ASSOCIATIONS AND PREDICTORS OF DEPRESSIVE SYMPTOMS AND SMOKING AMONG ADOLESCENTS IN UNITED STATES

by

BONNIE CHOI MO

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ABSTRACT

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Bonnie Choi Mo, MSSW

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Supervising Professor: Dr. Sung Seek Moon

Studies done on the association of smoking and depression found inconclusive results in the direction of the association. The present study utilized public data from the Add Health to examine the associations between depressive symptoms and smoking concurrently and prospectively in adolescents, predictors of current smoking, and associations between concurrent depressive levels and smoking cessation status.

The results indicate depressive symptoms promote concurrent and prospective smoking behavior. Higher levels of depressive symptoms at Wave I was characteristic of participants who smoked in the past and smoked regular. Higher depressive symptoms in Wave II were indicative of smoking initiation and regular smoking in Wave II. Also, depressive symptoms and age predicted current smoking status. Results did not find significant differences in depressive symptoms and smoking cessation.

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CHAPTER 1

INTRODUCTION

Adolescence signifies a dynamic period of growth that can be enjoyable and enriching, tumultuous and challenging, or a combination of both for many adolescents. During this time of development, adolescents' emotions and behaviors are more susceptible to influence by internal (physical) and external (social and environmental) factors than adults. Therefore, it is important to examine factors that affect their wellbeing and two such factors smoking and depression. Over the past three decades, considerable studies have been done on the area of smoking and depression, and some of these studies have examined the adolescent population. Research has suggested a strong association between smoking and depression (Fergusson, Goodwin, & Horwood, 2003; Goodman & Capitman, 2000; Kendler et al., 1993; Kendler, Neale, MacLean, & Heath, 1993; Lam et al., 2005; Lewinsohn, Rohde, & Seely, 1998; Nezami et al., 2005; Steuber & Banner, 2006; Upadhyaya, Deas, Brady, & Kruesi, 2002; Wu & Anthony, 1999). However, the direction of the association remains debatable. Some studies found that an increase in smoking increases depressive symptoms (Goodman & Capitman, 2000; Wu & Anthony, 1999) while others found that increased depressive symptoms increases smoking (Covey & Tam, 1990; Kandel & Davies, 1986; Lam et al., 2005; Nezami et al., 2005; Patton G., Hibbert M. Rosier M., Carlin J., Caust J., & Bowes G., 1996; Patton et al., 1998). In light of the inconsistency, this study hopes to provide further insight toward this ongoing directional association debate and shed light on the effects of depression and smoking comorbidity on smoking cessation. Using the data from waves I and II from the 1994-1996 National Longitudinal Adolescent Health Study, this study examines the associations between depressive symptoms and smoking concurrently and prospectively in adolescents of different races in America, whether levels of depressive symptoms, gender, and age predicts smoking, and associations between concurrent depressive levels and successes in smoking cessation.

1.1 Smoking

Nicotine, in its chief form of tobacco use, is one of the most addictive drugs and the leading preventable cause of death. In 2000, tobacco use attributed to approximately 435,000 deaths (18.1% of total death) and was the primary cause of death in United States (Mokdad, Marks, Stroup, & Gerberding, 2004). This cause of death could largely be prevented if people chose not to smoke. Even though smoking related deaths tend to occur after 35, smoking initiation usually takes place during adolescence (Rivara et al., 2004). The National Survey on Drug Use and Health found that 29.4% of Americans age 12 and older used tobacco products at least one time in the month prior to interview (National Institute of Drug Abuse, 2007). Of that group, 13.1 % of adolescents age 12 to 17 used some type of tobacco products. Furthermore, the Center of Disease Control and Prevention found that 23% of high school students and 8% of middle school students are current smokers (Center of Disease Control, 2006). These statistics are alarming because 89% of adult daily smokers initiated smoking by or at the age of 18, 71% of those adults initiated smoking daily by or at the age of 18, and almost

no adult initiated smoking after the age of 25 (Centers for Disease Control and Prevention, as cited in McMahon, 1999; Rivara et al., 2004). Adolescent smoking has serious consequences that last well into adulthood. Therefore, effective smoking cessation and prevention programs for the adolescent population are crucial and essential in decreasing the number of young smokers.

1.1.1 Physical and Behavioral Effects of Smoking

Smoking has detrimental effects on adolescents' physical and behavioral health. Several studies have examined the health consequences of smoking in adolescence. Brook, Brook, Zhang, and Cohen (2004) conducted a prospective longitudinal study to examine study the association between lifetime tobacco use and health problems by 30. 749 participants were interviewed at mean ages of 14, 16, 22, and 27. The study found that daily tobacco use during any of the four ages predicts respiratory issues, neurobehavioral and cognitive problems, and general sickness by the age of 30.

Mathers, Toumbourou, Catalano, Williams, and Patton (2006) conducted a literature search on longitudinal adolescent tobacco studies with large representative samples and found that adolescent tobacco use predicts alcohol use, mental health issues, adverse behaviors such as (poor academics, crime, and early parenthood), and poor sleep and health problems. Adolescent smokers are prone to developing respiratory infections, becoming short-breathed, and experiencing more asthma and allergy symptoms than their non-smoker counterparts (Working Party on Smoking and the Young, Adams et al., and Townsend et al. as cited in Mathers, Toumbourou, Catalano, Williams, & Patton, 2006).

A number of papers have indicated strong links in psychiatric comorbidity and cigarette smoking (Breslau, 1995; Lasser et al., 2000; Mathers et al., 2006; McMahon, 1999; Upadhyaya et al., 2002). Upadhyaya et al. (2002) conducted a literature search on this area in adult and child and adolescent studies and found that adolescent smoking have some association with psychiatric disorders. The study found a link between smoking and attention-deficit hyperactivity disorder, anxiety disorder, and drug and alcohol abuse/dependence and it found a strong link between oppositional defiant disorder, conduct disorder, and major depression. Lasser et al. (2000) found similar results in the analysis of 4411 respondents from the National Comorbidity Survey. The study found that respondents with mental illness are twice as likely to smoke as respondents without mental illness. Breslau (1995) examined data from 1000 young adults in an epidemiological study and found that people with nicotine dependence had a higher chance for alcohol and substance abuse and dependence, major depression, and anxiety disorders than non-nicotine dependent smokers and non-smokers. The strong link between psychiatric comorbidity and smoking in these studies indicate the need to address tobacco use and psychiatric disorder simultaneously in the adolescent population. One of the most common psychiatric disorders that occurs across the life span but is becoming a serious health issue is depression.

