A LONGITUDINAL TEST OF SOCIAL LEARNING THEORY: ADOLESCENT SMOKING

Ronald L. Akers
Gang Lee

A general social learning theory of deviance is applied to adolescent smoking as a form of substance use and tested with data from a 5-year longitudinal study of a panel (N=454) of respondents in grades 7 through 12 in an Iowa community. The major components of the process specified in the theory are differential association, differential reinforcement, definitions (attitudes), and modeling. The process is one in which the operation of these variables produces abstinence or smoking, but with some reciprocal effects of smoking behavior on the social learning variables. Previous research on various kinds of deviance and substance use has been supportive of the theory. The findings in this study from LISREL models of the overall social learning process and each of the component of association, reinforcement, and definitions are also supportive.

Introduction

The purpose of this paper is to report findings from research testing a social learning theory of substance use with longitudinal data on adolescent smoking. Tobacco use has come increasingly to be seen as part of the overall problem of substance use and abuse in society. Smoking at all ages, but especially during adolescence, has become defined as deviant behavior and habitual smoking as an addiction. The evidence on the health hazards of smoking is clear, and this social disapproval is based not only on smoking as a violation of norms of healthful behavior, but also as a violation of norms of good and acceptable behavior. The disapproval has become more strident as smokers of all ages are increasingly defined as pariahs and as morally condemned. The restrictive nature of legal regulation has made smoking in most public accommodations illegal. Organized interest groups have been working for many years to secure ever more restrictive regulations on smoking and to ban all tobacco advertising (Troyer and Markle 1983).

Ronald L. Akers, Ph.D., is Professor of Sociology and Criminology and Director of the Center for Studies in Criminology and Law at the University of Florida. He is past president of the American Society of Criminology and past president of the Southern Sociological Society. He is author of Criminological Theories (1994), Drugs, Alcohol and Society (1993), Deviant Behavior: A Social Learning Approach (3rd ed., 1985), and numerous journal articles and book chapters. He has conducted extensive research in his areas of interest in criminological theory, alcohol and drug behavior, sociology of law, juvenile delinquency, and corrections. Gang Lee is a doctoral student in the Department of Sociology at the University of Florida with a specialty in criminology and deviance. He has published articles on gambling and deviance. Correspondence and requests for reprints should be addressed to Dr. Ronald L. Akers, Center for Studies in Criminology and Law, PO Box 115950, University of Florida, Gainesville, FL 32611.

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AKERS, LEE

The notion that smokers are drug dependent has been formalized by the declaration by the Surgeon General and the Food and Drug Administration that nicotine is an addictive drug, producing an addiction that is more difficult to overcome than heroin addiction. Prevention and treatment of smoking has become a major industry, and nicotine addiction, relapse, and treatment have come to be treated in the literature as essentially the same as other drug addiction, relapse, and treatment. The textbooks on drug use and abuse routinely include sections or chapters on tobacco (Akers 1992; Goode 1993; Ray and Ksir 1993; Fishbein and Pease 1996), and journals on alcohol and drug behavior regularly carry articles on the etiology, prevention, and treatment of nicotine addiction (Carmody 1992), including adolescent smoking (Urberg et al. 1991).

Smoking, even by adolescents, is still apt to be seen as less serious than illegal drug use, but there can be little doubt that it is now considered by public opinion and in the research community to be a drug that is used and abused with serious negative consequences. Among teenagers, smoking is correlated with the use of alcohol, marijuana, and other drugs and bears a substantial relationship to involvement in serious delinquency (Jessor and Jessor 1977; Akers 1992). As with all drug use, most of those who ever use tobacco begin in the adolescent years, and there have been recent increases in tobacco smoking among teenagers (along with increases in the use of other substances). The issue of explaining and predicting smoking among adolescents, then, is related to that of drug use of all kinds, and general models of substance use should apply to smoking as well as to consumption of other substances.

A Social Learning Theory of Drug Use

The designation of “social learning theory” has been used to refer to any social behavioristic approach (Bandura 1977; Jessor and Jessor 1977; Rotter 1982), and social learning perspectives incorporating processes of behavioral modeling and reinforcement have been applied in a number of areas including alcohol and substance use (see Stumphauer 1983; Pomerleau and Pomerleau 1988; White et al. 1990, 1991). The social learning theory tested here is that developed by Ronald L. Akers, originally proposed in collaboration with Robert L. Burgess (Burgess and Akers 1966). It is a general theory that Akers has applied to a range of deviant and criminal behaviors (Akers 1985, 1994) as well as to drug use and abuse (Akers 1992).

Social learning theory proposes that the same process is involved in both conforming and deviant behavior. The difference lies in the direction of the process in which these mechanisms operate. It is seldom an either-or, all-or-nothing process. Rather it is the balance of influences on behavior. That balance usually exhibits some stability over time, but it can become unstable and change with time or circumstances. Conforming and deviant behavior is learned by all of the mechanisms in this process, but the theory proposes that the principal mechanisms are, differential association (direct and indirect interaction with others), differential reinforcement (instrumental learning through rewards and punishments), imitation (observational learning), and cognitive definitions (attitudes) that are favorable or unfavorable, functioning as discriminative (cue) stimuli, for the behavior.

