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RISK FOR EARLY SMOKING ONSET IN A HIGH-RISK POPULATION: CONTRIBUTIONS OF EARLY CHILD BEHAVIORAL RISK AND HETEROGENEITY OF PARENTAL SMOKING AND ALCOHOLISM

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By

Eun Young Mun

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ABSTRACT

RISK FOR EARLY SMOKING ONSET IN A HIGH-RISK POPULATION: CONTRIBUTIONS OF EARLY CHILD BEHAVIORAL RISK AND HETEROGENEITY OF PARENTAL SMOKING AND ALCOHOLISM

By

Eun Young Mun

This study examined early smoking onset in a high-risk population of adolescents. Participants were 281 families with biological parents and their children (281 sons and 88 daughters) residing in the Mid-Michigan area who completed at least two of the first four assessments of the ongoing University of Michigan – Michigan State University Longitudinal Study. Parental cigarette smoking and alcoholism were analyzed using a group-based semi-parametric modeling approach, resulting in three types of smokers (Heavy smokers, Light smokers, and Heavy-to-light smokers) and two types of alcoholics (Alcoholism I and Alcoholism II). Long-term paternal Heavy smoking, in combination with long-term maternal smoking, sufficiently elevated the chance of early smoking onset in offspring. Similarly, children in two-parent families where both parents were chronically alcoholic (Alcoholism II) had an increased likelihood of early smoking onset. However, smoking or alcoholism by just one parent in two-parent families did not pose much risk for early smoking onset in offspring.

Adolescents who started smoking by age 14 were different on domains that traced back to as early as prenatal development. Adolescents with early smoking onset showed a higher exposure to maternal daily cigarette smoking. Moreover, they were different on temperament and behavioral characteristics as early as ages three to five. Adolescents with early smoking onset were more reactive and approaching. In addition, their mothers

perceived them as having higher levels of negative affect (anxious/depressed), attention problems, delinquent behavior, and aggressive behavior. Structural equation modeling analysis on these parental and individual risk factors for early smoking onset revealed that maternal cigarette smoking during pregnancy led to early smoking onset in offspring via early child negative affect (i.e., anxious/depressed).

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It took a while to this end but I am jubilant that it is time to express my gratitude to many who have helped me along the way. I have greatly enjoyed my time at Michigan State University both academically and personally. Dr. Hiram E. Fitzgerald and Dr. Alexander von Eye have nurtured and cared for my growth as a psychologist and I am forever indebted to them for their unwavering support and encouragement. I would also like to thank Dr. Robert A. Zucker for his guidance in the literature of alcoholism. Despite being busy all the time, he has always responded to my needs and provided insightful suggestions and comments. I would like to express my gratitude to Dr. Joel Nigg for his valuable observations and suggestions. Finally, I would like to thank my friends and family. Without them the long journey would not have been as enjoyable and fun.

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INTRODUCTION

Cigarette smoking is a major cause of preventable premature death and disease in the United States. During 1990 and 1994, 351 out of 100,000 deaths were attributed to smoking in the U.S. (Centers for Disease Control and Prevention (CDC), 2001), and the total annual average mortality due to smoking was approximately 430,000 (CDC, 1999; Malarcher et al., 2000). In addition, the economic burden of tobacco use, in parallel to the health burden, has been estimated annually at $53.3 billion in direct medical costs alone (CDC, 1999; Malarcher et al., 2000). Indirect costs associated with morbidity and premature mortality from cigarette smoking have been estimated at $6.9 billion and $40.3 billion, respectively (CDC, 1999; Malarcher et al., 2000). These statistics indicate that smoking is a major health problem in the United States.

Epidemiological studies have reported an overall decrease in smoking prevalence over the last three decades in the United States, due to successful smoking cessation among adults, since the first report by the Surgeon General on detrimental health consequences of smoking appeared in 1964 (U.S. Department of Health and Human Services (USDHHS), 1994). However, there is an indication that smoking prevalence, especially among girls and women, may be increasing in recent years (USDHHS, 2001).

Furthermore, there are growing concerns that rates of smoking initiation have not decreased over the past decade (USDHHS, 1994) and data for the 1990s suggest that rates of smoking initiation among adolescents are on the rise (Mendez, Warner, & Courant, 1998). Increasing rates of smoking initiation are a major concern especially since risk for initiation into cigarette smoking is mostly over by age 20 (Chassin, Presson, Rose, & Sherman, 1996b; Chassin, Presson, Sherman, & Edwards, 1991; Chen &

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Kandel, 1995; USDHHS, 1994), and that changes in smoking status after age 20 mostly reflect a series of cessation attempts and relapses rather than new initiation (Chassin et al., 1996b; Chen & Kandel, 1995). Adolescents with early smoking initiation are at risk for the development of tobacco dependence, and are subject to higher rates of the morbidity and mortality associated with cigarette smoking in adulthood (Heishman, Kozlowski, & Henningfield, 1997; Jackson, 1998). Onset of cigarette smoking prior to age 13, in particular, is related to increased risks for adult daily smoking (Chassin et al., 1996b; Chassin, Presson, Sherman, & Edwards, 1990; Hanna & Grant, 1999; Jackson, 1998; Patton, Carlin, Wolfe, Hibbert, & Bowes, 1998), lower cessation and higher relapse rates (Patton et al., 1998), and DSM-IV diagnoses of drug dependence/abuse and lifetime depressive disorder (Hanna & Grant, 1999). In addition, tobacco is often the gateway drug used by adolescents who later use alcohol, marijuana, and other drugs (USDHHS, 1994).

Thus, early onset of cigarette smoking is a behavior of significance whose etiology needs to be studied in order to set up effective prevention as well as intervention programs against smoking. So far, public health efforts to control smoking initiation among adolescents have been geared toward school-based programs and reinforcement of increased restrictions on the advertisement and sale of tobacco products (CDC, 1994, 2000; USDHHS, 1994). The underlying rationale for the school-based programs is provided by the findings from mostly cross-sectional or retrospective studies that show smoking initiation is predicted by an epidemic or exposure model that requires contacts with smoking peers, parents, and/or siblings (Bobo & Husten, 2000; Rowe, Chassin,

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Presson, & Sherman, 1996; Rowe & Rodgers, 1991), with the presence of smoking peers as the single best predictor of adolescent smoking (USDHHS, 1994).

There is little doubt that socialization with smoking peers plays an important role in smoking behavior among adolescents, but some caution is warranted. First, predictors of smoking from cross-sectional studies, including socialization factors, are not fully supported in longitudinal studies of smoking (Chassin, Presson, Montello, Sherman, & McGrew, 1986; Engels, Knibbe, & Drop, 1999). On the contrary, there is evidence suggesting that influences of smoking peers may not be as potent as previously understood when studied longitudinally. A study on non-smoking children initially in the third or fifth grade found that it was not smoking peers but smokers at home that predicted higher rates of smoking initiation three years later (Patton et al., 1998).

The issue of comparability of evidence from cross-sectional and longitudinal research studies is not limited to smoking research (e.g., Ge, Lorenz, Conger, Elder, Jr., & Simons, 1994). It is difficult to establish direction of the causal relationship between variables from a snap shot approach because it is not clear what occurred first out of two related events. Although temporal priority does not always establish the causal direction between events, it is the "single most effective means" of doing so, and that fulfils one of the three conditions of causation (i.e., isolation, association, and direction of causation; Bollen, 1989, pp. 40-79). The influences of peer socialization on substance abuse among adolescents in cross-sectional studies, for example, may reflect both peer-selection (i.e., smoking-prone adolescents choose to socialize with peers of the same kind), and peer socialization (i.e., adolescents start smoking because of peer pressure and modeling of smoking behavior) processes (Curran, Stice, & Chassin, 1997).

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Second, recent studies suggest that the risk structure of smoking initiation may exist well before adolescents try a first cigarette, possibly involving genetic, biological, and psychosocial factors. It has long been wondered "why, with extensive exposure and widespread experimentation with tobacco, some people become nicotine dependent, others develop a pattern of occasional use, and still others avoid it entirely" (O. F. Pomerleau, Collins, Shiffman, & C. S. Pomerleau, 1993). Although initial sensitivity to nicotine has widely been recognized as a factor for smoking initiation, it is relatively recent that initial sensitivity to nicotine and its inverse relation to tolerance have been proposed as a key to nicotine dependence (i.e., "sensitivity" model of tolerance; see Pomerleau et al., 1993). According to this model of nicotine dependence, regular smokers may be those who are constitutionally sensitive to nicotine, react aversely to initial smoking, and quickly develop tolerance to nicotine.

New epidemiological evidence sheds further light on a constitutional or pathological model of etiology of smoking. As the prevalence of cigarette smoking among adults in the U.S. declined from 42% in 1965 to approximately 25% in 1990s (CDC, 1999), smoking has increasingly become linked to people with conditions such as depression, alcoholism, attention deficit-hyperactivity disorder, conduct disorder, antisocial personality disorder, schizophrenia, and Alzheimer's disease (Hanna & Grant, 1999; Picciotto, 1998; C. S. Pomerleau, 1997). In line with the observation of the comorbidity of nicotine with other conditions, nicotine is known to help relaxation, reduce stress, enhance attention, improve cognitive function, and regulate mood, and it has increasingly been studied as a medication for many medical conditions (Benowitz, 1996).

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Thus, it is plausible that nicotine is more reinforcing as a stimulant for individuals with a vulnerability factor, including behavioral and affective conditions (e.g., Hughes, Rose, & Callas, 2000; Picciotto, 1998; O. F. Pomerleau et al., 1993). In addition to the comorbidity of nicotine, over the past three decades quit attempts have been more successful among people with higher education and socioeconomic status compared to people with lower socioeconomic background (C. S. Pomerleau, 1997). These findings suggest that there are some individuals who are more susceptible to cigarette smoking above and beyond the influences of peer socialization, and that their susceptibility may be identified earlier.

The majority of studies that revealed the associations of smoking with other psychopathologies, however, relied on retrospective, self-reported age of onset of smoking, drinking, and use of other illicit drugs. The reliability and accuracy of self-reported age of onset can be inconsistent even within a one-year time period (Johnson & Mott, 2001). Therefore, it is important that the individual risk attributes of adolescents prior to smoking onset, and smoking onset itself should be identified prospectively and concurrently. The current study aims to investigate predictors of early smoking onset in a high-risk population from a prospective longitudinal study of adolescents and their parents.

Intergenerational Transmission of Smoking

In the literature, the relationship between parental smoking and smoking of the offspring is relatively understudied. The limited existing studies show inconsistent findings whether parental smoking is a risk factor for adolescent offspring's smoking (USDHHS, 1994). However, there is a growing body of new evidence that parental

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smoking is related to children's smoking as indicated by a greater prevalence rate of smoking in children of smokers than in children of nonsmokers (Chassin et al., 1996b; Chassin, Presson, Todd, Rose, & Sherman, 1998; Cornelius, Leech, Goldschmidt, & Day, 2000; Griesler, Kandel, & Davies, 1998; Kandel, Wu, & Davies, 1994; Patton et al., 1998; Rowe et al., 1996).

This phenomenon of intergenerational transmission of smoking is open to a number of explanations, including 1) genetic transmission of susceptibility to nicotine (Koopmans et al., 1999; Madden et al., 1999; True et al., 1997), 2) a common familial vulnerability represented by family history of alcoholism (Hanna & Grant, 1999), 3) prenatal exposure to maternal smoking (Cornelius et al., 2000; Griesler et al., 1998; Kandel et al., 1994), 4) attentional and behavioral problems in childhood (Griesler et al., 1998), and 5) parenting behavior (Chassin et al., 1998). These five explanations are neither exhaustive nor competing with one another as the mechanism of adolescent smoking onset. It is more likely that early smoking onset in adolescence reflects a complex interplay among distal and proximal factors via direct and indirect pathways. The current study focuses on the interplay among a familial vulnerability and an individual vulnerability. Parental smoking and alcoholism represent the former, while an prenatal exposure to maternal smoking, child early temperament, and behavioral problems in childhood embody the latter in the present study.

Heterogeneity of Parental Smoking

In the literature on parental smoking as a risk factor for adolescent children's smoking initiation, the possibility of differential influences of parental smoking has relatively been overlooked. Studies on clinical populations of smokers have noted

differences between heavy smokers and light smokers (O. F. Pomerleau et al., 1993), or regular smokers and "chippers" (Kassel, Shiffman, Gnys, Paty, & Zettler-Segal, 1994; Shiffman, Kassel, Paty, Gnys, & Zettler-Segal, 1994a, Shiffman, Paty, Kassel, Gnys, & Zettler-Segal, 1994b) on various smoking-related measures, including sensitivity to nicotine, attitude toward smoking, and familial history of smoking. In particular, Shiffman and his colleagues have reported that there are a small number of long-term light smokers or "chippers" who smoke no more than one to five cigarettes a day but do not develop nicotine dependence (Kassel et al., 1994; Shiffman et al., 1994a, 1994b). In addition, the age of smoking onset appears to be related to nicotine dependence (Breslau, Fenn, & Peterson, 1993). Of those who have ever smoked, only a quarter to a third develop nicotine dependence, and those who initiated smoking between 14 and16 were more likely to become dependent than those who initiated smoking at an older age (Breslau et al., 1993).

Unfortunately, parental smoking has rarely been studied as a risk factor for adolescent children's smoking initiation, with the exception of studies of familial resemblance (e.g., Eysenck, 1980; cf. O.F. Pomerleau et al., 1993), due in part to the prevailing epidemic view of smoking initiation. In contrast to the study of alcoholism where the heterogeneous nature of alcoholism (Cloninger, 1987; Hill, White, Chung, Hawkins, & Catalano, 2000; Schulenberg, O'Malley, Bachman, Wadsworth, & Johnston, 1996; Zucker, 1987, 1994) and the differential risks to their offspring have been hypothesized and tested (Zucker, Ellis, Bingham, & Fitzgerald, 1996), studies of smoking initiation have focused more on differential factors involved in early stages of smoking rather than individual differences in smoking pathways. For example, social contact with

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smoking peers and parents has been suggested as a major mechanism for the initiation of smoking by adolescents (Rowe et al., 1996; Rowe & Rodgers, 1991; Rowe, Rodgers, & Gilson, 2000) whereas genetic contributions have been suggested to be more prevalent for persistent smoking compared to the initiation of smoking (Madden et al., 1999; True et al., 1997) or high quantity of smoking (Koopmans et al., 1999).

Variable-centered approaches, however, do not capture individual differences in intra-individual change across the life span as well as the person-oriented approaches do (Bates, 2000; Bergman & Magnusson, 1997; Magnusson, 1998, 1999, 2000).
One of the critical issues related to smoking research is that variable-centered approaches are cumbersome when it comes to handling the timing of smoking transitions across time. In some studies, age differences are studied as a covariate for the changes from non-smoker to experimenter (i.e., initiation of smoking), to regular smoker (i.e., persistence of smoking), or to quitter. However, there is great variability in timing of smoking – smoking onset, regular smoking, and quit attempts. Furthermore, in developmental research not only age differences but also age changes are of interest. Age differences (inter-individual variability) refer to differences found among different age groups, whereas age changes (intra-individual variability) refer to changes within the same individual over time (Wohlwill, 1973). Studies of distinctive growth patterns from person-oriented approaches, in particular, can be characterized by many parameters that include the presence, direction, rate, level, and timing of change, general shape of a developmental pattern, and age corresponding to specified values of any of those characteristics (Wohlwill, 1973; cf. Bates, 2000).

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A recent study by Chassin and her colleagues (Chassin, Presson, Pitts, & Sherman, 2000) is one of the few studies that investigated individual differences in intra-individual changes in smoking behavior from a population-based cohort sample over a lengthy period. Using a semiparametic mixture group-based modeling (Nagin, 1999; Nagin & Tremblay, 1999, 2001) designed to investigate heterogeneous developmental patterns (B. Muthén & L. K. Muthén, 2000; B. Muthén, 2001; Nagin, 1999), four developmental patterns of cigarette smoking from ages 11 to 31 were identified: Early Stable (12%), Late Stable (16%), Experimenter (6%), and Quitter (5%) groups. The abstainers or non-smokers (approximately 60%) and relatively a small number of erratic smokers were a priori identified and excluded from the semiparametic mixture group-based modeling analysis.

Early Stable smokers had an early onset of smoking with a steep escalation to a stable and high consumption level over time. Late Stable smokers were those who established their regular smoking relatively late, and whose levels of smoking were not as high as the Early Stable group. Experimenters were those who started smoking early but never reached levels of smoking by either the Early Stable or the Late Stable smokers and who stopped smoking before age 20. And finally, Quitters were those who started smoking relatively late, who reached high levels of chronic smoking, and who quit smoking before age 25.

These four groups of smokers and quitters were different in various psychosocial measures, with Early Stable smokers reporting high tolerance for deviance, high external locus of control, low levels of parental support, and least likelihood of obtaining college education. In addition, they were also more likely to have parents and friends who

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smoked, and held most positive beliefs about the health and psychological consequences of smoking. On the other hand, Late Stable smokers had few friends and parents who smoked, held relatively negative beliefs about smoking, and reported high internal locus of control and parental support. Thus, even though smokers of the Early Stable and Late Stable trajectories were habitual smokers, they were distinctively different on measures of smoking consumption and psychosocial measures.

From the findings of Chassin et al.'s study (2000), we can naturally ask the following two key questions: 1) Can heterogeneous developmental courses of smoking also be found during the much wider age span in adulthood, ranging from the 20s to 50s? and, 2) Are adolescent children of smokers at different risks for early smoking onset? The first issue addresses the fact that Chassin et al.'s study (2000) covered a developmental period from adolescence to young adulthood where smoking initiation and experimentation most often occur. Therefore, changes in the patterns of cigarette smoking are more likely. Once smoking is established, however, the possibility exists that there may not be much variation in smoking. Alternatively, adult smokers may consist of distinctively different subpopulations that differ on psychosocial characteristics as well as their smoking characteristics such as quantity and duration of cigarette smoking. The second question is a natural extension to the first issue: Are there different levels of risk tied to parental smoking subtypes for early smoking onset? Are there specific patterns of parental smoking linked to adolescent children's early smoking onset? For example, are habitual heavy smokers, occasional smokers, and current abstainers with a past history of heavy smoking the same in levels of risk for early smoking in offspring?

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A Family History of Alcoholism

There is evidence that alcohol and tobacco go hand in hand for many individuals in adolescent as well as adult populations (Grant, 1998; Hughes, 1995; Hughes et al., 2000; Madden, Heath, Starmer, Whitfield, & Martin, 1995; O. F. Pomerleau, 1995; Shiffman & Balabanis, 1995) and that both alcoholism and habitual smoking run within families (Bierut, Schuckit, Hesselbrock, & Reich, 2000; Madden, Bucholz, Martin, & Heath, 2000). On the basis of this comorbidity, some researchers suggest that there may be a common familial vulnerability for the use of alcohol and tobacco among individuals with a positive family history of alcoholism (Grant, 1998; Hanna & Grant, 1999; Sher, Gotham, Erickson, & Wood, 1996). The mechanisms of why a family history of alcoholism is related to use of alcohol and tobacco in offspring are not yet understood.

One possibility is that the common genetic mechanism may exist that is associated with but not limited to both alcohol and tobacco use (Lerman et al., 1999). SLC6A3-9 genotypes[1] that are known to be associated with late initiation of smoking and smoking cessation, for example, may also account for reduced need for novelty and reward by external stimuli such as alcohol and tobacco (Lerman et al., 1999; Sabol et al., 1999). In addition, there is speculation that family resemblance in the manifestation of alcoholism is, in part, accounted for by a genetic liability of a general state of CNS disinhibition/hyperexcitability which can also be found in high risk individuals for alcoholism, substance abuse, antisocial personality, and attention deficit hyperactive disorder (Begleiter & Projesz, 1999).

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1 SLC6A3 is the dopamine transporter gene that regulates synaptic dopamine by coding for a reuptake protein. The SLC6A3-9 genotype is a variant of the SLC6A3 gene.

A positive family history of alcoholism also reflects environmental as well as genetic risks that canalize developmental pathways of children toward maladaptive behavior, including smoking and drinking (Fitzgerald et al., 1993; Zucker et al., 1996). Paternal antisocial alcoholism, in particular, is associated with a number of risk factors, including low family socioeconomic status (Fitzgerald & Zucker, 1995), relationship disturbances between spouses (Ichiyama, Zucker, Fitzgerald, & Bingham, 1996), neuroticism (Piejak, Twitchell, Loukas, Fitzgerald, & Zucker, 1996), and depression and family violence (Ellis, Zucker, & Fitzgerald, 1997). In relation to smoking of adolescent children, parents who habitually smoke and drink may hold more favorable attitudes and beliefs toward smoking and drinking, which indirectly influence their children's drinking and smoking. Furthermore, alcoholism of parents may interfere with their parental roles as a major supervisor of their children's activities.

Thus, it appears that a family history of alcoholism can be considered as a general risk factor for developmental outcomes in children as well as a more specific risk factor for substance use, including early onset of smoking. However, a positive family history of alcoholism has many dimensions. Alcoholism subtype is one aspect that is underexplored. Although the heterogeneous nature of alcoholism has often been hypothesized and studied in the literature (Babor et al., 1992; Cloninger, 1987; Zucker, 1987; Zucker, Chermack, & Curran, 2000; Zucker, Ellis, Fitzgerald, Bingham, & Sanford, 1996; Zucker, Fitzgerald, & Moses, 1995), alcoholism subtypes have never been studied before in relation to early smoking onset among adolescent children of alcoholics. Furthermore, a positive family history of alcoholism has many variables, including the

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number of alcoholic parents in family and the specificity of alcoholic parent in relation to developmental outcomes in children.

Prenatal Exposure to Maternal Cigarette Smoking

Risk factor for cigarette smoking in offspring. It is well known that prenatal exposure to maternal cigarette smoking influences the developing fetus by altering maternal physiology that limits the amount of oxygen and nutrients to the fetus (Weitzman, Gortmaker, & Sobol, 1992). A new wave of studies suggests that maternal smoking during pregnancy may directly be related to smoking initiation by the adolescent offspring (Cornelius et al., 2000; Griesler et al., 1998; Kandel et al., 1994). A study by Kandel et al. (1994) provides the first glimpse of a link between prenatal exposure to smoking and smoking of offspring. A subset of 192 mothers and their first-born children aged 9-17 years was drawn from a larger representative sample of adolescents in grades 10 and 11 in New York State public high schools in 1971/72. A significant association between maternal smoking during pregnancy and the child's smoking 13 years later was found with stronger associations for the child's smoking during the last year than for ever-smoking, and for daughters than for sons.

Kandel et al. speculated that maternal smoking during a critical prenatal period of brain development may modify the dopaminergic system structurally and functionally, predisposing the child to smoke and to persist in smoking later in life. The speculation of altered dopaminergic system in the brain due to prenatal exposure to nicotine has also been echoed by many researchers as a possible mechanism for the association of maternal smoking with behavior problems (Weitzman et al., 1992), conduct disorder (Wakschlag et al., 1997; Weissman, Warner, Wickramaratne, & Kandel, 1999), attention deficit

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hyperactivity disorder (ADHD) (Milberger, Biederman, & Faraone, 1997; Millberger, Biederman, Faraone, Chen, & Jones, 1996, 1997), and attention problems and impulsive behaviors (Fried, Watkinson, & Gray, 1992).

Taken together, evidence so far in the literature suggests that prenatal exposure to nicotine is one of early risk factors for later smoking behavior among adolescents via alterations in the structure and function of the brain. However, it is also suggested in the literature that there may be mediating pathways to smoking behavior in adolescence (Chassin et al., 1998; Griesler et al., 1998). Mediating pathways may include many psychosocial and ecological dimensions such as positive attitudes toward smoking in family, availability of cigarettes, and a lower level of awareness of the negative health consequences of smoking. It is also plausible that prenatal exposure to nicotine may also play a role in smoking onset among adolescents via behavioral characteristics, including attention deficit problems and conduct problems in childhood, known antecedents to a cluster of problem behaviors including smoking in adolescence.

Indirect link to early smoking onset via behavioral characteristics. Just as prenatal exposure to cigarette smoking presents risks for adolescents' smoking initiation, so too do behavioral problems in childhood and adolescence. A study by Weitzman et al. (1992) was the one of the early studies that observed higher behavior problems among children who were exposed to smoking prenatally. The observed association remained significant even when variables known for their association with behavior problems were controlled for, including birth weight, prenatal alcohol consumption, family income, and parental education and intelligence. Similar relationships were reported in children as early as three years of age (Day, Richardson, Goldschmidt, & Cornelius, 2000;

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Townsend, 1998) and adults as late as 34 years old in terms of arrests for nonviolent and violent crimes in a dose-response relationship (Brennan, Grekin, & Mednick, 1999). In addition, the associations between prenatal smoking exposure and a host of behavioral problems including delinquency (Bagley, 1992) were reported not only from maternal ratings but also independent observer ratings (Fergusson, Horwood, & Lynskey, 1993).

From the findings, we can speculate that the risks of prenatal exposure to maternal cigarette smoking for early smoking onset in children may indirectly be transmitted via behavioral characteristics. The association between behavior problems and substance use among adolescents, including smoking and drinking, has long been modeled and reported in the frame of problem behavior theory (R. Jessor & S. L. Jessor, 1977). According to this theory, there is an underlying tendency toward deviancy that is manifested in various forms: smoking, drinking, early sexual behavior, poor school performance, and association with deviant peers in adolescence. While these behaviors may be found more often together than in isolation, recent studies suggest that there may be an alternative explanation that addresses the more specific nature of the associations of cigarette smoking with attention, behavior, and affect, and cigarette smoking.

Alternatively, cigarette smoking is suggested to influence selective populations with negative affect and stress, poor attention, and problems with inhibition, possibly due to stimulant effects of nicotine (Downey, Pomerleau, & Pomerleau, 1996; Lambert & Hartsough, 1998; Levin et al., 1996; Milberger et al., 1997; Patton et al., 1998; O. F. Pomerleau, Downey, Stelson, & C. S. Pomerleau, 1995; Riggs, Mikulich, Whitmore, & Crowley, 1999; Tizabi et al., 1999). This self-medication hypothesis is supported in studies of animals (Tizabi et al., 1999) and adolescents. Smoking initiation is observed

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more often in adolescents with depression and anxiety (Patton et al., 1998; Riggs et al., 1999; M. Windle & R. C. Windle, 2001) and with ADHD (Burke, Loeber, & Lahey, 2001; Milberger et al., 1997; Whalen, Jamner, Henker, Delfino, & Lozano, 2002), conduct disorder, and behavior problems (Lambert & Hartsough, 1998; Levin et al., 1996; Milberger et al., 1997).

Taken together, the existing studies appear to converge on an individual sensitivity or vulnerability to cigarette smoking. However, it is still not clear which behavioral characteristics are vulnerability factors that can early be identified for smoking initiation among adolescents. It is due in part to selective populations used in studies. Participants in many studies previously mentioned were sampled from a special population such as those who were in treatment for symptoms of ADHD and behavioral problems (e.g., Milberger et al., 1997). In addition, behavioral characteristics were often measured concurrently for adolescent populations or retrospectively for adult populations in the majority of existing studies. For these reasons, for example, it is still not clear whether symptoms of ADHD are uniquely associated with smoking above and beyond the behavioral problems and vice versa (Lynskey & Hall, 2001).

In summary, the literature on adolescent smoking initiation suggest that an individual vulnerability to cigarette smoking may be traced back to their prenatal development. Negative affect, inattention, and unruly, and unrestrained behaviors may be potential early behavioral characteristics that mediate the link between prenatal exposure to maternal smoking and early onset of smoking in adolescent children. We then ask naturally whether parental smoking and alcoholism are associated with early

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onset of smoking above and beyond the more specific mediational pathways. Insignificant direct paths in the presence of the mediating pathways would suggest that intergenerational transmission of smoking is largely accounted for by the mediating pathways via prenatal exposure to daily maternal cigarette smoking and early behavioral characteristics. On the contrary, significant direct paths from parental smoking and alcoholism to early onset of smoking would strongly suggest that there are unaccounted mechanisms of parental smoking and alcoholism in leading to early smoking onset by the present study.

The Current Study

This study seeks to advance the research literature of intergenerational transmission of smoking and early smoking onset in a number of aspects using the prospective, long-term longitudinal study of a population-based, high-risk sample of early adolescents and their parents. Figure 1 illustrates a heuristic model of intergenerational transmission of smoking where a familial risk structure is linked to early smoking onset in children by mediators of individual vulnerability factors. Following the heuristic model illustrated in Figure 1, the current study plans to separately examine parental smoking, alcoholism, and early characteristics of child as risk factors for early smoking onset, and then to investigate their roles played in early smoking onset simultaneously.

More specifically and first, it was hypothesized that intergenerational transmission of smoking can be observed reliably. The following three factors related to parental smoking were explored in association with early smoking onset in offspring of smokers: 1) Parental smoking subtypes derived from a long-term prospective follow-up

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study of cigarette smoking, 2) the number of smoking parents, and 3) the relative potency of maternal versus paternal smoking as a risk factor for early smoking onset in offspring. Second, parental alcoholism was expected to predict early smoking onset in offspring. As in parental smoking, the same three factors related to parental alcoholism were explored in association with early smoking onset: 1) Parental alcoholism subtypes derived from a long-term prospective study of alcoholism, 2) the number of alcoholic parents, and 3) the relative potency of maternal versus paternal alcoholism as a risk factor for early smoking onset in offspring.

Third, it was hypothesized that prenatal exposure to daily maternal cigarette smoking would be related to early smoking onset in offspring. Heavier exposure to maternal cigarette smoking was hypothesized to trace to adolescents who have smoked by age 14. In addition, it was hypothesized that adolescents who start smoking by age 14 can reliably be differentiated on measures of early temperamental and behavioral characteristics as well as concurrent behavioral characteristics.

Finally, direct and indirect paths to early smoking onset were hypothesized and simultaneously tested. Maternal and paternal smoking subtypes, parental alcoholism, and prenatal exposure to maternal smoking were hypothesized to directly lead to early smoking onset in children. In addition to direct paths, a mediational path from maternal smoking subtype to early smoking onset was hypothesized via prenatal exposure and early behavioral characteristics. Figure 2 illustrates a simplified model of pathways to early smoking onset. Paths 1 – 3 indicate direct associations, with parental smoking and alcoholism, and prenatal exposure to smoking predicting early smoking onset in offspring (Paths 1 – 3). The first path stands for intergenerational transmission of smoking, while

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the second path represents a familial alcoholism as a risk factor for early onset of smoking. Path 3 reflects the direct association between prenatal exposure to daily maternal cigarette smoking and early smoking onset in children. And finally, Path 4 indicates indirect mediational pathway from parental smoking to prenatal exposure to smoking (Path 4-1), to early behavioral characteristics (Path 4-2), and then to early smoking onset (Path 4-3).

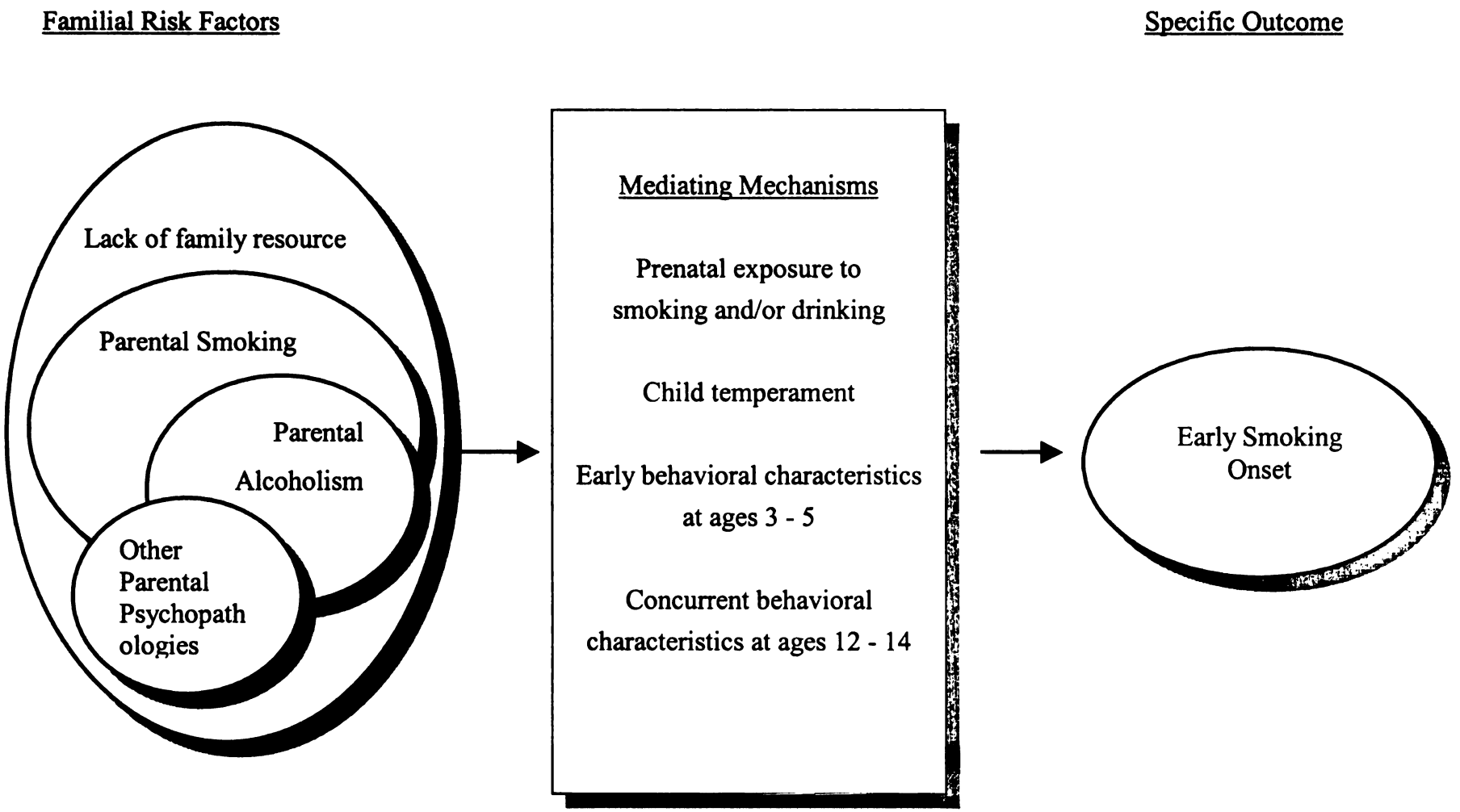

Figure 1.

A heuristic model of early smoking onset in adolescent children of smoking parents.

Parental smoking

Parental alcoholism

Prenatal exposure to cigarette smoking

Behavioral characteristics

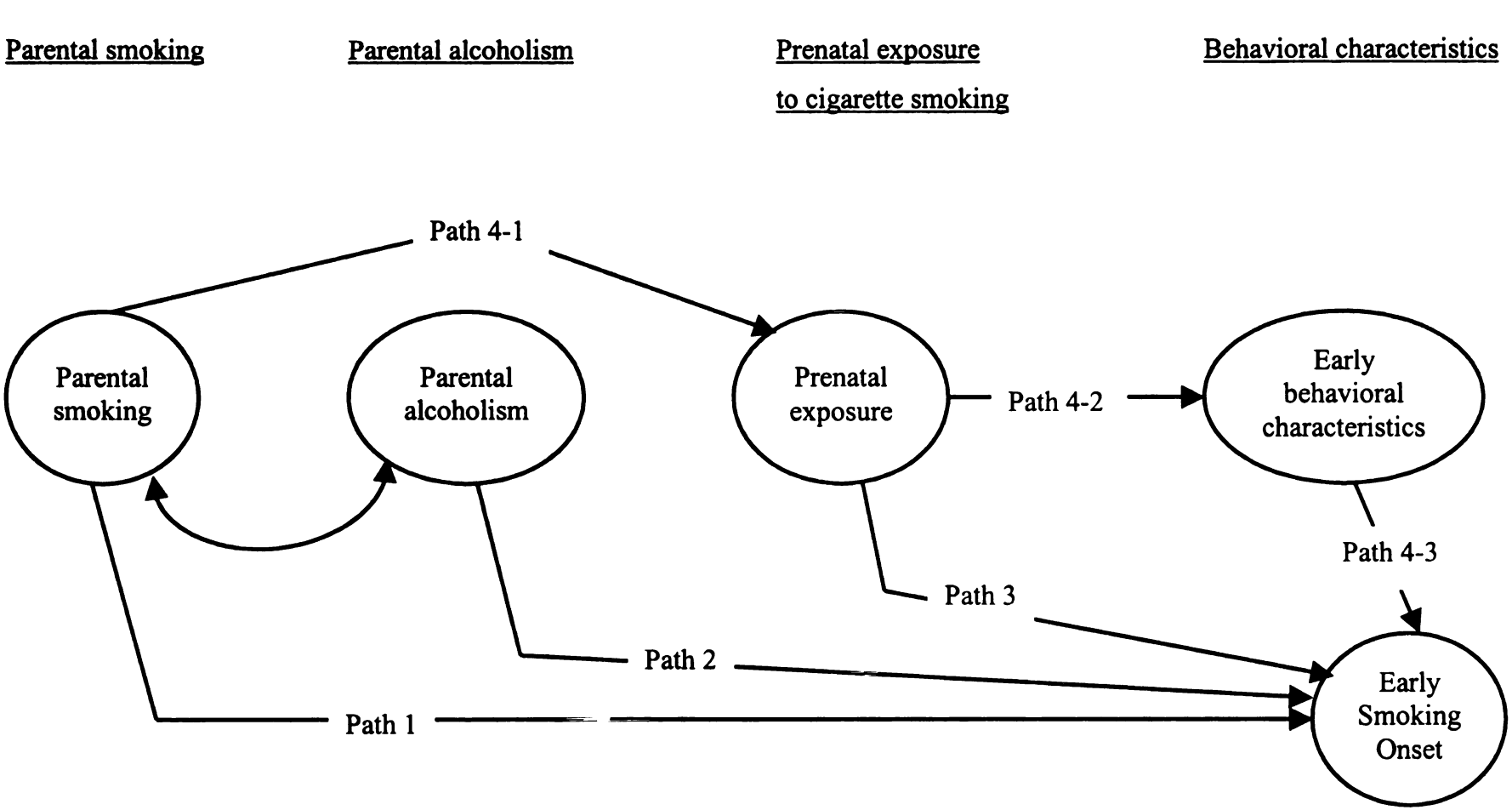

Figure 2.

A simplified model of pathways to early smoking onset.

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METHOD

Participants

Participants for the current study were 281 families with both biological parents and their sons (N = 281) and daughters (N = 88), who completed at least two of the first four waves of the larger University of Michigan – Michigan State University (UM-MSU) Longitudinal Study (Zucker et al., 2000) during the seventeen-year-span starting from 1985 until mid-2001. The UM-MSU Longitudinal Study is an ongoing longitudinal study designed to understand the risk and protective factors that affect etiologic pathways leading toward, or away from, alcohol abuse or dependence and co-active forms of psychopathology from a high risk sample of families in the mid-Michigan area with alcoholic and non-alcoholic fathers (Zucker et al., 2000). Maternal alcoholism was also assessed, but it was neither a requirement for study inclusion, nor a basis for exclusion. Families with children who manifested characteristics in the three areas required for a diagnosis of FAS (e.g., prenatal or postnatal growth retardation, central nervous system involvement, and characteristic facial dysmorphology; Cooper, 1987) were excluded (Fitzgerald et al., 1993; Noll, Zucker, Fitzgerald, & Curtis, 1992).

Almost all parents included in the present study were non-Hispanic European Americans (542 of 555; 97.7%) with the exception of eight Hispanic Americans, three Native Americans, one Asian American, and one of other ethnic heritage other than African American. All resided in the mid-Michigan area at initial assessment. Family income levels mostly fell within the lower to low-middle class range, although there were also some higher income families. Trained interviewers, who were blind to the family diagnostic status, collected the data. Since many alcoholics are known to be smokers in

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general and population-based community alcoholics don't seek out treatment, the participants in the current study provide an opportunity to investigate the natural developmental patterns of cigarette smoking and alcoholism among parents over a long period of time, and the mechanisms of how they are related to early smoking initiation among their adolescent and preadolescent children.