1.2 Depression

Depression affects approximately 14.8 million adults or 6.7% of the U.S. population in a given year and is the primary cause of disability for ages 15-44 (National Institute of Mental Health, 2006). Garrison et al. (as cited in Keyes, 2006)

pointed out close to 1 in every 10 children has one episode of major depression before their 14th birthday. Lifetime prevalence of depression during adolescence is approximately 15-20% and can affect 5.8% of adolescents in a given time (Kessler & Walters, 1998; Kubik, Lytle, Birnbaum, Murray, & Perry, 2003). Lewinsohn, Rohde, & Seely. (1998) indicated that 28% of adolescents will experience an episode of depression before age 19. Kessler and Walters (1998) found that 73.9% of the adolescents who participated in the National Comorbidity Survey have recurrent episodes, and 36.3% of the participants reported that their depression hindered them from doing activities and living their lives. Of those participants that were depressed, only half talked to a professional about the depression and only a small group of those actually received professional help. These statistics point out that depression is a major health problem, and the adolescent population is highly susceptible to depression, which can have lasting affects into adulthood.

Depression in adolescents can appear differently than from adults, and thus can be difficult to detect. In addition, adolescent can have difficulty describing their symptoms to match the DSM IV criteria for depression, so it is important to talk to family and other sources to assess comprehensively. Depression in children and adolescents can manifest as irritability and not gaining expected weight (Lagges & Dunn, 2003). According Lagges and Dunn, adolescents must meet five of the following criteria for at least two weeks to be diagnosed with a major depressive disorder: depressed or irritable mood, anhedonia, weight disturbance, change in appetite, change in sleep, psychomotor retardation or agitation, fatigue, feelings of worthlessness or guilt, difficulty concentrating, suicide ideation, and suicide. Also, adolescents often do not meet full diagnostic criteria for depression, and those adolescents can fall through the cracks and not receive help to manage their depression. Mental health professionals need to be mindful of the differences in diagnosing adolescent and adult depression and provide the appropriate treatment accordingly.

Studies have shown depression predicts and is associated with risky behaviors. Depression in teenage years is a predictor of academic issues, teenage pregnancy, heightened risk for substance use, suicide, and poor psychosocial functioning (Kubik et al., 2003). According to Deykin et al. and Kandel and Davies (as cited in Birmaher & Ryan, 1996), adolescence is associated with suicidal and homicidal ideation, tobacco use, alcohol and other illegal drugs abuse. Birmaher and Ryan noted a 4.5 year gap between depression and the onset of alcohol and substance abuse, and this time can be an excellent opportunity for prevention with depressed teens. Aside from depression, a higher level of depressive symptoms also predicts substance use, lower academic functioning, poor psychosocial skills, thoughts of suicide, and depression in adulthood (Kubik et al., 2003). These studies suggest the need for adolescent treatment for depression to reduce symptoms and in turn decrease risky behaviors.

1.3 Depression and Smoking

Depression and smoking have been strongly linked by numerous studies.(Breslau, 1995; Escobedo, Reddy, & Giovino, 1998; Fergusson et al., 2003; Goodman & Capitman, 2000; Kendler et al., 1993; Lam et al., 2005; Lewinsohn et al., 1998; Martini, Wagner, & Anthony, 2002; Mathers et al., 2006; Nezami et al., 2005; Steuber & Banner, 2006; Upadhyaya et al., 2002; Wu & Anthony, 1999). Despite the strong association from the wealth of studies, the direction of association remains unknown. To date, there is no consensus on the direction of association for depression and smoking. Some studies found that smoking leads to depression (Goodman & Capitman, 2000; Martini et al., 2002; Steuber & Banner, 2006; Wu & Anthony, 1999), and others found that depressive symptoms influence smoking (Covey & Tam, 1990; Escobedo et al., 1998; Kandel & Davies, 1986; Lam et al., 2005; Martini et al., 2002; Nezami et al., 2005; Patton G. et al., 1996; Patton et al., 1998).

Two studies conducted secondary data analysis on two separate sets of data (Martini et al., 2002; Wu & Anthony, 1999). Martini examined cross-sectional data of 13,826 adolescents from National Household Survey on Drug Abuse. The study categorized participants into one of three smoker categories: never, former, and current smoker, and assessed for depression by utilizing eight self-reported questions from the Youth Self Report. The findings show that current smoker have higher levels of depression when compared to former and never smoker, and depression level decreased as time elapsed from last smoking. Although it is possible that smoking cessation can be related to a decrease in risk of depression associated with smoking, the cross-sectional research design cannot examine causation in time. Wu and Anthony (1999) conducted secondary data analysis on the longitudinal data from Prevention Research Center at Johns Hopkins University conducted in the mid-Atlantic region from mid 1980s and 1990s. The study collected data related to depression and tobacco use on 1731 youths.

subsequent result and tobacco smoking as the antecedent and vice versa. The study concluded that smoking indicated a slight risk for subsequent depressed mood, but several limitations existed. For one, the study used a sample in mid-Atlantic region which restricts the generalizability of the results to all adolescents in United States. Secondly, the study excluded all youth with current depressed moods from the study in order to assess for new cases of depression within the year of the study, and also excluded all youth with current tobacco use in order to assess for new cases of tobacco in the study. In other words, this study only examined smoking initiation and depression onset, but not the effects that current depression and smoking cessation have on participants. These analyses only lend insight to current comorbidity in the year sampled but not prospective analysis.

Two studies examined the National Longitudinal Study of Adolescent Health (Addhealth) data and found that smoking can lead to depression (Goodman & Capitman, 2000; Steuber & Banner, 2006) Goodman and Capitman first examined 8704 non-depressed adolescents at baseline for tobacco use development of at least 1 pack of cigarettes per week at 1 year follow-up. Secondly, 6947 non-smokers for the past 30 days at baseline were assessed for developing high depressive symptomatology. Results from multivariate modeling found current smoking predicted high depressive symptoms and high depressive symptoms did not predict heavy smoking. However, bivariate analyses showed that high depressive symptoms did increase the risk of heavy smoking by three times. Similar to previous studies, this study only examined concurrent

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symptoms but not prospective symptoms. In addition, this study did not take into account that varying levels of smoking can also affect levels of depressive symptoms.

Steuber and Danner (2006) also used Addhealth to examine the association of depression and smoking. Participants were categorized into four smoking status groups: never, starter, quitter, and maintainer. A modified version of CES-D was used to measure depression, and the score of 24 and 22 were used as depression cut-offs for females and males, respectively. Results indicate that the starters, quitters, and maintainers were 1.5, 1.4, and 2.0 times likelier to be depressed than the nevers at time 2, and regular smokers are more likely to show higher levels of depression. Although these results indicate that smoking predicts depression, it could not prove that smoking leads to depression. In addition, the cut-offs scores from CES-D only predicted depression but could not diagnose.

The studies that found smoking increases due to depressive symptoms all had different limitations due to restricted generalizability, lack of research on prospective associations, narrow focus on only high levels of depression, and using predictive depression cut-off scores. This current study hopes to address these issues to provide more insight to the debate on smoking and depression.

Although some studies found that smoking promotes depressive symptoms, other studies found the depressive symptoms promotes smoking (Covey & Tam, 1990; Escobedo et al., 1998; Kandel & Davies, 1986; Kubik et al., 2003; Lam et al., 2005; Martini et al., 2002; Nezami et al., 2005; Patton G. et al., 1996; Patton et al., 1998).

