smoking to be stronger for girls than for boys. In addition, friends' smoking may be more salient for White than for Black youth (Headen et al. 1991; Landrine et al. 1994; Robinson et al. 2006b), although several studies observed common effects of friends' smoking on White and minority youth (Flay et al. 1994; Gritz et al. 2003; Kandel et al. 2004).

According to two studies (Chassin et al. 1986; Bauman et al. 2001), the influence of friends' smoking on progression of smoking stage remains constant throughout adolescence, although some studies suggest that peer influence may decrease as the levels of prior smoking by the adolescent increase (Hu et al. 1995), with transitions in smoking stage (Flay et al. 1998; Bricker et al. 2006b), and during later stages of adolescence (Chassin et al. 2000; Tucker et al. 2003).

Furthermore, several studies have suggested that the influence of friends' smoking fails to predict initia-

tion of smoking in young adulthood (Ellickson et al. 2001; Choi et al. 2003; Tercyak et al. 2007; White et al. 2007), but the findings of these studies have been inconsistent. Several other studies observed no influence of friends' smoking on various measures of smoking in young adults (Oygard et al. 1995; Brook et al. 1997; Wetter et al. 2004; Patton et al. 2006), but other studies did observe such an influence (West et al. 1999; Andrews et al. 2002; Hu et al. 2006; Pederson et al. 2007; Tucker et al. 2008). Explanations for the mixed findings may rest on differences in the smoking measures examined (e.g., current smoking, daily smoking, nicotine dependence) and the timeframe for measuring the influence of friends' smoking, whether adolescence or closer to young adulthood. West and colleagues (1999), for example, found that friends' smoking at 18 years of age, but not at 15 years of age, predicted smoking among young adults between the ages of 18 and 21 years. Overall, however, studies suggest that friends' smoking may be less relevant to the initiation and progression of smoking during young adulthood than during adolescence.

Disapproval among one's peers is one of the few peer factors, other than friends' smoking, that longitudinal studies have examined in both adolescents and young adults. In general, adolescents who perceive that their friends disapprove of smoking are less likely than their peers (who perceive that their friends approve of smoking) to initiate smoking (Chassin et al. 1986; Wang et al. 1999; Gritz et al. 2003). However, some studies have found no effects of peer disapproval on initiation (Flay et al. 1994; Carvajal and Granillo 2006). A longitudinal study of college students found that peer disapproval predicted decreased progression in smoking but not its initiation (Choi et al. 2003). Another study, however, found no effects of peer disapproval of smoking on transition to regular smoking between grade 12 and 23 years of age (Tucker et al. 2003).

Most longitudinal studies of tobacco use among youth have not measured changes in friendships or tobacco use by friends. Clearly, these data are needed to assess the contribution of selection of friends to the homogeneity of tobacco use among adolescents and their friends. Evidence dating from the late 1970s and 1980s suggests that adolescents are influenced to smoke by their friends and to select friends with similar tobacco use (Cohen 1977; Fisher and Bauman 1988). Fisher and Bauman (1988) examined the contributions of selection of friends and socialization (influence by friends) to homogeneity of cigarette smoking in adolescent friendship pairs; the authors collected linked information about the identity of friends and daily smoking from seventh and ninth graders at two time points 1 year apart. Selection effects, with smokers acquiring friends who smoked and nonsmokers acquiring friends who did not, were stronger than the effects of socialization as reflected by smokers influencing nonsmoking friends to smoke.

In a cohort of students assessed five times from grades six to nine, Simons-Morton and colleagues (2004) used growth modeling methods to examine relationships between the progression of smoking stage and affiliation with friends who smoked. Findings were consistent with the idea of selection effects but not with socialization effects; that is, adolescents with higher initial levels of smoking acquired over time more friends who smoked, but having friends who smoked did not predict progression in smoking. Similarly, in a cohort of 6,527 adolescents surveyed at the ages of 13, 16, 18, and 23 years, Tucker and colleagues (2008) estimated adolescents' and their friends' cigarette smoking (as well as parental smoking and approval of smoking). The study found reciprocal associations between smoking by youth and smoking by their peers. In support of the concept of selective affiliation, having friends who smoked was predicted at all ages by prior smoking of the adolescent, but smoking by peers (socialization) predicted smoking among young people only when adolescents reached 23 years of age.

Other longitudinal studies on similarities in cigarette smoking within friendship groups or among friends have found evidence for both selection and socialization processes, with the two processes contributing about equally (Ennett and Bauman 1994; Mercken et al. 2007; Go et al. 2010), or with stronger evidence for selection than for socialization effects (Engels et al. 1997, 1999, 2004; Wang et al. 2000; de Vries et al. 2006; Hoffman et al. 2007; Mercken et al. 2009, 2010). In one of the few studies of selection and socialization processes among college students, McCabe and colleagues (2005) found that current cigarette use was higher among fraternity and sorority members than among students who did not belong to these organizations but that the difference could be attributed to selection effects rather than to the influence of membership.

For adolescents, both selection (of friends) and socialization likely contribute to the homogeneity of tobacco use among friends. For example, Hall and Valente (2007), using social network methods to explore peer influence (socialization) and peer selection simultaneously, demonstrated effects of the selection of friends (i.e., choosing relatively more friends who smoked) in sixth grade on smoking behavior in seventh grade. At the same time, processes of influence (in this case being selected as a friend by relatively more smokers) in the sixth grade shaped the peer environment in the seventh grade and increased susceptibility to smoking in that grade.

An important implication of the findings on the contribution of selection of friends to the homogeneity of

tobacco use among peers is that when adolescents' inclination to select friends similar to themselves with regard to smoking is not considered, whether in cross-sectional or longitudinal studies, the effects of peer influence through selection may be overstated. At the same time, when the role of peer influence through selection is inflated, explanatory variables in the social environment other than selection of friends (e.g., characteristics of one's family as well as tobacco advertising and other attributes of the media) may be inappropriately discounted (Bauman and Ennett 1996; Kobus 2003; de Vries et al. 2006; Arnett 2007).

Aside from the selection and socialization processes, external factors may account for some similarities in tobacco use among adolescent friends. Adolescent friendships align along demographic, behavioral, and attitudinal characteristics, with the background characteristics of race/ethnicity, gender, and age or grade in school forming the largest divides (Kandel 1978; Shrum and Creek 1987; McPherson et al. 2001). Eiser and colleagues (1991) found that youth between the ages of 11 and 16 years strongly resembled their three matched friends on smoking behavior, background attributes, and a range of other attitudinal and behavioral characteristics. Future studies should continue to use analytic models that control for background and other shared characteristics to accurately assess the contributions of peers to tobacco use.

Interaction-Based Versus Identity-Based Peer Groups

Assessing the role of peers in tobacco use has become increasingly complicated because adolescents interact (network) within multiple peer groups and these multiple interactions may generate different personal perceptions within each group network (Brown 2004). Investigating interaction-based social networks is a relatively recent but growing area of inquiry in adolescent tobacco use and is accomplished by analyzing friendship linkages (Kobus 2003; Valente et al. 2004; Ennett et al. 2006). In contrast, studies of peer group identification have a long history in research on tobacco use among youth and demonstrate that adolescents' perceptions of their peers' and their own social identity are related to tobacco use (Sussman et al. 2007). Both social network and peer group identification studies are concerned with relating attributes of the larger peer group, typically all same-school peers, to adolescent tobacco use. When the larger peer network is the focus, investigating adolescent social position, social standing, reputation, and perceived norms becomes a salient consideration.

Peer Social Networks

Most social network studies of tobacco use among youth measure social networks within schools because most friendships are anchored at the school and the school is the easiest location in which to measure whole groups (Blyth et al. 1982). Youth networks, however, also exist outside of schools—in neighborhoods, sports leagues, clubs, faith organizations, cyberspace, and other places. A social network can be described as the entire set of relationships identified by adolescents' naming of other youth as friends or best friends. Researchers map these nominations by youth to discover nonrandom relational patterns of direct and indirect links between adolescents and reciprocated (mutual friendship) and absent (no friendship) linkages. Studies of social networks assume that relational patterns have implications for behavior (Wasserman and Faust 1994), and social network analysis is a set of techniques with specific mathematical algorithms and associated software (Valente et al. 2004). The techniques are used to identify and measure the characteristics of relational patterns, such as the social position of each adolescent in the network or the density of relationships in the network (Wasserman and Faust 1994; Valente et al. 2004).

An advantage of data obtained on social networks is that measures of the friends' tobacco use can be based on the friends' own reports rather than on adolescents' perceptions of their friends' use. Adolescents tend to project their own tobacco use behavior onto their friends, thereby spuriously inflating the similarity in tobacco use between adolescents and their friends (Sherman et al. 1983; Bauman and Fisher 1986; Urberg et al. 1990; Bauman and Ennett 1996). By using social network data, investigators can avoid such bias.

From the pattern of friendship links in a social network, adolescents can be categorized into three mutually exclusive social positions (Shrum and Creek 1987; Brown 2004): group members; liaisons or peripherals (those who have friendships with adolescents in different groups while not belonging to any group); and relative outsiders or isolates. Analyses of social networks have shown groups to be generally homogeneous in smoking behavior, whether characterized as predominantly smoking or nonsmoking (Ennett et al. 1994; Urberg et al. 1997; Pearson and Michell 2000). However, several studies have found that, with some qualifications, adolescents who are group members or liaisons are less likely to smoke than adolescents who are relative isolates (Ennett and Bauman 1993; Pearson and Michell 2000; Abel et al. 2002; Fang et al. 2003; Pearson et al. 2006). Forming relationships with peers may indicate social competence in navigating

the school social environment and in one study appeared to be protective against smoking when compared with social marginalization (Ennett et al. 2008). In support of this possibility, multiple studies that did not use social network methods have suggested that higher social and personal competencies protect against smoking (e.g., Botvin et al. 1993; Jackson et al. 1994; Epstein et al. 2000; Finkelstein et al. 2006).

Other studies, however, found that liaisons had higher rates of smoking than did relative isolates or group members (Henry and Kobus 2007) and that liaisons (Ennett and Bauman 1994) and group peripherals (Pearson and Michell 2000) with links to smoking groups had an increased likelihood of smoking (versus those with links to nonsmoking groups). Pollard and colleagues (2010) used a multimethod analytic approach to determine whether adolescents' friendship network position (i.e., group member, liaison, or isolate) predicted membership in one of six developmental smoking trajectories. Belonging to a smoking group, or having ties to a smoking group, predicted membership in higher use smoking trajectory groups over a 6-year period. Importantly, network position accounted for variance in smoking trajectory group above and beyond that which could be explained simply by the number of smoking friends. Using a different measurement approach, Aloise-Young and colleagues (1994) found that group outsiders with a best-friend smoker were significantly more likely to become smokers 1 year later than group members with a best-friend smoker. Dishion and colleagues (1999) found that youth with fewer social skills may gravitate to peers and groups characterized by smoking and initiate smoking as a way of fitting in. Indeed, several studies point to adolescents' desire for gaining acceptance or approval by their peers as a reason for smoking (Barton et al. 1982; Perry et al. 1987; NCI 2008).