During the initial contact, all families were invited to participate in a long-term study of family health and child development starting at the male child ages of three to five (wave 1). Families then have been followed up once in every three years when the male child's age reached six to eight (wave 2), nine to eleven (wave 3), and twelve to fourteen (wave 4). Parental information on the areas of psychosocial functions, including their drinking and smoking has also been collected once in every three years. Daughters of participating families were recruited into the study a few years later. More information on the recruitment procedures and sample characteristics are available elsewhere (Zucker et al., 1996; Zucker & Fitzgerald, 1991; Zucker et al., 2000). The median year of assessment for each of four waves and the corresponding age at assessment for all participants included in the current study are presented in Table 1 (for more detailed information on participants, see also Appendices A and B).

Table 1

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Descriptive Statistics of All Participants

	Father[2]	Mother[2]	Son	Daughter
	N = 275	N = 280	N = 281	N = 88
Age				
Wave 1	33.15 (5.11)	30.93 (4.23)	4.31 (1.00)	4.86 (0.90)
Wave 2	36.58 (4.97)	34.47 (3.91)	7.58 (1.00)	7.56 (0.86)
Wave 3	39.54 (5.00)	37.25 (4.09)	10.40 (0.94)	10.26 (0.87)
Wave 4	42.58 (4.95)	39.99 (4.29)	13.44 (0.93)	13.29 (0.80)
Year of birth				
Median	1956	1958	1985	1986
Range	1938 - 1966	1943 - 1970	1979 - 1988	1981 - 1992
Year of assessment				
Wave 1	1989	1989	1989	1993
Wave 2	1993	1993	1993	1994
Wave 3	1996	1996	1996	1996
Wave 4	1998	1998	1998	1998

Note. The numbers in parentheses are standard deviations. Year of assessment is shown in median (For more detailed information, see also Appendices A and B.).

2 The number of fathers and mothers included in the present study were different from the number of sons because six fathers and one mother did not complete two of the first four assessments, failing to meet the criteria of the study inclusion. The cases were not deleted listwise since not all subsequent analyses required all information.

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Parental Measures

Parental smoking at waves 1 through 4. Parental smoking was determined by a single item question, "How frequently have you smoked cigarettes during the past 30 days?" Responses were 0 = Not at all; 1 = Less than one cigarette per day; 2 = One to five cigarettes per day; 3 = About one-half pack per day; 4 = About one pack per day; 5 = About one and one-half packs per day; 6 = Two packs or more per day. This seven-level variable reflects quantity of smoking at the time of measurement. This question is very widely used and accepted in the literature of smoking.

Parental alcohol use disorder at waves 1 through 4. Parental alcohol use disorder was diagnosed at each assessment wave based on the Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (DSM-IV; American Psychiatric Association, 1994) criteria. Information for the diagnosis was obtained via administration of the Drinking and Drug History Questionnaire (DDHQ; Zucker, Fitzgerald, & Noll, 1990), the Short Michigan Alcohol Screening Test (SMAST; Selzer, Vinokur, & van Rooijien, 1975), and the NIMH Diagnostic Interview Schedule (DIS; Robins, Helzer, Croughan, & Ratcliffe, 1980). The DIS and the DDHQ provided detailed information on past and current consumption of alcohol and smoking patterns and problems related to excessive alcohol use.

The DIS is a structured diagnostic interview that allows trained lay interviewers to gather extensive information about physical, alcohol-related, and drug-related symptoms, as well as other areas of psychiatric symptomatology. The DDHQ consists of items from the 1978 National Institute on Drug Abuse Survey (Johnston, Bachman, & O'Malley, 1978), the American Drinking Practice Survey (Cahalan, Cisin, & Crossley, 1969), and

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the Veterans Administration Medical Center Research Questionnaire for Alcohol (Schuckit, 1978). The DDHQ gathers information about the informant's alcohol/other drug use and problems with regard to the amount of alcohol consumption during the past six months and it also inquires about the largest amount of alcohol consumed during a 24-hour period at any point in the participant's life. The instrument also asks the participant whether s/he has experienced various problems as a result of alcohol use and, if so, the frequency of these problems. The SMAST is a well-validated screening inventory used to assess alcohol problems, consisting of 13 items with "Yes/ No" responses.

Using all three sources of information, an experienced clinician established alcohol abuse/dependence diagnoses for the following three periods: the subject's entire lifetime, the past three years, and the current year leading up to the assessment. Diagnoses were coded from zero to three: 0 = No diagnosis; 1 = Alcohol abuse; 2 = Alcohol dependence without physical dependence; 3 = Alcohol dependence with physical dependence. In the current study, alcohol use disorder diagnoses were made over four exclusive time periods and the time prior to the first assessment.

Rearrangement of data on parental smoking and alcoholism. The current study utilized age of parents as a time variable instead of assessment wave when analyzing parental data on smoking and alcoholism. The interest of the current study with regard to the measures of parental smoking and alcoholism was to see whether the change and/or stability of the developmental patterns could be found over chronological age, not over the assessment wave at which variables related to smoking and alcoholism were measured. However, parental data were collected based on their son's assessment

schedule (i.e., age), resulting in a rather heterogeneous sample of parents in terms of ages and birth cohorts. This scheme was originally designed to match parental functions to those of their son.

The research questions in the current study, however, focus more on identifying several clusters of parents based on their own functions over time. For that reason, parental data on smoking and alcoholism were rearranged so that age rather than measurement wave was the time variable for analysis. Avoiding aggregation of the data across different ages for each measurement by grouping individuals by age, allowed one to identify change and stability of smoking and alcoholism in adulthood as a function of chronological age. In addition, we can gain knowledge about a longer period of the life span in a shorter amount of time to maximize efficiency of the available data. In the current study, parental smoking was investigated for the time period from ages 24 to 50. Parental alcoholism was investigated for the time period from ages 14 to 54 for men, and ages 14 to 49 for women.

There is one shortcoming of the design that needs to be noted here. The rearrangement of the data results in a higher rate of missing values because the data were converted from four measurements in nine to twelve years to twelve plus measurements in as many years (The number of observed cases used in the TRAJ procedure is reported in Appendix D.). The key to the issue was to find the balance between maximum utilization of the available longitudinal data and integrity of the data. Given that both smoking and alcoholism are phenomena of high stability and convergence and that the analytical tool (TRAJ) utilized in the current study handles missing data well, it was decided that the reliable findings were attainable in the current study.

Parental antisocial behavior at wave 1. Parental antisocial behavior was measured by the Antisocial Behavior Checklist (ASBCL; Zucker, Noll, Ham, Fitzgerald, & Sullivan, 1994) when parents were first recruited into the project. The ASBCL is a 45-item questionnaire that assesses the frequency of aggressive and antisocial activity in both childhood (e.g., lying to parents, being suspended from school) and adulthood (e.g., being fired, resisting arrest). Chronbach's alphas were .832 and .834, respectively for the subscales of childhood and adulthood in the current study.

Parental depression at wave 1. Parental depression was measured by the revised Beck Depression Inventory (BDI; Beck, Rush, Shaw, & Emery, 1979) and the Hamilton Rating Scale for Depression (HRSD; Hamilton, 1960). The BDI was designed for assessment of the severity of depression in adolescents and adults (Beck & Steer, 1993). The BDI has been one of the most frequently used instruments for assessing the intensity of depression in psychiatric patients and for screening possible depression in normal populations since its first introduction in 1961 (Piotrowski, Sherry & Keller, 1985; Steer, Beck, & Garrison, 1986). The items represent symptoms and attitudes based on clinical observations and descriptions of symptoms frequently mentioned by depressed patients, and are rated on a 4-point severity scale ranging from zero to three. In the current study, the short version of BDI with 13 items was used (Chronbach's alpha = .751).

The HRSD was originally designed for use with patients already diagnosed as suffering from depression measuring behavioral and somatic symptoms of depression. The HRSD has been utilized most frequently as an interviewer-based measure for patient selection and later assessment (Grundy, Lunnen, Lambert, Ashton, & Tovey, 1994). The HRSD was coded following administration of the DIS by the clinician who conducted the

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interview. The score was based on both the participant's responses to interviewer's questions during the DIS administration as well as the interviewer's judgment upon completion of the DIS administration. The interviewer made both a Current Depression rating and a rating of the level of the subject's depression at the point in their life when they were most depressed (Worst Ever rating). The Worst Ever episode was selected on the basis of the period with the largest number of depressive symptoms reported. Inter-rater reliabilities obtained on this project were .78 for current depression and .80 for worst-ever depression using a sample of 16 individuals (Reider, 1991).

Parental education and occupation at wave 1. The current study included parental education measured by years of education completed and parental occupation coded using the U.S. Census occupation codes. Parental occupation was recoded based on the revised Duncan Socioeconomic Index (RDSEI-TSEI2; Stevens & Featherman, 1981). Higher scores on parental occupation indicate socially more prestigious occupations.

Child Measures

Prenatal exposure to maternal smoking and drinking. The Health History - Prenatal Form (HHPF; Carpenter & Lester, 1980) was used to obtain information on mothers' cigarette smoking and alcohol use during pregnancy. Mothers recalled how many cigarettes they smoked per day and how many drinks they had per week during pregnancy. The mean length of time between the child's birth and the mother's report on their smoking during pregnancy was 4.18 years post-delivery for boys and 9.05 years post-delivery for girls. Compared to a prospective measure of prenatal exposure to smoking and drinking, the retrospective measure used in the current study was limited. However, the time lag between the child's birth and the mother's recall was relatively

short in the current study and the retrospective report of maternal cigarette smoking during pregnancy appeared valid (Griesler et al., 1998).

Child temperament at wave 1. Child temperament was measured by the Dimensions of Temperament Survey (DOTS; Lerner, Palermo, Spiro III, & Nesselroade, 1982) rated by each parent at Wave 1. The DOTS is a 34-item questionnaire that measures five dimensions of temperament: Activity Level, Attention Span/Distractibility, Adaptability/Approach-Withdrawal, Rhythmicity, and Reactivity. The dimension scores are based on sums of the items for that dimension (1 = True; 0 = False). Three of the temperament dimensions were used in the present study as precursors of smoking behavior in adolescence: Attention Span/Distractibility, Adaptability/Approach-Withdrawal, and Reactivity. High scores on the dimensions reflect greater longer attention span and higher persistence to distraction, higher approach, and greater reactivity. Chronbach's alphas were .803, .765, and .519, respectively. Activity Level and Rhythmicity were not included for reasons of the very limited range of possible scores (0-3) and irrelevance to child outcomes in earlier work (Tarter & Vanyukov, 1994), respectively.

Behavioral characteristics[3]: Anxious/depressed, Attention problems, Delinquent behavior, Aggressive behavior at waves 1 and 4. Based on the existing literature, four of eight syndromes from the Child Behavior Checklist for Ages 4-18 (CBCL; Achenbach, 1991) were tested in the current study in relation to smoking onset in adolescence:

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3 A term, behavioral characteristics was used in place of behavioral problems or syndromes in the present study since only a few children scored over the borderline clinical cutoff score on each of four syndrome scales.

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Anxious/depressed, Attention problems, Delinquent behavior, Aggressive behavior. Syndrome scale scores were computed by summing individual items. The CBCL consists of 118 items measuring the prevalence and degree of child behavior problems. Items were rated on a three-point scale (0 = Not True; 1 = Somewhat or Sometimes True; 2 = Often or Very True). The CBCL is well known for its robust construct and discriminant validity, as well as its reliability (Achenbach, 1991). In the current study, ratings from each parent were obtained between the time when the child was three to five (wave 1), and twelve to fourteen (wave 4). Cronbach's alphas for Anxious/depressed, Attention problems, Delinquent behavior, Aggressive behavior were .725, .690, .560, and .866 at wave 1 and .801, .785, .750, and .888 at wave 4, respectively.

Smoking onset. Smoking onset was determined by responses on two questions from the Drinking and Drug History Questionnaire (DDHQ; Zucker et al., 1990) that were slightly adapted for youth. The questions were asked during their regular wave 4 (at ages 12, 13, or 14) assessment and also annually at ages 11, 12, 13, and 14 for the majority of adolescents. The questions asked whether adolescents smoked during lifetime and the past 12-month period (i.e., "Have you ever smoked cigarettes?" and "Have you smoked cigarettes during the past 12 months?"). Their response was recoded as "Never," "Once or twice," "Occasionally but not regularly," "Regularly for a while during this year, but not now," and "Regularly now."

Teenagers were grouped into three categories based on the following criteria. First, adolescents who acknowledged their smoking (at least "Once or twice" or more) in any time during ages between 11 and 14 were considered as having experimented with cigarette smoking by age 14 (Smoking-onset; $\underline{n}$ = 79; 28.1% for boys; $\underline{n}$ = 21; 23.9% for

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girls). Second, adolescents who never smoked by age 14 were grouped as "Non-smoker" (n = 114; 40.6% for boys; n = 20; 22.7% for girls). For some adolescents of early birth cohorts, data from later assessments (e.g., wave 5) were available. If they continued to remain "Non-smoker" at age 15 or older, they were then grouped as "Non-smoker." The last category, "Smoking-Onset Unknown" (or smoking-onset remains to be seen) was for those adolescents who have not smoked but younger than 14 years old at the time of the latest assessment (n = 88; 31.3% for boys; n = 47; 53.4% for girls). This category also included a small number of adolescents who never smoked by the last measurement prior to age 14 but whose annual information at age 14 was not available. The decision to create a "Smoking Onset Unknown" category rather than to treat it as missing was made since the category conveys meaningful implications. The higher percentage of girls in the "Smoking Onset Unknown" category reflects their younger birth cohorts so that later assessments have not been completed.

Missing Data Estimation

Although there is no rule of thumb for an acceptable rate of missing data, the current study included any participants who had completed at least two out of four measurements. Missing data were handled at two levels. At the first level, the data on parental smoking and alcoholism were used with missing data in the subsequent analyses of trajectories of parental smoking and alcoholism. The SAS macro, TRAJ, developed by Jones, Nagin, and Roeder (2001) uses all the available data while neither imputing nor deleting the missing data. Once the trajectories of parental smoking and alcoholism were identified, the remaining missing data were imputed separately for parents and adolescent children using Schafer's NORM version 2.03 (Schafer, 2000), the most accessible

implementation program of multiple imputation (MI) method (Graham & Hofer, 2000; Schafer, 1997, 1999).

MI is the technique that substitutes each missing value m times with a value representing a distribution of possibilities in m datasets (Rubin, 1987). MI is superior to other popular procedures of single imputation because it provides a valid basis for computing standard errors of the parameter estimates by treating missing data as an explicit source of random variability (Graham & Hofer, 2000; Rubin, 1987; Schafer, 2001). Utilizing MI for missing data imputation in an analysis usually involves three steps: multiple imputation of m datasets, repeated analyses over m datasets, and combining results from m datasets to obtain a single set of results.

The NORM program used in the current study is originally designed for normally distributed data. However, it is shown to yield good results even with seriously non-normal data (Graham & Schafer, in press; cf. Graham & Hofer, 2000). Under the NORM procedure, data augmentation procedure alternates between the imputation step (I-step), in which missing data are simulated from their conditional distribution given the current estimate of the covariance matrix, and the posterior step (P-step), in which new values are simulated by drawing them from a Bayesian posterior distribution given the current values of the data (Graham & Hofer, 2000; Schafer, 1997, 1999).

In the current study, parental data and adolescent data were imputed separately. The number and percentage of cases with missing values and the matrix of missingness patterns for all variables used in the current study are presented separately for parents and adolescents in Appendix C. Missing data on parental measures were very few (for more information see Table C1 in Appendix C). Parental data needed only 38 iterations for

Expectation Maximization (EM) convergence. The diagnostics on the mean, and the variance and covariance parameters showed that the autocorrelation reached near zero instantly. Following the rule of thumb (i.e., doubling the number of iterations for EM convergence) and the diagnostics, 760 total steps were specified in data augmentation step. Although no more than five multiply imputed datasets are sufficient for most occasions (Rubin, 1987; Schaffer, 1997), conservatively ten datasets were imputed per every 76 steps of iterations for parents in the present study.

The adolescent children's data included parental reports of behavior problems assessed when the target child was between the ages of three and five (wave 1) and again between the ages of 12 and 14 (wave 4). However, a substantial portion of adolescent daughters in the current study had missed data collections at waves 1 and/or 4 (see Table C3 in Appendix C for more information) due to the recruitment design of the UM-MSU Longitudinal Project. Either they were not in the age range to complete wave 4 or they were recruited to the study from wave 2 and on. Therefore, child behavior problems reported by both parents at waves 2 and 3 were also included in the missing data estimation procedure to assure a better solution, although they were not part of the analyses in the current study. This is a generally recommended procedure for imputing missing data because the imputed values do not depend on what is included in the subsequent analyses (Little, 2001; Schafer, 2000).

Adolescent children's data required 1,243 iterations for EM convergence, due in part to the large number of variables included and to a higher proportion of missing information especially for girls. Following the same procedure used for the parental data, 2,600 total steps were selected with an imputed data set per every 260 steps. Tables C2

and C3 in Appendix C present matrices of missingness pattern of the data used in the subsequent analyses for sons and daughters, respectively. Results from daughters were mostly considered exploratory in nature, due to a small sample size ($\underline{N}$ = 88) and a higher rate of missing data (see Table C3 in Appendix C), with the exception of prenatal exposure to maternal smoking. Results from daughters are briefly noted in the following sections when applicable and tables and figures pertaining to daughters are presented in Appendix G.

RESULTS

Results are organized as follows: 1) Intergenerational Transmission of Smoking asks whether and how parental smoking plays a role of a risk factor for children's smoking, 2) A Family History of Alcoholism investigates alcoholism subtypes in relation to early onset of smoking, 3) Early and Concurrent Characteristics of Adolescents with Early Smoking Onset investigates prenatal exposure to maternal cigarette smoking, early temperamental dimensions, and behavioral characteristics across different groups of adolescents with different smoking onset status, and 4) Paths to Early Onset of Smoking investigates direct and indirect paths to early onset of smoking in children. Each of the four results addresses the mechanisms of early smoking onset from slightly unique perspective and methods.

The first two sections of results address the notions of intergenerational transmission of smoking and a familial alcoholism as a shared vulnerability factor for early smoking onset. First, in order to identify subtypes of smoking and alcoholism among parents in the first two sections of Results, a group-based semiparametric modeling approach[4] (Nagin, 1999; Nagin & Trembly, 1999, 2001) was utilized. Unlike hierarchical and latent growth curve modeling analyses that focus on an average growth curve under the assumption that all individuals belong to one homogeneous population, this new approach was designed to identify qualitatively different, prototypical multiple developmental growth patterns. Following identification of the number of groups, posterior probabilities of group membership are estimated for all individuals. Multiple

4 It is also called Latent Class Growth Analysis (LCGA; B. Muthén, 2001).

growth patterns are conventionally dealt with by modeling a growth function separately for a subset of the sample in hierarchical and latent growth curve modeling analyses, based on a priori theories and hypotheses. However, this procedure is limited in the sense that there is no statistical ground to test whether there exist multiple groups and how many subgroups exist (Nagin, 1999).

In the current study, the SAS (SAS Inc., 1999) procedure, TRAJ (Jones et al., 2001) was used to fit semiparametric mixtures[5] of censored[6] normal distributions of parental smoking and alcoholism. Once the identification of group membership for all parents was completed based on posterior probabilities, the current study investigated whether these groups are different on psychosocial measures such as years of education, occupation, conduct problems in childhood, antisocial behavior, and depression. Multivariate Analysis of Variance (MANOVA), followed by subsequent univariate analysis of variance were conducted using S Plus (MathSoft Inc., 1998) and SPSS (SPSS Inc., 1999) software programs.

Second, using the group membership identified in the previous step, Configural Frequency Analysis (CFA; von Eye, 2001a) was conducted to see whether and how parental smoking and alcoholism are related to early smoking onset in offspring. CFA is

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5 A mixture refers to the situation where the measurements of a random variable are taken under two or more different conditions, resulting in the distribution of the mixture of two or more subpopulations. Instead of treating the distribution as bi- or multi-modal, in analysis of mixture models, a number of subpopulations are identified and parameterized so that a relatively simple model can normally be applied to each of subpopulations (Gelman, Carlin, Stern, & Rubin, 1995).

6 Censored data means that the upper and lower limits of the data have been truncated by various reasons when the underlying distribution is normal.

a multivariate method for typological research that involves categorical variables in both exploratory and confirmatory research. Using CFA, researchers ask whether cells contain fewer or more cases than expected from some chance model (von Eye, 1990, 2002). CFA is the only method application of person-centered approaches that shows whether a group of observations (or configuration) does not occur beyond statistical significance (von Eye, personal communication, 2001b). Therefore, CFA application is well-suited for the current research question.

Third, the current study investigated whether adolescents who started smoking by age 14 can be identified based on measures of prenatal exposure to maternal daily cigarette smoking and weekly drinking, child temperament, and child behavioral characteristics. Concurrent behavioral characteristics measured at ages twelve to fourteen were also investigated in relation to their smoking onset status. Multivariate analysis of variance (MANOVA) was conducted using S Plus (MathSoft Inc., 1998) and SPSS (SPSS Inc., 1999).

Fourth, using the selected key factors of intergenerational transmission of smoking, manifest variable structural equation modeling (SEM) analysis was conducted using LISREL program version 8.51 (Jöreskog & Sörbom, 2001). Given that at least a few key variables were either categorical (e.g., smoking onset status) or ordinal (smoking subtypes), other numerical variables were recoded into ordinal variables while maintaining as much information as possible. Due to the ordinal nature of the variables and the moderate sample size in the current study, a limited number of variables were selected to test direct and indirect paths of intergenerational transmission of smoking simultaneously.

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Developmental Patterns of Smoking in Adulthood

A mixture modeling was used to identify distinctive clusters of parental smoking patterns using a customized SAS macro, TRAJ developed by Jones et al. (2001). Parental smoking measured in the current study was censored normal. Smoking behavior in adulthood was modeled by means of a latent variable y_{it}^{*j}, measuring potential for smoking for individual *i*'s age at time *t* given membership in group *j*. Following Nagin (1999)'s notations, for example, a quadratic relationship between smoking and age is expressed:

$$y_{it}^{*j} = \beta_0^j + \beta_1^j Age_{it} + \beta_2^j Age_{it}^2 + \varepsilon_{it}$$

where Age_{it} and Age_{it}^2 are the subject *i*'s age and the square of the age at time *t*, respectively, and ϵ is a residual term assumed to be normally distributed, with the expected mean of zero and constant variance σ^2. The expected value of the latent variable, y_{it}^{*j}, is $y_{it}^{*j} = \beta_0^j + \beta_1^j Age_{it} + \beta_2^j Age_{it}^2$. The expected value of the observed variable, $E(Y_{it}^j)$, assuming group membership *j* observed, is expressed:

$$E(Y_{it}^j) = \Phi_{min}^j S_{min} + \beta^j x_t (\Phi_{max}^j - \Phi_{min}^j) + \sigma(\phi_{min}^j - \phi_{max}^j) + (1 - \Phi_{max}^j) S_{max},$$

where Φ and ϕ denote cumulative normal distribution function and the normal density function, respectively, and S_{min} and S_{max} denote the minimum and maximum possible scores on the measurement scale, respectively (For more information, see Nagin, 1999).

The determination of the optimal model of parental smoking trajectories involved two important issues: 1) determination of the number of groups to compose the mixture; and 2) determination of the order of the growth patterns (e.g., linear, quadratic, cubic,

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etc.). The determination of the number of groups that best describes the data was based on the Bayesian information criterion (BIC), following the lead of D'Unger, Land, McCall, and Nagin (1998) and Nagin (1999). The model with the smallest absolute BIC value is generally selected as the best-fitting model, with BIC rewarding parsimony. In addition to the BIC criteria, two additional criteria were used to determine the optimal number of groups to compose the mixture in the present study: 1) significant parameter estimates for growth terms, and 2) at least 5% of participants in each group. With regard to the order of growth polynomial, the models were specified using a linear growth parameter given that studies of natural history of smoking have reported stability or small drop-offs in adulthood (Anthony & Echeagaray-Wagner, 2000; Chassin et al., 1996b, 2000).

Using these procedures, one-, two-, three-, and four-group models of trajectories of parental smoking were tested for ages 24 to 50. Non-smokers ($\underline{n}$ = 293; 52.8% of the 555 parents in the analyzed sample) were a priori identified and excluded from the analyses. Gender and birth cohort were not meaningful factors when added as covariates; therefore, subsequent analyses were conducted across genders and birth cohorts. Based on the BIC criterion as well as other additional criteria, a three-group model was selected as the best-fitting model. The results are summarized in Tables 2 and 3. Individuals are then assigned to the group that best fits their observed smoking behavior according to the maximum posterior probability of group membership.[7] This model selection procedure resulted in the best-fitting model with adequate sample sizes for subsequent analysis.

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7 The formulas for derivation of likelihood for each individual can be found in Nagin (1999, pp. 156-157).

Table 2

Model Comparisons of Parental Smoking (n = 262)

Model	BIC	Δ BIC
1	-1667.17	
2	-1532.86	134.31
3	**-1530.50**	**2.36**
4	-1530.64	-0.14

Note. Bolded row indicates the model selected. BIC: Bayesian information criterion.

Table 3

Growth Parameter Estimates for Each of Three Smoking Groups

Group	Parameter	Estimate	SE	t
1. Light Smoker (n = 57)	Intercept	3.68	1.08	3.42*
	Linear	-.09	.03	-3.11*
2. Heavy Smoker (n = 186)	Intercept	2.21	.51	4.35*
	Linear	.06	.02	3.87*
3. Heavy-to-Light Smoker (n = 19)	Intercept	9.44	2.01	4.70*
	Linear	-.18	.06	-3.15*

Note. * $p < .05$, 293 of 555 parents (52.8% of the total parents in the sample) were non-smokers. SE = Standard error. There were 25 men and 32 women in the Light smoker category; 105 men and 81 women in the Heavy smoker category; and 13 men and 6 women in the Heavy-to-light smoker category.

Table 3 shows growth parameter estimates for each group, and three distinctive growth patterns of smoking in adulthood are illustrated in Figure 3. Solid lines represent observed means of smoking whereas dashed lines denote expected value of smoking. All parameters were significant in each of three smoking groups. The first group of Light smokers (n = 57; 22.1% of 262 smoking parents; 10% of the 555 smoking and non-smoking parents) was characterized by a lower level of smoking in quantity and the gradual decline throughout adulthood (linear growth parameter = -.09, $p < .05$). Their level of smoking was confined within one to five cigarettes per day.

This group of smokers may also be called "tobacco chippers" who smoke less than five cigarettes per day for a long-term without developing nicotine dependence (Shiffman et al., 1994a, 1994b). It is estimated that five percent (O. F. Pomerleau et al., 1993), or five to ten percent (USDHHS, 1988) of smokers are tobacco chippers or light smokers in the population at large. A "chipper" was originally used to describe casual opiate users who use opiates in moderation for a long term without developing addiction (Shiffman et al., 1994a, 1994b). Based on their history and pattern of smoking, Shiffman and his colleagues speculated whether "chippers" are the "smoking equivalent of a moderate social drinker (Shiffman et al., 1994b)." They smoke cigarettes regularly and they find cigarette smoking reinforcing, but their smoking is often tied to social contexts and positive affect. Furthermore, their cigarette smoking is not driven by dependence to compulsive use.

As expected, the majority of smokers belong to the second group, Heavy smokers (n = 186; 71% of smoking parents; 33.5% of all parents in the sample). They showed a persistent, high level of smoking throughout adulthood, ranging from a half pack per day

to one and a half pack per day, in average. Moreover, their smoking showed a slight increase over years in smoking quantity (linear growth parameter = .06, $p < .05$). This group of smokers may overlap with habitual smokers with nicotine dependence (with and without physiological dependence) based on the DSM-IV criteria (American Psychiatric Association, 1994) in the literature.

The third group ($n = 19$; 7.3% of smoking parents; 3.4% of all parents in the sample) consisted of a smaller number of people whose smoking was indistinguishable from Heavy smokers until early 30s but who either quit smoking or reduced their smoking under one to five cigarettes per day (linear growth parameter = -.18, $p < .05$). Shiffman et al. (1994b) named a small number of smokers who were previously nicotine dependent but could not be distinguished from pure "chippers" based on current pattern of smoking as "converted chippers." Results confirmed the notion of heterogeneity of smokers. However, the results from the current study indicate that after late 20s smokers can be differentiated mostly by the quantity of their cigarette smoking, and that smokers consist of three groups: Heavy, Light, and Heavy-to-light smokers.

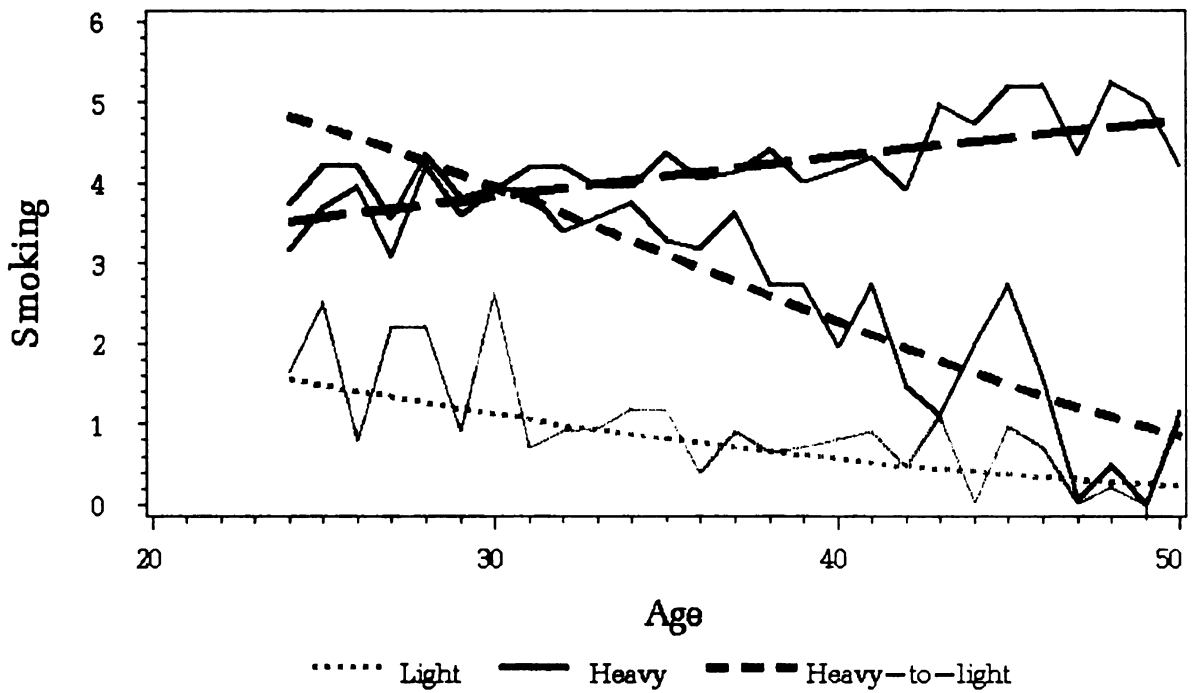

Figure 3.

Three distinctive developmental patterns of smoking in adulthood. Values of the daily smoking: 0 = not at all; 1 = less than one cigarette per day; 2 = one to five cigarettes per day; 3 = one-half pack per day; 4 = one pack per day; 5 = one and one-half packs per day; 6 = two packs or more per day.

Characteristics of Subtypes of Smokers

I next attempted to determine whether smokers of different subtypes differ from each other on dimensions other than smoking, such as educational level, occupation, conduct problems in childhood, antisocial behavior in adulthood, and depression. In addition to smoking subtypes, gender was also investigated as a fixed factor since smoking subtypes were originally generated from one sample pool across men and women.

Multivariate analysis of variance (MANOVA) was repeated ten times for each of ten multiply imputed data sets. Means and standard deviations from each of the ten imputed datasets were averaged, and test statistics such as F statistic and Wilk's λ were reported in range.[8] Main effects of smoking subtype and gender, and the interaction between the two were significant at $p < .05$ (see Table 4). Smoking, gender, and their interaction explained 20-21%, 21-22%, and 3% of the variance, respectively. This part of the MANOVA computation was done using S Plus, version 4.5 (MathSoft Inc., 1998). At the next step, each of seven variables was examined separately using SPSS MANOVA module, version 10 (SPSS Inc., 1999) and the unique contribution of each of three

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8 Averaging parameters of interest is a common practice to obtain a combined set of results from multiply imputed datasets. However, standard errors (and accordingly test statistics) need to be calculated based on both between and within variances of parameters across m datasets (Rubin, 1987; Schafer, 1997). Using the standard error and t statistic calculated, the null hypothesis, a parameter of interest = 0 can be tested as in regression analysis or path analysis (see p. 96 in the present study for more information). However, there is no practical application to obtain a single set of combined results from multiple mean comparisons. Therefore, alternatively, the range of F statistic and Wilk's λ was reported here in the current study.

sources (smoking subtype, gender, and interaction) was investigated utilizing Type III sum of squares.[9] This procedure takes into account that all three factors (smoking subtype, gender, interaction between the two) contribute to each of seven variables, and that a significant F statistic represents unique input of each factor. A descriptive summary including standard deviations for each measure is provided in Tables E1 and E2 in Appendix E.

Post-hoc tests were conducted separately for men and women using the Bonferroni method to protect familywise error rate.[10] Each of ten multiply imputed datasets was serially analyzed, with results combined to a single set of results using Rubin's method (1987) to compute standard error (for more details, see p. 96 in the following section of Results in the present study). Although the Bonferroni method tends to be more conservative than other methods, it was adopted in the present study given the small number of comparisons conducted (three to six comparisons per post-hoc test in the present study) and the reliance on the t distribution for combining results from multiply imputed datasets.

Figures 4 – 10 reveal results of post-hoc tests. Bars that do not share letters in both men and women indicate that means differed at $p < .05$ (a Bonferroni-adjusted alpha per post-hoc test = .05/6 = 0.0083). Among men, Heavy smokers were different from others on years of education, occupational status, conduct problems, antisocial behavior,

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9 Type III sum of squares is a method to divide variance by attributing a unique portion to each source. It does not depend on the entry order of sources. On the other hand, Type I sum of squares divides variance sequentially by the entry order as in hierarchical regression analysis.

10 Familywise error rate is the probability to reject at least one true null hypothesis, where the family refers to the collection of all pairwise null hypotheses.

and both current and worst-ever depression. Heavy smokers had a fewer year of education, a lower level of occupational status, a higher level of conduct problems and antisocial behavior, and a higher level of both current and worst-ever depression, compared to non-smokers. On the other hand, on all measures, neither Light nor Heavy-to-light smokers differed from non-smokers. Among women, Heavy smokers had a fewer year of education, a lower occupational status, a higher level of conduct problems and antisocial behavior, and worst-ever depression, compared to non-smokers. Heavy smoking women had a fewer year of education than Light smokers. With the exceptions of conduct problems and antisocial behavior that smokers of all types showed higher levels but no differences among them, neither Light nor Heavy-to-light smokers differed from non-smokers.

The results suggest that Heavy smoking in both men and women were associated with a higher level of psychopathologies and a lower level of socioeconomic status. Although, the direction of the relationship is not clear, it is plausible that these men and women pose smoking-specific as well as common risks for their children. In addition, there was a "dose-response" pattern between smoking subtypes and variables tested, albeit insignificant statistically: Heavy smokers, followed by Light and Heavy-to-light smokers manifested an elevated level of antisocial behaviors and depression, and a more disadvantageous socioeconomic status.

Table 4

MANOVA Results on Demographic Characteristics and Psychopathologies by Smoking Subtype and Gender

	Source		
	Smoking subtype	Gender	Interaction
Multivariate F(7, 545)	19.80* – 20.57*	20.63* – 21.93*	2.61* – 3.09*
Wilk's λ	.791* – .797*	.780* – .791*	.962* – .968*

	Tests of unique contribution			
Variable	Smoking subtype	Gender	Interaction	Total
Education in years	25.74* – 27.47*	3.18 – 3.42	.08 – .14	12.02* – 12.88*
Occupation	13.73* – 14.67*	4.83* – 5.06*	1.97 – 2.56	9.09* – 9.77*
Conduct problems in childhood	24.61* – 26.50*	1.10 – 1.50	3.07* – 3.39*	18.38* – 19.30*
Antisocial behavior in adulthood	20.66* – 21.56*	14.92* – 16.36*	4.88* – 5.77*	23.27* – 24.47*
Hamilton depression – current	5.88* – 6.98*	2.73 – 4.14*	.52 – .96	3.61* – 4.12*
Hamilton depression – worst	6.97* – 8.36*	4.46* – 6.32*	.21 – .56	4.28* – 4.90*
Beck depression index	1.79 – 2.43	3.99* – 5.41*	.43 – .69	1.59 – 2.06*

Note. * $p < .05$. Degrees of freedom for univariate F tests were 3 and 547 for smoking subtype and interaction, 1 and 547 for gender 7 and 547 for total model. The model explained 13.8%, 10.8%, 19.5%, 23.4%, 4.8%, 5.6%, and 2.3% of variance for each of seven measures, respectively.

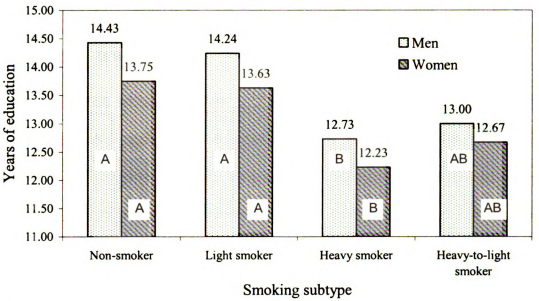

Figure 4.

Years of education and smoking subtype. In both men and women, means that do not share letters differ at $p < .05$ using the Bonferroni method.

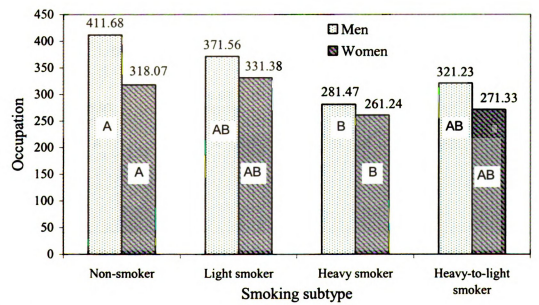

Figure 5.

Occupational status and smoking subtype. In both men and women, means that do not share letters differ at $p < .05$ using the Bonferroni method.

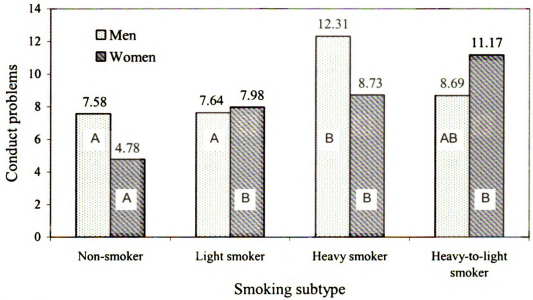

Figure 6.

Childhood conduct problems and smoking subtype. In both men and women, means that do not share letters differ at $p < .05$ using the Bonferroni method.

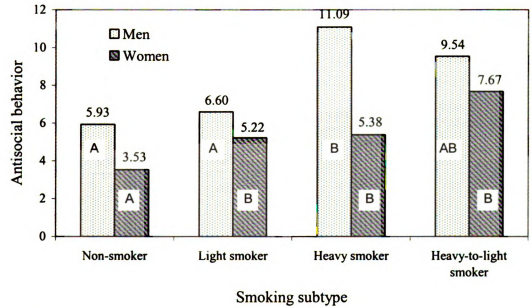

Figure 7.

Adulthood antisocial behavior and smoking subtype. In both men and women, means that do not share letters differ at $p < .05$ using the Bonferroni method.