Escobedo et al. (1998) and Lam et al. (2005) in particular, by a conducted secondary data analysis on two separate longitudinal studies.

Escobedo et al (1998) analyzed data from the Teenage Attitudes and Practices Survey collected from 7885 adolescents between the ages of 12-18. The depressive symptoms scale created by Mellinger was used to assess depressive symptoms and 21.8 was the cut-off value for depression. Questions related to their smoking use categorize participants into different groups: occasional smokers, regular smokers, heavy smokers, non-smokers, and quitters. Results found that depressive symptoms increase the likelihood of smoking during adolescence. However, the Mellinger scale has shown to detect greater incidences of depressive symptoms than clinical diagnoses, so it is questionable whether the scale over-reported depressive symptoms.

Lam et al. (2005) conducted a prospective longitudinal study with 1894 7th grade students in Hong Kong with two waves. Anonymous self-reported surveys were utilized, and those students were reassessed 12 months later (Time 2). The students were categorized by smoking status ('never smokers,' 'ex-smokers,' and 'current smokers') and depressive symptoms ('low depressive symptoms' are students with scores lower than the 75th percentile and 'high depressive symptoms' are students with score higher than the 75th percentile). Students who were smokers and ex-smokers were further categorized by their smoking cessation status. Current students who never tried or wanted to quit smoking were put in the 'non-dissonant smokers' group, students who were current smokers who attempted to quit but were unsuccessful were classified as

'unsuccessful quitters.' Lam et al. found that 'never smokers' with high depressive symptoms predicted 50% likelihood of being a smoker at Time 2, and ex-and current smokers in Time 1 had higher depressive symptoms than never smokers at Time 2. Among the groups, persistent smokers had the highest depressive symptoms followed by new smokers. The relationship between depressive symptoms and difficulty quitting cigarette smoking was also assessed. After 12 months, unsuccessful and non-dissonant smokers had higher depressive symptoms than successful quitters. Although Lam et al. suggests that depressive symptoms affect smoking, this study was done in only one grade level and in Hong Kong. Therefore, the generalizability of the study particularly to adolescents in United States is questionable due not only to its geographic location but also due to cultural factors. The vast majority of the ethnic make-up in Hong Kong is Chinese in contrast with the ethnic diversity in the United States.

In addition to longitudinal studies confirming the association of depression promoting smoking, other studies on smoking and psychiatric comorbidity also affirms this association(Kubik et al., 2003; Upadhyaya et al., 2002). For example, Kubik et al. conducted a cross-sectional survey with 3621 seventh grade students from 16 middle schools. The study used Center for Epidemiological Studies Depression scale (CES-D) to assess for depressive symptoms used two measures for assessment of smoking (one for monthly smoking and one for weekly smoking). Results found that monthly smoking was independently associated with elevated symptoms especially among girls.

1.4 Benefits of the Current Study

In sum, the literature provides a wealth of information on depression and smoking, but limited insight on how depression and smoking associate with each other in practice. Furthermore, research in this well-studied area has been conducted in a myriad of methods such that there is no consensus on how this area is studied, and this can be a factor of the discrepancy in the findings in this area.

All these discrepancies in the area indicate a clear need to continue to explore the association between depression and smoking. With this issue in mind, this current study will examine the associations between depressive symptoms and smoking concurrently and prospectively in adolescents in America, evaluate depressive symptoms according to different group's smoking status, and assess associations between concurrent depressive level and success in smoking cessation.

This current study will utilize a prospective longitudinal study from Lam et al. (2005) as its framework to assess adolescents of different ages in various areas of the United States. The current study will analyze data from the first and second waves of the National Longitudinal Study of Adolescent Health (Addhealth) from 1994-1996. This research design clearly separates the experimental, current, and past smokers. This is crucial because failure to separate participants by smoking status can result in confounding factors interfering with the group. Examining each group of smokers separately ensures that each course of smoking can be studied independently. In addition, resurveying after approximately a year allows researchers to identify new smokers without risking the current smokers to be affected by extraneous depressive

symptoms from continual tobacco use. More importantly, this study uses a nationwide longitudinal sample that comprises of a large sample size that is representative of the adolescent population across age and race. This large population sample will increase the generalizability and decrease biases toward one group of adolescents over another. Although two other studies have been done using this data set and found smoking increases the likelihood of depression, this study's design differs from both in separating smoking status and treating depressive symptoms as a continuous variable. Participants who scored 75% percentile or above are placed in the high depressive symptoms group. In this way, the participants' symptoms are relative to the other subjects in the study group.

This study proposes that depressive symptoms increase the likelihood of smoking in adolescents because of the self-medication theory (Breslau, 1995; Escobedo et al., 1998; Kandel & Davies, 1986; Lam et al., 2005). Longitudinal studies from Lam et al. (2005) and Escobedo et al. (1998) mentioned earlier provides strong evidence for depression promoting smoking through this theory. Cigarettes contain a substance that acts as a monoamine oxidase (MAO) inhibitor similar to the first generation of antidepressants such as Nardil, Parnate, and Marplan (Klimek et al., 2001). Cigarettes also contain nicotine, which blocks nicotinic receptors and exhibited anti-depressant (Schelling; Brodkin et al.; Ferguson, Brodkin, and Lloyd; as cited in Klimek et al., 2001). Klimek et al. examined the effects of cigarette smoking and antidepressant drugs and found that cigarette smoking affects the noradrenergic proteins in the locus coeruleus in the human brain, similar to the effects of antidepressants in chronic

smokers. This finding suggests that smoking-induced changes in the locus coeruleus might strengthen smoking in depressed participants. A study on French depressed inpatient smokers reported patients were more likely to smoke in negative situations regardless of dependence (Carton, Jouvent, & Widlöcher, 1994). Carton also found nicotine dependence correlates with smoking to improve emotions. Because depressed adolescents have the tendency to smoke to improve their mood, they might smoke to elevate depressive symptoms and the addictive chemical changes can reinforce the nicotine dependence. Thus, smokers can become caught in a cycle of smoking to alleviate existing depressive symptoms and feeling depressed when they attempt to quit. In theory, smoking can potentially help a depressed individual to feel less depressed, but the harmful effects of smoking hurt more than help an individual's mental health.

1.5 Hypotheses

This present study will examine the association and predictors of depression and smoking. The study hypothesizes the following:

- 1. High levels of depressive symptoms will increase current and prospective smoking predisposition levels regardless of smoking status.
- 2. Depressive symptoms predict current status as a smoker.
- 3. Current depressive symptoms predict difficulty in smoking cessation.