Social networks are the point of reference for an adolescent's social standing, as indicated by the youth's popularity or centrality. Results of the few social network studies that have examined whether elevated standing in the social network is associated with smoking have been inconsistent, with findings that have found greater popularity to be predictive of smoking initiation (Valente et al. 2005), no relationship to increases in cigarette smoking (Ennett et al. 2006), and a dependence on other attributes of the school environment (Alexander et al. 2001; Pearson et al. 2006) for its effect on smoking. For example, Alexander and colleagues (2001) found that the level of smoking in the school moderated the association between popularity and current smoking, such that greater popularity was associated with lower risk of smoking in schools with a lower prevalence of smoking but with a higher risk of smoking in schools with a higher prevalence of smoking.

Similar to the findings of social network studies, studies of sociometric status suggest that smoking is influenced by social marginalization and by social impact. In studies of sociometric status, youth name the peers they like the most and the least, and researchers use the choices to classify or rate individuals as popular (well liked and not often disliked), rejected (disliked and not often liked), neglected (rarely mentioned as liked or disliked), controversial (frequently mentioned as liked and disliked), or average (Brown 2004). In a longitudinal sample of 7th, 8th, and 9th graders, youth classified as rejected and controversial were more likely than average youth to report lifetime smoking in 7th grade and to begin smoking 1 year later, while popular youth were marginally less likely than average youth to report ever smoking (Aloise-Young and Kaeppner 2005). Similar results were reported in a long-term study of boys in which the onset of smoking was more common in 5th through 10th grades among those who, in 4th grade, received more "disliking" than "liking" nominations (Dishion et al. 1999) and were classified as rejected and isolated (Dishion et al. 1995). Moreover, in a long-term longitudinal study of Swedish youth, students rated by teachers as unpopular in school were more likely to smoke at 16 years of age, and being unpopular during adolescence had an indirect effect on smoking in young adulthood (Novak et al. 2007).

Identification with a Peer Group

Adolescents use such factors as perceived popularity, academic inclination, participation in athletics, substance use, and other behaviors to place themselves and their peers into peer groups or "peer crowds" (Brown 2004; Sussman et al. 2007). Identifying youth with a particular type of peer group, such as "nerds" or "jocks," makes a statement about that individual's identity within youth culture, although it may not reflect direct interactions among adolescents in the group.

In an early study of peer group identity, Mosbach and Leventhal (1988) found that higher percentages of current smoking were reported by seventh and eighth graders self-identified as "dirts," who were mainly boys who smoked cigarettes, used other drugs, were poor students, and engaged in a variety of problem behaviors (62.5% prevalence of smoking among this group), and "hotshots," who were popular and academically successful students (27.8%), than by "regulars," who did not belong to any group and were typical of junior high students (9.2%), and "jocks," those with a strong interest in organized sports (4.3%). Findings from a review of identification research in peer groups mirrored these results (Sussman et al. 2007). This review collapsed group names across studies into five general categories of peer groups: elites, athletes,

academics, deviants, and others. Among the 14 studies that investigated cigarette smoking, 13 found that youth in the "deviants" group were most likely to smoke; in the remaining study, in which a deviant group was not identified, students in the "elites" group were most likely to smoke. In another analysis of the same 14 studies, "elites" were also very likely to smoke, but were not as likely to do so as deviants (Sussman et al. 2007). A concern with the studies on peer group identity is the possible redundancy in measurement of drug use or smoking stemming from the fact that adolescents may use drug behaviors to identify and differentiate peer groups. Indeed, in several studies, "druggies" were one of the peer groups included under the "deviant" classification (Sussman et al. 2007). Clearly, if smoking contributes to peer group identity, the correlation between peer group identity and smoking will be inflated. This issue can be reduced in importance, however, in longitudinal studies that control for adolescents' prior smoking behavior when predicting smoking from peer group identity.

Only two of the studies, one from 1994 and the other from 2000, that were reviewed by Sussman and colleagues were longitudinal. After adjusting for prior smoking, one study found that identification as a member of a deviant group predicted cigarette smoking 1 year later (Sussman et al. 1994), but no effects were found in the other study (Sussman et al. 2000). However, the likelihood of detecting effects on smoking among those self-identified in the deviant group may have been compromised by the restriction of the sample to youth already identified as high risk. Thus, this sample may have had less variability in deviance across peer groups than other samples of youth have had.

Normative Expectations of Peers

A large peer group, typically peers at school, is generally the reference group that adolescents use to estimate the prevalence of smoking among their peers, and this is used as an indication of their normative expectations about smoking (Sherman et al. 1983; Sussman et al. 1988; Botvin et al. 1992b). As with their estimates of smoking by close friends, adolescents' estimates of the prevalence of peer smoking reflect to some degree a projection of their own behavior in a phenomenon known as the "false consensus effect" (i.e., assuming in error that others do the same thing as one does) (Sherman et al. 1983; Bauman et al. 1992; Botvin et al. 1992b). Regardless of their own smoking status, adolescents tend to overestimate actual smoking rates among their peers, and overestimation of these rates has predicted the initiation of smoking (Botvin et al. 1992a; Simons-Morton 2002; Forrester et al. 2007), experimentation (Flay et al. 1998), and progression in smoking stage (Simons-Morton and Haynie 2003). Cunningham and Selby (2007) found that young adult smokers exhibited the same tendency to overestimate the prevalence of smoking among their peers. Earlier, Ellickson and colleagues (2003) conducted a rare study that investigated both the actual and perceived school-level prevalence of smoking. The study adjusted for individual smoking at baseline and reports of close friends' smoking. The findings indicated that the seventh graders' perceived prevalence of smoking, but not the actual prevalence of smoking among their peers, predicted smoking 1 year later among the seventh graders in this study. The results suggest that adolescents' perceptions of their peers' smoking matter more to their own smoking behaviors than what their peers are actually doing.

Family Context

The family is a source of social, genetic, and biological factors (see "Genetic Factors and Neurobiological and Neurodevelopmental Processes" later in this chapter), and its effects must be assessed as well. As with the peer context, the content and quality of interactions between youth and their family members, rather than the actions of parents alone, contribute to tobacco use among youth. Studies of the family context have focused primarily on four factors: smoking by parents and older siblings, dimensions of parenting behavior, family relationships, and parental reactions to smoking by their children (Conrad et al. 1992; Tyas and Pederson 1998; Avenevoli and Merikangas 2003).

Smoking by parents is the most frequently assessed parental risk factor for smoking by youth, given the central role that parents serve in young people's lives, but this factor has been assessed much less often in studies of young adults. Many studies have found that exposure to parental smoking is predictive of the onset, progression, and developmental trajectories of smoking by youth (e.g., Biglan et al. 1995; den Exter Blokland et al. 2004; Hill et al. 2005; Brook et al. 2006; Peterson et al. 2006; Chassin et al. 2008; Gilman et al. 2009), but other studies have failed to find any such effects (e.g., Cohen et al. 1994; Flay et al. 1994; Distefan et al. 1998). In addition, several studies suggest that the influence of exposure to parental smoking persists into young adulthood (Oygard et al. 1995; Chassin et al. 1996, 2000; Brook et al. 1997; Hu et al. 2006; Patton et al. 2006; Otten et al. 2011), but other studies have found it does not (West et al. 1999; Pederson et al. 2007). The inconsistent findings in studies of smoking among youth may be attributable to differences in the extent to which such studies have included other parenting variables, peer-smoking variables, or perhaps other variables (Tyas and Pederson 1998; Avenevoli and

Merikangas 2003). Notably, a recent meta-analysis concluded that parental smoking is strongly associated with smoking among youth (Leonardi-Bee et al. 2011). The effects of parental smoking on smoking among youth can be seen in both boys and girls (Andrews et al. 1997), but the effects may be stronger for girls (Hu et al. 1995). Findings by race/ethnicity are mixed, with several studies suggesting that parental smoking may be more salient for White than for African American/Black youth (Landrine et al. 1994; Hu et al. 1995; Griesler et al. 1998), but Gritz and colleagues (2003) drew a different conclusion, that African American youth were susceptible to smoking if anyone in their household smoked. In addition, Hu and colleagues (2006) found that parental smoking may be more important for young adults than for youth, and two studies found that such smoking may be relatively more important for Hispanic youth (Landrine et al. 1994; Griesler and Kandel 1998), but Hu and associates (1995) and Gritz and coworkers (2003) obtained contrasting results (that demonstrated the importance of household smoking and youth smoking among Hispanic youth). One study found that parental smoking predicted transition to daily smoking for three racial/ethnic groups: White, Black, and Hispanic adolescents (Kandel et al. 2004). The effects of parental smoking on smoking by adolescents appear to remain constant over the adolescent period (Chassin et al. 1986; Hu et al. 1995; Bauman et al. 2001) or may even increase (Bricker et al. 2007) throughout this time.

Longitudinal studies of effects on smoking among youth have looked at older siblings less often than they have looked at parents. Studies have found that smoking by older siblings influences smoking among youth more consistently than does smoking by parents (Conrad et al. 1992; Tyas and Pederson 1998; Avenevoli and Merikangas 2003), and this includes effects on the behaviors of initiation (e.g., Rajan et al. 2003; Forrester et al. 2007) as well as progression to higher levels of tobacco use (e.g., Hill et al. 2005; Bricker et al. 2006a). Bricker and colleagues (2006a), who followed 4,576 youth from 3rd through 12th grades, found that after controlling for smoking by parents and close friends, smoking by older siblings—measured in early childhood—predicted daily smoking by adolescents 9 years later. In fact, the effects of siblings' smoking were as strong as the effects of smoking by close friends. In contrast, some studies of smoking by young adults suggest that siblings' smoking may not be an important risk factor for the initiation or persistence of smoking in this older group (Oygard et al. 1995; West et al. 1999; White et al. 2002; Pederson et al. 2007).

Multiple studies of youth indicate that a higher quality of parent-adolescent relationships—variously defined by such indicators as closeness, supportiveness, and involvement—protects youth against smoking (e.g., Doherty and Allen 1994; Scal et al. 2003; Kandel et al. 2004; Mahabee-Gittens et al. 2011). In addition, several studies suggest that parental monitoring of their child's activities, whereabouts, and friends may reduce the likelihood of smoking (e.g., Biglan et al. 1995; Dishion et al. 1999; Simons-Morton 2002). Conversely, other studies find that some family supervisory practices (e.g., disciplinary practices) are not likely to deter youth from smoking (Chassin et al. 1986; Côté et al. 2004; Hill et al. 2005). Some studies have considered dimensions of both parental support and behavioral control by combining selected variables to define parenting styles: authoritative (high support, high control), authoritarian (low support, high control), indulgent (high support, low control), and disengaged (low support, low control) (Baumrind 1985). Jackson and colleagues (1994) observed that adolescents with authoritative parents were less likely to initiate smoking, while Chassin and coworkers (2005) found that adolescents with disengaged parents were more likely to smoke, even after controlling for parental smoking.

