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CIGARETTE SMOKING, DEPRESSIVE SYMPTOMS, AND SOCIAL COGNITIONS: AN EXAMINATION OF THEIR INTERCORRELATIONSHIPS AMONG HIGH SCHOOL STUDENTS

by

Wilbeth Lugo-Morales

A thesis submitted in partial fulfillment of the requirements for the Doctor of Philosophy degree in Psychological and Quantitative Foundations in the Graduate College of The University of Iowa

August 2019

Thesis Supervisor: Professor Stewart Ehly
I dedicate this dissertation to my beloved family, the “Lugo Morales.” Thank you mami & papi for believing in me, for giving me wings to fly even when that meant being more than two thousand miles apart. Thank you for taking me to the library, and for always keeping books at home that taught me to dream big. Thank you for teaching me the value of education and hard work. There is no doubt that my first ethics class was at home with you. Your care packages, velas, and oraciones paid off! Thank you for giving me and my sister the opportunity for a better future. ¡Gracias mami y papi por enseñarme que el cielo es el límite!
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ABSTRACT

Cigarette smoking and depressive symptoms are two problems that affect adolescents’ health. Although it has been well-documented that a relationship exists between these two concerns, most researchers have used self-report methods to study smoking behaviors. While adolescents are typically accurate when reporting depressive symptoms, they tend to be less accurate reporting smoking. Moreover, research supporting the Prototype Willingness Model (PWM) has shown that the social cognitions of willingness to smoke, and the prototypes or images of smokers are predictors of smoking in adolescents. Little is known about the association between social cognitions and depressive symptoms in adolescence.

In this study, I examined the relationship between cigarette smoking and depressive symptoms in high school students using a biological measure of cigarette smoking. First, I investigated whether depressive symptoms and cigarette smoking were related in a sample of 440 high school sophomore students from Iowa. Then, using data from 264 of the same participants, I examined whether smoking during or before the sophomore year of high school predicted depressive symptoms a year later when participants were in their junior year of high school. Conversely, I also examined whether depressive symptoms during the sophomore year of high school predicted cigarette smoking in the junior year of high school. Finally, I studied the relationship between social cognitions (i.e., prototypes and willingness) and depressive symptoms. An additional section explored whether the social cognitions predicted cigarette smoking.

The findings did not provide evidence supporting a relationship between cigarette smoking and depressive symptoms when smoking was measured by a biological measure. Only self-report of smoking cigarettes significantly predicted depressive symptoms during the
sophomore year of high school. Cigarette smoking during or before the sophomore year of high school did not predict depressive symptoms a year later. Similarly, depressive symptoms reported in the sophomore year of high school did not predict cigarette smoking a year later. For the social cognitions, willingness to engage in smoking behaviors and the prototypes or images adolescents have about teenagers who smoke were significantly associated with depressive symptoms. Only willingness to engage in smoking behaviors was a significant predictor of cigarette smoking. These results support the idea that the relationship between depressive symptoms and cigarette smoking varies when using different methodologies to assess smoking status. Also, the finding that social cognitions correlates with depressive symptoms could motivate further investigation. This work can also alert adults about other ways in which elevated depressive symptoms in adolescents may influence their perceptions.
PUBLIC ABSTRACT

Cigarette smoking and depressive symptoms are two problems that affect adolescents. Exploring concerns such as smoking and depression, which affect the well-being of adolescents, is crucial to keep young people healthy. Most studies exploring the relationship between smoking and depressive symptoms have used self-report measures. While adolescents are typically accurate reporting depressive symptoms, they are less accurate on reporting cigarette smoking. In the current study, I examined the relationship between cigarette smoking and depressive symptoms in high school students using a biological measure of cigarette smoking. The results did not support a relationship between depressive symptoms and cigarette smoking. Cigarette smoking during or before the sophomore year of high school did not predict elevated symptoms of depression a year later during the junior year of high school. Similarly, having elevated depressive symptoms during the sophomore year did not predict cigarette smoking in the junior year of high school.