CHAPTER 2

METHOD

2.1 Add Health Study Overview

The National Longitudinal Study of Adolescent Health (Add Health) studied the health status of teenagers, investigated causes of health-related behaviors, and examined environment effects on adolescents (Kelley & Peterson, 1997). The Add Health research design revolved around three factors that effect adolescent health: social environment (from family to community), health-related behaviors, and vulnerabilities and strengths that environment and behaviors can assert on the individual. The study recognizes that the individual and the individual's environment interact and affect each other. The study uses school as the hub for recruitment to provide easy access to participants as well the peers of those participants.

The Add Health study collected self-reported data related to adolescent behaviors and outcomes (Udry, 1998). The data set from baseline to follow-up used various questions and instruments to gather information through the in-school and inhome interviews on adolescent's physical, emotional, and mental health as well as social behaviors and environment. The current study utilized the public data from Add Health for analysis (Udry).

2.1.1 Adolescent: In-school questionnaire

The questionnaire was administered from September 1994 through April 1995 to 7th to 12th grade students. The in-school questionnaire consisted of questions about demographics, parental background (education level, occupation, and more), and health-related behaviors. The self-report was designed for optical scanning since participants were only given a 45- to 60-minute class period to complete it.

2.1.2 Adolescent: In-home interview.

Interviewers conducted in-home interviews on computer laptops. The two portions, Computer-Assisted Personal Interview and an Audio Computer-Assisted Self-Interview made up the entire in-home interview. Participants answered questions on a wide range of topics including demographics, academics, parental relationships, friendships, sexual behaviors and delinquent behaviors. The Computer-Assisted Personal Interview utilized the computer to prompt the interviewer with questions, and the interviewer in turn entered the data directly into the laptop. The Audio Computer-Assisted Self-Interview helped the interviewer to administer sensitive questions related to sexual activity, drug and alcohol use, fighting, suicide, and more. Students listened to prerecorded questions via earphones before entering their responses directly into the laptop. This design helped to increase confidentiality and decrease the chances of fictitious answers that could result from the presence of family, friends, interviewer, and others during the interview. The in-home interview took approximately one to two hours depending on the adolescent. The in-home interviews were administered in both waves of the Add Health study while the in-school interview was administered in the first wave of the study.

Wave I of the in-home interviews was conducted on April and December of 1995 and Wave II was conducted from April through August of 1996. The two in-home interviews were similar, but some questions including race were taken out of Wave II and some questions related to sun exposure and nutrition were inserted.

In addition to in-home and in-school interviews, Add Health conducted parent interviews on areas such as health, school and community involvement, education, employment, income, parent-adolescent relationship, and involvement in adolescents' friendship and activities. School administrators also completed self-reported questions in Wave I related to characteristics of the school, teachers, and students. In addition, Add Health gathered information on the residential communities of the students.

2.2 Participants

The Adolescent Health Study's sample came from students from 80 high schools and the additional 54 middle schools that feed students into those high schools. The schools were randomly chosen and stratified across the United States by geography, community type (urban/suburban/rural), type of school (private/public/parochial), ethnic composition, and size. Any student on the rosters of the 134 schools was eligible to participate in the Adolescent Health Wave I in-home survey and in-school questionnaire. Students who responded to age, gender, race, depressive symptoms, and smoking habits in both Wave I and II were included in the current study. The final sample for this current study included 2735 boys (48.6%) and 2890 girls (51.4%), with a mean age of 16.04 (SD = 1.62).

2.3 Measures

The data in this current study were obtained from the initial and follow-up (Wave I and II) from the In-Home Interview portion of Add Health from 1994-1996.

2.3.1 Cigarette Use

In order to assess for cigarette use, two questions were taken from the Add Health in Wave I:

- "Have you ever tried cigarette smoking, even just 1 or 2 puffs?"
- "Have you ever smoked cigarettes regularly, that is, at least 1 cigarette every day for 30 days?"

and three questions from Wave II:

- "Since {Month of Last Interview}, have you ever tried cigarette smoking, even just 1 or 2 puffs?"
- "Since {Month of Last Interview}, have you ever tried cigarette smoking, even just 1 or 2 puffs?"
- "During the past 6 months, have you tried to quit smoking cigarettes?"

Participants answered yes or no to these questions.

From these questions, participants were divided into three smoking status: 'nonsmoker', 'experimenter', and 'smoker'. 'Non-smoker' participants answer "No" in "Have you ever tried cigarette smoking, even just 1 or 2 puffs?" in Wave I and "No" in "Since {Month of Last Interview}, have you ever tried cigarette smoking, even just 1 or 2 puffs?" in Wave II.

'Experimenters' are participants that answer "Yes" on "Ever smoked a cigarette (Have you ever tried cigarette smoking, even just 1 or 2 puffs?)" in Wave I and/or "Smoked a cigarette (Since {MOLI}, have you ever tried cigarette smoking, even just 1 or 2 puffs)" in Wave II, but "No" on "Smoked cigarettes regularly (Have you ever smoked cigarettes regularly, that is, at least 1 cigarette every day for 30 days?)" in Wave I and/or "Smoked cigarettes regularly (Since {MOLI}, have you ever smoked cigarettes regularly, that is, at least 1 cigarette every day for 30 days?)" in Wave II and/or "Smoked cigarettes regularly (Since {MOLI}, have you ever smoked cigarettes regularly, that is, at least 1 cigarette every day for 30 days?)" in Wave II.

'Smoker' participants answer "Yes" to both "Smoked cigarettes regularly (Have you ever smoked cigarettes regularly, that is, at least 1 cigarette every day for 30 days?)" in Wave I and "Smoked cigarettes regularly (Since {MOLI}, have you ever smoked cigarettes regularly, that is, at least 1 cigarette every day for 30 days?)" in Wave II.

In order to assess for smoking cessation, participants in the smoker category were further divided into three groups according to their smoking cessation status: 'successful quitter,' 'unsuccessful quitter,' and 'non-dissonant smoker,' 'Successful quitter' is a smoker that answered "Yes" to "Smoked cigarettes regularly (Have you ever smoked cigarettes regularly, that is, at least 1 cigarette every day for 30 days?)"

in Wave I and "No" to "Smoked cigarettes regularly (Since {MOLI}, have you ever smoked cigarettes regularly, that is, at least 1 cigarette every day for 30 days?)" in Wave II.

Smokers that answered "Yes" to "Smoked cigarettes regularly (Have you ever smoked cigarettes regularly, that is, at least 1 cigarette every day for 30 days?)" in Wave I, "yes" to "Smoked cigarettes regularly (Since {MOLI}, have you ever smoked cigarettes regularly, that is, at least 1 cigarette every day for 30 days?)" in Wave II, and "Yes" to "Past 6 months, tried quit smoking (During the past 6 months, have you tried to quit smoking cigarettes?)" in Wave II are labeled as 'unsuccessful quitter'.

'Non-dissonant smoker' is a participant that answered "Yes" to "Smoked cigarettes regularly (Have you ever smoked cigarettes regularly, that is, at least 1 cigarette every day for 30 days?)" in Wave I, and "No" to "Past 6 months, tried quit smoking (During the past 6 months, have you tried to quit smoking cigarettes?)" in Wave II.