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which control sources and patterns of reinforcement, provide normative definitions, and expose one to behavioral models. . .Deviant behavior can be expected to the extent that it has been differentially reinforced over alternative behavior (conforming or other deviant behavior) and is defined as desirable or justified when the individual is in a situation discriminative for the behavior. (Akers 1985:57-58)

The theory explains abstinence, use, and abuse of drugs as socially influenced behavior acquired and sustained through a learning process in which these four main sets of variables operate.

Whether individuals will abstain from or take drugs (and whether they will continue or desist) depends on the past, present, and anticipated future rewards and punishments perceived to be attached to abstinence and use (differential reinforcement). The person learns attitudes, orientations, or evaluative knowledge which are favorable or unfavorable to using drugs (definitions). These are themselves verbal and cognitive behavior which can be directly reinforced and can also act as cue stimuli for drug use. The more individuals define the behavior as good or at least justified or excusable rather than holding to general beliefs or specific attitudes counter to a drug, the more likely they are to use it.

The reinforcers and punishers can be nonsocial, as in the direct physical effects of drugs and alcohol, but the principal behavioral effects come from interaction in or under the influence of those groups (primary groups of family and friends but also secondary groups and the media) with which one is in differential association which control sources and patterns of reinforcement, exposure to norms and behavioral models. Drug use is predicted to the extent that it has been differentially reinforced over abstinence and is defined by the individual as desirable or justified when he or she is in a situation discriminative for the behavior. (Akers 1992:12-13)

The theory hypothesizes, then, a positive relationship between the frequency of drug use, in this case abstinence/frequency of smoking by adolescents, and the social learning variables of differential association, differential reinforcement, definitions, and imitation. The adolescent is more likely to smoke and to smoke frequently the greater the extent to which he or she associates with others who are smokers and hold attitudes favorable to smoking, is in a position to imitate behavioral models of smoking, has experienced and/or anticipates greater positive outcomes than negative outcomes of smoking in the reactions of others and the effects of smoking, and endorses positive or neutralizing definitions of smoking.

Sequence, Causal Ordering, and Reciprocal Effects in Social Learning Theory

Stafford and Ekland-Olson (1982) and Strickland (1982) argue that social learning theory applies only to the “socialization” model of direct one-way causal effects of peer associations on deviance. Therefore, they conclude that any empirical finding of a “selective” process in which deviants are self-selected into association with one another disconfirms the theory. Similarly, Thornberry (Thornberry 1987; Thornberry et al. 1991) has criticized social learning theory on the grounds that it posits only one-way causal mechanisms in which the social
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Once the acts have been performed, the actual consequences (social and nonsocial reinforcers and punishers) of the specific behavior come into play to affect the chances that the deviant behavior will be continued and at what level. . . . Initial acts may and do occur in the absence of definitions favorable to them; rather the definitions get applied retroactively to excuse or redefine the initial deviant acts. To the extent that they successfully mitigate others' or self-punishment, they become discriminative for repetition of the deviant acts and, hence, precede the future commission of the acts. . . . [A]fter the deviant activity has begun and the consequences accompanying it are experienced, the associational patterns may themselves in turn be altered so that . . . further interaction with others is based, at least in part, on whether they too are involved in the deviant activity and to what degree. (Akers 1985:60)

Differential peer influence is not the only influence, however. Parents, other family members, neighbors, church and religious groups, schoolteachers, physicians, authority figures, and other individuals and groups in the community as well as mass media and other more remote sources of attitudes and models have varying degrees of effect on drug use and abstinence. When these act in harmony to move youngsters in the direction toward either using or not using, the chances of their behaving that way are maximized. When these sources are in conflict, adolescents will most often behave similarly to close peers. It is in peer groups that drugs typically are first made available and opportunities for use provided. The process is one of peer influence, however, and does not usually involve peer "pressure."

After use has begun beyond the initial attempts and experimentation, imitation becomes less important, although the effects of definitions and consequences of use, themselves affected by the experience of a person's having used drugs, continue. These consequences include the actual or perceived physiological effects of the drugs, which may be affected by what the person has previously learned to expect. . . . Using and the consequences of using now may begin to have an effect on choice of friends and social settings and therefore may have some feedback effects on differential association (Akers 1985:115-116).

Social learning admits that birds of a feather do flock together, but it also admits that if the birds are humans, they also will influence one another's behavior, in both conforming and deviant directions. (Akers 1991:210).

[协会] with peers and others are most often formed initially around attractions, friendships, and circumstances, such as neighborhood proximity, that have little to do directly with co-involvement in some deviant behavior. However, after the associations have been established and the reinforcing or punishing consequences of the deviant behavior are experienced, both the continuation of old and the seeking of new associations (over which one has any choice) will themselves be affected. But the theory proposes that the sequence of events, in which deviant associations precede the onset of delinquent behavior, will occur more
frequently than the sequence of events in which the onset of delinquency precedes the beginning of deviant associations.

Akers applies this general model of deviance and substance use specifically to adolescent smoking as follows:

Social learning is a complex, ongoing behavioral process, and the sequence of events is variable depending on the particular behavior and individuals in question. Nevertheless, the typical process envisioned by the theory is that individuals interact and identify with different groups [e.g., family and peer groups] in which they are exposed to behavioral models, norms, and reinforcement patterns tending toward abstinence or smoking. The balance of definitions favorable and unfavorable to smoking combined with imitation and the anticipated balance of reinforcement produce the initial smoking. After initiation, imitation becomes less important (although facilitative effects of modeling may remain) while the effects of norms and definitions (themselves now affected by the consequences of initial deviant acts) and reinforcement continue. Differential association has a strong effect in both initiation and maintenance. Those associations are most often formed initially on bases other than coinvolvement in smoking [such as family background and proximity in the community, neighborhood, or school].

