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The effect of peer and parental smoking on adolescent smoking initiation: Exploring potential moderators

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THE EFFECT OF PEER AND PARENTAL SMOKING ON ADOLESCENT
SMOKING INITIATION: EXPLORING POTENTIAL MODERATORS

BY

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DISSERTATION

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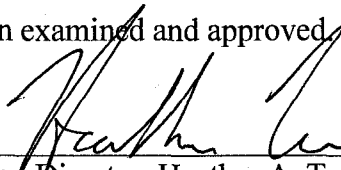
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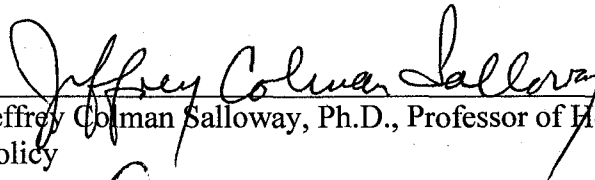
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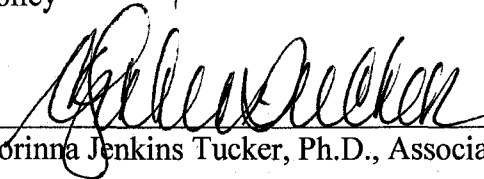
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DEDICATION

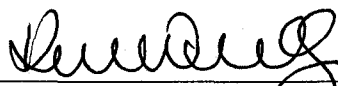Many people deserve my gratitude in helping me to achieve the completion of this dissertation. Thanks to my entire family, especially my wife Janet, my children Carla, Colin and Cody who have sacrificed along with me so that I might achieve this goal. My committee has provided much guidance along the way, and I will forever remember their help and kindness. Professor Heather Turner especially deserves recognition due to her perseverance with me through this extended process. I would also like to thank Pam DiNapoli and others in the Nursing Department at UNH whose assistance was instrumental along the way.

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University of New Hampshire, December, 2008

The factors that contribute to smoking initiation among adolescents are poorly
understood. The current approaches to smoking prevention may have achieved their
maximum potential as evidenced by a stalling in the decline in smoking rates. To date,
approaches to smoking prevention based on social and individual factors have previously
met with limited success. A promising new approach will be to examine the interaction
between social and individual factors and the effects of their interaction on smoking
initiation. Parental and peer smoking behaviors are well-known risk factors for smoking
initiation. Several theoretical models suggest that perceptual or interpretative processes
may moderate the influence of factors such as these on the smoking initiation process.
This study looks at age (as a proxy for adolescent development), depression and school
performance as potential moderators of the impact of parental or peer smoking. This
study uses a large longitudinal sample (The Teenage Attitudes and Practices Surveys –
1989 and 1993) to explore for these relationships. Results show very limited support for
the impact of potential moderated relationships, with only one of the six hypothesized
interactions being supported (peer smoking and school performance). This would suggest
that theoretical models which include concepts of perceptual or interpretative processes
as moderating influences need to continue to evaluate their validity. Another finding of
the study is a significant main effect of school performance on smoking initiation –a
relationship which has not been previously reported in a national longitudinal sample.
This study also found support for depression as an antecedent to smoking initiation – a
relationship whose causal direction continues to be controversial. Continued exploration
of the complex relationships between these social and individual factors may allow for
the development of more effective evidence-based smoking prevention programs.

CHAPTER I

STATEMENT OF THE PROBLEM

Research on tobacco smoking clearly demonstrates its association with an

extensive list of physical illnesses and disorders (Appendix 1). This has been widely

recognized since the Surgeon General's 1964 Report on Smoking and Health. Although

much progress has been made in controlling smoking since that time, and smoking rates

have declined from 45% of the American population to about 20%, the decline has stalled

in recent years. In fact, there was no change in smoking rates between 2004 and 2005

(20.9%) (CDC 2006). Even more concerning is the number of young people who *start*

smoking. Smoking initiation rates among adolescents continue to be well above goals set

by the Healthy People 2010 initiative from the United States Office of Disease

Prevention and Health Promotion, with adolescent smoking rates currently near 35%.

Early smoking research explored many elements associated with the smoking

process, including adolescent smoking initiation. More recently, however, smoking

research has turned away from initiation and increasingly focused on the areas that

seemed to result in greater reductions in smoking rates. For example, the "Best Practices

for Comprehensive Tobacco Control Programs", issued by the CDC in 1999, suggested

that statewide programs should focus on "promoting media advocacy, implementing

smoke-free policies, and reducing minors' access to tobacco" (CDC 1999). This focus

results in an emphasis on general public education campaigns ("counter marketing"),

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increased law and school policy enforcement, and increased taxes to decrease adolescent access to cigarettes. Another element of the 1999 report was an emphasis on cessation programs, with large amounts of the funding being focused on helping those who already smoke to stop.

Emphasis on counter marketing and tax increases have resulted in a de-emphasis of those interventions focused on preventing adolescents from initiating smoking. This represents a significant shift from the approaches used immediately after the Surgeon General's report of 1964 which included evidence of correlations between adolescent smoking initiation and social factors such as peer smoking, parental smoking, and depression rates. Since that time, programs such as DARE have attempted to address social predictors, but have been met with very limited success. Adolescent smoking initiation rates have now stalled at about 20%. This would seem to suggest that it is time to re-explore the other social risk factors in an effort to develop new insights into the adolescent smoking initiation process. This exploration will need to utilize new approaches. Approaches which consider *combinations* of social and individual factors have the potential to inform intervention approaches. Patterns of combinations may allow researchers to identify high-risk adolescents. In order to identify patterns, we will need to better understand how combinations of factors might exert their influence on smoking initiation among adolescents.

Many individual risk factors for adolescent smoking initiation are well known. For example, past studies suggest that peer and parental smoking are significant risk factors. The processes by which these risk factors exert their effects, and the conditions under which those effects are greatest, are not well understood. However, studies that

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build on and expand earlier work by considering how some factors are influenced by other factors may provide a greater understanding of these processes and conditions and allow for the development of interventions that may help prevent adolescent smoking initiation.

A number of conceptual models that have been utilized in smoking research suggest processes that involve a dynamic interaction between social and individual factors. Conceptual approaches such as Social Attachment Theory, Social Learning Theory, and Protection Motivation Theory all include suggestions of this interaction. In addition, each of these theories includes, as a key element, the interaction of social factors and moderating processes which would alter the perception of social factors. Analyses which explore and explain the nature of these interactions can help us in our overall understanding of the smoking initiation process and in the development of a theoretical basis for effective prevention strategies. These kinds of explorations can also provide a test of the assumptions of these conceptual approaches. If these assumptions are not supported, it would suggest the need for refining these theories or replacing them with new theories for smoking prevention.

This study will build on earlier studies by exploring for the existence of conditional relationships between parent and peer smoking behavior and individual factors such as age, school performance and depression.

Age is considered in this discussion because, during the various stages of adolescent development, relationships with parents and peers might be expected to vary in character and intensity, reflecting changes in social and cognitive development, although the empirical evidence for this assertion is mixed. Defining how parents' or

peers' influence on smoking behavior may change during adolescent development could allow for the tailoring of prevention efforts.

School performance is a well-documented risk factor for smoking initiation (Tyas and Pederson 1998), but one which has received very limited attention since the early 1990s. School performance may reflect a number of characteristics. Logically, we would expect that school performance should reflect an adolescent's intellectual abilities-abilities which should allow for an accurate assessment of the risk of smoking. If school success is based on intelligence and problem-solving abilities, these skills would be expected to impact the smoking initiation process. This study will explore the relationship between school performance and smoking initiation in a national longitudinal sample. No studies were found that tested for either a main effect of school performance in this type of sample or the possibility of an interaction effect between school performance and factors such as peer and parental smoking effecting the probability of smoking initiation.

Another individual characteristic that would be expected to impact the adolescent's perceptual and interpretative processes regarding smoking initiation is depression. Depression has been found to be associated with smoking in numerous studies. Moreover, some evidence already exists suggesting that peer smoking behavior may interact with depression effecting smoking initiation (Patton, Carlin, Coffey, Wolfe, Hibbert, and Bowes 1998; Ritt-Olson, Unger, Valente, Nezami Chih-Pingchou, Trinidad, Milam, Earleywine, Tan, and Anderson Johnson 2005). This raises the possibility that depression makes an adolescent more vulnerable to peer influence. No known studies have tested for an interaction between *parental* smoking and depression, but this might

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also create a vulnerability to smoking initiation. The main focus of this study will be defining what impact, if any, factors such as depression have on the influence that peer or parental smoking exert on adolescent smoking initiation.

A number of other factors such as gender, race and socioeconomic status have also been found to have a relationship to smoking initiation. These factors will be considered in these analyses primarily as control variables, although their recent historical trends do also provide support for the importance of looking at these kinds of social factors in the etiology of cigarette smoking.

Further progress in reducing smoking rates will require new approaches. Research that explores and identifies the factors and processes involved in smoking initiation can provide important knowledge that may then be used to design interventions that can effectively prevent smoking initiation. This study will provide new ways of looking at the relationships between some well known risk factors for smoking initiation in a national, longitudinal dataset.

CHAPTER II

LITERATURE REVIEW

Looking at combinations of smoking risk factors has the potential to enrich our overall understanding of the smoking initiation process. It may also provide a practical understanding of how these factors can be influenced to reduce smoking initiation rates. This section will start by addressing relevant theoretical approaches. Next, the existing literature for the main effects on smoking initiation for each of the key variables in the model will be described. For relationships that have previously received extensive attention, I will provide a brief overview, and only studies specifically relevant to the current study will be discussed. This section is sub-divided by predictor variables (peer and parental smoking), moderator variables (age, depression and school performance), and control variables (sex, race and family income). Lastly, studies which have looked at combinations of factors will be reviewed.

The six hypotheses of this study which address the proposed interactions between the predictor and moderating variables (for example, peer smoking interacting with depression) will guide the discussion of the existing literature that has explored for the presence of interaction effects. This section will also provide a discussion of the potential implications of the existence of each of the proposed conditional relationships.

Theoretical Considerations

A number of lifestyle issues negatively impacting the health of adolescents – including obesity, lack of exercise, risk-taking and smoking – perplex health behavior researchers as well as health professionals and the laity. One of the most difficult issues is the initiation of smoking. A number of theories suggest that smoking initiation is a function of a dynamic interaction between social factors and individual perceptual or interpretative processes. To date, however, there has been limited success in achieving a comprehensive understanding of the smoking initiation process. This ultimately has limited the creation of effective prevention programs.

A theoretical understanding of the processes of smoking initiation would have great practical benefits. This study will be informed by several theories that have in common the use of the combination of social factors and perceptual or interpretative processes as an explanation for the initiation of smoking. Using the elements that these theories have in common, a conceptual framework will be established which will guide the current study. Three theories that have been frequently cited in the smoking literature will be influential in the development of this conceptual framework: Social Attachment Theory, Social Learning Theory and Protection Motivation Theory. There are numerous examples of the use of these theories in smoking research – Social Learning Theory (Bard and Rodgers 2003; Collins and Ellickson 2004; Flay, Hu, Siddiqui, Day, Hedeker, Petraitis, Richardson, and Sussman 1994; Kobus 2003), Social Attachment Theory (Collins and Ellickson 2004; Gossop, Griffiths, and Strang 1994; Gress and Boss 1996), and Protection Motivation Theory (Daniels 1999; Greening 1997; Leas and McCabe 2007; Pechmann, Zhao, Goldberg, and Reibling 2003) have all been referenced

extensively – an EBSCO search of "social learning theory" and "smoking" yields over one hundred studies.

Interest in Social Attachment Theory (SAT) in sociology goes as far back as Durkheim's (1897/1951) classic study which looked at levels of social attachment and the corresponding probability of suicide. Social attachment is most commonly defined by one's relationships with immediate family, friends and extended family, and social participation in church and other activities. SAT suggests that the kinds of bonds that an adolescent has with family, school, and church, and the level of approval of smoking by these groups, will predict smoking behavior. It also suggests that these bonds will change over time with the relative focus of the adolescent gradually moving from family connections to peer connections. Thus, the relative influence of family and peers on smoking behavior should vary as the adolescent develops. As the importance of these groups change, the normativeness of smoking behavior might also change and affect the probability of adolescent smoking initiation.

There is significant support for the influence of peers (Conrad, Flay, and Hill 1992), families (Fleming, Kim, Harachi, and Catalano 2002), and other elements consistent with SAT (e.g., low religious connectivity) (van den Bree, Whitmer, and Pickworth 2004) on the probability of smoking initiation. Past studies have focused on these relationships, but have not considered how moderating processes might alter the processes involved. It is possible that, by examining how some factors might alter the perceptions or interpretations of these adolescents, we may be able to understand why some adolescents, whose social attachments include smokers, will begin smoking while others do not.

Social Learning Theory (SLT) is another theory commonly used in the study of adolescence. It posits that adolescents learn behaviors by observing and imitating the behaviors of those in the social environment around them. Models could be parents, other adults or peers. Several theories of the adolescent development process (reviewed in Kobus 2003) suggest that the relative impact of these groups should change as the adolescent develops. For example, the relative importance of parents might be expected to be greater in early adolescence and decline as the adolescent becomes more independent and focuses more on relationships with peers.

Like SAT, Social Learning Theory also considers the influence of peers, families and social organizations, but focuses on how they model social behavior. It emphasizes the impact of modeling on the adolescent. Bandura (1977) suggests that, while observed behavior and the social response to that behavior is key to the probability of an adolescent engaging in any particular behavior, the cognitive processes of the adolescent are important in *interpreting* those behaviors and the social responses to them.

The appeal of SLT in understanding smoking initiation is that, if smoking in the family or among friends is normative and accompanied by either a positive or neutral response, the adolescent is likely to adopt that behavior. SLT also suggests, however, that anything that changes cognitive processes might alter this process (Bandura 1977). A theoretical approach that includes a better understanding of variables that impact these interpretative processes may be the key to smoking initiation.

A model from the public health literature, Protection Motivation Theory (PMT) as described by Rogers (1975) considers how evaluation of external threats and self assessment of coping resources, results in adaptive or maladaptive health behaviors. This

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theory uses the assumption that behaviors will be driven in great part by a desire to protect oneself and one's health. There is significant research supporting the PMT model as a predictor of health behaviors (Rogers and Prentice-Dunn 1997). PMT originated as a theory to explain how fear motivates change in health behavior. However, over time, it has evolved (Rogers and Prentice-Dunn 1997) to include the interaction of social and cognitive factors in the prediction of whether individuals will participate in adaptive or maladaptive responses when given the option of healthy or unhealthy behaviors. In this model, cognitive processes such as threat appraisal and coping appraisal moderate the probability of adaptive coping outcomes. Alteration of interpretative processes will change the probability of adaptive versus maladaptive coping choices.

Each of these three models includes processes which affect the perception or interpretation of social factors and thus, the probability of health behaviors such as smoking. Depression is an example of an individual characteristic that might affect perception or interpretation of social factors. Much evidence has established a correlation between depression and smoking, though the causal sequencing and hence the nature of this relationship is unclear. In the SAT, SLT and PMT models, depression would be likely to alter cognitive processes that would then alter the probability of smoking initiation. Depression might change the perception of "severity" or "vulnerability" regarding the risks associated with smoking. It may influence the adolescent to choose a behavior that provides short term benefits (such as stress reduction) rather than longer term benefits (such as avoidance of cancer and other illnesses). Depression might also decrease the adolescent's sense of self-efficacy, thus making the adolescent less resistant to the influence of peers who smoke.

Other variables included in the SAT, SLT and PMT models are adolescent development and school performance. These variables may have similar effects. As an adolescent develops, ability to accurately assess health risks should improve. Adolescents' underlying intellectual abilities (which should be reflected in their school performance) may also result in a greater ability to make positive health choices.

These three theoretical approaches each suggest a potential importance for the interaction of well-known smoking risk factors such as parental or peer influence, and refer to perceptual or interpretative processes therein that may impact the likelihood of smoking behavior in adolescents. If supported by empirical data, these kinds of conditional relationships would support the need for more refined theoretical approaches that could more effectively explain smoking initiation, and could then serve as the basis for future prevention programs. If these conditional relationships are not found to be supported by empirical data, the possibility would have to be considered that social and interpretative factors may not interact in the ways that these theories have suggested. Thus, until adequate empirical data can be collected, any interventions based on these models should be carefully evaluated to determine whether they have the potential to be effective.

This study considers the shared elements of these theories. Using the relationships suggested, a conceptual model was created (Figure 1). Use of this conceptual framework allows for a general assessment of elements suggested by these theoretical approaches.

The conceptual model guiding the current research is as follows:

Depression

↓

Peer smoking

--→ Smoking

↑   ↑   Initiation

Parental smoking

School   Age (Adolescent Development)
Performance
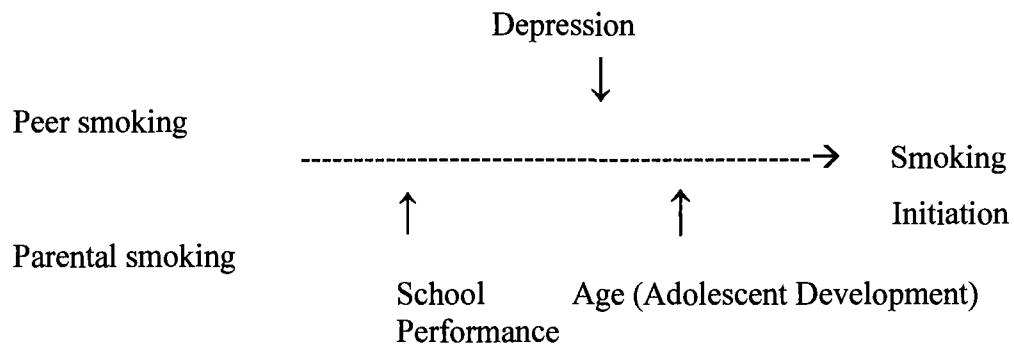
Figure 1: Conceptual Model for the Study (will also control for gender, race and family income)

In this model, depression, school performance and age (as a proxy for adolescent development) are expected to moderate the relationships between peer smoking and smoking initiation and between parental smoking and smoking initiation. If the existence of conditional relationships is supported, then future approaches to smoking prevention that consider the interaction of these factors can be developed. It will be important to control for gender, race, and family income in all of these analyses because these factors may be related to the variables of interest, and are also known to be related to the likelihood of smoking initiation.

Previous Research on Main Effects of Variables of Interest

This section will review the existing evidence related to the main effects of the variables that have been chosen for this analysis. This review serves purposes: 1. before we look at combinations of variables, it is useful to review the overall evidence of how those variables independently influence smoking initiation and, 2. we will also be looking

specifically at two of these relationships in the main effects analysis to try to provide insight regarding the relationships between depression and smoking initiation, and school performance and smoking initiation. The sample that I will be using (the TAPS) has been evaluated for the relationship of adolescent smoking and depression before (Escobedo, Reddy, and Giovino 1998), but this study will use a different dependent variable – a measure of those adolescents who become "regular" smokers. Escobedo had used "smoked on five or more days in the previous month" as the dependent variable. The issue of variability in the definition of the dependent variable in smoking research will be discussed further in the methods chapter.

This dataset has not been previously evaluated for the possibility of a main effect of school performance on the probability of smoking initiation. No studies were found that specifically looked at this relationship in a national longitudinal sample.

The subsections on control variables provide a historical context for the changing smoking patterns associated with gender and race. These discussions not only document the importance of including these variables as controls in the model, but also show how smoking patterns have changed over time. The most likely explanation for these changes is the social and cultural changes associated with race and gender that were occurring over this time period, supporting the suggestion that these kinds of social factors are important to the smoking initiation process.

Predictor Variables

Peer Influence and Smoking

There is substantial evidence that peers are an important influence on an adolescent's transition to becoming a smoker. Bauman and Ennett (1996) have said, "The accumulated wisdom of more than two decades of research on adolescent drug use (including smoking) is that peer influence is a prominent cause, if not the most important factor, among a complicated set of circumstances and risk factors" (p. 185). Numerous studies have found that peer smoking is one of the strongest predictors of adolescent smoking initiation. Conrad, Flay and Hill's (1992) review of 27 prospective studies between 1980 and 1990 found that "peer bonding received consistently positive support in the prediction of smoking initiation" (p. 1720). This finding has been found to be consistent across multi-ethnic groups (Alexander, Allen, Crawford, and McCormick 1999), urban youth (Botvin, Epstein, Schinke, and Diaz 1994), and rural youth (Epstein, Botvin, and Spoth 2003).

While there is general agreement that a relationship between peer smoking and smoking initiation exists, the precise nature of this relationship is not clear. A variety of theoretical approaches have been used to try to explain it based on the assumption that smoking peers will exert social influence on an adolescent. In research combining social learning theory and social identity theory, Kobus' (2003) findings suggest that "the influences of peers are more subtle than commonly thought and need to be examined more carefully, including consideration of larger social contexts" (p.37). Other researchers have also explored the nature of the relationship between peer smoking and smoking initiation using concepts such as peer pressure and social conformity with

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Simons-Morton, Haynie, Crump, Eitel and Saylor (2001) finding support for the contention that adolescents who felt peer pressure from friends were more likely to smoke.