2.3.2 Depressive Symptoms

To measure depressive symptoms, this study used a modified version of the Center for Epidemiologic Studies Depression (CES-D) scale that had 19 items instead of the original 20. Radloff (1977) designed this scale to assess current depressive symptomatology and to study relationships between depression and other variables. This short self-report can be administered by lay interviewers, which makes it ideal for surveying large populations. The CES-D is highly reliable and internally consistent with sufficient test-retest repeatability, and the scale is also reliable when used with the

adolescent population (Radloff, 1977; Radloff, 1991). The scale originally had 20 items, but two items, "My sleep was restless" and "I had crying spells" were omitted and one item, "I felt that life was not worth living" was inserted. Items were scored on a frequency range of 0-3 (0 = rarely or none of the time; 1 = some or a little of the time; 2= occasionally or moderate amount of time; 3 = most or all the time). The scale took a sum of the responses, with a maximum score of 57. The higher the score, the higher the depressive symptoms. The CES-D is shown to be highly reliable and internally consistent (Cronbach's α) with .85 for Wave I and .86 for Wave II (Hann, Winter, & Jacobsen, 1999). The CES-D was created with an arbitrary cutoff of 16 as indication of current depressive symptoms. However, this study did not use that cutoff score as the indicator for depressive symptomatology because the cutoff was derived from the adult population. It is questionable whether the cutoff at 16 will be indicative of adolescent depressive symptoms. Because depressive symptom is a continuous variable in this study, this study will use a cutoff from the 75th percentile as differentiation between high and low depressive symptoms. Adolescents in the 75th percentile and above are considered to have 'High' depressive symptoms, and those below the 75th percentile are considered to have 'Low' depressive symptoms. This percentage relates to the depressive-symptoms population from the epidemiological study by Rushton, Forcier, and Schectman (2002), whose results derive from the same data as the current study in Add Health. Rushton et al. (Rushton, Forcier, & Schectman, 2002) examined prevalence of depressive symptoms based on two different cut off scores: 24 and 16. Prevalence of depressive symptoms based on a score of 24 was 9.2% at time 1 and 9.4%

at time 2, and a score of 16 resulted in 28.7% at time 1 and 28.6% at time 2. Based on these results, the cutoff score of 16 is comparable to the 75th percentile cutoff in this study. However, cutting off highly depressive symptoms at the 75th percentile allows this study to assess symptomatology in relationship to the participants and not to an arbitrary external parameter.

2.3.3 Demographics

The current study also examined depression and smoking in association with three demographic variables: gender, age, and race. Gender was assessed by the interviewer at the time of interview. The age of the participant was determined by birth date in month and year from the Add Health study. Six questions were used to assess ethnicity and race. Participants were asked the following:

- "Are you of Hispanic or Latino origin?"
- "What is your race? White"
- "What is your race? Black or African American"
- "What is your race? American Indian or Native American"
- "What is your race? Asian or Pacific Islander"
- "What is your race? Other"

From there, the current study categorized participants into the following groups by race: White, Black, Hispanic, Asian American and Pacific Islander, American Indian, and Other. Although Hispanic is considered an ethnicity and not a race in accordance to the Office of Management and Budget, when a respondent answered "yes" to "Are you of Hispanic or Latino origin?", that respondent was given a race of 'Hispanic' and eliminated from all other race categories in the Add Health data . In other words, participants assigned to White, Black, Asian American and Pacific Islander, American Indian, and Other are all non-Hispanic participants (Add Health, 2004). The 'Other' category is for participants who were unable to identify with other categories.

CHAPTER 3

RESULTS

Of the 6504 adolescents who responded in the public-use data set, only data from 5625 participants were utilized because 879 participants entered multiple answers for the race and/or Hispanic ethnicity question. Overall, about half (50.9%) of the sample were non-smokers (n = 2,863; 1372 males and 1491 females), followed by 26.3% experimenters (n = 1477; 729 males and 748 females) and 22.8% regular smokers (n=1285; 634 males and 651 females).

The racially diverse sample is comprised of 51.6% Whites (2900), 23.4% Blacks (1,315), 11.1% Hispanics (624), 4.1% Asians and Pacific Islanders (232), 3.6% American Indians (203), and 6.2% Other (351). The Black (67.2%) and Asian and Pacific Islander (62.1%) group had the highest percentages of non-smokers. Aside from the Other group (29.1%), Hispanic (28.7%) and American Indian (28.6%) had the highest percentages of experimenters. The White (28.3%) and American Indian (25.1%) had the most smokers according to race. The mean age of the participants is 16.04 years \pm 1.62. There were age differences in all three smoking status groups; non-smoker group (15.73 \pm 1.65) was the lowest followed by experimenter (16.20 \pm 1.47) and smoker (16.59 \pm 1.47).

Sample demographics by smoking status is shown in Table 3.1. Gender ($\chi^2 =$ 11.72, p = .003) was significantly related to smoking status but race ($\chi^2 = 12.21$, p =

.142) was not significantly related to smoking status. The results of a one-way ANOVA showed that age was also significantly related to smoking status with regular smokers being significantly older than either experimenters (p < .001) or non-smokers (p < .001).

	Table 3.1 Sample Demographics By Smoking Status					
	Total Sample	Smoking Status				
	<i>(n)</i>					
		Non-Smoker	Experimenter	Smoker		
Gender						
Male	2735 (48.6%)	1372 (50.1%)	729 (26.7%)	634 (23.2%)		
Female	2890 (51.4%)	1491 (51.6%)	748 (25.9%)	651 (22.5%)		
Race						
White	2900 (51.6%)	1296 (44.7%)	783 (27%)	821 (28.3%)		
Black	1315 (23.4%)	884 (67.2%)	306 (23.3%)	125 (9.5%)		
Hispanic	624 (11.1%)	328 (52.6%)	179 (28.7%)	117 (18.8%)		
Asian	232 (4.1%)	144 (62.1%)	53 (22.9%)	35 (15.1%)		
American	203 (3.6%)	94 (46.3%)	58 (28.6%)	51 (25.1%)		
Indian						
Other	351 (6.2%)	182 (51.9%)	102 (29.1%)	67 (19.1%)		
Age						
Mean (SD)	16.04 (1.62)	15.73 (1.65)	16.20 (1.47)	16.59 (1.47)		

3.1 Concurrent associations between smoking and depression

Table 3.2 presents the concurrent distribution of depressive symptoms for nonsmokers, experimenters, and regular smokers at the two waves of the study. At T1, 77.1% (n = 4320) of the participants were in the low depressive symptoms group, and 22.9% (n = 1280) were in the high depressive symptoms group. Similar percentages appeared in T2: 77.7% (n = 3223) of the participants were in the low depressive symptoms while 22.3% (n = 927) were in the high depressive symptoms group. Although there were no significant changes in the percentage of participants with depressive symptoms in Wave I and Wave II, there were significant differences between the depressive symptoms and smoking status groups. Among the non-smokers group, 16.6 % of the participants had high depressive symptoms and that proportion doubled for regular smokers at Wave I (34.5%) and Wave II (32.7%). The proportion of participants with high depressive symptoms increased from non-smokers to experimenters in Wave I but not in Wave II.