This is obviously the case within the family (one does not choose his family), and it is also hypothesized to be the typical case with peer associations. However, formation or continuation of peer friendships will be affected in part by one's own attitudes and prior or anticipated parental and peer reinforcement. Further, after smoking has begun and the consequences accompanying it are experienced, the associational patterns (over which one has any choice) may themselves be altered so that the fact that one is drawn to or chooses further interaction with others is based, at least in part, on whether they too are smokers. (Akers 1992: 249-250)

The social learning model specified below is explicitly based on, and allows a test of, the hypothesized relationships among the variables in this process.

Previous Research on Social Learning Variables
Previous research provides considerable support for social learning theory as an explanation of deviant behavior and drug use. There is abundant evidence to show the significant impact on deviant behavior of learning in the family, especially in the early years. The family is usually a conventional socializer providing antirelational definitions, conforming models, and the reinforcement of conformity through parental discipline, but can also produce deviant behavior through parental models, socialization failures, and reinforcement of deviance (Patterson 1975; Snyder and Patterson 1995; Adler and Adler 1978; McCord 1991). Other than one's own prior deviant behavior, the best single predictor of the onset, continuance, or desistance of crime and delinquency is differential association with conforming or law-violating peers (Loeber and Dishion 1987). More frequent, longer-term, and closer association with peers who do not support deviant behavior is strongly correlated with conformity, whereas greater

association with peers who commit and approve of delinquency is predictive of one's own delinquent behavior. The differential peer association variable is usually the most strongly related to delinquency, alcohol and drug use and abuse, adult crime, and other forms of deviant behavior. There is a sizable body of research literature that supports the theory's hypotheses about the importance of differential association, imitation, definitions, and differential reinforcement in explaining crime, delinquency, and drug use (see for example, Jensen 1972; Krohn 1974; Burkett and Jensen 1975; Minor 1980; Winfree and Griffiths 1983; Elliott et al. 1985; Patterson and Dishion 1985; LaGrange and White, 1985; Dembo et al. 1986; Fagan and Wexler 1987; Winfree et al. 1989; Sellers and Winfree 1990; Loeber et al. 1991; McGee 1992; Benda 1994; Warr and Stafford 1991; Agnew 1991, 1993; Warr 1993). When empirically evaluated against other theories, using the same data collected from the same samples, variables taken from social learning theory are usually found to have stronger effects than variables derived from the other theories (see Akers and Cochran 1985; Matsueda and Heimer 1987; White et al. 1986; McGee 1992; Benda 1994).

Research on adolescent smoking is also consistent with social learning, particularly the peer association component of the theory (Charlton and Blair 1989; Urberg et al. 1991). The findings from several studies of adolescent drug use and delinquency favor the typical peer process in which a youngster associates differentially with peers who are deviant or hold tolerant attitudes toward deviance, learns new or additional definitions favorable to delinquent behavior, is exposed to deviant models who reinforce delinquency, then initiates or increases that behavior, which in turn has an effect on his definitions and associations (Sellers and Winfree 1990; Agnew 1991, 1993; Elliott and Menard 1991; Empey and Stafford 1991; Warr 1993).

Previous Research on Social Learning Theory by Akers
Research conducted by Akers and his associates on a range of behavior from minor deviance to serious criminal behavior also provides support for social learning theory. The first of these was the Boys Town study, a survey of adolescent substance use and abuse among approximately 3,000 students in grades 7 through 12 in eight communities of the midwest (Akers et al. 1979; Krohn et al. 1982; Lanza-Kaduce et al. 1984; Akers and Cochran 1985; Akers 1992). The second was a 5-year longitudinal study of smoking among junior and senior high school students in Muscatine, Iowa (Lauer et al. 1982; Krohn et al. 1985; Akers et al. 1987; Spear and Akers 1988; Akers 1992). The third project was a 4-year longitudinal study of conforming and deviant drinking among 1,410 elderly respondents in four communities in Florida and New Jersey (Akers et al. 1989; Akers and La Greca 1991; Akers 1992). The fourth was a study of rape and sexual coercion among two samples of college males (Boergerer et al. 1991; Boergerer 1992; Boergerer and Akers 1993).

In the elderly drinking study, an OLS regression model with three sets of social learning variables (differential association, definitions, and differential reinforcement) accounted for a very sizable 59% of the variance in the frequency of drinking in the past year and 52% of the variance in a quantity/frequency measure of drinking, and LISREL and logistic regression models also provided support for the theory as an explanation of frequency of use and problem drinking (Akers et al. 1989; Akers and La Greca 1991). In the first study of college males (N=285), the social learning variables of association, reinforcement, definitions,
and imitation accounted for 53% of the variance in their perceived likelihood of using force in sex or committing rape, 41% of the variance in using nonphysical coercion in sex, and 15% of the variance in rape. In the second study (N=477), the social learning model accounted for 21% of the variance in rape and 54% of the variance in rape proclivity (readiness to commit rape). Although all of these studies produced findings in support of social learning theory, the most relevant to the present analysis of adolescent smoking are the Boys Town and Iowa studies.