Though there is substantial evidence for the effect of peer influence on smoking initiation, the relationship between the two may actually be more complex than it first appears: peers who smoke may influence adolescents to become smokers, and in addition, smokers may tend to choose other smokers to be their friends. Bauman and Ennett (1994) found that peer group choice was an influence on smoking initiation, and that smoking behavior was a factor in choosing the peer group – suggesting that reciprocal causality may exist. However, in a subsequent study, Baumann and Ennett (1996) suggested that there was greater support for what they called the "peer influence axiom".

Another possibility is that this relationship may be spurious with some other factor influencing both choice of friends and smoking initiation. Choice of friends and smoking initiation may serve the single purpose of demonstrating rebelliousness from parent control. Simons-Morton, Chen, Abroms and Haynie (2004) found that both smoking behavior and choice of friends are influenced by parental factors. The current study will try to address this issue by using control variables (such as parental smoking behavior) whenever possible in its analyses of peer smoking, being mindful of the possibility of spurious causality.

The correlation between peer smoking and smoking initiation is well established. While being aware of the possibility of reciprocal causality or the potentiality of a spurious relationship, it appears that there is extensive evidence providing substantial

support for the importance of peer influence as a factor in smoking initiation. Additional studies further clarifying the character of that relationship would enhance our overall understanding of this correlation and may provide opportunities for theoretical and practical approaches to lower adolescent smoking rates in the future. The conceptual model would suggest that the influence of peers will increase throughout adolescence. The current evidence for and against this assumption will be discussed in the section reviewing the literature on studies looking at interactions.

Parental and Family Influence and Smoking Initiation

Family smoking behavior, especially that of parents, has also been strongly associated with adolescent smoking initiation. In a review of the existing evidence: the Surgeon General's report of 1994 (Elders, Perry, Eriksen, and Giovino 1994) regarding smoking initiation suggested that a preponderance of evidence supported an association between parental smoking and smoking initiation; a systematic review by Tyas and Pederson (1998) was in agreement with this conclusion; and recent studies continue to support the relationship between parental smoking (Fleming, Kim, Harachi, and Catalano 2002; Miller and Volk 2002) and adolescent smoking initiation.

Though the evidence for the importance of parental smoking behavior is extensive, the potential theoretical explanations for the relationship between parental smoking and adolescent smoking initiation are even more diverse than those suggested for peers. Family smoking could exert its influence through modeling, verbal persuasion, greater access to cigarettes, or levels of parental control of adolescent behavior; and it could also reflect the fact that parents and children share genetic factors. Combinations of

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these factors could also be at work. For example, Fleming, Kim, Harachi and Catalano (2002)found support for a complex relationship whereby parental bonding provides a protective factor, and parental smoking a risk factor, for adolescent smoking initiation.

In this complex relationship, it is plausible that the proposed moderating factors exert an influence through their impact on perceptual or interpretative processes. Existence of a moderated relationship would support the idea that perceptual or interpretative processes could affect the likelihood that parental smoking behavior will have an influence on the smoking initiation process of an adolescent. The conceptual model provided and some theories of adolescent development suggest that the influence of family would be greater in early rather than late adolescence, and some evidence supports this contention (Krosnick and Judd 1982). This topic will be further addressed in the discussions of the proposed moderating variables.

Like the theorized increase in peer influence as an adolescent ages, the theorized waning of parental influence is controversial. The theories which serve as a foundation for this study suggest that parental influence will decrease in later adolescence, though some empirical evidence has shown a stable influence of parents throughout adolescence(Bauman, Carver, and Gleiter 2001; Beyers and Goossens 2008). This controversy will be further addressed in the section on interactions. Additional exploration of these issues may help in our understanding of the relative influences throughout adolescence.

Moderating Variables

The variables that are addressed in the following discussions – age, depression, and school performance – are of interest in the current study as factors which may moderate the effects of peer or parental smoking behavior. The foundational theories to the model guiding this study suggest that these factors should alter the impact of peer or parental smoking by changing the perception or interpretation of the smoking behavior of parents or peers. Each of these variables could also be thought of as proxies for broader concepts. Age can be conceived as a reflection of adolescent development. Depression is an example of an emotional state which should alter the adolescent's perception. School performance may be indicative of an adolescent's intellectual abilities and, by extension, the ability to interpret behavior. These are, of course, imprecise measurements of these concepts, but if evidence is found for interactions, then additional studies which use better measures could be designed. This section will look at the main effects of these variables, and the next section will then address studies which have specifically looked for an interaction of these variables with peer or parental smoking.

Age: The Significance of Adolescent Development in Smoking Research

Smoking initiation is primarily an issue of adolescence, with most studies finding that the average age of smoking initiation is approximately 13 years old, examples being studies which have found it to be 12.3 years old (Harrell, Bangdiwala, Deng, Webb, and Bradley 1998), and 13.3 years old (Siqueira, Diab, Bodian, and Rolnitzky 2000). It is also documented that 89% of smokers begin smoking before the age of 18 (Nelson, Giovino, Shopland, Mowery, Mills, and Eriksen 1995).

"Adolescence" is a relatively young concept. Most dictionary definitions simply define adolescence as the period between puberty and maturity. Since maturity is the state of full development into adulthood, this means that adolescence is merely that period between childhood and adulthood. This transition is defined uniquely by each society, thus, adolescence may be thought of as "socially constructed" (Larson 2002). Larson suggests that it is a "Western invention of the late 19th and early 20th century" (p.1). Historically, adolescence was initially described by Hall (1916) as a period of "sturm und drang" (storm and stress), and this interpretation influenced our concepts of adolescence for many years. By looking at other cultures, later researchers, including Margaret Mead, suggested that Hall's concept of adolescence might be specific to Western societies. Mead (1950) suggested that the process of adolescent development was not as "stormy" in other cultures and therefore was not, by nature, a period filled with great conflict.

Erik Erikson (1950) described adolescence as a stage in which the individual is wrestling with issues of identity development. He identified its central developmental task as the resolution of a conflict between "role diffusion and identity confusion." Essentially all theories of adolescence address the element of identity development associated with this stage. The development of 'identity' in adolescence is influenced by many factors including parents, friends, and social institutions such as schools and the media. Several studies have contended that the relative influence of these factors varies as adolescence progresses, with early adolescents being primarily influenced by a combination of family and peers, and later adolescents by peers and other social conditions as they gradually become more independent (Irwin 1986; Rice 2002). These

changes are accompanied by other transitions, including moving into higher level school settings where adolescents are expected to demonstrate progressively greater decision-making skills.

One's behaviors help to define one's identity, and smoking is an example of a behavior that may be influenced by this developmental process. Although smoking initiation most often occurs in adolescence, the relevance of developmental processes is still unclear. Some adolescents begin to regularly smoke cigarettes, others do not smoke at all, and another group may continually smoke at very low levels and may not even become daily smokers. This variability has provided additional challenges to researchers trying to develop explanations for smoking initiation.

Since, as noted, a number of models of adolescent development suggest that adolescent behaviors differ in early versus late adolescence, one would expect this to impact the probability of smoking initiation. In other words, as adolescents develop and move from the primary attachments of the family to developing friendships, the influence of parental smoking may be greater for early adolescent initiators, and the influence of peer smoking greater for late adolescent initiators. Thus, peer influence might be expected to have greater impact on older adolescents.

Empirical evidence for this assumption of a transition from parental to peer influence over the course of adolescence as related to smoking initiation has been mixed. Trying to provide a test for this assumption, Krosnick and Judd (1982) found support for an increase in peer influence over the course of adolescence. They also found that parental influence declined, although not at a significant level. Other studies have found evidence for a stable and consistent effect of parental influence throughout adolescence

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(Bauman, Carver, and Gleiter 2001; Beyers and Goossens 2008). Jang (2002) found that peer influence increased in early adolescence but then stabilized. Bricker and colleagues (Bricker, Peterson, Sarason, Andersen, and Rajan 2007) found that peer influence was greater at younger ages and parental at older. A greater understanding of the important influences at various stages of adolescent development could be useful in designing smoking prevention programs. This study will explore for possible interactions between age, as a proxy for adolescent development, and peer and parental smoking behavior.

Previous efforts to provide smoking prevention programs for adolescents have not been very effective. Several school-based prevention programs, including DARE (Drug Abuse Resistance Education) have been developed but have had very modest success. Ennett, Tobler, Ringwalt and Flewelling (1994) did a meta-analysis of eight DARE evaluation studies and found that the effect size ranged from 0.00 to 0.11. They concluded that DARE had a very small effect and that it was inferior when compared with programs that promoted social and general competencies. When evaluating a ten year follow up, Lynam, Milich, Zimmerman, Novak, Logan, Martin, Leukefeld and Clayton (1999) concluded that DARE had no demonstrable benefits.

Interventions that have taken a broader approach have had somewhat better outcomes. Several studies that included family and peer support approaches (Bauman, Ennett, Foshee, Pemberton, King, and Koch 2002; Cameron and Brown 1999; Skara and Sussman 2003) have had generally positive but still mixed effects. In a meta-analysis, Rooney and Murray (1996) estimated that the benefit of these kinds of programs was in the range of a 5% reduction in smoking.

The relative lack of success of these smoking prevention and cessation programs would suggest that programs that address these kinds of individual and social factors may not be effective. It is also possible, however, that programs which are built on a greater understanding of how these factors interact in the smoking initiation process may be effective in preventing smoking initiation.

Depression and Smoking

A topic of interest in the smoking literature is the complex relationship between depression and smoking. The majority of current evidence suggests that being depressed results in an increased probability of smoking initiation. The most common explanation provided for this association is that smoking may be a form of "self-medication" for depression. The exact nature of this suggested relationship, however, is not well understood and the existence of a *causal* relationship between depression and smoking is controversial.

Early research was cross sectional in nature and established a correlation between depression and smoking (Covey and Tam 1990; Patton, Hibbert, Rosier, Carlin, Caust, and Bowes 1996). Later longitudinal research also supported the idea that adolescents with depressive symptoms are more likely to become regular smokers (Escobedo, Reddy, and Giovino 1998; Patton et al. 1998). Recently, several studies (Goodman and Capitman 2000; Wu and Anthony 1999) have taken an alternative view suggesting that smoking may *precede* depression. Four tables are provided in Appendix II which include twenty-eight articles supporting the various causal pathways to smoking initiation. The existence

of evidence for causal directions, reciprocality, and spuriousness demonstrates that the nature of the relationships between these factors has not been adequately defined.

The tables in Appendix II provide a summary of the studies that support the various "causal pathways". A brief discussion of the key elements is also provided here. Tables 1 through 4 review studies that have explored this issue. Appendix II, Table 1 includes 13 studies that support depression as an antecedent to smoking initiation. Several of these studies base this assumption on correlation alone (Covey and Tam 1990; Lenz 2004). Others, however, have tried to address the limitations of cross sectional research by asking about intent to smoke (Carvajal, Hanson, Downing, Coyle, and Pederson 2004; Nezami, Unger, Tan, Mahaffey, Ritt-Olson, Sussman, Nguyen-Michel, Baezconde-Garbanati, Azen, and Johnson 2005). Some studies have asked for an adolescent's self report by which to assess previous behavior (Carvajal et al. 2004) or known correlates to depression such as physical and sexual abuse (Nichols and Harlow 2004) or suicidal thoughts (Tomori, Zalar, Plesnicar, Ziherl, and Stergar 2001). Four studies used longitudinal designs (Kandel and Davies 1986; Patton et al. 1998; Repetto, Caldwell, and Zimmerman 2005) with support found in Patton,et al. (1998) for depression as an antecedent to smoking initiation, and by Repetto,et al. (2005) and Kandel and Davies (1986) for increased risk of smoking initiation in those with a previous history of depression. Orlando, Ellickson and Jinnett (2001) found that tenth graders with depressive symptoms were more likely to be smokers by the twelfth grade. Using a multi-ethnic sample, Nezami, et al (2005) also found that depression was associated with the intention to smoke..

As indirect evidence of the depression-to-smoking pathway, Paperwalla, Levin, Weiner, and Saravay (2004) have reviewed the prevalence of smoking in people with psychiatric illnesses. The prevalence of smoking in the United States over the past several years has been 20-25% in the overall population but among people with a psychiatric illness it consistently exceeds 50% (Paperwalla, et al. 2004). It is even higher among people with certain specific conditions, with schizophrenia having the highest prevalence at 88%. People with depression smoke at a rate of approximately 49%. This is more than double the rate of non-depressed individuals. It is also interesting to note that mortality rates among people with depression are higher than those of non-depressed individuals, with major depression having roughly *double* that of the normal mortality rate (Penninx, Geerlings, Deeg, van Eijk, van Tilburg, and Beekman 1999). Though one would expect that suicide and other risk behaviors would be important in affecting these mortality rates, the mechanisms responsible for the higher death rate among those with depression have not been completely explained, and it makes sense that higher smoking rates may be responsible at least in part for this higher death rate. An increased understanding of this relationship might allow for smoking prevention interventions targeted specifically to those with depression.

The evidence connecting depression to smoking initiation is also buoyed by our increasing understanding of the neurotransmitters that are involved in depression. Smoking alters the levels of norepinephrine and serotonin (Paperwalla, Levin, Weiner, and Saravay 2004), chemicals that are known to be key in the physiological status of depression. Thus, adolescents may find relief from depressive symptoms by smoking

cigarettes. As such, "self-medication" for depression may be a factor in the smoking process.

Recently a number of researchers have focused on the possibility that the causal relationship runs in the opposite direction with smoking resulting in higher levels of depression (Appendix II- Table 2). Wu and Anthony (1999), in a sample of 2000 adolescents, found that smokers were more likely to develop depression (OR 1.66, 95% CI 1.28-2.16). A similar relationship was found in 8704 adolescents in the National Longitudinal Study of Adolescent Health (Goodman and Capitman 2000) after controlling for a number of variables and using "smoking one pack per week" as the dependent variable. Other studies have also found support for the contention that smoking precedes depression (Brook, Schuster, and Zhang 2004; Stein, Newcomb, and Bentler 1996; Steuber and Danner 2006). They are still few in numbers, however, and further explanations must be provided explaining the existence of temporal ordering supporting a depression-to-smoking pathway before this alternative pathway could be accepted as representing a primary process.

There is also the possibility of a reciprocal relationship between smoking and depression. Depression may result in an increased probability of smoking, *and* smoking may result in an increase in the prevalence of depression. Wang and Fitzhugh (1996) used a cross lagged analysis that supported reciprocal causality. A number of other studies have suggested this possibility although most of these seem to simply represent correlational studies that have been cautious not to make strong causal assumptions (Appendix II- Table 3).

Other studies have suggested that depression and smoking may be caused by some other variable or variables (Appendix II- Table 4). Examples of other possible variables include rebelliousness (Albers and Biener 2002; Koval and Pederson 1999; Koval, Pederson, and Chan 2004) and family characteristics (Jarvelaid 2004). These studies may also be examples of researchers taking a cautious approach to the causality issue with recognition that depression and smoking have a number of correlates in common (Breslau, Peterson, Schultz, Chilcoat, and Andreski 1998). Other studies have also seen common correlates and found stress as an antecedent to both depression and substance abuse in general (Turner 2003).

Though it is important to recognize that there is evidence for a number of different causal relationships in smoking initiation, there is substantial evidence for a direct effect of depression on the probability of smoking initiation. The presence of a relationship in numerous longitudinal studies provides a strong case for depression as antecedent to smoking initiation. Using this sample (The TAPS) Escobedo, Reddy, and Giovino (1998) have already found support for depression as an antecedent to smoking initiation, although Escobedo, et al, used a definition of "smoking initiation" which included all adolescents who had smoked on 5 or more days in the last month. Further exploration and testing of this relationship using a better measure of smoking initiation while including exploration of interaction effects could help provide a greater understanding of the overall phenomenon. The current study will look at the main effects of depression but will also look for evidence that depression may alter the influence of peers' and/or parents' smoking behaviors. Choice of depression as a variable is consistent with the conceptual model, since depression has been shown to influence decision

making; and in fact having problems with decision making is actually part of the diagnosis of depression (http://www.nimh.nih.gov/publicat/depression.cfm#ptdep3).

School Performance and Smoking

A number of early studies established a connection between school performance and smoking initiation (Young and Rogers 1986). Since that time, however, little clarity has been achieved regarding the nature of that relationship other than the observation that "most researchers agree that there is an inverse relationship between adolescent substance use *(including smoking)* and high academic grades" (Cox, Zhang, Johnson, and Bender 2007).

Several researchers have pointed to the issue of causal ordering with regard to the relationship between school performance and smoking, asking the questions: Does poor academic performance precede smoking initiation? Or, does smoking initiation precede poor academic performance? Is it a spurious association perhaps due to their association with a third factor? Some cross sectional studies have been interpreted to imply that substance abuse may impair academic performance. Supporting this contention are studies that have found that substance abusers place little value on academic performance (Beman 1995) and demonstrate impaired cognitive abilities related to the use of various substances (Johnson and Kaplan 1990). Other researchers have used models which suggest that clusters of behaviors such as substance abuse and poor academic performance are all caused by various underlying social factors (Conwell, O'Callaghan, Andersen, Bor, Najman, and Williams 2003; Thomas 2002; Wang 2001; Zhu, Liu, Shelton, Liu, and Giovino 1996). Only one longitudinal study which evaluated this

question could be found, and this study supported poor academic performance as antecedent to smoking initiation (Cox, Zhang, Johnson, and Bender 2007). This study looked at a longitudinal sample of Mississippi adolescents. No study could be found which explored for this relationship in a national longitudinal sample, thus, the current study will provide a contribution to this discussion.

If, in fact, impaired academic performance does precede substance abuse including smoking, there are at least four processes by which this could occur: 1. adolescents who are poor school performers may lack the ability to fully understand the risks associated with smoking, resulting in a poor health behavior choice, 2. adolescents might use smoking to improve cognitive processing (through processes described in this section), 3. they might use smoking to "self medicate" to compensate for such deficits as low self-esteem associated with their poor school performance, or 4. they may use smoking as a way to reframe self identity as rebellious and thus devalue academic performance.

It is difficult to believe that anyone could be unfamiliar with the risks of smoking by this point in history, but it is possible that lower academic performers do not have a really meaningful understanding of the risks of smoking. Higher academic performers may be able to understand the risks in a more complete way and thus make better judgments about health behaviors than lower school performers. Conversely, if understanding of risks is less complete, the influence of the behavior of others such as parents or peers might be more likely to result in smoking initiation. If this is true, educational approaches which recognize this issue may be more effective in smoking prevention.

Though I have suggested that school performance may be a proxy for intellectual abilities, we should also consider other ways in which school performance may be connected to smoking initiation. Some adolescents may try smoking and feel that it improves their thinking processes. It is perhaps not unexpected that the "benefits" of nicotine on mental processing have not been emphasized in the lay or professional literature. There is evidence, however, of several positive cognitive effects of nicotine, with research finding that nicotine improves cognitive processing speed and attention abilities, and, therefore, may have the potential of improving academic performance (Poltavski and Petros 2005; Poltavski and Petros 2006). It is important to note, however, that, for smoking to have this effect, the adolescent would have to have smoked very near to the time of academic performance. Thus, benefits would be much greater for homework rather than for performance in class or for examinations. It is possible that some students have discovered these benefits, consciously or unconsciously. There is no direct evidence of this, although no study could be found that specifically evaluated for this possibility. Indirect evidence may be found for smoking as self medication to improve cognitive function in the high prevalence of smoking among adolescents with Attention Deficit Disorder (ADD). Adolescents with ADD may find that smoking improves their symptoms resulting in a conscious or unconscious self-medication process. Lerman, Audrain, Tercyak, Hawk, Bush, Crystal-Mansour, Rose, Niaura, and Epstein (2001) found support for a "positive" effect of smoking in adolescents with ADD, in other words, smoking improved their attention and performance. Though there has been speculation that the higher rates of smoking among those with ADD are due to side effects from medication treatments, Whalen, Jamner, Henker, Gehricke, and King

(2003) found greater support for smoking as self-medication for ADD. These studies raise the interesting possibility that some of the reason for smoking, at least in this population, is short term cognitive benefits (or perceived benefits).

Smoking may also be an attempt to deal with low self-esteem and depression that may result from inadequate academic performance. As noted in the previous discussion, smoking alters the level of certain key neurotransmitters. Adolescents may learn this "coping mechanism" from one another, and may then find that their depressive symptoms are improved at least in the short term.