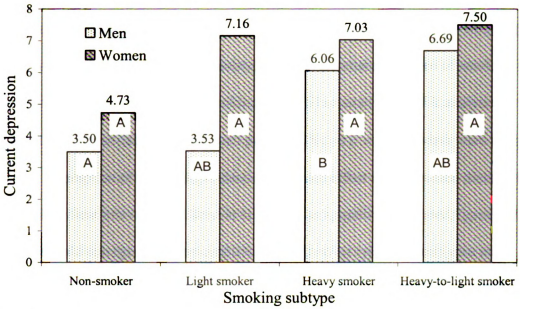

Figure 8.

Current depressive symptoms and smoking subtype. In both men and women, means that do not share letters differ at $p < .05$ using the Bonferroni method.

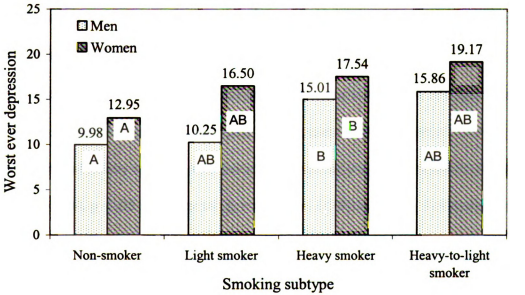

Figure 9.

Worst-ever depressive symptoms and smoking subtype. In both men and women, means that do not share letters differ at $p < .05$ using the Bonferroni method.

Parental Smoking Subtype and Early Smoking Onset in Adolescent Children

Parental smoking was examined in relation to adolescent children's smoking onset. Maternal (M) and paternal (P) subtypes of developmental pattern of smoking were crossed with adolescent children's smoking onset (S) separately in CFA analysis, yielding a 4 × 3 cross-tabulation for each pair, M × S and P × S. The data were analyzed under the assumption of total independence (i.e., main effect model) which dictates that all three classifications are not related at all. If the assumption is violated, types and/or antitypes should emerge. If the assumption is met, neither types nor antitypes are expected. Lehmacher's test (L; Lehmacher, 1981) was used for significance testing of types and antitypes with a Bonferroni-adjusted alpha level ($\alpha^* = 0.05/12$ cells = 0.004167). The Bonferroni adjustment of alpha was adopted to control for inflated alpha first due to, simultaneous multiple testing of types and antitypes and second, the mutual dependency of tests (see von Eye, 1990, 2002).

This analysis resulted in Pearson's $X^2 = 21.08$ for $df = 6$; $p < .05$ for maternal smoking subtypes (M × S), and Pearson's $X^2 = 23.37$ for $df = 6$; $p < .05$ for paternal smoking subtypes (P × S). Types and antitypes were found in the same configurations for maternal and paternal smoking subtypes. In both maternal and paternal smoking subtypes, parental Heavy smoking was associated with early onset smoking in adolescent children, $L = 2.84$, $p < .05$ for maternal; $L = 4.27$, $p < .05$ for paternal. Fewer cases than expected whose smoking onset was unknown were found among children of Heavy smokers, $L = -2.91$, $p < .05$ for maternal; $L = -3.36$, $p < .05$ for paternal. Among children of non-smoking parents, more cases were found than expected of unknown smoking onset, $L = 2.66$, $p < .05$ for maternal; $L = 2.97$, $p < .05$ for paternal. Conversely, fewer

cases than expected started experimenting with cigarettes, $\underline{L} = -4.05$, $\underline{p} < .05$ for maternal; $\underline{L} = -3.75$, $\underline{p} < .05$ for paternal.

The results of pair-wise CFA supported the notion of heterogeneous risks of parental smoking for adolescent children's smoking onset. In particular, parental long-term heavy smoking presented an elevated risk for their children's early smoking onset. In contrast, neither Light nor Heavy-to-light smoking parents elevated the chances of their children's early smoking onset. Parental smoking had the same effects on children's smoking onset, regardless of whether a smoking parent was a mother or father.

At the next step, cumulative risks of parental smoking were investigated by examining maternal and paternal smoking subtypes altogether. Parental smoking subtypes were simultaneously crossed with adolescent children's smoking onset (M × P × S in 4 × 4 × 3 cross-tabulation). Due to a small number of parents in the Heavy-to-light subtype (six mothers and thirteen fathers), expected frequencies in a couple of cell configurations fell below 0.5 in this analysis. Therefore, "Heavy-to-light" category was collapsed with "Heavy" in subsequent analyses.[11] Both maternal (M) and paternal (P) smoking patterns were crossed with adolescent son's smoking status (S), yielding the 3 × 3 × 3 cross-classification (M × P × S). Table 5 shows the observed and expected frequencies and types and antitypes from CFA analysis for boys, and Figure 10 illustrates the three-way associations among maternal and paternal smoking, and smoking onset of

11 Alternatively, "Heavy-to-light" category was collapsed with "Light" category. Results were almost identical to those results reported in the text, with the exception of one antitype found in cell configuration 131.

adolescent boys[12]. Again, the data were analyzed under the assumption of total independence (i.e., main effect model).

As expected, the data on sons showed a poor fit. The Pearson $X^2 = 80.81$, for $df = 20$, $p = .00$, suggests that there were associations above and beyond main effects. Four types and three antitypes emerged. Types were found in configurations 111, 113, 232, and 332. The first two types (i.e., 111 and 113) indicate that there were more cases than expected of non-smoking parents with adolescent boys who have never smoked by age 14 or whose smoking onset remains to be seen, respectively. The latter two types (232 and 332) suggest that maternal smoking, regardless of its type, when paired with Heavy smoking on the paternal side was more often associated with early smoking onset in offspring. Antitypes were found in configurations 133 and 313, indicating that among adolescent boys whose smoking onset was unknown, fewer cases than expected were found when they had one Heavy smoker parent and one non-smoker parent.

Figure 10 also captures the associations between subtypes of smoking and early smoking onset in a mosaic display.[13] A glance at Figure 10 reveals that smoking

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12 Data on daughters showed a similar pattern to that of sons in the sense that there were more cases of a combination of two Heavy smoking parents paired with daughters who started smoking (Configuration 332), and of a combination of non-smoking parents with non-smoking daughters (Configuration 111; for more information see Table G1 in Appendix G).

13 The mosaic display, proposed by Hartigan and Kleiner (1981, 1984) is a graphical method for examining cross-tabulated data. A mosaic, defined as the collection of tiles or rectangles for the n-way contingency table is formed by dividing a square *n* times vertically and then horizontally until all cell configurations are displayed. All mosaic displays in the current study were generated using MOSAICS developed for the SAS software (SAS Institute, 1999) by Friendly (1992, 1994). More detailed information

subtypes of parents were strongly associated in a way that not only was smoking of one parent was related to the other parent's smoking, but smoking type was also related to the other parent's smoking type. For example, among the three clustered columns of maternal smoking, the two columns on the right represent smoking mothers. Of smoking mothers, only a fraction were paired with a non-smoking spouse (the bottom clusters of rectangles). Of Heavy smoking mothers (the column of clusters of rectangles on the right), the majority of spouses were also Heavy smokers (the elongated oblong in the middle of the clusters in the upper right corner). In addition, Figure 10 exhibits that adolescent sons with early smoking onset were more often found in families with both parents being Heavy smokers or with Heavy smoking father and Light (and Heavy-to-light) smoking mothers (i.e., Types 332 and 232; see the two hatch-marked oblongs in the upper right corner). On the other hand, in families where neither parents smoked, more adolescent boys than expected were found to have never smoked by age 14 or to remain non-smoker (i.e., Types 111 and 113; see the two hatch-marked oblongs in the lower left corner). Two antitypes marked by cross-hatched rectangles (at configurations 133 and 313) suggest that fewer than expected were cases of one Heavy smoking parent and the other non-smoking parent with sons whose smoking status was unknown or remains to be seen.

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about utilizing mosaic displays for CFA is available elsewhere (Mun, von Eye, Fitzgerald, & Zucker, 2001).

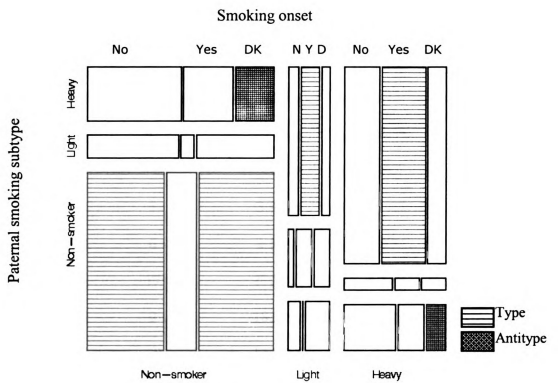


Figure 10.

Parental smoking subtypes and adolescent children's smoking onset. Yes = Smoking onset by age 14, No = Non-smoker, DK = Smoking onset unknown or remain to be seen.

Table 5

Configurations of Parental Smoking Patterns and Early Smoking Onset Among Adolescent Sons

MPS	Obs. Freq.	Exp. Freq.	L	Type/Antitype
111	46	30.88	3.79*	Type
112	18	20.59	-.75	
113	45	23.65	5.84*	Type
121	7	5.94	.52	
122	1	3.96	-1.67	
123	6	4.55	.78	
131	17	27.32	-2.67	
132	9	18.21	-2.75	
133	7	20.92	-3.95*	Antitype
211	2	6.14	-1.93	
212	0	4.09	-2.24	
213	4	4.70	-.36	
221	1	1.18	-.17	
222	3	.79	2.57	
223	3	.90	2.28	
231	5	5.43	-.21	
232	9	3.62	3.10*	Type
233	4	4.16	-.09	

(table continues)

Table 5 (Continued)

Configurations of Parental Smoking Patterns and Early Smoking Onset Among Adolescent Sons

MPS	Obs. Freq.	Exp. Freq.	L	Type/Antitype
311	8	16.43	-2.58	
312	4	10.95	-2.48	
313	3	12.58	-3.23*	Antitype
321	2	3.16	-.72	
322	1	2.11	-.81	
323	1	2.42	-.98	
331	23	14.53	2.70	
332	29	9.69	7.20*	Type
333	12	11.13	.31	

Note. M = maternal smoking pattern; P = paternal smoking pattern; S = smoking onset by adolescent boys. Numerals in MPS column represent ordered triples of variable categories. Response categories for parental smoking were 1 = Non-smoker, 2 = Light/Heavy-to-light smoker, and 3 = Heavy smoker for parental smoking, and options for adolescent smoking onset were 1 = Never smoked, 2 = Smoked by age 14, and 3 = Smoking-onset unknown. L stands for Lehmacher's test statistic (1981); Bonferroni-adjusted alpha, $\alpha^* = 0.00185$ was used; * significant at $\alpha^* = 0.00185$.

The results indicate that parental smoking poses different levels of risks for their children's smoking onset. Smoking type appears to be a very useful concept for studying parental smoking as a risk factor for adolescent smoking onset. Long-term Heavy smoking by parents, regardless of whether it was maternal or paternal was tied to early smoking onset among adolescent children. It was well demonstrated in pair-wise associations of adolescent children's smoking onset with maternal and paternal smoking subtype. However, when both parental smoking subtypes in two-parent families were crossed with children's smoking onset it was the presence of both smoking parents that sufficiently elevated the likelihood of early smoking onset among adolescent children. Although one spouse's smoking was highly related to the other's smoking, parental smoking did not influence sons' smoking onset above and beyond statistical significance, when only one parent smoked. There also appears to be specificity of parental smoking in the relation to early onset of smoking in adolescent children. Whereas Light smoking by fathers did not pose much risk, Light smoking by mothers, was a significant risk factor when linked to paternal Heavy smoking.

A FAMILY HISTORY OF ALCOHOLISM AS A RISK FACTOR

Developmental Patterns of Alcoholism

Natural developmental patterns of alcoholism were modeled in the present study using the same procedures as in the analysis of parental smoking trajectories. Males and females were analyzed separately for a number of theoretical and empirical reasons. First, in the literature of alcoholism, the etiology of women's alcoholism is considered to be somewhat different from that of men, although it is uncertain as to what extent as well as to what kind (Babor et al., 1992; Cloninger, 1987; Fitzgerald, Zucker, Puttler, Caplan, & Mun, 2000; Zucker, 1987; Zucker et al., 2000; Zucker et al., 1995). Second, the age range covered for men and women in the current study was different, with women's alcoholism documented over a shorter span of adulthood (e.g., ages 14 to 49 for women versus 14 to 54 for men). Separate analysis by gender was necessary in the context of unequal end point of observations since the prevalence of both alcoholism and drinking tend to dwindle with increasing age (Anthony & Echeagaray-Wagner, 2000; Zucker et al., 2000) and therefore, it was likely that the different end point of data observations for men and women prevent one from revealing the true developmental patterns of alcoholism in adulthood, if analyzed in one sample. Third, men and women in the ongoing larger UM-MSU project were recruited based on different criteria. Women's alcoholism was neither a requirement nor a basis for exclusion, whereas alcoholic men were deliberately recruited in the study. Fourth, gender was a significant covariate when all participants were pooled and analyzed together in the initial TRAJ procedure.

Because the natural courses of alcoholism and alcohol use show patterns of gradual decline after peaking during the twenties (Anthony & Echeagaray-Wagner, 2000;

Zucker et al., 2000), the models were specified using linear and quadratic growth parameters. One-, two-, and three-group models of trajectories of alcoholism were tested separately for men and women. Non-alcoholic men (n = 73; 26.5% of 275 men in the sample) and women (n = 171; 61.1% of 280 women in the sample) were a priori identified and excluded in the subsequent analyses. Birth cohort was not a meaningful factor when added as a covariate; therefore, subsequent analyses were conducted across birth cohorts. A two-group model was selected as the best-fitting model for both men and women. The results are summarized in Table 6. Table 7 shows growth parameter estimates for each group and Figures 11 and 12 depict distinctive developmental patterns of alcoholism for men and women in adulthood, respectively. Solid lines represent observed means of alcoholism whereas dashed lines denote predicted value of alcoholism diagnosis.

Table 6

Model Comparisons of Parental Alcoholism

Men (n = 202)			Women (n = 109)		
Model	BIC	Δ BIC	Model	BIC	Δ BIC
1	-1976.79		1	-944.98	
2	**-1885.94**	**90.85**	2	**-899.67**	**45.31**
3	-2221.47	-335.53	3	-957.70	-58.03

Note. Bolded row indicates the model selected. BIC: Bayesian information criterion.

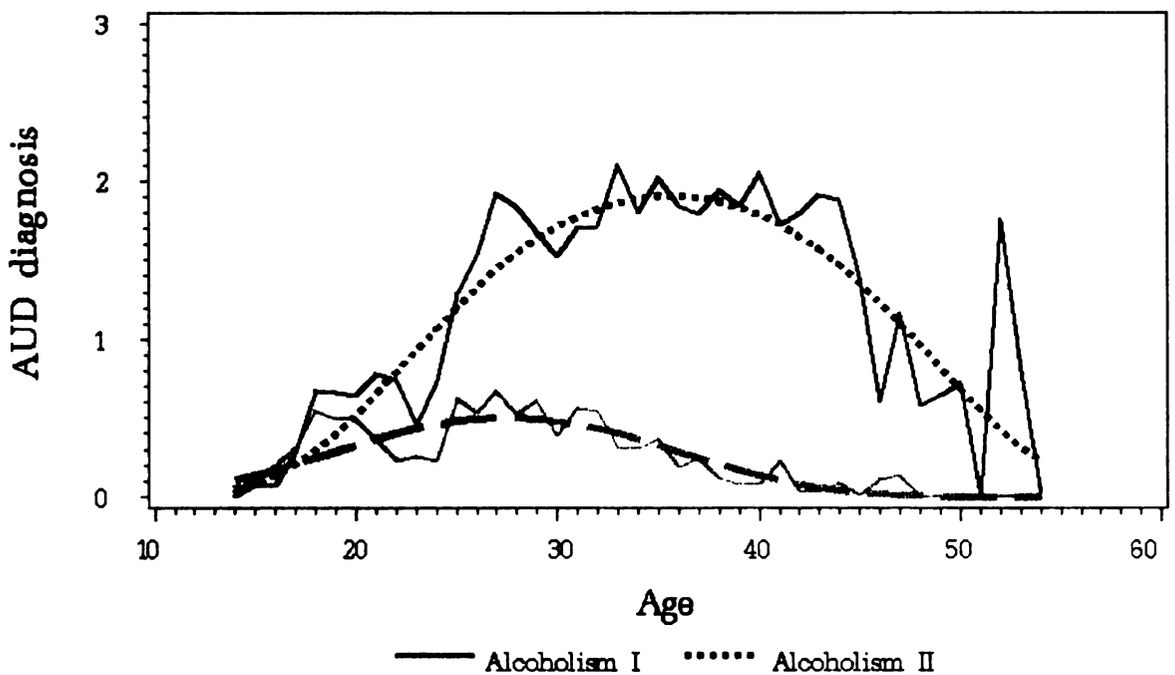


Figure 11.

Developmental patterns of alcoholism among men. AUD diagnosis = Alcohol use disorder diagnosis. Values of AUD diagnosis: 0 = No diagnosis; 1 = Alcohol abuse; 2 = Alcohol dependence without physical dependence; 3 = Alcohol dependence with physiological dependence.

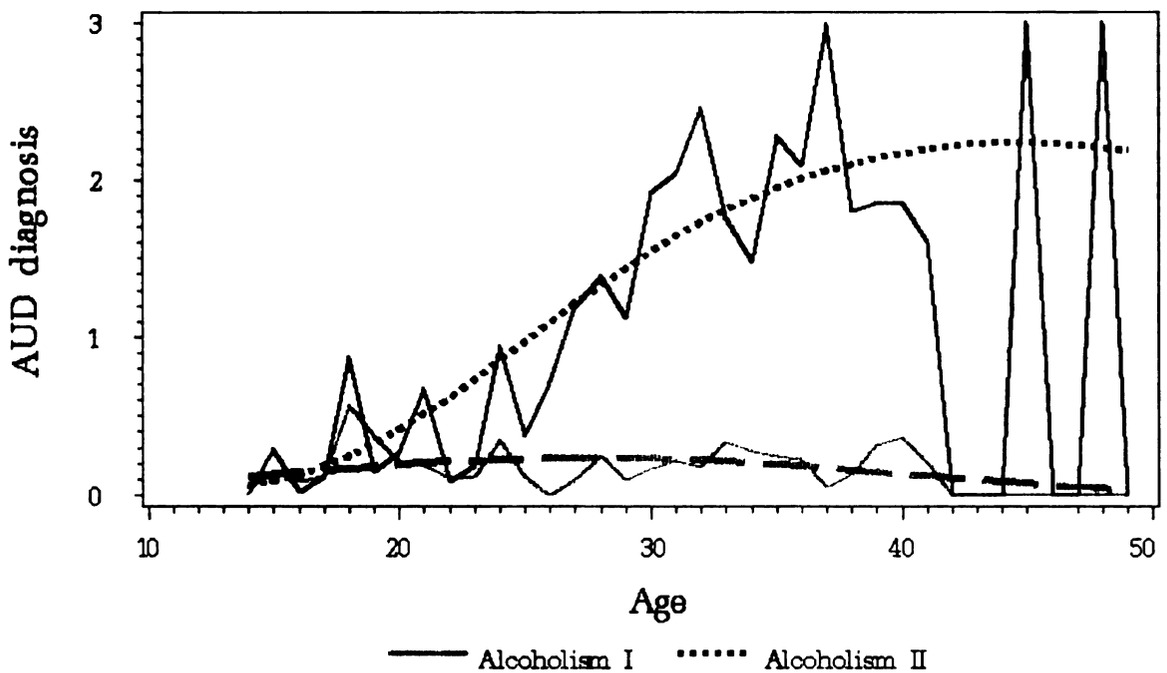


Figure 12.

Developmental patterns of alcoholism among women. AUD diagnosis = Alcohol use disorder diagnosis. Values of AUD diagnosis: 0 = No diagnosis; 1 = Alcohol abuse; 2 = Alcohol dependence without physiological dependence; 3 = Alcohol dependence with physiological dependence.

Table 7

Growth Parameter Estimates for Each of Two Alcoholisms

Group	Parameter	Estimate	SE	t
Men (n = 202)				
1. Alcoholism I (n = 103)	Intercept	-21.01	2.80	-7.51*
	Linear	1.29	.22	5.88*
	Quadratic	-.02	.00	-5.68*
2. Alcoholism II (n = 99)	Intercept	-30.30	2.63	-11.50*
	Linear	1.88	.18	10.24*
	Quadratic	-.03	.00	-8.98*
Women (n = 109)				
1. Alcoholism I (n = 79)	Intercept	-12.93	3.00	-4.32*
	Linear	.52	.22	2.35*
	Quadratic	-.01	.00	-2.34*
2. Alcoholism II (n = 30)	Intercept	-24.37	5.86	-4.16*
	Linear	1.31	.46	2.86*
	Quadratic	-.01	.01	-1.78*

Note. * $p < .05$. 73 (13.2%) of 275 men and 171 (30.8%) of 280 women never met a positive alcoholism diagnosis. SE = Standard error.

In both men and women, two types of developmental patterns of alcoholism emerged as expected from the literature of alcoholism. All linear and quadratic growth parameters were significant (see Table 7). The first group of alcoholic men (Alcoholism I, $\underline{n}$ = 103; 51.0% of alcoholic men; 37.5% of all 275 men in the sample) showed an idiosyncratic developmental pattern characterized by a less severe kind of alcoholism diagnosis (i.e., alcohol abuse) over the course of adulthood, with a peak at late 20s and gradual decline thereafter. The second group of alcoholic men (Alcoholism II, $\underline{n}$ = 99; 49.0% of alcoholic men; 36% of all men in the sample) revealed a developmental course characterized by a severe type of alcoholism diagnosis (i.e., alcohol dependence) for the most of adulthood life span that peaked at mid 30s but gradually declined afterwards (see Figure 11).

Among women, the first group of alcoholic women (Alcoholism I, $\underline{n}$ = 79; 72.5% of 109 alcoholic women; 28.2% of all 280 women in the sample) showed a pattern of alcoholism that was confined within the diagnosis of alcoholism abuse. Although there was no sharp peak or drop-off in their alcoholism pattern, the pattern of alcoholism diagnosis at ages 14 and 49 for this group of women showed a combined flat shape of linear (.52, $\underline{p} < .05$) and quadratic (-.01, $\underline{p} < .05$) components. The second group of alcoholic women (Alcoholism II, $\underline{n}$ = 30; 27.5% of 109 alcoholic women; 10.7% of all 280 women in the sample) revealed a developmental course characterized by a severe diagnosis of alcoholism over time with a slight decline in 40s (-.01, $\underline{p} < .05$; one-tailed), without a clear drop-off but as illustrated in Figure 12. It can be attributed to the scarcity of data observations after age 40 among women, causing relatively large standard error as indicated by the fluctuations after age 40 depicted in Figure 12. It remains to be seen

whether Alcoholism II among women tapers off after late 40s from future follow-up studies.

Although there are existing terms for the subtypes of alcoholism (e.g., Types I and II (Cloninger, 1987), Types A and B (Babor et al., 1992), and Antisocial alcoholism and Non-antisocial alcoholism (Zucker et al., 1996), Alcoholism I and Alcoholism II were used throughout the present study. The rationale is as follows. In theoretical and empirical studies of typology of alcoholism, different samples (and populations) of alcoholics as well as various methods were used to derive alcoholism subtypes. Although there are some convergence in the literature in that one type (Type II, Type B, and Antisocial alcoholism) is generally regarded as a more severe expression of alcoholism than the other, with other co-active psychopathologies and a denser family history of alcoholism, it is not clear that, to what extent, the two types of alcoholics in the present study are equivalent to the types of alcoholism in the extant literature. Therefore, instead of adopting the existing terminology, Alcoholisms I and II were used in the current study to differentiate the two kinds of developmental patterns of alcoholics.

Characteristics of Subtypes of Alcoholism.

Since subtypes of alcoholism were derived separately for men and women, they were separately tested on the following measures: Education in years, occupational status, conduct problems in childhood, antisocial behavior in young adulthood, and depression. In both groups of men and women, alcoholism subtype was a significant factor (see Table 8). It explained approximately 25% of the variance in both groups of men and women (Wilk's lambda = .764 – .775; $p < .05$ for men, and .742 – .761; $p < .05$ for women). Univariate analysis on each of the seven measures revealed that in all

measures with the exception of self-reported depression, there was a group difference across subtypes of alcoholism for men (see Table 8 and Figures 13 – 19). As for women, with the exception of occupational status, alcoholism subtype differentiated developmental patterns of alcoholism (see Table 8 and Figures 13 – 19).

Post-hoc tests were conducted separately for men and women using the Bonferroni method. Ten multiply imputed datasets were analyzed separately and then results were combined following the previous procedure used in post-hoc tests of smokers. In both men and women, bars that do not share letters indicate that means differed at $p < .05$. Among men, alcoholics had a fewer year of education, a lower occupational status, a higher level of conduct problems, antisocial behavior, and worst-ever depression. Among alcoholic men, men with Alcoholism II were associated with a higher level of antisocial behavior and depression, compared to Alcoholism I. Among women, alcoholic women had a higher level of conduct problems childhood and antisocial behavior, and worst-ever depression, compared to non-alcoholics. Among alcoholic women, women with Alcoholism II had a higher level of conduct problems and antisocial behavior, compared to women with Alcoholism I.

Table 8

MANOVA Results on Demographic Characteristics and Psychopathologies of Two Alcoholisms for Men and Women

	Men	Women
Multivariate F	11.07* – 11.76*	12.17* – 13.53*
Wilk's Λ	.764* – .775*	.742* – .761*
Variable	Univariate analysis	
Education in years	10.55* – 11.69*	3.24* – 3.39*
Occupation	9.28* – 10.22*	.39 – .47
Conduct problems in childhood	17.78* – 19.21*	29.52* – 34.16*
Antisocial behavior in adulthood	29.08* – 30.72*	29.99* – 33.54*
Hamilton depression – current	9.02* – 10.00*	4.00* – 4.81*
Hamilton depression – worst	14.12* – 15.92*	6.78* – 8.39*
Beck depression index	2.55 – 3.22*	4.44* – 6.00*

Note. * $p < .05$. Degrees of freedom for multivariate analysis of variance for men and women were 1, 273 and 1, 278, respectively; degrees of freedom for univariate F for men and women were 2, 272 and 2, 277, respectively. Alcoholism subtype explained 7.7%, 6.8%, 12.0%, 17.9%, 6.5%, 10.0%, and 2.1% of variance respectively for each of seven measures for men. Alcoholism subtype explained 2.4%, 0%, 18.6%, 18.7%, 3%, 5.1%, and 3.8% of variance respectively for each of seven measures for women.

Figure 13.

Years of education and alcoholism subtype. In both men and women, means that do not share letters differ at $p < .05$ using the Bonferroni method.

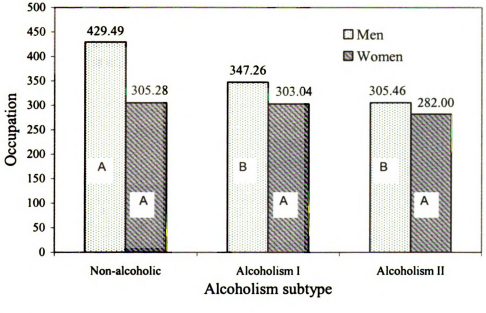


Figure 14.

Occupational status and alcoholism subtype. In both men and women, means that do not share letters differ at $p < .05$ using the Bonferroni method.

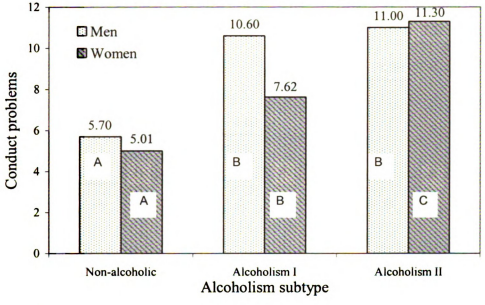


Figure 15.

Childhood conduct problems and alcoholism subtypes. In both men and women, means that do not share letters differ at $p < .05$ using the Bonferroni method.

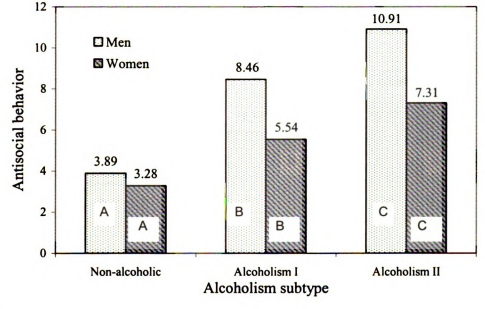


Figure 16.

Adulthood antisocial behavior and alcoholism subtypes. In both men and women, means that do not share letters differ at $p < .05$ using the Bonferroni method.

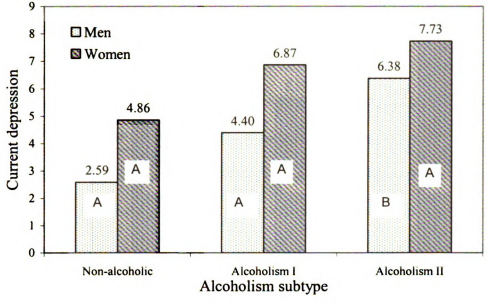


Figure 17.

Current depressive symptoms and alcoholism subtype. In both men and women, means that do not share letters differ at $p < .05$ using the Bonferroni method.

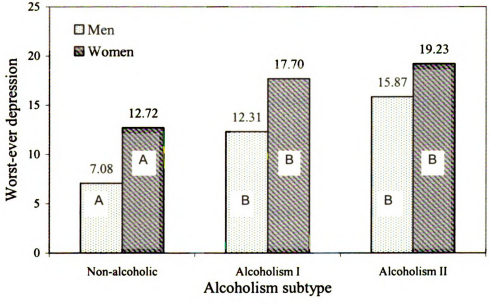


Figure 18.

Worst-ever depressive symptoms and alcoholism subtype. In both men and women, means that do not share letters differ at $p < .05$ using the Bonferroni method.

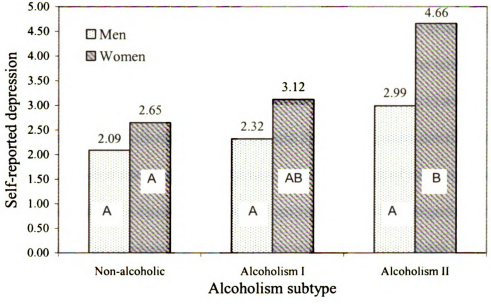


Figure 19.

Self-reported depressive symptoms and alcoholism subtype. In both men and women, means that do not share letters differ at $p < .05$ using the Bonferroni method.

Commonality Between Subtypes of Smokers and Alcoholics

The commonality between subtypes of smokers and subtypes of alcoholics was investigated using a two-sample CFA with the z-approximation of the binominal test (z^*). The major purpose of the two-sample CFA analysis was to see 1) whether smoking types were associated with alcoholism types, and 2) whether gender discriminated the relationship between smoking and alcoholism. Four categories of smoking types (S) were crossed with three categories of alcoholism types (A). This categorization scheme yielded the 4 × 3 cross-classification (S × A) for men and women. Table 9 and Figure 20 show the observed and expected frequencies and types and antitypes from CFA analysis for both men and women. Following a two-sample CFA, I subsequently analyzed the data under the assumption of total independence and Lehmacher's test was used for significance testing of types and antitypes with a Bonferroni-adjusted alpha level ($\alpha^* = 0.0042$).

As expected, the data showed a poor fit, Pearson $X^2 = 69.01$, for $df = 6$, $p = .00$ for men; Pearson $X^2 = 37.38$, for $df = 6$, $p = .00$ for women, suggesting associations above and beyond main effects. Two types and two antitypes emerged for both men and women at the same configurations. Types were found at configurations 11 and 33 while antitypes were found at configurations 13 and 31. The results indicated that regardless of gender, there were more cases of neither smoker nor alcoholic than expected (Configuration 11; a hatch-marked rectangle in the lower left corner in Figure 20) and that Heavy smokers were more often than expected Alcoholism II (Configuration 33; a hatch-marked rectangle in the upper right corner in Figure 20). Antitypes indicate that cases of Heavy smokers who were non-alcoholic (Configuration 31: a cross-hatched

rectangle in the lower right corner) and cases of non-smokers with Alcoholism II (Configuration 13: a cross-hatched rectangle in the upper left corner) were found less frequently than expected.

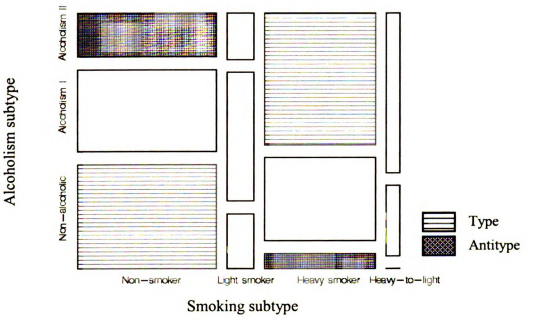
In addition, a two-sample CFA test showed that men and women had unequal number of cases in five configurations (see Table 9). Five discrimination types (DT) were found to differentiate men from women. More cases of women than men were found in configurations 11, 21, and 31, while more observations of men than women were found in configurations 13 and 33. In more detail, there were more women than men in the categories of Non-smoker and Non-alcoholic, $\underline{z}^* = -4.63$, $\underline{p} < .05$ (Configuration 11), of Light smoking and Non-alcoholic, $\underline{z}^* = -2.91$, $\underline{p} < .05$ (Configuration 21), and of Heavy smoking and Non-alcoholic, $\underline{z}^* = -4.54$, $\underline{p} < .05$ (Configuration 31). On the other hand, there were more cases of men than women in the categories of Heavy smoking with Alcoholism II, $\underline{z}^* = 4.63$, $\underline{p} < .05$ (Configuration 33) and of Non-smoker with Alcoholism II, $\underline{z}^* = 3.56$, $\underline{p} < .05$ (Configuration 13).

The discrepancy between men and women can also be shown in Figure 20. Although types and antitypes were found at the exactly same locations for men and women, the number of cases (i.e., the size of rectangle) was visibly and statistically different for men and women in the five configurations identified as discrimination type (DT) above. For example, a configuration 31 (a cross-hatched rectangle in the bottom right) can be interpreted as follows: Although there were fewer cases than expected of Heavy smokers who were non-alcoholic in both men and women, these rare cares were observed more often in women than men. Results illustrate that an overall pattern of

association between smoking and alcoholism holds true for both men and women, with some gender-specific characteristics.

The discrepancies between men and women in the observed frequencies of two-way associations in the present study were not due to the recruitment criteria; one-way marginals of both alcoholism types and smoking subtypes were taken into consideration in examining two-way associations across gender. The results confirm the well-known association between alcoholism and smoking. Furthermore, the results support the notion that the nature of the relation between alcohol and tobacco-related phenomena may depend on levels of involvement with alcohol and cigarette smoking (use versus dependence), with dependence more linked to two specific but related factors (Prescott & Kendler, 1995). The common thread between Alcoholism II and Heavy smoking in the present study was dependency. Unique pathways for each of these dependences as well as the related risk factors and mechanisms remain to be studied.

Men



Women

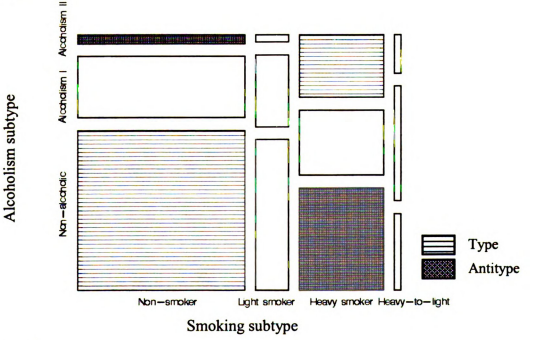


Figure 20.

Associations among subtypes of smoking and alcoholism

Table 9

Associations Among Subtypes of Smokers and Alcoholics

	Men (N = 275)				Women (N = 280)				
SA	OF	EF	L	T/A	OF	EF	L	T/A	DT
11	60	35.04	6.81*	Type	112	98.33	3.38*	Type	DT
12	47	49.44	-.61		43	45.43	-.65		
13	25	47.52	-5.65*	Antitype	6	17.25	-4.39*	Antitype	DT
21	6	6.64	-.30		21	19.54	.56		DT
22	14	9.36	2.01		10	9.03	.41		
23	5	9.00	-1.75		1	3.43	-1.47		
31	7	27.87	-5.86*	Antitype	36	49.47	-3.63*	Antitype	DT
32	38	39.33	-.34		23	22.85	.04		
33	60	37.80	5.73*	Type	22	8.68	5.67*	Type	DT
41	0	3.45	-2.22		2	3.66	-1.41		
42	4	4.87	-.51		3	1.69	1.20		
43	9	4.68	2.55		1	.64	.48		

Note. S = smoking subtype; A = alcoholism subtype. Numerals in SA column represent ordered doubles of variable categories. Response categories for smoking were 1 = Non-smoker, 2 = Light smoker, 3 = Heavy smoker, and 4 = Heavy-to-light smoker. Options for alcoholism subtype were 1 = Non-alcoholic, 2 = Alcoholism I, and 3 = Alcoholism II. OF = Observed frequency, EF = Expected frequency, T/A = Presence of Type or Antitype, L = Lehmacher's test statistic (1981), DT = Discrimination type. Bonferroni-adjusted alpha, $\alpha^* = 0.0042$ was used; * significant at $\alpha^* = 0.00185$.

Parental Alcoholism Subtypes and Early Smoking Onset in Adolescent Children

To address whether parental alcoholism subtypes are associated with adolescent children's smoking onset, frequencies of parental alcoholism and smoking onset were examined. Three categories of maternal (M_A) and paternal (P_A) alcoholism patterns were crossed with adolescent children's smoking status (S). This cross-tabulation yielded the $3 \times 3 \times 3$ cross-classification ($M_A \times P_A \times S$)[14]. Table 10 shows the observed and expected frequencies and types and antitypes from CFA analysis for sons. The same assumption of total independence, and the same statistical procedure used in the MPS data were adopted. Adolescent sons' data showed a poor fit, Pearson $X^2 = 60.73$, for $df = 20$, $p = .00$, suggesting that there were associations among the three classifications. Two types and one antitype emerged (see also Figure 21). Types were found in cell configurations 111 and 332. Results indicated that there were more cases of non-alcoholic parents whose adolescent son has not tried smoking (Configuration 111; a hatch-marked rectangle in the lower left corner), and that there were more cases of Alcoholism II by both parents whose adolescent son started smoking cigarettes by age 14 (Configuration 332; a hatch-marked oblong in the upper right corner). There was one antitype at configuration 133 indicating that cases of adolescents whose smoking onset

---

14 Paternal alcoholism and maternal alcoholism were separately examined in their associations with smoking onset among adolescent children (3×3 cross classification). Results revealed that maternal alcoholism subtypes were not associated with children's smoking onset (Pearson' $X^2 = 2.11$, $p = .72$). However, paternal alcoholism subtypes were related to children's smoking onset (Pearson' $X^2 = 13.79$, $p < .05$) and one type and one antitype were found. There were more cases than expected of early smoking onset with paternal Alcoholism II, $L = 3.01$; $p < .05$. Fewer cases than expected were found among adolescents with paternal Alcoholism II whose smoking onset was not known, $L = -2.70$; $p < .05$.

was not known in two-parent families in which mother was non-alcoholic mother but father had Alcoholism II were fewer than expected (see the cross-hatch marked rectangle on top in Figure 21).

Results suggest that the salience of paternal alcoholism over maternal alcoholism as a risk factor for early smoking onset in offspring. Furthermore, results point out that the risks of early smoking onset were higher for sons of fathers with Alcoholism II. A milder form of alcoholism over long-term did not appear to pose much risk to children. In a striking resemblance to the results of smoking parents, results appear to emphasize the importance of both parents in two-parent families in the sense that it took two alcoholic parents to steer children to the smoking path. Even with a severe type of alcoholism (i.e., Alcoholism II), it was the combined effects of alcoholism on both parents' side that elevated the risks for adolescent sons' smoking onset.[15]

15 Data on daughters was inconclusive, due to the small observed and expected frequencies. One antitype was found at configuration 113, indicating that there were fewer cases than expected of adolescent daughters, whose smoking onset was unknown, had non-smoking parents (for more information, see Table G2 in Appendix G).