The main focus of the Boys Town study was on explanation of abstinence and frequency of use of alcoholic beverages and marijuana, including variations among users in abuse of these substances as a specific test of the general social learning theory. Social learning models of drinking and marijuana use were constructed incorporating measures of the four main sets of variables of differential association, imitation, definitions, and differential reinforcement. The findings from that study offered strong support for the theory. Strong bivariate relationships were found with peer associations and attitudes, one’s own positive/negative definitions, reinforcement balance, and the rewards-costs of use. Multiple regression analyses showed that more than half of the total variance ($R^2=.54$) in frequency of drinking and over two-thirds of the variance ($R^2=.68$) in marijuana use were accounted for by the theoretical variables.

Not only the full model, but each of the major subsets of social learning variables in the models explained a substantial portion of variance in drinking and marijuana use, with the exception of the measure of imitation, which explained modest amounts of variance. The weaker effects of imitation were not unexpected. The imitation measure captured the narrowest range of empirical phenomena in the social learning process, and the interrelationships among the independent variables expected in the theory meant that much of the impact of imitation was absorbed by the other variables. The role of imitation in social learning theory is considered to be more important in first starting to use than in frequency of use, maintenance of use, and abuse which were the dependent variables in this study.

The social learning variables also substantially affected the probability that the adolescent who begins use will move on to more serious involvement in drugs and alcohol. The analysis of abuse of drugs among users produced findings in support of social learning theory similar to those for differences between users and nonusers. The amount of variance explained in drug abuse was lower than it was for drug use (partly as a function of the more truncated variance in the abuse variables), but it was still substantial (31% and 39%, respectively, of the variance in alcohol and marijuana abuse).

The Boys Town data also supported the hypothesis that the social learning variables will differentiate between those who begin and then stop using drugs and those who begin and continue to use. That analysis went beyond drinking and marijuana use to measure cessation of alcohol, marijuana, stimulants, depressants, and stronger drugs such as heroin and cocaine. Cessation was related to a preponderance of nonusing associations, aversive drug experiences, negative social sanctions, exposure to abstinent models, and definitions unfavorable to continued use of each of these drugs. The squared canonical correlations (from discriminant function analyses) ranged from .18 for cessation of alcohol to .35 for marijuana and .36 for stronger drugs (Lanza-Kaduce et al. 1984).

In the Iowa study, social learning theory was first tested by analyzing data from the first year survey (Spear and Akers 1988). Indicators of each of the four major social learning concepts were moderately to strongly correlated with smoking frequency and in the expected direction ($r=.20$ to $.63$). The social learning model explained more than half of the variance in smoking behavior ($R^2=.54$). Thus, the cross-sectional findings of the Boys Town study of adolescent drinking and use of marijuana and other drugs were essentially replicated in the results from the Iowa study of adolescent smoking. Indeed, the level of explained variance in smoking in the latter study is exactly the same as the level of explained variance in teenage alcohol behavior found in the former study. Just as in the Boys Town study, the imitation variable in the Iowa smoking study was correlated with the dependent variable, but its zero-order correlation and net regression effects were less than those of the other social learning variables. The influence of both parents and peers was seen in the findings. When neither parents nor friends smoked, the adolescent was very likely to abstain from cigarettes and was almost certain (99%) not to be a regular smoker. On the other hand when both parents and friends smoked, 84% of the youngsters also smoked, at least experimentally, and 44% were regular smokers (Akers 1992).

The next step in the Iowa study was to test the social learning model longitudinally with a panel of respondents who were in the study for the first 3 years. In this analysis, two path models were constructed, one for initiation of smoking and one for maintenance of smoking. The initiation model included only those respondents who were abstainers in the first year and second year of the study. The goal was to predict who in this cohort would still be abstinent and who would smoke at least once or twice in the third year of the study. The direct and indirect effects of the learning variables on initiation of smoking were in the direction expected but the effects were weak ($R^2=.03$). At the same time, the social learning maintenance model did a very good job of predicting the continuation or cessation of smoking. The social learning variables measured in the first and second year accounted for 41% of the variance in smoking or quitting by the third year (Krohn et al. 1985).

These previous analyses of social learning theory with the Iowa smoking data have tested both cross-sectional and longitudinal models predicting smoking behavior as the dependent variable based on 3 years of data. The present analysis includes 5 years of data and tests for sequencing and reciprocal effects proposed in social learning theory.

Methodology

Sample and Procedure

The project to test this theory of adolescent smoking was a longitudinal study of secondary school students in Muscatine, Iowa, with a population of about 23,000. The research was a self-report questionnaire survey of students attending the two junior high schools (grades 7 through 9) and the one senior high school (grades 10 through 12) in the town. The sample each year (about 2,000) included students who had participated in one or more years previously, new respondents in the seventh grade, and new students coming into any grade who had not taken part the year before. Of course, graduating seniors and students in lower grades who left the school system the next year or who were not in attendance on the day of the annual survey were lost to further survey. Embedded within this overall sample is a panel of 454 students (in grades 7 and 8 in the first year of the study) who participated in the study for all 5 years. The analysis here is of the longitudinal data collected on this panel.
Validation of Self-reported Smoking Behavior with Biochemical Measures and Randomized Response

The truthfulness of oral or written reports of behavior is an issue regardless of the behavior measured, but it is especially salient if the information asked for in the interview or survey questionnaire is sensitive, potentially incriminating, or embarrassing. As we argue above, smoking by teenagers is socially disapproved. When teenagers smoke, they do so knowing that it is generally disapproved, and for many it is specifically without parental knowledge or permission. Therefore, it can be assumed that for many respondents there will be some motivation to conceal or underreport smoking for fear of disclosure. At the same time some may be motivated by a desire to be different or to show willingness to flout parental and social rules to falsely report themselves as smokers or to overreport how much they smoke. When the reported acts involve consumption of some substance such as tobacco, we have the opportunity to check response validity by measuring the metabolized residues of the substance in body fluids and comparing this with self-reported measures of consumption. This provides an independent, biochemical indicator of behavior that respondents cannot modify to produce a false reading either overreporting or underreporting actual use.