Since adolescence is a time of significantly increasing depression rates, this will also need to be considered in the analyses. Since adolescence is defined in part by puberty, the physiological and social elements of this transition would suggest that depression rates would rise. This is supported by statistics provided by the National Institute of Mental Health (www.nimh.nih.gov 2007), which show that mental health disorders are estimated to affect 5% of children but, by adulthood, may affect as many as 26% of adults, with depression accounting for over a third. I will include the measure of depression as a control variable in all analyses not specifically addressing its main effect in an effort to address this issue.

Smoking could also be a mechanism by which adolescents re-frame their self perceptions with regard to their school performance. Several studies have found support for smoking as a sign of rebelliousness (Albers and Biener 2002; Choi, Harris, Okuyemi, and Ahluwalia 2003; Koval and Pederson 1999). By redefining their values, they no longer need to perform well academically to be consistent with their own self-image. This reframing might occur either as a result of low school performance or might be due to

other factors and subsequently result in a lower school performance. Either way, to support this re-framing of self perceptions, the adolescent might initiate smoking in an effort to appear nonconformist to usual standards.

Thus, the relationship between smoking and low academic performance may be a result of several different processes. While recognizing these different possibilities, it is useful to consider how intellectual ability might be important to the ability to assess the risk of smoking and make good health behavior decisions. Some suggest that the way to address the association between poor school performance and smoking initiation is to develop remedial educational approaches to teaching about the risks of smoking (Hu, Lin, and Keeler 1998; Thomas 2002). Greater understanding of the possible direct and interacting effects involving school performance is probably necessary before these kinds of programs could be developed. Others have suggested (Thomas 2002) that efforts to improve academic performance in general may provide protection against smoking initiation. If, in fact, intellectual ability is important in this process, then defining the mechanisms by which this occurs should enhance our ability to design effective smoking prevention programs and will also have implications for educational policies and practices.

Control Variables

Many factors have been found to be associated with an increased likelihood of smoking. Excellent reviews of the evidence related to these factors have been previously published (Elders, Perry, Eriksen, and Giovino 1994; Tyas and Pederson 1998). Other subsequent studies have also provided continued support for factors including gender

(Pampel 2001), race (Flint, Yamada, and Novotny 1998), and socioeconomic status

(Escobedo and Peddicord 1996; Pampel and Rogers 2004). The process or processes that

underlie the associations between these variables and the initiation of smoking are not

completely clear. It is clear, however, that any study that explores the relationships

between variables such as peer and parental smoking, and depression and age, needs to

include factors such as gender, race and socioeconomic status in any causal model.

The effect of factors such as race and gender can be better understood using

historical research methods. Looking at the trends of smoking initiation associated with

race and gender demonstrates that the relationship between smoking and these social

variables has changed significantly over time. Though the reasons for these changes are

not completely clear, the fact that the relationships have changed over time provides

support for the importance of social factors in the smoking initiation process. The period

between 1960 and 1990 was a time in which many things changed regarding the social

status of females and ethnic minorities and, though we cannot be sure that these changes

are responsible for the concurrent changes in smoking behavior, it is a logical conclusion.

In general, gender, race, and family income have clear associations with smoking

behaviors and, thus, it is necessary to include these as control variables in the model.

Gender

The association between gender and smoking behavior is well documented, with

males being more likely to smoke throughout history (Table 1). There are clearly

differences between male and female smoking patterns and, since these differences have

varied with sociohistorical trends, they are most likely related to the differing social

experiences of males and females. Rates for females have increased significantly since the 1960s (Anderson and Burns 2000) with at least one researcher finding evidence that the trend is related to an increase in gender equality of females (Pampel 2001). Over the same period, male smoking rates have declined.

Due to these differences, statistical analyses must take gender into account. For this study, I will control for gender in analyses that explore for the interactions of interest.

Year	Percent of Males age 18-24 who are smokers	Percent of Females age 18-24 who are smokers
1965	54.1	38.1
1974	42.1	34.1
1979	35	33.8
1983	32.9	35.5
1985	28	30.4
1990	26.6	22.5
1992	28	24.9
1993	28.8	22.9
1994	29.8	25.2
1995	27.8	21.8
1997	31.7	25.7
1998	31.3	24.5

Table 1: Smoking Rates by Gender: 1965-1998.

Race

African Americans' smoking rates differ from those of white Americans, though these differences have varied significantly over recent history (Table 2). Prior to 1984, African Americans consistently smoked at rates higher than whites. Since that time, however, their rates have lowered. The reasons for this are unclear, though most explanations for this change include the recognition of changing social factors for African Americans during this time period.

Weinrich, Hardin, Valois, and Gleaton (1996) found that white students are more likely to engage in stress-related smoking than African American students. The differences in smoking rates do not appear to be related to different experiences of experimenting with smoking. Two studies found African American teens were more likely to try smoking but less likely to progress to becoming regular smokers (Ellickson, Orlando, Tucker, and Klein 2004; Flint, Yamada, and Novotny 1998). Flint, et al., found that only 10.3% of African American experimenters went on to become regular smokers as compared with 25.7% of white experimenters.

Another difference between African American and white adolescents is that the influence of peers may be different. Unger, Rohrbach, Cruz, Baezconde-Garbanati, Howard, Palmer, and Johnson (2001) found that peer influence on smoking was greater among white adolescents than among African American adolescents. Gritz, Prokhorov, Hudmon, Jones, Rosenblum, Chang, Chamberlain, Taylor, Johnston and de Moor (2003) had similar findings with regard to peer influence, and found that African American adolescents were also less influenced by parental smoking.

Year	White Males age 18-24	Black Males age 18-24	White Females age 18- 24	Black Females age 18-24
1965	53	62.8	38.4	37.1
1974	40.8	54.9	34	35.6
1979	34.3	40.2	34.5	31.8
1983	32.5	34.2	36.5	32
1985	28.4	27.2	31.8	23.7
1990	27.4	21.3	25.4	10
1992	30	16.2	28.5	10.3
1993	30.4	19.9	26.8	8.2
1994	31.8	18.7	28.5	11.8
1995	28.4	14.6	24.9	8.8
1997	34	23.5	29.4	11.5
1998	34.1	19.7	28	8.3

Table 2: Smoking Rates by Gender and Race: 1965-1998.

African Americans have lower rates of depression (and psychiatric disorders in general) than Caucasians (Kessler and Zhao 1999). Guthrie, Young, Williams, Boyd and Kintner (2002) found that the effect of race on smoking initiation was dependent on the stress level of the individual. In support of this, Ganz (2000) found that the smoking rates among African Americans in Harlem were mediated by the level of exposure to violence.

Clearly, race has an impact on the probability of smoking initiation. It is not clear however, what mechanisms are involved in this process. The analyses for this study will use race as a control variable though later exploration of the differential experience of African Americans regarding parental smoking, peer smoking, and depression could be an important area for study.

Socioeconomic Status

Social class in general has also been shown to have an inverse relationship with smoking rates (Isohanni, Moilanen, and Rantakallio 1991; Millar and Hunter 1990; Stanton, Oei, and Silva 1994; Zhu et al. 1996). Factors linked to social class such as financial distress (Siahpush, Borland, and Scollo 2003) have also been associated with smoking rates. This association holds only to a certain level and is thus limited by cigarette prices that are "accessible" since higher cigarette prices have been found to lower smoking rates (Farrelly, Nimsch, Hyland, and Cummings 2004; Lee and Cubbin 2002), and, in some cases, smoking rates have been found to be higher among the more wealthy (Ennett, Flewelling, Lindrooth, and Norton 1997). Socioeconomic status (SES) may exert an effect in various ways. As previously noted, stress, which is usually

associated with lower SES, may result in higher rates of substance abuse including smoking. Conversely, higher SES may provide greater resources which allow for the acquisition of cigarettes.

Another factor related with SES is low education, which has been found to be a correlate of smoking (Sheahan and Latimer 1995; Zhu, Giovino, Mowery, and Eriksen 1996). People of lower socioeconomic status may lack knowledge regarding the dangers of smoking. With many years of health-focused counter marketing, it seems improbable at this point, but low SES individuals' understanding of the full implications of this information may still be less than those who are more highly educated. The issue of socioeconomic status will be handled in this study by controlling for family income, although, as will be discussed in the methods section, the measure in this study is of a lower quality than we would like, since there is not adequate measurement of the upper ranges of income.

Interaction Effects and Smoking Initiation

The major objective of this study is to help determine whether combinations of social factors result in an increased risk of smoking initiation. Smoking rates, which were in decline from the 1960s through the 1990s have since stabilized and may even be increasing among adolescents. New approaches must be developed if we are to see a reduction in adolescents who take up the smoking habit. This section reviews the evidence for combinations of social factors that may increase the risk of smoking initiation and discusses how understanding the nature of these relationships may assist in the development of smoking prevention strategies.

A few studies have found evidence for the interaction of the variables of interest (peer smoking and parental smoking), though these studies are small in number. An EBSCO host search for studies looking at interaction effects involving the variables of interest (key words: smoking initiation, peer smoking or parental smoking, and interaction or moderating) was carried out using the following databases: Academic Search Premier, Sociological Abstracts, Medline, ERIC, PsycArticles, and Health Source Professional. Resulting abstracts did not include any studies which tested for interactions of the variables of interest in this study. The search did reveal interest in looking at other moderated relationships including interactions between depression and tobacco advertising (Tercyak, Goldman, Smith, and Audrain 2002), biological factors (such as testosterone and estrogen levels), and social factors (Bauman, Foshee, and Haley 1992; Foshee, Ennett, Bauman, Granger, Benefield, Suchindran, Hussong, Karriker-Jaffe, and DuRant 2007). However, a small number of articles was found which address interactions of the variables of interest.

To review the findings of these studies, I will use the framework of the theoretical model for this study, which suggests exploration of the proposed predictor variables (peer smoking and parental smoking) and the three moderator variables (age, depression, and school performance) to structure the discussion of the proposed hypotheses, the potential implications of these relationships, and the evidence which currently exists for these interaction effects. These variables are of the greatest interest as they are seen as reflecting characteristics that may be useful in the development of smoking prevention interventions. If it is found that adolescent development (reflected in this study by age), depression, or school performance result in an increased risk of smoking initiation

through an increased vulnerability to peer or parental smoking behavior, then educators

or health professionals may be able to take steps to protect adolescents from this

increased risk.

The proposed predictor and moderator variables result in six combinations that

are expressed by the following hypotheses:

Hypothesis 1:
The effect of parental smoking on smoking initiation will be greater for younger versus older adolescents

Hypothesis 2:
The effect of parental smoking on smoking initiation will be greater in the presence of higher levels of depression

Hypothesis 3:
The effect of parental smoking on smoking initiation will be greater for those with lower levels of school performance

Hypothesis 4:
The effect of peer smoking on smoking initiation will be greater for older versus younger adolescents

Hypothesis 5:
The effect of peer smoking on smoking initiation will be greater in the presence of higher levels of depression

Hypothesis 6:
The effect of peer smoking on smoking initiation will be greater for those with lower levels of school performance

Considering the interaction of variables is complex, conceptually and statistically.

The use of this approach for research on smoking initiation has been limited, though

some studies have published analyses which have explored for conditional relationships

between variables. These include studies that have specifically explored for interactions,

but also include separate analysis by different groups. Analysis by separate groups would

infer – without providing a statistical test for – the existence of conditional relationships.

Jaccard (2001) contends that a product term analysis is superior to this type of analysis

although both types of studies will be included in this discussion.

Hypotheses of Proposed Parental Smoking Interactions

Hypothesis 1:
The effect of parental smoking on smoking initiation will be greater for younger versus
older adolescents

Hypothesis 2:
The effect of parental smoking on smoking initiation will be greater in the presence of
higher levels of depression

Hypothesis 3:
The effect of parental smoking on smoking initiation will be greater for those with lower
levels of school performance

No studies have reported evidence of an interaction between parental smoking

and age, depression, or school performance. This is contrary to what we might expect

based on the conceptual model of this study, as well as a number of the models that have

been used in smoking research and prevention models. There is some empirical

evidence, as noted in the discussion of the main effect of parental smoking, that would

suggest that the influence of parents is more stable throughout adolescence than these

models would suggest (Bauman, Carver, and Gleiter 2001; Beyers and Goossens 2008).

Many of the theoretical models that have been used to study smoking would

suggest that during early adolescence the behavior of parents would have a greater impact

than those behaviors would have in later adolescence. The conceptual model of this study

would also suggest that being depressed would make an adolescent more vulnerable to

the influence of parental smoking. This same logic would indicate that poor school

performance would make an adolescent more vulnerable to the influence of parental

smoking. Lack of evidence for these interactions might suggest that these models are incorrect. There might also be other alternative explanations however such as the possibility that the influence of parental smoking may occur in the preadolescent stage.

It is important to remember that, when we are talking about combinations of factors in this study, we are talking about how the combination would result in rates different from those expected by an accumulation of the rates of individual factors. Several studies have suggested an accumulation effect of factors such as parental smoking and depression (or related concepts such as self esteem). Wilkinson and Abraham (2004) found that including multiple factors, in this case, self esteem, parental smoking, sibling smoking, and peer smoking in a path analysis resulted in an R^2 of 0.56 when trying to predict smoking status six months after initial measurement. Models that look at additive effects may also have potential in the development of smoking prevention programs, but that is a different question than is being addressed in the current study.

The lack of previously published studies might indicate that no one has looked at these relationships, or it may reflect the well-known bias against publishing negative findings. In other words, these relationships have been tested for, but no evidence has been found for their existence. Even negative evidence which suggests a lack of importance for these combinations may be helpful, as it can help us avoid prevention efforts which are based on assumptions rather than solid evidence.

Hypotheses of Proposed Peer Smoking Interactions

Hypothesis 4:
The effect of peer smoking on smoking initiation will be greater for older versus younger adolescents

The theoretical models on which the conceptual framework of this study is based suggest that the influence of peers will increase throughout the adolescent period, although, as has been described, the empirical evidence for this contention is mixed. As noted, there is some support for increasing risk of smoking in older adolescents (Jang 2002; Krosnick and Judd 1982) as well as empirical evidence that the influence of peers on a number of risk behaviors such as alcohol and drug use is greater for older rather than younger adolescents (Stoff 1997). Despite these findings, no study could be found which specifically tested for a conditional relationship between age and peer smoking behavior using a national, longitudinal sample.

Several models of the adolescent developmental process would suggest that, as the adolescent ages, the importance of peers increases, and that family influences decrease. This would suggest that peer smoking behavior should interact with age. Smoking prevention interventions that were based on the premise that peer influence was greater at certain ages would target those age groups regarding peer choice or counteracting peer influence. Lack of a relationship between peer smoking behavior and age would suggest that prevention efforts could be similar at various ages. If we are going to shift back to an emphasis on social factors in prevention, as public health professionals we will need to determine if risk factors change by age (and if so, how) to be able to tailor prevention efforts to various age groups.

Hypothesis 5:
The effect of peer smoking on smoking initiation will be greater in the presence of higher levels of depression

The conceptual model of this study suggests that being depressed will make the adolescent more vulnerable to the influence of peers who are smokers. Two studies have found evidence for interaction effects between peer smoking and depression on smoking initiation. Patton, et al (1998) found that, in a sample of 2032 Australian teenagers, the presence of depressive symptoms in adolescents increased the probability of smoking initiation – but only in those adolescents who had smoking peers. In the 12-17 year old age group, the hazard ratio for daily smoking in adolescents with a high depression score and who reported the most friends smoking was reported as 2.6 (95% CI 1.3-5.6). Ritt-Olsen, et al. (2005) also found that peer influence interacted with depression but only for females. Other studies including Tercyk, Goldman, Smith, and Audrain (2002) tested for an interaction between peer smoking and depression but did not find one. The sample used in Tercyk, et al, however included only high school freshman, so it did not address whether a relationship might exist in other age groups.

There has been a recent focus on cigarette smoking as being one of a number of concerning risk behaviors which may occur in adolescence. Escobedo, Reddy and Durant (1997) found that other behaviors such as use of smokeless tobacco, having multiple sexual partners, not using bicycle helmets, carrying weapons, marijuana use, binge drinking, and fighting are correlates of cigarette smoking. Interaction effects have been found in research on many of these behaviors including an interaction between depression and peer behavior impacting the use of alcohol and other substances

(Prinstein, Boergers, and Spirito 2001). Similar relationships might be expected to have an influence on smoking.

Though results have been inconsistent, two studies have found evidence of an increased vulnerability to smoking associated with depression. The findings of Patton, et al. (1998) and Ritt-Olsen, et al. (2005) are consistent with the findings predicted by the conceptual model. Thus we might expect that the combination of these factors could have similar findings in this sample.

Hypothesis 6:
The effect of peer smoking on smoking initiation will be greater for those with lower levels of school performance

No studies could be found which specifically tested for the influence of an interaction of school performance or scholastic competence and peer smoking behavior on smoking initiation.

If the combination of peer smoking and school performance increases the risk of smoking initiation, it may have practical implications for the decisions made by educators. One area of controversy in education today regards the use of "tracking" or placing students in groups based on academic ability. A number of educators and researchers have expressed concerns regarding the use of this approach (Dornbusch, Glasgow, and Lin 1996; Kozol 1992; Oakes 1985). If the influence of peers on smoking initiation is conditional on the level of school performance, putting poor school performers together with a peer group who are smokers could result in an unintended increase in the risk of smoking initiation. Thus, greater understanding of the nature of the

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relationship between peer smoking, school performance, and smoking initiation may have implications for educational policies and practices.

Some researchers have explored for conditional relationships between other variables thought to be important in smoking initiation. Tercyak,et al (2002) found evidence for an interaction between depression and cigarette advertising on smoking initiation. Trinidad, Unger, Chou and Johnson (2005) found that level of acculturation had a moderating influence on the relationship between emotional intelligence and smoking initiation. Bauman, Foshee, and Haley (1992) have found evidence of interactions between physiologic factors and social factors. Their most recent study looked at male and female hormone levels in adolescents and their relationship to social factors regarding the likelihood of smoking initiation (Foshee et al. 2007). Though these studies do not involve the variables of interest in the conceptual model of the current study, they do support the contention that combinations of variables may have utility in predicting smoking initiation.

It is clear that much evidence connects social factors and smoking. As noted, reviews were published in the 1990s, including the Surgeon General's report of 1994 (Elders, Perry, Eriksen, and Giovino 1994) and Tyas and Pederson (1998), both of whom provide excellent reviews of the extant knowledge at that time. However, little progress has been made since the late 1990s in exploring and explaining the complex relationships among the variables that they identify. Research that provides connections between currently known factors, and then suggests ways to apply this knowledge in educational

and clinical settings, could be very useful. Before we can talk about applying this knowledge, however, a better understanding of the kinds of "risks" associated with age, race, gender, social class, and peer and family behaviors in smoking initiation is necessary. Exploration of whether some of these factors may work synergistically may provide a better overall understanding of the smoking initiation process.

This study will look at the main effects of the key variables to see if relationships similar to those found in the existing literature exist in this sample. Special attention will be given to the issues of causal sequencing between depression and smoking initiation, and the relationship between school performance and smoking initiation. The data will then be examined for the existence of moderating effects consistent with the conceptual model. Exploration for conditional effects may also help to explain inconsistencies in the literature and provide insights concerning the process of smoking initiation.

Cigarette smoking is a major health problem. Despite knowledge of the dangers of smoking, fifty to sixty per cent of adolescents still try smoking. Of that number, about one half (CDC 2004) progress to become regular smokers. There will be great benefit if we can understand why a large number of adolescents still become regular smokers despite its well-known health risks. Certain factors, or combinations of factors, may make adolescents more vulnerable to smoking initiation.

If moderating factors, such as age, depression, or school performance alter the perception or interpretation of peer or parental smoking, this could put adolescents at higher risk of being influenced to smoke. An understanding of these processes may then

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be important in the development of effective smoking prevention programs. Understanding the factors that contribute to individuals initiating smoking, despite its known health risks, is essential before a more effective approach to smoking prevention can be achieved. In addition, exploration of these relationships in early versus late adolescence can help determine whether different processes may be at work at different stages of adolescent development. An understanding of these processes would be useful for researchers, health care professionals, and educators. Understanding how these factors interact could provide potential for the development of much more effective smoking prevention programs than currently exist, and may also have theoretical implications for the approach to other health risk behaviors.

CHAPTER III

METHODOLOGY

Sample- The Teenage Attitudes and Practices Survey (TAPS)

The study uses longitudinal data on a subset of a sample of 7,960 adolescents who

took part in the United States National Center for Health Statistics Teenage Attitudes and

Practices Surveys (TAPS) carried out in 1989 (United States Department of Health and

Human Services 1989) and 1993 (United States Department of Health and Human

Services 1993). The TAPS was a supplemental component of the National Health

Interview Survey (NHIS) conducted during those years. The NHIS is administered

annually by the National Center for Health Statistics to provide information about the

health status and behaviors of non-institutionalized Americans. This subset consists of

2,966 adolescents who were nonsmokers at the time 1 of the TAPS (1989) and looks at

how their characteristics at time 1 relate to their smoking status at time 2 (1993).