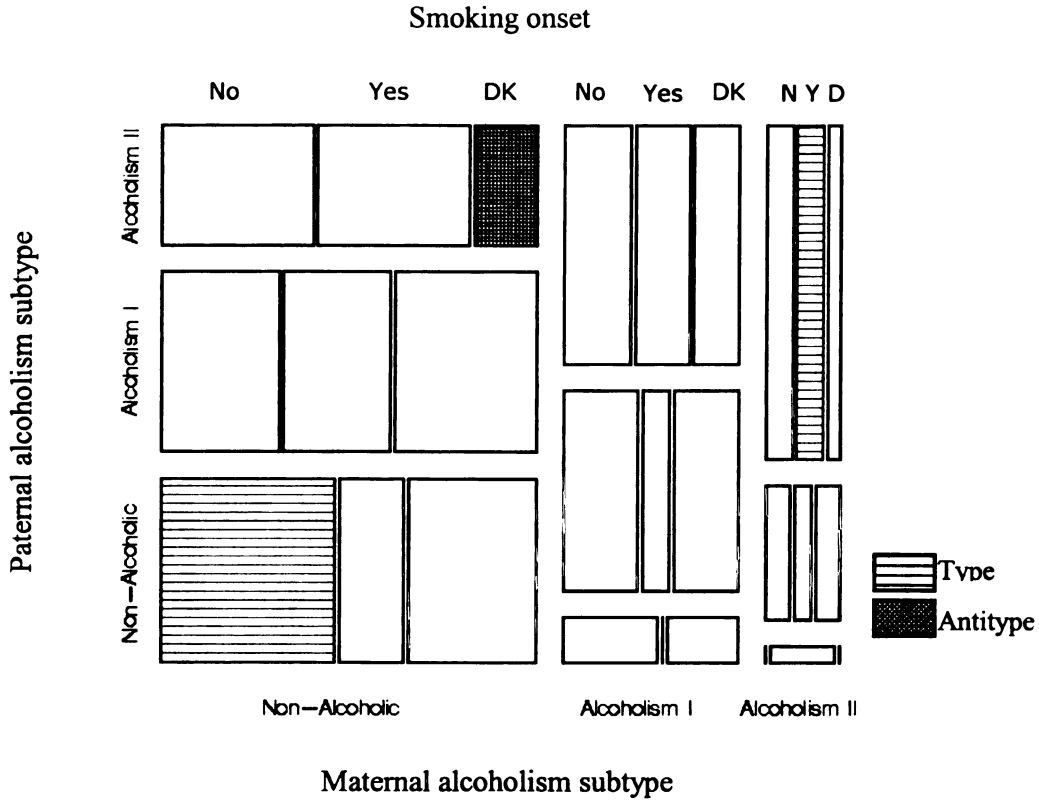


Figure 21.

Parental alcoholism and smoking onset among adolescent sons. Yes = Smoking onset by age 14, No = Non-smoker, DK = smoking onset unknown.

Table 10

Configurations of Parental Alcoholism Patterns and Early Smoking Onset Among Adolescent Sons

M_AP_AS	Obs. Freq.	Exp. Freq.	L	Type/Antitype
111	30	17.95	3.64*	Type
112	11	11.96	-.34	
113	22	13.74	2.73	
121	20	25.53	-1.47	
122	18	17.02	.30	
123	24	19.55	1.29	
131	17	24.77	-2.09	
132	17	16.51	.15	
133	7	18.97	-3.52*	Antitype
211	4	8.11	-1.65	
212	0	5.41	-2.57	
213	3	6.21	-1.44	
221	14	11.53	.86	
222	5	7.69	-1.10	
223	12	8.83	1.22	
231	15	11.19	1.34	
232	12	7.46	1.88	
233	10	8.57	.56	

(table continues)

Table 10 (Continued)

Configurations of Parental Alcoholism Patterns and Early Smoking Onset Among Adolescent Sons

M_AP_AS	Obs. Freq.	Exp. Freq.	L	Type/Antitype
311	0	3.14	-1.93	
312	1	2.09	-.80	
313	0	2.40	-1.66	
321	3	4.46	-.77	
322	2	2.97	-.61	
323	3	3.42	-.25	
331	8	4.33	1.97	
332	8	2.89	3.26*	Type
333	4	3.31	.41	

Note. M_A = maternal alcoholism pattern; P_A = paternal alcoholism pattern; S = smoking onset by adolescent sons. Numerals in M_AP_AS column represent ordered triples of variable categories. Response categories for parental alcoholism were 1 = Non-alcoholic, 2 = Alcoholism I, and 3 = Alcoholism II. Options for adolescent smoking onset were 1 = Never smoked, 2 = Smoked by age 14, and 3 = Smoking-onset unknown. L stands for Lehmacher's test statistic (1981); Bonferroni-adjusted alpha, $\alpha^* = 0.00185$ was used; * significant at $\alpha^* = 0.00185$.

In summary, so far results indicate that there are differential risks involved, with dependent types of usage patterns of cigarette smoking and alcoholism related to a higher risk for their adolescent sons' smoking onset. However, in both cases of parental smoking and alcoholism, it was the combination of both parents that exerted any impact on adolescent children's smoking onset. As for the relative importance of paternal versus maternal, it appears that at least in the case of alcoholism, paternal alcoholism is a more important factor for early smoking onset in offspring.

From the previous results on parental smoking and alcoholism where dependent types of smoking and alcoholism were found associated with one another, it is plausible that children's risks for early smoking onset would accordingly increase, as parental dependence on smoking is stacked upon Alcoholism II. Unfortunately, it was not possible to investigate all five factors together (i.e., maternal smoking and alcoholism subtypes, paternal smoking and alcoholism subtypes, and adolescent children's smoking onset) in the current study since it requires at least 243 cell configurations ($3 \times 3 \times 3 \times 3 \times 3$). However, a large scale national longitudinal data may provide further insights into this issue of aggregated risks of parental substance abuse for their children's smoking onset and usage, and the specific patterns of risks in the future.

EARLY AND CONCURRENT CHARACTERISTICS OF ADOLESCENTS WITH EARLY SMOKING ONSET

In this section, adolescents who start experimenting with cigarette smoking early were compared on measures of prenatal exposure to maternal smoking and drinking, early temperament and behavioral characteristics as well as concurrent child behavioral characteristics. In particular, it was hypothesized that adolescents with early smoking onset would be characterized by 1) a higher level of prenatal exposure to maternal daily cigarette smoking and/or weekly drinking, 2) early temperament dimensions assessed at ages three and five, and 3) a higher level on each of the four CBCL syndrome scales rated by both mother and father at child ages three and five, and once again at child ages twelve and fourteen: Anxious/Depressed, Attention problems, Delinquent behavior, and Aggressive behavior. MANOVA was conducted for each of ten multiply imputed datasets, and the results are presented in average parameters (i.e., average mean and standard deviation) and the range of test statistics.

Prenatal Exposure to Maternal Smoking and Drinking

Prenatal exposure to maternal smoking and drinking was examined for both sons and daughters. Information on their prenatal history was obtained for almost all children included in the current study. Therefore, prenatal exposure to maternal smoking and drinking was investigated and presented for both sons and daughters. Prenatal exposure to daily maternal cigarette smoking was related to smoking onset of sons, $F(2, 278) = 3.29 - 4.26$, $p < .05$, and daughters, $F(2, 85) = 6.12 - 7.81$, $p < .05$ (see Figure 22 and Tables F1 and G3 in Appendices F and G).

Post-hoc tests using the Bonferroni method revealed that sons who started cigarette use by age 14 had a higher level of prenatal exposure to maternal daily smoking, compared to those whose smoking onset was unknown. No significant difference was found between sons with early smoking onset and those who never smoked. Daughters who started smoking by age 14 had a higher level of maternal smoking, compared to all others. Results supported the latest research findings that prenatal exposure to maternal smoking is a risk factor for early smoking onset in offspring.

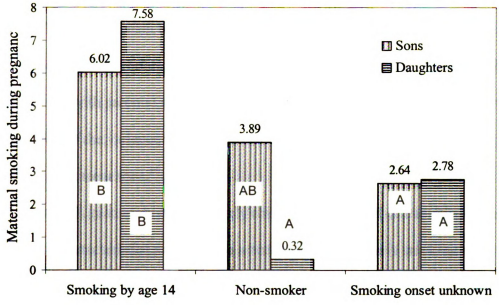


Figure 22.

Prenatal exposure to maternal smoking and adolescent children's smoking. Means that do not share letters differ at $p < .05$ using the Bonferroni method.

In contrast, results on maternal weekly drinking during pregnancy as a risk factor for early smoking onset were inconsistent. It turned out to be a significant factor for early onset of smoking in only four of ten multiply imputed datasets among sons, $F(2,$

278) = 2.28 – 4.47; p = .104 – .012. It was not a significant factor in all ten datasets among daughters, F(2, 85) = 1.80 – 3.08, ns.

Precursors and Concurrent Characteristics of Adolescents of Early Smoking Onset

In this analysis, multivariate analysis of variance was conducted to see whether early smoking onset status was related to the host of early measures, including prenatal exposure to daily smoking and weekly drinking, child temperament and behavioral characteristics measured at ages between three and five, and behavioral characteristics measured at ages between twelve and fourteen. Sons of various smoking onset status were different, multivariate F = 2.44 – 3.52, p < .05 with maternal ratings; F = 2.59 – 3.66, p < .05 with paternal ratings (see Table 11 and Figures 23 - 30). Approximately 11% – 15% of the variance for variables entered in MANOVA was explained by smoking status of adolescent sons.[16]

Overall, maternal ratings of early child temperamental characteristics and behavioral characteristics proved to be a better indicator of early smoking onset among adolescent sons than paternal ratings. Based on maternal ratings, early measures of child temperament and four behavioral syndromes and concurrent measures of behavioral syndromes distinguished adolescents who subsequently started cigarette use early from those who did not (see Figures 23 – 26). On the other hand, paternal ratings were not as predictive as maternal ratings (see Table 11). Based on paternal ratings, a temperament

16 The data on daughters was inconclusive, albeit some apparent resemblance to results of sons, due to a small sample of younger cohorts. MANOVA results are presented in Appendix G (see Table G3), and descriptive statistics in Tables G4 and G5.

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dimension, Approach-Withdrawal at ages three to five, and concurrent measures of Anxious/depressed, Attention problems, Delinquent behavior, and Aggressive behavior were predictive of smoking onset in sons.

Post-hoc tests were conducted on each of significant measures using the Bonferroni method of pairwise comparisons. The same procedure used previously was again adopted to combine results of post-hoc tests from ten multiply imputed datasets (For more details, see p. 96 in the present study). Adolescents who never smoked were rated by mother more attentive (and less distractible) at ages three to five, compared to those whose smoking onset was unknown (see Figure 23). In addition, adolescent sons who started cigarette use by age 14 were rated more reactive, compared to those who never smoked, based on maternal ratings. There were no differences across three groups of adolescents on a temperamental dimension, "Approach-Withdrawal." However, results from paternal ratings were different from those of maternal ratings in that no differences were found on both temperamental dimensions of Attention span and Reactivity. Yet, those who started cigarette use by age 14 were rated more approaching to new stimuli and people at ages three to five by father (see Figure 24).

Early behavioral characteristics were markedly different across three groups of adolescents based on maternal ratings. Adolescents who started cigarette use by age 14 were rated by mother as being more anxious and depressed, having more attention problems, and more often displaying both delinquent and aggressive behavior at ages three and five (see Figures 25 – 28). Paternal ratings of child early behavioral characteristics did not differ across three groups of adolescents (see Figures 29 – 30). Concurrent behavioral characteristics rated by mother revealed that adolescents with

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early onset of smoking had more often attention problems, delinquent behavior, and aggressive behavior at ages 12 to 14 (see Figures 26 – 28). Parental ratings of behavioral characteristics converged more on measures of concurrent behaviors. Paternal ratings on delinquent and aggressive behaviors were discriminating adolescents of different smoking onset statuses (see Figures 29 – 30). In all measures of behavioral characteristics, adolescents whose smoking onset was unknown were not different from those who never smoked by age 14.

In summary, results in this section support the hypotheses of 1) prenatal exposure to maternal cigarette smoking and 2) early discriminating child characteristics as precursors of early smoking onset. Overall, results support the notion that children who were parentally exposed to maternal daily cigarette smoking are at risk for early smoking onset, and that there are identifiable early precursors of early smoking onset temperamentally and behaviorally. Results so far point to the possibility that there may exist some early constitutional vulnerability manifested in child temperament and behavioral characteristics as well as familial vulnerability. The next section addresses questions related to the mechanisms of how these factors contribute to early smoking onset.

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Table 11

MANOVA Results on Precursors and Concurrent Characteristics of Adolescents with Differing Smoking Onset Status

	Maternal rating	Paternal rating
Multivariate F	2.44* – 3.52*	2.59* – 3.66*
Wilk's Λ	.854* – .894*	.849* – .888*
Variable	Univariate analysis	
Prenatal exposure		
Daily maternal smoking	3.29* – 4.26*	
Weekly maternal drinking	2.28 – 4.47*	
Early temperament (Ages 3 – 5)		
Attention span	3.26* – 4.35*	2.00 – 3.30*
Approach/Withdrawal	.27 – .76	3.35* – 4.40*
Reactivity	3.08* – 3.56*	1.65 – 2.65
Early child behavior problems (Ages 3 – 5)		
Anxious/Depressed	6.42* – 8.67*	1.33 – 2.17
Attention problems	3.77* – 5.24*	1.31 – 1.60
Delinquent behavior	7.15* – 9.65*	1.24 – 1.69
Aggressive behavior	2.85 – 5.35*	1.23 – 1.56
Concurrent adolescent behavior problems (Ages 12 – 14)		
Anxious/Depressed	2.87 – 6.20*	1.38 – 9.62*
Attention problems	5.30* – 7.99*	3.74* – 9.47*
Delinquent behavior	17.02* – 22.92*	13.22* – 18.98*
Aggressive behavior	8.31* – 11.80*	5.32* – 6.86*

Note. * $p < .05$. Degrees of freedom for multivariate analysis of variance 1, 279 for both maternal and paternal ratings; degrees of freedom for univariate F test were 2, 278.

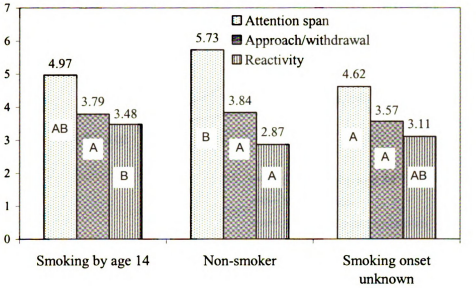

Figure 23.

Maternal ratings of child temperament and smoking onset among sons. Means that do not share letters differ at $p < .05$ using the Bonferroni method.

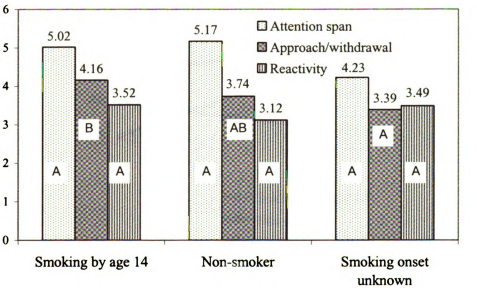

Figure 24.

Paternal ratings of child temperament and smoking onset among sons. Means that do not share letters differ at $p < .05$ using the Bonferroni method.

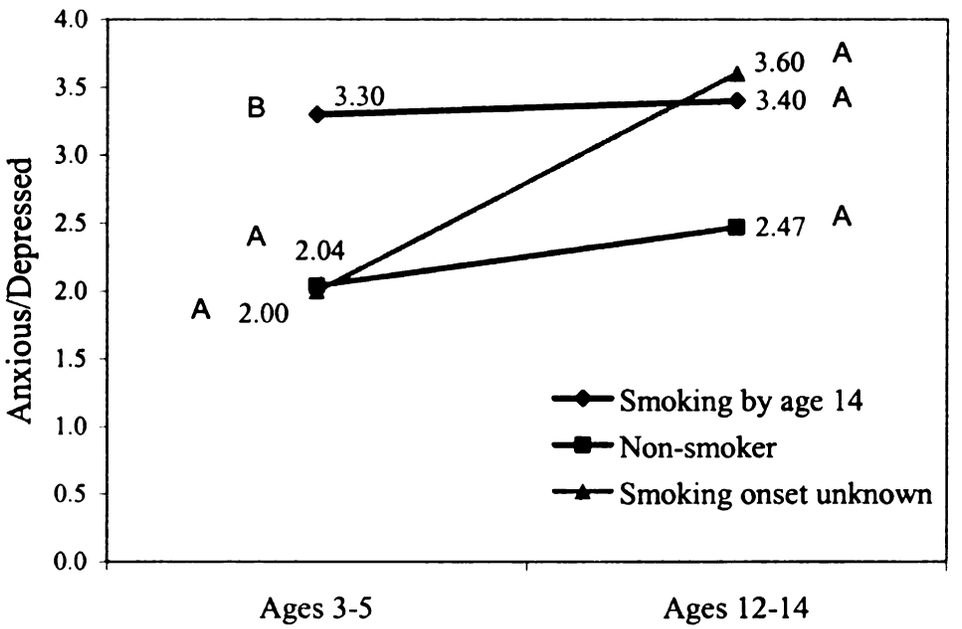

Figure 25.

Maternal ratings of Anxious/depressed and smoking onset among sons. Means that do not share letters differ at $p < .05$ using the Bonferroni method.

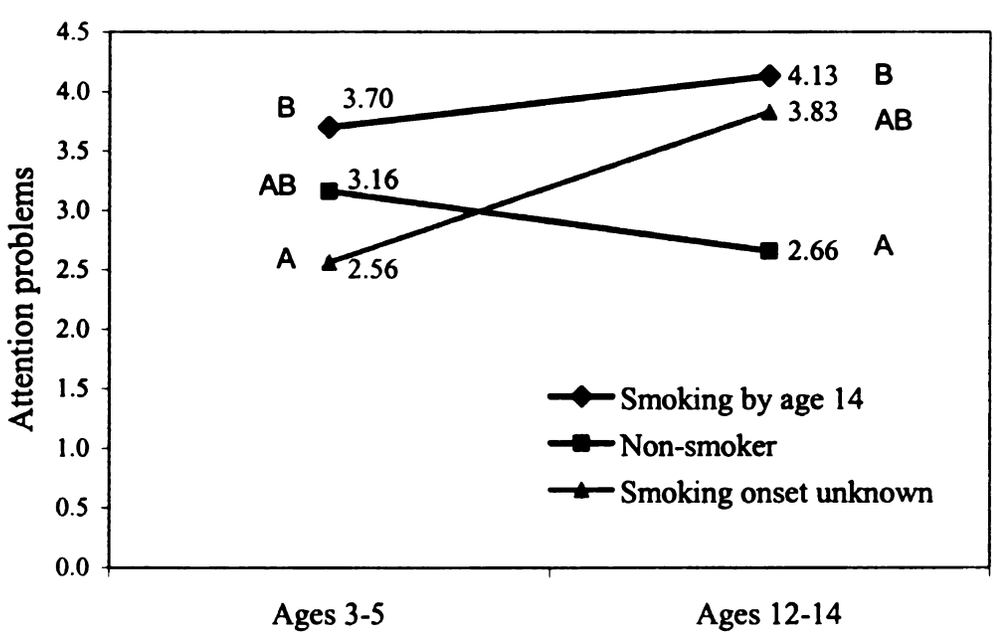

Figure 26.

Maternal ratings of Attention problems and smoking onset among sons. Means that do not share letters differ at $p < .05$ using the Bonferroni method.

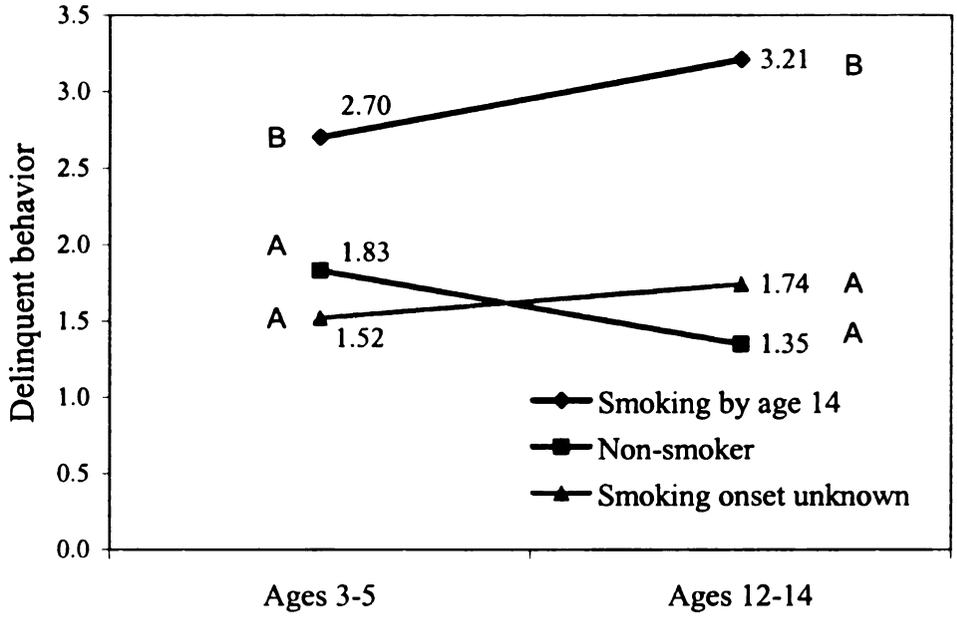

Figure 27.

Maternal ratings of Delinquent behavior and smoking onset among sons. Means that do not share letters differ at $p < .05$ using the Bonferroni method.

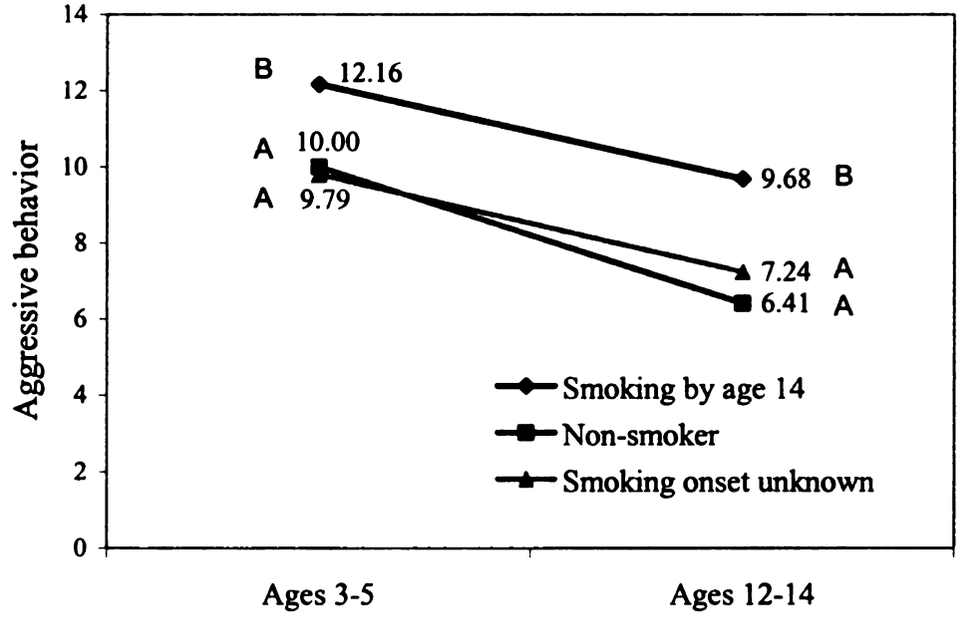

Figure 28.

Maternal ratings of Aggressive behavior and smoking onset among sons. Means that do not share letters differ at $p < .05$ using the Bonferroni method.

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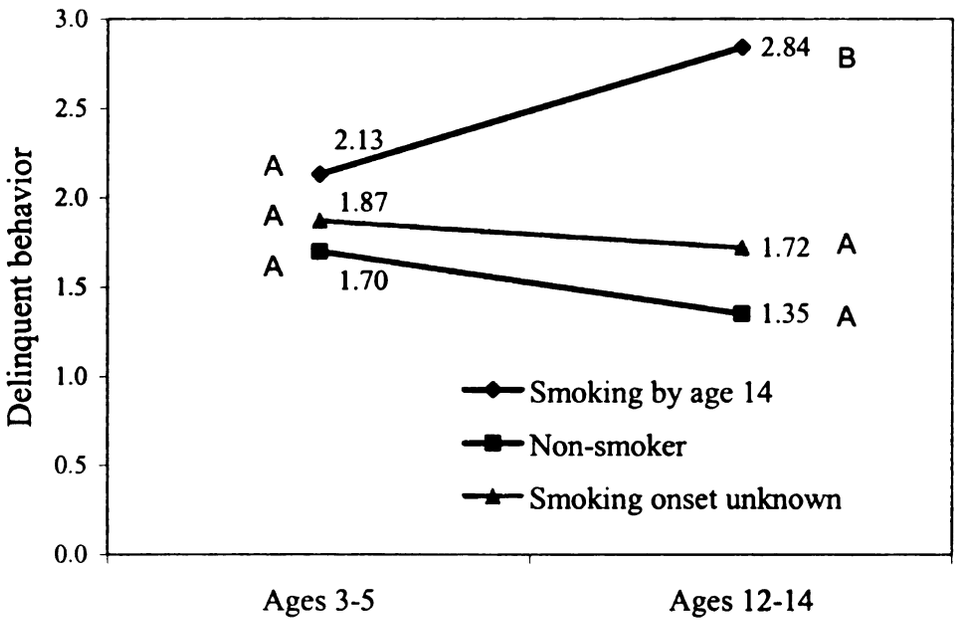

Figure 29.

Paternal ratings of Delinquent behavior and smoking onset among sons. Means that do not share letters differ at $p < .05$ using the Bonferroni method.

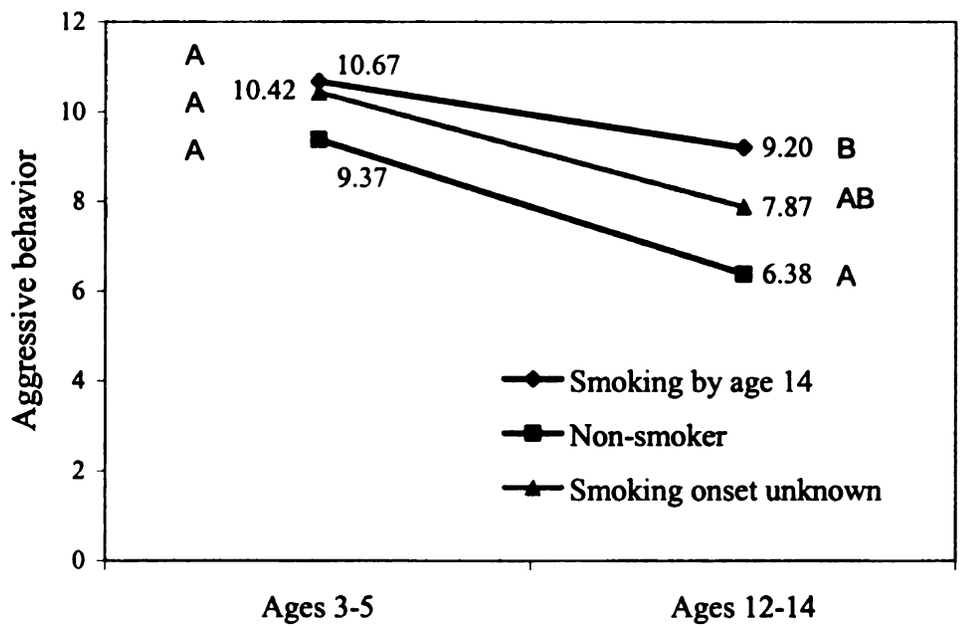

Figure 30.

Paternal ratings of Aggressive behavior and smoking onset among sons. Means that do not share letters differ at $p < .05$ using the Bonferroni method.

PATHS TO EARLY SMOKING ONSET

The current study so far demonstrated that early smoking onset was robustly observed among adolescent boys in families where both parents are smokers, with at least one parent is a Heavy smoker for their entire adulthood (see Figure 10). In addition, results showed that parental alcoholism also played a role in early smoking onset among adolescent sons (see Figure 21). When both parents were alcoholics with a lifetime pattern of dependence, adolescent sons were more likely to experiment with or smoke cigarettes. The current study also showed that the smoking subtype and alcoholism subtype were associated with one another, and that these subtypes were related to a host of demographic and psychopathological measures. In addition, findings showed that adolescents with early smoking onset could be distinguished from others, based on the measures of prenatal exposure to cigarette smoking, child temperament, and behaviors as early as three to five as well as concurrent adolescent behaviors at ages twelve to fourteen. However, due to the co-existing nature of risk factors involved in the process of growing up in families with smoking parents, it is desirable that the unique role of each of the risk factors related to smoking onset in children be investigated simultaneously.

Manifest variable structural equation modeling (SEM) analysis was conducted. Based on the previous results of the current study, the following key nine variables were selectively chosen: Maternal smoking subtype, paternal smoking subtype, parental alcoholism composite score, prenatal exposure to daily maternal cigarette smoking, four syndrome scale scores of Anxious/depressed, Attention problems, Delinquent behavior, and Aggressive behavior rated at ages three to five, and smoking onset status. The

variables were carefully selected in number and kind due to the overall sample size and the ordinal scale level of the variables. Asymptotic covariance matrix with the Generalized Weighted Least-Squares (WLS) estimation method normally requires very large samples for reasonably robust estimation (Jöreskog & Sörbom, 1996, pp. 21-23).

Maternal and paternal smoking subtypes were coded on a four-point ordinal scale: 0 = Non-smoker, 1 = Light smoker, 2 = Heavy-to-light smoker, and 3 = Heavy smoker. Parental alcoholism composite score was created by adding maternal and paternal alcoholism subtypes, yielding a five-point ordinal scale (0 = Neither parent alcoholic, 1 = Only one alcoholic parent with Alcoholism I, 2 = One parent with Alcoholism II and the other non-alcoholic parent, or both parents with Alcoholism I, 3 = One parent with Alcoholism II and the other with Alcoholism I, and 4 = Both parents with Alcoholism II). Prenatal exposure to maternal daily cigarette smoking was originally measured in the number of cigarettes smoked per day but recoded on a six-point ordinal scale: 0 = None, 1 = One to five cigarettes per day, 2 = Six to ten cigarettes per day, 3 = 11 to 19 cigarettes per day, 4 = 20 cigarettes (or one pack) per day, and 5 = More than one pack per day. The cutoff points for each category of prenatal exposure were decided based on the smoking literature, and the frequencies of responses. Four CBCL behavioral syndrome scales rated by mother were recoded on a three-point ordinal scale: 0 = Lower quartile (bottom 25 percent), 1 = Middle 50 percent, and 2 = Upper quartile (top 25 percent). Smoking onset status was coded as follows: 0 = Never smoked, 1 = Smoking-onset unknown, and 2 = Smoked by age 14.

Due to the ordinal level scale of variables, a polychoric correlation matrix and an asymptotic covariance matrix were created for each of ten multiply imputed datasets by

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PRELIS using LISREL, version 8.51 (Jöreskog & Sörbom, 2001). Once all ten sets of polychoric correlation matrices and asymptotic covariance matrices were calculated, they were analyzed using LISREL with the WLS estimation method (Jöreskog & Sörbom, 1996). A polychoric correlation matrix using all ten imputed datasets is presented in Table 12. Statistical inferences on parameters followed Rubin's suggestion (1987; Schafer, 1997, pp. 108-110). The overall estimate ($\overline{Q}$) is the average of the individual estimates ($\hat{Q}$). The overall standard error ($\sqrt{T}$) is $\sqrt{\overline{U} + \left(1 + \frac{1}{m}\right) * B}$, where within-imputed variance ($\overline{U}$) is the mean variance (i.e., squared standard error in the current situation) of m parameter estimates from m datasets, and the between-imputed variance (B) is the variance of imputed estimates ($\hat{Q}$) from the overall estimate ($\overline{Q}$). The overall degrees of freedom is calculated by $df = (m-1) * \left(1 + \frac{m * \overline{U}}{(m+1) * B}\right)^2$.

It was hypothesized that there are direct links from maternal and paternal smoking subtypes to early smoking onset in adolescent sons. In addition, parental alcoholism and prenatal exposure to maternal daily smoking were hypothesized to directly link to early smoking onset. The most interesting part of the hypothesized structural relationships was the indirect path from maternal smoking subtype to early smoking onset via prenatal exposure to maternal daily cigarette smoking and early child behavioral characteristics (i.e., Anxious/Depressed, Attention problems, Delinquent behavior, and Aggressive behavior). Four early child behavioral syndrome scales were allowed to covary, and three exogenous variables (parental smoking subtypes and parental alcoholism) were automatically set to correlate with each other.

The hypothesized model addresses several research questions: 1) whether the direct links from parental smoking to early smoking onset in offspring stays significant above and beyond other direct and indirect paths to early smoking onset in offspring; 2) whether parental alcoholism plays an important role in leading children toward paths to early smoking onset above and beyond other more specific paths; 3) whether prenatal exposure to maternal daily smoking plays a direct and immediate role in early smoking onset; and finally 4) whether the indirect path from maternal smoking to early smoking onset via prenatal exposure and early child behavioral characteristics is significant above and beyond the effects of other direct mechanisms of early smoking onset.

Reports of the analyses followed the guidelines suggested in the literature (Hoyle & Panter, 1995; Raykov, Tomer, & Nesselroade, 1991). The hypothesized model fit the data very well, $\underline{X}^2$ (20) = 14.664 – 18.213, ns; Root Mean Square Error of Approximation (RMSEA) = 0.000; Standardized Root Mean Square Residual (SRMR) = 0.038 – 0.042; Goodness of Fit Index (GFI) = 0.996; Comparative Fit Index (CFI) = 1.000. Goodness-of-fit indices and residuals were all within the acceptable range. Modification indices of un-estimated path coefficients were very small, ranging from .00 to 6.31. Only one modification index for the path coefficient from Aggressive behavior to prenatal exposure to maternal daily smoking was slightly bigger than the critical value of 3.84 (expected amount of $\underline{X}^2$ change significant for one degree of freedom) in seven of ten analyses. The hypothesized model was not modified due to the improbable nature of the direction of the un-estimated path. Overall, excellent goodness of fit statistics, and small modification indices and residuals suggest that the hypothesized mechanisms of early

smoking onset were all in the expected directions and that there were no other substantial relations left out in the hypothesized model (see Figure 31).

All direct links from exogenous factors to early smoking onset were not significant. Neither parental smoking subtypes nor parental alcoholism were directly involved in intergenerational transmission of smoking. In addition, prenatal exposure to maternal daily cigarette smoking was not directly related to early smoking onset in adolescent sons. However, there was an indirect link between maternal smoking subtype and early smoking onset via prenatal exposure and observed child behavior of Anxious/Depressed at ages three to five.

The results suggest that when all things were considered simultaneously, it was an indirect path via early child behaviors of Anxious/depressed that led to early smoking onset in adolescent sons. Although prenatal exposure to maternal daily cigarette smoking was related to all four domains of child behavior problems at child age three to five, it was only via the Anxious/depressed route that led to early smoking onset in adolescent sons. Young children as early as three to five who were rated high by their mother on the CBCL items such as "Lonely," "Cries," "Unloved," "Fearful," and "Worries" were more likely to engage in early smoking experimentation and smoking. Results suggest that the link between parental smoking and early smoking onset in offspring can be accounted for by mediating factors of prenatal exposure to cigarette smoking and negative affect as early as age three to five. In addition, paternal smoking or parental alcoholism do not appear to have true associations with early smoking onset in offspring. Their ties to early smoking onset in offspring in the first two sections of Results can be mostly attributed to their shared variance with maternal smoking type.

Table 12

Estimated Polychoric Correlation Matrix

	1	2	3	4	5	6	7	8	9
1. Maternal smoking subtype	1.000								
2. Paternal smoking subtype	0.682	1.000							
3. Parental alcoholism	0.481	0.517	1.000						
4. Prenatal exposure to nicotine	0.888	0.623	0.406	1.000					
5. Anxious/Depressed	0.266	0.156	0.099	0.235	1.000				
6. Attention problems	0.180	0.073	0.217	0.125	0.359	1.000			
7. Delinquent behavior	0.290	0.267	0.133	0.259	0.390	0.379	1.000		
8. Aggressive behavior	0.273	0.181	0.224	0.172	0.519	0.591	0.434	1.000	
9. Early smoking onset	0.217	0.215	0.111	0.173	0.214	0.101	0.197	0.123	1.000

Note. This matrix was created using all ten imputed datasets.

Ages 12 14

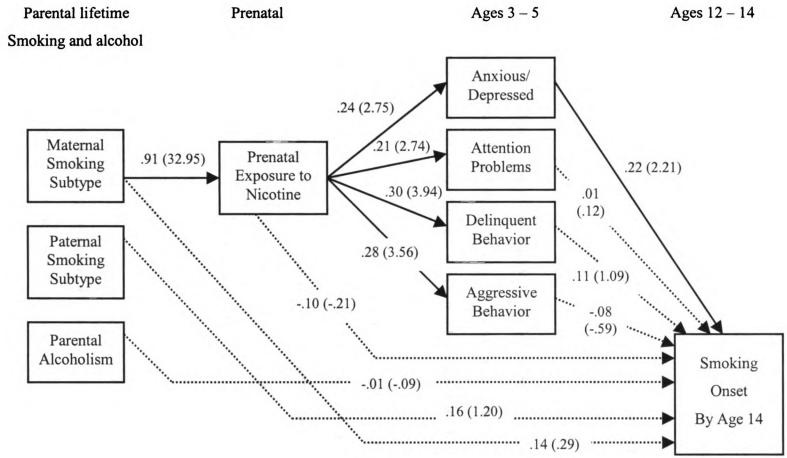

Figure 31.

A model of intergenerational transmission of smoking: Anxious/Depressed as a behavioral mediator. Dotted lines indicate non-significant paths. Numbers in parentheses are t statistic values. X^2 (20) = 14.664 – 18.213, ns; Root Mean Square Error of Approximation (RMSEA) = 0.0; Goodness of Fit Index (GFI) = 0.996.

DISCUSSION

The present study examined initiation of cigarette use among adolescents from a pathological perspective where individual sensitivity and vulnerability to cigarette smoking is emphasized as a mechanism of early smoking onset. This perspective leads our attention to early observable factors with some constitutional basis in children and familial vulnerabilities. These emphases sharply contrast to a widely popular epidemic or exposure model of adolescent smoking where exposure to smoking by peers and family members are construed as a key factor in the context of modeling and pressure. The perspective of individual sensitivity (and vulnerability) to smoking as a key is not new in the literature of nicotine dependence and intervention for cessation of smoking in adult populations. However, it has not been a focal point in the studies of smoking onset and in preventive efforts to control smoking initiation.

In line with individual differences in sensitivity to cigarette smoking, the present study examined whether the phenomenon of intergenerational transmission of smoking can reliably be observed in the manifestation of early smoking onset. Although some researchers suggested that habitual smoking runs in families (e.g., Bierut et al., 2000; Prescott & Kendler, 1995), parental smoking as a risk factor for adolescent smoking initiation has been relatively underexplored. To fill the gap, the present study investigated smoking onset among adolescents from four major angles. While intergenerational transmission of smoking can be attributed to numerous factors, the present study limited its focus to early risk factors in children and parents. Each of four major results sections provides insight to the phenomenon of early onset of smoking among adolescents. Findings of the current study are recaptured and discussed in here in

the order of 1) pathways to early smoking onset in a high-risk population of adolescents, 2) heterogeneous developmental patterns of smoking and alcoholism among parents, and finally 3) limitations and future directions.