In the Iowa study (Akers et al. 1983) we gauged the truthfulness of responses to our questions about smoking by matching the answers with the level of thiocyanate (a chemical residue of nicotine) found in saliva samples taken from the same respondents. Through matching code numbers assigned to each respondent that were placed on the questionnaire and the vial containing the saliva sample (3 ml), an individual’s self-report responses to a series of questions about smoking could be matched with the amount of thiocyanate in his or her saliva. Response validity was also checked with a technique known as “randomized response.” In this technique the group rate of self-reported smoking given by respondents on the confidential questionnaire were checked against the rate obtained from an anonymous response to one question randomly selected from two questions. One question asks something innocuous (e.g., my birthday is in April) and the other asks the more sensitive question of interest (e.g., I have smoked cigarettes yesterday or today). The response is given on a single sheet with nothing on it except the two questions and a place to check the response (in this case Yes or No). Because there is no way for anyone except the respondent to know which question was answered by that individual and because even the respondent’s reply does not identify which question has been selected, the procedure makes it obvious to the respondent that anonymity cannot be compromised. Group proportions measured by the randomized response can be used as a validity check by comparing them with group proportions obtained from responses to the questionnaire.

The levels of thiocyanate found in the saliva corresponded closely to the self-reports of the teenagers in the study about their smoking within the last 48 hours, how frequently they smoked, how many cigarettes they smoked each day, and how they described themselves as a nonsmoker, moderate smoker, or heavy smoker. The randomized response procedure also showed a high level of response validity to the questionnaire. Both the biochemical measure of nicotine residues found in the saliva of respondents and the randomized response procedure indicated that valid self-reports of smoking behavior were obtained.

Measurement of Variables

The measure of Smoking is the frequency of cigarette smoking. Respondents were asked how often they smoke cigarettes along a 6-point scale from never smoked to smoke every day or almost every day. Scales of the social learning variables measured with multiple questionnaire items were created by summing responses for items for which the standardized score loaded .40 or above on the factor in a factor analysis. The reliability of each scale was established by Cronbach alpha coefficients of .50 to .91 (see Krohn et al. 1983; Spear and Akers 1983).

The measures of Differential Reinforcement involved asking about both positive and negative consequences. Some of these are social and others have to do with the immediate and longer term direct physical effects of smoking. The social reinforcement measures are Parental Reinforcement and Friends Reinforcement. These are the respondents’ reports of the positive/negative reactions of father and mother and of their best friends to the adolescent’s smoking. The other measure of differential reinforcement is based on responses to a list of “good things” and a list of “bad things” that “happen or you think would happen to you from smoking cigarettes.” This overall Balance of Reinforcement was calculated by subtracting the sum of the negative consequences from the sum of the positive consequences checked by the respondents.

The measures of Differential Association include three subsets of variables, differential peer association, parents’ definitions, and friends’ definitions. Differential peer association was measured by asking “How many of your friends smoke?” for friends known the longest time (duration), friends most often associated with (frequency), and best friends (intensity). The response categories are none, less than half, more than half, all, or almost all. These three items are highly intercorrelated and were formed into a scale of Differential Peer Association. The measure of peer modeling or imitation attempted to measure general awareness of peer smoking (on the assumption that this indicates observation of models), but this overlapped with the differential peer association measure and, indeed, is similar to measures used in the literature as a peer association variable. Moreover, in our study, the measures of differential peer association and modeling are highly intercorrelated (r=.63). Therefore, we do not include a measure of imitation in this analysis. This should not make a major difference, because imitation is more important in initiation than maintenance and the intercorrelation indicates that much of the effects of the imitation variable will be picked up by the peer association measure. There is no comparable measure for “differential parental association,” and we did not attempt constructing one.
However, directly comparable measures were devised for both parents and friends as the most important primary groups in which one is exposed to attitudes favorable and unfavorable toward smoking. This was done by asking respondents to report for both parents and friends “What is the general attitude of each of the following toward teenagers smoking?” The response categories are approve, sometimes approve and sometimes disapprove, disapprove, and strongly disapprove. From this list we devised scale measures of Parents’ Definitions and Friends’ Definitions.

One’s own Definitions of smoking behavior were measured by a general question on one’s general Positive/Negative Definition of teenage smoking. Definitions were also measured by a series of attitudinal questions to which the respondents indicated, on a 4-point Likert scale, strong agreement to strong disagreement. Three of these attitudinal questions formed a scale of Neutralizing Definitions favorable to smoking (“takes many years for bad health effects from smoking,” “smoking is all right if you do not get the habit,” and “is all right with parental permission”). Two of the items were summed to form a scale of Negative Definitions counter to smoking (“cigarette advertising should be stopped” and “sales of cigarettes should be outlawed”).