This archival data is especially well-suited for the current study's purpose since it

is a large stratified random sample of adolescents, and includes data from two different

time periods, four years apart, at a time when smoking rates had essentially stabilized

nationally and were relatively stable among adolescents.

In 1989, a sample of 12,097 non-institutionalized 12-18 year olds were chosen to

take part in the TAPS supplement to the NHIS using stratified multistage probability area

sampling. The data were collected over the last two quarters of 1988 and the first two

quarters of 1989. Computer Assisted Telephone Interviewing (CATI) was used to obtain

the data. In addition, teens from non-telephone households and those who could not be reached by the end of CATI interviewing were sent a mail questionnaire containing a portion of the CATI questions. Items included questions about smoking prevalence and those items felt to be the most important predictors of smoking uptake.

In 1993, a follow up to the first TAPS was completed by surveying 9,135 people from the initial TAPS sample who were then between the ages of 15 and 22. Of the 9,135 people chosen from the initial sample 7,960 responded to the survey (87%). Siddiqui, Flay and Hu (1996) have suggested that smokers may have been more likely to drop out of the TAPS since a number of the social variables associated with smoking might also promote subject loss. This will need to be considered in the interpretation of the results of this study. Phone questionnaires are commonly used to collect this type of data. Supporting the accuracy of this type of data collection, Caraballo, Giovino and Pechacek (2004) found that self-report and serum cotinine levels (a chemical marker of cigarette smoking) in a sample of adolescents ages 12-17 (n=2,107) varied by only about 2.7% suggesting a good level of accuracy for self report techniques in this population.

The final sample for analysis used a subsample of the 7,960 original subjects. Since the issue of interest was smoking initiation, the 4,384 of the original sample who were nonsmokers were eligible. Of this number 2,489 were still nonsmokers at time 2 and 477 were classified as regular smokers resulting in a final sample of 2,966. The remaining 1,418 were in various stages of experimentation.

This dataset has already provided much information regarding smoking patterns in adolescents. Analyses of the TAPS data along with numerous other studies, have provided support, that social influences are predictive of smoking initiation. In the TAPS

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data, several relationships have been explored, such as the cross sectional correlation of peer group smoking behavior with individual smoking behavior (Wang and Eddy 2000), the association of risk behaviors with smoking initiation (Wang 2001), and the influence of several sociodemographic risk factors (such as age, gender, and ethnicity) as being associated with smoking initiation (Wang 1998).

In an earlier study utilizing the TAPS, Escobedo, Reddy and Giovino (1998), based on a process previously described by Kandel and Davies (1986), created a depression scale using six items. Escobedo, et al then used a cut-off value to define whether adolescents were depressed. They found that a considerable proportion of adolescents in this sample met the criteria for depression, consisting of roughly 15% of the males and 20% of the females. Data on depression variables were not included in the mail surveys, resulting in a lack of data on 44 subjects. In this study, and in the survey's coding guide, variables not included on the mail surveys are indicated by an asterisk.

Variables/Instrument

One of the great challenges in doing research on smoking initiation is defining when a person has really "initiated" smoking. Is it when they smoke the first cigarette, when they have smoked 100 cigarettes, or when they smoke greater than a certain number of cigarettes on a daily basis? This study will use a recoded variable created by NCHS which classifies smoking status. This recoded variable (see Appendix III) uses multiple other items in the TAPS to classify smoking status. These include the adolescent's previous smoking behavior, current smoking behavior, and experience with experimentation. The dependent variable for this study collapses these categories into two

categories consistent with the conceptual model of this study, in which the issue of interest is smoking initiation. To look at smoking initiation, those identified as "never smokers" at time 1 (TAPS I - 1989), and still identifying themselves as "never smokers" at time 2 (TAPS II – 1993), are coded as non-smokers. Those who have never smoked at time 1 but are smoking regularly at time 2 are initiators (coded as 3, 4, or 6 in the NCHS classification).

Since I am trying to predict smoking behavior at time 2, all of the independent, moderating, and control variables reflect the characteristics of the adolescent at time 1. The means, standard deviations, and ranges of the variables are included in Table 5.

There are two primary independent variables. The variable for peer smoking was created by adding two items on the TAPS which ask "number of male friends who smoke" (*Q29) and "number of female friends that smoke" (*Q30). The second independent variable is a measure of parental smoking. This is a dummy variable, with those adolescents who do not have a parent in the household who smokes being coded 0, and those with either, or both, smoking parents coded as 1.

The three moderating variables are measures of age, depression, and school performance. Age is included as a moderating variable since it may be thought of as a proxy for adolescent development. A depression scale was created from six items in the TAPS (see Table 3). Respondents had the option to rate their level of distress on a scale of 1-4 (which for this study has been recoded as 0-3). A scale has previously been created with the dataset by Escobedo, et al (1998) as noted above and has been validated. These items have a Cronbach's Alpha of 0.72. Using principal factor analysis, we can see that these questions appear to load on a common factor (Table 4).

Q48A	During the past year, how often have you felt too tired to do things?
Q48B	During the past year, how often have you had trouble going to sleep or staying asleep?
Q48C	During the past year, how often have you felt unhappy, sad, or depressed?
Q48D	During the past year, how often have you felt hopeless about the future?
Q48E	During the past year, how often have you felt nervous or tense?
Q48F	During the past year, how often have you worried too much about things?

Table 3: Items from the TAPS used to create a Depression Scale (items previously used by Escobedo, Reddy and Giovino (1998)

(principal factors; 2 factors retained)

Factor	Eigenvalue	Difference	Proportion	Cumulative
1	1.73636	1.68064	1.3133	1.3133
2	0.05571	0.09442	0.0421	1.3554
3	-0.03871	0.05340	-0.0293	1.3261
4	-0.09211	0.06683	-0.0697	1.2565
5	-0.15894	0.02121	-0.1202	1.1363
6	-0.18015	.	-0.1363	1.0000

Table 4: Factor Analysis of Items to be included in Depression Scale (n=7885)

Factor Loadings

Variable		1	2	Uniqueness
DtiredR		0.44721	0.08317	0.79308
DsleepR		0.49451	0.05300	0.75265
DsadR		0.60009	0.04459	0.63790
DhoplesR		0.50152	0.11029	0.73631
DtenseR		0.55624	-0.13877	0.67134
DworryR		0.60892	-0.11214	0.61664

(Table 4- continued from previous page)

A scree chart also supports that these appear to represent one underlying variable (Figure 2).
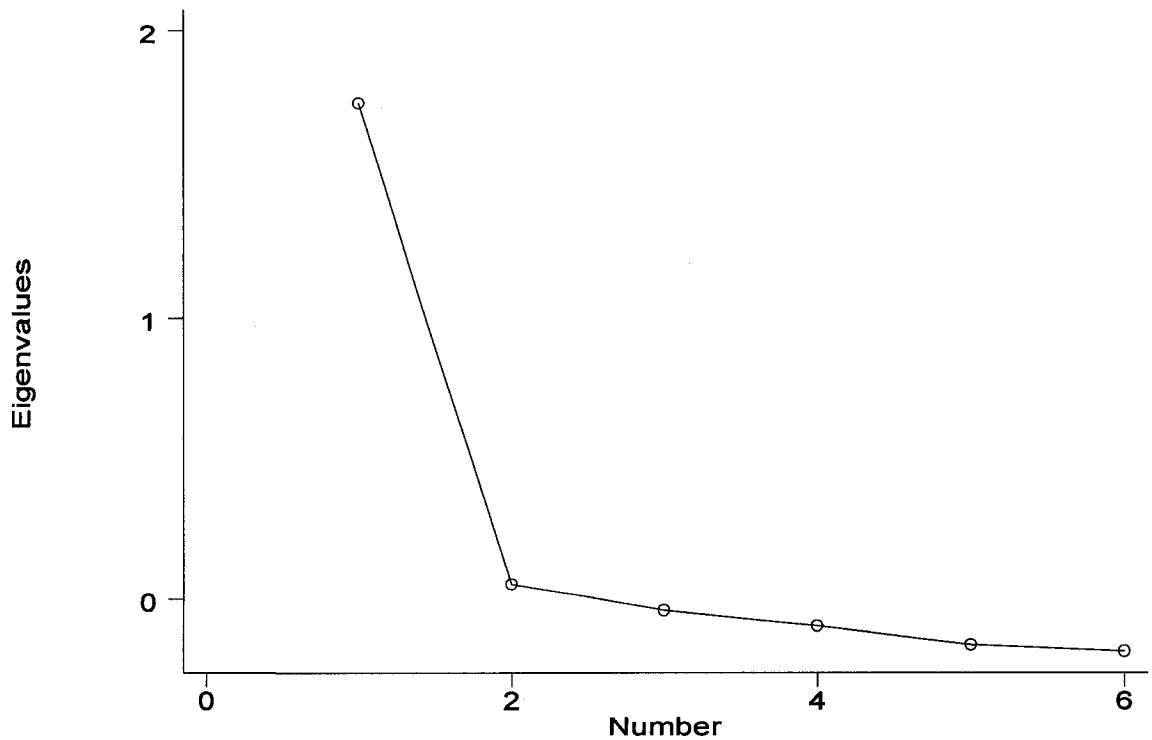
Figure 2: Scree Chart of Factor Analysis of Depression Scale Items

School performance is measured by a self report, meaning a student's perception of their own performance. Choices include "below average", "average", "better than average" or "much better than average". This variable was recoded such that higher performance was reflected by a higher value.

Age is a continuous variable. Though we would normally think of age as a control variable, in this case I am using it as a proxy for adolescent development. As an adolescent ages, it is a reasonable expectation that perceptual and interpretative abilities will change and that these changes may be reflected in the nature of any moderating relationships that might exist with the proposed prediction variables of peer and parental smoking.

Control variables include sex, race, and socioeconomic status. Sex is the usual two category variable with males coded as 1. Race is defined only as white or black, a limitation that exists in the original data. Those identifying themselves as black have been coded 1. Socioeconomic status is defined by categories divided by each 1,000 dollars of family income with a few of the higher categories being increments of 5,000 and the highest category being "over 50,000 dollars". This variable reflects the income at time 1 (1989) and compares to an average income in the United States of $47,184 in 1990 (os.dhhs.gov, retrieved March 5, 2007). The poverty level at that time was $13,359 for a family of four (as defined by the U.S. Bureau of the Census) (http://www.census.gov/hhes/www/poverty/prevcps/p60-175.pdf retrieved March 5, 2007).

Six interaction variables were created to explore for evidence of a moderated relationship. This was done by multiplying the prediction variables – parental smoking

(parsmo) and peer smoking (peers) by the proposed moderating variables – depression (depscalR), school performance (dosch), and age (age) variables. This resulted in six multiplicative interaction terms – par*dep, par*age, par*do, peer*dep, peer*age, and peer*do, which were used for analysis.

Analysis Plan

Since the dependent variable is a two value categorical variable, the data were analyzed using logistic regression methods. The data were analyzed using Stata since it has the capability of analyzing large stratified samples. Stata requires three additional variables to utilize the "survey" commands, which adjust for multistage random sampling. These variables must stipulate the Strata, the Primary Sampling Unit (PSU), and the sampling weight, which reflects the probability of a particular observation being included in the overall sample based on the sampling design. The Strata variable used is CSTRATUM which is a variable created by the National Center for Healthcare Statistics (NCHS) to adjust for certain factors such as age, sex, and race in the sampling process. The PSU variable for the NHIS and the TAPS is based on Metropolitan Statistical Areas (MSAs). The variable CPSU adjusts for the relative sizes of these MSAs. I used CFINALWT as the sampling weight, since this is the weight that reflects the probability of any particular adolescent having his or her data collected using the CATI technique. Subjects who completed the mail survey were not asked to respond to the depression items, thus they have not been included in this sample. Additional description of the sampling process is provided in Appendix 4.

Variable	Variable Name	Total Sample	Standard deviation	Range
		Mean		
Control				
Gender (Male)	sex	0.479	0.50	0-1
Race (Black)	race	0.186	0.39	0-1
Family Income	faminc	20.7	5.98	0-26
Prediction				
Number of Peers who smoke	peers	0.64	1.32	0-8
Parent who smokes?	parsmo	0.37	0.48	0-1
Moderating				
Age	Age	14.5	1.99	11-19
Depression	depscalR	7.75	3.51	0-18
School Performance	dosch	2.18	0.78	1-4
Outcome				
Initiated smoking?	dumsmok	0.162	0.37	0-1

Table 5: Means, Standard Deviations, and Ranges of Variables

Appropriate diagnostics are used to assess the analysis for evidence of

multicollinearity. Logistic regression can also be sensitive to outliers, so the sample was

assessed for any extreme values (Mertler and Vannatta 2005). Hosmer and Lemeshow

(2000) and Hamilton (1992) suggest using diagnostic graphs including:

> a.) Change in Pearson chi-square versus predicted probability

> b.) Change in deviance versus predicted probability

> c.) Influence (dbeta) versus predicted probability

As noted by Jaccard (2001), any interaction effects must be interpreted with caution since the statistical tests indicate only that there is a synergistic effect, and it may or may not be the one that has been indicated in any explanatory model. The first step in this process is to look for evidence of conditional relationships statistically by the use of a multiplicative interaction term. If appropriate, these relationships are explored using conditional effect plots to examine them graphically.

IRB

Approval to conduct the study was obtained from the University of New Hampshire Institutional Review Board (Appendix 5).

CHAPTER IV

RESULTS

This study addresses the question of whether looking at combinations of variables may provide additional benefit in predicting smoking initiation beyond that provided by looking at individual variables. The exploration for the existence of relationships between the outcome variable (smoking initiation) and the suggested predictors (peer and parental smoking) and the proposed moderating variables (age, depression, and school performance) were addressed using Stata. The results of these analyses are presented as follows. First, the characteristics of the individual variables will be described. Second, the main effects of the variables of interest on smoking initiation will be addressed. Next, interaction effects which explore for the existence of the proposed moderated relationships are examined. Lastly, assessment for threats to the analysis using logistic regression diagnostics as recommended in the literature is described.

The analyses were carried out using "survey" commands, which utilize weighting that has been designed for large stratified random samples such as this. The specific variables used in the Stata survey commands to adjust for the research design are described in the methods section and in Appendix 4. Certain individuals or groups are more likely to be included in the sample based on their location or characteristics, not considering this might make it appear that these individuals or groups were over or underrepresented in a multistage random sample such as this. Use of the stratification, primary sampling unit, and probability weight variables can adjust for this over or underrepresentation and result in more accurate point estimates. Using design-based

analysis results in more accurate estimates of standard errors, which is necessary for accurate significance tests. In general, if a survey has been carried out with a stratified sample, these weights are provided and they should be utilized whenever possible (Chantala 1999).

The research design called for taking only those adolescents who were non-smokers at time 1, and then looking at the characteristics at time 1 of those who initiated between time 1 and time 2 (which turned out to include 477 adolescents) versus those who did not (2489 adolescents).

It is, however, also interesting to look at those who were regular smokers at Time 1. Table 6 shows that the characteristics associated with being a regular smoker at time 1 are similar to the characteristics that are well known to be associated with smoking initiation. We can see by comparing tables 6 and 7 that the factors associated with being a smoker are similar in the cross-sectional (Table 6) and longitudinal (Table 7) analyses but several interesting differences are seen. The regular smokers at time 1 are different from those who initiated between times 1 and 2 with regard to age (16.3 vs. 14.1, p<0.05), number of smoking peers (4.27 vs. 0.88, p<0.05), and school performance (2.19 vs. 2.61, p<0.05). By definition, those who initiated between time 1 and time 2 initiated at or after age 12. There are at least two notable differences between the cross sectional and longitudinal data. The cross sectional sample at time 1 would also include early initiators (those who had initiated before age 12) and these smokers might have different characteristics. Secondly, if there was any reciprocal causality of smoking, which might cause one to choose smoking peers or cause a decline in academic performance, this

might account for the difference in peer smoking rates and school performance, although a cross sectional sample would not be able to address issues of sequencing.

Variable	Variable Name	Non-Smokers at Time 1 (n=3,524)	Regular Smokers at time 1 (n=957)	P value from T-test comparing means
Control				
Gender (Male)	sex	0.49 (0.48-0.51)	0.55 (0.53-0.56)	0.00*
Race (Black)	race	0.19 (0.18-0.21)	0.06 (0.05-0.66)	0.00*
Family Income	faminc	19.7 (19.3-20.2)	20.6 (19.9-21.3)	0.93
Prediction				
Number of Peers who smoke	peers	0.60 (0.58-0.61)	4.27 (3.73-4.64)	0.00*
Parent who smokes?	parsmo	0.37 (0.43-0.49)	0.46 (0.41-0.51)	0.00*
Moderating				
Age	Age	14.4 (14.4-14.6)	16.3 (16.1-16.5)	0.00*
Depression	depscalR	7.38 (7.36-7.40)	8.62 (8.52-8.90)	0.00*
School Performance	dosch	2.69 (2.53-2.74)	2.19 (2.11-2.40)	0.00*

Table 6: Means and 95% Confidence Intervals of Selected Variables Comparing Nonsmokers and Those Already Regularly Smoking at Time 1. (Weighted to reflect stratified random sample) (* signif at <0.05)

Table 7 shows the characteristics of the adolescents who were nonsmokers at the time of the first wave of TAPS (1989). Initiators were those that were defined as regular smokers at time 2. It further shows the characteristics of those who initiated smoking versus those who did not.

Variable	Non-Initiators (n=2489)	Initiators (n=477)	P value for difference in initiators v. noninitiators
Control			
Gender (Male)	0.474 (0.45-0.50)	0.502 (0.46-0.54)	0.15
Race (Black)	0.204 (0.174-0.232)	0.094 (0.059-0.130)	0.00*
Family Income	20.7 (20.4-21.1)	20.6 (19.9-21.3)	0.94
Prediction			
Number of Peers who smoke	0.58 (0.54-0.64)	0.88 (0.73-1.04)	0.00*
Parent who smokes?	0.35 (0.33-0.38)	0.46 0.41-0.51)	0.00*
Moderating			
Age	14.5 (14.4-14.6)	14.1 (14.0-14.3)	0.00*
Depression	7.58 (7.44-7.73)	8.62 (8.25-9.00)	0.00*
School Performance	2.85 (2.82-2.89)	2.61 (2.53-2.69)	0.00*

Table 7: A Comparison of the Characteristics (at Time 1, 1989) of Initiators Versus Non-initiators at Time 2: Control, Prediction, Proposed Moderating and Outcome Variables (Weighted to reflect stratified random sample) (n=2966)

Several of the variables in Table 7 are dummy variables. Their means thus equal the proportion that have been coded as 1. This would include gender (which shows that 47.4% of the noninitiators are male compared with 50.2% of initiators, NS), race (which shows that 20.4% of the noninitiators are black compared with 9.4% of initiators, p<0.05), parental smoking (35% of the noninitiators had at least one parent who smoked compared with 46% among the initiators, P<0.05). We can see by looking at the mean of smoking initiation that 16.2% of the sample started smoking between 1989 and 1993. The differences between initiators and non-initiators are consistent with those found

when evaluating main effects in this study, and they will be discussed further in the section on main effects.

Looking at the overall characteristics of the sample (in Table 5) we can see that sample compares well to the percentages seen in the overall United States population. The percentage of males and females in the sample suggests that the sample approximates the actual percentages in the actual population: 47.9% male (95% CI 0.46-0.49) as compared to the population estimate of 49.8% male from the US census. Race shows a slightly higher than expected value at 18.6% "black". United States census figures suggest that the prevalence of African Americans in the United States was 12.9% at the time of the study (www.census.gov) but this survey (TAPS) did not provide the option for choosing Hispanic or other options. Respondents who determined themselves to be "non-white" may have chosen the "black" option. Family income was measured by the respondent choosing the range of income which most closely reflected the family income as assessed by the adolescent. The average income in the United States at the time was $47,184 with the median being $35,225. Though it is difficult to estimate what precisely the number 20.7 would translate to in real dollars, it would probably fall into the range of $20,000-30,000. Though this is not directly reflective of the population at large, the measure probably does provide some understanding of the adolescent's subjective relative assessment of the family's means. Income measures have been improved in later versions of the National Health Interview Survey, although income is well known to be a difficult variable to measure.

The variable "number of peers who smoke" provides an actual measurement albeit through self report. The number of peers who smoked averaged 0.64 (95% CI

0.59-0.69). A large percentage (71.5%) of the sample had no friends who smoked. Among those with at least one smoking friend, the number of smoking friends averaged 2.3. This can be compared to the sample at time 1 in which only 52.4% of the sample had no smoking friends. This again raises the issue that those who initiate during adolescence may be different from those who initiate during pre-adolescence or that reciprocal causality causes smokers to choose smoking friends.

Parental smoking was coded as "1" if either parent smoked since this was felt to reflect an acceptance of smoking in the home. In this sample, 37% of the respondents had at least one parent who smoked.