Pathways to Early Smoking Onset

Parental smoking. Results revealed that Heavy smoking by either parent, characterized by a long-term, high-level smoking was related to children's smoking initiation, when either paternal or maternal smoking was studied alone. However, when smoking by two parents was simultaneously considered in their association with smoking onset in offspring, it was the presence of both smoking parents that was related to an increased likelihood for children to start cigarette use early. Heavy smoking by either parent alone was not sufficiently related to an increased likelihood of early smoking onset. It was the combination of heavy smoking by fathers, with either light or heavy smoking by mothers that was associated with an increased risk of early onset of smoking in children.

The results suggest that parental smoking be studied on both parents in two-parent families. In the presence of heavy smoking by one parent, smoking by the other parent becomes a vital factor for children's smoking onset. Although it is uncertain how the risk for early smoking onset increases as both parents in a two-parent family smoke, the presence of a non-smoking parent appears to ameliorate and counter-balance the facilitators of early smoking onset in children, whereas the presence of two smoking parent exacerbates them. In addition, the results point to the pattern of cigarette smoking (i.e., quantity and duration of smoking) by parents as an important factor for early onset of smoking in offspring. The current study hints that a long-term habitual heavy smoking

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(or nicotine dependence) by parents may be a significant marker for an underlying susceptibility to cigarette smoking in children, and also a potent marker for an array of risk factors related to growing up in families with a higher level of parental psychopathologies and a lower level of socioeconomic status.

Parental alcoholism. Among many possibilities, one pathway to early onset of smoking investigated in the present study was via a common familial vulnerability represented by parental alcoholism. Given the high co-occurrences of habitual smoking and alcoholism, it is natural to speculate that the intergenerational transmission of smoking may potentially be marked by a parental alcoholism diagnosis that can be attributed in part to the common correlates of a positive family alcoholism and habitual smoking.

The results lend support to the studies that found a relationship between parental alcoholism and early smoking initiation in offspring (i.e., Hanna & Grant, 1999), and further provide some insight on the reported relationship between the two. The current study particularly points to the specific nature of parental alcoholism as a risk factor for early smoking onset in offspring. A long-term dependence on alcohol by both parents appears to link to an increased likelihood of early smoking onset in offspring. Paternal but not maternal alcoholism subtype, when crossed with adolescent smoking onset, was related to an increased the likelihood of children's early engagement in cigarette use, with parental Alcoholism II (a long-term dependence on alcohol) closely tied to early child smoking onset. However, when alcoholism subtypes by both parents were simultaneously crossed with children's smoking onset, it was the presence of both

alcoholic parents with Alcoholism II that were associated with early onset of smoking in offspring.

The results for alcoholism were parallel to those of parental smoking as a risk factor for early onset of smoking in the sense that 1) both results revealed that in two-parent families, it involved two parents to increased a chance for their child to start cigarette use at an earlier age, 2) out of the two subtypes of alcoholism only Alcoholism II, representing a more dependent type of alcoholism was related to an increased likelihood of early onset of smoking in offspring, 3) Alcoholism II was related to a higher level of psychopathologies and a lower level of socioeconomic status. Furthermore, Heavy smoking and Alcoholism II were related to one another. The strong relationship between smoking types and alcoholism types and the compatibility between parental smoking and alcoholism in their associations with early smoking onset in offspring prompt many more questions than answers.

Among many others, we can ask what it is about Heavy smoking and/or Alcoholism II by parents that might set children at risk for early onset of smoking? And what are possible synergistic influences on children' smoking onset when parents smoke and drink so heavily for the majority of their adulthood during while their children move into adolescence. In addition, how does the risk for children's early smoking onset step over the threshold as both parents exhibit a dependent type of smoking and alcohol use? It is plausible that heavy smoking and Alcoholism II by both parents reflect the increased likelihood that the child has some constitutional susceptibility to cigarette smoking among other problems, with one possible source resulting from prenatal and postnatal exposure to cigarette smoking. At the same time, co-existing psychopathologies and lack

of resources in families may play indirect roles in promoting early smoking onset in offspring via numerous pathways, including parenting behaviors, coercive family environment, and association with deviant peers. The current study focused on early observable characteristics of children as antecedents of early smoking onset in adolescence.

Early characteristics of adolescents with early smoking onset. One can shift the attention to children and ask whether there were early identifiable factors in children with early smoking onset. Results revealed that adolescents could be traced back to their early development for their vulnerability to early cigarette use. Adolescents with early cigarette use were different, on several characteristics typically considered as early risk factors for many later developmental outcomes. Adolescents with early onset of smoking had more prenatal exposure to maternal daily cigarette smoking. In addition, their mother viewed them as more reactive, and the father viewed them as more approaching to new stimuli and people as early as three to five. In addition, adolescents who never smoked by age 14 were observed by their mother as less distractible at ages three to five. Behaviorally, they were rated high at ages three to five by mothers on the CBCL scales of Anxious/Depressed, Attention problems, and Delinquent and Aggressive behaviors. These behavioral characteristics were again confirmed at ages 12 to 14, with the exception of Anxious/Depressed. Paternal ratings produced slightly different results, showing adolescent sons with smoking onset rated high on Delinquent and Aggressive behavior at ages 12 to 14.

Results lend support to the recent findings of the literature of child behavioral characteristics and smoking. There are burgeoning debates on 1) which aspects of

ADHD (e.g., inattention versus hyperactivity) are related to smoking (Burke et al., 2001), and 2) whether ADHD is truly related to smoking, regardless of conduct disorder (CD) and vice versa among various populations of adolescents (Milberger et al., 1997; Whalen et al., 2002). Although the current study is neither designed nor equipped for these issues, it supplies evidence that these co-existing conditions of attention problems, hyperactivity, and CD-related behaviors are manifested and observed as early as three to five years of age among children with early smoking onset.

In addition, results indicate the importance of Anxious/Depressed behavior in relation to early smoking onset. The relationship between smoking and negative affect/depressive symptoms is well established in adult populations of normative (e.g., M. Windle & R. C. Windle, 2001) as well as clinical samples (e.g., Pomerleau et al., 1997; Shiffman et al., 1994a). However, Anxious/Depressed behavior has not been studied in a sample of young children in relation to smoking. The current study highlights that early negative affect measured using questions such as "Lonely," "Cries," "Fearful," and "Worries" can meaningfully be observed in children as early as three to five, and that it appears to be a significant early risk factor for early smoking onset.

The current study also showed that maternal and paternal ratings had low convergence on child temperament and behavioral problems, yielding distinct results based on maternal and paternal ratings of child characteristics. Correlations between maternal and paternal ratings on child temperament ranged in average from .269 to .373, and from .198 to .384 for four behavioral syndrome scales at ages three to five. Parental agreement on children's behavior appeared improved at child's ages 12 to 14, with correlations ranging from .250 to .527. Low to moderate agreement between maternal

and paternal ratings is nothing new. Substantially different findings, depending on the source of information (e.g., ratings of self, parents, peers, or teachers), have been noted in psychiatric and family research (O'Connor & Rutter, 1996). Multiple sources of information have been suggested as a strategy to obtain a robust solution that can reliably be generalized, especially when concurrent relations are of interest in a study (O'Connor & Rutter, 1996; Rothbart & Bates, 1998). In the current study, however, early child temperament and early behavioral characteristics temporally preceded smoking onset in young adolescence, therefore minimizing possible reporting biases that one's perception of problematic behavior clouds judgment of other behaviors. In the current study, maternal observations and perceptions of child temperament and behavioral problems were overall a better indicator than paternal ratings for later smoking onset in children. Results were consonant with a summarized report that maternal observations are, in most situations, relevant and reliable (see Rothbart & Bates, 1998).

Paths to early smoking onset. The primary purpose of the manifest variable SEM analysis was to provide some clues to the mechanisms of intergenerational transmission of smoking at the stage of smoking onset in offspring. It was hypothesized that parental smoking and alcoholism are risk factors for early smoking onset in children, not because they have direct influences on it, but because they reflect indirect paths via prenatal exposure to maternal cigarette smoking and early child behavioral characteristics. With the exception of maternal smoking subtype, neither paternal smoking type nor parental alcoholism nor even prenatal exposure to smoking was a sufficient condition to cultivate early smoking onset in children.

Maternal smoking, however, appeared to be a causal spark in a series of chains leading up to early smoking onset via mother's continued smoking during pregnancy. Prenatal exposure to maternal cigarette smoking was then related to anxious/depressed behavior (negative affect), attention problems, aggressive and delinquent behaviors at child ages three to five. Of these negative affect predicted early smoking onset many years later. Paternal smoking subtype and parental alcoholism were remotely related to early smoking onset only due to the fact that they shared variance with maternal smoking subtypes. Therefore, roles of paternal smoking and parental alcoholism appear to be spurious in nature in relation to early smoking onset in offspring. The existence of heavy smoking and Alcoholism II by both parents in two-parent families may be suggestive of perilous undercurrents of heavy smoking by mothers and accordingly exposure to a higher level of cigarette smoking during prenatal development.

The origin of individual vulnerability to cigarette smoking may be rooted in a long-term heavy smoking by mother that is directly and closely related to continued smoking during pregnancy. Indeed, maternal smoking type and prenatal exposure to maternal smoking were almost inseparable constructs, as indicated by the path coefficient of .91 in Figure 31 and also by the correlation coefficient of .888 in Table 12. And the exposure to maternal cigarette smoking during prenatal development may have altered, to some extent, the function and structure of the brain of the child (Wakschlag et al., 1997; Weitzman et al., 1992). Although we do not know the full details about prenatal exposure to cigarette smoking, it appears that it certainly leads to an increased level of behavior problems (Anxious/Depressed, Attention problems, Delinquent and Aggressive behaviors) in young children as early as three to five. Among these early child

behavioral characteristics, at least one domain, Anxious/Depressed appears to be directly associated with early smoking onset in adolescent offspring. Anxious/Depressed syndrome or negative affect in a more general term has not been studied in the literature in relation to prenatal smoking exposure nor to smoking onset. The current study provided evidence that negative affect shown by young children is an important mediator of early smoking onset.

There are several important implications to these findings with regard to preventive efforts to control cigarette initiation among adolescents. The report of the Surgeon General in 1994 concluded that school-based programs, coupled with youth-oriented mass media campaigns and tobacco tax increase are effective measures to prevent tobacco use among youth (USDHHS, 1994). Recently, the Task Force on Community Preventive Services reconfirmed the previous conclusions and recommended that increase the unit price of tobacco products and long-term, high-intensity mass media campaigns are an effective deterrence to smoking initiation among youth (CDC, 2000). Findings of the current study suggest that prevention programs targeted for special populations at risk for early smoking onset may also be effective. Adolescents with increased susceptibility to cigarette smoking may benefit more with preventive and intervention programs tailored uniquely for them. Findings of the current study showed that adolescents with early smoking onset are different from others from very early on, tracing back to as early as their prenatal development followed by differences in temperament and behavioral characteristics at ages three to five. Therefore, children's susceptibility to cigarette smoking may be identified much earlier before their first cigarette.

Heterogeneous Developmental Patterns of Smoking and Alcoholism

Although it was not the focal point of the current study, a pathological model of smoking could also be examined in a high-risk population of alcoholic and control parents. This served two purposes. It provided parental information in connection with adolescent children's smoking initiation, and a parent equivalent portrait of vulnerability to smoking. The former has been discussed; the latter, the second focus of the current study is discussed in this section. The investigation of parental smoking and alcoholism in the present study was unique in many aspects, including utilization of population-based, prospective longitudinal data and new analytical techniques. Its implications are discussed as follows.

Smoking types. The current study asked whether the findings of two major types of smokers from Chassin and her colleagues (2000) could be extended into the age period that goes beyond age 31. Results supported the notion of the heterogeneity of smokers, and confirmed the general pattern of decline in smoking prevalence (Anthony & Echeagaray-Wagner, 2000). However, the number and the kind of distinctive smoking patterns over time proved to be convergent more with the literature on smoking by habitual smokers in adult clinical populations, including Shiffman and his colleagues (1994a, 1994b) than with findings of Chassin et al. (Early Stable, Late Stable, Experimenter, and Quitter). Three distinctively different types of smokers (i.e., Heavy smokers, Light smokers, and Heavy-to-Light smokers) were identified in the present study, based on their long-term patterns of cigarette smoking during adulthood ranging from ages 24 to 50.

Differences in results between the current study and Chassin et al. (2000) can be attributed to the differences in measurement of smoking, the age period, and participants between two studies. First, in Chassin et al's study smoking was measured in a way that once participants smoke weekly, they were differentiated into three categories based on their quantity of smoking: 10 or fewer cigarettes per day, 11 – 20 cigarettes a day, and 20 or more cigarettes a day. Therefore, it is plausible and acknowledged by authors that Light smokers and/or chippers may have been included in both Late and Early stable smokers in Chassin et al's study (2000). In the present study, on the other hand, smoking was based on a finer quantity measure of seven categories that captured light smoking as well as heavy smoking. Therefore, the current study was able to show the distinctive developmental pattern for Light smokers.

Second, in Chassin et al's study, smoking was measured from adolescence to young adulthood (up to age 31) where smoking initiation and experimentation most often occur, with frequent changes in both smoking status and quantity of smoking. Therefore, a lot more fluctuations in smoking can be expected and consequently captured in Chassin et al's study. However, the age period covered in the present study ranged from ages 24 to 50, well past the time of smoking initiation and experimentation. Three types of smokers found in the present study, accordingly, reflect stabilized, long-term habitual smoking in adulthood. The patterns of stabilized habitual smoking in mid 20s to 50 in the present study generally match with findings of epidemiological studies of nation-wide, representative, cross-sectional data of all ages on tobacco use and dependence (Anthony & Echeagaray-Wagner, 2000).

Third, participants in the present study were population-based alcoholic and non-alcoholic men and their spouse, whereas Chassin et al (2000) featured population-based adolescents. Given a higher rate of smoking among alcoholics, it is more likely that smokers were over-represented in the present sample than in populations at large. In fact, 47.2% of smokers in the present sample were substantially more than 39.1% of smokers (including quitters and experimenters) in Chassin et al's study. In addition to a higher number of smokers among alcoholics, alcoholics tend to smoke more heavily. Therefore, results from the current study may be over representative of heavy smokers than in populations at large.

However, findings of the current study hold up the notion that there are two types of smokers. The majority belongs to a group of long-term regular, heavy smokers who are chronically dependent on nicotine, and the second group consists of a small proportion of long-term regular but light smokers without nicotine dependence (i.e., chippers or light smokers) (O. F. Pomerleau et al., 1993). A much smaller number of smokers are suggested to be in transition from heavy smoking to reduced light smoking (e.g., converted chippers) or even to smoking cessation. Findings from the current study appear to fit into these three categories of smokers described in the literature in terms of prevalence and patterns of smoking, with Heavy smokers equivalent of "regular and heavy" smokers, Light smokers of "regular but light" smokers or chippers, and Heavy-to-light smokers of converted chippers.

The notion of two major types of smokers was empirically supported in the present study for the first time, to the author's knowledge, from population-based, long-term prospective longitudinal data. New revelations made by the current study were that

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first, there were substantially more smokers (22.1%) in the category of Light smokers than previously thought (an estimated 5% of smokers); second, although non-significant, the types of smokers showed systematic differences on measures of psychopathologies and socioeconomic status in a dose-response pattern; third, the specificity of the relationship between smoking types and other characteristics varied across gender, with more differences found between Heavy and non-Heavy smokers among men, whereas more differences found between Non-smokers and smokers among women; fourth, in both men and women Heavy smokers were statistically different from all others in the number of years of education and conduct problems and antisocial behavior. Findings by the current study should prompt initiatives to examine typology of smokers and its related antecedents, concurrent relations, and health outcomes in future studies.

Alcoholism types. There have been many theoretical and empirical studies in the literature that point out the heterogeneous nature of alcoholism (Babor et al., 1992; Cloninger, 1987; Zucker, 1987; Zucker, Fitzgerald, & Moses, 1995). However, typologies of alcoholism were rarely derived from an empirical study based on population-based long-term prospective longitudinal data. Empirical results from the current study appear convergent with the literature on alcoholism although more extensive studies are needed to calibrate the nature of these two alcoholic groups. Based on findings of the current study, Alcoholism II appears to overlap, to certain extent, with Antisocial alcoholism, Type II, or Type B in the literature. However, results were limited by the modest sample size of alcoholics in the present study, and the generalization of the results beyond the homogeneous population featured in this study should be cautioned. Limitations of the current study are later discussed in more detail.

As expected, types of smoking and alcoholism were highly associated in both men and women. Results support the long-held notion using a different analytical approach and perspective that smoking and drinking are related. Non-smokers were very unlikely to have a long-term severe type of alcoholism (i.e., Alcoholism II). Likewise, long-term Heavy smokers were very unlikely to be non-alcoholics but more likely to have Alcoholism II. In addition, there were gender-specific patterns of association between alcoholism and smoking. While it was unlikely that heavy smokers were non-alcoholics, those cases were found more often in women than men. Similarly, while it was unlikely that non-smokers were those who showed Alcoholism II, more men than women belonged to the category.

Although the co-occurrence of smoking and drinking or the co-morbidity of nicotine dependence and alcohol dependence were outside the focus of the present study, results nonetheless provided interesting issues for future studies. First, there may be different mechanisms underlying the link between smoking and drinking for men and women. The present study showed that there are patterns of gender-specific as well as general in the way that smoking is related to alcoholism. Second, there may be some shared psychosocial characteristics unmeasured in the present study. The current study used only a limited number of variables to test whether empirically driven types of smoking and alcoholism could be distinctively discriminated. However, it is highly plausible that people with dependence on nicotine and alcohol may share some other psychosocial and personality characteristics, including impulsivity and sensation seeking (Little, 2000). Similarly, nicotine and alcohol may share some neuropharmacological

properties as expressed in hypotheses of cross-tolerance and cross-reinforcement[17] (O. F. Pomerleau, 1995). More studies need to follow from all directions to reveal the mechanisms of co-occurrence of smoking and drinking/alcoholism.

Quantitative differences versus qualitative typology. In the current study, parental smoking and alcoholism were utilized as typologies as opposed to quantitative differences over time. Subtypes of smoking and alcoholism were examined as an indicator for an unmeasured risk structure typified as a lower level of socioeconomic status and resources, and a higher level of antisociality and depression. An alternative to this approach is to study fluctuations of parental functions across time as a predictor for child outcomes. It is natural to assume that familial and child outcomes may vary over time as parental functions fluctuate (DeLucia, Belz, & Chassin, 2001). Although this alternative approach did not turn out as expected in DeLucia et al's study, it merits future investigations on both approaches to parental functions over time as predictors of child developmental outcomes.

Limitations and Future Directions

The findings of the present study should be seen in light of its several limitations. First, findings of the present study regarding the natural developmental patterns (or trajectories) of smoking and alcoholism need to be replicated using a full longitudinal analysis that matches the same age period and descriptions of the population analyzed in the present study. The current study utilized an available four-wave longitudinal data to

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17 Cross-tolerance refers to the possibility that nicotine increases tolerance to the aversive effects of alcohol and vice versa, whereas cross-reinforcement refers to the possibility that nicotine increases reinforcing effects of alcohol and vice versa.

its maximum efficiency, with possible compromise on robustness of the data. Parental alcohol use and smoking were assessed up to four times with a three-year interval between measurements in this study. Information on parental smoking and alcoholism of each individual were then placed and overlapped with others over age, creating developmental patterns over a much wider age span. This method produced incomplete data with gaps in between ages per person that pertained to a relatively smaller age period per participant.

The current study design can be construed as a blend between a full longitudinal study and a cross-sectional study in that results were derived from four-wave longitudinal data stretched over an age span at least twice as long. In addition, analysis on parental alcoholism utilized a retrospective report on the age of first alcoholism diagnosis, expanding the time period up to 40 and 35 for fathers and mothers, respectively. One of the drawbacks was that in both extreme ends of the age period, there were only a few observations made, resulting in relatively large standard errors. It especially influenced analysis on women's alcoholism. In an ideal situation where no limits are placed on time and financial resource, a more controlled forty-year longitudinal study may suit better to investigate developmental patterns of smoking and alcoholism. However, its merits are also traded off with the hefty price tag of such a longitudinal study and other related issues, including a substantial subject attrition rate. In the present study, the issue of robustness and reliability of the data was weighed against the efficient use of the data. Although the present study had a higher rate of unobserved data points across chronological age, it was justified given the highly convergent nature of the phenomena of our interests (i.e., smoking and alcoholism).

Second, one should exercise caution when generalizing the findings from this racially and geographically homogeneous sample of the current study. Participants in the present study were mostly Caucasian families residing in mid-Michigan area. In addition, when they were initially recruited, families were intact. Although a number of families were separated, divorced, and/or remarried, the UM-MSU Longitudinal Study followed up almost all biological and stepparents who were separated from or added into the families. In the present study, three stepparents met the criteria of the present study (e.g., completion of at least two of the first four assessments). However, in all three cases information on biological parents' smoking and alcoholism was available and therefore used in the present study. Consequently, parental smoking and alcoholism in the present study refer to those of biological parents in two-parent families who reside with the children in all but three cases. It is important for future studies to investigate parental smoking and alcoholism with adolescent children in families of different structures. The roles of parental smoking and alcoholism may differ among children in single-parent families or two-parent families with stepparents. In addition, it is important for future studies to replicate the present findings with adolescent children and their parents from other geographic locations, and from other racial, ethnic and cultural groups.

Third, there may be more factors that lead children of smoking parents to and away from cigarette smoking that were not included in the current study. By no means do results from the current study imply that the tested path model is the only way to early smoking onset in a high-risk population of adolescents. On the contrary, the current study aimed to show that some children are at increased risks for early smoking onset and their susceptibility to cigarette smoking exists prior to children's first cigarette smoking

from the perspective of the multiple pathways to substance abuse, with smoking included (Chassin, Curran, Hussong, & Colder, 1996a; Zucker, 1994). Although the research focus could not be extended due to sample size and the nature of the data, it is more reasonable that there may be multiple routes to early onset of smoking via diverse combinations of risk factors and mechanisms (i.e., equifinality; Cicchetti & Rogosch, 1996; Cicchetti & Toth, 1998). For example, it is very plausible that families with parents of certain subtypes of smoking and alcoholism may foster smoking-friendly environment in which children take on more favorable attitudes toward smoking, and have an easy and early access to cigarettes. In addition, findings of the current study point out that children in these families are exposed to a higher level of parental psychopathologies and a lower level of socioeconomic resource. Future studies can address whether there exist additional mediating pathways to early smoking onset in a high-risk population of adolescents.

Likewise, maternal heavy smoking and prenatal exposure to cigarette smoking may also lead to multiple outcomes, with early smoking onset being just one outcome of many (i.e., multifinality; Cicchetti & Rogosch, 1996; Cicchetti & Toth, 1998). In addition, parental smoking subtypes and alcoholism may serve as a proxy for other developmental outcomes in children other than cigarette smoking. Considering that Heavy smoking and Alcoholism II were associated with other known risk factors for children's less advantageous developmental outcomes, it is highly likely that children of those parents may be at risk for other developmental outcomes, including early onset of drinking, higher behavioral problems, and lower academic achievement. Children of smoking parents have not been considered as a risk population in the literature. However

the current study provided evidence that they may be at increased risk for other aspects of development as well. Further efforts are necessary to investigate the specificity as well as aggregation of the risk factors identified in the current study in relation to a wide range of developmental outcomes.

Fourth, data on daughters were not sufficient to ensure reliable findings for many analyses conducted in the current study, due to the recruitment design of daughters. Daughters were brought into the UM-MSU Longitudinal Study systematically later than sons and it resulted in either their first participation starting at later assessment wave or younger birth cohorts at the first measurement wave. Due to this design, assessments of either the first wave or the 4^{th} wave were not carried out, resulting in a higher rate of missingness. Since many analyses of the current study required information collected when the target child was three to five and twelve to fourteen years old, it was decided that data on daughters data be exploratively used with caution. Although results from daughters showed similar trends to those of sons in some analyses, they were not as statistically reliable. Comprehensive research is needed in the future to clarify whether there exists an equivalent mechanism of intergenerational transmission of smoking for boys and girls.

APPENDICES

APPENDIX A

Table 1A

Number of Adolescents by Birth Cohort

	Count	
Cohort	Son	Daughter
1992	0	1
1991	0	1
1990	0	6
1989	0	12
1988	24	12
1987	50	11
1986	37	18
1985	57	7
1984	25	11
1983	31	5
1982	22	1
1981	20	3
1980	10	0
1979	5	0
Total	281	88

Table 2A

Number of Parents by Birth Cohort

	Count			Count	
Cohort	Father	Mother	Cohort	Father	Mother
1938	2	0	1955	21	18
1940	1	0	1956	22	25
1941	1	0	1957	24	21
1943	1	1	1958	23	26
1944	3	0	1959	20	33
1945	2	0	1960	16	19
1946	2	0	1961	19	18
1947	5	3	1962	13	23
1948	4	0	1963	6	22
1949	6	3	1964	10	9
1950	10	5	1965	3	6
1951	12	9	1966	1	4
1952	15	4	1967	0	1
1953	13	14	1970	0	1
1954	20	15	Total	275	280

APPENDIX B

Table B1

Distribution of Birth Cohort Across Year of Assessment at Wave 1: Sons

		Year of assessment																
Cohort	n	1985	1986	1987	1988	1989	1990	1991	1992	1993	1994	1995	1996	1997	1998	1999	2000	2001
1988	22							15	7									
1987	49						14	27	8									
1986	37					7	18	9	3									
1985	54				14	16	15	9										
1984	24			3	7	9	5											
1983	28	2	1	14	7	4												
1982	18	2	5	6	4	1												
1981	20	8	8	3	1													
1980	10	5	4	1														
1979	5	3	2															

Table B2

Distribution of Birth Cohort Across Year of Assessment at Wave 4: Sons

		Year of assessment															
Cohort	n	1985	1986	1987	1988	1989	1990	1991	1992	1993	1994	1995	1996	1997	1998	1999	2000	2001
1988	11																10	1
1987	36															12	19	5
1986	27														4	15	8	
1985	55												1	9	14	16	14	1
1984	24												2	8	7	7		
1983	31											3	14	7	7			
1982	22										4	7	6	5				
1981	18									2	6	5	5					
1980	9									2	5	2						
1979	4									1	3							

Table B3

Distribution of Birth Cohort Across Year of Assessment at Wave 1: Daughters

		Year of assessment																
Cohort	n	1985	1986	1987	1988	1989	1990	1991	1992	1993	1994	1995	1996	1997	1998	1999	2000	2001
1992	1							1										
1991	1							1										
1990	5					1	1	3										
1989	11					7	4											
1988	9				3	3	3											
1987	8				5	2	1											
1986	5				5													
1985	1			1														
1984	1		1															
1983	3	1	1	1														
1982	1		1															
1981	3	2	1															

Table B4

Distribution of Birth Cohort Across Year of Assessment at Wave 4: Daughters

		Year of assessment																
Cohort	n	1985	1986	1987	1988	1989	1990	1991	1992	1993	1994	1995	1996	1997	1998	1999	2000	2001
1992	0																	
1991	0																	
1990	0																	
1989	0																	
1988	4															3	1	
1987	8														1	4	3	
1986	14													3	5	6		
1985	7												3	3		1		
1984	11												3	6	2			
1983	5											1	4					
1982	1										1							
1981	3										2	1						

Table B5

Distribution of Birth Cohort Across Year of Assessment at Wave 1: Fathers

Cohort	n	Year of assessment							
		1985	1986	1987	1988	1989	1990	1991	1992
1938	2						1		1
1940	1			1					
1941	1						1		
1943	1								1
1944	3	1				1	1		
1945	2			1			1		
1946	1		1						
1947	5				1		2	1	1
1948	4	1		1	1	1			
1949	6	1	1	2	1	1			
1950	10		3	1		2	1	3	
1951	12	2			2	2	3	3	
1952	14	3	2		2	2	2	2	1

(table continues)

Table B5 (Continued)

Distribution of Birth Cohort Across Year of Assessment at Wave 1: Fathers

Cohort	n	Year of assessment							
		1985	1986	1987	1988	1989	1990	1991	1992
1953	13	2		1		5	3	1	1
1954	19		1	3	4	3	5	1	2
1955	21	1	3	1	6	2	4	3	1
1956	22	3	3	2	1	5	2	6	
1957	23		2	3	3	5	5	4	1
1958	22	4	1	2	2	1	5	5	2
1959	20	1	2	3	1	2	2	6	3
1960	16	1				2	4	7	2
1961	18			3	4	1	2	5	3
1962	13		1	1	1	2	4	4	
1963	6			1	1		1	3	
1964	10			1		1	2	6	
1965	3							3	
1966	1						1		

Table B6

Distribution of Birth Cohort Across Year of Assessment at Wave 2: Fathers

Cohort	N	1990	1991	1992	1993	1994	1995
1938	2			1		1	
1943	1				1		
1944	2			1	1		
1945	1				1		
1946	1			1			
1947	4			1	2	1	
1948	3	2		1			
1949	4	2		1	1		
1950	7	1		3		3	
1951	10	2	1	1	2	4	
1952	11	1	1	4		3	2
1953	9			3	4		2

(table continues)

Table B6 (Continued)

Distribution of Birth Cohort Across Year of Assessment at Wave 2: Fathers

Cohort	n	1990	1991	1992	1993	1994	1995
1954	13	1		5	3	3	1
1955	15	3	1	2	3	4	2
1956	17	3	1	4	3	4	2
1957	19	1	1	5	6	4	2
1958	17	1	2	3	1	8	2
1959	14	1		3	2	5	3
1960	14				3	7	4
1961	18	2	1		7	3	5
1962	12		3	2	3	4	
1963	6	1	1			4	
1964	9				2	5	2
1965	3					3	
1966	1				1		

Table B7

Distribution of Birth Cohort Across Year of Assessment at Wave 3: Fathers

		Year of assessment									
Cohort	n	1990	1991	1992	1993	1994	1995	1996	1997	1998	1999
1938	2						1		1		
1940	1			1							
1941	1					1					
1943	1								1		
1944	1								1		
1945	2					1		1			
1946	1						1				
1947	2							1		1	
1948	4				2	1	1				
1949	4				3		1				
1950	8			1	1		3		2	1	
1951	10				3	1	1	2	3		
1952	12	2				2	3	1	2	2	

(table continues)

Table B7 (Continued)

Distribution of Birth Cohort Across Year of Assessment at Wave 3: Fathers

		Year of assessment									
Cohort	n	1990	1991	1992	1993	1994	1995	1996	1997	1998	1999
1953	12		1		2		6	1		1	1
1954	16			1	2	4	3	2	2	2	
1955	17				2	6	1	5	1	2	
1956	17		2	1	1	2	3	3	3	1	1
1957	19				2	1	8	4	3	1	
1958	15		1		1	1	3	3	3	3	
1959	17			1	2		2	3	5	4	
1960	12					1	2	2	3	4	
1961	17				2	1	5	2	3	4	
1962	12					2	4	3	3		
1963	4				1			1	1	1	1
1964	9						1	4	1	2	
1965	2							1	1		
1966	1							1			

Table B8

Table B8

Distribution of Birth Cohort Across Year of Assessment at Wave 4: Fathers

		Year of assessment								
Cohort	n	1993	1994	1995	1996	1997	1998	1999	2000	2001
1938	1						1			
1940	1			1						
1941	1					1				
1943	1								1	
1944	2			1	1					
1945	2				1			1		
1946	2		1		1					
1947	4				1		1	1	1	
1948	2			1		1				
1949	5	1		1	2			1		
1950	10			2	1		3	1	2	1
1951	12			2	1	2	2	2	3	
1952	11	2	2		1	1	2		1	2
1953	11		1		2	1	5	1		1

(table continues)

Table B8 (Continued)

Table B8 (Continued)

Distribution of Birth Cohort Across Year of Assessment at Wave 4: Fathers

		Year of assessment								
Cohort	n	1993	1994	1995	1996	1997	1998	1999	2000	2001
1954	18		2	3	2	3	2	3	1	2
1955	17		2	2	1	4	2	3	3	
1956	15		1	3		1	3	4	3	
1957	19	1	1		1	3	3	5	2	3
1958	17		3	2	2	1	3	1	3	2
1959	16		2	1	2	1	1	3	4	2
1960	8		1					2	3	2
1961	14				1	2	3	2	2	4
1962	12					2	3	3	4	
1963	4				1			1	1	1
1964	6				1			3	2	
1965	2								2	
1966	1								1	

Table B9

Table B9

Distribution of Birth Cohort Across Year of Assessment at Wave 1: Mothers

Cohort	n	Year of assessment							
		1985	1986	1987	1988	1989	1990	1991	1992
1943	1						1		
1947	3			1	1	1			
1949	3		1			1	1		
1950	5	1	1	1		1		1	
1951	9	2	1	1	2	2	1		
1952	4		1	1		1		1	
1953	14	4	1	1	3	2	1	1	1
1954	14	1	1	2	2	2	2	4	
1955	18	2	3				7	4	2
1956	25	4	1	1	5	6	3	3	2
1957	21		2	3	2	5	1	5	3
1958	24	2	4	2	1	1	10	4	
1959	31	2	1	5	2	5	7	8	1
1960	19	1	2	1	2		4	9	

(table continues)

Table B9 (Continued)

Distribution of Birth Cohort Across Year of Assessment at Wave 1: Mothers

Table B9 (Continued)

Distribution of Birth Cohort Across Year of Assessment at Wave 1: Mothers

		Year of assessment							
Cohort	n	1985	1986	1987	1988	1989	1990	1991	1992
1961	17	2		1	1	4	3	3	3
1962	23	1	1	2	2	3	6	4	4
1963	22			2	4	3	3	10	
1964	9			1	2	2	2	2	
1965	6			1			1	3	1
1966	4			1	2			1	
1967	1							1	
1970	1						1		

Table B10

Table B10

Distribution of Birth Cohort Across Year of Assessment at Wave 2: Mothers

		Year of assessment					
Cohort	n	1990	1991	1992	1993	1994	1995
1947	1			1			
1949	2			1	1		
1950	4	2		1		1	
1951	6	2	1	2		1	
1952	3	1		1		1	
1953	11	3	1	2	3	2	
1954	8			3		5	
1955	14	2			5	5	2
1956	19	2	1	7	3	4	2
1957	18	2		4	5	3	4
1958	19	2	1	3	5	7	1
1959	27	3	2	3	9	6	4
1960	14	1		1	3	6	3

(table continues)

Table B10 (Continued)

Distribution of Birth Cohort Across Year of Assessment at Wave 2: Mothers

		Year of assessment					
Cohort	n	1990	1991	1992	1993	1994	1995
1961	11		1	2	3	3	2
1962	21	1	1	4	5	6	4
1963	22		3	3	4	9	3
1964	7	1	1	2	1	2	
1965	5				1	3	1
1966	2		1			1	
1967	1					1	
1970	1					1	

Table B11

Table B11

Distribution of Birth Cohort Across Year of Assessment at Wave 3: Mothers

		Year of assessment									
Cohort	n	1990	1991	1992	1993	1994	1995	1996	1997	1998	1999
1947	3					2	1				
1949	2						1		1		
1950	4				2		1		1		
1951	8				4	1	2	1			
1952	3				1		1		1		
1953	11	2		1	1	2	2	1	2		
1954	13	1	1		1	2	3	1	3	1	
1955	14				2			6	2	4	
1956	21			2	4	3	6	3	1	2	
1957	18					2	7	2	4	3	
1958	19			1	2	3	3	6	2	2	
1959	32		1		5	4	5	8	5	4	
1960	16		1		1	1		4	4	4	1

(table continues)

Table B11 (Continued)

Distribution of Birth Cohort Across Year of Assessment at Wave 3: Mothers

		Year of assessment									
Cohort	n	1990	1991	1992	1993	1994	1995	1996	1997	1998	1999
1961	16				1	2	3	4	2	4	
1962	19			1	1	1	6	3	3	4	
1963	19					1	6	6	4	1	1
1964	8				1	1	2	3	1		
1965	5					1		1	1	1	1
1966	4				1	1	1	1			
1967	1							1			
1970	1								1		

Table B12

Distribution of Birth Cohort Across Year of Assessment at Wave 4: Mothers

		Year of assessment								
Cohort	n	1993	1994	1995	1996	1997	1998	1999	2000	2001
1943	1					1				
1947	3				1	1	1			
1949	2			1			1			
1950	5	1		1	1		1		1	
1951	9		1	2	1	1	3	1		
1952	4		1		1		1			1
1953	13	1	2	1	3	1	1	2	2	
1954	12		1		2	2	3	1	2	1
1955	16		2	3				6	3	2
1956	21		1	2	2	4	3	4	4	1
1957	19		3			3	6	1	4	2
1958	22	1	2	3	1	3	3	2	5	2
1959	28		1	2	3	2	4	9	5	2
1960	17	1	2	1	1	1		3	5	3

(table continues)

Table B12 (Continued)

Table B12 (Continued)

Distribution of Birth Cohort Across Year of Assessment at Wave 4: Mothers

		Year of assessment								
Cohort	n	1993	1994	1995	1996	1997	1998	1999	2000	2001
1961	13		1	1	1	2	1	3	4	
1962	19		1		2	2	4	4	3	3
1963	18				2	3	1	6	5	1
1964	9		1		1		3	2	2	
1965	3				1			1		1
1966	4				1	2			1	
1967	1								1	
1970	1								1	

APPENDIX C

Table C1

Matrix of Missingness Pattern for Parental Measures (N = 555)

Count	Smoking	Alcohol	Educ.	Occ.	Conduct	Antisocial	Ham_C	Ham_W	BDI
529	1	1	1	1	1	1	1	1	1
11	1	1	1	1	1	1	0	0	1
7	1	1	1	1	1	1	1	1	0
4	1	1	1	1	0	0	1	1	0
2	1	1	0	0	0	0	1	1	0
2	1	1	0	0	1	1	0	0	0
# Missing	0	0	4	4	6	6	13	13	15
% Missing	0	0	0.72	0.72	1.08	1.08	2.34	2.34	2.70

Note. Matrix of missingness patterns (1 = observed, 0 = missing), Count = number of observations with the specified pattern. Smoking = smoking subtype; Alcohol = alcoholism subtype; Educ. = education in years; Occ. = occupation; Conduct = conduct problems in childhood; Antisocial = antisocial behavior in adulthood; Ham_C = Hamilton depression: current level; Ham_W = Hamilton depression: worst ever; BDI = Beck depression index.