Specification of the Social Learning Model of Adolescent Smoking

The model tested here incorporates social learning variables and smoking behavior in the first, third, and fifth year of the survey, designated here, respectively, as Time 1 (T1), Time 2 (T2), and Time 3 (T3). Therefore, it is a model of abstinence and continuation or cessation of cigarette smoking rather than a model of the initiation of smoking. We measure both the social learning and smoking variables at each time in order to examine the cross-lagged effects as a way of approximating the process specified in the theory. This avoids a problem with the common model of change in which a concept at one point is explained by other variables after controlling for earlier measures of the concept, identified by Lanza-Kaduce and Hollinger (1994:13) in their study of college cheating. “If the effects of prior cheating are removed (because they have a feedback effect on learning) so the effects of the social learning variables can be studied, the only variance left to explain is that for persons who changed their cheating patterns. Inasmuch as social learning theory is purported to explain both change . . . and maintenance of deviance . . . introducing prior deviance as a control results in an analysis that addresses only the change part of the process.”

The model we propose in figure 1 incorporates both change and stability in behavior. In this model, adolescents’ smoking is regressed on social learning variables (β₄1 and β₆3) while measuring stability in smoking behavior across time (β₃1 and β₅3), and social learning variables are regressed on smoking (β₃2 and β₅4) while measuring stability in the learning variables across time (β₄2 and β₆4). Two sets of structural equations are developed to assess this model. The first is a model of the overall process with reinforcement, differential association, and definitions as the indicators of a latent construct of the general social learning process. The second is a series of 3w²n models for each of the three subsets (each with three indicators) of social learning variables, differential association, differential reinforcement, and definitions.

All of these are based on the model in figure 1, with smoking measured in the first year (T1), the third year (T2), and the fifth year (T3) and with each of the social learning variables measured at each of those times. The variables have parallel form across time because the same items were used through the 5 years. Therefore, in the analysis of the models we assume that the parallel forms of each indicator have equal factor loadings and equal error variances by equating λ-coefficients across time and by equating error terms across time (Jöreskog 1971). The model posits covariances between two different latent variables across time (φ₂₁, φ₄₃, and φ₆₅). The model includes the T1 to T2 and T2 to T3 stability paths for latent variables, and also allows T1 variables to influence other T2 variables and T2 variables to influence other T3 variables. The general model estimates cross-lagged paths of adolescents’ smoking at T1 to social learning at T2, social learning at T1 to adolescent’s smoking at T2, adolescents’ smoking at T2 to social learning at T3, and social learning at T2 to adolescents’ smoking at T3.

The other models are identical in form to the overall model, except that the overall social learning construct is replaced in the different models with, respectively, differential reinforcement, definitions, and differential association. Each of these learning variables has three indicators.

**Figure 1**

A Cross-lagged Social Learning Model for Adolescent Smoking

![Figure 1](image)

**Findings**

Table 1 presents the matrix of bivariate correlations. All of the relationships are in the expected direction. The cross-sectional relationships between smoking and the social learning variables are the strongest, especially in the fifth year (T3) when all the correlations, except for parents definitions, are .4 or higher, with...
peer associations and one’s own definitions showing the strongest bivariate relationships with smoking (r = .6 and .7, respectively). The social learning variables measured at T1 and T2 are also substantially related to the expected direction to subsequent smoking behavior. As expected, the smoking at T1 is related to smoking at T2 (.547) and smoking at T2 with smoking at T3 (.527). These 2-year lag relationships are stronger than the 4-year lag relationship of smoking at T1 with smoking at T3 (.340). Not surprising smoking behavior at one time is predictive of smoking behavior at a later time, but it becomes progressively less predictive the farther into the future one goes (Akers et al. 1987).

The correlations among social learning variables across the years are also positively correlated. The magnitude of the correlations ranged from .18 for parents' definition at T1 with T2 to .45 for adolescents' negative definition at T2 with T3. Thus, social learning variables are related, but the relationships are not at a level to suggest problems of multicollinearity (less than 21% of shared variance).

As noted above, the model was tested for overall social learning (fig. 2) and each of the major social learning dimensions of differential reinforcement (fig. 3), definitions (fig. 4), and differential association (fig. 5). Beta coefficients and factor loadings are reported in each figure. Factor loadings for latent constructs and standardized regression coefficients were estimated. The estimations of these models were calculated with the LISREL VII statistical program (Jöreskog and Sörbom 1989). LISREL VII is based on maximum likelihood statistical theory and allows for multiple indicators of constructs, adjusts parameter estimates for the unreliability of measurement when multiple indicators are used, permits correlated residuals, and provides a test of the extent to which overidentified models fit the data. LISREL is well suited to testing longitudinal models because it takes into account correlated measurement errors that are usually present when the same measure is used at two or more points in time.

Figure 2 shows that the overall social learning model of adolescent smoking is supported. The model fits the data well; X² with 41 degrees of freedom = 60.88, p = .023. Although the X² is significant at the .05 level, its ratio to degrees of freedom (1.5) is low (Carmine and McIver 1981). The goodness-of-fit index and adjusted goodness-of-fit index are greater than .90 (Jöreskog and Sörbom 1989). Factor loadings of social learning variables are relatively high and stable across indicators.