Two studies found that family conflict may increase the risk of smoking among youth (Duncan et al. 1998; Flay et al. 1998). Earlier, Biglan and colleagues found an indirect effect of family conflict on smoking by youth (Biglan et al. 1995). In addition, smoking-specific parental attitudes and practices appear to influence youth smoking: youth who perceive that their parents disapprove of smoking have been found to be less likely to smoke (Sargent and Dalton 2001; Miller and Volk 2002; Simons-Morton and Haynie 2003), but some studies found no such effects (Hill et al. 2005; Carvajal and Granillo 2006) or effects at only particular stages of smoking (Distefan et al. 1998) or at certain ages (Tucker et al. 2008), with effects less likely in young adulthood (Ellickson et al. 2001; Tucker et al. 2003). Similarly, studies have found parent-child communication about smoking to be a protective factor (Huver et al. 2006), but this may be the case only in nonsmoking families (Chassin et al. 2005) or at certain stages of smoking (Distefan et al. 1998). Ennett and colleagues (2001) found that multiple dimensions of parent-child communication about tobacco use had no effects on initiation of smoking among youth but that harsher parent-child communication on the rules about smoking and discipline for smoking had detrimental effects (i.e., it escalated smoking).

Additional insights into how parents influence adolescent smoking have come from complex longitudinal models that included both parental and peer factors. For example, in a longitudinal sample of 14- to 17-year-olds, Biglan and colleagues (1995) observed that family conflict led to poor parental monitoring that, in turn, led to an increased risk of smoking. Several studies found that parental smoking indirectly influenced adolescents'

smoking through their selection of friends who smoked (Chassin et al. 1998; Engels et al. 1999, 2004; Tucker et al. 2003; Simons-Morton et al. 2004) and through cognitive factors, such as adolescents' expectations of the outcomes of smoking, perceptions of whether their parents approved of smoking, and intentions to smoke (Flay et al. 1994). Other studies have observed that the effects of affiliating with friends who smoked were diminished when parents were perceived to disapprove of smoking (Sargent and Dalton 2001). In general, studies suggest that parental risk factors tend to become less important relative to peer risk factors along with increasing age (Flay et al. 1994).

Summary

The literature on the contributions of small social groups to tobacco use among youth, and to a lesser extent to tobacco use among young adults, points to the importance of peers and family in the initiation of tobacco use as well as its continuation and progressive use, particularly of cigarettes. How peers and family actually affect and potentially support or deter tobacco use among youth is a complex question that is not reducible to single causal factors. Instead, the literature suggests that the entire social context (i.e., the interrelations and attributes within and between peers and family and adolescents' perceptions of their own social environment) helps to shape smoking behavior among youth.

Understanding the influence of friends' smoking is an important component of understanding the complex etiology of smoking among youth. As noted earlier in this chapter, one can expect findings on the effects of friends' tobacco use to be inflated when studies do not account for selective affiliation (i.e., the tendency for adolescents to choose friends who are similar to themselves) or for adolescents' perceptions of their friends' tobacco use, which may or may not reflect actual use. Moreover, because the effects of friends' tobacco use may be stronger for females than for males and for White than for minority youth, estimates of friends' tobacco use may be misleading if these specific effects are not considered. Indeed, assessing the causal role of friends' smoking is incomplete without these and other considerations, such as adolescents' relationships with peers.

To conclude that there is a causal linkage between parental smoking and smoking among youth, more longitudinal research is needed, perhaps focusing on varying trajectories of smoking over time in parents and their offspring, since the data to date have not been consistent or conclusive (Chassin et al. 2008). And yet, because some studies have shown that parental variables may indirectly affect adolescents' choices of friends or their thoughts about smoking, parental smoking and other family effects may be both directly and indirectly important, again suggesting the need for more sophisticated research in this area.

This review did not find sufficient evidence to implicate parental factors as being causal agents in the use of tobacco among young adults, but the evidence is suggestive of a potential causal role for parental smoking and a causal role for peer group influences.

Cognitive and Affective Processes

Mood and Affect

Affective processes appear to play an important role in the uptake, progression, and persistence of adolescent smoking. Numerous investigators have examined the role of negative affective states and affect regulation in the initiation and development of cigarette smoking behavior. In cross-sectional studies, regular and experimental smoking among youth is associated with higher levels of negative affect compared with nonsmoking peers (Mitic et al. 1985; Coogan et al. 1998; Escobedo et al. 1998; McKenzie et al. 2010). Longitudinal studies in this area demonstrate that higher levels of negative affect are not only characteristic of adolescent tobacco users but also are likely related to smoking initiation and transitions along a trajectory of use. Patton and colleagues (1998) prospectively examined the association between depression, anxiety, and smoking initiation among youth and determined that depression and anxiety predicted initiation of experimental smoking. This association was mediated by the presence or absence of smoking peers. Wills and associates (2002) showed that high levels of negative affect and life stress in a sample of adolescents predicted increases in tobacco use over a 3-year period. Siqueira and colleagues (2000) found that when teenage smokers were directly asked about the reasons for their progression from experimental to regular tobacco use, stress was identified as a primary catalyst, with endorsement by 72% of the sample. Audrain-McGovern and colleagues (2009) followed a large cohort of students (n = 1,093) from 9th grade until 12th grade to

examine the temporal relationship between smoking and depression. Students were assessed annually for smoking, depression, smoking among their peers, and other potential covariates. The authors found that increased depression symptoms predicted elevated smoking levels and progression in smoking. Interestingly, greater smoking at baseline predicted a deceleration in the number of smoking peers across time, which predicted a deceleration in depression symptoms. The comorbidity of depression and smoking can possibly be explained through peer influences, since the number of smoking peers mediated the relationships between smoking and depression.

Mood benefits derived from smoking may be an important driver of smoking behavior among youth. Like adults, many youth report smoking for reasons related to affect regulation (e.g., tension reduction, negative affect relief) (Scales et al. 2009). A growing body of evidence suggests that cigarette smoking can produce immediate, reinforcing changes in both positive and negative moods among adolescents (Kassel et al. 2003, 2007; Hedeker et al. 2008, 2009). Kassel and colleagues (2007) used a matched case-comparison study design of 15- to 18-year-old smokers (n = 45) and nonsmokers (n = 27) to determine the effects that nicotine has on both positive and negative affect. Smokers in this study experienced reductions in both their positive and negative affect scores after smoking a cigarette; these reductions were moderated by nicotine dependence, the nicotine content of the cigarette (high yield vs. denicotinized), and cigarette craving. In addition, smoking expectancy moderated negative but not positive affect. Nonsmokers had no reduction in either positive or negative affect over a 10-minute interval (Kassel et al. 2007). Importantly, adolescents who expect to receive greater mood benefits from smoking experience it as more reinforcing, compared with those without strong mood-related expectancies. Colvin and Mermelstein (2010) sought to determine whether expectancies of negative affect influenced mood expectancies directly after smoking. Using handheld computers for a week to assess changes in mood throughout the day, the participating adolescents (n = 461) were given surveys to measure smoking expectancies, nicotine dependence, number of biological parents who were ever smokers, and current smoking behaviors (Colvin and Mermelstein 2010). Increased amounts of negative-affect expectancies were related to a greater decrease in negative mood and an increase in positive mood immediately following smoking.

Evidence is also accumulating to suggest that adolescents who experience greater subjective mood benefits of smoking are more likely to progress in their smoking. In a prospective study of adolescent smokers, Mermelstein and colleagues (2007) found that subjective mood benefits of smoking predicted escalation in a cohort of adolescents. Adolescents who progressed in their smoking were those who reported substantial in-the-moment mood benefits following smoking; adolescents who tried smoking but stopped did not report any subjective mood benefits following smoking. Further evidence that mood-stabilizing effects may reinforce and maintain smoking among youth comes from Weinstein and colleagues (2008), who examined variability in negative moods as it related to smoking patterns among adolescents. Students in 8th and 10th grades (n = 517) were assessed at baseline, 6 months, and 12 months on cigarette use; for 1 week, students used palmtop computers to provide momentary assessments of negative moods. Increased variability in negative mood at baseline was significantly associated with subsequent escalation of smoking compared with students who did not progress beyond experimentation.

Smoking-related expectancies are associated with many aspects of smoking motivation and behavior (Brandon and et al. 1999). Studies of adolescent smokers have demonstrated a strong relation between positive expectancies for smoking (e.g., relaxation, mood enhancement) and smoking status, such that more experienced smokers appear to have more positive expectancies for smoking (Gordon 1986; Covington and Omelich 1988). Heinz and colleagues (2010) followed a group of 568 adolescents for 2 years at four time points to determine the influence that negative affect relief expectancies (NAREs) have on smoking behavior and nicotine dependence; both were measured at the four assessments. When controlled for anxiety and depression symptoms, NAREs predicted both the progression of smoking and nicotine dependence (Heinz et al. 2010). The NAREs were measured as a subscale of 10 items (e.g., "smoking helps calm me down when I'm nervous"); responses were recorded on a 4-point scale ranging from 1 = disagree to 4 = agree. Taken together, the evidence suggests that adolescents who hold more favorable positive expectancies for smoking are more likely to begin smoking and to smoke more cigarettes.

In addition to the substantial body of evidence implicating negative affect in the etiology and progression of youth smoking, a number of studies have shown that smoking during adolescence may increase the risk for subsequent development of mood disorders. For example, Jamal and colleagues (2011) examined the relationship between age at smoking initiation and subsequent onset of mood disorders in a sample of 1,055 current and former smokers. Only smokers who were nondepressed or nonanxious when they started smoking were included in the study. Relative to late-onset smokers, early-onset smokers experienced onset of depression and/or anxiety disorders 5 years earlier, suggesting that a young age at

smoking onset increases vulnerability for the subsequent development of psychopathology. Furthermore, a growing body of evidence provides support for a bidirectional relationship between smoking and negative affect. Windle and Windle (2001) used a large four-wave panel design to examine the temporal relationship between depressive symptoms and cigarette smoking in a large sample of 10th and 11th graders (n = 1,218). Students completed surveys about their depressive symptoms and smoking behavior at baseline and every 6 months thereafter, for a total of 1.5 years. Symptoms of depression predicted increases in cigarette smoking over time. Over the same study period, heavy and persistent smoking prospectively predicted increases in depressive symptoms. Similarly, Orlando and colleagues (2001) tested the hypothesis that smoking was dynamically related to emotional distress in a cohort of 2,961 adolescents. The authors examined concomitant changes in smoking behavior and emotional distress over time and found that baseline emotional distress in grade 10 predicted increased smoking in grade 12; this increase in smoking was, in turn, associated with increased emotional distress in young adulthood.