An additional component of the study examined the relationship between willingness to smoke and prototypes or images of smokers, which are part of a model of a health-risk behavior decision-making model, and their associations with depressive symptoms and cigarette smoking. The findings showed that willingness and prototypes were associated with depressive symptoms. Expressing more willingness to smoke was related to adolescents smoking cigarettes.
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CHAPTER I

INTRODUCTION

Although the prevalence of adolescent smoking has declined over time, smoking is still common in this population. In 2018, 8% of high school students were current cigarette smokers in the U.S. (Centers for Disease Control and Prevention [CDC], 2019). Furthermore, 6% of middle school and 20% of high school students reported the use of any tobacco product (e.g., e-cigarettes, cigars, and cigarettes) in the past 30 days, which is usually the determined time to be considered a smoker (CDC, 2019; U.S. Department of Health and Human Services [DHHS], 2014). Taken together, this would be 3.05 million adolescents who are using products that may kill them in the long run, considering that smoking is the number one preventable cause of death in the U.S. (DHHS, 2014). Although the majority of high school students do not become regular smokers, earlier initiation predicts subsequent regular smoking without discriminating of sex or racial identities (Reidpath, Davey, Kadirvelu, Soyiri, & Allotey, 2014).

In contrast, depression has significantly escalated among adolescents in the last decade. A recent study about trends in depression prevalence in the U.S. revealed that the rate of adolescent depression increased from 8.7 percent in 2005 to 12.7 percent in 2015 (Weinberger et al., 2017). Depression has been strongly associated with smoking, particularly in adult population (Dierker et al., 2015; Fluharty, Taylor, Grabski, & Munafò, 2017; Tjora et al., 2014). Depression interferes with learning, social, health, and everyday functioning (American Psychiatric Association, 2013). Further exploration of factors that might influence adolescent smoking and depression is vital for prevention and intervention programs to keep students mentally and physically healthy. In the current study, I addressed the association between these two concerns among high school students using a biological measure of cigarette smoking. Also,
it explored social cognitions from a model of health-risk behavior decision-making and its association to depressive symptoms and cigarette smoking.

**Background**

Smoking is a health risk behavior that typically starts during adolescence (Bernat, Erickson, Widome, Perry, & Forster, 2008; DHHS, 2014). Cigarette smoking is detrimental in several ways (Thompson, Tebes, & McKee, 2015). Smoking cigarettes increases the likelihood of health issues later in life (e.g., respiratory problems and lung cancer) (Park, 2011). According to Brook, Brook, Zhang, and Cohen (2004), smoking during adolescence (13-17 years old) and young adulthood (18-23 years old) predicts several health issues, such as respiratory ailments, neurobehavioral and cognitive problems by age 30. Adolescents who smoke cigarettes regularly start to experience changes in affect, cognition, and neurobiology (Lydon, Wilson, Child, & Geier, 2014), and those who smoke by the age of 16 are less likely to complete a college degree (Maralani, 2014). Furthermore, smoking usually occurs in the context of other externalizing behaviors such as drinking, unsafe sex, and marijuana use (Hale & Viner, 2016). Given the multiple challenges that adolescents experience, it is important to understand changes that may be happening in connection to adolescents’ well-being.

**The Association between Smoking and Depression**

Research into the association between smoking and depression are not new. For more than 30 years researchers have been exploring this topic after observing that individuals with psychiatric conditions, such as depression, had a high prevalence of smoking (Breslau, Kilbey, & Andreski, 1991; Hughes, Hatsukami, Mitchell, & Dahlgren, 1986). However, most of the studies about the association between smoking and depression were conducted with adult samples. As a result, although many studies have shown that smoking and depression are associated,
the directionality of the association remains debatable (Tjora et al., 2014; Wilkinson, Halpern, & Herring, 2016). Four possible pathways have been proposed to explain the association: 1) smoking leads to depression, 2) depression leads to smoking, 3) bi-directional association, in which both variables influence each other, and 4) there is no association at all.