The stability path coefficients for adolescents' smoking (β31 and β53) and social learning (β42 and β64) show that adolescents' earlier smoking is associated with later smoking (.35 and .32) and that the social learning variables at one time are highly associated with the same variables at a subsequent time (.73 and .56). This strong influence of variables at an earlier stage on the same variables at a later stage, especially for the social learning variables, suggests that the stability is relatively autonomous (Hertzog and Nesselroade 1987). The model allows T1 latent variables to influence other T2 variables and T2 variables to influence other T3 variables and includes estimates of our cross-lagged paths to address the issue of change: effects of social learning at T1 and T2 on adolescents smoking, respectively, at T2 and T3, and effects of smoking at T1 and T2 on social learning at T2 and T3.

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**Table 1**

Bivariate Correlations for Adolescent Smoking Behavior and Social Learning (N = 440)
The cross-sectional relationship between social learning and smoking at T1 is strong; the cross-sectional relationships in subsequent years are not as strong, but remain high. The model accounts for 33% of the variance in smoking at T2 and 31% of the variance in smoking at T3. The magnitude of effects (in the .3 range) of the model variables on smoking shows that prior social learning predicts later smoking almost as well as does prior smoking behavior itself. The model also accounts for 43% of the variance in social learning at T2 and 39% of the variance at T3. Unlike smoking behavior, however, most of the effect on the social learning construct comes from the same social learning construct at an earlier time, rather than from prior smoking behavior. Social learning at T1 is significantly and positively associated with adolescent smoking behavior at T2 (.27) and smoking behavior at T2 is positively, but not significantly, associated with social learning at T3. In turn, adolescent smoking behavior at T1 is nonsignificantly, and unexpectedly negatively, associated with social learning at T2, and social learning at T2 is significantly, positively associated with smoking behavior at T3 (.27). Only the variables measuring social learning have significant effect on adolescents' smoking behavior whereas their abstinence or frequency of smoking has some, but nonsignificant, lagged effects on social learning variables. Therefore, there is some evidence of reciprocal effects between the social learning variables and smoking, but the lagged effects of social learning on smoking are stronger than those of smoking behavior on social learning.
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We turn now to findings on the other models for each of three major dimensions of social learning—differential reinforcement, definitions, and differential association. The findings on these models are similar to those on the overall model and are supportive of the theory. The variance explained in smoking behavior in the three models ranges from 29% to 40%, and the variance explained in social learning ranges from 28% to 47%.

Figure 3
Model with Standardized Estimates for Adolescent Smoking and Differential Reinforcement
(N = 440, \*p < .05)

Chi-square (41) = 174.70 (p = .000)
Good-of-fit index = .959
Adjusted good-of-fit index = .933

The model for differential reinforcement (fig. 3) shows findings consistent with those on the overall model (fig. 2). Adolescent smoking and social learning variables are highly associated with later smoking and social learning (stability coefficients .31, .26, .62, and .46, respectively). Adolescents smoking at T1 is not significantly related to differential reinforcement at T2 (-.02), but smoking at T2 is positively, although not significantly, associated with differential reinforcement at T3 (.18). The effects of differential reinforcement on smoking are positive and significant both from T1 to T2 (.31) and from T2 to T3 (.33). The fit for this model, while acceptable, is less than the other models. The X^2 with 41 degrees of freedom = 174.70 is significant and the ratio of degrees of freedom to chi-square is high compared to the other models, but it is less than 5. Moreover, it is a good fitting model when measured by the goodness-of-fit index (above .90). Factor loadings of differential reinforcement variables are moderate and stable across indicators. The model for definitions favorable and unfavorable to smoking (fig. 4) is very similar to the model for differential reinforcement, except both

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coefficients measuring magnitude of effect of smoking on subsequent definitions are essentially zero. Apparently, although one's attitudes toward smoking have a clear influence on abstaining or smoking, one's smoking behavior does not have much of a subsequent effect on his or her positive/negative or neutralizing attitudes toward smoking. This model also fits the data quite well. Again, the X^2 of 82.27 with 41 degrees of freedom is significant, but the ratio of chi-square to degrees of freedom is low. The goodness-of-fit index and adjusted goodness-of-fit index are above .90.

Figure 4
Model with Standardized Estimates for Adolescent Smoking and Adolescent's Own Definition
(N = 440, \*p < .05)

Chi-square (41) = 82.27 (p = .000)
Good-of-fit index = .969
Adjusted good-of-fit index = .941

Figure 5 shows the LISREL model for differential association. This model also looks similar to the other models for T1 to T2 effects, but shows greater reciprocal effects at T2 to T3 than was found for the other models. The lagged effects of smoking at T2 on differential association at T3 is significant and of about the same magnitude as the effects of peer association at T2 on smoking at T3.

Because the only measure of parental association in figure 5 is parents definitions and that indicator loads weakly with the peer association and peer definitions indicators, we ran another model with only the latter two indicators of differential peer association. This peer-only model (figure not shown) fit the data better, with a lower chi-square and higher goodness-of-fit index, but the magnitude and significance of the coefficients remained similar to those in figure 5. Smoking at T2 continued to have a significant effect (and indeed slightly increased in magnitude) on peer associations at T3. These findings would indicate
that as smoking behavior develops, it is shaped by association with peers, exposure to their normative definitions of smoking, and social reinforcement, but then over time, one’s own smoking behavior comes to exert influence over patterns of association with friends. Again, this pattern of peer association > behavior > peer association is consistent with social learning theory.