Table C2

Matrix of Missingness Pattern for Adolescent Sons' Measures (N=281)

Count	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27
165	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
2	1	1	0	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
19	1	1	1	1	0	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
3	1	1	0	1	0	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
3	1	1	1	1	1	1	1	1	1	0	0	0	0	1	1	1	1	1	1	1	1	1	1	1	1	1	1
3	1	1	1	1	1	0	0	0	0	1	1	1	1	0	0	0	0	1	1	1	1	1	1	1	1	1	1
26	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	0	0	0	0	1	1	1	1	1	1
2	1	1	1	1	0	1	1	1	1	1	1	1	1	1	1	1	1	0	0	0	0	1	1	1	1	1	1
37	1	1	1	1	1	1	1	1	1	0	0	0	0	1	1	1	1	0	0	0	0	1	1	1	1	1	1
1	1	1	0	1	1	1	1	1	1	0	0	0	0	1	1	1	1	0	0	0	0	1	1	1	1	1	1
8	1	1	1	1	0	1	1	1	1	0	0	0	0	1	1	1	1	0	0	0	0	1	1	1	1	1	1
1	1	1	1	0	0	1	1	1	1	0	0	0	0	1	1	1	1	0	0	0	0	1	1	1	1	1	1
1	1	1	1	1	1	0	0	0	0	0	0	0	0	1	1	1	1	0	0	0	0	1	1	1	1	1	1
2	1	1	1	1	1	0	0	0	0	1	1	1	1	0	0	0	0	0	0	0	0	1	1	1	1	1	1
1	1	1	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1	1	1	1	1	1

(table continues)

Table C2 (Continued)

Matrix of Missingness Pattern for Adolescent Sons' Measures (N=281)

Count	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27
1	1	1	1	1	1	1	1	1	1	0	0	0	0	1	1	1	1	0	0	0	0	0	0	0	1	1	1
1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	0	0	0
1	1	1	1	1	1	1	1	1	1	0	0	0	0	1	1	1	1	0	0	0	0	1	1	1	0	0	0
2	1	1	1	1	1	0	0	0	0	1	1	1	1	0	0	0	0	1	1	1	1	0	0	0	0	0	0
1	1	1	0	1	1	0	0	0	0	1	1	1	1	0	0	0	0	1	1	1	1	0	0	0	0	0	0
1	1	0	0	0	0	0	0	0	0	1	1	1	1	0	0	0	0	1	1	1	1	0	0	0	0	0	0
# missing	0	1	8	2	35	11	11	11	11	54	54	54	54	10	10	10	10	81	81	81	81	5	5	5	6	6	6

Note. Matrix of missingness patterns (1 = observed, 0 = missing), Count = number of observations with the specified pattern. The variables included in the table are 1 = smoking onset status; 2 = prenatal smoking exposure; 3 = quantity of prenatal smoking exposure; 4 = prenatal drinking exposure; 5 = quantity of prenatal exposure to maternal drinking; 6 – 9 = maternal ratings of the four CBCL syndrome scales (anxious/depressed, attention problems, delinquent behavior, and aggressive behavior) at wave 1; 10 – 13 = maternal ratings of the four CBCL syndrome scales at wave 4; 14 – 17 = paternal ratings of the four CBCL syndrome scales at wave 1; 18 – 21 = paternal ratings of the four CBCL syndrome scales at wave 4; 22 – 27 = maternal and paternal ratings of child

temperament (attention span/distractibility, approach/withdrawal

temperament (attention span/distractibility, apporoach/widthrawal, and reactivity). The percentage of missing for each of 27 variables in Table C2 were 0.0, 0.36, 2.85, 0.71, 12.46, 3.91, 3.91, 3.91, 3.91, 19.22, 19.22, 19.22, 19.22, 3.56, 3.56, 3.56, 3.56, 28.83, 28.83, 28.83, 28.83, 1.78, 1.78, 1.78, 2.14, 2.14, and 2.14, respectively.

Table C3

Matrix of Missingness Pattern for Adolescent Daughters' Measures (N = 88)

Count	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27
13	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
1	1	1	1	1	1	1	1	1	1	1	1	1	1	0	0	0	0	1	1	1	1	1	1	1	1	1	1
3	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	0	0	0	0	1	1	1	1	1	1
26	1	1	1	1	1	1	1	1	1	0	0	0	0	1	1	1	1	0	0	0	0	1	1	1	1	1	1
1	1	1	0	1	0	1	1	1	1	0	0	0	0	1	1	1	1	0	0	0	0	1	1	1	1	1	1
1	1	1	1	1	1	1	1	1	1	0	0	0	0	0	0	0	0	0	0	0	0	1	1	1	1	1	1
1	1	1	1	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1	1	1	1	1	1
1	1	1	1	1	1	0	0	0	0	0	0	0	0	1	1	1	1	0	0	0	0	0	0	0	1	1	1
1	1	1	1	1	1	0	0	0	0	1	1	1	1	0	0	0	0	0	0	0	0	1	1	1	0	0	0
2	1	1	1	1	1	1	1	1	1	0	0	0	0	0	0	0	0	0	0	0	0	1	1	1	0	0	0
1	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1	1	1	0	0	0
1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	0	0	0	0	0	0
27	1	1	1	1	1	0	0	0	0	1	1	1	1	0	0	0	0	1	1	1	1	0	0	0	0	0	0
1	1	1	0	1	1	0	0	0	0	1	1	1	1	0	0	0	0	1	1	1	1	0	0	0	0	0	0

(table continues)

Table C3 (Continued)

Matrix of Missingness Pattern for Adolescent Daughters' Measures (N = 88)

Count	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27
4	1	1	1	1	1	0	0	0	0	1	1	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0
1	1	1	1	1	1	1	1	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
2	1	1	1	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
1	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
# missing	0	2	4	2	3	39	39	39	39	37	37	37	37	43	43	43	43	45	45	45	45	38	38	38	41	41	41

Note. Matrix of missingness patterns (1 = observed, 0 = missing). Count = number of observations with the specified pattern. The variables included in the table are 1 = smoking onset status; 2 = prenatal smoking exposure; 3 = quantity of prenatal smoking exposure; 4 = prenatal drinking exposure; 5 = quantity of prenatal exposure to maternal drinking; 6 – 9 = maternal ratings of the four CBCL syndrome scales (anxious/depressed, attention problems, delinquent behavior, and aggressive behavior) at wave 1; 10 – 13 = maternal ratings of the four CBCL syndrome scales at wave 4; 14 – 17 = paternal ratings of the four CBCL syndrome scales at wave 1; 18 – 21 = paternal ratings of the four CBCL syndrome scales at wave 4; 22 – 27 = maternal and paternal ratings of child temperament (attention span/distractibility, apporoach/widthrawal, and reactivity). The percentage of missing for each of 27 variables

in Table C3 were 0.0, 2.27, 4.55, 2.27, 3.41, 44.32, 44.32, 44.32, 44.32, 42.05, 42.05, 42.05, 42.05, 48.86, 48.86, 48.86, 48.86, 51.14, 51.14, 51.14, 51.14, 43.18, 43.18, 43.18, 46.59, 46.59, and 46.59, respectively.

APPENDIX D

Table D1

Number of Cases in the TRAJ Analysis of Smoking (n = 262)

Age	Number of cases	Mean	Standard Deviation
22	2	4.50	.71
23	5	3.80	.45
24	11	2.91	1.51
25	11	3.55	1.57
26	17	3.53	1.70
27	27	2.85	1.63
28	29	3.90	1.45
29	32	3.09	1.69
30	32	3.75	1.34
31	52	3.02	1.99
32	49	3.41	1.82
33	59	3.25	1.65
34	58	3.52	1.65
35	55	3.31	1.86
36	69	2.99	1.87
37	47	3.30	1.86
38	51	3.12	2.03

(table continues)

Table D1 (Continued)

Number of Cases in the TRAJ Analysis of Smoking (n = 262)

Age	Number of cases	Mean	Standard Deviation
39	43	3.16	1.76
40	44	2.89	2.03
41	28	3.00	1.83
42	30	2.37	2.06
43	19	3.21	2.32
44	17	3.06	2.19
45	11	4.00	2.14
46	9	3.22	2.59
47	8	2.25	2.43
48	7	3.14	2.67
49	2	2.50	3.54
50	6	3.67	1.75
51	3	4.67	1.53
52	2	5.50	.71
53	1	4.00	.00
54	2	6.00	.00
55	1	5.00	.00

Table D2

Number of Cases in the TRAJ Analysis of Alcoholism: Men (n = 202)

Age	Number of cases	Mean	Standard Deviation
14	200	.02	.21
15	199	.09	.48
16	192	.14	.62
17	183	.29	.84
18	163	.61	1.13
19	123	.58	1.10
20	94	.56	1.10
21	73	.56	1.11
22	57	.46	1.04
23	49	.33	.90
24	45	.40	.94
25	42	.83	1.19
26	39	.87	1.20
27	39	1.03	1.25
28	35	.89	1.21
29	41	.93	1.29
30	56	.73	1.09
31	58	.88	1.23

(table continues)

Table D2 (Continued)

Number of Cases in the TRAJ Analysis of Alcoholism: Men (n = 202)

Age	Number of cases	Mean	Standard Deviation
32	64	.81	1.08
33	69	.84	1.21
34	67	.72	1.07
35	73	.82	1.21
36	79	.65	1.05
37	77	.57	1.04
38	77	.68	1.14
39	64	.66	1.12
40	59	.78	1.25
41	45	.73	1.12
42	32	.63	1.13
43	25	.96	1.24
44	25	1.04	1.21
45	19	.89	1.20
46	15	.33	.72
47	16	.69	1.01
48	13	.38	.77
49	7	.29	.76

(table continues)

Table D2 (Continued)

Number of Cases in the TRAJ Analysis of Alcoholism: Men (n = 202)

Age	Number of cases	Mean	Standard Deviation
50	9	.33	1.00
51	6	.00	.00
52	7	.57	.98
53	4	.25	.50
54	4	.00	.00

Table D3

Number of Cases in the TRAJ Analysis of Alcoholism: Women (n = 109)

Age	Number of cases	Mean	Standard Deviation
14	109	.04	.30
15	107	.19	.72
16	100	.07	.36
17	95	.12	.50
18	89	.65	1.10
19	63	.32	.82
20	54	.22	.72
21	51	.31	.86
22	47	.11	.37
23	46	.13	.54
24	47	.47	.97
25	39	.15	.54
26	35	.02	.51
27	41	.32	.79
28	47	.47	.95
29	46	.22	.70
30	47	.34	.87
31	47	.53	1.04

(table continues)

Table D3 (Continued)

Number of Cases in the TRAJ Analysis of Alcoholism: Women (n = 109)

Age	Number of cases	Mean	Standard Deviation
32	53	.66	1.09
33	48	.50	.90
34	52	.42	.87
35	51	.57	1.10
36	45	.47	.94
37	33	.39	1.00
38	34	.56	1.05
39	30	.43	.97
40	24	.67	1.24
41	20	.55	1.10
42	15	.00	.00
43	10	.00	.00
44	6	.00	.00
45	7	.43	1.13
46	7	.00	.00
47	4	.00	.00
48	3	1.00	1.73
49	1	.00	.00

APPENDIX E

Table E1

Descriptive Statistics of Smoking Subtypes: Men

Variable	Non-smoker		Light smoker		Heavy smoker		Heavy-to-light	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Education in years	14.43	2.33	14.24	2.24	12.73	1.60	13.00	2.73
Occupation	411.68	199.97	371.56	184.01	281.47	132.21	321.23	231.25
Conduct problems in childhood	7.58	5.66	7.64	4.21	12.31	7.23	8.69	3.51
Antisocial behavior in adulthood	5.93	4.61	6.60	4.86	11.09	7.74	9.54	5.08
Hamilton depression – current	3.50	4.51	3.53	5.11	6.06	7.05	6.69	5.62
Hamilton depression – worst	9.98	9.94	10.25	10.75	15.01	11.42	15.86	9.21
Beck depression index	2.26	2.46	1.95	1.89	3.04	2.95	1.62	2.28

Note. * $p < .05$.

Table E2

Descriptive Statistics of Smoking Subtypes: Women

	Non-smoker		Light smoker		Heavy smoker		Heavy-to-light	
Variable	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Education in years	13.75	1.97	13.63	2.03	12.23	1.40	12.67	.75
Occupation	318.07	137.38	331.38	151.23	261.24	95.39	271.33	54.30
Conduct problems in childhood	4.78	3.36	7.98	6.47	8.73	4.95	11.17	2.88
Antisocial behavior in adulthood	3.53	2.74	5.22	2.48	5.38	3.86	7.67	5.45
Hamilton depression – current	4.73	6.10	7.16	7.05	7.03	6.63	7.50	6.21
Hamilton depression – worst	12.95	11.86	16.50	10.46	17.54	11.65	19.17	12.34
Beck depression index	2.81	3.20	2.99	3.33	3.32	2.98	3.50	2.65

Note. * $p < .05$

Table E3

Descriptive Statistics for Alcoholism Subtypes: Men

Variable	Non-alcoholic		Alcoholism I		Alcoholism II	
	Mean	SD	Mean	SD	Mean	SD
Education in years	14.62	2.15	13.67	2.20	13.03	2.11
Occupation	429.49	200.07	347.26	172.66	305.46	174.59
Conduct problems in childhood	5.70	4.11	10.60	7.05	11.00	6.34
Antisocial behavior in adulthood	3.89	3.00	8.46	5.71	10.91	7.48
Hamilton depression – current	2.59	3.60	4.40	4.90	6.38	7.39
Hamilton depression – worst	7.08	7.99	12.31	10.89	15.87	11.17
Beck depression index	2.09	2.24	2.32	2.84	2.99	2.64

Note. * $p < .05$

Table E4

Descriptive Statistics for Alcoholism Subtypes: Women

Variable	Non-alcoholic		Alcoholism I		Alcoholism II	
	Mean	SD	Mean	SD	Mean	SD
Education in years	13.41	1.88	13.29	2.16	12.43	1.34
Occupation	305.28	130.62	303.04	135.03	282.00	109.87
Conduct problems in childhood	5.01	3.74	7.62	5.02	11.30	4.92
Antisocial behavior in adulthood	3.28	2.35	5.54	3.37	7.31	4.68
Hamilton depression – current	4.86	5.73	6.87	7.67	7.73	6.16
Hamilton depression – worst	12.72	10.20	17.70	13.28	19.23	13.79
Beck depression index	2.65	3.01	3.12	3.08	4.66	3.57

Note. * $p < .05$

APPENDIX F

Table F1

Precursors and Concurrent Factors of Early Smoking Onset: Maternal Ratings of Sons

	Smoking onset by age 14		Non-smoker		Smoking onset unknown	
Variable	Mean	SD	Mean	SD	Mean	SD
Prenatal exposure to maternal smoking and drinking						
Daily smoking	6.02	9.51	3.89	7.47	2.64	6.62
Weekly drinking	.24	.53	.51	1.37	.22	.57
Early temperament (Ages 3 – 5)						
Attention span	4.97	2.74	5.73	3.03	4.62	2.72
Approach/Withdrawal	3.79	1.90	3.84	1.89	3.57	1.93
Reactivity	3.48	1.51	2.87	1.63	3.11	1.71
Early child behavior problems (Ages 3 – 5)						
Anxious/Depressed	3.30	3.37	2.04	1.86	2.00	2.33
Attention problems	3.70	2.79	3.16	2.40	2.56	2.25
Delinquent behavior	2.70	2.84	1.83	1.68	1.52	1.19
Aggressive behavior	12.16	5.87	10.00	5.75	9.79	5.11
Concurrent adolescent behavior problems (Ages 12 – 14)						
Anxious/Depressed	3.40	3.68	2.47	2.47	3.60	2.90
Attention problems	4.13	3.48	2.66	2.55	3.83	3.14
Delinquent behavior	3.21	2.76	1.35	1.53	1.74	1.83
Aggressive behavior	9.68	6.18	6.41	4.56	7.24	5.14

Note. * $p < .05$

Table F2

Precursors and Concurrent Factors of Early Smoking Onset: Paternal Ratings of Sons

Variable	Smoking onset by age 14		Non-smoker		Smoking onset unknown	
	Mean	SD	Mean	SD	Mean	SD
Early temperament (Ages 3 – 5)						
Attention span	5.02	3.16	5.17	3.03	4.23	2.86
Approach/Withdrawal	4.16	1.67	3.74	1.67	3.39	1.91
Reactivity	3.52	1.53	3.12	1.45	3.49	1.44
Early child behavior problems (Ages 3 – 5)						
Anxious/Depressed	2.79	2.89	2.14	2.16	2.43	2.46
Attention problems	3.62	2.87	3.01	2.36	3.11	2.40
Delinquent behavior	2.13	1.71	1.70	1.46	1.87	1.96
Aggressive behavior	10.67	6.50	9.37	5.48	10.42	5.39
Concurrent adolescent behavior problems (Ages 12 – 14)						
Anxious/Depressed	2.63	2.87	2.56	2.63	3.92	3.41
Attention problems	3.71	3.01	2.70	2.51	4.24	3.78
Delinquent behavior	2.84	2.39	1.35	1.51	1.72	1.77
Aggressive behavior	9.20	6.76	6.38	4.73	7.87	5.83

Note. * $p < .05$

APPENDIX G

Table G1

Configurations of Parental Smoking Patterns and Early Smoking Onset Among Adolescent Daughters

MPS	Obs. Freq.	Exp. Freq.	L	Type/Antitype
111	13	5.92	3.54	Type
112	3	6.24	-1.44	
113	20	14.03	2.24	
121	2	1.08	.45	
122	0	1.13	-.66	
123	2	2.55	-.04	
131	2	4.31	-1.07	
132	2	4.54	-1.18	
133	6	10.20	-1.64	
211	1	1.30	.19	
212	2	1.37	.12	
213	2	3.09	-.41	
221	0	.24	.56	
222	0	.25	.52	
223	2	.56	1.33	
231	0	.95	-.50	
232	3	1.00	1.62	

(table continues)

Table G1 (Continued)

Configurations of Parental Smoking Patterns and Early Smoking Onset Among Adolescent Daughters

MPS	Obs. Freq.	Exp. Freq.	L	Type/Antitype
233	1	2.25	-.58	
311	0	2.73	-1.56	
312	0	2.87	-1.63	
313	3	6.45	-1.52	
321	0	.50	.01	
322	0	.52	-.03	
323	2	1.17	.34	
331	1	1.98	-.38	
332	10	2.09	5.76	Type
333	7	4.69	1.03	

Note. M = maternal smoking pattern; P = paternal smoking pattern; S = smoking onset by adolescent girls. Numerals in MPS column represent ordered triples of variable categories. Response categories for parental smoking were 1 = Non-smoker, 2 = Light/Heavy-to-light smoker, and 3 = Heavy smoker for parental smoking, and options for adolescent smoking onset were 1 = Never smoked, 2 = Smoked by age 14, and 3 = Smoking-onset unknown. L stands for Lehmacher's test with continuity correction (1981); Bonferroni-adjusted alpha (0.00185) was used. Pearson's X^2 = 70.35 for df = 20, p = .00.

Table G2

Configurations of Parental Alcoholism Patterns and Early Smoking Onset Among Adolescent Daughters

M_AP_AS	Obs. Freq.	Exp. Freq.	L	Type/Antitype
111	8	3.85	2.57	
112	0	4.05	-2.15	
113	16	9.11	2.95	Type
121	3	3.85	-.21	
122	4	4.05	.27	
123	9	9.11	.18	
131	1	3.85	-1.45	
132	7	4.05	1.48	
133	3	9.11	-2.59	
211	2	1.73	-.20	
212	1	1.83	-.27	
213	1	4.11	-1.55	
221	3	1.73	.64	
222	1	1.83	-.27	
223	5	4.11	.23	
231	1	1.73	-.20	
232	2	1.83	-.27	
233	7	4.11	1.43	

(table continues)

Table G2 (Continued)

Configurations of Parental Alcoholism Patterns and Early Smoking Onset Among Adolescent Daughters

M_AP_AS	Obs. Freq.	Exp. Freq.	L	Type/Antitype
311	0	.75	-.31	
312	0	.79	-.35	
313	0	1.79	-1.10	
321	1	.75	-.31	
322	2	.79	.85	
323	0	1.79	-1.10	
331	0	.75	-.31	
332	3	.79	2.05	
333	4	1.79	1.47	

Note. M_A = maternal alcoholism pattern; P_A = paternal alcoholism pattern; S = smoking onset by adolescent girls. Numerals in M_AP_AS column represent ordered triples of variable categories. Response categories for parental alcoholism were 1 = Non-alcoholic, 2 = Alcoholism I, and 3 = Alcoholism II. Options for adolescent smoking onset were 1 = Never smoked, 2 = Smoked by age 14, and 3 = Smoking-onset unknown. L stands for Lehmacher's test with continuity correction (1981); Bonferroni-adjusted alpha (0.00185) was used. Pearson's $X^2 = 45.58$ for df = 20, $p = .00$.

Table G3

MANOVA Results on Precursors and Concurrent Characteristics of Adolescents with Different Smoking Onset Status Among Daughters

	Maternal rating	Paternal rating
Multivariate F	1.29 – 2.63*	.83 – 3.26*
Wilk's Λ	.684* – .812	.636* – .872
Variable	Univariate analysis	
Prenatal exposure		
Daily maternal smoking	6.12* – 7.81*	
Weekly maternal drinking	1.80 – 3.08	
Early temperament (Ages 3 – 5)		
Attention span	.18 – 2.52	.10 – 1.62
Approach/Withdrawal	.03 – 3.52*	.04 – 4.28*
Reactivity	.06 – 2.00	.20 – 2.50
Early child behavior problems (Ages 3 – 5)		
Anxious/Depressed	.02 – 3.77*	.08 – 3.05
Attention problems	1.04 – 3.50*	.06 – 2.46
Delinquent behavior	2.61 – 10.37*	.87 – 5.78*
Aggressive behavior	1.74 – 6.15*	.37 – 6.07*
Concurrent adolescent behavior problems (Ages 12 – 14)		
Anxious/Depressed	.59 – 2.05	.16 – 3.15*
Attention problems	1.61 – 8.74*	2.13 – 8.83*
Delinquent behavior	2.89 – 6.06*	3.02 – 11.42*
Aggressive behavior	1.25 – 3.62*	2.09 – 7.37*

Note. * $p < .05$. Degrees of freedom for multivariate analysis of variance 1, 86 for both maternal and paternal ratings; degrees of freedom for univariate F test were 2, 85.

Table G4

Precursors and Concurrent Factors of Early Smoking Onset: Maternal Ratings of Daughters

	Smoking onset by age 14		Non-smoker		Smoking onset unknown	
Variable	Mean	SD	Mean	SD	Mean	SD
Prenatal exposure to maternal smoking and drinking						
Daily smoking	7.58	9.70	.32	1.31	2.78	5.48
Weekly drinking	.48	1.22	0.09	.35	.09	.32
Early child behavior problems (Ages 3 – 5)						
Attention span	4.37	2.94	5.85	2.76	5.38	3.75
Approach/Withdrawal	3.83	1.78	3.38	2.03	3.68	1.98
Reactivity	2.96	1.61	2.81	1.71	2.71	1.49
Child behavior problems at wave 1 (Ages 3 – 5)						
Anxious/Depressed	2.81	2.88	1.97	1.75	2.33	2.00
Attention problems	3.11	2.38	2.38	2.00	2.07	1.75
Delinquent behavior	2.29	1.85	1.79	1.58	1.22	1.28
Aggressive behavior	10.57	5.49	7.86	5.08	7.63	4.82
Concurrent adolescent behavior problems (Ages 12 – 14)						
Anxious/Depressed	4.18	4.50	2.82	2.65	3.36	2.93
Attention problems	2.65	3.23	1.25	1.85	3.18	2.66
Delinquent behavior	2.81	3.01	1.18	1.20	1.64	1.56
Aggressive behavior	8.54	6.15	5.56	4.13	6.85	4.86

Note. * $p < .05$

Table G5

Precursors and Concurrent Factors of Early Smoking Onset: Paternal Ratings of Daughters

Variable	Smoking onset by age 14		Non-smoker		Smoking onset unknown	
	Mean	SD	Mean	SD	Mean	SD
Early temperament (Ages 3 – 5)						
Attention span	5.21	2.88	5.52	3.18	5.40	2.99
Approach/Withdrawal	3.39	1.97	3.57	1.74	4.07	1.82
Reactivity	3.28	1.61	3.15	1.73	2.79	1.70
Early child behavior problems (Ages 3 – 5)						
Anxious/Depressed	2.88	2.43	2.23	2.39	2.08	2.09
Attention problems	2.80	2.18	2.39	2.22	2.37	1.75
Delinquent behavior	2.21	1.30	1.58	1.58	1.48	1.29
Aggressive behavior	10.31	5.66	7.44	5.63	8.23	5.35
Concurrent adolescent behavior problems (Ages 12 – 14)						
Anxious/Depressed	2.92	2.70	1.92	2.96	3.41	2.64
Attention problems	2.93	2.68	1.39	2.19	3.28	2.54
Delinquent behavior	2.36	1.96	.84	.88	1.51	1.56
Aggressive behavior	8.42	5.58	3.53	4.33	6.78	5.41

Note. * $p < .05$

APPENDIX H

DRINKING AND OTHER DRUG USE (PARENT)
Follow-Up Information - Form B; 12/97

This questionnaire takes about 15 minutes to complete. All information will be used for research only and will be kept strictly confidential. If you are not sure of the answer to a question please answer the best you can. Please try to answer each item.

A. THE FOLLOWING QUESTIONS ARE ABOUT YOUR DRINKING OF ALCOHOLIC BEVERAGES DURING THE PAST 6 MONTHS (that is, since last _____ to now.):

1. OVER THE LAST 6 MONTHS, ON THE AVERAGE, HOW MANY DAYS A MONTH HAVE YOU HAD A DRINK?
_____ days a month.

2. OVER THE LAST 6 MONTHS, ON A DAY WHEN YOU ARE DRINKING, HOW MANY DRINKS DO YOU USUALLY HAVE IN 24 HOURS? (A DRINK IS A 12 OZ. CAN, GLASS OR BOTTLE OF BEER; A 4 OZ. GLASS OF WINE; A SINGLE SHOT; OR A "SINGLE MIXED DRINK.")
_____ drinks per 24 hours.

3. OVER THE PAST 6 MONTHS, WHEN YOU GOT DRUNK, HOW BAD WAS YOUR HANGOVER?

_____ Never bad
_____ Not bad
_____ A little less than average
_____ Average
_____ A little more than average
_____ Pretty bad
_____ Terrible
_____ Worst possible
_____ Never drank enough to get a hangover

IF YOU DRANK NO ALCOHOLIC BEVERAGES AT ALL (NOT EVEN A FEW SIPS) IN THE LAST 6 MONTHS, GO NOW TO PAGE 6, SECTION C.

ALL OTHERS CONTINUE ON THE NEXT PAGE

B. THE FOLLOWING QUESTIONS ARE ABOUT YOUR DRINKING PATTERNS. IN ANSWERING THE QUESTIONS, PLEASE THINK ABOUT WHAT YOU HAVE DONE ON THE AVERAGE OVER THE LAST SIX MONTHS.

1. WHEN DRINKING WINE:

a. HOW OFTEN DO YOU USUALLY HAVE WINE OR A PUNCH CONTAINING WINE?

_____ 3 or more times a day
_____ 2 times a day
_____ Once a day
_____ Nearly every day
_____ 3 or 4 times a week
_____ once or twice a week
_____ 2 or 3 times a month
_____ About once a month
_____ Less than once a month, but at least once a year
_____ Less than once a year
_____ NEVER [If checked, go to question #2a]

b. THINK OF ALL THE TIMES YOU HAD WINE OR A PUNCH CONTAINING WINE RECENTLY. WHEN YOU DRINK WINE, HOW OFTEN DO YOU HAVE 10 OR MORE GLASSES?

_____ Nearly every time: SKIP TO QUESTION #2 BELOW
_____ More than half the time: SKIP TO QUESTION #2 BELOW
_____ Less than half the time
_____ Once in a while
_____ NEVER

c. WHEN YOU DRINK WINE OR A PUNCH CONTAINING WINE, HOW OFTEN DO YOU HAVE AS MANY AS 7 TO 9 GLASSES?

_____ Nearly every time: SKIP TO QUESTION #2 BELOW
_____ More than half the time: SKIP TO QUESTION #2 BELOW
_____ Less than half the time
_____ Once in a while
_____ NEVER

d. WHEN YOU DRINK WINE OR A PUNCH CONTAINING WINE, HOW OFTEN DO YOU HAVE AS MANY AS 5 to 6 GLASSES?

_____ Nearly every time: SKIP TO QUESTION #2 BELOW
_____ More than half the time: SKIP TO QUESTION #2 BELOW
_____ Less than half the time
_____ Once in a while
_____ NEVER

e. WHEN YOU DRINK WINE OR A PUNCH CONTAINING WINE, HOW OFTEN DO YOU HAVE AS MANY AS 3 to 4 GLASSES?

_____ Nearly every time: SKIP TO QUESTION #2 BELOW
_____ More than half the time: SKIP TO QUESTION #2 BELOW
_____ Less than half the time
_____ Once in a while
_____ NEVER

f. WHEN YOU DRINK WINE OR A PUNCH CONTAINING WINE, HOW OFTEN DO YOU HAVE 1 TO 2 GLASSES?

_____ Nearly every time
_____ More than half the time
_____ Less than half the time
_____ Once in a while
_____ NEVER

2. <u>WHEN DRINKING BEER</u>

a. HOW OFTEN DO YOU USUALLY HAVE BEER?

_____ 3 or more times a day
_____ 2 times a day
_____ Once a day
_____ Nearly every day
_____ 3 or 4 times a week
_____ Once or twice a week
_____ 2 or 3 times a month
_____ About once a month
_____ Less than once a month, but at least once a year
_____ Less than once a year
_____ NEVER [If checked, go to question #3a]

b. THINK OF ALL THE TIMES YOU HAD BEER RECENTLY. WHEN YOU DRINK BEER, HOW OFTEN DO YOU HAVE 10 OR MORE CANS, GLASSES OR BOTTLES?

_____ Nearly every time: SKIP TO QUESTION #3 BELOW
_____ More than half the time: SKIP TO QUESTION #3 BELOW
_____ Less than half the time
_____ Once in a while
_____ NEVER

c. WHEN YOU DRINK BEER, HOW OFTEN DO YOU HAVE AS MANY AS 7 TO 9 CANS, GLASSES OR BOTTLES?

_____ Nearly every time: SKIP TO QUESTION #3 BELOW
_____ More than half the time: SKIP TO QUESTION #3 BELOW
_____ Less than half the time
_____ Once in a while
_____ NEVER

d. WHEN YOU DRINK BEER, HOW OFTEN DO YOU HAVE AS MANY AS 5 TO 6 CANS, GLASSES OR BOTTLES?

_____ Nearly every time: SKIP TO QUESTION #3 BELOW
_____ More than half the time: SKIP TO QUESTION #3 BELOW
_____ Less than half the time
_____ Once in a while
_____ NEVER

e. WHEN YOU DRINK BEER, HOW OFTEN DO YOU HAVE AS MANY AS 3 TO 4 CANS, GLASSES OR BOTTLES?

_____ Nearly every time: SKIP TO QUESTION #3 BELOW
_____ More than half the time: SKIP TO QUESTION #3 BELOW
_____ Less than half the time
_____ Once in a while
_____ NEVER

f. WHEN YOU DRINK BEER, HOW OFTEN DO YOU HAVE 1 TO 2 CANS, GLASSES OR BOTTLES?

_____ Nearly every time
_____ More than half the time
_____ Less than half the time
_____ Once in a while
_____ NEVER

3. <u>WHEN DRINKING WHISKEY OR LIQUOR</u>

a. HOW OFTEN DO YOU USUALLY HAVE WHISKEY OR LIQUOR (SUCH AS MARTINIS, MANHATTANS, HIGHBALLS, OR STRAIGHT DRINKS INCLUDING SCOTCH, BOURBON, GIN, VODKA, RUM, ETC.)?

_____ 3 or more times a day
_____ 2 times a day
_____ Once a day
_____ Nearly every day
_____ 3 or 4 times a week
_____ Once or twice a week
_____ 2 or 3 times a month
_____ About once a month
_____ Less than once a month, but at least once a year
_____ Less than once a year
_____ NEVER [If checked, go to question #4]

b. THINK OF ALL THE TIMES YOU HAD DRINKS CONTAINING WHISKEY OR OTHER LIQUOR RECENTLY. WHEN YOU HAVE HAD THEM, HOW OFTEN DO YOU HAVE 10 OR MORE DRINKS?

_____ Nearly every time: SKIP TO QUESTION #4 BELOW
_____ More than half the time: SKIP TO QUESTION #4 BELOW
_____ Less than half the time
_____ Once in a while
_____ NEVER

c. WHEN YOU HAVE HAD DRINKS CONTAINING WHISKEY OR OTHER LIQUOR, HOW OFTEN DO YOU HAVE AS MANY AS 7 TO 9 DRINKS?

_____ Nearly every time: SKIP TO QUESTION #4 BELOW
_____ More than half the time: SKIP TO QUESTION #4 BELOW
_____ Less than half the time
_____ Once in a while
_____ NEVER

d. WHEN YOU HAVE HAD DRINKS CONTAINING WHISKEY OR OTHER LIQUOR, HOW OFTEN DO YOU HAVE AS MANY AS 5 TO 6 DRINKS?

_____ Nearly every time: SKIP TO QUESTION #4 BELOW
_____ More than half the time: SKIP TO QUESTION #4 BELOW
_____ Less than half the time
_____ Once in a while
_____ NEVER

e. WHEN YOU HAVE HAD DRINKS CONTAINING WHISKEY OR LIQUOR, HOW OFTEN DO YOU HAVE 3 TO 4 DRINKS?

_____ Nearly every time: SKIP TO QUESTION #4 BELOW
_____ More than half the time: SKIP TO QUESTION #4 BELOW
_____ Less than half the time
_____ Once in a while
_____ NEVER

f. WHEN YOU HAVE HAD DRINKS CONTAINING WHISKEY OR LIQUOR, HOW OFTEN DO YOU HAVE 1 TO 2 DRINKS?

_____ Nearly every time
_____ More than half the time
_____ Less than half the time
_____ Once in a while
_____ NEVER

4. WHEN DRINKING ANYTHING, CHECK HOW OFTEN YOU HAVE ANY DRINK CONTAINING ALCOHOL, WHETHER IT IS WINE, BEER, WHISKEY OR ANY OTHER DRINK. MAKE SURE THAT YOUR ANSWER IS NOT LESS FREQUENT THAN THE FREQUENCY REPORTED ON ANY OF THE PRECEDING QUESTIONS.

_____ 3 or more times a day
_____ 2 times a day
_____ Once a day
_____ Nearly every day
_____ 3 or 4 times a week
_____ Once or twice a week
_____ 2 or 3 times a month
_____ About once a month
_____ Less than once a month, but at least once a year
_____ Less than once a year
_____ Never

C. NOW, WE'D LIKE YOU TO SHIFT GEARS AND THINK ABOUT THE PERIOD FOR THE 2 AND A HALF YEARS BEFORE THIS YEAR

1. OVERALL DURING THAT TIME, WOULD YOU SAY YOUR DRINKING WAS PRETTY MUCH THE SAME AS IN THIS PAST 6 MONTHS, MORE THAN IN THIS PAST 6 MONTHS, OR LESS THAN IN THIS PAST 6 MONTHS?

My drinking was:

_____ A lot more than in this past 6 months
_____ Somewhat more than in this past 6 months
_____ About the same as in this past 6 months
_____ Somewhat less than in this past 6 months
_____ A lot less than in this past 6 months

2. OVER THOSE TWO AND A HALF YEARS (BETWEEN 19____ AND 19__), ON THE AVERAGE, HOW MANY DAYS A MONTH DID YOU HAVE A DRINK?

_____ days a month.
[If you did not drink at all during that time, go to section E]

3. OVER THOSE TWO AND A HALF YEARS, ON A DAY WHEN YOU WERE DRINKING, HOW MANY DRINKS DID YOU USUALLY HAVE IN 24 HOURS? (A DRINK IS A 12 OZ. CAN, GLASS OR BOTTLE OF BEER; A 4 OZ. GLASS OF WINE; A SINGLE SHOT; OR A "SINGLE MIXED DRINK.")

_____ drinks per 24 hours.

4. OVER THOSE TWO AND A HALF YEARS, WHEN YOU GOT DRUNK, HOW BAD WAS YOUR HANGOVER?

_____ Never bad
_____ Not bad
_____ A little less than average
_____ Average
_____ A little more than average
_____ Pretty Bad
_____ Terrible
_____ Worst possible
_____ Never drank enough to get hangover

5. WAS THERE ANY PERIOD IN HERE DURING WHICH YOU DID NOT DRINK AT ALL?
YES NO

IF YES:

a. For how long a time did that last?
I did not drink at all for _____ months.

b. When was this?
From _____/___ to _____/____.
(month) (yr) (month) (yr)

c. What led you to stop when you did?
_____
_____

d. What led you to begin drinking again, if you did?
_____
_____

D.

1. OVER THE LAST 3 YEARS, THINK OF THE 24 HOUR PERIOD WHEN YOU DID THE MOST DRINKING. ON THAT DAY, HOW MANY DRINKS DID YOU HAVE? (A DRINK IS A 12 OZ. CAN, GLASS OR BOTTLE OF BEER; A 4 OZ. GLASS OF WINE; A SINGLE SHOT; OR A SINGLE MIXED DRINK).

_____	30 or more drinks
_____	25 - 29 drinks
_____	20 - 24 drinks
_____	15 - 19 drinks
_____	10 - 14 drinks
_____	7 - 9 drinks
_____	5 - 6 drinks
_____	3 - 4 drinks
_____	1 - 2 drinks
_____	None

2. APPROXIMATELY WHEN DID THIS HAPPEN? _____, _____.
(month) (year)

ANSWER KEY FOR QUESTIONS BELOW:

1 2 3-5 6-10 11-20 21-50 51-100 101-250 251-500 500+ (more than 500)

E. NOW SOME QUESTIONS ABOUT OUTCOMES PEOPLE SOMETIMES HAVE BECAUSE OF DRINKING. DURING THE LAST 3 YEARS, HAVE YOU HAD ANY OF THE FOLLOWING HAPPEN BECAUSE OF YOUR DRINKING?

	YES (check one)	NO	IN THE LAST 3 YEARS: HOW MANY TIMES (Use key)*+	JUST IN THE LAST YEAR-- Last 12 months. HOW MANY TIMES? (Use key)*
1. Missed school or time on job	_____	_____	_____	_____
2. Thought I was drinking too much	_____	_____	_____	_____
3. Gone on a binge of constant drinking for 2 or more days	_____	_____	_____	_____
4. Lost friends	_____	_____	_____	_____
5. My spouse or others in my family (my parents or children) objected to my drinking	_____	_____	_____	_____
6. Felt guilty about my drinking	_____	_____	_____	_____
7. Divorce or separation	_____	_____	_____	_____

	YES (check one)	NO	IN THE LAST 3 YEARS: HOW MANY TIMES (Use key)*+	JUST IN THE LAST YEAR-- Last 12 months. HOW MANY TIMES? (Use key)*
8. Took a drink or two first thing in morning	_____	_____	_____	_____
9. Restricted my drinking to certain times of day or week in order to control it or cut down (like after 5PM, or only on weekends, or only with other people)	_____	_____	_____	_____
10. Been fired or laid off	_____	_____	_____	_____
11. Once started drinking, kept on going till completely intoxicated	_____	_____	_____	_____
12. Had a car accident when I was driving	_____	_____	_____	_____
13. Kept on drinking after I promised myself not to	_____	_____	_____	_____
14. Had to go to a hospital (other than accidents)	_____	_____	_____	_____
15. Had to stay in a hospital overnight	_____	_____	_____	_____
16. Had the shakes "the morning after"	_____	_____	_____	_____
17. Heard or saw or felt things that weren't there (hallucinations), several days after stopping drinking	_____	_____	_____	_____
18. Had blackouts (couldn't remember later what you'd done while drinking)	_____	_____	_____	_____
19. Been given a ticket for drunk driving (DWI or DUIL)	_____	_____	_____	_____
20. Had jerking or fits (convulsions) several days after stopping drinking	_____	_____	_____	_____

	YES (check one)	NO	IN THE LAST 3 YEARS: HOW MANY TIMES (Use key)*+	JUST IN THE LAST YEAR-- Last 12 months. HOW MANY TIMES? (Use key)*
21. Been given a ticket for public intoxication, drunk and disorderly or other non-driving alcohol arrest	_____	_____	_____	_____
22. Had the D.T.'s (delirium tremens, shakes, sweating, rapid heart, etc.) within 2 - 3 days after stopping drinking	_____	_____	_____	_____
23. Found that I had a strong craving for a drink at some time each day	_____	_____	_____	_____
24. Needed to drink a lot more in order to get an effect, or found that I no longer could get high on the amount I used to drink	_____	_____	_____	_____
25. Found that I was able to drink a lot more than I used to before I would get drunk	_____	_____	_____	_____
26. Had days where I drank much more that I expected to when I began	_____	_____	_____	_____
27. Found that I often continued drinking for more days in a row than I had planned to	_____	_____	_____	_____
28. Found that I tended to gulp my drinks rather than just drink them	_____	_____	_____	_____
29. Been arrested for a drinking related offense	_____	_____	_____	_____

	YES (check one)	NO	IN THE LAST 3 YEARS: HOW MANY TIMES (Use key)*+	JUST IN THE LAST YEAR-- Last 12 months. HOW MANY TIMES? (Use key)*
30. Been court ordered to get alcohol treatment	_____	_____	_____	_____
31. Been put on probation or parole for a drinking related offense.	_____	_____	_____	_____

F. THE LAST SECTIONS OF THIS QUESTIONNAIRE DEAL WITH YOUR USE OF VARIOUS DRUGS OTHER THAN ALCOHOL. WE HOPE THAT YOU CAN ANSWER ALL QUESTIONS; BUT IF YOU FIND ONE WHICH YOU FEEL YOU CANNOT ANSWER HONESTLY, WE WOULD PREFER THAT YOU LEAVE IT BLANK.