For example, Kang and Lee (2010) studied the association between depression and smoking using longitudinal data with adults who were 20 years old or older at the beginning of the study. After controlling for socioeconomic and confounding variables, they showed that smoking status at the beginning of the study was significantly related to depression one year later. Similarly, Bakhshaie, Zvolensky, and Goodwin (2013) through using logistic regression in data from a sample of middle age adults, found that smoking significantly increased the likelihood of a major depressive disorder even 10 years later. Moreover, adults who identified as current smokers more often reported a major depressive episode in the past in comparison to non-smokers (Berg, Wen, Cummings, Ahluwalia, & Druss, 2013). This is consistent with older studies in which depression predicted not only cigarette smoking, but also greater intensity of smoking (Breslau et al., 1991). The medical community tended to use the self-medication hypothesis to explain the increase of smoking in individuals with depression (Khantzian, 1997). According to this hypothesis, the nicotine found in cigarettes reliefs depressive symptoms; therefore, reinforcing individuals to continue smoking.

In comparison to studies that suggest unidirectional association, fewer studies show a bi-directional relationship between depression and smoking. For example, An and Xiang (2015) found that depression predicted future smoking among those who were nonsmokers at baseline, whereas smoking predicted future depression among non-depressed adults at baseline. Wilkinson, Halpern, and Herring (2016) found similar results thorough linear mixed effects
models in a sample of young adults. In this study, depressive symptoms at baseline were associated with an increase in smoking frequency in following waves. Similarly, they found that smoking frequency was associated with an increase in depressive symptoms. Unlike those studies in which an association between the variables has been found, a smaller group of studies have found no association after controlling for other variables, suggesting that the association between smoking and depression is based on confounding association with both variables (Chaiton, Cohen, Rehm, Abdulle, & O’Loughlin, 2015; Wiesner & Ittel, 2002). Others have found that the association changes at different stages of life (Arnold, Greco, Desmond, & Rotheram-Borus, 2014; O’Loughlin, Karp, Kouliis, Paradis, & DiFranza, 2009).

Given the strength of these findings, a question of importance has been whether the association between smoking and depression is causal. Some researchers have been able to explore the causal direction of the association between depression and smoking through Mendelian randomization analysis (Bjørngaard et al., 2013; Taylor et al., 2014). This is a methodology used in epidemiological studies by using genetic variation to determine whether a particular exposure in the environment is caused by a specific trait (Burgess, Timpson, Ebrahim, & Smith, 2015). In this case, both studies explored whether smoking was the cause of depression and anxiety. However, the results of these studies did not support a causal relationship between the variables; in other words, smoking is not a cause for depression. In summary, mixed results have been found about the association between depression and smoking. Understanding the association between these two constructs is complex and differences in results depend on the construct studied, the methods used, and the developmental period in which the relationship is explored (Goodwin, 2017).
The Association of Smoking and Depression in Adolescents

Studies focused on the relationship in adolescents reveal similar results. For example, Moon, Mo, and Basham (2010) studied the association between depressive symptoms and smoking in a large sample of adolescents in the U.S. The results showed that adolescents who had higher levels of depressive symptoms at the beginning of the study were more likely to start smoking and become regular smokers two years later. Also, results from the same study indicated that depressive symptoms predicted smoking status concurrently. Similarly, Clark, Ringwalt, & Shamblen (2011) investigated whether depressive symptoms predicted future smoking among middle school students and found that depressed mood while in 6th grade predicted cigarette use in the 8th grade. In contrast, Beal, Negriff, Dorn, Pabst, and Schulenberg (2014) explored the association in a sample of adolescents girls from ages 11 to 20 using latent dual change scores. They found that higher levels of smoking predicted an increase in depressive symptoms across the years. Other studies have supported this direction of smoking, predicting depression in adolescence (Maslowsky & Schulenberg, 2013).

Findings from several studies of adolescents suggest bi-directional association between smoking and depression (Audrain-McGovern, Rodriguez, & Kassel, 2009; Tjora et al., 2014; Wilkinson et al., 2016). For example, Audrain-McGovern et al. (2009) showed that depressive symptoms since the age of 14, predicted smoking progression until the age of 18. However, smoking at 14 was associated with less depression later in life, which may be explained by the self-medication hypothesis, which consists of using substances to decrease negative symptoms (Khantzian, 1997). Tjora et al., (2014), showed a different type of bidirectional association. They investigated the association using both cross-sectional analyses and longitudinal models following individuals from ages 13 to 30. They found that daily smoking and depression were
significantly associated in almost all waves with cross-sectional analyses; similarly, the longitudinal models showed good fit in both directions.