The observed bidirectional influences described above support the plausibility of shared etiologies between negative affect and smoking behavior. However, it is also possible that unique causal mechanisms are operating in each direction. For example, self-medication of depressed mood could be influencing smoking progression, whereas the effects of nicotine on neurotransmitter systems linked to depression could be driving the association with negative affect. More research is needed to explain these mechanisms.

Cognitive Processes

Two kinds of cognitive processes play roles in the development of regular smoking among youth: (1) those that are conscious, explicit, and planned and (2) those that are unconscious, implicit, and relatively automatic. These processes can act independently or interact as dual-process models; a fuller discussion follows below.

Explicit or Controlled Cognitive Factors and the Deliberate Processing of Information

The role of cognition in tobacco use can be understood more fully by examining social learning theory and cognitive-behavioral principles of learning (Brandon et al. 2004). Investigations into the etiology of tobacco use have studied three key cognitive constructs: expectancy (Goldman et al. 1999), self-efficacy (Bandura 1977a), and coping (Wills and Filer 1996). Expectancy refers to the perceived outcomes of tobacco use, and positive outcome expectancy is related to the theory of positive reinforcement of addiction. Self-efficacy is related to an individual's confidence in achieving goals through personal efforts, such as the ability to resist smoking or to remain smoke-free after quitting. Coping theories view tobacco use as a mechanism to deal with stress and other negative states; such theories include the self-medication and performance-enhancement models.

The Smoking Consequences Questionnaire (Brandon and Baker 1991), a well-known instrument for measuring expectancies, has been adapted for use among adolescents and young adults (Myers et al. 2003; Lewis-Esquerre et al. 2005; Wahl et al. 2005). This instrument measures several positive outcome expectancies about smoking, including:

- pleasant taste
- relief from boredom
- reduction in negative affect
- weight regulation
- positive social consequences
- favorable outcomes related to the health hazards of smoking

Various studies have associated these outcomes with increased intention of smoking, initiation of smoking, escalation in smoking behavior, regular smoking, and/or current smoking (Flay et al. 1998; Ausems et al. 2003; Myers et al. 2003; Lewis-Esquerre et al. 2005; Wahl et al. 2005).

Two studies linked low self-efficacy early in adolescence with smoking behavior later during the adolescent period. In one, Flay and colleagues (1998) associated low self-efficacy in skills for refusal of cigarettes from peers in 7th grade with smoking experimentation (versus never smoking) in 12th grade. Later, Ausems and colleagues (2003) found that low self-efficacy in refusal skills among 11- and 12-year-olds led to a higher likelihood of experimentation with smoking (compared with never smoking) and regular smoking (compared with experimentation).

In a multivariate analysis, Lewis-Esquerre and associates (2005) found that perceptions that both the sensory and motor aspects of smoking were pleasant constituted a significant risk factor for smoking in 7th- to 12th-grade youth and that a belief in the negative social consequences of smoking was strongly protective for this group. Among students in the 2nd through 5th grades, Hampson and

colleagues (2007) found an association between a more positive social image of cigarette smoking (i.e., youth who smoke are "liked by other youth," are "exciting," and are "cool or neat") with increased intentions to smoke. Using the same sample, an analysis by Andrews and colleagues (2008) found that a positive social image of smoking was related to willingness to smoke that, in turn, predicted smoking rather than simply intention to smoke.

Among sixth- and seventh-grade urban youth, poor decision-making and lower self-efficacy were found to be related to perceived social benefits of smoking in the seventh and eighth grades, which, in turn, were positively associated with smoking 1 year later (Epstein et al. 2000). However, there was no direct relationship between decision-making skills or self-efficacy in the sixth and seventh grades and smoking 2 years later (Epstein et al. 2000). These results suggest an important role for the perceived social aspects of smoking in mediating whether smoking will be taken up.

Belief in the negative health consequences of smoking was found to be a robust protective factor against the risk that youth would smoke (Rodriguez et al. 2007). Even in 16-year-old tobacco users, Myers and coworkers (2003) found that belief in the negative health consequences of smoking was associated with lower smoking frequency, lower scores on tobacco dependence, and more quit attempts.

Velicer and colleagues (2007) identified four clusters of ninth graders on the basis of their beliefs about the negative and positive consequences of smoking and their self-efficacy for resisting the temptations of smoking. At 3-year follow-up, the cluster with the most-negative and least-positive beliefs, which also demonstrated low levels of smoking temptations, had the lowest prevalence of smoking initiation (13.2%). A second cluster, characterized by high levels of smoking temptation, and a third cluster, characterized by the least-negative outlook on the consequences of smoking, had the highest proportions of smoking initiators at follow-up (26.5% and 28.7%, respectively). The same three constructs—low self-efficacy for resistance, belief in the positive consequences of cigarette smoking, and lack of belief in the negative consequences of that behavior—have been associated with onset of smoking or rapid escalation to regular smoking following experimentation (Chassin et al. 2000; Orlando et al. 2004; Bernat et al. 2008).

Executive function, which involves such tasks as reasoning, processing speed, and the ability to inhibit a reflexive response, is another explicit cognitive factor that may affect adolescent smoking. Fried and colleagues (2006) found that slower processing speed and worse performance on tasks requiring sustained attention and abstract reasoning at 9–12 years of age were associated with smoking (≥9 cigarettes per day) at 17–21 years of age. In this study, however, performance on these tasks did not distinguish between eventual groups of lighter smokers (1–8 cigarettes per day), former smokers, and those who never became regular smokers. In addition, performance on vocabulary, memory, and tasks requiring spatial ability did not distinguish between any of the smoking groups. Elsewhere, from cross-sectional data describing 14-year-olds, Lawlor and colleagues (2005) found a higher prevalence of smoking at lower levels of nonverbal reasoning and reading abilities. These two studies suggest that specific deficits in executive function may be related to an increased risk of smoking, but neither study focused explicitly on the relationship between performance on cognitive tasks and smoking, and so adjustments for covariates could not be made in the comparisons cited.

Automatic/Implicit Cognitive Processes

Research in social cognition indicates that the acquisition of automatic behaviors (i.e., behaviors that are not consciously mediated) develops through frequent and consistent experiences with a particular social behavior that, in turn, affects the likelihood of engaging in that behavior. Conscious choice drops out as it becomes a superfluous step in the process (Bargh and Chartrand 1999).

Currently, there is a research focus on the evaluation of implicit or spontaneously activated cognitions on behaviors, such as regular tobacco use and other behaviors involving addictions (for a review, see Wiers and Stacy 2006). Implicit cognitions result from information processing of associations involving tobacco-related outcomes, such as feeling good because of dopamine-dependent associations, tobacco-relevant stimuli (e.g., cigarette advertising, cigarette packages, lighters, ashtrays), or tobacco-related situations or environmental contexts (e.g., smoking with friends at a party). These types of associations are strengthened in memory through repetitive experiences (Stacy 1995, 1997) and come to influence or guide behavior through a relatively spontaneous process that circumvents rational decision-making (Stacy 1997; Wiers and Stacy 2006). Implicit cognitive processes can influence thought processes and the interpretations of situations, contexts, and other stimuli, and they can also either make more accessible or inhibit the memory of behavioral alternatives (e.g., healthy behavioral options).

Numerous studies have evaluated the influence of implicit cognitive processes on smoking behavior among

adolescents. Although a variety of cross-sectional studies on implicit associations have had robust outcomes, only a few prospective studies have evaluated the influence of such associations on subsequent smoking while controlling for potential confounders (Stacy 1997; Kelly et al. 2005, 2008; Thush et al. 2007). In a prospective study, Kelly and colleagues (2008) used the Memory Association Test (Kelly et al. 2005) to evaluate the effects of implicit tobacco-related memory associations on smoking in adolescents; this test is a variation of an indirect cue-association paradigm (Stacy et al. 1994, 1996) that contains no explicit reference to the behavior being assessed. Among high school youth, the study found that tobacco-related memory associations assessed at baseline were predictive of smoking 6 months later when they were controlled for within-subject variability in smoking and other variables. These findings suggest that youth with strong memory associations related to tobacco use may be at increased risk for subsequent smoking.

In an extensive review of the literature involving the influence of nonconsciously mediated processes on smoking dependence and cessation, Waters and Sayette (2006) found across a range of cross-sectional studies that smoking status among young adults (college students) was frequently associated with indirect tests of association, such as the Implicit Association Test (IAT).

The IAT is a categorization task that provides a method of indirectly assessing the relative strength of memory associations among different concepts (Greenwald et al. 1998). The basic assumption is that past learning (e.g., experience with smoking) is represented by facilitated information processing of associated concepts as measured by reaction time on the task. During the task, participants sort stimuli into two categories of attributes (e.g., positive vs. negative and approach vs. avoid) and two target categories (e.g., tobacco-related objects and non-tobacco-related objects). Faster responses to observed paired stimuli (e.g., a cigarette and feels bad) are interpreted to mean that the two stimuli are more strongly associated in memory than are other pairs of stimuli. Stronger implicit associations between a behavior and a variety of cues or outcomes (e.g., smoking a cigarette and being social or feeling good) are potentially significant in promoting the behavior (for reviews, see Ames et al. 2006; Waters and Sayette 2006; Wiers et al. 2006).

Several studies of adults who completed the IAT indicate that both smokers and nonsmokers have some negative implicit attitudes toward smoking when asked to categorize smoking and nonsmoking stimuli with positive and negative attributes, but smokers have relatively fewer negative attitudes toward smoking than do nonsmokers (Swanson et al. 2001; Sherman et al. 2003; Huijding et al. 2005; DeHouwer et al. 2006). In addition, DeHouwer and colleagues (2006) found that smokers reacted faster when categorizing smoking stimuli with an "I like" label, and nonsmokers reacted faster when categorizing smoking stimuli with an "I dislike" label. Such results, however, may be more reflective of individual differences in implicit attitudes and less sensitive to societal influences or attitudes toward smoking (Olson and Fazio 2004). More research with the IAT is needed to support previous findings. With an approach-avoid IAT, DeHouwer and colleagues (2006) further found that smokers associated smoking with more "approach words" than "avoid words" and that nonsmokers associated smoking with more "avoid words" than "approach words."

Sherman and colleagues (2003) found that heavier smokers had significantly more positive implicit associations toward smoking than did lighter smokers and less negative implicit associations toward that activity than did nonsmokers. Perugini (2005) reported similar findings when comparing smokers with nonsmokers on an IAT; that is, smokers' implicit attitudes toward smoking were significantly more positive than those of nonsmokers, and their explicit attitudes, also measured, were significantly more positive as well. McCarthy and Thompsen (2006) reported similar findings with a tobacco-related IAT: they found correlations between positive implicit associations and self-reported smoking behavior but no significant relationship between negative implicit associations and smoking behavior. Using a single-target IAT, Huijding and de Jong (2006) found that smokers had positive implicit affective associations toward smoking, but nonsmokers had negative (implicit) affective associations. In addition, self-reported craving correlated with negative implicit affective associations but not with self-reported attitudes. In a subsequent IAT study among smokers only, Waters and colleagues (2007) found that implicit attitudes toward smoking were robustly and positively related to self-reported craving and nicotine dependence.