REMEMBER THAT YOUR ANSWERS WILL BE KEPT STRICTLY CONFIDENTIAL AND THEY ARE NEVER CONNECTED WITH YOUR NAME. THAT IS WHY THIS QUESTIONNAIRE IS IDENTIFIED ONLY WITH A CODE NUMBER.

THE FOLLOWING QUESTIONS ARE ABOUT CIGARETTES (CHECK THE BEST ANSWER):

1. HAVE YOU SMOKED CIGARETTES DURING THE PAST 3 YEARS?

_____ Never (GO TO SECTION G on page 13)
_____ Once or twice
_____ Occasionally but not regularly
_____ Regularly for a while during this year, but not now.
_____ Regularly now

2. HAVE YOU SMOKED CIGARETTES DURING THE PAST 12 MONTHS?

_____ Never (GO TO QUESTION 4)
_____ Once or twice
_____ Occasionally but not regularly
_____ Regularly for a while during this year, but not now.
_____ Regularly now

3. HOW FREQUENTLY HAVE YOU SMOKED CIGARETTES DURING THE PAST 30 DAYS?

_____ Not at all
_____ Less than one cigarette per day
_____ One to five cigarettes per day
_____ About one-half pack per day
_____ About one pack per day
_____ About one and one-half packs per day
_____ Two packs or more per day

(ANSWER QUESTIONS 4-9 FOR THE MOST RECENT TIME YOU HAVE BEEN SMOKING.)

4. How soon after you wake up do you smoke your first cigarette? **(Circle one answer)**
   - Within 5 minutes 3
   - 6-30 minutes 2
   - 31-60 minutes 1
   - After 60 minutes 0

5. Which cigarette would you hate most to give up? **(Circle one answer)**
   - The first one in the morning 1
   - Any others 0

6. How many cigarettes a day do you smoke? **(Circle one answer)**
   - 10 or less 0
   - 11-20 1
   - 21-30 2
   - 31 or more 3

		(Circle one answer) YES	NO
7.	Do you find it difficult to refrain from smoking in places where it is forbidden, such as in church, the library, or the theater?	1	0
8.	Do you smoke more frequently during the first hours after waking than during the rest of the day?	1	0
9.	Do you smoke if you are so ill that you are in bed most of the day?	1	0

G. THE FOLLOWING QUESTIONS ARE ALL ABOUT NON-PRESCRIPTION USE OF DRUGS, EITHER FOR RECREATION OR FOR SELF-MEDICATION

(MARK ONE SPACE FOR EACH LINE).

1. ON HOW MANY OCCASION (IF ANY) HAVE YOU USED MARIJUANA (GRASS, POT) OR HASHISH (HASH, HASH OIL)	0 occasions	1-2 occasions	3-5 occasions	6-9 occasions	10-19 occasions	20-39 occasions	40-99 occasions	100-249 occasions	250-499 occasions	500 & above
During the last 3 years?	( )	( )	( )	( )	( )	( )	( )	( )	( )	( )
During the last 12 mos?	( )	( )	( )	( )	( )	( )	( )	( )	( )	( )
During the last 30 days?	( )	( )	( )	( )	( )	( )	( )	( )	( )	( )

2. ON HOW MANY OCCASION (IF ANY)HAVE YOU USED LSD (ACID).	0 occasions	1-2 occasions	3-5 occasions	6-9 occasions	10-19 occasions	20-39 occasions	40-99 occasions	100-249 occasions	250-499 occasions	500 & above
During the last 3 years?	( )	( )	( )	( )	( )	( )	( )	( )	( )	( )
During the last 12 mos?	( )	( )	( )	( )	( )	( )	( )	( )	( )	( )
During the last 30 days?	( )	( )	( )	( )	( )	( )	( )	( )	( )	( )

3. ON HOW MANY OCCASIONS (IF ANY) HAVE YOU USED PSYCHEDELICS OTHER THAN LSD (LIKE MESCALINE, PEYOTE, PSILOCYBIN, PCP)	0 occasions	1-2 occasions	3-5 occasions	6-9 occasions	10-19 occasions	20-39 occasions	40-99 occasions	100-249 occasions	250-499 occasions	500 & above
During the last 3 years?	( )	( )	( )	( )	( )	( )	( )	( )	( )	( )
During the last 12 mos?	( )	( )	( )	( )	( )	( )	( )	( )	( )	( )
During the last 30 days?	( )	( )	( )	( )	( )	( )	( )	( )	( )	( )

4. ON HOW MANY OCCASIONS (IF ANY) HAVE YOU USED COCAINE (COKE, CRACK, ROCK COCAINE, SNOW)	0 occasions	1-2 occasions	3-5 occasions	6-9 occasions	10-19 occasions	20-39 occasions	40-99 occasions	100-249 occasions	250-499 occasions	500 & above
During the last 3 years?	( )	( )	( )	( )	( )	( )	( )	( )	( )	( )
During the last 12 mos?	( )	( )	( )	( )	( )	( )	( )	( )	( )	( )
During the last 30 days?	( )	( )	( )	( )	( )	( )	( )	( )	( )	( )

4a. Which of the following is the way that most accurately describes how you use coke?
(Please circle only one answer)

a. All or mostly nasal (snorting).
b. All or mostly smoking crack.
c. All or mostly freebase.
d. Both nasal and smoking crack.
e. Both nasal and freebase.
f. Both smoking crack and freebase.

(MARK ONE SPACE FOR EACH LINE)

5.
AMPHETAMINES ARE SOMETIMES PRESCRIBED BY DOCTORS TO HELP PEOPLE LOSE WEIGHT OR TO GIVE PEOPLE MORE ENERGY. THEY ARE SOMETIMES CALLED UPPERS, UPS, SPEED, CRYSTAL, CRANK, BENNIES, DEXIES, PEP PILLS AND DIET PILLS.

ON HOW MANY OCCASIONS (IF ANY) HAVE YOU TAKEN AMPHETAMINES ON YOUR OWN--THAT IS, WITHOUT A DOCTOR TELLING YOU TO TAKE THEM.	0 occasions	1-2 occasions	3-5 occasions	6-9 occasions	10-19 occasions	20-39 occasions	40-99 occasions	100-249 occasions	250-499 occasions	500 & above
During the last 3 years?	( )	( )	( )	( )	( )	( )	( )	( )	( )	( )
During the last 12 mos?	( )	( )	( )	( )	( )	( )	( )	( )	( )	( )
During the last 30 days?	( )	( )	( )	( )	( )	( )	( )	( )	( )	( )

6.

ON HOW MANY OCCASIONS (IF ANY) HAVE YOU USED QUAALUDES (QUADS, SOAPERS, METHAQUALONE) ON YOUR OWN--THAT IS, WITHOUT A DOCTOR TELLING YOU TO TAKE THEM.	0 occasions	1-2 occasions	3-5 occasions	6-9 occasions	10-19 occasions	20-39 occasions	40-99 occasions	100-249 occasions	250-499 occasions	500 & above
During the last 3 years?	( )	( )	( )	( )	( )	( )	( )	( )	( )	( )
During the last 12 mos?	( )	( )	( )	( )	( )	( )	( )	( )	( )	( )
During the last 30 days?	( )	( )	( )	( )	( )	( )	( )	( )	( )	( )

7.
BARBITURATES ARE SOMETIMES PRESCRIBED BY DOCTORS TO HELP PEOPLE RELAX OR GET TO SLEEP. THEY ARE SOMETIMES CALLED DOWNS, DOWNERS, GOOFBALLS, YELLOWS, REDS, BLUES, RAINBOWS.

ON HOW MANY OCCASIONS (IF ANY) HAVE YOU TAKEN BARBITURATES ON YOUR OWN-- THAT IS, WITHOUT A DOCTOR TELLING YOU TO TAKE THEM.	0 occasions	1-2 occasions	3-5 occasions	6-9 occasions	10-19 occasions	20-39 occasions	40-99 occasions	100-249 occasions	250-499 occasions	500 & above
During the last 3 years?	( )	( )	( )	( )	( )	( )	( )	( )	( )	( )
During the last 12 mos?	( )	( )	( )	( )	( )	( )	( )	( )	( )	( )
During the last 30 days?	( )	( )	( )	( )	( )	( )	( )	( )	( )	( )

(MARK ONE SPACE FOR EACH LINE)

8.
TRANQUILIZERS ARE SOMETIMES PRESCRIBED BY DOCTORS TO CALM PEOPLE DOWN, QUITE THEIR NERVES, OR RELAX THEIR MUSCLES. LIBRIUM, VALIUM, AND MILTOWN ARE ALL TRANQUILIZERS.

ON HOW MANY OCCASIONS (IF ANY) HAVE YOU TAKEN TRANQUILIZERS ON YOUR OWN--THAT IS, WITHOUT A DOCTOR TELLING YOU TO TAKE THEM.	0 occasions	1-2 occasions	3-5 occasions	6-9 occasions	10-19 occasions	20-39 occasions	40-99 occasions	100-249 occasions	250-499 occasions	500 & above
During the last 3 years?	( )	( )	( )	( )	( )	( )	( )	( )	( )	( )
During the last 12 mos?	( )	( )	( )	( )	( )	( )	( )	( )	( )	( )
During the last 30 days?	( )	( )	( )	( )	( )	( )	( )	( )	( )	( )

9.

ON HOW MANY OCCASIONS (IF ANY) HAVE YOU USED HEROIN (SMACK, HORSE, SKAG).	0 occasions	1-2 occasions	3-5 occasions	6-9 occasions	10-19 occasions	20-39 occasions	40-99 occasions	100-249 occasions	250-499 occasions	500 & above
During the last 3 years?	( )	( )	( )	( )	( )	( )	( )	( )	( )	( )
During the last 12 mos?	( )	( )	( )	( )	( )	( )	( )	( )	( )	( )
During the last 30 days?	( )	( )	( )	( )	( )	( )	( )	( )	( )	( )

10.
THERE ARE A NUMBER OF NARCOTICS OTHER THAN HEROIN, SUCH AS METHADONE, OPIUM, MORPHINE, CODEINE, DEMEROL, PAREGORIC, TALWIN, AND LAUDANUM. THESE ARE SOMETIMES PRESCRIBED BY DOCTORS.

ON HOW MANY OCCASIONS (IF ANY) HAVE YOU TAKEN NARCOTICS OTHER THAN HEROIN ON YOUR OWN – THAT IS, WITHOUT A DOCTOR TELLING YOU TO TAKE	0 occasions	1-2 occasions	3-5 occasions	6-9 occasions	10-19 occasions	20-39 occasions	40-99 occasions	100-249 occasions	250-499 occasions	500 & above
During the last 3 years?	( )	( )	( )	( )	( )	( )	( )	( )	( )	( )
During the last 12 mos?	( )	( )	( )	( )	( )	( )	( )	( )	( )	( )
During the last 30 days?	( )	( )	( )	( )	( )	( )	( )	( )	( )	( )

11.

ON HOW MANY OCCASION (IF ANY) HAVE YOU SNIFFED GLUE, OR BREATHED THE CONTENTS OF AEROSOL SPRAY CANS, OR INHALED ANY OTHER GASES OR SPRAYS IN ORDER TO GET HIGH	0 occasions	1-2 occasions	3-5 occasions	6-9 occasions	10-19 occasions	20-39 occasions	40-99 occasions	100-249 occasions	250-499 occasions	500 & above
During the last 3 years?	( )	( )	( )	( )	( )	( )	( )	( )	( )	( )
During the last 12 mos?	( )	( )	( )	( )	( )	( )	( )	( )	( )	( )
During the last 30 days?	( )	( )	( )	( )	( )	( )	( )	( )	( )	( )

ANSWER KEY FOR QUESTIONS BELOW:

1 2 3-5 6-10 11-20 21-50 51-100 101-250 251-500 500+ (more than 500)

H. NOW SOME QUESTIONS ABOUT NONPRESCRIPTION USE OF DRUGS. DURING THE LAST 3 YEARS, HAVE YOU HAD ANY OF THE FOLLOWING OUTCOMES BECAUSE OF YOUR USE OF NONPRESCRIPTION DRUGS ASKED ABOUT IN SECTION G. (The last section). (Use the answer key at the top of page for column 3 & 4)

	YES (check one)	NO	IN THE LAST 3 YEARS: HOW MANY TIMES (Use key)*+	JUST IN THE LAST YEAR--Last 12 months. HOW MANY TIMES? (Use key)*
1. Missed school or time on job	_____	_____	_____	_____
2. Lost friends	_____	_____	_____	_____
3. Been divorced or separated	_____	_____	_____	_____
4. Been fired or laid off	_____	_____	_____	_____
5. Had a car accident when you were driving	_____	_____	_____	_____
6. Had to go to a hospital (other than accidents)	_____	_____	_____	_____
7. Had to stay in hospital overnight	_____	_____	_____	_____
8. Had to see a doctor because of drug use (unintentional overdose) or had a doctor say drugs had harmed your health	_____	_____	_____	_____
9. Gone through physical withdrawal from drugs	_____	_____	_____	_____
10. Been arrested more than once for possession or sale of drugs other than marijuana	_____	_____	_____	_____
10a. Been arrested for any drug related offense	_____	_____	_____	_____
10b. Been court ordered to get substance abuse treatment	_____	_____	_____	_____
10c. Been put on probation or parole for a drug related offense	_____	_____	_____	_____

11a. DURING THE PAST 3 YEARS HAVE YOU TAKEN DRUGS INTRAVENOUSLY (USING A NEEDLE)? DON'T COUNT SHOTS YOU WERE GIVEN BY A DOCTOR OR NURSE OR SHOTS YOU MAY HAVE TAKEN FOR TREATMENT OF DIABETES.

_____NO _____YES

IF YES, WHAT ABOUT IN THE LAST 12 MONTHS?

11b. NO_____ YES_____

11c. IF YES, (to 11a or 11b) WHAT DRUGS HAVE YOU TAKEN INTRAVENOUSLY (IV)?

SECTION I. TREATMENT PROGRAMS

These next questions ask about your experiences getting help for problems with drugs or alcohol during the past three years.

1. During the past 3 years, have you been in a **formal** treatment program for alcohol or drug problems?

(Circle One)

Yes 1
No 2

1a. How many **times**? ..... _____ times.
(If none, enter 0)

1b. What were your age(s) when you were in a formal program for alcohol or drug problems? (e.g. 34-36, 27) ..... _____age(s).

2. During the past 3 years have you attended a **self-help group** like Alcoholics Anonymous (AA) or Narcotics Anonymous (NA) or another similar self-help group for alcohol or drug problems?

(Circle One)

Yes 1
No ..... 2 [Go to Question 3]

2a. What were your age(s) when you attended such a group?_____age(s).

2b. Overall, how many times have you attended such a group? (Circle One)

1-2 3-5 6-10 11→

2c. When was the most recent time? ..... _____, _____.
month year

2d. Have you asked for **or** received sponsorship in AA at any time?

(Circle One)

Yes 1
No 2

3. During the past 3 years have you been in an outpatient therapy, or other formal treatment program for emotional or mental health problems other than drugs or alcohol?

(Circle One)

Yes 1
No 2

3a. How many different **times** during the past 3 years?_____times
(If none, enter 0)

3b. What were your age(s) when you were in such therapy? .....
_____age(s).

3c. Overall, during the past three years, how many sessions of treatment have you had? ..... _____sessions

DEMOGRAPHIC BACKGROUND INFO (PARENT)
MSU-UM Family Project; 5/01

Background Information

We would like to ask you a few questions about yourself. The questions ask about your life during the time you were growing up as well as now. Please answer all of them as completely as possible. (PLEASE PRINT)

1. What is your date of birth?

_____ _____ _____
MONTH DAY YEAR

2. Where were you born?

_____ _____ _____
CITY/TOWN (COUNTY IF RURAL) STATE COUNTRY (IF NOT U.S.)

Number 3 intentionally left out.

4. Until you were 18, about how many times did your family move.
CIRCLE ONE 0 1 2 3 4 5 6 7 or more.

5a. Did you live together with both of your natural parents for most of the time from birth to 18? CIRCLE ONE.
YES (If Yes, go to question 6) NO (If No, go to question 5b)

5b. What was the main reason your parents did not live together with you during that time? CIRCLE ONE
1. Mother died
2. Father died
3. Both parents died
4. Parents divorced or separated
5. Parents never lived together
6. Other (Please explain) _____

5c. Which adult(s) did you live with most of the time from birth to 18? CIRCLE ONE
1. Mother, but no adult male
2. Father, but no adult female
3. Mother and step-father
4. Father and step-mother
5. Other (Please explain) _____

6. Who was the main wage earner in your home while you were growing up? CHECK ONE
a) your father _____
b) your mother _____
c) someone else _____ What was their relationship to you_____

ABOUT YOUR NATURAL (BIOLOGICAL) FATHER

7a. Where was he born? _____ _____
State Country (If not U.S.A.)

ABOUT THE ADULT MALE WHO LIVED WITH YOU MOST OF THE TIME UNTIL YOU WERE 18. (This could be your natural father, or stepfather, or someone else.)

7b. What kind of work did this adult male do (the adult male who lived with you most of the time until you were 18?) That is what was his occupation?

_____

(For example: electrical engineer, machinist, stock clerk, assembly line worker, farmer)

7c. What were his most important activities or duties?

_____

(For example: keep account books, filing, selling cars, operate printing press, finish concrete)

7d. What kind of business or industry was this?

_____

(For example: TV and radio mfg., retail shoe store, automobile manufacturing, [Oldsmobile], State Labor Dept., farm work.

7e. What was the highest grade of school he completed? CIRCLE THE HIGHEST GRADE COMPLETED

None	0							
Elementary	1	2	3	4	5	6	7	8
High School	9	10	11	12				
College	1	2	3	4	Degree? _____			
Graduate School	5	6	7	8+	Degree? _____			

7f. Would your father identify as Latino/Hispanic/Spanish? CIRCLE ONE

a) No
b) Yes: Mexican, Mexican American, Chicano
c) Yes: Puerto Rican
d) Yes: Cuban, Cuban-American
e) Yes: Central American
f) Yes: Other Latino/Hispanic/Spanish group (print group): _____

7g. Which of the following best identifies your father's race? CIRCLE ONE

a) White
b) Black, African American
c) Native American, American Indian
d) Asian American, Pacific Islander
e) Some other race (please print): _____

ABOUT YOUR NATURAL (BIOLOGICAL) MOTHER

8a. Where was she born? _____ _____
State Country -- If not U.S.A.

ABOUT THE ADULT FEMALE WHO LIVED WITH YOU MOST OF THE TIME UNTIL YOU WERE 18.
(This could be your natural mother, or stepmother, or someone else.)

8b. What kind of work did this adult female do (the adult female who lived with you most of the time until you were 18?) That is what was her occupation?

_____
(For example: electrical engineer, file clerk, assembly line worker, bookkeeper, sales clerk)

8c. What were her most important activities or duties?

_____
(For example: keep account books, filing, selling clothes, teach fifth graders)

8d. What kind of business or industry was that?

_____
(For example: TV and radio mfg., retail shoe store, automobile manufacturing [Oldsmobile], state labor dept.)

8e. What was the highest grade of school she completed? CIRCLE THE HIGHEST GRADE COMPLETED

None	0							
Elementary	1	2	3	4	5	6	7	8
High School	9	10	11	12				
College	1	2	3	4	Degree? _____			
Graduate School	5	6	7	8+	Degree? _____			

AGAIN, A QUESTION ABOUT YOUR NATURAL (BIOLOGICAL) MOTHER:

8f. Would your mother identify as Latino/Hispanic/Spanish? CIRCLE ONE

a) No
b) Yes: Mexican, Mexican American, Chicano
c) Yes: Puerto Rican
d) Yes: Cuban, Cuban-American
e) Yes: Central American
f) Yes: Other Latino/Hispanic/Spanish group (print group): _____

8g. Which of the following best identifies your mother's race? CIRCLE ONE

a) White
b) Black, African American
c) Native American, American Indian
d) Asian American, Pacific Islander
e) Some other race (please print): _____

9x. Do you identify as Latino/Hispanic/Spanish? CIRCLE ONE

a) No
b) Yes: Mexican, Mexican American, Chicano
c) Yes: Puerto Rican
d) Yes: Cuban, Cuban-American
e) Yes: Central American
f) Yes: Other Latino/Hispanic/Spanish group (print group): _____

9y. Which of the following best identifies your race? CIRCLE ONE

a) White
b) Black, African American
c) Native American, American Indian
d) Asian American, Pacific Islander
e) Some other race (please print:) _____

9z. Until you were 18, what religion was practiced in your home most of the time? CIRCLE ONE

88	None	21	Episcopalian
1	Buddhist	22	Full Gospel (Tabernacle)
2	Christian Scientist	23	Fundamentalist
3	Hindu	24	Lutheran
4	Islam	25	Methodist
5	Jehovah's Witness	26	Moravian
6	Jewish	27	Nazarene
7	Mormon (Latter Day Saints)	28	Pentecostal
8	Orthodox (Eastern, Greek, Russian, etc.)	29	Presbyterian
12	Other Eastern (e.g. Shinto, Taoism)	31	Quaker
13	Roman Catholic	32	Reformed Church
14	Assembly of God	33	Dutch Reformed Church
15	Baptist	34	Seventh Day Adventist
16	Church of Brethren	35	Unitarian
17	Church of Christ	36	United Brethren
18	Church of God	37	Wesleyan
19	Congregational	98	Other (name)
20	Disciples of Christ		

_____
(Name of "other")

9c. Until you were 18, how often did you attend religious services? CIRCLE ONE

1. several times a week
2. about once a week
3. 2-3 times a month
4. once a month or less
5. never

10z. Please circle the denomination/type of church that best represents your religious preference now.

88	None	21	Episcopalian
1	Buddhist	22	Full Gospel (Tabernacle)
2	Christian Scientist	23	Fundamentalist
3	Hindu	24	Lutheran
4	Islam	25	Methodist
5	Jehovah's Witness	26	Moravian
6	Jewish	27	Nazarene
7	Mormon (Latter Day Saints)	28	Pentecostal
8	Orthodox (Eastern, Greek, Russian, etc.)	29	Presbyterian
12	Other Eastern (e.g. Shinto, Taoism)	31	Quaker
13	Roman Catholic	32	Reformed Church
14	Assembly of God	33	Dutch Reformed Church
15	Baptist	34	Seventh Day Adventist
16	Church of Brethren	35	Unitarian
17	Church of Christ	36	United Brethren
18	Church of God	37	Wesleyan
19	Congregational	98	Other (name)
20	Disciples of Christ		

_____
(Name of "other")

10c. About how often did you attend religious services in the last year? CIRCLE ONE

1. several times a week
2. about once a week
3. 2-3 times a month
4. once a month or less
5. never

10d. Regardless of your attendance at religious services, how religious do you consider yourself to be?

1. not religious at all
2. not very religious
3. fairly religious
4. very religious

11. What is the highest grade of school you have completed? CIRCLE THE HIGHEST GRADE COMPLETED.

None	0	
Elementary	1 2 3 4 5 6 7 8	
High School	9 10 11 12	
	POST HIGH SCHOOL	
College	1 2 3 4	Degree _____
Graduate School	5 6 7 8+	Degree _____
Vo-Tech School	1 2 3 4	Certificate _____

12a. What kind of work are you doing now? (What is your occupation?)

_____

_____

(For example: Electrical engineer, machinist, stock clerk, assembly line worker, teacher, farmer).

12b. What are your most important activities or duties?

_____

(For example: keep account books, filing, selling cars, operate printing press, finish concrete, teach fifth graders, answer phone).

12c. What kind of business or industry is this?

_____

(For example: TV and radio manufacturing, retail shoe store, automobile manufacturing [Oldsmobile], State Labor Department, farm work)

12d. Are you: (Check one)

An employee of a PRIVATE company, business or individual [who works] for wages, salary or commission? _____

A GOVERNMENT employee (federal, state, county, or local government? _____

Self-employed in OWN business, professional practice, or farm?

own business **not incorporated** _____

own business **incorporated** _____

working **without pay** in a family business or farm _____

12e. Approximately what is your present annual <u>family</u> income?

CIRCLE ONE

1.	$4,000 or under	6.	$16,001 -- $20,000
2.	$ 4,001 -- $ 7,000	7.	$20,001 -- $30,000
3.	$ 7,001 -- $10,000	8.	$30,001 -- $50,000
4.	$10,001 -- $13,000	9.	$50,001 -- $75,000
5.	$13,001 -- $16,000	10.	$75,000 -- $100,000
		11.	Over $100,000

12e1. How often do you have problems paying for basic necessities like food, clothing and rent?

1. Hardly ever 2. Sometimes 3. Often

Compared to other people, do you have enough money to pay for:

		More than enough	Just enough	Not Enough
12e2a.	The food you need?	1	2	3
12e2b.	The clothing you need?	1	2	3
12e2c.	The medical care you need?	1	2	3

12e3. How would you describe your family's money situation while you were growing up?

1. Very poor, not enough to get by.
2. Had enough to get by, but that's all.
3. Had more than enough to get by.

12f. How many months out of the last 3 years have you been without a regular paid job? **(DO** count months you were retired, in school full-time, a home maker or too ill to work) **(Your answer may range from 0-36).** _____ months.

12g. Please list the jobs you have had in the last three years as well as the periods during which you were not working. Start with your current job (or if not working, your current activities) and work backwards. We do not need to know who your employer was, but list your approximate dates of employment and what type of work you were doing. For each different employment, list (1) type of work/occupation and (2) most important job duties.

DATES OF EMPLOYMENT		TYPE OF WORK/OCCUPATION
month/year	month/year	
(a)_____	to _____	_____
(b)_____	to _____	_____
(c)_____	to _____	_____
(d)_____	to _____	_____

NOW ABOUT YOUR MARITAL STATUS

13. How many times have you been married? CIRCLE ONE
0 1 2 3 4+

13a. Which answer best fits your current marital situation? CIRCLE ONLY ONE
1. Married or living a partner
2. Divorced
3. Separated

14a. What was the date of your marriage to your (present) spouse?
_____

14b. If married more than once, what was the date of your first marriage?
_____

NOW SOME QUESTIONS ABOUT ALL YOUR CHILDREN

15a. List all biological children (children born to you) from all relationships including your current marriage or relationship, as well as all previous ones. * **"Lives with you now"** means with you all the time or most of the time. If you are primary custodial parent or share custody equally, circle yes ("Y") for "lives with you now."

FIRST NAME ONLY	BIRTH DATE mo/day/yr	SEX (circle one)	LIVES WITH YOU NOW* (circle one)	DECEASED (GIVE DATE)
1. _____	_____	M / F	Y / N	_____
2. _____	_____	M / F	Y / N	_____
3. _____	_____	M / F	Y / N	_____
4. _____	_____	M / F	Y / N	_____
5. _____	_____	M / F	Y / N	_____

15b. Now circle the names of the biological children who are from your present marriage or relationship. If all are from your present marriage or relationship, mark a check here _____.

15c. Now list all the other (nonbiological) children you have from another marriage or relationship.

FIRST NAME ONLY	BIRTH DATE mo/day/year	SEX (circle one)	TYPE OF RELATIONSHIP (step, adopted, foster, relative, etc.)
1._____	_____	M / F	_____
2._____	_____	M / F	_____
3._____	_____	M / F	_____
4._____	_____	M / F	_____
5._____	_____	M / F	_____
6._____	_____	M / F	_____
7._____	_____	M / F	_____
8._____	_____	M / F	_____
9._____	_____	M / F	_____
10._____	_____	M / F	_____

Many of us have had adventures during our lives...times that were exciting and carefree, even though they may have been a bit impulsive or happy-go-lucky. Please read each of the following items. Indicate (with a check) if you have ever done any of the following activities and how often.

NEVER	RARELY	SOMETIMES	OFTEN	NEVER - You have never done this RARELY - Once or twice in your life SOMETIMES - Three (3) to nine (9) times in your life OFTEN - More than ten (10) times in your life
				1. Skipped school without a legitimate excuse for more than 5 days in one school year.
				2. Been suspended or expelled from school for fighting.
				3. Been suspended or expelled from school for reasons other than fighting.
				4. Lied to a teacher or principal.
				5. Cursed at a teacher or principal (to their face).
				6. Hit a teacher or principal.
				7. Repeated a grade in school.
				8. Taken part in a gang fight.
				9. "Beaten up" another person.
				10. Broken street lights, car windows, or car antennas just for the fun of it.
				11. Gone for a ride in a car someone else stole.
				12. Teased or killed an animal (like a dog or cat) just for the fun of it.
				13. Defied your parent's authority (to their face).
				14. Hit your parents.
				15. Cursed at your parents (to their face).
				16. Stayed out overnight without your parent's permission.

				17. Run away from home for more than 24 hours.
				18. Lied to your parents.
				19. Snatched a women's purse.
				20. Rolled drunks just for the fun of it.
				21. Shoplifted merchandise valued over $25.
				22. Shoplifted merchandise valued under $25.
				23. Received a speeding ticket.
				24. Been questioned by the police.
				25. Taken part in a robbery.
				26. Taken part in a robbery involving physical force or a weapon.
				27. Been arrested for a felony.
				28. Resisted arrest.
				29. Been arrested for any other non-traffic police offenses (except fighting or a felony).
				30. Been convicted or any non-traffic police offense.
				31. Defaulted on a debt.
				32. Passed bad checks for the fun of it.
				33. Ever used an alias.
				34. Gone AWOL from the military.
				35. Received a bad conduct or undesirable discharge from the military.
				36. Performed sexual acts for money.
				37. This item was deleted.
				38. Had intercourse with more than one person in a single day.
				39. "Fooled around" with other women/men after you were married.
				40. Hit your husband/wife during an argument.
				41. Lied to your spouse.
				42. Spent six months without any job or permanent home.

				43. Been fired for excessive absenteeism.
				44. Been fired for poor job performance (except absenteeism).
				45. Changed jobs more than 3 times in one year.
				46. Lied to your boss.

Thank you very much for your cooperation.

HEALTH HISTORY QUESTIONNAIRE - FORM W

The following questions are mainly about your medical history, health history and health habits. At the start, there also are some questions about your child _____, that ask about the pregnancy and early developmental history. Please complete each item carefully. If you have questions about any item, ask the interviewer. Remember that all information is confidential and will not be disclosed to anyone.

I. Pregnancy History

During your pregnancy with _____, did you:

1. Ever have high blood pressure? ..... YES( ) NO( )
2. Have diabetes, or have sugar in your urine? ..... YES( ) NO( )
3. Have albumin or protein in your urine? ..... YES( ) NO( )
4. Have toxemia? ..... YES( ) NO( )
5. Have any infections? ..... YES( ) NO( )

   If yes, please

   specify_____
6. Have German (3 day) measles? ..... YES( ) NO( )
7. Take medicines prescribed by your doctor? ..... YES( ) NO( )

   If yes, what medications?_____
8. Did you smoke cigarettes? ..... YES( ) NO( )

   If yes, about how many cigarettes a day? _____per day
9. Have a venereal disease such as gonorrhea, syphilis or herpes? .....

   YES( ) NO( ) DON'T KNOW( )

   If yes, please specify_____
10. Did you drink alcoholic beverages? ..... YES( ) NO( )

    If yes, about how many drinks per day_____

    per week_____
11. Did you use any nonprescription drugs? ..... YES( ) NO( )

    If yes, what drugs?_____

12. During your pregnancy with _____, did you threaten to miscarry or have premature labor? ..... YES( ) NO( )

If yes, please explain_____

_____

13. Get hurt or injured? ..... YES( ) NO( )

If yes, please explain_____

_____

14. Have Rh or other blood group incompatibility?

YES( ) NO( ) DON'T KNOW( )

15. Have other problems, diseases or conditions? ..... YES( ) NO( )

If yes, please explain_____

_____

16. How long was your pregnancy? _____months.

17. How early did you start seeing a doctor? Starting at _____months.

18. What was your child's weight at birth? ..... _____lb. _____oz.

19. Was your labor longer than 12 hours? ..... YES( ) NO( )

20. Was your labor less than 2 hours? ..... YES( ) NO( )

21. Did you have a difficult deliver? ..... YES( ) NO( )

If yes, please explain_____

_____

22. Was it a breech (bottom first) deliver? ..... YES( ) NO( )

23. Was it a caesarean delivery? ..... YES( ) NO( )

24. Did you have a multiple birth (twins or triplets)? ..... YES( ) NO( )

25. Were you given an anaesthetic for the delivery? ..... YES( ) NO( )

If so, what_____

26. Have you had premature births, miscarriages or stillbirths?

..... YES( ) NO( )

II. Delivery and Newborn History During _____'s delivery:

1. Was she born with the cord around her neck? ..... YES( ) NO( )
2. Was she injured during birth? ..... YES( ) NO( )
3. Was anything wrong with your child at birth? ..... YES( ) NO( )
   If yes, what?_____
   _____

During _____'s newborn period (4 weeks): did she:

4. Have any breathing problems? ..... YES( ) NO( )
   If yes, please explain_____
   _____
5. Need to receive oxygen? ..... YES( ) NO( )
6. Turn blue (cyanosis)? ..... YES( ) NO( )
7. Turn yellow (jaundice)? ..... YES( ) NO( )
   If yes, did she receive: blood transfusions .... YES( ) NO( )
   phototherapy (lights) ..... YES( ) NO( )
8. Have any infections? ..... YES( ) NO( )
   If so, what were they? _____
9. Receive medication? ..... YES( ) NO( )
   If so, what kind? _____
10. Have seizures (fits, convulsions)? ..... YES( ) NO( )
11. Have feeding problems? ..... YES( ) NO( )
12. Was _____ born with any birth defects? ..... YES( ) NO( )
    If so, what _____
13. Did _____ have any other problems? ..... YES( ) NO( )
    If yes, please explain_____
    _____
14. Was she born in a hospital? ..... YES( ) NO( )
    If yes, what hospital? _____
    address: (city and state)_____

15. What kinds of doctor(s) or clinic(s) have provided your child's health care?

_____	_____
_____	_____
_____	_____
_____	_____

16. Up to what age was your child breast-fed?

( ) My child was not breast-fed

( ) My child was breast-fed until the age of _____ months.

17. Have you had <u>any</u> premature births? ..... YES( ) NO( )

18. Have you had <u>any</u> caesarean births? ..... YES( ) NO( )

This questionnaire takes about 20 minutes. All information will be used for research only and will be kept strictly confidential. If you are not sure of the answer to a question, please answer the best you can. Please try to answer each item. These questions are to find out how you feel about drinking, drug use, and other topics having to do with your attitudes and behavior. Please remember that no one will see your answers except members of the research staff.

A. THE FOLLOWING QUESTIONS ASK ABOUT YOUR EXPERIENCE DRINKING ALCOHOLIC BEVERAGES, (BEER, WINE, AND LIQUOR):

1. HOW OLD WERE YOU THE FIRST TIME YOU EVER TOOK A DRINK? DO NOT COUNT THE TIMES WHEN YOU WERE GIVEN A "SIP" BY AN ADULT.

_____ years old.

IF YOU'VE NEVER TAKEN A DRINK AT ALL, GO TO PAGE 11 (SECTION E), QUESTION 1.

---

2. OVER THE LAST 6 MONTHS, ON THE AVERAGE, HOW MANY DAYS A MONTH HAVE YOU HAD A DRINK _____ days a month.

2a. DURING THE 6 MONTHS BEFORE THAT PERIOD, ON THE AVERAGE, HOW MANY DAYS A MONTH DID YOU HAVE A DRINK?
_____ days a month.

3. OVER THE LAST 6 MONTHS, ON A DAY WHEN YOU ARE DRINKING, HOW MANY DRINKS DO YOU USUALLY HAVE IN 24 HOURS? (A DRINK IS A 12 OZ. CAN, GLASS OR BOTTLE OF BEER; A 4 OZ. GLASS OF WINE; A 12 OZ. WINE COOLER BOTTLE; A SINGLE SHOT; OR A SINGLE "MIXED DRINK.") _____ drinks per 24 hours.

3a. DURING THE 6 MONTHS BEFORE THAT PERIOD ,ON A DAY WHEN YOU WERE DRINKING, HOW MANY DRINKS DID YOU USUALLY HAVE IN 24 HOURS? (A DRINK IS A 12 OZ. CAN, GLASS OR BOTTLE OF BEER; A 4 OZ. GLASS OF WINE; A 12 OZ. WINE COOLER BOTTLE; A SINGLE SHOT; OR A SINGLE "MIXED DRINK.")
_____ drinks per 24 hours.

4. OVER THOSE 6 MONTHS, WHEN YOU GOT DRUNK, HOW BAD WAS YOUR HANGOVER?

_____ Never bad
_____ Not bad
_____ A little less than average
_____ Average
_____ A little more than average
_____ Pretty Bad
_____ Terrible
_____ Worst possible
_____ Never drank enough to get hangover

IF YOU DRANK NO ALCOHOLIC BEVERAGES AT ALL (NOT EVEN A FEW SIPS) IN THE LAST 6 MONTHS, GO TO PAGE 5, QUESTION 5.

B. THE FOLLOWING QUESTIONS ARE ABOUT YOUR DRINKING PATTERNS. IN ANSWERING THE QUESTIONS, PLEASE THINK ABOUT WHAT YOU HAVE DONE ON THE AVERAGE OVER THE LAST SIX MONTHS.

1. WHEN DRINKING BEER

a. HOW OFTEN DO YOU USUALLY HAVE BEER?

_____ 3 or more times a day
_____ 2 times a day
_____ Once a day
_____ Nearly every day
_____ 3 or 4 times a week
_____ Once or twice a week
_____ 2 or 3 times a month
_____ About once a month
_____ Less than once a month, but at least once a year
_____ Less than once a year
_____ NEVER [If checked, go to question #2a]

b. THINK OF ALL THE TIMES YOU HAD BEER RECENTLY, WHEN YOU DRINK BEER, HOW OFTEN DO YOU HAVE 10 OR MORE CANS, GLASSES OR BOTTLES?

_____ Nearly every time: SKIP TO QUESTION #2 BELOW
_____ More than half the time: SKIP TO QUESTION #2 BELOW
_____ Less than half the time
_____ Once in a while
_____ NEVER

c. WHEN YOU DRINK BEER, HOW OFTEN DO YOU HAVE AS MANY AS 7 TO 9 CANS, GLASSES OR BOTTLES?

_____ Nearly every time: SKIP TO QUESTION #2 BELOW
_____ More than half the time: SKIP TO QUESTION #2 BELOW
_____ Less than half the time
_____ Once in a while
_____ NEVER

d. WHEN YOU DRINK BEER, HOW OFTEN DO YOU HAVE AS MANY AS 5 TO 6 CANS, GLASSES OR BOTTLES?

_____ Nearly every time: SKIP TO QUESTION #2 BELOW
_____ More than half the time: SKIP TO QUESTION #2 BELOW
_____ Less than half the time
_____ Once in a while
_____ NEVER

e. WHEN YOU DRINK BEER, HOW OFTEN DO YOU HAVE AS MANY AS 3 to 4 CANS, GLASSES OR BOTTLES?

_____ Nearly every time: SKIP TO QUESTION #2 BELOW
_____ More than half the time: SKIP TO QUESTION #2 BELOW
_____ Less than half the time
_____ Once in a while
_____ NEVER

f. WHEN YOU DRINK BEER, HOW OFTEN DO YOU HAVE 1 TO 2 CANS, GLASSES OR BOTTLES?

_____ Nearly every time
_____ More than half the time
_____ Less than half the time
_____ Once in a while
_____ NEVER

2. <u>WHEN DRINKING WINE</u>:

a. HOW OFTEN DO YOU USUALLY HAVE WINE OR A WINE COOLER, OR A PUNCH CONTAINING WINE?