Recent studies also reveal that the association varies by gender. For example, Wilkinson et al.’s (2016) results supported a bi-directional relationship between smoking and depressive symptoms for females only; whereas for males the results support the self-medication hypothesis in which smoking is used as a relief for depressive symptoms. Similar results were found by Fröjd, Ala-Soini, Marttunen, and Kaltiala-Heino (2013) in a longitudinal study in which they found that depression at age 15 emerged as a risk factor for smoking at age 17 among girls but not among boys. Overall, researchers agree that more studies are needed regarding the differences in the association between depression and smoking by gender.

**Internal Influences of Smoking: Social Cognitions**

Adolescents’ decisions to smoke do not occur in a vacuum. There are multiple influences, internal and external, that lead individuals to try their first cigarette (Villanti, Boulay, & Juon, 2011; D. Wilkinson & Abraham, 2004). For example, some of the external influences are social pressure, easy access to cigarettes in the neighborhood, friends who smoke, and family members who are smokers (O’Loughlin et al., 2017; Villanti et al., 2011). Additionally, some of the internal influences are desire to experiment and attitudes toward individuals who smoke (Haas & Schaefer, 2014). The way these internal influencers operate have been explained by different models of decision making. Smoking is considered a health risk behavior, and like other risk behaviors, dual processing models of decision making have been valuable to explain those behaviors (Gerrard, Gibbons, Houlihan, Stock, & Pomery, 2008). Dual processing models explain that there are two different processes that can work simultaneously in decision making.
The idea is that individuals have the capacity to engage in analytic and heuristics processes at the same time (Gibbons, Kingsbury, & Gerrard, 2012).

The Prototype Willingness Model (PWM) (Gibbons, Gerrard, Blanton, & Russell, 1998) is a dual processing model that has been found effective for predicting health risk behaviors in adolescents. The main tenet of this model is to conceptualize decision making as a process that is not always deliberate. The PWM model explains that there are two types of information processing that influence decision making: a heuristic process and a mode that is more rational. In the PWM, the less reasoned process, the heuristic one, is more important. This is especially true in adolescents, who are more likely to engage in behaviors without being able to premeditate the consequences of their actions (Gibbons et al., 2012). This heuristic process is usually called “the social reaction path” because it is image-based (Gerrard et al., 2008). In this path, there are cognitive schemas that influence decision making. In the context of smoking, the social cognitions of prototypes or images of smokers and willingness to engage in smoking behaviors (e.g., taking a puff) have been found to be antecedents of smoking (Gerrard et al., 2008; Gerrard, Gibbons, Stock, Vande Lune, & Cleveland, 2005). An antecedent in this context means that the perceptions of others’ behaviors (i.e., cigarette smoking), and the willingness to engage in that behavior lead to actually engaging in that behavior later.

**Limitations of Existing Literature and Rationale for the Current Study**

Although there are considerable differences in the methodologies used, many researchers concluded that more studies are needed to understand the association between depression and smoking (Fluharty, Taylor, Grabski, & Munafò, 2017; Goodwin, 2017; Wilkinson et al., 2016). Goodwin (2017) pointed out that if this association starts during adolescence, more studies should focus on this population. One of the main limitations in previous studies is the almost
exclusive use of self-report to measure cigarette smoking behaviors (Dube, Thompson, Homa, & Zack, 2013; Kang & Lee, 2010; Tjora et al., 2014). It is known that the reliability of self-report measures for substance use is low in adolescents (Andersen, Philibert, Gibbons, Simons, & Long, 2017; Dube et al., 2013). For example, Dube et al., (2013) compared two measures of cigarette smoking (i.e., self-report vs. a biological measure) in a large sample of adolescents ages 12 to 17 and found a discrepancy in the prevalence of smoking. This prevalence was higher when measuring serum cotinine levels in the blood (13.4%) than in self-report, where only 10% of the adolescents reported smoking. Some researchers have recognized this limitation in their studies and have suggested the use of biological measures to confirm tobacco use in future studies (Moon et al., 2010; Thompson et al., 2015). The current study tackled this limitation by using a biological quantitative measure of smoking consumption.