The average age of the sample was 14.5 which is roughly what one would expect in a large sample of 11-19 year olds. The average age of smoking initiation in this sample was 12.6 which is comparable to most other reports. A dummy variable for older versus younger adolescents was created, but no evidence of a threshold effect was found so those analyses are not reported here.

The depression variable is a summed scale. Explanation of the creation of this scale is provided in the methods chapter. Results of the depression measure show that rates in this analysis are consistent with previous analysis of this sample as reported by Escobedo (1998). Escobedo and other studies, however, have used primarily a "cut-point" approach to defining depression whereby those who reached a certain score were defined as depressed, and those who did not were considered to be depression-free. While this approach may possess a certain logic in a "clinical" definition of depression where decisions are made to treat or not treat, it may be more appropriate to think of depression on a continuum in this case. Figure 3, which is a bar chart of the percentage of smokers at

each level of depression shows only limited support for the existence of a threshold effect. This study will explore the relationship between depression and smoking initiation using depression as a continuous variable.
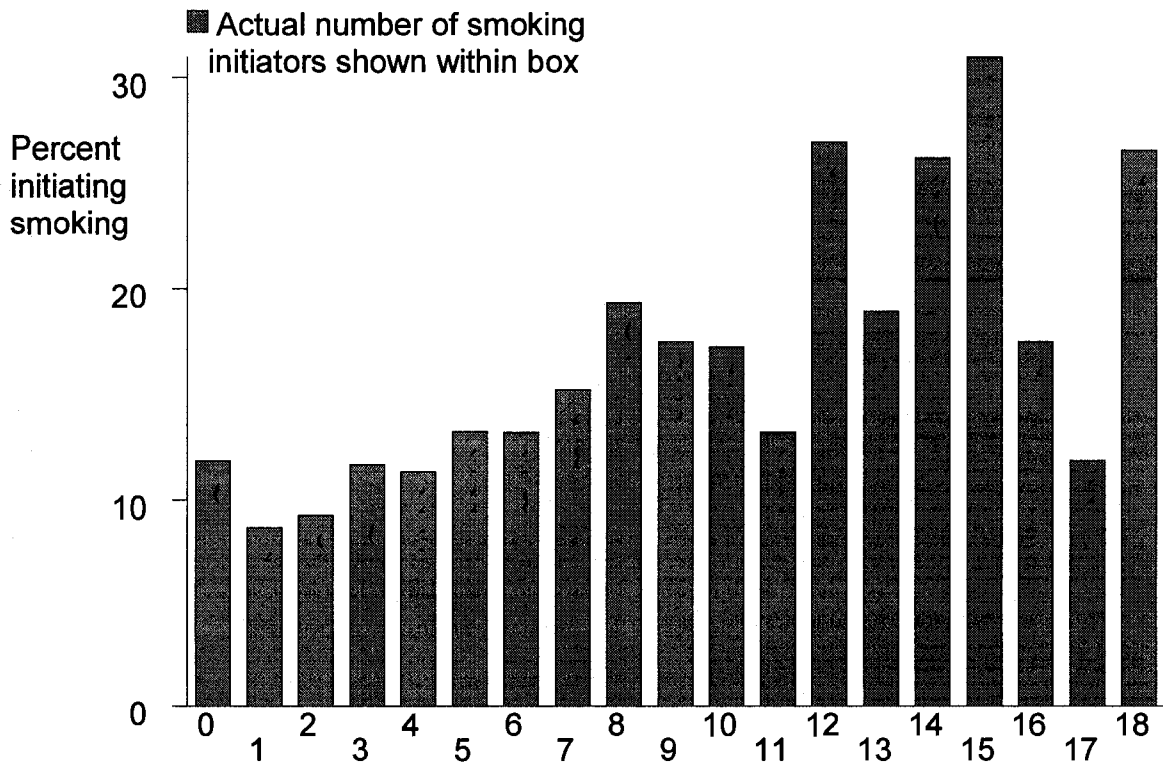
Figure 3: Bar Chart Comparing the Percentage of Smoking Initiation at Each Level of the Depression Score Showing Only Limited Evidence of a Threshold Effect (Number within the bar represents actual number of smoking initiators)

School performance is a 4 option choice. It was recoded such that 4=much better than average, 3=better than average, 2=average, and 1= below average. Table 8 provides the percentages estimations of the adolescents in each category in the overall population using weighted data. It is interesting to note that few students rate themselves below average (2%, or 52 adolescents). The "average" students in fact seem to represent the

lower performers. This may reflect a cultural tendency not to label students "below average" and needs to be considered in comparisons of these adolescents.

	Proportion of students reporting their academic performance as:	Observations
Much better than average	21.6%	639
Better than average	40.5%	1195
Average	35.9%	1068
Below Average	2.0%	52

Table 8: Distribution of Adolescents Self-reported School Performance (transformed to percents) (weighted to reflect stratified random sample)

Main Effects

Analyses which addressed the main effects of control, prediction and moderating variables on the probability of smoking initiation in this sample revealed results that are quite similar to those found in other samples.

Most studies have found higher rates of smoking among males. This trend exists in this sample as well, but the lack of a statistically significant effect involving gender is consistent with the narrowing in smoking rates between the genders over recent history. Smoking initiation rates by gender show the pattern expected by recent historical trends with minimal difference between males (11.5%) and females (10.7%) (OR 1.22, NS). Historical evidence would, however, still suggest that the processes that affect male and female smoking rates may be different, so I will continue to use gender as a control variable in other analyses.

Variable		Odds Ratio	95% CI
Control			
Gender (Male)	Sex	1.22	0.95-1.55
Race (Black)	Race	0.35*	0.23-0.55
Family Income	Faminc	0.99	0.97-1.01
Prediction			
Number of Peers who smoke	Peers	1.16*	1.06-1.27
Parent who smokes?	Parsmo	1.38*	1.10-1.73
Moderating			
Age	Age	0.88*	0.83-0.93
Depression	depscalR	1.09*	1.05-1.13
School Performance	Dosch	0.69*	0.60-0.82

Table 9: Logistic Regression of Smoking Initiation on Selected Variables (sex, race, faminc, peers, parsmo, age, depscalR, dosch) expressed in odds ratios (* p<0.05) (weighted to reflect stratified random sample) (n=2387)

With regard to race, smoking initiation rates in this sample are also consistent with recent historical trends with 17.9% of whites and 8.1% of those who called themselves black initiating during the 1989-1993 period, showing a significant difference (OR 0.35 95% CI 0.23-0.55). As previously noted, the dramatic change in smoking rates among African Americans is also evidence that changing social factors (and social factors in general) are important in the initiation of smoking.

No significant relationship (OR 0.99, NS) was found between family income and smoking initiation. This may have had to do with the poor quality of the measure. Despite the fact that the measure provides only an estimation of income, income would be expected to be important in the smoking initiation process so I will still include this variable as a control in the analyses.

Both peer smoking and parental smoking increased the risk of smoking initiation. Parental smoking was a binary variable, so having either parent smoke increased the odds by 38%. Peer smoking was a measurement variable, so for each additional smoking friend the odds of smoking initiation increased by 16%.

The risk of smoking initiation decreased as age increased (OR 0.88 95% CI 0.83-0.93) with 20.5% of 11 year olds initiating and 10.7% of 19 year olds initiating. This pattern is similar to previously published studies.

Depression increased the risk of smoking initiation (OR 1.09 95% CI 1.05-1.12) such that, for each increase in the depression score (measured 0-18), the odds of smoking initiation increased by 9%. This result provides support for depression preceding smoking initiation in this sample. The implications for this finding in relation to previous studies will be further addressed in the next chapter.

School performance also had a significant relationship to smoking initiation (OR 0.69 (95% CI 0.60-0.82). Thus, higher school performance resulted in a lower risk of smoking initiation. If the variable is coded as poor school performance with higher values, the OR is 1.43 (95% CI 1.23-1.67), showing that, for each increment of worsening school performance, the odds of initiating smoking increase by 43%. It is interesting to note that while some studies have supported school performance as a factor in smoking initiation, no other study could be found which provided that support in a national longitudinal sample such as this one. This finding may have important implications, and the possible factors which may contribute to this relationship and the practical implications will be discussed in Chapter 5. It is important to note that this relationship has been found in cross sectional studies (Carvajal, Wiatrek, Evans, Knee,

and Nash 2000; Young and Rogers 1986) and in a statewide longitudinal study (Cox, Zhang, Johnson, and Bender 2007), but this is the only known case where it has been documented in a longitudinal national sample.

Interaction Effects

The exploration for interaction effects will be divided into two parts. The first three hypotheses address interactions between age, depression, and school performance and *parental smoking*; and they will be addressed together. The second three hypotheses which include similar interactions with *peer smoking* will then be discussed.

Before interaction terms were created, the variables of interest were "centered". Centering is achieved by subtracting the mean of each variable from the individual values for that variable. Though there are differences of opinion regarding the importance of centering, it is generally felt to result in less danger of multicollinearity. The results reported here utilize the interaction terms, which were created using centered variables.

The three hypotheses involving parental smoking and proposed moderating variables are:

Hypothesis 1:
The effect of parental smoking on smoking initiation will be greater for younger versus older adolescents

Hypothesis 2:
The effect of parental smoking on smoking initiation will be greater in the presence of higher levels of depression

Hypothesis 3:
The effect of parental smoking on smoking initiation will be greater for those with lower levels of school performance

	Hypothesis 1	Hypothesis 2	Hypothesis 3
Sex (Male)	1.21 (0.95-1.54)	1.22 (0.96-1.55)	1.22 (0.96-1.56)
Race (Black)	0.35 (0.23-0.55)*	0.35 (0.23-0.55)*	0.35 (0.23-0.55)*
Faminc	0.99 (0.97-1.01)	0.99 (0.97-1.01)	0.99 (0.97-1.01)
Peers	1.16 (1.06-1.27)*	1.16 (1.06-1.27)*	1.16 (1.06-1.27)*
Parsmo	1.31 (1.04-1.64)*	1.39 (1.11-1.74)*	1.38 (1.10-1.73)*
Age	0.88 (0.83-0.93)*	0.88 (0.83-0.93)*	0.88 (0.83-0.93)*
depscalR	1.09 (1.05-1.13)*	1.09 (1.05-1.13)*	1.09 (1.05-1.13)*
Dosch	0.70 (0.60-0.82)*	0.70 (0.60-0.82)*	0.70 (0.58-0.85)*
parsmoXage	0.90 (0.80-1.02)		
parsmoXdepscalR		0.98 (0.92-1.04)	
parsmoXdosch			0.99 (0.72-1.36)

Table 10: Logistic Regression of Smoking Initiation and Interaction Terms of Parental Smoking and Proposed Moderating Variables (age, depression, and school performance) expressed in odds ratios (weighted to reflect stratified random sample) (* $p<0.05$) (n=2387)

Table 10 shows that none of the proposed interactions involving parental smoking were supported. This would suggest that the proposed moderating variables do not change the influence that parental smoking has on the probability of smoking initiation. This is contrary to the relationship proposed in the conceptual model of this study. Alternatively, we should also consider the possibility that the primary influence of parental smoking, and thus the time in which it would be most likely to interact with "cognitive processes," might take place at a time earlier than the age group we are studying in this sample. This and other possibilities which might explain the lack of support of these relationships will be discussed further in the next chapter.

Table 11 shows the results for the hypotheses involving peer smoking. These include:

Hypothesis 4:
The effect of peer smoking on smoking initiation will be greater for older versus younger adolescents

Hypothesis 5:
The effect of peer smoking on smoking initiation will be greater in the presence of higher levels of depression

Hypothesis 6:
The effect of peer smoking on smoking initiation will be greater for those with lower levels of school performance

	Hypothesis 4	Hypothesis 5	Hypothesis 6
Sex (Male)	1.22 (0.95-1.56)	1.22 (0.96-1.56)	1.21 (0.95-1.55)
Race (Black)	0.35 (0.23-0.55)*	0.35 (0.23-0.55)*	0.36 (0.23-0.58)*
faminc	0.99 (0.97-1.01)	0.99 (0.97-1.01)	0.99 (0.96-1.01)
peers	1.16 (1.06-1.28)*	1.15 (1.06-1.26)*	1.53 (1.16-2.03)*
parsmo	1.37 (1.09-1.72)*	1.38 (1.10-1.73)*	1.38 (1.10-1.74)*
Age	0.88 (0.83-0.93)*	0.88 (0.83-0.93)*	0.88 (0.83-0.93)*
depscalR	1.09 (1.05-1.13)*	1.09 (1.05-1.13)*	1.09 (1.05-1.13)*
dosch	0.70 (0.60-0.82)*	0.70 (0.60-0.82)*	0.76 (0.64-0.90)*
peersXage	0.98 (0.94-1.03)		
peersXdepscalR		1.01 (0.99-1.03)	
peersXdosch			0.89 (0.81-0.99)*

Table 11: Logistic Regression of Smoking Initiation and Interaction Terms of Peer Smoking and Proposed Moderating Variables (age, depression, and school performance) expressed in odds ratios (weighted to reflect stratified random sample) (* $p < 0.05$) (n=2387)

One of the three hypotheses involving interactions between peer smoking behavior and proposed moderating variables was supported: the interaction of peer smoking and school performance. When an interaction effect exists, it is evidence that a conditional or moderating effect exists. One way to better understand this moderating effect is to do a conditional effect plot. If the effect of one variable varies depending on the level of another variable, it can be demonstrated graphically. In an interaction effect, the appearance is different than what would be expected in an additive effect. The

interaction of peer smoking behavior and school performance is shown by the different slopes in Figure 4.

In graphing the levels of school performance, we can see that students with lower levels of school performance seem to have a greater change in the probability of smoking initiation as the number of smoking peers increases; compared to higher academic performers, as demonstrated by the slight difference in the slopes of the top and bottom lines. Incidentally, we can also clearly see the direct effect of school performance in the conditional effect plot, with lower school performers having higher rates of smoking initiation.
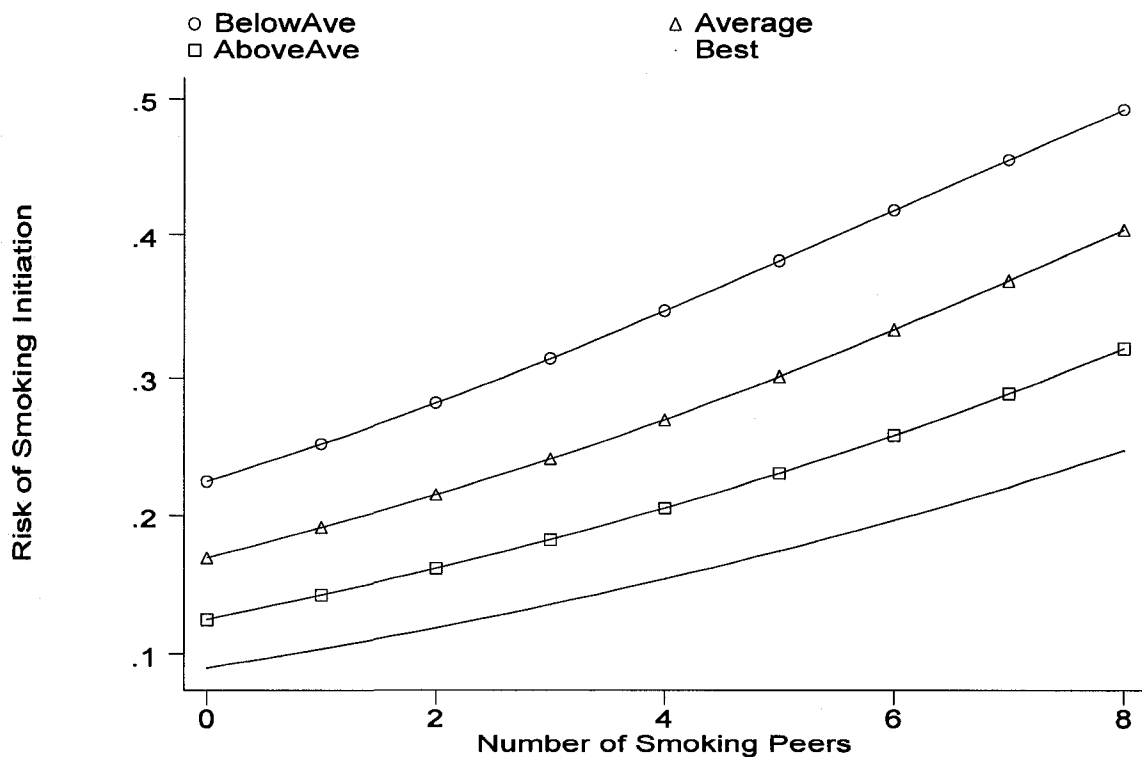
Figure 4: Likelihood of Smoking Initiation for Four Levels of Academic Performance at Various Levels of Peer Smoking Behavior (sex, race, faminc, parsmo, age, depscalR held at their means) (weighted to reflect stratified random sample)

70

The potential interaction of peer smoking and school performance is of particular interest because the conceptual model of this study suggested that poor school performance might reflect a lowered ability to comprehend the risks of smoking, and thus increase the vulnerability to the influences of peer smoking behavior. The implications of this finding are further discussed in the next chapter.

Though the differences in the slopes on the conditional effect plot appear to be quite modest, it does provide some limited support for Hypothesis 6, "the effect of peer smoking on smoking initiation will be greater for those with lower levels of school performance". Figure 5 represents a diagram of the proposed moderating relationship.

Peer Smoking ----------------------→ Smoking Initiation

↑

School Performance
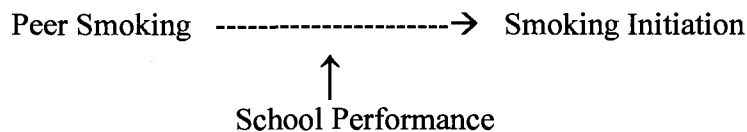
Figure 5: Diagram Indicating a Relationship between Peer Smoking Behavior and Smoking Initiation which is Conditional on the level of School Performance

Another way to think about this is, that the combination of these two factors – peer smoking and school performance – has a different effect than one would expect from the sum of their separate individual effects. In other words, the effect of peer smoking seems to be greater when poor academic performance is also present. This could also be expressed as: a poor student might be more vulnerable to the influence of peers. Chapter 5 provides additional discussion of this finding.

Diagnostics

Table 12 shows that Zero order correlations among variables, including the multiplicative interactions variables, do not raise any concerns about simple collinearity. The highest correlation is 0.4047 (between peer smoking and the interaction of peers and age) and at this level any effect on standard errors would not have a meaningful effect.

The Stata command "collin" assesses for multicollinearity (Table 13). This assessment shows that the highest Variance Inflation Factor (VIF) is 1.41 and the highest R squared is 0.2915. This would indicate that multicollinearity is also not an issue in this sample. VIFs are not a concern unless the largest VIF is greater than ten, or the mean of all VIFs is considerably larger than one (Stata 1999).

Regression Diagnostics

The diagnostic plots mentioned in the methods section – specifically change in Pearson chi-square versus predicted probability, change in deviance versus predicted probability, and influence (dbeta) versus predicted probability – were carried out for the specific analysis of greatest interest in the overall analysis approach, that of the regression of smoking initiation on the interaction of school performance and peer smoking behavior.

Table 12: Zero Order Correlations

	age	sex	faminc	dosch	peers	depscalR	parsmo
age	1.0000						
sex	-0.0330	1.0000					
faminc	0.0206	0.0143	1.0000				
dosch	-0.0283	0.0737	-0.1294	1.0000			
peers	0.2570	-0.0886	-0.0528	0.0990	1.0000		
depscalR	0.1095	-0.1428	-0.0202	0.0736	0.1495	1.0000	
parsmo	-0.0139	-0.0128	-0.0956	0.0850	0.0899	0.0444	1.0000
race	0.0076	-0.0165	-0.3174	0.0419	-0.0682	-0.0092	0.0274
peerXage	0.0133	-0.0037	0.0062	0.0380	0.4047	0.0137	0.0079
peerXdo	0.0339	-0.0121	0.0186	-0.0084	0.1824	0.0178	0.0041
peerXdep	0.0108	-0.0483	0.0007	0.0183	0.2040	0.0512	0.0057
parXage	-0.0061	-0.0306	0.0275	-0.0229	0.0098	-0.0208	-0.0029
parXdo	-0.0232	-0.0026	0.0389	0.0129	0.0061	0.0041	0.0220
parXdep	-0.0208	0.0303	0.0083	0.0045	0.0082	0.0492	0.0152

	race	peerXage	peerXdo	peerXdep	parXage	parXdo	parXdep
race	1.0000						
peerXage	-0.0613	1.0000					
peerXdo	-0.0392	0.0430	1.0000				
peerXdep	-0.0239	0.1516	0.0820	1.0000			
parXage	0.0199	0.0823	0.0183	0.0041	1.0000		
parXdo	-0.0335	-0.0051	0.1191	0.0177	-0.0372	1.0000	
parXdep	0.0167	-0.0101	0.0267	0.1171	0.1021	0.0647	1.0000

Table 13: Collinearity Diagnostics

VIF= Variance Inflation factor

Collinearity Diagnostics

Variable	VIF	SQRT VIF	Tolerance	R-Squared
age	1.10	1.05	0.9105	0.0895
sex	1.04	1.02	0.9609	0.0391
race	1.12	1.06	0.8933	0.1067
faminc	1.14	1.07	0.8769	0.1231
peers	1.41	1.19	0.7085	0.2915
parsmo	1.03	1.01	0.9720	0.0280
dosch	1.05	1.02	0.9545	0.0455
depscalR	1.06	1.03	0.9409	0.0591
peerXage	1.24	1.11	0.8047	0.1953
peerXdo	1.05	1.03	0.9507	0.0493
peerXdep	1.08	1.04	0.9225	0.0775
parXage	1.03	1.01	0.9754	0.0246
parXdo	1.03	1.01	0.9753	0.0247
parXdep	1.04	1.02	0.9599	0.0401
Mean VIF	1.10			

Regression diagnostics can serve multiple functions. Values that are outliers may exert undo leverage and actually influence the statistical outcomes of analyses. In a large sample such as this, the influence of a few values would not be expected to effect significance levels to any great extent and, in fact, on reanalysis after dropping the outlier values, no difference is seen in logistic regression results. A second benefit to regression diagnostics is that we can look at the outliers to see how they may differ from the other values, and see if meaningful information related to the patterns of those outliers can be gleaned.