Figure 5
Model with Standardized Estimates for Adolescent Smoking and Differential Association
(N=440, *p < .05)

Chi-square (41) = 113.30 (p = .000)
Good-of-fit index = .958
Adjusted good-of-fit index = .921

Discussion and Conclusions
The purpose of this study was to test a social learning model of adolescent smoking with 5-year longitudinal data from the Iowa smoking study. The general conclusion is that the theory is supported. Therefore, the research reported here adds to the extant body of research favoring a social learning explanation of deviant behavior. It goes beyond much of that previous research in testing not only for the independent effects of the social learning variables on deviant behavior, but also for the more complex sequential learning process recognized in social learning theory. The behavioral process in smoking remains relatively stable through time, and the social learning variables continue to have an effect on smoking behavior through the years even when taking into account behavioral stability and the effect of prior smoking behavior on social learning variables. The findings in support of the model are very similar in all four versions—the overall model, differential reinforcement model, definitions model, and differential association model. However, the findings show that the reciprocal effects of smoking behavior on social learning is clearer for peer associations than for the other learning variables of reinforcement and definitions.

One of the problems with this kind of model is that the magnitude of differences in factor loadings (λ) between different variables (in this case, smoking behavior and social learning variables), and the difference in stability may influence the cross-lagged coefficients (Rogosa 1980). The stability of smoking behavior is less than the stability of the social learning variables and this difference may have suppressed somewhat the effect of smoking behavior on the social learning variables. This suppression is not sufficient to challenge the overall conclusions for adolescent smoking. It suggests, however, that for other kinds of adolescent substance use or deviance, the reciprocal effects of the deviant behavior on the social learning variables may be stronger than they have been shown to be here for teenage smoking. Future research should examine the extent to which the same sequencing and magnitude of effects prevail with other types of drug and deviant behavior.

Akers et al. (1987) mention that, while social learning variables, as expected, account for deviance for both male and female, the magnitude of the relationships are somewhat weaker for adolescent girls than for adolescent boys. Further, teenage girls are more influenced in their smoking behavior by their boyfriends than boys are by their girlfriends. Another issue concerns the relative influence of parents and peers on adolescent deviant behavior. Theoretically, both parents and peers substance-use attitudes and behaviors should have a significant impact on adolescent substance use, but the relative influence of parents and peers is expected to vary somewhat by age and substance. Thus, future research should test different models testing for effects of gender and of the relative effects of parents and peers. Also, the smoking initiation model tested by Krohn et al. (1985) with 3 years of data should be modified and retested using the 5-year panel. There were some problems of measurement and misspecification in that earlier analysis that may be corrected with more years of data and the kind of structural equation models used here.

Finally, it should be noted that the same general learning process is hypothesized to operate at any stage in the life cycle, but with different parts of the process and the empirical measures of the concepts varying somewhat by age (Akers and LaGreca 1991). For instance, parental models and reinforcement should be more important at a younger age and peer influences more important in the adolescent years; peer associations and reinforcement in adolescence are in school-related and neighborhood groups whereas later in life they are work and leisure groups and organizations. Therefore, it should be compatible with lifestyles or developmental perspectives ( Sampson and Laub 1993). Thus far, a preponderance of research on the theory (as is true for research on most of the leading theories of deviance and drug use) has been with adolescent subjects. There need to be additional efforts made to test the explanatory power of modeling, reinforcement, definitions, associations, and other learning variables on deviance and drug use in groups at all stages of the life cycle.

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Fagan, J., and S. Wexler

Fishbein, D.H., and S.E. Pease

Goode, E.

Gottfredson, M., and T. Hirschi

Hertzog, C., and J.R. Nesselroade

Hirschi, T.

Jensen, G.F.

Jessor, R., and S.L. Jessor

Jöreskog, K.G.

Jöreskog, K.G., and D. Sörbom

Kessler, R.C., and D.F. Greenberg

Krohn, M.D.

Krohn, M.D., R.L. Akers, M.J. Radosevich, and L. Lanza-Kaduce

Krohn, M.D., W.F. Skinner, J.L. Massey, and R.L. Akers

LaGrange, R.L., and H.R. White

Lanza-Kaduce, L., R.L. Akers, M.D. Krohn, and M. Radosevich

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Lanza-Kaduce, L., and R. Hollinger

Lauer, R.M., R.L. Akers, J. Massey, and W.Clarke

Loeb, R., and T.J. Dishion

Loeb, R., M. Stouthamer-Loeber, W. Van Kammen, and D.P. Farrington

Matsueda, R.L., and K. Heimer

McCord, J.

McGee, Z. T.

Minor, W.W.

Patterson, G.R.

Patterson, G.R., and T.J. Dishion

Pomerleau, O.F., and C.S. Pomerleau

Ray, O., and C. Keir

Rogosa, D.

Rotter, J.

Sampson R. J., and J.H. Laub

Sellers, C.S., and T.L. Winfree
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Snyder, J. J., and G. R. Patterson

Spear, S., and R.L. Akers

Stafford, M.C., and S. Ekland-Olson

Strickland, D.E.

Stumphauzer, J.S.

Thornberry, T.P.


Troyer, R. J., and G. E. Markle

Urberg, K.A., C. Cheng, and S. Shyu

Warr, M.

Warr, M., and M. Stafford

White, H.R. M. Bates, and V. Johnson

White, H.R. M. Bates, and V. Johnson

White, H.R., V. Johnson, and A. Horowitz

Winfree, L.T., and C.T. Griffiths

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Winfree, L.T., C.T. Griffiths, and C.S. Sellers