Chassin and colleagues (2002) included an IAT in a study with both implicit and explicit attitudes when they evaluated the influence of parental smoking/cessation on adolescent smoking. These authors found that mothers with positive implicit attitudes toward smoking were more likely to have children who smoked. In addition, the IAT differentiated between smoking and nonsmoking/formerly smoking mothers, with mothers who smoked having more positive implicit attitudes toward the behavior. However, implicit attitudes toward smoking among youth in the study failed to correlate with parental smoking, and implicit attitudes of both fathers and youth did not corre-

late with the youth's smoking behavior, even though there was a correlation between mothers' attitudes and smoking by their children. Although more studies are needed on the influence on youth of the implicit attitudes of parents toward smoking, findings suggest that in conjunction with prevention efforts among youth, programs might want to target parental (particularly maternal) implicit attitudes toward smoking to help efforts to prevent this behavior.

The Go/No-go Association Test (GNAT), an indirect test of association developed by Nosek and Banaji (2001), assesses implicit associations with a single target category, thus eliminating competing or contrasting categories as in the IAT. Using a portable version of the GNAT, Bassett and Dabbs (2005) differentiated implicit attitudes of smokers from nonsmokers among 39 adults in a university environment. Smokers reacted faster to "smoking words" that were paired with "good words," and nonsmokers responded faster to "smoking words" that were paired with "bad words."

In summary, findings from IAT studies and others that have focused on implicit cognitive processes appear to reflect some differences between smokers and nonsmokers in tobacco-related implicit associations. The differences found may suggest differences in neurobiology, early-life experiences, or exposure to tobacco use. Refinement of methodologies may help further elucidate the influence of implicit associations on smoking behavior among youth. These contributions will be important to the literature and future studies of nonconsciously mediated influences on behavior (Waters and Sayette 2006; Wiers and de Jong 2006).

Dual-Process Models

Dual-process models of behavior acknowledge that goal-directed behaviors, such as tobacco use, are influenced by a range of cognitive processes, including both implicit or automatic processes and more controlled, deliberate, or executive processes (Tiffany 1990; Stanovich and West 2000; Evans 2003; Kahneman 2003; for dual-process approaches to addiction, see Wiers and Stacy 2006). In general, researchers on addiction have accepted this dual-process approach to cognition and have acknowledged the influence of both implicit and explicit processes in the development and maintenance of addictive behaviors (Bechara and Damasio 2002; Wiers and Stacy 2006). Furthermore, many studies have shown the additive and independent predictive ability of implicit and explicit processes in usage models for tobacco and other drugs (Stacy 1995, 1997; Chassin et al. 2002; Wiers et al. 2002; Sherman et al. 2003; Huijding et al. 2005; Perugini 2005; Ames et al. 2007; Thush et al. 2008). In one study, Grenard and associates (2008) found possible tobacco-related associations to be stronger predictors of smoking among youth with lower-capacity working memories than among those with a higher capacity. Ongoing research about dual-process models of addiction will help to elucidate the influence of explicit and implicit processes on goal-directed behaviors as well as explain how certain cognitive functions may inhibit behavioral tendencies that arise from more spontaneously activated implicit associations.

Summary

A robust association between youth smoking and negative affect has been demonstrated in the literature. Prospective studies suggest that this association may be bidirectional. Negative affect has been shown to be an influential factor for the onset and continuation of youth smoking. At the same time, smoking during adolescence has been found to prospectively predict subsequent negative affect and depressive symptoms. It can be concluded that smoking and mood are related to one another, but more research is needed to understand the temporal relationship. A key question regarding the association between negative affect and youth smoking is whether it reflects a direct causal influence, in one or both directions.

The cognitive processes that influence the initiation of tobacco use, continued use, and dependence include executive, or more explicit, processes and implicit processes (those that are more automatically associative). Executive processes are relevant to inhibitory control over behaviors and to counteracting the influence of more spontaneous (or implicit) cognitive processes. Evidence suggests that executive processes moderate behavior (e.g., the capacity of working memory) during decision making in complex situations (Finn and Hall 2004; Payne 2005; Grenard et al. 2008). For example, complex social situations involving cues to use tobacco or ambiguous contexts are likely to tax aspects of executive functioning for many youth, reducing their ability to inhibit intentions to resist smoking. For most youth, tobacco use is unlikely to be motivated solely by rational decision-making processes. The influence of implicit cognitive processes on behavior has been demonstrated in numerous studies across a variety of drugs and populations (for reviews, see Ames et al. 2006 for drugs; Waters and Sayette 2006 for tobacco; and Wiers et al. 2006 for alcohol). Implicit associations, or more spontaneously activated cognitions, may help to explain why some people engage in apparently irrational behaviors, such as smoking, while clearly knowing that the behavior can have negative consequences.

Both automatic and controlled cognitive processes (incorporated in dual-process models) influence behavior and, therefore, both should be considered potential targets of interventions (Wiers and Stacy 2006). However, more research is needed to evaluate the ability of dual-process models to predict the use of tobacco and other substances, the interaction between the two processes, and individual variations in these processes. Future research should focus on increasing the understanding of the role of cognitive mediators in complex social behaviors, such as the use of tobacco and other drugs, and the decision making behind engaging or not engaging in the particular behavior.

Genetic Factors and Neurobiological and Neurodevelopmental Processes

This section considers the role of genetic factors and their interaction with measured environmental factors; neurobiological processes, including addiction to nicotine; and neurodevelopmental processes (USDHHS 2010). The term "genetics" refers to a person's biological coding scheme, which may become a phenotype (expression) that at times depends on context and previous experience and exposures. The term "neurodevelopmental processes" refers to the influences of environmental experiences and maturation processes on cognitive function and, in this case, the likelihood that a person may yield to perceived social influences or curiosity and use tobacco products. Neurobiological processes are neurologic transmissions across brain structures that may predispose a person to seek out the use of tobacco or other drugs or that may be affected by tobacco or other drug use. Importantly, adolescence is a time of considerable neurodevelopmental plasticity and change (Steinberg 2007; Windle et al. 2008; Giedd and Rapoport 2010). Brain development in regions associated with impulsivity, motivation, and addiction continues well into young adulthood (Lebel and Beaulieu 2011). Maturational changes that occur during adolescence may contribute to neurologic factors that underlie vulnerability to addiction, such as increases in novelty seeking and impulsivity (Chambers et al. 2003). Some individual traits such as sensation-seeking and temperament might predispose young people toward certain problem behaviors in particular social contexts (Wills et al. 2000; Bisol et al. 2010).

Genetic Influence on Smoking Behaviors

Genetic influences have been documented at each stage in the continuum of smoking, from initiation to dependence, in twin and family studies. This broad topic was covered in depth in the 2010 Surgeon General's report on how tobacco smoke causes disease, with the conclusion that inherited genetic variation contributes to differing patterns of smoking behavior and cessation (USDHHS 2010). Some of the supporting evidence is also summarized in Chapter 2 of this report and in the present section.

This evidence in support of the heritability of smoking behavior has prompted researchers to identify specific genes and biological mechanisms that play a role in smoking behavior and nicotine dependence using a variety of genetic study designs.

Until recently, research has focused many of the genetic efforts on candidate gene and linkage studies rather than on more powerful genomewide association studies or sequencing. Some of these candidate gene studies have been fruitful, mainly because the genetics of addiction benefits from a vast knowledge of a given drug's mechanism of action; consequently, many genes are plausible candidates, and some associations have been reported. However, the more recent high-throughput approaches have provided consistent and compelling results that have advanced the science base on genetics and smoking behavior. Although incomplete, the overview below provides a picture of the approaches and findings to date.

Genetic Linkage Analyses

Genetic linkage analyses seek to identify genetic variants associated with an outcome of interest by testing genetic markers across the genome. Regions of the genome that appear strongly linked to the outcome have a higher likelihood of containing influential genetic regions or genes. Several large family-based genetic linkage analyses have been conducted to identify the chromosomal regions associated with different smoking outcomes,

including smoking status, tobacco dependence, and even cigarettes smoked per day. The results of these studies have been somewhat inconsistent, however, pointing to different regions on a number of chromosomes (Munafó and Johnstone 2008; Uhl et al. 2008). Even so, the implicated regions likely contain susceptibility loci and several candidate genes whose genetic variation may explain differences in phenotypes. For example, a region on chromosome 9q22 has been linked to tobacco dependence (Li et al. 2003), a finding corroborated by three other independent studies (Bergen et al. 1999; Bierut et al. 2004; Gelertner et al. 2004). In addition, a location on chromosome 5q (D5S1354) has been strongly linked to smoking behavior in at least two studies (Bergen et al. 1999; Duggirala et al. 1999). Research has also found associations between the gamma amino butyric acid receptor subunit B2 (*GABA-B2*) and neurotrophic tyrosine kinase receptor type 2 (*NTRK2*) genes and tobacco dependence (Beuten et al. 2005, 2007). Unfortunately, only a few studies go on to identify the specific genetic variants located on the chromosomes implicated in genetic linkage analyses (Munafó and Johnstone 2008).

Candidate Gene Studies

Candidate gene studies, on the other hand, compare the prevalence of specific genetic variants by using a case-control design. The variants are selected on the basis of evidence from earlier studies that are related to the outcome of interest. Most candidate gene studies on tobacco use have evaluated the influence of genes that operate in neurotransmitter pathways (e.g., the dopamine and serotonin pathways), nicotine metabolism, and nicotinic receptors (Munafó and Johnstone 2008). The majority have focused on genes involved in the dopamine pathway, particularly the dopamine receptor D2 (*DRD2*) gene. The *DRD2 Taq1A* polymorphism has been implicated in the majority of studies, while others have found no such association (Munafó et al. 2004). Two meta-analyses have shown that the *Taq1* A1 allele is significantly more likely to be found among smokers than among nonsmokers. Other genes in the dopaminergic reward system have been investigated in the context of tobacco use and dependence, such as the dopamine transporter (*DAT*), other dopamine receptors (*DRD1*, *DRD4*, *DRD5*), catechol-*O*-methyltransferase (*COMT*), monoamine oxidases A and B, and tyrosine hydroxylase (*TH*), although none of these variants has shown a strong relationship with smoking behaviors (Munafó and Johnstone 2008). In the serotonin pathway, most studies investigating the *5-HTTLPR5* polymorphism within the *SLC6A4* gene, including one meta-analysis, found a relationship with smoking behavior (Munafó et al. 2004).