The first figure related to diagnostics (Figure 6) analyzes change in Pearson chi-square versus predicted probability, and it shows that there is a single potential outlier. This outlier is actually on the slope that would be expected, and would not be expected to

change the statistical analytic outcome. On further investigation, case # 882 is an 18-year-old girl who started smoking, had three peers who smoked, and was a very high performer academically. This young woman had a lower than average family income (9), a low depression rating (1), and was black. This case demonstrates some of the difficulties with trying to predict smoking initiation, since, on several counts, she would be considered low risk for smoking initiation. It would of course be interesting to look at other characteristics of this young woman to see why she does not fit our expectations. She might be a high academic performer, but may be cohorted for whatever reason with others who are not. Unfortunately the data available in this sample does not allow us to address that question. Future research may want to look at atypical cases such as this to try to determine the factors that are associated with atypical initiators.

Figure 6: Change in Pearson Chi-Square versus Predicted Probability for the Logistic Regression of Smoking Initiation on peerXdo (interaction of peer smoking and school performance). Other variables included in the analysis include age, sex, race, faminc, parsmo, depscalR, peers, dosch. (weighted to reflect stratified random sample)

The second figure related to diagnostics (Figure 7) looks at change in deviance versus predicted probability. In this analysis, we see a small cluster of cases that seem to vary from the usual pattern (cases 492, 773, 797, and 1080). These four cases share some interesting similarities. All are 12-14 year old girls who initiated smoking, all are average school performers, none have peers who smoke, three of the four have parents who smoke, and most have depression scores a bit above the mean (2, 11, 11, 12 – the sample mean was 7.75). Although they vary with regard to family income, values for this variable for the four cases are 10, 13, 18, and 26 (the sample mean was 20.7).

The similarities in these cases are quite interesting. Since they are near the mean age of smoking initiation in this sample, they may not yet have any peers that are smokers, and yet they seem to have other risk factors which may contribute to their initiating smoking. It is possible that they are "smoking pioneers", who, from a public health view, could be looked at as potential index cases of an outbreak. Since many adolescents initiate smoking at a young age, looking further at this age group (and at pre-adolescents) may have greater potential for understanding the smoking initiation process. Factors which are associated with initiation at those times may be substantially different from the factors that relate to adolescent smoking initiation in general.
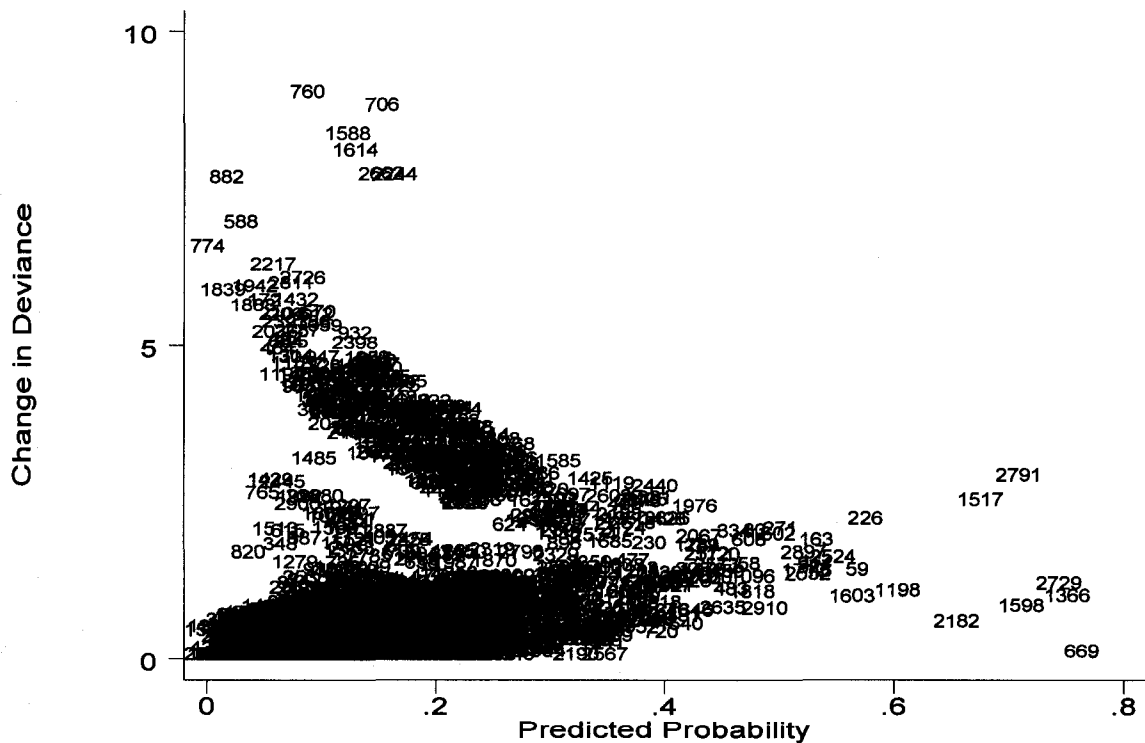
Figure 7: Change in Deviance Versus Predicted probability for the Logistic Regression of Smoking Initiation on peerXdo (interaction of peer smoking and school performance). Other variables included in the analysis include age, sex, race, faminc, parsmo, depscalR, peers, and dosch. (weighted to reflect stratified random sample)

The third figure looking at diagnostics (Figure 8) Influence versus Predicted

Probability, shows evidence of the same cluster and an additional outlier. On looking at

case #1945, it is an 18-year-old, white, non-smoking male. He does, however, have seven

smoking friends, is a poor school performer, has a higher than average depression score

(11), and has at least one smoking parent. Research which looks at the ability of

adolescents such as this to resist smoking might help identify other factors which could
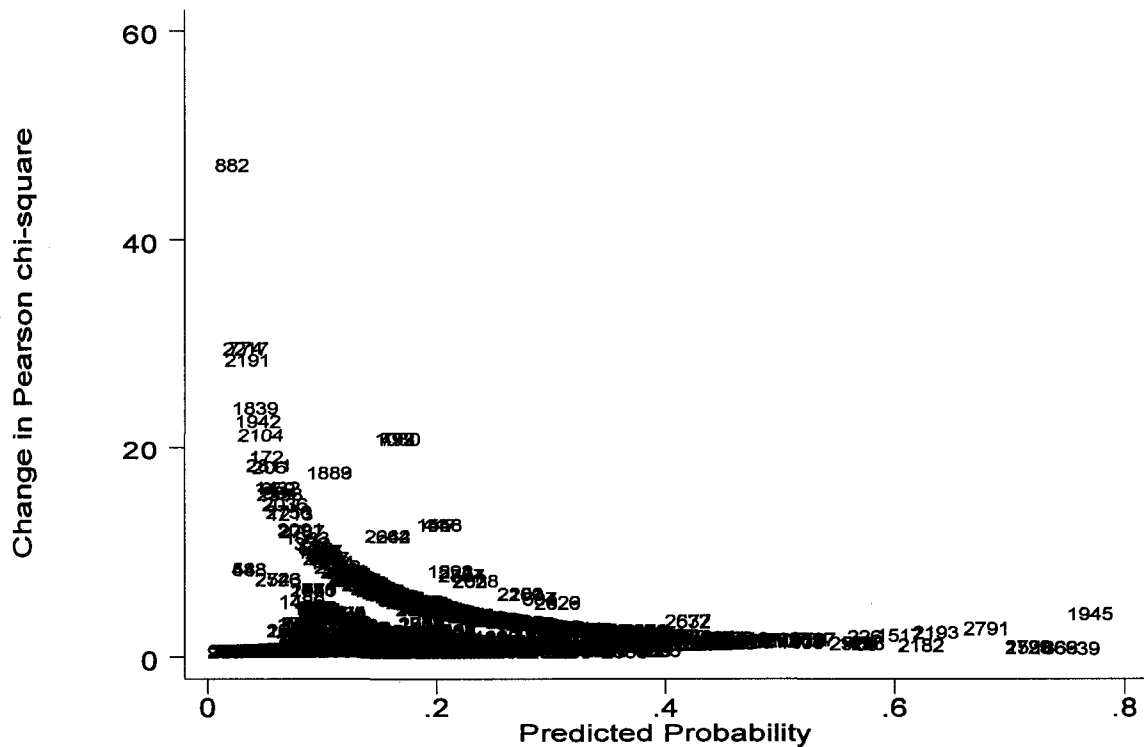
provide an adolescent with the ability to avoid smoking.
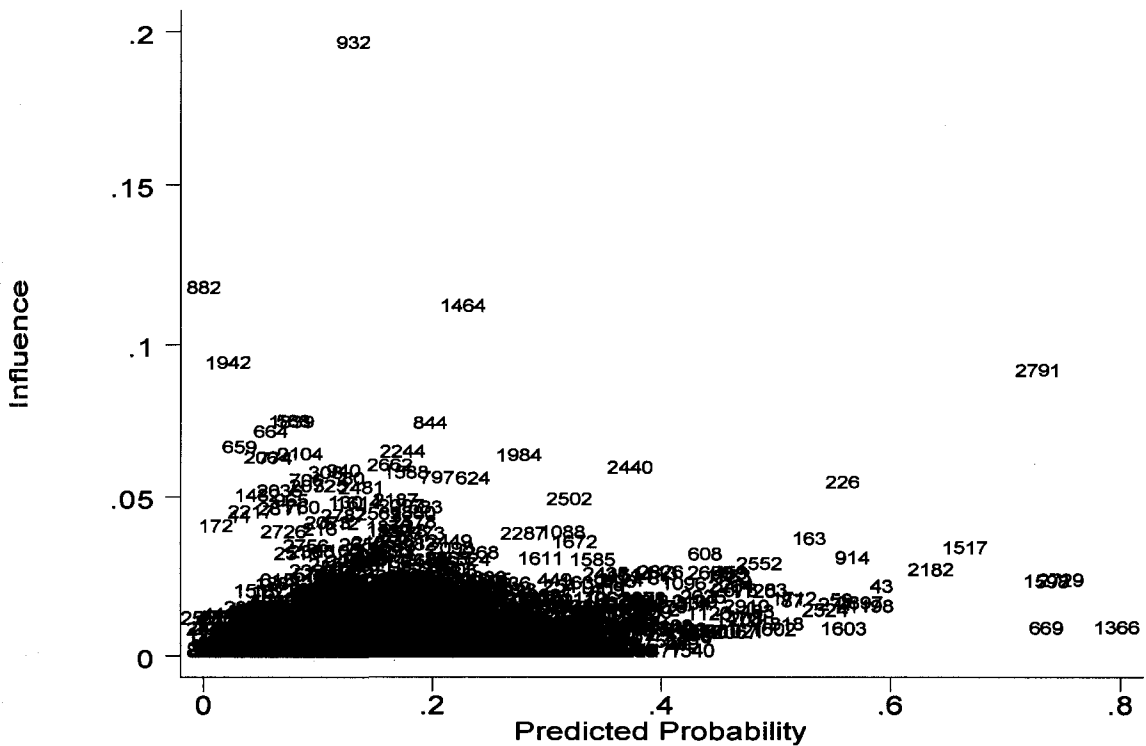
Figure 8: Influence Versus Predicted Probability for the Logistic Regression of Smoking
Initiation on peerXdo(interaction of peer smoking and school performance). Other
variables included in the analysis include age, sex, race, faminc, parsmo, depscalR, peers,
and dosch. (weighted to reflect stratified random sample)

Overall, logistic regression diagnostics would suggest that there are no significant

threats to our overall interpretation. We do, however, see several interesting patterns.

These patterns remind us that, while we may have evidence for risk factors that may

eventually be used to develop smoking prevention programs, smoking is a complex

phenomenon; and specifically that additional exploration of the behavior of

preadolescents may provide additional key insights into the smoking initiation process.

CHAPTER V

SUMMARY, DISCUSSION AND RECOMMENDATIONS

Diseases directly caused by cigarette smoking cause significant suffering throughout the world. Appendix 1 lists over forty serious illnesses in which the link to smoking is well documented. Making this fact even more tragic is what appears to be the voluntary nature of smoking. Unfortunately, even after extensive study, the processes that lead to smoking initiation are poorly understood. This study has tried to take a somewhat different approach by focusing on the *combinations* of selected social and individual factors in an effort to contribute to the understanding of the smoking initiation process.

Most of the current efforts in smoking research and prevention are being focused on approaches such as tax increases and improved policy enforcement as efforts to prevent smoking. Based on evidence that suggests that those kinds of interventions may have reached their maximum potential, specifically a stalling in the decline of smoking rates, this study represents a departure from that approach and a return to a focus on such social factors as peer and parental smoking behaviors. Unlike most previous studies, however, it focuses on these factors' interaction with other potentially moderating factors – specifically age, depression, and school performance – that might be expected to alter the adolescent's perception or interpretation of those behaviors. Several theoretical models suggest that these kinds of processes may moderate the impact of other social factors on the smoking initiation process. This study has generated a number of findings which can help us to make progress in our understanding of the issues of interest.

This discussion will be organized using the following approach. Initially, the discussion will address the results of the analyses which looked for the existence of interactions between variables that may have an impact on the likelihood of smoking initiation. Special attention will be given to the evidence found for an interaction effect between school performance and peer smoking behavior – a relationship that has not been previously reported. The implications of these findings will also be considered in relation to the proposed conceptual framework, and in relation to the theories which provided the foundation for the development of that conceptual framework. Next, findings which are primarily replications of previous studies of main effects will be reviewed with discussion of the implications of those findings. A special emphasis will be placed on the issues of depression and school performance. The remaining sections will include thoughts regarding the limitations of the current study, implications for further research, immediate implications for clinical and policy actions, and some concluding remarks.

Interaction of Prediction and Moderation Variables on Smoking Initiation

The sample was analyzed for the presence of interaction effects using multiplicative interaction terms. Several interesting findings, both in relation to suggested relationships that were supported and those that were not supported, provide information which can be used to further our understanding of the phenomena involved. The overall conceptual model of the study proposed that interactions would exist between peer or parental smoking, and several variables that could be thought of as moderators of perception or interpretation of those smoking behaviors (i.e., age, depression, and school

performance) in their influence on smoking initiation. Exploration for conditional effects

provided only limited support for this conceptual model. Figure 9 reviews the conceptual

model.

Depression

↓

Peer smoking

--→   Smoking

↑   ↑   Initiation

Parental smoking

School   Age (Adolescent Development)
Performance
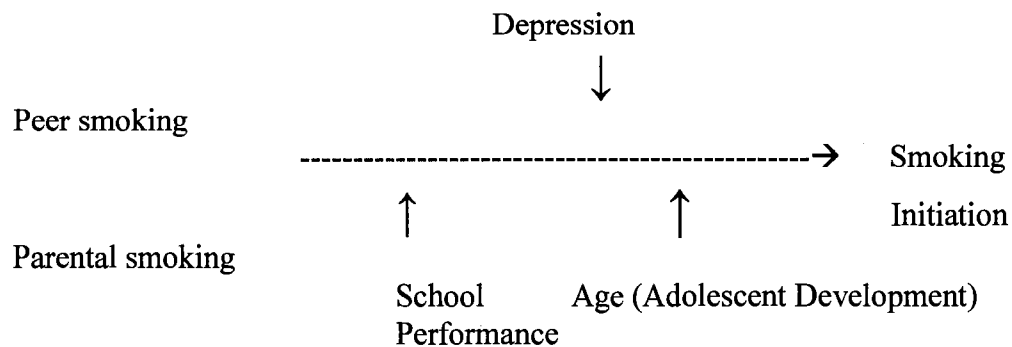
Figure 9: Conceptual Model Guiding the Study (with controls for gender, race, and
family income)

The conceptual framework identified two prediction variables and three proposed

moderators resulting in six hypotheses:

Hypothesis 1:
The effect of parental smoking on smoking initiation will be greater for younger
versus older adolescents

Hypothesis 2:
The effect of parental smoking on smoking initiation will be greater in the
presence of higher levels of depression

Hypothesis 3:
The effect of parental smoking on smoking initiation will be greater for those with
lower levels of school performance

Hypothesis 4:
The effect of peer smoking on smoking initiation will be greater for older versus
younger adolescents

Hypothesis 5:
The effect of peer smoking on smoking initiation will be greater in the presence of
higher levels of depression

Hypothesis 6:
The effect of peer smoking on smoking initiation will be greater for those with lower levels of school performance

No evidence of interaction was found for the first three hypotheses which addressed *parental smoking* and the proposed moderators – age, depression, or school performance. Though the possibility of a type II error must be considered, the lack of interaction effects in these analyses suggests that there may not be a conditional relationship between these variables in this sample.

Based on my analysis, the effect of parental smoking does not seem to change as adolescents age through the 12-18 year-old period, at different levels of depression or at different levels of school performance. It could be tempting to abandon approaches which focus on combinations of variables based on the lack of support in these analyses. However, we should also consider the possibility that relationships might exist between these variables that were not detected by my approach. In fact, two of the theories which were used as a foundation for the model, social learning theory and social attachment theory, might have suggested that parental smoking effects were exerted earlier in the young person's development than this sample can measure, in which case roughly half of those initiating would have already initiated by the time they were old enough to be included in this sample. Smoking initiation is a phenomenon that occurs more than half of the time at or before the age of twelve. Thus, since this sample was predominantly made up of adolescents rather than pre-adolescents, it might not have been expected to show a substantial impact of the influences of parental smoking. The proposed relationships might be found in earlier initiators (pre-adolescents) but not in later

initiators. This possibility would need to be tested on a sample of pre-adolescents, if in fact the issue is that parental influence is greater in children younger than twelve. The findings of this study, though not conclusive, suggest that the influence of parents may be stable throughout adolescence. This may have practical implications in that parents may assume that their influence is declining when in fact they may still have more influence on their adolescent children than they believe.

No evidence was found for interactions between parental smoking and depression and parental smoking and school performance. This would seem to indicate that the theorized increase in vulnerability to the impact of parental smoking behavior associated with an adolescent being depressed or a poor school performer may not exist. Alternatively, these young people might have been vulnerable to the impact of that behavior at an earlier age.

We must also consider the possibility that I have not chosen the right moderating factors to include in the analyses. There may be other factors that alter perception or interpretation that would thus increase or decrease the vulnerability of adolescents to the influence of parental smoking.

Testing the model using *peer smoking* as the predictor variable yielded somewhat different results. No evidence was found supporting interactions between peer smoking and age or peer smoking and depression level. An interaction between peer smoking and self-perceived school performance on the probability of smoking initiation was supported. This does provide at least some limited support for the proposed model. The existence of an interaction between school performance and peer smoking has not been previously reported and may also have practical implications. School performance seems

to differ from the other proposed variables, although how it differs is not clear. The model would suggest that this may reflect actual cognitive abilities of the adolescent to assess risk leading to a greater understanding of the health risks of smoking, but as noted in the literature review, self medication for ADD, self medication for self-esteem issues, or reframing the importance of school performance by assuming a rebellious persona are all possible alternative explanations.

School performance may reflect overall cognitive abilities but, since the measure was a self-report, it might also represent a characteristic of the adolescent such as self-confidence or self-esteem. It is also possible, as noted in the literature review, that smoking may be "self medication" for ADD and that, in certain subsets of the population, it may actually serve to improve academic performance at least partially. Efforts to explicate this relationship should continue but, in the short term, just knowing that the combination of peer smoking and school performance may create risk for smoking initiation could have practical implications. For example, educators who are determining whether "tracking" or "mainstreaming" approaches are to be used for at-risk students might want to consider how grouping poor students together, who are often also smokers, might increase risk for smoking initiation.