The association between depressive symptoms and smoking in adolescents is complex. While mixed results have been found regarding directionality, to the knowledge of this researcher no study had used a biomarker of cigarette smoking to explore this association in a group of students who are at-risk of smoking. Therefore, the purpose of the current study was to explore the association between depressive symptoms and cigarette smoking among high school students using both self-report and a new technique that allows confirmation of smoking status. In addition to investigate whether social cognitions play a role in smoking decisions (Gerrard, et al., 2008).

Although there is evidence showing that the social cognitions of prototypes and willingness influence smoking, it is unknown if experiencing depressive symptoms is associated with the social cognitions that adolescents adopt according to the PWM (willingness and prototypes). In the current study, depression and smoking were observed from the individual and
contextual levels. Adolescents do not engage in smoking behaviors with a blank slate; they carry dispositional characteristics, genetic predispositions, and situational states. Therefore, it seemed relevant to examine whether depressive symptoms were associated with social cognitions among adolescents. Higher levels of depressive symptoms may have an impact in the way individuals perceive their own intentions and the actions of others.

Little is known regarding possible association of those social cognitions and depressive symptoms. The research available on this topic focuses mostly on a concept called “susceptibility of smoking,” which refers to the absence of a commitment to not smoke in the future (Pierce, Choi, Gilpin, Farkas, & Merritt, 1996). Although it is similar to what is presented in the PWM, it differs in the conceptualization of decision making in the context of smoking. More specifically, susceptibility is seen from an individual level, whereas the variables in the PWM include the less deliberate decision making influenced by the environment. Investigating the social cognitions among students with different severity of depressive symptoms contributes to the understanding of literature on the association of smoking and depression.

**Definition of Concepts**

1. **Cigarette smoking** refers to regular cigarette consumption; and to the way it is measured in the current study by a biomarker that allows to estimate the probability of consumption.

2. **Depressive symptoms** refer to experiences common among individuals with depression. In this study, the symptoms are the following: pleasure, feeling down, sleep disruption, energy levels, poor appetite, feeling a failure, trouble concentrating, speaking slowly or being fidgety and having negative thoughts concerning suicide or self-harm.
3. **DNA methylation** refers to a process in which a DNA segment can be modified altering its function or expression without changing its sequence. It is through this process that the biomarker can detects cigarette smoking.

4. **Household income** refers to the gross income received from one or both parents or guardians ranging from none to $150,000 or more per year.

5. **Race/ethnicity** is categorized for this study in two categories: Caucasian/White of non-Hispanic origin and Others (including all other ethnicities due to small numbers of participants from other races/ethnicities).

6. **Sex** is categorized as female or male, as perceived by the interviewer.

7. **Smoking behaviors** refer to different types of smoking (e.g., regular cigarettes, cigars, e-cigarettes, and hookahs) and processes involving smoking (e.g., smoking initiation or onset, regular smoking, daily smoking, and occasional smoking).

8. **Social cognitions** refer to cognitive processes related to perceptions of others (Frith & Blakemore). In this study, the social cognitions studied are constructs from a model of health-risk behaviors decision making.

**Conclusion of Introduction**

Although smoking has decreased among adolescents, it is still common and represents a threat to adolescents’ well-being. Using new methodologies to understand factors associated with smoking is valuable to future prevention efforts. Given that smoking typically initiates in adolescence, it would be ideal to avoid smoking altogether. One way to do this is creating intervention programs that target specific adolescents who are at-risk for smoking. Cigarette smoking during adolescence is characterized by a period of experimentation of light smoking. It has been documented that a relationship exists between depression and smoking. However, most
studies have used self-report measures to report smoking, which is typically inaccurate among adolescents. Investigating whether light exposure to smoking is related to depressive symptoms among adolescents represents progress toward intervention programming. No study has been conducted using a biomarker that has a high probability of detecting light smoking. This study investigated the association between smoking and depression using a biological measure of smoking, in addition of exploring social cognitions and its association with depressive symptoms.