Candidate gene studies have also looked at genes in the nicotine metabolism pathway; variants in these genes might be expected to cause individual differences in susceptibility to different doses of nicotine. The most commonly studied gene in this category is the *CYP2A6* gene; there is evidence to suggest that *CYP2A6* variants that reduce nicotine metabolism are associated with reduced smoking quantity (Malaiyandi et al. 2006) and increased likelihood of cessation (Munafó et al. 2004).

Several nicotinic receptor genes have been examined, with some studies finding that *CHRNA4* plays a role in tobacco dependence. In addition, a large case-control study found several other nicotinic receptor genes to be associated with tobacco dependence, including *CHRNA5* and *CHRNB3* (Saccone et al. 2009). Replication of these findings is a necessary step toward validating the roles of these genetic variants (see below). The region on chromosome 15 that includes a group of nicotinic receptor genes has been associated in multiple populations with the quantity smoked and the risk of becoming nicotine dependent (Bierut 2010), thereby demonstrating the importance of this region.

Findings in both genetic linkage analyses and candidate gene studies demonstrate great heterogeneity, indicating that genetic influence on tobacco use and nicotine dependence is complex and likely involves multiple genes.

Genomewide Association Studies and Sequencing

Over the last decade, the science of genetics has made important progress through conceptual insights and technological breakthroughs. In 1999, the idea of evaluating hundreds to thousands of genetic variants—namely, single nucleotide polymorphisms (SNPs)—at once was beginning to take shape. One key turning point was the understanding that the genome is built by many sets of correlated SNPs, called haplotypes, which meant that rather than screening the entire SNP collection, a subset of "proxy" SNPs, or TagSNPs, could be screened without loss of information, using the so-called genomewide association study (GWAS) design. GWAS is powerful for honing in on relevant areas of the genome related to the phenotype in an unbiased way. Once a "hit" is discovered through a GWAS scan, it needs to be replicated and evaluated further to determine if it contributes to the phenotypic outcome. Therefore, GWAS provides the first step in identifying key regions for deep sequencing and functional characterization. This approach resulted in one of the most replicated findings in addiction genetics—the A5/A3/B4 nicotinic cholinergic receptor subunit cluster on chromosome 15 associated with tobacco dependence across populations (Saccone et al. 2009, 2010; Liu et al.

2010; The Tobacco and Genetics Consortium 2010; Thorgeirsson et al. 2010).

Science and genetics technologies continue to evolve at a rapid pace, and it is now possible to conduct whole-genome sequencing (complete sequence of an individual's genome) and "deep" sequencing (sequencing specific regions of a genome) of targeted regions in many people. In either approach, sequencing allows for a single-base examination of the genetic architecture within the target region, and it also allows for a higher order view of the genomic structure (e.g., copy number variation, structural variations such as deletions, insertions, inversions, and epigenetic targets).

The whole-genome sequencing and deep sequencing approaches are starting to be used to uncover additional rare genetic variants that also contribute to smoking-related phenotypes (Wessel et al. 2010). The GWAS evidence and subsequent replications showing association with tobacco dependence phenotypes with the nicotinic subunit receptor cluster on chromosome 15 (CHRNA5/A3/B4) supports the next steps of deep sequencing and functional analyses to understand the relationships and mechanisms of how those genetic variants contribute to the smoking phenotype; this work is ongoing and shows that the genetic changes in this gene cluster have effects on receptor function (Wang et al. 2009, Hong et al. 2010; Fowler et al. 2011; Smith et al. 2011).

Genetic Factors in Tobacco Use Among Youth

Studies of Twins

From data obtained from pairs of twins reared together, latent genetic and environmental contributions to phenotypic variation can be estimated. Twin models compare the correlations of twin pairs across zygosity groups. If the resemblance of twin pairs is determined by additive genetic effects transmitted from parents to their offspring, then the correlation in monozygotic (MZ) twin pairs is predicted to be twice that of dizygotic (DZ) twin pairs because MZ twins share 100% of their genes, while DZ twins share, on average, 50% of their genes and are no more similar than are any other pair of full siblings. If the resemblance of twin pairs is determined by shared environmental influences, or factors common to family members, such as home or school environment, then equal MZ and DZ correlations are expected, because both MZ and DZ twin pairs are assumed to share 100% of the shared environmental factors. If the MZ correlation is greater than, but less than twice, the DZ correlation, then both genetic and shared environmental influences contribute to phenotypic variation. Residual variation not accounted for by genetic or shared environmental factors is termed a nonshared or individual-specific environmental variance. Residual variation contributes to the dissimilarity of twin pairs and includes measurement error.

In 12- to 19-year-old adolescents, heritability for initiation of smoking (defined as having ever smoked) has been estimated to be between 36% and 56% across different samples, and the effect of shared environmental factors on initiation has been estimated to be between 30% and 44% (Han et al. 1999; McGue et al. 2000; Rhee et al. 2003). Estimates of heritability for regular cigarette smoking (defined by the frequency of smoking in the past month) range from 27% to 52%, and the range of estimates for the effect of shared environmental factors (7–43%) is wider than that for the initiation of smoking (Rende et al. 2005; Slomkowski et al. 2005; Young et al. 2006). Similar estimates have been found for dependent smoking (i.e., smoking in which the smoker is dependent on nicotine) in adolescents (heritability, 44–49%; shared environmental factors, 15–37%) (McGue et al. 2000; Young et al. 2006). Slomkowski and colleagues (2005), who looked at regular smoking, reported the lowest heritability (23%) and the highest estimate for shared environmental factors (43%) in 15-year-olds, with a shift in the relative values at 1-year follow-up (43% for heritability and 34% for shared environmental factors). In a study of 13- to 16-year-olds, questions about cigarette and other tobacco products were combined into one item of "ever consuming more than 1 cigarette or other tobacco products per day" (Maes et al. 1999, p. 295); this definition of "ever use" produced an estimate for heritability of 65% (Maes et al. 1999) and may capture daily smoking at a later stage of smoking than initiation. Overall, these results support the idea that the relative contribution of genetic influences increases from earlier (initiation) to later (regular/daily or dependent smoking) stages of tobacco use. The results also suggest that the same behavioral measure (frequency of smoking in the past month) may index different types of risk at different ages.

In a sample of 12- to 24-year-olds, heritability for smoking initiation was 39%, and the estimate for shared environmental factors was 53% (Boomsma et al. 1994; Koopmans et al. 1999). Across age groups, however, significant differences in the relative contribution of genetic and environmental factors to the initiation of smoking were not found (Boomsma et al. 1994). A substantial genetic influence was found on quantity smoked in two studies: 86% in a study by Koopmans and colleagues (1999) and 52% in a sample of young adults (aged 18–24 years) in a study by Haberstick and colleagues (2007). Neither study contained evidence for a significant influence of shared environmental factors on the quantity smoked.

Traditionally, the number of cigarettes smoked per day has been used as an indicator of tobacco dependence, the final stage of tobacco use. Latency (time) to first cigarette after waking, another indicator of dependence, was significantly heritable in young adults (55%) in the study by Haberstick and coworkers, with no significant shared environmental factors for this marker (Haberstick et al. 2007). The Heaviness of Smoking Index (HSI), which combines scores on quantity and latency measures, was strongly heritable (61%) in the Haberstick study, with no significant shared environmental factors (Haberstick et al. 2007). In contrast, the Fagerström Test for Nicotine Dependence (FTND) score, which is comprised of the two HSI items and four other items, was modestly heritable (17%) in that study, with a relatively large contribution from shared environmental factors (25%). This is not surprising in that the four additional FTND items showed no evidence for genetic influences (Haberstick et al. 2007).

Taken together, these studies suggest that the prominent role played by shared environmental factors at earlier stages of cigarette smoking, such as initiation, disappears at later stages of regular or dependent use, when genetic influences predominate. For example, Kendler and colleagues (2008) specifically examined the interplay of genetic and environmental factors over time through the use of retrospective life history data (from calendars) among 13- to 35-year-olds and found that genetic influences for number of cigarettes smoked per day first appeared around 16 years of age (about 10% heritability) and increased to about 60% by 35 years of age. In contrast, the contribution of shared environmental factors decreased from about 50% at 13–17 years of age to 0% by 35 years of age.

Studies in adults have shown that the age of smoking initiation is significantly heritable (Heath et al. 1999; Broms et al. 2006), but genetic influence on the age of initiation is independent of the genetic influence on such variables as the quantity smoked and quitting smoking (Broms et al. 2006). Furthermore, Pergadia and colleagues (2006) found similar genetic and environmental influences for regular smoking and dependence measures in adult twin pairs who first tried smoking cigarettes on the same occasion in a comparison with pairs who first tried smoking at different times or ages. These results suggest that varying ages for initiation do not appear to bias genetic and environmental estimates on later stages of smoking, perhaps because initiation and later-stage smoking may not share common genes (Broms et al. 2006).

Schmitt and associates (2005) examined the contribution of genetic and environmental factors to the use of tobacco products other than cigarettes among 20- to 58-year-olds. The relative contributions of genetic and shared environmental factors were, respectively, 43% and 28% for regular use of dip (moist snuff), 19% and 21% for use of chewing tobacco, 0% and 32% for pipe use, and 0% and 26% for cigar use. These results suggest substantial variation in the genetic contribution to regular use of different forms of tobacco.

Interaction Effects Between Genetic and Environmental Factors

The previous section summarized the evidence that genetics plays an important role in smoking behavior, particularly at later stages of smoking. Although genetic risk for cigarette smoking may be a vulnerability with which persons are born, it is not a static and obligatory influence on smoking behavior (for review, see Lynskey et al. 2010). In fact, the expression of genetic risk depends on certain environmental circumstances. For example, smoking by one's peers is a robust predictor of current smoking, regular smoking, and the transition to regular smoking and has a strong influence in adolescence, but it is also significant in adulthood even after controlling for genetic risk for smoking (Vink et al. 2003a,b). Thus, smoking by peers may inhibit the expression of genetic influences on smoking behavior. In a study by White and colleagues (2003), a heritability estimate of 15% for regular (past week) smoking by 13- to 18-year-olds was reduced to 0% after accounting for peer smoking. Two waves of follow-up assessments, about 3 years apart, showed a progressive increase in the heritability estimate for regular smoking to 20% at the second wave (sample aged 16–21 years) and 35% at the third wave (sample aged 20–25 years). In contrast, smoking by peers showed a decreasing influence across waves, explaining 37% of the variance in heritability at the second wave and 12% at the third wave. In another study, Harden and colleagues (2008) found that genetic risk for tobacco and alcohol use in adolescents correlated with best-friend's substance use, a case of gene-environment correlation, and that adolescents at high genetic risk for tobacco and alcohol use also appeared to be more sensitive to adverse peer influences, a case of gene-environment interaction.