My theoretical model had suggested that school performance would alter the impact of peer smoking behavior on the probability of smoking initiation, with lower school performers being more vulnerable to the influence of peers. The model might also suggest that higher school performance would correlate with higher abilities to understand the implications of one's health behavior choices. The conditional effect plot supported the nature of this relationship although the effect shown was small. If, in fact,

peer influence is conditional on the adolescent's school performance, this could have important practical implications. Interventions could be designed to address peer factors among those with the lowest academic performance. Alternatively, promoting improved school performance might actually provide protection to these students. Students that are at risk academically could be identified, and specific individual interventions to prevent smoking initiation could be used.

The lack of evidence for an interaction with age could be explained by a stable level of peer influence throughout this age range, as has been suggested in relation to parental smoking. There may be a difference between pre-adolescent and adolescent initiators, but this would not have been detected by analyzing this sample.

Support had been found by two previous studies (Patton et al. 1998; Ritt-Olson et al. 2005) for an interaction effect between peer smoking and depression on the probability of smoking initiation. My analysis did not find support for this relationship. Others have also tested for this interaction without finding evidence (Tercyak, Goldman, Smith, and Audrain 2002). Lack of support found in this and other studies might suggest that an interaction does not exist and that the two studies cited above have found positive results by chance. The lack of evidence in other samples might also suggest that there is something different about the samples used. Patton, et al. (1998) used a 6 wave sample of Australian students starting at age fourteen in 1992 and ending in 1995. It is possible that there is something special about Australian culture, or the social factors that existed at the time that might explain this finding. Ritt-Olsen, et al. (2005) had found evidence of an interaction between peer approval of smoking (1-4 rating) and depression (dichotomous with 23 on the CES-D being the cut point) only among females. The CES-D is usually

weighted, and scores can be as high as 53. Their sample was drawn from southern California and consisted only of 12 and 13 year olds. Their data were cross-sectional and were collected in 2001. I analyzed this sample (the TAPS) using only females and found no evidence of an interaction effect between peer smoking and depression (OR 1.01 -95% CI 0.99-1.03). Focusing on younger adolescents may have been a factor in the results of Ritt-Olsen, et al, as well as other factors specific to the southern California population. Further study will be needed to explore possible explanations for the variations in findings. Determining whether depressed adolescents are more vulnerable to the impact of smoking peers could be useful information for educators and mental health professionals.

When we look at the specific conceptual model for this study in relation to the findings, we see that the support for the model was quite limited. In the one significant interaction that was detected, the effect size was modest and the confidence interval of this odds ratio approached 1.00 (OR 0.89 95% CI 0.81-0.99). This emphasizes the need to replicate this finding before making any concrete decisions based on it. It may be that the variables chosen do not reflect the key moderating processes that are involved in any interactions with parental smoking. Other factors such as self-esteem and self-efficacy, which could be possible influences on the smoking initiation process, deserve further study as possible factors which may be involved in interaction effects. Though we should be cautious, the findings of this study do indicate potential for practical use and they deserve further study.

Implications for the Foundational Theories

The conceptual model is based on three theories which have commonly been used to guide smoking research: Social Attachment Theory, Social Learning Theory, and Protection Motivation Theory. This discussion will briefly describe how the findings relate to each of the individual theories.

Social Attachment Theory (SAT) suggests that bonds exist between an adolescent and family, friends, and social "organizations" such as churches, and that these bonds will have an impact on adolescent behavior. This occurs through the adolescent's assessment of a sense of "normativeness" of behavior which, in turn, determines the probability of a behavior being adopted. My model, partially based on SAT, suggested that this assessment of normativeness would have been influenced by adolescent development, depression, and school performance. The support found for an interaction between peer smoking and school performance also suggests some support for this theory. Peers may be the primary defining group for normativeness. Devaluation of school performance might also be a norm among certain groups of adolescents. The changes that we see among females and African Americans also suggest that social changes of normativeness may be at play here. Increased self-perception as a "good student" could be at least partially responsible for the decline in rates of smoking initiation among African American adolescents. Exploring and explaining these changes may help us in our understanding of how the issue of normativeness impacts smoking initiation.

Social Learning Theory suggests that actual modeling of behavior influences the adolescent's behavior, with the interpretation of the actual consequences of these

behaviors being a key element. In this case, seeing ones' peers smoke may result in a higher likelihood of smoking initiation in the adolescent. A recent study which may broadly relate to the use of SLT in smoking research, found that seeing smoking in movies was associated with a much higher probability of smoking initiation (Sargent, Beach, Adachi-Mejia, Gibson, Titus-Ernstoff, Carusi, Swain, Heatherton, and Dalton 2005). In this study, seeing more movies in which the star smoked resulted in a much higher incidence of smoking initiation. The study was carried out on a national sample of 6,522 adolescents, it was found that seeing these movies explained 38% of the variance in smoking initiation. Though this study supports the idea that this modeling resulted in a much higher probability of smoking initiation, it also points out some of the methodological issues which make smoking initiation so difficult to study. No note of a variable controlling for "disposable income" was included in this study. Those with more disposable income might be more likely to attend the movies and also more likely to be able to afford cigarettes. In addition, the researchers defined smoking as "ever tried a cigarette, even a puff?" This would not necessarily reflect those adolescents who went on to become regular smokers. The study was also cross sectional so that causal sequencing cannot be defined. Despite these shortcomings the researchers "doubt(ed) that there was an unmeasured confounding variable".

If we look specifically at the issue of school performance in relation to SLT, we might suggest that the likelihood of adopting smoking behavior may be dependent on how it affects outcomes related to school performance. Smoking might actually improve school performance in some cases (this may be the case if smoking is self-medication for

ADD) or, alternatively, smoking associated with assuming a more rebellious persona may reframe academic performance, diminishing its importance.

Protection Motivation Theory (PMT) would suggest that certain cognitive processes might interfere with the adolescent's ability to define a threat to one's health. The support for the impact of peer smoking being conditional on the level of school performance could be consistent with this theory. "Smarter kids" may be able to see smoking for the health threat that it is, and thus may be more able to resist the influence of their smoking peers. PMT also involves an assessment of coping appraisal. Peers might also influence the adolescent with poor academic performance to see smoking as a way to cope.

Use of any of the theories that were the foundation of the conceptual model of this study (SAT, SLT, or PMT) to plan smoking prevention programs, though common, has only limited empirical support, and this study would suggest that each element of those theories must be critically evaluated with regard to its impact on smoking initiation. Research or practical approaches that are based upon the assumption that the proposed moderating processes will alter social risk factors (such as has been seen with DARE) have not been met with the successful outcomes that they expected. In any instance, the implication is that conceptual models which are based on assumptions that perceptual or interpretative processes will moderate the influence of social variables had very limited support in this study. Approaches based on these assumptions need to continue to test them with regard to various perceptual or interpretative factors.

Main Effects

Overall, exploration for main effects yielded findings consistent with previous studies, although a few interesting differences were found as well. This discussion will address the findings for main effects for prediction (peer and parental smoking), proposed moderating variables (age, school performance, and depression), and control variables (gender, race, and family income) on the probability of smoking initiation in that order.

Main Effects of Prediction Variables – Peer and Parental Smoking

The direct effect of peer smoking behavior has been a consistent finding over many years, and this study also supports that relationship with an odds ratio of smoking initiation of 1.16 (95% CI 1.06-1.27) for each additional smoking friend. This is a relationship that has been difficult to translate into practical approaches to smoking prevention. Most educational approaches have seemed to assume that peers are important in the transition to trying cigarettes, although approaches such as DARE which have tried to counter peer influence with "resistance" education have had very limited success.

The issue of causal direction in this relationship is also not clear. As discussed in the literature review, we are not clear whether peer smoking influences the adolescent to smoke, if smokers tend to choose smoking friends, or whether some third factor causes both smoking initiation and association with smoking peers. The main effect analysis of the relationship between peer smoking and smoking initiation in this study does not, of course, provide conclusive evidence regarding the issue of causal direction, although, like many other studies, it does show that peer smoking at time 1 was clearly associated with being a smoker at time 2. This would seem to support peer influence as a cause, although

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adolescents may already have a favorable attitude to smoking at time 1, and, though their peers are not yet smoking, they may have chosen as friends peers who had a similar attitude toward smoking. Control variables did not change this relationship but other factors which were not included in the model might account for a spurious relationship.

It is because of this clearly demonstrated, but poorly understood, relationship that this study has focused on the possible impact of combinations of factors rather than specific factors, in an effort to explore under what conditions associating with peers who smoke may result in smoking initiation.

The findings for parental smoking were similar to those of previous studies which showed a positive relationship between parental smoking and smoking initiation. In this study a dummy variable which indicated whether either parent smoked had an odds ratio of 1.38 (95% CI 1.10-1.73). Similarly to peer smoking though, our greater interest is in understanding under what conditions this relationship may exist. Another issue that is subtly different between peer and parental smoking is that of causal ordering. Unlike choosing peers, adolescents who smoke cannot choose parents who are smokers, thus we would assume that parental smoking behavior is antecedent to smoking initiation. Another factor which I have not included in the model that could account for a spurious relationship between parental smoking and smoking initiation is that the relationship between parental smoking and adolescent smoking could represent some sort of genetic tendency toward substance use. There could also be a genetically determined biological factor which might increase the probability of abuse/addiction once use began. There could also be cultural or social parenting behaviors that might increase the probability of smoking initiation.

Like the findings with peer smoking, the presence of findings similar to previous studies regarding parental smoking supports the contention that this sample is similar to those previously studied, although our greatest interest is in looking at the combinations of factors.

Main Effects of Proposed Moderating Variables-Age, Depression and School Performance

The analysis found support for younger ages being more likely to initiate smoking (odds ratio 0.88 95% CI 0.83-0.93). This finding is consistent with numerous other studies. The similarity of this finding to previous studies looking at adolescent smoking initiation supports the validity of the findings, but they apply only to the 12-18 year old age group. By looking at numerous studies, we can conclude that the average age of smoking initiation is about twelve (Harrell et al. 1998; Siqueira, Diab, Bodian, and Rolnitzky 2000). This probably means that roughly half of all adolescents who initiate smoking do so before the age of twelve. Thus, when we think about these findings we may need to think of the TAPS sample which ranges in age from 12-18 as "later initiators" and consider this in any conclusions that are drawn. The factors involved in smoking initiation for earlier versus later initiators may be different.

I chose age as a potential moderating variable due to the association of age with biological, social, and emotional development, but we must also consider the possibility that the developmental issues of greatest interest in smoking initiation may not occur during the period of adolescence. Thus, exploration for similar relationships among pre-adolescents might yield different results.

This study supported depression as a factor which is antecedent to smoking initiation. The odds ratio was 1.09 (CI 1.05-1.13) so that, for each one point increase in the depression scale, there was a 9% increase in the odds of smoking initiation. As noted in the literature review, the sequencing of depression and smoking initiation is controversial. The results of the current study clearly show depression as antecedent to smoking initiation in this sample. In this analysis, depression was treated as a continuous rather than threshold variable. In using this continuous variable, support was found for depression being antecedent to smoking, a finding dissimilar to several notable studies which have treated depression as a threshold variable (Goodman and Capitman 2000). Though we cannot be sure that the difference in measurement is the only issue here, the issue of the definition and measurement of depression may be important to our eventual understanding of this phenomenon, and needs further exploration and explanation. A meta-analysis, or some other similar research approach which compares studies using continuous versus threshold measures, might be enlightening. If depression is a moderating factor, it is intuitive to think that it would have a greater effect when it reached a threshold at which it would affect perceptual or interpretative processes. We found only limited evidence for such a threshold effect, however, when looking at the data. In graphing the relationship of depression and smoking initiation, we could see that, in general, as the level of depression symptoms rose, so did the probability of smoking initiation and that this was not conclusively a threshold relationship.

With regard to school performance, this study supports earlier studies that suggested that school performance was related to smoking initiation. Most previous studies had been based on cross sectional correlations (Tyas and Pederson 1998), with the

exception of Cox, et al (2007) who had found evidence in a statewide longitudinal sample. No previous studies could be found which tested this using a national, longitudinal sample. This analysis does provide support using a large, longitudinal national sample. The mechanisms by which school performance might change the probability of smoking initiation are not fully clear, but the conceptual model suggests that low school performance may reflect an impaired ability to fully comprehend the health implications of smoking. Whether or not this is the actual mechanism by which school performance is associated with smoking initiation, it appears that school performance may be a tangible marker which can help us identify adolescents at risk, even if there are other factors which may eventually identify the relationship as "spurious". Factors, such as rebelliousness, which might also be related to school performance have been found to be associated with smoking initiation (Albers and Biener 2002; Koval and Pederson 1999) and these kinds of relationships should continue to be explored. In the interim, school performance may be a useful marker for targeting prevention efforts. It may be especially useful since it is measured on a regular basis and educators may be in a position to use prevention efforts for high risk individuals.

School performance is likely reflective of other characteristics of the individual adolescent. As has been mentioned in the literature review, there is some evidence that smoking may act as "self medication" for Attention Deficit Disorder (ADD) (Lerman et al. 2001; Whalen et al. 2003) most likely through the stimulant effect of nicotine. The relationship between school performance and peer smoking may also be indirectly related to ADD. Cigarettes may also be a form of self medication for ADD, and having peers who smoke may provide explicit or implicit motivation to use this as a coping

mechanism. Further exploration of these relationships may help us to understand these processes.

Main Effects of Control Variables- Gender, Race and Family Income

As noted in the review of literature, patterns of smoking behavior have historically varied by gender over the years, with recent data indicating that males have consistently smoked more than females, with that difference diminishing in recent years. In this sample, we find no significant difference between male and female smoking initiation rates although the actual odds ratios are still higher for males (1.22, NS), as we might have expected based on historical trends. This consistency with the previously noted trend provides an indication that this sample is reflective of the overall population. This finding regarding gender may be specific to a particular society, however. Gender roles continue to change in American society, and smoking is one area where we see this change. We should remember that this sample is drawn from the United States, and that samples drawn from other countries might have very different results. Current smoking rates in China, for example, are 62% among males and 3.8% among females (www.chinatoday.com 2008).

Analysis of this sample shows results consistent with historical trends, in that the likelihood of smoking initiation that is associated with being African American (OR 0.35, $p<0.05$), has shown a dramatic decline over time as described in the literature review. Though interesting in itself, this data also provides support for the contention that these kinds of social factors are important in explaining changes in smoking initiation rates. Though it is not clear what has caused the dramatic decline in smoking rates for African

Americans, it is not unreasonable to suggest that changes in social factors for African Americans have been important in this decline. Very few other possible explanations exist. It is not clear however, what social factors are associated with this decline, and how they have impacted smoking rates. This should be a focus of future research.

No evidence for a main effect for family income on the probability of smoking initiation was detected. This could be due to the well-known problem in social science research of getting an accurate measurement of income. This is compounded in this study by the fact that the measure is a relatively imprecise measurement. Another possibility exists, however, that family income may be becoming less important in the smoking initiation process. Traditionally, smoking initiation has been higher among those with lower income. Recent efforts at smoking prevention have had tax increases as a major focus. We would expect that tax increases would cause a greater decline in smoking rates among those with lower incomes, since discretionary items such as cigarettes would be competing with other, more essential, needs. The less-than-optimal measure of income is a limitation of this study. This issue will need to be explored in other samples with a better measure of income to determine what processes are at work.

Limitations

This study provides some useful insights but, like all research, there are issues that must be considered in its interpretation. Large public-use datasets such as this provide opportunities for multiple researchers to address questions of interest. The data, however, are collected without a specific question in mind, and thus, the data may not have all of the variables desired for a specific purpose.

Using a secondary dataset has a number of advantages but it also comes with a number of limitations. In this case, several of the variables which I have used may not have been measured in a way which optimally facilitated the study goals. In this dataset, the measure of income was less precise than we would have liked. As previously mentioned, family income was an imprecise measure thus limiting its utility in analyses. Race provided important information but its utility was diminished by the dichotomous white/black choice. One area in which a potential issue of interest was not measured was the issue of ADD. ADD has clearly been associated with a higher risk of smoking. In the literature on school performance and smoking, some have suggested that smoking could improve academic performance (Poltavski and Petros 2006). A measure of ADD in this sample might have allowed us to explore this issue.

Other measurement issues are independent of the issue of this being a secondary dataset. The definition and measurement of smoking initiation is likely to be an ongoing problem in smoking initiation research. The time at which a child or adolescent "begins to smoke" can be defined in a number of different ways. This must be considered in evaluating any study which addresses smoking initiation. The issue of depression measurement is also likely to be an ongoing issue. Depression can be thought of as either a state specific to a certain time, or a stable ongoing trait. Differences in opinion regarding the definition of depression may result in dramatically different findings. As noted, consideration of depression as existing on a continuum, versus a threshold, clinical problem also may result in quite different approaches and results.

As noted in the methods section, this survey lost 1,175 participants between time 1 and time 2, and the characteristics of those who might be lost (e.g., income) might

suggest that a higher number of participants that would have progressed to smoking might have been lost (Siddiqui, Flay, and Hu 1996).

The most important limitation of the study may be the age group that we are evaluating. This sample included 12-18 year-olds but almost half of the young people who begin smoking in the United States would already have initiated before this time. The age group in this sample may provide important information about adolescent initiators, but we should always keep in mind the fact that more than half of young people who will smoke have already initiated before the age of thirteen, when they might be thought of as pre-adolescents.

It is also possible that a third variable (an unmeasured confounding variable), which has not been included in the model, is creating the appearance of an interaction effect. A third variable that is highly correlated to one of the variables in the interaction might be the actual factor that is creating the interaction (in other words a spurious relationship). For example, if peer smoking is highly associated with something like rebelliousness, then this may be the actual factor that is involved in the interaction.

I tested for multiple interaction effects and this increases the probability of a Type I error, in which we might conclude that a relationship exists but in fact it is due to chance associations. We have no specific reason to believe this, but replication of studies increases the confidence that a relationship is real.

Recommendations for Future Research

A number of challenges exist for those who are studying smoking initiation. Smoking research may suffer from a problem that is relatively unusual in research, and

that is the perception that it has already been very extensively studied. Oftentimes, researchers want to study a unique aspect of an issue and, as a topic receives more study, this becomes progressively more difficult as more research is done. Another challenge for smoking initiation research is that the smoking initiation process appears to be an extremely complex process. Despite these difficulties, the public health implications of smoking require that we continue to study the issue.

This study would also suggest that looking at younger children might be more fruitful. Research with children comes with a number of practical issues regarding access and consent. Despite these challenges, studying younger children may help to uncover the key processes in the transition to becoming a smoker.

Future studies that might be fruitful include: more longitudinal studies with more time points, quasi-experimental studies in which students change peer groups, studies that make greater use of qualitative research methods, and studies that continue to explore other social variables including media factors.

Though this study was longitudinal, additional exploration using a dataset with more time points might allow greater options for analysis. Elements such as additional information about depression as a stable or transient characteristic would provide important information about the relationship between depression and smoking initiation. A longitudinal sample which included multiple waves (such as Add Health) could also further address the issue of how school performance and peer smoking are related temporally. If at least three time points are included, trending and sequencing can be better assessed.

Looking at students who change academic ability cohorts by choice or by chance may help define how the impact of peers varies related to academic ability. One group that might be useful to study would be students who change schools due to parental relocation. If there is a change in those students' behavior related to the change in peer group, it would be evidence for the importance of peers as a key influence, although relocation in itself would have to be considered. Looking at schools that have more mixed-ability classes, as compared to the more traditional tracking approaches, might also provide insights.

Use of qualitative or mixed methodology could also create important insights useful in understanding the relationships of interest. For example, a qualitative research approach might more effectively explore students' attitudes and beliefs about academic performance and smoking behaviors.

How factors such as the media interact with the factors included in this study may also yield additional insights. Research that supports the importance of smoking viewed in movies in the smoking initiation process (Sargent et al. 2005) could be expanded to further explore that relationship with other variables.

Continued evaluation and refinement of the theoretical models used in smoking research is essential. Studies which explore other theoretical models, such as Brofenbrenner's ecological theory (Brofenbrenner 2004), or that look at models such as Social Attachment Theory with a greater emphasis on the elements of community than were included in this study, could be used to look at "larger" social attachments. These theoretical models must constantly evaluate their assumptions with empirical findings.

Clinical and Policy Implications

One of the initial points of this study was to return to research that may help define factors which can be used in the development of "individual-based" prevention programs. This study provided confirmatory evidence for already well-known risk factors for smoking initiation such as depression, peer smoking, parental smoking, and race. It also showed the expected historical changes in risk associated with gender and race. This supports the contention that social factors continue to be important. Exploration for interaction effects provided support for only one interaction (peer smoking and school performance) consistent with the conceptual model. Though our understanding of how perceptual and interpretative processes impact vulnerability to the already identified social risk factors is clearly incomplete, the evidence for the one interaction found raises some interesting possibilities.