_____ 3 or more times a day
_____ 2 times a day
_____ Once a day
_____ Nearly every day
_____ 3 or 4 times a week
_____ Once or twice a week
_____ 2 or 3 times a month
_____ About once a month
_____ Less than once a month, but at least once a year
_____ Less than once a year
_____ NEVER [If checked, go to question #3a]

b. THINK OF ALL THE TIMES YOU HAD WINE OR A WINE COOLER OR A PUNCH CONTAINING WINE RECENTLY, HOW OFTEN DO YOU HAVE 10 OR MORE GLASSES OR WINE COOLERS?

_____ Nearly every time: SKIP TO QUESTION #3 BELOW
_____ More than half the time: SKIP TO QUESTION #3 BELOW
_____ Less than half the time
_____ Once in a while
_____ NEVER

c. WHEN YOU DRINK WINE, HOW OFTEN DO YOU HAVE AS MANY AS 7 TO 9 GLASSES OR WINE COOLERS?

_____ Nearly every time: SKIP TO QUESTION #3 BELOW
_____ More than half the time: SKIP TO QUESTION #3 BELOW
_____ Less than half the time
_____ Once in a while
_____ NEVER

d. WHEN YOU DRINK WINE, HOW OFTEN DO YOU HAVE AS MANY AS 5 to 6 GLASSES OR WINE COOLERS?

_____ Nearly every time: SKIP TO QUESTION #3 BELOW
_____ More than half the time: SKIP TO QUESTION #3 BELOW
_____ Less than half the time
_____ Once in a while
_____ NEVER

e. WHEN YOU DRINK WINE, HOW OFTEN DO YOU HAVE AS MANY AS 3 to 4 GLASSES OR WINE COOLERS?

_____ Nearly every time: SKIP TO QUESTION #3 BELOW
_____ More than half the time: SKIP TO QUESTION #3 BELOW
_____ Less than half the time
_____ Once in a while
_____ NEVER

f. WHEN YOU DRINK WINE, HOW OFTEN DO YOU HAVE 1 TO 2 GLASSES OR WINE COOLERS?

_____ Nearly every time
_____ More than half the time
_____ Less than half the time
_____ Once in a while
_____ NEVER

3. WHEN DRINKING WHISKEY OR LIQUOR

a. HOW OFTEN DO YOU USUALLY HAVE WHISKEY OR LIQUOR (SUCH AS MARTINIS, MANHATTANS, HIGHBALLS, OR STRAIGHT DRINKS INCLUDING SCOTCH, BOURBON, GIN, VODKA, RUM, ETC.)?

_____ 3 or more times a day
_____ 2 times a day
_____ Once a day
_____ Nearly every day
_____ 3 or 4 times a week
_____ Once or twice a week
_____ 2 or 3 times a month
_____ About once a month
_____ Less than once a month, but at least once a year
_____ Less than once a year
_____ NEVER [If checked, go to question #4]

b. THINK OF ALL THE TIMES YOU HAD DRINKS CONTAINING WHISKEY OR OTHER LIQUOR RECENTLY, WHEN YOU HAVE HAD THEM, HOW OFTEN DO YOU HAVE AS MANY AS 10 OR MORE?

_____ Nearly every time: SKIP TO QUESTION #4 BELOW
_____ More than half the time: SKIP TO QUESTION #4 BELOW
_____ Less than half the time
_____ Once in a while
_____ NEVER

c. WHEN YOU HAVE HAD DRINKS CONTAINING WHISKEY OR OTHER LIQUOR, HOW OFTEN DO YOU HAVE AS MANY AS 7 TO 9 DRINKS?

_____ Nearly every time: SKIP TO QUESTION #4 BELOW
_____ More than half the time: SKIP TO QUESTION #4 BELOW
_____ Less than half the time
_____ Once in a while
_____ NEVER

d. WHEN YOU HAVE HAD DRINKS CONTAINING WHISKEY OR OTHER LIQUOR, HOW OFTEN DO YOU HAVE AS MANY AS 5 TO 6 DRINKS?

_____ Nearly every time: SKIP TO QUESTION #4 BELOW
_____ More than half the time: SKIP TO QUESTION #4 BELOW
_____ Less than half the time
_____ Once in a while
_____ NEVER

e. WHEN YOU HAVE HAD DRINKS CONTAINING WHISKEY OR LIQUOR, HOW OFTEN DO YOU HAVE 3 TO 4 DRINKS?

_____ Nearly every time: SKIP TO QUESTION #4 BELOW
_____ More than half the time: SKIP TO QUESTION #4 BELOW
_____ Less than half the time
_____ Once in a while
_____ NEVER

f. WHEN YOU HAVE HAD DRINKS CONTAINING WHISKEY OR LIQUOR, HOW OFTEN DO YOU HAVE 1 TO 2 DRINKS?

_____ Nearly every time
_____ More than half the time
_____ Less than half the time
_____ Once in a while
_____ NEVER

4. WHEN DRINKING ANYTHING, CHECK HOW OFTEN DO YOU HAVE ANY DRINK CONTAINING ALCOHOL, WHETHER IT IS WINE, BEER, WHISKEY OR ANY OTHER DRINK. MAKE SURE THAT YOUR ANSWER IS NOT LESS FREQUENT THAN THE FREQUENCY REPORTED ON ANY OF THE PRECEDING QUESTIONS.

_____ 3 or more times a day
_____ 2 times a day
_____ Once a day
_____ Nearly every day
_____ 3 or 4 times a week
_____ Once or twice a week
_____ 2 or 3 times a month
_____ About once a month
_____ Less than once a month, but at least once a year
_____ Less than once a year
_____ NEVER

NOW SOME QUESTIONS ABOUT OTHER TIME PERIODS.

---

5. NOW ABOUT THE PAST YEAR, ABOUT HOW MANY TIMES HAVE YOU DRUNK JUST ENOUGH TO FEEL A LITTLE HIGH OR LIGHT-HEADED?

_____ None
_____ 1 time in the past year
_____ 2-3 times in the past year
_____ 4-5 times in the past year
_____ 6-10 times in the past year
_____ Once a month
_____ Twice a month
_____ Once a week
_____ Twice a week
_____ More than twice a week

6. DURING THE PAST YEAR, ABOUT HOW MANY TIMES HAVE YOU GOTTEN DRUNK OR VERY, VERY HIGH?

_____ None	_____ Once a month
_____ 1 time in the past year	_____ Twice a month
_____ 2-3 times in the past year	_____ Once a week
_____ 4-5 times in the past year	_____ Twice a week
_____ 6-10 times in the past year	_____ More than twice a week

7. Now a question about earlier in your life; HOW OLD WERE YOU THE FIRST TIME YOU EVER DRANK ENOUGH TO GET DRUNK?

_____ years old; if you have never been drunk, check here _____.

8a. WE ARE ALSO INTERESTED IN THE OCCASIONS THAT MAY BE RARE (OR NOT), WHEN PEOPLE DRINK A LOT MORE THAN THEY USUALLY DO. IN THE LAST YEAR, THINK OF THE 24 HOUR PERIOD WHEN YOU DID THE MOST DRINKING; THIS WOULD BE A DAY SOMEWHERE IN THE PERIOD BETWEEN _____, _____ AND NOW.
(month) (year)

On that day, how many drinks did you have? (A drink is a 12 oz. can, bottle or glass of beer, a 4 oz. glass of wine, a 12 oz. wine cooler bottle, a single shot, or a single mixed drink)

_____	30 or more drinks
_____	25 - 29 drinks
_____	20 - 24 drinks
_____	15 - 19 drinks
_____	10 - 14 drinks
_____	7 - 9 drinks
_____	5 - 6 drinks
_____	3 - 4 drinks
_____	1 - 2 drinks
_____	None

8b. APPROXIMATELY WHEN DID THIS HAPPEN? _____, _____.
(month) (year)

8c. NOW ANSWER THE QUESTION FOR ANY TIME IN YOUR LIFE BEFORE THIS LAST YEAR. IN THE 24 HOUR PERIOD WHEN YOU DID THE MOST DRINKING, HOW MANY DRINKS DID YOU HAVE?

_____	30 or more drinks
_____	25 - 29 drinks
_____	20 - 24 drinks
_____	15 - 19 drinks
_____	10 - 14 drinks
_____	7 - 9 drinks
_____	5 - 6 drinks
_____	3 - 4 drinks
_____	1 - 2 drinks
_____	None

8d. APPROXIMATELY WHEN DID THIS HAPPEN? _____, _____.
(month) (year)

C. NOW SOME QUESTIONS ABOUT WHERE YOU DRINK:

PLEASE INDICATE HOW OFTEN YOU DRINK BEER, WINE, OR LIQUOR IN EACH OF THE FOLLOWING SETTINGS, PLACES, OR OCCASIONS. MARK X ON ONE BLANK LINE IN EACH ROW.

Never drink or don't drink in this setting	**Sometimes**	**Frequently**	**Most of the time**	
____	____	____	____	1. At parties when other kids are drinking and your parents or other adults are not present.
____	____	____	____	2. At a party when other kids are drinking and when your parents or other adults are present.
____	____	____	____	3. At home on special occasions such as birthdays, or holidays such as Thanksgiving, etc.
____	____	____	____	4. At dinner at home with your family.
____	____	____	____	5a. At places where kids hang around when their parents or other adults are not present. If you answer YES here, answer Q. 5b and Q. 5c.

5b. Where? _____

5c. What are you usually doing? _____

Never drink or don't drink in this setting	**Sometimes**	**Frequently**	**Most of the time**	
____	____	____	____	6. During or after a school activity such as a dance or football game, when your parents or other adults you know <u>are not</u> present or can't see you.
____	____	____	____	7. Driving around or sitting in somebody's car at night.
____	____	____	____	8. Alone-- when no one else is around.
____	____	____	____	9. When a grownup I know offers it to me (not a parent).

D. NOW SOME QUESTIONS ABOUT OUTCOMES PEOPLE SOMETIMES HAVE BECAUSE OF DRINKING. HAVE YOU EVER HAD ANY OF THE FOLLOWING HAPPEN BECAUSE OF YOUR DRINKING?

<u>ANSWER KEY FOR QUESTIONS BELOW</u>:

1	2	3-5	6-10	11-20	21-50	51-100	101-250	251+

		<u>YES</u>	<u>NO</u> (check one)	HOW MANY TIMES (approx.- see key)*	AGE first time	AGE most recent time
1.	Got into trouble with my teachers or principal because of my drinking.	____	____	_____	_____	_____
2.	Got into difficulties of any kind with my friends.	____	____	_____	_____	_____

ANSWER KEY FOR QUESTIONS BELOW:

1	2	3-5	6-10	11-20	21-50	51-100	101-250	251+

	YES	NO	HOW MANY TIMES (approx.- see key)*	AGE first time	AGE most recent time
3. Driven a car when I'd had a good bit to drink.	____	____	_____	_____	_____
4. Been criticized by someone I was dating because of my drinking.	____	____	_____	_____	_____
5. Gotten in trouble with the police because of my drinking.	____	____	_____	_____	_____
6. Gotten in trouble with my parents because of my drinking.	____	____	_____	_____	_____
7. Missed school (or time on job) because of my drinking.	____	____	_____	_____	_____
8. Thought I was drinking too much.	____	____	_____	_____	_____
9. Gone on a binge of constant drinking.	____	____	_____	_____	_____
10. Lost friends because of my drinking.	____	____	_____	_____	_____
11. Felt guilty about my drinking.	____	____	_____	_____	_____
12. Took a drink or two first thing in the morning.	____	____	_____	_____	_____
13. Restricted my drinking to certain times of day or week in order to control it or cut down (like after 5PM, or only on weekends, or only with other people).	____	____	_____	_____	_____

ANSWER KEY FOR QUESTIONS BELOW:

1	2	3-5	6-10	11-20	21-50	51-100	101-250	251+

	YES	NO	HOW MANY TIMES (approx.- see key)*	AGE first time	AGE most recent time
	(check one)				
14. Once started drinking, kept on going till drunk.	____	____	_____	_____	_____
15. Had a car accident when I was drinking and driving.	____	____	_____	_____	_____
16. Kept on drinking after I promised myself not to.	____	____	_____	_____	_____
17. Had the shakes "the morning after".	____	____	_____	_____	_____
18. Heard or saw or felt things that weren't there (hallucinations), several days after stopping drinking.	____	____	_____	_____	_____
19. Had blackouts (couldn't remember later what you'd done while drinking).	____	____	_____	_____	_____
20. Been given a ticket for drunk driving (DWI or DUIL).	____	____	_____	_____	_____
21. Been given a ticket for public intoxication, drunk and disorderly or other non-driving alcohol arrest.	____	____	_____	_____	_____
22. Found that I had a strong need for a drink at some time each day.	____	____	_____	_____	_____

ANSWER KEY FOR QUESTIONS BELOW:

1	2	3-5	6-10	11-20	21-50	51-100	101-250	251+

	YES (check one)	NO (check one)	HOW MANY TIMES (approx.- see key)*	AGE first time	AGE most recent time
23. Needed to drink a lot more in order to get an effect, or found that I no longer could get high on the amount I used to drink.	____	____	_____	_____	_____
24. Found that I was able to drink a lot more than I used to before I would get drunk.	____	____	_____	_____	_____
25. Had days where I drank much more that I expected to when I began	____	____	_____	_____	_____
26. Found that I often continued drinking for more days in a row than I had planned to	____	____	_____	_____	_____
27. Found that I tended to gulp my drinks rather than just drink them	____	____	_____	_____	_____

* SELECT ANSWERS FROM THE KEY AT THE TOP OF THE PAGE

E. SO FAR THE QUESTIONS HAVE ASKED FOR THE FACTS ABOUT YOUR DRINKING. IN THIS SECTION YOU WILL BE ASKED ABOUT YOUR BEHAVIOR AND THE BEHAVIOR OF YOUR FRIENDS WHEN DRINKING; AND, MOST IMPORTANTLY, WHAT YOU AND YOUR FRIENDS THINK ABOUT DRINKING.

1. Have any of your friends suggested that you try drinking?

_____ Never
_____ Once or twice
_____ Several Times
_____ Often

2. Do you think that your father (stepfather, mother's partner) ever takes a drink of beer, wine or whiskey?

_____ Yes fairly regularly
_____ Yes, sometimes
_____ No
_____ I don't know

3. Do you think that your mother, (stepmother, father's partner) ever takes a drink of beer, wine or whiskey?

_____ Yes fairly regularly
_____ Yes, sometimes
_____ No
_____ I don't know

4. How do you think your parents (or your family) feel about boys your age drinking?

_____ Strongly approve
_____ Approve
_____ Don't care one way or the other
_____ Disapprove
_____ Strongly disapprove
_____ I don't know

5. How do you think your parents (or your family) feel about girls your age drinking?

_____ Strongly approve
_____ Approve
_____ Don't care one way or the other
_____ Disapprove
_____ Strongly disapprove
_____ I don't know

6. How do most of the kids you hang around with feel about kids your age drinking?

_____ Strongly approve
_____ Approve
_____ Neither approve nor disapprove
_____ Disapprove
_____ Strongly disapprove
_____ I don't know
_____ Does not apply

7. Please mark the blank which indicates the answer to the question on the right side. Give one answer for each question. Mark X on one blank line in each row.

None	1-2	Several	Less than half	More than half	All of them	
_____	_____	_____	_____	_____	_____	a. As far as you know, about how many of the kids in your school class drink alcohol at least sometimes?
_____	_____	_____	_____	_____	_____	b. About how many of the kids you hang around with drink alcohol at least sometimes?

8. Can you get alcoholic beverages when you want them?

____ I don't ever want them (check here if no drinking in last year)
____ No
____ Sometimes
____ Usually
____ Always

9. Where do you most often get the alcohol you and your friends drink?

____ I don't ever get it (check here if no drinking in last year)
____ From my home
____ A friend gives it to me
____ A friend or someone else buys it for me
____ I buy it myself
____ Other (Please explain)_____
_____

10. Does your school show films or have discussion groups or other programs to teach students about alcohol and drinking?

____ Yes ____ No

F. THE LAST SECTIONS OF THIS QUESTIONNAIRE DEAL WITH VARIOUS DRUGS OTHER THAN ALCOHOL. THERE IS STILL A LOT OF TALK THESE DAYS ABOUT THIS SUBJECT, BUT VERY LITTLE ACCURATE INFORMATION.

WE HOPE THAT YOU CAN ANSWER ALL QUESTIONS TRUTHFULLY; BUT IF YOU FIND ONE WHICH YOU FEEL YOU CANNOT ANSWER HONESTLY, WE WOULD PREFER THAT YOU LEAVE IT BLANK.

REMEMBER THAT YOUR ANSWERS WILL BE KEPT STRICTLY CONFIDENTIAL AND THEY ARE NEVER CONNECTED WITH YOUR NAME. THAT IS WHY THIS QUESTIONNAIRE IS IDENTIFIED ONLY WITH A CODE NUMBER.

THE FOLLOWING QUESTIONS ARE ABOUT CIGARETTES (CHECK THE BEST ANSWER):

1a. HAVE YOU EVER SMOKED CIGARETTES?

_____ Never
_____ Once or twice
_____ Occasionally but not regularly
_____ Regularly in the past
_____ Regularly now

1b. HAVE YOU SMOKED CIGARETTES DURING THE PAST 12 MONTHS?

_____ Never
_____ Once or twice
_____ Occasionally but not regularly
_____ Regularly for a while during this year, but not now
_____ Regularly now

2. HOW FREQUENTLY HAVE YOU SMOKED CIGARETTES DURING THE PAST 30 DAYS?

_____ Not at all
_____ Less than one cigarette per day
_____ One to five cigarettes per day
_____ About one-half pack per day
_____ About one pack per day
_____ About one and one-half packs per day
_____ Two packs or more per day

3a. Have you ever been around anyone else who has been smoking cigarettes?

Yes _____ No _____

3b. How many times (circle one)

1-2 3-5 6-9 10-19 20-39 40-99 100-1000 More than 1000

3c. Who was smoking? (check all that apply)

_____ Parents
_____ Other adults I know well
_____ Other adults I know, but not so well
_____ Adults I didn't know
_____ Kids I know well
_____ Kids I know, but not so well
_____ Kids I didn't know

G. THE FOLLOWING QUESTIONS ARE ALL ABOUT NON-PRESCRIPTION USE OF DRUGS, EITHER FOR RECREATION OR FOR SELF-MEDICATION.

1a. ON HOW MANY OCCASIONS (IF ANY) HAVE YOU USED MARIJUANA (GRASS, POT) OR HASHISH (HASH, HASH OIL)	0 occasions	1-2 occasions	3-5 occasions	6-9 occasions	10-19 occasions	20-39 occasions	40-99 occasions	100-1000 occasions	more than 1000
In your lifetime?	( )	( )	( )	( )	( )	( )	( )	( )	( )
During the last 12 months?	( )	( )	( )	( )	( )	( )	( )	( )	( )
During the last 30 days?	( )	( )	( )	( )	( )	( )	( )	( )	( )

1b. HOW OLD WERE YOU WHEN YOU FIRST USED MARIJUANA? _____ years old.

1c. HAVE YOU EVER BEEN AROUND ANYONE ELSE WHO HAS BEEN USING IT?

YES _____ NO _____

1d. HOW MANY TIMES? (Circle one)

1-2 3-5 6-9 10-19 20-39 40-99 100-1000 More than 1000

1e. WHO WAS USING IT? (CHECK ALL THAT APPLY)

___ Parents
___ Other adults I know well
___ Other adults I know, but not so well
___ Adults I didn't know
___ Kids I know well
___ Kids I know, but not so well
___ Kids I didn't know

2a. ON HOW MANY OCCASIONS (IF ANY) HAVE YOU SNIFFED GLUE, OR BREATHED THE CONTENTS OF AEROSOL SPRAY CANS, OR INHALED ANY OTHER GASES OR SPRAYS IN ORDER TO GET HIGH (LIKE LIGHTER FLUID, NAIL POLISH REMOVER, PAINT THINNER, AND PAINT)

	0 occasions	1-2 occasions	3-5 occasions	6-9 occasions	10-19 occasions	20-39 occasions	40-99 occasions	100-1000 occasions	more than 1000
In your lifetime?	( )	( )	( )	( )	( )	( )	( )	( )	( )
During the last 12 months?	( )	( )	( )	( )	( )	( )	( )	( )	( )
During the last 30 days?	( )	( )	( )	( )	( )	( )	( )	( )	( )

2b. HOW OLD WERE YOU WHEN YOU FIRST USED ANY OF THESE INHALANTS?
_____ years old.

2c. HAVE YOU EVER BEEN AROUND ANYONE ELSE WHO HAS BEEN USING IT?

YES _____ NO _____

2d. HOW MANY TIMES? (Circle one)

1-2 3-5 6-9 10-19 20-39 40-99 100-1000 More than 1000

2e. WHO WAS USING IT? (CHECK ALL THAT APPLY)

___ Parents
___ Other adults I know well
___ Other adults I know, but not so well
___ Adults I didn't know
___ Kids I know well
___ Kids I know, but not so well
___ Kids I didn't know

3a. ON HOW MANY OCCASIONS (IF ANY) HAVE YOU USED AMYL OR BUTYL NITRATES (POPPERS, SNAPPERS, LOCKER ROOM, VAPORALE, RUSH, KICK, BULLET).	0 occasions	1-2 occasions	3-5 occasions	6-9 occasions	10-19 occasions	20-39 occasions	40-99 occasions	100-1000 occasions	more than 1000
In your lifetime?	( )	( )	( )	( )	( )	( )	( )	( )	( )
During the last 12 months?	( )	( )	( )	( )	( )	( )	( )	( )	( )
During the last 30 days?	( )	( )	( )	( )	( )	( )	( )	( )	( )

3b. HOW OLD WERE YOU WHEN YOU FIRST USED AMYL? _____ years old.

3c. HAVE YOU EVER BEEN AROUND ANYONE ELSE WHO HAS BEEN USING IT?

YES _____ NO _____

3d. HOW MANY TIMES? (Circle one)

1-2 3-5 6-9 10-19 20-39 40-99 100-1000 More than 1000

3e. WHO WAS USING IT? (CHECK ALL THAT APPLY)

___ Parents
___ Other adults I know well
___ Other adults I know, but not so well
___ Adults I didn't know
___ Kids I know well
___ Kids I know, but not so well
___ Kids I didn't know

4a. AMPHETAMINES ARE SOMETIMES PRESCRIBED BY DOCTORS TO HELP PEOPLE LOSE WEIGHT OR TO GIVE PEOPLE MORE ENERGY. THEY ARE SOMETIMES CALLED UPPERS, UPS, SPEED, CRYSTAL, CRANK, BENNIES, DEXIES, PEP PILLS, GREENIES, SPLASH AND DIET PILLS.

ON HOW MANY OCCASIONS (IF ANY) HAVE YOU TAKEN AMPHETAMINES ON YOUR OWN--THAT IS, WITHOUT A DOCTOR TELLING YOU TO TAKE THEM	0 occasions	1-2 occasions	3-5 occasions	6-9 occasions	10-19 occasions	20-39 occasions	40-99 occasions	100-1000 occasions	more than 1000
In your lifetime?	( )	( )	( )	( )	( )	( )	( )	( )	( )
During the last 12 months?	( )	( )	( )	( )	( )	( )	( )	( )	( )
During the last 30 days?	( )	( )	( )	( )	( )	( )	( )	( )	( )

4b. HOW OLD WERE YOU WHEN YOU FIRST USED AMPHETAMINES?

_____years old.

4c. HAVE YOU EVER BEEN AROUND ANYONE ELSE WHO HAS BEEN USING IT?

YES _____ NO _____

4d. HOW MANY TIMES? (Circle one)

1-2 3-5 6-9 10-19 20-39 40-99 100-1000 More than 1000

4e. WHO WAS USING IT? (CHECK ALL THAT APPLY)

___ Parents
___ Other adults I know well
___ Other adults I know, but not so well
___ Adults I didn't know
___ Kids I know well
___ Kids I know, but not so well
___ Kids I didn't know

5a. ON HOW MANY OCCASIONS (IF ANY) HAVE YOU USED LSD (ACID)	0 occasions	1-2 occasions	3-5 occasions	6-9 occasions	10-19 occasions	20-39 occasions	40-99 occasions	100-1000 occasions	more than 1000
In your lifetime?	( )	( )	( )	( )	( )	( )	( )	( )	( )
During the last 12 months?	( )	( )	( )	( )	( )	( )	( )	( )	( )
During the last 30 days?	( )	( )	( )	( )	( )	( )	( )	( )	( )

5b. HOW OLD WERE YOU WHEN YOU FIRST USED LSD? _____ years old.

5c. HAVE YOU EVER BEEN AROUND ANYONE ELSE WHO HAS BEEN USING IT?

YES _____ NO _____

5d. HOW MANY TIMES? (Circle one)

1-2 3-5 6-9 10-19 20-39 40-99 100-1000 More than 1000

5e. WHO WAS USING IT? (CHECK ALL THAT APPLY)

___ Parents
___ Other adults I know well
___ Other adults I know, but not so well
___ Adults I didn't know
___ Kids I know well
___ Kids I know, but not so well
___ Kids I didn't know

6a. ON HOW MANY OCCASIONS (IF ANY) HAVE YOU USED PSYCHEDELICS OTHER THAN LSD (LIKE MESCALINE, PEYOTE, PSILOCYBIN, PCP, ANGEL DUST)

	0 occasions	1-2 occasions	3-5 occasions	6-9 occasions	10-19 occasions	20-39 occasions	40-99 occasions	100-1000 occasions	more than 1000
In your lifetime?	( )	( )	( )	( )	( )	( )	( )	( )	( )
During the last 12 months?	( )	( )	( )	( )	( )	( )	( )	( )	( )
During the last 30 days?	( )	( )	( )	( )	( )	( )	( )	( )	( )

6b. HOW OLD WERE YOU WHEN YOU FIRST USED PSYCHEDELICS OTHER THAN LSD? _____ years old.

6c. HAVE YOU EVER BEEN AROUND ANYONE ELSE WHO HAS BEEN USING IT?

YES _____ NO _____

6d. HOW MANY TIMES? (Circle one)

1-2 3-5 6-9 10-19 20-39 40-99 100-1000 More than 1000

6e. WHO WAS USING IT? (CHECK ALL THAT APPLY)

___ Parents
___ Other adults I know well
___ Other adults I know, but not so well
___ Adults I didn't know
___ Kids I know well
___ Kids I know, but not so well
___ Kids I didn't know

7a. ON HOW MANY OCCASIONS (IF ANY) HAVE YOU USED CRACK.

	0 occasions	1-2 occasions	3-5 occasions	6-9 occasions	10-19 occasions	20-39 occasions	40-99 occasions	100-1000 occasions	more than 1000
In your lifetime?	( )	( )	( )	( )	( )	( )	( )	( )	( )
During the past 12 months?	( )	( )	( )	( )	( )	( )	( )	( )	( )
During the last 30 days?	( )	( )	( )	( )	( )	( )	( )	( )	( )

7b. HOW OLD WERE YOU WHEN YOU FIRST USED CRACK? _____ years old.

7c. HAVE YOU EVER BEEN AROUND ANYONE ELSE WHO HAS BEEN USING IT?

YES _____ NO _____

7d. HOW MANY TIMES? (Circle one)

1-2 3-5 6-9 10-19 20-39 40-99 100-1000 More than 1000

7e. WHO WAS USING IT? (CHECK ALL THAT APPLY)

___ Parents
___ Other adults I know well
___ Other adults I know, but not so well
___ Adults I didn't know
___ Kids I know well
___ Kids I know, but not so well
___ Kids I didn't know

8a. ON HOW MANY OCCASIONS (IF ANY) HAVE YOU USED COCAINE (COKE)

	0 occasions	1-2 occasions	3-5 occasions	6-9 occasions	10-19 occasions	20-39 occasions	40-99 occasions	100-1000 occasions	more than 1000
In your lifetime?	( )	( )	( )	( )	( )	( )	( )	( )	( )
During the past 12 months?	( )	( )	( )	( )	( )	( )	( )	( )	( )
During the last 30 days?	( )	( )	( )	( )	( )	( )	( )	( )	( )

8b. HOW OLD WERE YOU WHEN YOU FIRST USED COCAINE? _____ years old.

8c. HAVE YOU EVER BEEN AROUND ANYONE ELSE WHO HAS BEEN USING IT?

YES _____ NO _____

8d. HOW MANY TIMES? (Circle one)

1-2 3-5 6-9 10-19 20-39 40-99 100-1000 More than 1000

8e. WHO WAS USING IT? (CHECK ALL THAT APPLY)

___ Parents
___ Other adults I know well
___ Other adults I know, but not so well
___ Adults I didn't know
___ Kids I know well
___ Kids I know, but not so well
___ Kids I didn't know

9a. STEROIDS, OR ANABOLIC STEROIDS, ARE SOMETIMES USED FOR BODY BUILDING OR TO IMPROVE ATHLETIC PERFORMANCE. ON HOW MANY OCCASIONS (IF ANY) HAVE YOU USED STEROIDS ON YOUR OWN--THAT IS, WITHOUT A DOCTOR TELLING YOU TO TAKE THEM.

	0 occasions	1-2 occasions	3-5 occasions	6-9 occasions	10-19 occasions	20-39 occasions	40-99 occasions	100-1000 occasions	more than 1000
In your lifetime?	( )	( )	( )	( )	( )	( )	( )	( )	( )
During the last 12 months?	( )	( )	( )	( )	( )	( )	( )	( )	( )
During the last 30 days?	( )	( )	( )	( )	( )	( )	( )	( )	( )

9b. HOW OLD WERE YOU WHEN YOU FIRST USED STEROIDS? _____ years old.

9c. HAVE YOU EVER BEEN AROUND ANYONE ELSE WHO HAS BEEN USING IT?

YES _____ NO _____

9d. HOW MANY TIMES? (Circle one)

1-2 3-5 6-9 10-19 20-39 40-99 100-1000 More than 1000

9e. WHO WAS USING IT? (CHECK ALL THAT APPLY)

___ Parents
___ Other adults I know well
___ Other adults I know, but not so well
___ Adults I didn't know
___ Kids I know well
___ Kids I know, but not so well
___ Kids I didn't know

10a. ON HOW MANY OCCASIONS (IF ANY) HAVE YOU USED QUAALUDES (QUADS, SOAPERS, METHAQUALONE) ON YOUR OWN--THAT IS, WITHOUT A DOCTOR TELLING YOU TO TAKE THEM	0 occasions	1-2 occasions	3-5 occasions	6-9 occasions	10-19 occasions	20-39 occasions	40-99 occasions	100-1000 occasions	more than 1000
In your lifetime?	( )	( )	( )	( )	( )	( )	( )	( )	( )
During the last 12 months?	( )	( )	( )	( )	( )	( )	( )	( )	( )
During the last 30 days?	( )	( )	( )	( )	( )	( )	( )	( )	( )

10b. HOW OLD WERE YOU WHEN YOU FIRST USED QUAALUDES? _____ years old.

10c. HAVE YOU EVER BEEN AROUND ANYONE ELSE WHO HAS BEEN USING IT?

YES _____ NO _____

10d. HOW MANY TIMES? (Circle one)

1-2 3-5 6-9 10-19 20-39 40-99 100-1000 More than 1000

10e. WHO WAS USING IT? (CHECK ALL THAT APPLY)

___ Parents
___ Other adults I know well
___ Other adults I know, but not so well
___ Adults I didn't know
___ Kids I know well
___ Kids I know, but not so well
___ Kids I didn't know

11a. TRANQUILIZERS ARE SOMETIMES PRESCRIBED BY DOCTORS TO CALM PEOPLE DOWN, QUIET THEIR NERVES, OR RELAX THEIR MUSCLES. LIBRIUM VALIUM, EQUANIL AND MILTOWN ARE ALL TRANQUILIZERS.

ON HOW MANY OCCASIONS (IF ANY) HAVE YOU TAKEN TRANQUILIZERS ON YOUR OWN--THAT IS, WITHOUT A DOCTOR TELLING YOU TO TAKE THEM.	0 occasions	1-2 occasions	3-5 occasions	6-9 occasions	10-19 occasions	20-39 occasions	40-99 occasions	100-1000 occasions	more than 1000
In your lifetime?	( )	( )	( )	( )	( )	( )	( )	( )	( )
During the last 12 months?	( )	( )	( )	( )	( )	( )	( )	( )	( )
During the last 30 days?	( )	( )	( )	( )	( )	( )	( )	( )	( )

11b. HOW OLD WERE YOU WHEN YOU FIRST USED TRANQUILIZERS?

_____ years old.

11c. HAVE YOU EVER BEEN AROUND ANYONE ELSE WHO HAS BEEN USING IT?

YES _____ NO _____

11d. HOW MANY TIMES? (Circle one)

1-2 3-5 6-9 10-19 20-39 40-99 100-1000 More than 1000

11e. WHO WAS USING IT? (CHECK ALL THAT APPLY)

___ Parents
___ Other adults I know well
___ Other adults I know, but not so well
___ Adults I didn't know
___ Kids I know well
___ Kids I know, but not so well
___ Kids I didn't know

12a. BARBITURATES ARE SOMETIMES PRESCRIBED BY DOCTORS TO HELP PEOPLE RELAX OR GET TO SLEEP. THEY ARE SOMETIMES CALLED DOWNS, DOWNERS, GOOFBALLS, YELLOWS, REDS, BLUES, RAINBOWS.

ON HOW MANY OCCASIONS (IF ANY) HAVE YOU TAKEN BARBITURATES ON YOUR OWN -- THAT IS, WITHOUT A DOCTOR TELLING YOU TO TAKE THEM.	0 occasions	1-2 occasions	3-5 occasions	6-9 occasions	10-19 occasions	20-39 occasions	40-99 occasions	100-1000 occasions	more than 1000
In your lifetime?	( )	( )	( )	( )	( )	( )	( )	( )	( )
During the last	( )	( )	( )	( )	( )	( )	( )	( )	( )
During the last 30 days?	( )	( )	( )	( )	( )	( )	( )	( )	( )

12b. HOW OLD WERE YOU WHEN YOU FIRST USED BARBITURATES?

_____ years old.

12c. HAVE YOU EVER BEEN AROUND ANYONE ELSE WHO HAS BEEN USING IT?

YES _____ NO _____

12d. HOW MANY TIMES? (Circle one)

1-2 3-5 6-9 10-19 20-39 40-99 100-1000 More than 1000

12e. WHO WAS USING IT? (CHECK ALL THAT APPLY)

___ Parents
___ Other adults I know well
___ Other adults I know, but not so well
___ Adults I didn't know
___ Kids I know well
___ Kids I know, but not so well
___ Kids I didn't know

13a. ON HOW MANY OCCASIONS (IF ANY) HAVE YOU USED HEROIN (SMACK, HORSE, SKAG, JUNK).	0 occasions	1-2 occasions	3-5 occasions	6-9 occasions	10-19 occasions	20-39 occasions	40-99 occasions	100-1000 occasions	more than 1000
In your lifetime?	( )	( )	( )	( )	( )	( )	( )	( )	( )
During the last 12 months?	( )	( )	( )	( )	( )	( )	( )	( )	( )
During the last 30 days?	( )	( )	( )	( )	( )	( )	( )	( )	( )

13b. HOW OLD WERE YOU WHEN YOU FIRST USED HEROIN? _____ years old.

13c. HAVE YOU EVER BEEN AROUND ANYONE ELSE WHO HAS BEEN USING IT?

YES _____ NO _____

13d. HOW MANY TIMES? (Circle one)

1-2 3-5 6-9 10-19 20-39 40-99 100-1000 More than 1000

13e. WHO WAS USING IT? (CHECK ALL THAT APPLY)

___ Parents
___ Other adults I know well
___ Other adults I know, but not so well
___ Adults I didn't know
___ Kids I know well
___ Kids I know, but not so well
___ Kids I didn't know

14a. THERE ARE A NUMBER OF NARCOTICS OTHER THAN HEROIN, SUCH AS METHADONE, OPIUM, MORPHINE, CODEINE, DEMEROL, PAREGORIC, TALWIN, AND LAUDANUM. THESE ARE SOMETIMES PRESCRIBED BY DOCTORS.

ON HOW MANY OCCASIONS (IF ANY) HAVE YOU TAKEN NARCOTICS OTHER THAN HEROIN ON YOUR OWN-- THAT IS, WITHOUT A DOCTOR TELLING YOU TO TAKE THEM	0 occasions	1-2 occasions	3-5 occasions	6-9 occasions	10-19 occasions	20-39 occasions	40-99 occasions	100-1000 occasions	more than 1000
In your lifetime?	( )	( )	( )	( )	( )	( )	( )	( )	( )
During the last 12 months?	( )	( )	( )	( )	( )	( )	( )	( )	( )
During the last 30 days?	( )	( )	( )	( )	( )	( )	( )	( )	( )

14b. HOW OLD WERE YOU WHEN YOU FIRST USED OTHER NARCOTICS?

_____ years old.

14c. HAVE YOU EVER BEEN AROUND ANYONE ELSE WHO HAS BEEN USING IT?

YES _____ NO _____

14d. HOW MANY TIMES? (Circle one)

1-2 3-5 6-9 10-19 20-39 40-99 100-1000 More than 1000

14e. WHO WAS USING IT? (CHECK ALL THAT APPLY)

___ Parents
___ Other adults I know well
___ Other adults I know, but not so well
___ Adults I didn't know
___ Kids I know well
___ Kids I know, but not so well
___ Kids I didn't know

H. NOW SOME OTHER QUESTIONS ABOUT NONPRESCRIPTION USE OF DRUGS. HAVE YOU EVER HAD ANY OF THE FOLLOWING THINGS HAPPEN BECAUSE OF YOUR USE OF THE NONPRESCRIPTION DRUGS ASKED ABOUT IN SECTION G (THE LAST SECTION)?

ANSWER KEY FOR QUESTIONS BELOW:

1	2	3-5	6-10	11-20	21-50	51-100	101-250	251+

	YES	NO	HOW MANY TIMES (approx) (see key)*	AGE first TIME	AGE most recent TIME
1. Been absent from school one or more times because of my use.	____	____	_____	_____	_____
2. Had my grades in school get worse than they were because of my use.	____	____	_____	_____	_____
3. Caused me to be stopped by the police or get a traffic citation.	____	____	_____	_____	_____
4. Caused some physical or medical problem (even a minor or unimportant one).	____	____	_____	_____	_____
5. Found it hard to concentrate on something I wanted to do, because of my use.	____	____	_____	_____	_____
6. Had trouble getting along with my parents (at least once) because they didn't want me to use any of the stuff.	____	____	_____	_____	_____
7. Found myself unable to control my moods when I used any of the stuff.	____	____	_____	_____	_____

8. Had trouble getting along with some of my friends because of use. ____ ____ _____ _____ _____

9. Missed school (or time on the job). ____ ____ _____ _____ _____

10. Lost friends because of use. ____ ____ _____ _____ _____

11. Been fired or laid off from a job because of use ____ ____ _____ _____ _____

12. Had a car accident when I was driving ____ ____ _____ _____ _____

13. Had to go to a hospital (other than accidents) ____ ____ _____ _____ _____

14. Had to stay in hospital overnight ____ ____ _____ _____ _____

15. Had to see a doctor because of drug use (unintentional overdose) or had a doctor say drugs had harmed your health ____ ____ _____ _____ _____

16. Gone through physical with-withdrawal from drugs ____ ____ _____ _____ _____

17. Been arrested for possession of marijuana ____ ____ _____ _____ _____

18a. Have you ever taken drugs intravenously (using a needle)? Don't count shots you were given by a doctor or nurse or shots you may have taken for treatment of diabetes.

_____NO _____YES

18b. IF YES, WHAT DRUGS HAVE YOU TAKEN INTRAVENOUSLY (IV)?

_____

18c. AT WHAT AGE DID YOU FIRST TAKE AN IV DRUG? _____ years old.

18d. AT WHAT AGE WAS THE MOST RECENT TIME? _____ years old.

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