Aside from peer influences, parental behavior may affect the expression of genetic risks for smoking. In a sample of 14-year-olds (with 67% shared environmental factors), Dick and colleagues (2007b) estimated a 21% heritability for lifetime quantity smoked, but this estimate decreased to 15% under conditions of high levels of perceived parental monitoring and increased to 60% with perceptions that parental monitoring was low. These results suggest that less perceived parental monitoring may provide conditions that are conducive for the expression of genetic risk for the smoking phenotype. In the study of 14-year-olds, the moderating effect of parental monitoring

was not influenced by whether the parents were smokers (Dick et al. 2007b).

Shared time with parents, another parental variable, may affect the expression of genetic risk on lifetime quantity smoked but in an unexpected direction. Among 14-year-olds, spending more time with parents was associated with 50% heritability for lifetime quantity smoked, but spending less time with parents was associated with almost no heritable effects (Dick et al. 2007a). The authors surmised that "spending more time with biologically related relatives may engender the expression of genetic predispositions" and that "for some children, spending time with parents may be beneficial, but for other children, it may not, depending on the behavior and predispositions of the parents" (Dick et al. 2007a, p. 323). Current smoking by parents also moderated the effects of genetic predispositions.

The school environment may also moderate genetic risk for smoking behavior in adolescents. Boardman and colleagues (2008) examined the effects of the social and demographic composition of 7th- to 12th-grade students (mother's education, student's race/ethnicity), school smoking norms (smoking status of popular students), institutional control of smoking (teachers not allowed to smoke on campus, penalties for smoking infractions), and the prevalence of student smoking, on the heritability of ever smoking (heritability estimate, 51%) and daily smoking (58%). They found no effects of these school characteristics on the heritability of ever smoking, but the heritability of daily smoking was significantly lower in schools with higher proportions of White (versus non-White) students and was significantly higher in schools in which the popular students were smokers.

A further layer of detail can be achieved by investigating the interaction between measured genetic and measured environmental factors. In a study of 9th- to 12th-grade students by Audrain-McGovern and colleagues (2006c), risk genotype was not related to smoking progression among those who had had at least one puff of a cigarette but was positively related to physical activity that, in turn, was negatively related to the progression of smoking. However, the relationships between risk genotype and physical activity and between physical activity and the progression of smoking were significant only in adolescents who participated in one or more team sports. Audrain-McGovern and associates (2006c) speculated that the type of physical activity or the social aspects of participation in team sports, or both, may be particularly rewarding in adolescents with risk genotypes, which would tend to decrease the rewarding value of cigarette smoking.

Peer influences, parental behaviors, school characteristics, and school-related activities, such as participation in team sports, are likely to be shared between twins and siblings and are, therefore, likely to be included in the overall estimate of shared environmental variance for smoking behavior unless their effects on genetic risk are explicitly tested. Considering the larger importance of shared environmental factors in the early stages of smoking behavior, it is important to understand the dynamics of measured and latent genetic risk and measured shared environmental factors on smoking behavior. Overall, the interactions of genetic and shared environmental factors are quite complex and call for continued research and careful analyses. More specifically, understanding how genes affect smoking behavior will necessitate identifying key specific factors or sets of factors in the adolescent environment that dynamically interact with genetic vulnerability to affect smoking or nonsmoking.

Neurotransmission and Brain Function in Tobacco Use

Overview of the Effects of Nicotine on the Brain

Upon inhalation of cigarette smoke, nicotine quickly crosses the blood-brain barrier and binds to nicotinic acetylcholine receptors (nAChRs) in the brain (Dani and Heinemann 1996). Activation of nAChRs stimulates the mesocorticolimbic dopamine system (a reward pathway) to produce the primary reinforcing effects of nicotine (Di Chiara 2000). Stimulation of dopamine neurons in the ventral tegmental area (VTA) by nicotine via high-affinity $\alpha4\beta2$ nAChRs (and by all drugs of abuse via specific receptor targets) causes increased firing in terminal dopaminergic fields, such as the nucleus accumbens, amygdala, and the prefrontal cortex (specifically the dorsolateral prefrontal cortex and orbitofrontal cortex). Activation of dopaminergic VTA neurons is also mediated by excitatory glutamatergic neurons projecting primarily from the prefrontal cortex (Taber et al. 1995), and presynaptic $\alpha7$ nAChRs located on glutamatergic projections enhance excitatory input (Mansvelder and McGehee 2000). The GABA interneurons in the VTA, which also express nAChRs and GABA-ergic projections from the nucleus accumbens to the VTA (Walaas and Fonnum 1980; Kalivas et al. 1993), mediate inhibitory and control processes of dopamine stimulation. Thus, the overall effect of nicotine in the VTA stems from the interactions of upstream and downstream effects (Mansvelder et al. 2003). Repeated exposure to nicotine in conjunction with environmental cues (Chaudhri et al. 2007) causes lasting changes in dopaminergic function that contribute to maintenance of smoking and the experience of withdrawal symptoms upon its cessation (Miyata and Yanagita 2001; Balfour 2002).

Studies by Fowler and colleagues (2008) and Salas and colleagues (2003) showed that withdrawal in mice after nicotine intake is linked to the medial habenula and α2 and α5 nicotine subunits. Mice lacking these receptors show a decrease in withdrawal symptoms. Also, mice lacking these receptors demonstrate increased intake of nicotine, possibly due to a difference in the inhibitory signals (i.e., diminished input) from the habenula in response to nicotine. Thus, some individuals (either through genetics or predisposition) may be more vulnerable to nicotine addiction.

Research Using Imaging in Children and Adolescents

Reward and cognitive control neural networks are implicated in the maintenance of addictive behaviors, including the use of nicotine (Kalivas and Volkow 2005; Brody 2006). Several studies have found that 9- to 19-year-old children and adolescents are at increased risk for smoking by virtue of a family history of drug use or personal history of psychiatric illness (e.g., attention-deficit hyperactivity disorder, conduct disorder). The same youth show blunted activation of the reward system (ventral striatum and frontal cortex) and relatively less activation in a distributed network of primarily frontal and cingulate cortex. They also show relatively less activation of temporal and parietal cortical regions that subserve decision making, performance monitoring, and cognitive control (Schweinsburg et al. 2004; Tamm et al. 2004; Sterzer et al. 2005; Scheres et al. 2007; McNamee et al. 2008; Rubia et al. 2008). Decreased activation may indicate deficits in impulse control coupled with dysregulation of reward sensitivity, which may help explain the etiology of psychiatric conditions.

Blunted activation of the brain to reward and challenges to cognitive control are observed in children who have not previously taken drugs. These conditions are also observed in adolescents at heightened risk for drug use relative to age-matched controls without psychopathology or a family history of drug use. This suggests that differences in reward and control processing may exist before exposure to drugs. These differences may contribute to comorbidity involving substance use and psychopathology and may explain why, in vulnerable persons, even a low level of exposure can tip the balance toward an addicted state (Gervais et al. 2006; DiFranza et al. 2007; Scragg et al. 2008).

Tobacco Dependence in Adolescence

Research demonstrates considerable variation in the length of time that youth report it takes to become addicted to using tobacco. The Hooked on Nicotine Checklist (HONC) was developed and validated specifically for assessing adolescents' dependence on tobacco; endorsement of any 1 of the 10 "yes/no" items indicates dependence (DiFranza et al. 2000, 2002):

- Have you ever tried to quit but couldn't?
- Do you smoke *now* because it is really hard to quit?
- Have you ever felt like you were addicted to tobacco?
- Do you ever have strong cravings to smoke?
- Have you ever felt like you really needed a cigarette?
- Is it hard to keep from smoking in places where you are not supposed to, like in school?
- When you tried to stop smoking (or when you have not used tobacco for a while):
  - Did you find it hard to concentrate because you couldn't smoke?
  - Did you feel more irritable because you could not smoke?
  - Did you feel a strong need or urge to smoke?
  - Did you feel nervous, restless, or anxious because you could not smoke?

In a study by DiFranza and colleagues (2007), approximately 10% of middle school adolescents endorsed one or more HONC symptoms within 2 days after having inhaled from a cigarette for the first time. In another study by Scragg and colleagues (2008), 25% of 14- and 15-year-olds endorsed at least one HONC symptom after having smoked just one cigarette in their lives.

Using longitudinal data, one study computed the length of time taken by 25% of a sample of 12- to 13-year-olds to transition from first cigarette puff to several milestones for cigarette use (Gervais et al. 2006). Reports of feeling "mentally addicted to smoking cigarettes" and smoking one entire cigarette were made 2 to 3 months after the first puff, cravings for cigarettes about 4 to 5 months later (than the first puff), and feeling "physically addicted to smoking cigarettes" about 5 to 6 months after the initial puff. Notably, these behaviors preceded monthly smoking, which was reported about 10 months after the first puff, and preceded having smoked 100 cigarettes, which was reached 20 months after the first puff.

These studies show that symptoms of tobacco dependence are seen in some adolescents well in advance of regular smoking. Thus, at least for a subgroup of adolescents, the conceptualization of a stagewise progression toward

tobacco dependence may not be appropriate because these youth are immediately or rapidly reinforced for initial smoking. In brief, these adolescents appear to transition rapidly from a tobacco-naive state to a tobacco-dependent state. Early-emerging symptoms of nicotine dependence during adolescence, however, have been found to be a poor prognostic indicator for chronicity of smoking in adulthood (Dierker and Mermelstein 2010).

Still, biological evidence is accumulating to suggest that the adolescent brain may be particularly susceptible to the addictive properties of nicotine (Chambers et al. 2003). Human and animal studies of the adolescent brain have demonstrated heightened neuronal sensitivity to nicotine and other constituents of cigarettes (Belluzzi et al. 2004, 2005; Cao et al. 2007). In addition, exposing the developing brain to nicotine has been shown to alter its structure and function in a way that introduces long-lasting vulnerability for addiction to nicotine and other substances of abuse (Leslie et al. 2004; Debry and Tiffany 2008; Dao et al. 2011).

Developmental Processes: Prenatal Exposure to Nicotine

More than 15% of pregnant women in the United States smoke (SAMHSA 2010) despite the significant perinatal and postnatal risks of this behavior to their offspring (Salihu and Wilson 2007). Of note is that more than 20% of pregnant adolescents 15–17 years of age smoke (SAMHSA 2010). Use of smokeless tobacco is common in Western Alaska Native pregnant women (58%), though less so over the entire state (17.8%), but still alarming rates in light of the prevalence in the general population of U.S. women of one-half of 1% (Renner et al. 2005; Kim et al. 2010). Use of smokeless tobacco is also prevalent (34%) among pregnant women in certain parts of India (Bloch et al. 2008). Nicotine (in tobacco smoke or in smokeless tobacco products) can have direct effects on nAChRs, which are already present in the brain and spinal cord of fetuses at 4 weeks of gestation (Hellström-Lindahl et al. 1998), suggesting that nAChRs play an important role in the development of the nervous system. Researchers performing animal studies (Slotkin 1998; Slikker et al. 2005) have surmised that prenatal exposure to nicotine affects neural development. Maternal smoking during pregnancy has been associated with increased risks for the offspring of ever smoking, regular (or current) smoking, and dependence on tobacco as preadolescents, adolescents, and young adults (Kandel et al. 1994; Kandel and Udry 1999; Cornelius et al. 2000; Buka et al. 2003; Al Mamun et al. 2006). However, some studies have not found such associations (Kandel et al. 1994; Silberg et al. 2003; Cornelius et al. 2005; Knopik et al. 2005; Roberts et al. 2005; O'Callaghan et al. 2006), and so there is need for further investigation.