The existence of an interaction between peer smoking behavior and school performance will need confirmation in other studies but, if supported, might have direct implications for educators and health professionals. Educators make daily decisions regarding how groups of students will interact. If they know that placing lower performing students with known smokers increases the risk of smoking initiation, it may allow for more strategic decisions that may lower the risk of smoking initiation. As noted previously, there are also practical implications for educators who are determining whether "tracking" or "mainstreaming" approaches are to be used for at-risk students. Health professionals could ask more about smoking behaviors and school performance in "Well Child Exams". It may be possible to tailor interventions and education to these adolescents if it turns out that they are at a higher risk. Parents may also be in a position

to influence their children's choice of peers, and, if they are fully aware of all risk factors, may be able to make better decisions.

If school performance behaviors create risk that is conditional on the smoking behavior of peers, this should be considered in larger policy issues. If, in fact, lower school performers are more vulnerable to the influence of peers, the use of alternative high schools in which at-risk students are grouped together may have unintended health risks. Educators should be aware of the potential health issues in approaches in which students with several "risk factors" for smoking initiation such as being male, having problems with school performance, and having multiple smoking peers, may be combined in such a way as to create a greater risk of smoking initiation. One study of Texas alternative high schools found a 62.4% smoking rate among students (Weller, Tortolero, Kelder, Grunbaum, Carvajal, and Gingiss 1999), which is of course much higher than rates of students in traditional school settings. Though we cannot be sure of the processes which create this higher smoking rate, further exploration of potential processes can be valuable in understanding the smoking initiation process and, ultimately, in the design of effective prevention efforts. Continued efforts should be made to define the relationship between peer smoking behaviors and school performance and how this combination might put adolescents at risk for smoking initiation. Despite this need for clarification of the mechanisms involved, school performance may still be a useful marker for the student at-risk for smoking initiation, especially in relation to decisions that might impact exposure to smoking peers.

These findings need further exploration, but should also be considered in curricular approaches which cohort adolescents who are both smokers and poorer school performers. Those designing smoking prevention programs are faced with great challenges since the current research is extensive, but by no means conclusive, regarding relative risk factors or the effectiveness of prevention efforts. Continued research must be accompanied by ongoing efforts to evaluate current smoking prevention interventions and programs and efforts which are successful must be promoted.

Conclusions

This study has added to currently existing knowledge. To recap the key findings:

1. A relationship of depression as antecedent to smoking initiation in this sample was supported. Future studies should explore how findings are different when using a continuous versus threshold measure of depression.

2. A direct relationship between school performance and smoking initiation was supported. This may have potential use as a guide in designing smoking prevention interventions.

3. The existence of an interaction between peer smoking and school performance on the probability of smoking initiation was supported. Additional understanding of this relationship may allow specific actions by educators or health professionals that might result in a lower risk of smoking initiation.

4.   No other interactions were supported, contrary to the theoretical model,

suggesting that some of the current theoretical frameworks that are

being used for smoking prevention may need to be reexamined and

tested.

To date, much of the effort to address smoking related health problems has been

primarily in the area of smoking cessation – helping people who have started smoking to

quit. However, the processes by which smokers start smoking are poorly understood.

The relative lack of success of medical and public health models in preventing smoking

initiation indicates that this process is clearly more complex than it would first appear.

Initiation of smoking is likely to be multifactorial in origin. A model that includes both

social factors and individual factors associated with initiation may provide greater

opportunities for decreasing smoking rates than addressing these factors separately. In

this study, however, no evidence was found for five of the six interaction effects

involving peer or parental smoking and age, depression, or school performance as

possible moderators. This very limited evidence for the proposed model suggests that

continued development of theoretical models with documented utility is needed.

With so many diseases being caused by smoking, it is clear that reduction in

smoking rates would result in significant improvement in public health. This would of

course result in a significant reduction in health care costs. Thus, any improvement in the

understanding of the process of smoking initiation could have great practical benefits

both in terms of health and financial considerations. This study has provided additional

evidence which can be used in these efforts. The findings of this study provide evidence

which may eventually contribute to the development of smoking initiation prevention strategies. It also provides incremental progress in the understanding of some key questions in the smoking initiation process, and information from this study may be useful in the design of other studies.

Continued efforts to understand the smoking initiation process are essential. Cigarette smoking is a complex, poorly understood behavior that results in millions of deaths and billions of dollars of health care expenditures annually worldwide. The World Health Organization predicts that smoking rates will increase such that 1.6 billion people are smoking by the year 2030, and that half a billion of those alive today will die of a smoking related illness (WHO Website 2005).

As noted, mixed support of the conceptual model of this study and its foundational theories suggests that it may be appropriate for prevention models to re-examine their assumptions and move toward approaches that are more evidence based. A re-evaluation of the role of perceptual or interpretative "moderating" processes in these models is needed, and prevention programs should not be based on models that do not reflect the current evidence. Studies which clarify the impact that perceptual or interpretative processes have on the smoking initiation process and define under what conditions these occur are key to the development of effective smoking prevention programs.

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APPENDIX 1
HEALTH EFFECTS OF SMOKING

1. Cancer Of The Stomach, **
2. Cancer Of The Uterine Cervix, **
3. Cancer Of The Pancreas, **
4. Cancer Of The Kidney **
5. Acute Myeloid Leukemia **
6. Pneumonia; **
7. Abdominal Aortic Aneurysm; **
8. Cataract; **
9. Macular Degeneration **
10. Periodontitis. **
11. Cancer Of The Bladder
12. Esophageal Cancer
13. Kidney Cancer
14. Colorectal Cancer
15. Laryngeal Cancer
16. Lung Cancer
17. Oral Cancer
18. Stomach Cancer
19. Atherosclerosis
20. Cerebrovascular Disease
21. Coronary heart disease
22. Worsening Of Multiple Sclerosis
23. Erectile Dysfunction
24. Chronic obstructive pulmonary disease (COPD)
25. Pneumonia
26. Reduced Lung Function In Neonates
27. Impaired Lung Growth
28. Asthma Related Symptoms
29. Sudden infant death syndrome (SIDS)
30. Reduced Fertility In Women And Men
31. Fetal Growth Restriction
32. Low Birth Weight
33. Premature Rupture Of The Membranes,
34. Placenta Previa,
35. Placental Abruption.
36. Preterm Delivery And Shortened Gestation
37. Enhance Transmission Of HIV To Fetus
38. Risks For Adverse Surgical Outcomes Related To Wound Healing And Respiratory Complications
39. The Evidence Is Sufficient To Infer A Causal Relationship Between Smoking And Hip Fractures
40. Low Bone Density
41. Peptic Ulcer Disease In Persons Who Are *Helicobacter Pylori* Positive

(from the Surgeon General's Report 2004 and the World Health Organization)
** = Diseases identified in the 2004 report to be caused by smoking that were not previously causally associated with smoking

APPENDIX 2

Table 1: Studies suggesting that Depression precedes Smoking Initiation

Author	Journal and Date	"theory"	Findings	Sample	
Anda, et al (Anda, Croft, Giles, Williamson, Giovino, Felitti, and Nordenberg)	JAMA, 2000	Adverse childhood events predict smoking	If adverse childhood events risk of ever smoking increased (OR, 3.1; 95% CI, 2.6-3.8)	9,215 adults average age 53 female and 58 male	Self report of pre-existing depression
Carvajal, et al	Journal of App. Social Psychology, 2004	Depression increases vulnerability	Depression predicted SI in those who had not yet smoked	2,004 US Middle School Students	Cross sectional-correlation with intent to smoke
Covey and Tam	AJPH, 1990	Depression results in smoking	Depression scores correlated with number of cigarettes smoked	205 eleventh graders	Early study to suggest that depression resulted in smoking based on cross sectional correlation
Escobedo	Addiction, 1996	Depression results in smoking	Increased risk of smoking initiation		
Kandel and Davies	Archives of General Psychiatry, 1986	Depression results in smoking	Increased risk of smoking initiation		Longitudinal - Adolescents with depressed mood at age 15-16 were reassessed 9

					years later
Koval and Pederson	Preventative Medicine, 2004	Depression (stress) results in smoking	Increased risk of smoking initiation but differences between males and females and older and younger adolescents	1,598 Canadian sixth graders	Cross sectional but data suggests self-report of self medication
Lenz	Journal of American College Health,2004	Depression history predicts smoking	Seven fold risk of SI	203 Freshman and sophomore US college students	Cross sectional
Lerman	Health Psychology, 1998	Cigarette smoking is self-medication for depression	Those with an abnormal DRD4 gene and depression had higher SI	231 Smokers	Cross sectional
Nezami	Nicotine and Tobacco Research, 2005	Depression results in higher SI in multiple ethnic groups	Depression associated with intent to smoke	800 seventh graders among an ethnically diverse population in Los Angeles	Cross sectional
Nichols	Journal of Epidemiology and Community Health, 2004	Not depression per se but physical or sexual abuse results in SI	Physical and sexual abuse predicted SI	722 women ages 36-45	Depression as mediating variable-self report
Orlando, et al	Journal of Consulting and Clinical Psychology,	Depression in 10th grade results in higher smoking	Hypothesis supported	2,961 adolescents	Longitudinal

	2001	probability in Grade 12			
Patton and Carlin	AJPH, 1998	Depression results in smoking experimentation	Hypothesis supported	2032 Australian 14 and 15 year olds	Longitudinal
Repetto	Health Psychology, 2005	Depression history predicts smoking	Depression predicted later cigarette use with greater affect for males	623 African American Adolescents	Longitudinal
Tomori	European Child and Adolescent Psychiatry, 2001	Descriptive study correlating psychosocial factors with SI	SI correlated with previous abuse or suicidal thoughts	2111 High school students	Cross sectional

Appendix 2: Table 2: Studies suggesting that Smoking Initiation precedes onset of Depression

Author	Journal and Date	"theory"	Findings	Sample	
Brook, Shuster, et al	Psychological Reports , 2004	Early cigarette smoking leads to later depression	Smoking during adolescence predicts depression rates in late twenties	688 adolescents surveyed over 13 years	
Goodman and Capitman	Pediatrics, 2000	Smoking precedes depression	Smoking predicted later depression when 18 other variables controlled	8,704 adolescents from the National Longitudinal Study of Adolescent Health	Analysis limited to those who were not depressed at time 1
Stein	Journal of Applied Social Psychology, 1996	Descriptive	Increased depression at times 2,3, and 4 among smokers	461 seventh through ninth graders assessed every four years	
Steuber and Danner	Addictive Behaviors, 2005	Smoking increases the probability of depression	Odds increased by about 50% for the probability of developing depression from time 1 to time 2	Add health 14,634 Adolescents	
Wu	AJPH, 1999	Smoking precedes depression	Modestly increased risk of depression after smoking	1731 youth ages 8-14	

Appendix 2: Table 3: Studies suggesting that Depression and Smoking Initiation are Reciprocal

Author	Journal and Date	"theory"	Findings	Sample	
Chang, et al	Journal of Adolescent Health, 2005	Depression and smoking co-exist	Correlation found	486 adolescents	
Coogan, et al	American Journal of Preventative Medicine, 1998	Correlation of depression and all risky behaviors (alcohol, smoking, sexual)	Hypothesis supported	31,861 children ages 9-19 in Connecticut	
Johnson	Nicotine and Tobacco Research, 2004	Shared familial risk	Family history of depression and parental smoking explained 73-95% of the variance in the relationship between depression and smoking	979 young adults in the US ages 26-35	
Wang	Psychological Reports, 1996	Exploratory	Suggest support for mutual causality	7,960 Teenage Attitudes and Practices Survey	

Appendix 2: Table 4: Studies supporting Confounding Variables

Author	Journal and Date	"theory"	Findings	Sample	
Albers and Biener	Preventative Medicine 2002	Rebelliousness may explain both smoking and depression	Predictive effect of smoking disappears when controlling for rebelliousness	N=522 Massachusetts Adolescents	
Koval and Pederson	Addictive Behaviors 1999	Rebelliousness and stress may explain both smoking and depression	Support for rebelliousness in both males and females	1,552 Canadian 11 and 12 year olds	
Koval and Pederson	Preventative Medicine 2004	Rebelliousness, level of mastery, and social conformity may explain both smoking and depression	Support for cross sectional correlations with correlations stronger in older adolescents	1,543 Canadian 6th and 8th graders	
Breslau and Peterson	Archives of General Psychiatry, 1998	Major depression correlated with smoking	OR 3.0 5 year long study	1,007 young adults	"May be shared etiologies"
Jarvelaid	Scandinavian Journal of Primary Health Care, 2004	Cross sectional correlations	Smoking correlated with depression and both correlated with family characteristics	977 schoolchildren ages 14-18 in Estonia.	

APPENDIX 3

TAPS smoking status recode

Smoking Status	Description
Never smoked, don't know type (coded 0)	Never smoked a cigarette, never tried or experimented with cigarettes, unknown if they will try a cigarette soon and/or unknown if they will be smoking one year from the time of the interview
Never smoked, no intention (coded 1)	Never smoked, never experimented, will not try a cigarette soon and will definitely not be smoking one year from the time of the interview
Never smoked, contemplator (coded 2)	Never smoked, never experimented, may try a cigarette soon and/or may be smoking one year from the time of the interview
Current regular smoker, light (coded 3)	Smoked 10-30 days in the past 30 and smoked less than five cigarettes each day
Current regular smoker, heavy (coded 4)	Smoked 10-30 days in the past 30 and smoked five or more cigarettes each day
Current occasional smoker (coded 5)	Smoked 1-9 days in the past 30
Current regular smoker, don't know type (coded 6)	Smoked 10-30 days in the past 30 and number of cigarettes smoked each day is unknown
Experimenter (coded 7)	Smoked or tried a cigarette but has not smoked 100 cigarettes and has not smoked in the past 30 days
Former smoker (coded 8)	Smoked 100 or more cigarettes but has not smoked in the past 30 days

APPENDIX 4

Description of multistage sampling process of the TAPS and variables used in this analysis

The following information is primarily taken from "Vital and Health Statistics: Design and Estimation for the National Health Interview Survey, 1985-94, Series 2: Data Evaluation and Methods Research: No. 110" (NCHS 1989) and additional information about the sampling process can be obtained from that document.

This study utilized the Teenage Attitudes and Practices Survey (TAPS) which was a supplement to the 1989 and 1993 National Health Interview Surveys (NHIS). The NHIS utilizes a stratified, multi-stage probability sampling process which is designed to reflect the overall noninstitutionalized U.S. population. The sampling process for the NHIS is reevaluated at intervals to assure that it is obtaining the best sample possible.

For the NHIS years of interest, those that included the TAPS, data were collected as a multistage probability sample using Primary Sampling Units (PSUs) that were defined using the most recent census. These PSUs were primarily those areas defined as Metropolitan Statistical Areas (MSAs). For areas that were not defined as MSAs county level data were used, or if the county was small adjoining counties were combined to create a comparable sample. These PSUs were then stratified based on certain criteria to assure that the sample obtained was representative of the population at large. During the 1985–94 design, NHIS PSUs were stratified based on geography, age, sex and gender, but were also stratified by factors felt to reflect health status. The publication noted above explains this process: "The best stratifiers would have been health variables, but health statistics were available only for sample PSU's and could not be used as stratifiers. Instead, variables that were highly correlated with health variables were sought for stratifiers." (NCHS 1989, p. 22). The stratification variables thus identified were the following: Hispanic; persons below poverty level; households with income less than $15,000; persons in urban areas; unemployed persons; and persons employed in manufacturing. Certain subgroups that are thought to be important in understanding health differences within the population were oversampled specifically those who were black, Hispanic, aged, and low income.

Several different options are provided in the TAPS to adjust for the PSU and Stratification factors depending on the software used and the research design. Based on the recommendation of a technical specialist from the NHIS (personal communication with Veronica Benson, September 2004) the variables CPSU and CSTRATUM were used. The recommendation to use these particular variables when using Stata to analyze the NHIS is also supported by a CDC publication (**http://www.cdc.gov/nchs/data/series/sr_02/sr02_110.pdf**). The data were also analyzed using PSU and STRATUM, as the psu and strata variables with similar findings

being obtained for the two main effects and the interaction effects of primary interest in this study.

The third element necessary for analysis of this type of sample is a probability weight. This weight defines the relative probability of a given subject being included in the sample. For the NHIS during the time period of interest this weight was created using a four step process. These four steps included: the inverse of the probability of selection based on the PSU and stratum variables, a household nonresponse adjustment, a first stage ratio adjustment which considers racial and residence factors, and finally, a poststratification adjustment based on age, sex, and race. The weight recommended by the NHIS technical specialist was CFINALWT (mean 2550.8, range 426-10204) as this is the weight best reflecting the characteristics of those adolescents participating in the computer assisted telephone interview (CATI).

The following is the output of the Stata command "svydes" which provides a description of the weight, primary sampling unit, and strata variables which were used along with the characteristics of each stratum.

```
svydes

pweight:   CFINALWT
Strata:    CSTRATUM
PSU:       CPSU
```

| | | | #Obs per PSU | | |
Strata CSTRATUM	#PSUs	#Obs	min	mean	max
1	4	181	30	45.2	53
2	4	41	5	10.2	19
3	4	33	4	8.2	11
4	4	59	13	14.8	17
5	4	45	4	11.2	17
6	4	39	3	9.8	15
7	4	22	2	5.5	9
8	4	57	5	14.2	21
9	4	41	7	10.2	17
10	4	54	10	13.5	17
11	4	113	18	28.2	39
12	4	48	6	12.0	21
13	4	27	4	6.8	9
14	4	70	13	17.5	22
15	4	49	10	12.2	15
16	4	62	8	15.5	31
17	4	41	6	10.2	19
18	4	48	9	12.0	16
19	4	31	5	7.8	11
20	4	52	7	13.0	19
21	4	45	10	11.2	12
22	4	48	8	12.0	19
23	4	51	4	12.8	20
24	4	46	9	11.5	14
25	4	54	8	13.5	18
26	4	60	12	15.0	16
27	4	45	6	11.2	15

28	4	39	5	9.8	12
29	4	44	6	11.0	13
30	4	26	4	6.5	10
31	4	32	2	8.0	15
32	4	48	6	12.0	18
33	4	52	4	13.0	18
34	4	42	5	10.5	15
35	4	35	7	8.8	12
36	4	34	5	8.5	12
37	4	30	6	7.5	9
38	4	40	6	10.0	17
39	4	39	2	9.8	16
40	4	52	8	13.0	18
41	4	52	7	13.0	16
42	4	33	7	8.2	11
43	4	32	4	8.0	12
44	4	29	3	7.2	13
45	4	23	4	5.8	8
46	4	60	10	15.0	20
47	4	23	4	5.8	8
48	4	57	10	14.2	18
49	4	19	2	4.8	8
50	4	27	3	6.8	12
51	4	55	11	13.8	16
52	4	139	20	34.8	51
53	4	39	4	9.8	20
54	4	36	6	9.0	14
55	4	31	5	7.8	11
56	4	53	7	13.2	20
57	4	87	7	21.8	35
58	4	42	5	10.5	13
59	4	48	6	12.0	17
60	4	39	3	9.8	21
61	4	26	2	6.5	13
62	4	41	2	10.2	22
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62	248	2966	2	12.0	53

APPENDIX 5

IRB Approval

UNIVERSITY of NEW HAMPSHIRE

April 3, 2006

Jeffrey Eaton
Sociology, Horton SSC
381 Main Street
Springvale, ME 04083

IRB #:   3693
Study: The Influences of Depression and Peer Smoking on Smoking
                Initiation: Main and Interacting Effects
Approval Date: 04/03/2006

The Institutional Review Board for the Protection of Human Subjects in Research (IRB) has reviewed and approved the protocol for your study as Exempt as described in Title 45, Code of Federal Regulations (CFR), Part 46, Subsection 101(b). Approval is granted to conduct your study as described in your protocol.

Researchers who conduct studies involving human subjects have responsibilities as outlined in the attached document, *Responsibilities of Directors of Research Studies Involving Human Subjects.* (This document is also available at http://www.unh.edu/osr/compliance/irb.html.) Please read this document carefully before commencing your work involving human subjects.

Upon completion of your study, please complete the enclosed pink Exempt Study Final Report form and return it to this office along with a report of your findings.

If you have questions or concerns about your study or this approval, please feel free to contact me at 603-862-2003 or Julie.simpson@unh.edu. Please refer to the IRB # above in all correspondence related to this study. The IRB wishes you success with your research.

For the IRB,

Julie F. Simpson
Manager

cc: File
        Heather Turner

Research Conduct and Compliance Services, Office of Sponsored Research, Service
Building, 51 College Road, Durham, NH 03824-3585 * Fax: 603-862-3564