Table 3.2 also presents means and standard deviations for the (continuous) depressive symptoms measure for the three smoking groups. Wave I depressive symptoms were lower for the non-smokers compared to experimenters and regular smokers. However, there were no significant differences between the experimenters and smokers at Wave I. At Wave II, the differences in depressive symptoms among the three groups were more evident in all paired comparisons. From this, smoking and depressive symptoms showed concurrent associations, regardless of whether depressive symptoms were treated as a continuous variable.

-		current smoking s	Comparisons between groups			
Depression	-			(Mean Differences and Standard		
				Errors)		
	Non-	Experimenter	Smoker	1 versus	1 versus	2 versus
	Smoker	(2)	(3)	2	3	3
	(1)					
Time1						
Low ^a	2375	1106 (75.1%)	839	-2.16	-4.13	-1.96
	(83.4%)		(65.5%)	(.24)***	(.25)***	(.28)
High	473	366 (24.9%)	441			
	(16.6%)		(34.5%)			
Group	2848	1472	1280			
Total						
Mean $(SD)^{b}$	9.46	11.62 (7.63)	13.59			
	(6.73)		(8.37)			
Time2						
Low	1823	811 (83.5%)	589	-2.39	-3.74	-1.35
	(83.5%)		(67.3%)	(.27)***	(.29)***	(.33)***
High	359	282 (16.5%)	286			
	(16.5%)		(32.7%)			
Group	2182	1093	875			
Total						
Mean $(SD)^{b}$	9.39	11.78 (7.56)	13.13			
	(6.70)		(8.37)			

Table 3.2 Concurrent Associations Among Smoking Status And Depression

^a Depression dichotomized into below ('low symptoms') and above ('high symptoms') 75th percentile for the group; ^b Depression treated as a continuous variable; * p < .05; ** p < .01; *** p < .001.

3.2 Prospective associations between smoking and depression

3.2.1 Prediction offered to smoking at Wave II by depressive symptoms at Wave I.

Table 3.3 shows the prospective analyses. Only non-smokers at Wave I were included in the first set. High versus low depressive symptoms at Wave I predicted future smoking status and experiment status of non-smokers. In parallel analyses for depressive symptoms as a continuous variable, non-smokers at Wave II had lower levels of depressive symptoms at Wave I than regular smokers and experimenters. Multivariate associations were also tested. Non-smokers with high depressive symptoms at Wave I had a 50% higher likelihood of becoming a smoker a year later [OR, 95% CI = 1.40 (1.04-1.89), p < .05] than those who reported low levels of depressive symptoms.

3.2.2 Prediction offered to depressive symptoms at Wave II by smoking at Wave I.

Only those who reported 'low' depressive symptoms at Wave I were included in these analyses. Many more experimenters and regular smokers than non-smokers (categorized at Wave I) had developed high levels of depressive symptoms a year later (Wave II). When depressive symptoms were treated as a continuous variable, experimenters and regular smokers at Wave I had higher depressive scores than nonsmokers a year later (Table 3.3). Also, regular smokers at Wave I had higher depressive scores than experimenters a year later. After controlling for age and gender, smoking status (ever versus never) at Wave I predicted high versus low depressive symptoms at Wave II [OR, 95% CI = .55 (.44-.68), p < .001]. Prediction by Wave I smoking status (β = .14, p < .001) was sustained when depressive symptoms were examined as a continuous variable in linear regression.

1 auto 3.3	_	e Associations An surrent smoking s			sons betwee			
Depression	Cone	different sintoking i	futus		n Difference			
2 • pression					Standard Errors)			
	Non-	Experimenter	Smoker	1 versus	1 versus	2 versus		
	smoker	(2)	(3)	2	3	3		
	(1)							
Time1								
Depression to								
Time 2								
Smoking ^a								
Low	1644	687 (84.4%)	567	-1.65	-2.37	72		
$(n = 2898)^{\rm b}$	(92.2%)		(82%)	(.26)***	(.29)***	(.33)		
High	178	127 (15.6%)	272					
(n = 577)	(9.8%)		(18%)					
Group Total	1822	814	839					
(n = 3475)								
Mean $(SD)^{c}$	8.01	9.66 (6.26)	10.38					
	(5.69)		(6.86)					
Time1								
Smoking to								
Time 2								
Depression ^d								
Low	1861	239 (76.4%)	60	-1.14	-3.33	-2.19		
$(n = 2160)^{\rm b}$	(82.3%)		(67.3%)	(.41)*	(.79)***	(.87)*		
High	400	74 (23.6%)	20					
(n = 494)	(17.7%)	212	(32.7%)					
Group Total	2261	313	80					
(n = 2654)	0.64	10.70 (6.00)	10.00					
Mean $(SD)^{c}$	9.64	10.78 (6.88)	12.98					
	(6.84)		(8.74)					

Table 3.3 Prospective A	Associations Amo	ng Smoking S	Status And Depression

^aAnalyses only 3475 participants with low depression at Time 1; ^bDepression dichotomized into below ('low symptoms') and above ('high symptoms') 75th percentile for the group; ^cDepression treated as a continuous variable; ^dAnalyses includes only 2654 'never smokers' at Time 1; * p < .05; ** p < .01; *** p < .001.

3.3 Predictors of current smoking status

The current study examined whether depressive symptoms, gender, and age predicts smoking status. Table 3.4 shows the multiple logistic regression analysis of predicting current smoking status. Depressive symptoms at Wave I and Wave II as well as age predicted current smoking status, but not gender.

	Smoker ($n = 5625$)	
Variable	Odds Ratio	95% Confidence Interval
Depression ^a at Time 1 (0:	1.41**	1.11-1.78
Low; 1: High)		
Depression ^a at Time 2 (0:	1.36**	1.08-1.71
Low; 1: High)		
Gender (1: Male; 2: Female)	1.04	.86-1.26
Age	1.26**	1.19-1.34

Table 3.4 Multiple Logistic Regression Analysis Predicting Current Status As A Smoker (n = 5625)

^aDepression dichotomized into below ('low symptoms') and above ('high symptoms') 75^{th} percentile for the group; * p < .05; ** p < .01; *** p < .001.

3.4 Depressive symptoms and smoking cessation

At Wave I and Wave II, successful quitters had fewer high depressive symptoms compared to unsuccessful quitters and non-dissonant smokers (Table 3.5). However, the differences between the three groups on a continuum of depressive symptoms were not significant.