Prenatal exposure to nicotine affects outcomes among offspring through established deleterious influences on fetal growth or as part of a maternal profile of substance use or comorbid psychopathology (Cornelius et al. 2011). This kind of prenatal exposure may also alter the sensitivity of the offspring to later environmental influences (Abreu-Villaça et al. 2004), which could predispose the offspring to a given behavioral trajectory. Thus, the environmental influences would become the salient proximal risk factors for behavior and might mask, in statistical analysis, the changes in sensitivity initially conferred by prenatal exposure to nicotine.

Summary

Future research should explore the influence of specific neural mechanisms at all stages of tobacco use and the relationships of such mechanisms with the underlying genetic architecture. Future work should also explore how the brain integrates information from large social and physical environments, small social groups, and cognitive factors to influence tobacco use behaviors in a measurable way.

At this time, research on neurobiological mechanisms that contributes to our knowledge of the etiology of tobacco use in humans lags significantly behind research on the other important influences on tobacco use summarized in this chapter. So far, the evidence from the literature on animals and adult humans indicates that nicotine activates brain reward pathways (Stein et al. 1998; Di Chiara 2000; Rose et al. 2003), the literature on adult humans indicates that smoking history is related to changes in the processing of reward and cognitive control (Anokhin et al. 2000; Martin-Sölch et al. 2001; Neuhaus et al. 2006; Musso et al. 2007), and the literature on adolescents indicates that the same changes in system responsiveness seen in adult smokers (vs. nonsmokers) are seen in tobacco-naive adolescents at risk for smoking (because of psychiatric history or familial substance use) relative to controls (Schweinsburg et al. 2004; Tamm et al. 2004; Sterzer et al. 2005; Scheres et al. 2007; McNamee et al. 2008; Rubia et al. 2008). These latter results suggest that differences in brain processing observed between adult smokers and nonsmokers may result from preexisting differences in brain processing between these groups. Some smokers' use of tobacco might be considered as part of a

general profile of psychopathology and high-risk behavior and may not be a direct effect of brain processing on tobacco use. Although evidence from neuroimaging is consistent in that observed group differences occur in the same direction as lower or higher neural activation and in overlapping brain regions, the evidence is inconclusive as to whether neural processing is related to or causes tobacco use specifically. The evidence that genes play an important role in tobacco use behaviors is increasing in the literature and consistent across samples, age groups, and age cohorts. However, the presence of genetic risk alone is not sufficient for the expression of a tobacco use behavior. Environmental factors can modify the expression of genetic risk, making it impossible to conclude that genetic variation causes a specific tobacco use behavior. Rather, genetic predisposition likely interacts in complex ways with a number of environmental factors across the large social and physical environments and among small social groups.

Evidence Summary

This chapter covered four general levels of predictors related to the etiology of tobacco use among youth. Risk factors at each of these levels are particularly potent for adolescents and young adults as they transition from childhood to adulthood. The changes in social expectations for these age groups, the further expansion of brain functioning, and the influence of peers provide a changing and challenging context with added vulnerability to tobacco use from 12 to 25 years of age.

Large Social and Physical Environments

Factors found in large social and physical environments may establish norms that affect tobacco use. For example, youth who participate in religious activity are less likely to smoke. The expression of other cultural values, such as using cigarettes as gifts, may, conversely, stimulate tobacco use. Educational attainment and academic achievement are consistently (and negatively) associated with tobacco use from early adolescence to young adulthood. In addition, persons of lower SES may be more likely to smoke because of differential norms or as a reaction to pressures, such as discrimination, or targeted marketing (see Chapter 5). Particularly in the developing world, women, who traditionally use tobacco products less often than men, have apparently been using tobacco more in recent years, perhaps as a reaction to increased marketing appeals directed at them. Physical environments favorable to tobacco use—as might be demonstrated by the availability of ashtrays or smoking areas or the presence of advertising displays—may also influence tobacco use through implicit norms that favor use.

Small Social Groups

Social influences are among the most robust and consistent predictors of adolescent smoking. Peer influences seem to be especially salient, perhaps because adolescence is a time during which school and peer group affiliations take on particular importance. Adolescents tend to overestimate the prevalence of smoking among their peers, and perceptions that one's peers smoke consistently predict use of tobacco. Another well-established finding is that adolescents are more likely to smoke if they have friends who smoke. Young smokers tend to affiliate with other young smokers, and both selection (of friends) and socialization (influences of friends) likely contribute to homogeneity in tobacco use among groups of friends. These processes that lead to homogeneity are not separate from, and are likely nested within, a similarity in factors in large social and physical environments, such as religion, social stratification, and ethnicity. In short, youth might be guided by those closest to them and by perceived social norms and then select and be influenced by peers to use or not use tobacco products.

Social network analyses have demonstrated that peer group structure uniquely contributes to the prediction of youth smoking behavior. Youth who are able to mix successfully within small social groups are relatively less likely to conform to the tobacco use behavior of others than are isolates, who perhaps have fewer social skills or experience a sense of being lower in social status within a group. The fact that popular youth are relatively more likely to smoke in schools that have relatively greater concentrations of smokers suggests that smoking behavior among peer networks is also contingent on school-level norms and attempts to be liked by others in the group.

Research on group identification indicates that youth who self-identify as belonging to deviant groups are most likely to be smokers. In addition to these peer-related effects, smoking by parents and older siblings and the quality of family relationships and parenting practices are generally predictive of all levels of smoking among adolescents. However, parental disapproval of smoking is inconsistently related to smoking by their children, and the effects of parental smoking may be mediated by such variables as the degree of monitoring and supervision provided by parents. Evidence from studies of young adults indicates there may be a continuing influence of parental smoking on the initiation and progression of smoking, although the studies are few and the findings are not sufficient for a definitive conclusion.

Intrapersonal Cognitive Processes

Beliefs about the consequences of tobacco use, decision-making capabilities, and the ability to regulate or monitor one's behavior, all of which reflect deliberate or controlled cognitive processes, are predictive of tobacco use. For example, beliefs that tobacco use leads to positive social outcomes and is relatively safe, along with poor decision-making skills and difficulties in self-monitoring, are predictive of later tobacco use. These cognitive factors may be moderated by family-level protective factors or sociocultural factors, such as relatively high SES. Alternatively, these cognitive factors may moderate the influence of sociocultural influences on the initiation of smoking. In addition, implicit attitudes (e.g., liking smoking) tend to be more positive among smokers, and measures of tobacco-related memory/implicit associations are predictive of subsequent tobacco use. Thus, both deliberate and implicit cognitive processes may predict later tobacco use among youth. However, tobacco-related implicit associations are also potent predictors of smoking among youth whose working memory has a relatively lower capacity. Cognitive processes clearly play a key role in whether a person engages in risky behaviors, but more research is needed to clarify the interplay of controlled and automatic cognitive processes.

Genetic Factors and Neurobiological and Neurodevelopmental Processes

Heritability for tobacco use is more strongly associated with regular use and dependence than with the early stages of tobacco use, suggesting that addiction to tobacco may have a relatively strong genetic component. However, the expression of genetic risk for smoking is moderated by small-group factors (e.g., peer smoking, parental monitoring, and engagement in team sports) and larger social environmental factors (e.g., school-level norms, the prevalence of smoking among popular kids). Youth at relatively greater risk for tobacco use show relatively less activation in brain structures associated with decision making and impulse control coupled with impairment in sensitivity to reward. Thus, neurobiological input into cognitive-level factors may be associated with tobacco use. More research is needed, but some evidence suggests that some youth become dependent on nicotine shortly after trying tobacco. In addition, although available studies show mixed results, some evidence indicates that a mother's smoking during pregnancy may increase the likelihood that her offspring will become regular smokers. All of these neurobiological factors are moderated by other environmental factors, although they may affect the operation of these other factors as well.

Multilevel Influences on Tobacco Use

This chapter has focused on four primary levels of influence related to the etiology of tobacco use among adolescents and young adults. There are increasing numbers of studies that consider interactions between multiple levels of influence. Innovations in statistical techniques now allow for more sophisticated models that are helping to disentangle the relative contributions of nested factors important in the onset and progression of tobacco use among youth. Multilevel models, for example, are now being used to examine the relative influence of community-level, school-level, and individual-level risk and protective factors for tobacco use (Ali and Dwyer 2009; Mayberry et al. 2009; Wen et al. 2009; Ennett et al. 2010; Kelly et al. 2010; McVicar 2011). These studies suggest that proximal social influences (e.g., individual, peer and parental influences) are particularly potent predictors of tobacco use among young people, having a stronger, more direct, and more immediate influence than do macrolevel factors (e.g., the school climate, community norms). However, these macrolevel factors are also powerful predictors, since they are pervasive in society and because they strongly affect the proximal social influences. These studies underscore corresponding findings from recent reviews on peer (Simons-Morton and Farhart 2010) and parental influences on youth tobacco use (Emory et al. 2010; Leonardi-Bee et al. 2011).

Summary

Adolescence represents a critical period of vulnerability for the onset and progression of smoking. Understanding the etiology of tobacco use in youth and young adults can be complex. The determinants of adolescent and young adult smoking are many and interrelated. Smoking uptake and progression is determined by the concurrent and joint contributions of the biological, psychosocial, and environmental factors identified in this chapter. The identified influences may exert small to large effects across adolescents' transitions from initiation to experimentation to regular use. Similarly, these factors may be more or less influential across developmental periods. For example, parental risk factors tend to become less important relative to peer risk factors with increasing age. New areas of research about the etiology of smoking among young people have been developing rapidly since the publication of the last Surgeon General's report on youth (USDHHS 1994) and have been summarized in this chapter. Much more remains to be learned, especially in the area of cognitions and the neurobiology of smoking risk and the development of tobacco dependence.

Conclusions

1. Given their developmental stage, adolescents and young adults are uniquely susceptible to social and environmental influences to use tobacco.

2. Socioeconomic factors and educational attainment influence the development of youth smoking behavior. The adolescents most likely to begin to use tobacco and progress to regular use are those who have lower academic achievement.

3. The evidence is sufficient to conclude that there is a causal relationship between peer group social influences and the initiation and maintenance of smoking behaviors during adolescence.

4. Affective processes play an important role in youth smoking behavior, with a strong association between youth smoking and negative affect.

5. The evidence is suggestive that tobacco use is a heritable trait, more so for regular use than for onset. The expression of genetic risk for smoking among young people may be moderated by small-group and larger social-environmental factors.

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