Depression	Concurrent smoking cessation status			Comparisons between groups (Mean Differences and Standard			
Depression				(Interni Di	Errors)		
	Successful	Unsuccessful	Non-	1 versus	1 versus	2 versus	
	(1)	(2)	dissonant	2	3	3	
			(3)				
Time1							
Low ^a	37	202 (65.8%)	163	-1.13	-1.20	06 (.70)	
	(68.5%)		(62.5%)	(1.22)	(1.24)		
High	17	105 (34.2%)	98				
	(31.5%)		(37.5%)				
Group	54	307	261				
Total							
Mean	12.89	14.02 (8.25)	14.08				
$(SD)^{\rm b}$	(7.17)		(8.49)				
Time2							
Low ^a	40	204 (66%)	173	-1.51	-1.28	.23 (.73)	
	(74.1%)		(65.8%)	(1.28)	(1.30)		
High	14	105 (34%)	90				
	(25.9%)		(34.2%)				
Group	54	309	263				
Total							
Mean	12.20	13.71 (8.51)	13.49				
$(SD)^{b}$	(8.14)		(8.99)				

Table 2.5 Cone	urrant Danra	action And	Smolting	Connetion	Status
Table 3.5 Conc	unent Deple	SSIOII AIIU	Smoking	Cessation	Status

^a Depression dichotomized into below ('low symptoms') and above ('high symptoms') 75th percentile for the group; ^b Depression treated as a continuous variable.

CHAPTER 4

DISCUSSION

Depression has been linked to academic issues, teenage pregnancy, risk for substance use, suicide, and poor psychosocial functioning (Kubik et al., 2003). These factors lead to the deterioration of adolescent well-being, and therefore studying the comorbidity of depression and these factors are of utmost importance. Results have indicated a strong comorbidity between depression and smoking (Fergusson et al., 2003; Goodman & Capitman, 2000; Kendler et al., 1993; Kendler et al., 1993; Lam et al., 2005; Lewinsohn et al., 1998; Nezami et al., 2005; Steuber & Banner, 2006; Upadhyaya et al., 2002; Wu & Anthony, 1999). While the causal relationship between smoking and depression is yet to be determined, the strong association between the two is well-documented and undeniable. This prospective adolescent study examined data on smoking and depressive symptoms collected from the National Longitudinal Adolescent Health Study between 1994 and 1996. This study aimed to examine the associations between depressive symptoms and smoking concurrently and prospectively in American adolescents, predictor of smoking, and associations between concurrent depressive symptoms and success in smoking cessation.

Results found higher levels of depressive symptoms at baseline (Wave I) were indicative of participants who smoked in the past and persisted to smoke regularly with low depressive symptoms. Also, higher levels of depressive symptoms at Wave II were characteristic of participants who smoked regularly. These results suggest that depressive symptoms promote concurrent smoking behavior, and further supports the directional association in previous findings (Covey & Tam, 1990; Escobedo et al., 1998; Kandel & Davies, 1986; Kubik et al., 2003; Lam et al., 2005; Martini et al., 2002; Nezami et al., 2005; Patton G. et al., 1996; Patton et al., 1998).

This study differs methodologically from other studies because of the prospective design. The prospective analysis indicates that participants with higher depressive symptoms in Wave I are more likely to start smoking in and become regular smokers in Wave II. Moreover, regression analysis found that depressive symptoms in both Wave I and II predict current smoking status. These results suggest that depressive symptoms promote both concurrent and prospective smoking behaviors and are supported by the self-medication theory, which says that adolescents smoke to reduce their depressive symptoms. Although association between depressive symptomatology and smoking can be made, it does not mean that there is a causal relationship. In addition, it is possible that smoking can also affect depressive symptoms based on these results. Experimenters and smokers at Wave I were more likely to develop high levels of depressive symptoms in Wave II. However, there is more evidence in this study that points to depressive symptoms leading to smoking than vice versa, and Upadhyaya et al. (2002) reported similar findings in his study.

Although, this current study examined participants by various smoking status to avoid confounding interference, it is possible that the study did not adequately filter out all confounding factors. The literature review on smoking and depression reveals both are predictors of adolescent's risky behaviors such as alcohol and substance use, poor academics, early parenthood, poor sleep, and other health problems (Breslau, 1995; Kubik et al., 2003; Mathers et al., 2006). It is probable that some confounding factors cut across smoking status. Aside for the causal explanation to the comorbidity of smoking and depression, an alternative is confounding and selection. These confounding factors can include genetic and environmental that increases the risk of depression and smoking. Fergusson et al. (2003) conducted a 21-year longitudinal study in New Zealand to examine major depression and cigarette use. Data gathered through interview found that participants that met DSM-IV criteria for major depression tend to smoke daily and become nicotine dependent. However after controlling for potential confounding factors such as ethnicity, childhood adversity, alcohol abuse/dependence, previous cigarette use, and nicotine dependence, the association between depression and smoking reduced considerably. Nevertheless, Fergusson et al. found small but evident tendencies for major depression associated with increased possibility for smoking even after considerable control for confounding factors.

In addition to associations of smoking and depressive symptoms, this study also examined depressive symptoms and smoking cessation. Results indicate differences in the depressive symptoms of successful quitters compare to unsuccessful quitters and non-dissonant smokers, but the differences are not enough to be significant. This indicates that smoking cessation is affected by multiple factors. Because of the addictive effects of nicotine, smokers have difficulty quitting. Predictors of smoking cessation include influences from other adolescent non-smokers, readiness to change, confidence to quit smoking, and negative beliefs about smoking predicted readiness for smoking cessation (Apodaca, Abrantes, Strong, Ramsey, & Brown, 2007; Stanton, Baade, & Moffatt, 2006). Zhu, Sun, Billings, Choi, Malarcher (1999) conducted on data from the National Teenage Attitudes and Practices Survey I and II. The study find frequency of smoking, length of past smoking cessation attempts, self-estimate of continual smoking, maternal smoking status, and depressive symptoms as significant baseline factors of smoking cessation. While the current study did not found significant differences between depression symptoms among the different smoking quit statuses, it recorded depressive symptom differences. Further study needs to be done to validate this predictor.

The associations between depression and smoking in relationship to gender and age revealed age is a predictor of current smoking status. In other words, the older the participant, the likelihood of current smoking increases. This can be attributed to the peer pressure face by older adolescents. Older adolescents tend to be more self-conscious and aware of peer's perception of their behavior, and studies support that peer effects on adolescent smoking status (Epstein, Bang, & Botvin, 2007; Miller, Burgoon, Grandpre, & Alvaro, 2006; Scal, Ireland, & Borowsky, 2003). In addition, older adolescents can potentially smoke to reduce stress. Older adolescents experience more stress from school, social system, and family in their stage of development compare to younger adolescent, and that can predispose them to smoke to alleviate stress. Although age is a predictor, gender differences does not predict current smoking status. This can be due to the similar environmental factors that adolescents experience despite gender

differences. Although, adolescent boys and girls develop differently and at different rates, they encounter similar general developmental milestones. Also, there can be gender differences in the stressors and risk factors that cause smoking, but they have common coping mechanisms both healthy and harmful. This explanation is supported by the similar percentages of non-smokers, experimenters, and smokers in this study in both males and females.

Although race was not examined in relation to depressive symptoms and smoking, this study examined racial differences and smoking status. African American adolescents had the highest rates of non-smokers and lowest rates of smokers. This finding is consistent with other studies on African Americans and smoking (Brook, Brook, Zhang, & Cohen, 2004; Brook, Balka, Ning, Whiteman, & Finch, 2006; Burke, Loeber, White, Stouthamer-Loeber, & Pardini, 2007; D'Amico & McCarthy, 2006; H. R. White, Nagin, Replogle, & Stouthamer-Loeber, 2004a; H. R. White, Violette, Metzger, & Stouthamer-Loeber, 2007; Wills et al., 2007). In contrast, White adolescents had the lowest rate of non-smokers and highest rates of regular smokers. This too is consistent with other studies (D'Amico & McCarthy, 2006; H. R. White, Nagin, Replogle, & Stouthamer-Loeber, 2004a). These findings might seem surprising and inconsistent because African American adults have higher prevalence of current smoking even though White adults report higher prevalence of lifetime smoking. This can be explained by the late on-set of African American smoking and lower rates of cessation (H. R. White, Nagin, Replogle, & Stouthamer-Loeber, 2004b; H. R. White et al., 2007). White et al. found that Whites initiated smoking earlier and smoked more cigarettes than African Americans.

An explanation to low rates of African American adolescent smoking is due to the tight knit family structure in African American families. African American families tend to be more hierarchical and strict, use demanding behavioral standards, and apply physical discipline. However, such strict disciplined is balanced by strong love and support. In addition, the family structure is not limited to immediate family. Extended family as well as close friends are considered part of kin, and therefore adolescents can receive support from multiple relatives. In turn, the family is able to monitor adolescents more closely to reduce the risk of smoking and other delinquent behaviors.

The Add Health allowed this current study to examine longitudinal data between two common problem, depressive symptoms and smoking, in a large sample across United States. However, limitations in the current study require caution in interpretation of the findings. First, the self-report nature of the Add Health can lead to unreliable and invalid data. Participants can choose to under-report, over-report, or provide factitious information which the study cannot safe-guard against. Interviewer information, diagnostic interviews, and biochemical markers for tobacco use could be used to determine actual use of tobacco and concrete cases of depressive symptoms. Second, the small smoker sample size for a longitudinal study can affect the association between depressive symptoms and smoking. In turn, random errors can influence results. Third, although the current study did not use the arbitrary cut-off point in the CES-D, the study set its own arbitrary cut-off at the 75th percentile to examine depressive symptoms

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relative to the participants. This arbitrary point could have potentially under and/or over-screened depressive symptoms. Fourth, CES-D does not clinically diagnose Major Depression, so the depressive symptoms can be indicative of other psychiatric disorders such as anxiety. In addition, CES-D might not be culturally sensitive to accurately identify depressive symptoms in different cultures. Hispanic cultures tend to be expressive while Asian cultures tend to be reserved, so depressive symptoms can manifest differently which in turn can affect CES-D. Fifth, the smoking questions on Add Health used in this study have not been standardized or validated, and therefore the reliability and validity of the smoking measures can be questionable.

Overall, these findings lend insight to the growing wealth of information on smoking and depressive symptoms. Even though this study supports other studies that found depressive symptoms promote smoking, other studies have found the opposite or the confounding factor explanation to be true. Therefore, additional studies need to be done to continue to investigate the mechanism behind this comorbidity. Meanwhile, the association between depressive symptoms endorses smoking calls for effective evidence-based intervention that targets depressive symptoms and healthy coping methods with adolescents. This is important for adolescents of all racial backgrounds but particularly the White and African American population. In addition, older adolescents are susceptible to smoking, so smoking prevention can provide alternatives to smoking. Schools can implement smoking education for young adolescents to reinforce the alternatives for the future.

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APPENDIX A

MODIFIED VERSION OF CENTER FOR EPIDEMIOLOGIC STUDIES DEPRESSION SCALE (CES-D)

Depression symptoms (19 items)

Please tell me how often you have felt this way during the past week.

Answers range: 1 (rarely or none of the time) to 5 (most or all of the time)

- 1. In Past week bothered by things.
- 2. In Past week had poor appetite.
- 3. In Past week had the blues.
- 4. In Past week felt just as good as other people.*
- 5. In Past week had trouble keeping mind focused.
- 6. In Past week felt depressed.
- 7. In Past week too tired to do things.
- 8. In Past week hopeful about the future.*
- 9. In Past week felt life had been a failure.
- 10. In past week felt fearful.
- 11. In past week felt happy.*
- 12. In past week talked less than usual.
- 13. In past week felt lonely.
- 14. In past week people unfriendly to you.
- 15. In past week enjoyed life.*
- 16. In past week felt sad.
- 17. In past week felt people dislike you.
- 18. In past week hard to start doing things.
- 19. In past week felt life not worth living.
- * The scoring of positive items is reversed.

APPENDIX B

SMOKING MEASURES FROM ADD HEALTH

Wave I

Ever smoked a cigarette (Have you ever tried cigarette smoking, even just 1 or 2 puffs?) 0: No, 1: Yes

Smoked cigarettes regularly (Have you ever smoked cigarettes regularly, that is, at least 1 cigarette every day for 30 days?)

0: No, 1: Yes

Wave II

Smoked a cigarette (Since {MOLI}, have you ever tried cigarette smoking, even just 1 or 2 puffs)

0: No, 1: Yes

Smoked cigarettes regularly (Since {MOLI}, have you ever smoked cigarettes regularly, that is, at least 1 cigarette every day for 30 days?)

0: No, 1: Yes

Past 6 months, tried quit smoking (During the past 6 months, have you tried to quit smoking cigarettes?)

0: No, 1: Yes

APPENDIX C

DEMOGRAPHICS

Age What is your birth date? month [and year]? What is your birth date? [month and] year?

Ethnity/Race

Are you of Hispanic or Latino origin? 0: No, 1: Yes

What is your race? White 0: No, 1: Yes

What is your race? Black or African American 0: No, 1: Yes

What is your race? American Indian or Native American 0: No, 1: Yes

What is your race? Asian or Pacific Islander 0: No, 1: Yes

What is your race? Other 0: No, 1: Yes

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BIOGRAPHICAL INFORMATION

Bonnie Choi Mo received for Bachelor of Science in Psychology at University of Texas at Austin. Ms. Mo was a clinician at the Adolescent Mood and Addiction Research Program in University of Texas Southwestern Medical School. She has also done field placement at the Collin County Children's Advocacy Center and Parkland Memorial Hospital's Psychiatric Social Work service in Dallas while obtaining her Masters of Science in Social Work from University of Texas at Arlington. Ms. Mo's research interest includes mental health and child and family issues in the Asian American population. She hopes to continue to work in a Psychiatric setting serving this population.