Following this introduction, Chapter II provides a literature review featuring the main topics: theoretical framework, depressive symptoms among adolescents, cigarette smoking during adolescence, methods used to study cigarette smoking, and the relationship between depression and smoking. Chapter III describes in detail the methods used in the current study including analysis procedures. Chapter IV includes the results for each of the aims and finally, Chapter V is a discussion of the results and future directions.
CHAPTER II

LITERATURE REVIEW

In this section, I review literature relevant to the aims of the current study. Most of the literature presented is based in studies with a focus on adolescents and within the scope of the decade of 2008 to 2018. It includes the following subsections: 1) theoretical framework, 2) cigarette smoking among adolescents, 3) depression and depressive symptoms, and 4) the relationship between depression and smoking in adolescents.

Theoretical Framework: The Prototype Willingness Model

The current study was informed by the same model that served as a theoretical framework for the main longitudinal study: The PWM of adolescents health risk behaviors (Gerrard et al., 2008; Gibbons & Gerrard, 1995). This model was developed to explain and predict adolescent health risk behaviors using a social-cognitive approach. The model incorporates two pathways to explain why adolescents engage in risky behaviors: a reasoned path and a social reaction path. The reasoned path involves a more logical and analytic process (e.g., "I am going to smoke"), whereas the social reaction path is a more heuristic process, in which adolescents use previous knowledge and context to engage in risky behavior (e.g., "it just happened" given the situation). This model has been studied extensively and its effectiveness has been shown by several researchers (Armenta, Hautala, & Whitbeck, 2015; Gerrard et al., 2005; Litt & Lewis, 2016; Myklestad & Rise, 2007).

Within the social reaction path two constructs are of great importance: risk prototypes, defined as images of people who engage in risk behaviors (e.g., the typical smoker), and behavioral willingness, defined as openness to engaging in the behavior (e.g., smoking) (Gerrard et al., 2008). Based on the PWM three assumptions have been developed about risky behaviors:
1) engaging in a health risk behavior is not always a conscious decision, it is rather unintentional and occurs as a reaction to particular circumstances, 2) risk behaviors are social events for adolescents, they rarely engage in those behaviors alone, 3) because of the social component, adolescents have clear images of the type of people that engage in those behaviors.

Although the reason and the social reaction paths seems to occur simultaneously, the social reaction path is the main tenet to explain risky behaviors using the PWM given the strong supporting evidence. Many studies have shown its effectiveness in predicting risky behaviors, such as alcohol use (Armenta et al., 2015; Litt & Lewis, 2016), unprotected sex (Myklestad & Rise, 2007), and smoking (Gerrard et al., 2005; Hukkelberg & Dykstra, 2009). For example, Gerrard et al. (2005) tested the PWM with a sample of African American preadolescents (mean age of 10.5) and they found that their images of smokers and willingness to smoke mediated the impact of other contextual factors, such as family environment on predicting smoking initiation. Similarly, Hukkelberg and Dykstra (2009) showed that the PWM is also useful to understand willingness and intentions to abstain from smoking among non-smoking adolescents. Based on a sample of Norwegian adolescents who reported not being regular smokers, they found that having a negative image of smokers is related to "willingness not to smoke," which predicts future non-smoking behavior.

Adolescents will make decisions based on their previous knowledge and experiences. The interpretations of the environment that are going to influence their willingness to smoke are going to be influenced by their family and individuals’ factors (e.g., thoughts and ideas they may experience when depressive symptoms are present). The PWM predicts smoking and non-smoking based on the prototypes risk and the behavioral willingness. Adolescents who have friends that smoke have a clear idea (or prototype) of people their age that smoke. PWM posits
that people are more willing to engage in behavior to the extent that they have a positive view of
the prototypical person who performs that behavior (Gerrard et al., 2008).

**Cigarette Smoking among Adolescents**

Cigarette smoking typically starts during adolescence and it follows a trajectory that may end in nicotine dependence (Bernat et al., 2008). During the first stage, the initial exposure to cigarettes occurs around 12 to 18 years old. Then follows the experimental smoking stage, in which adolescents have irregular periods of smoking. The last stage is when smoking becomes regular, and there is a physiological and psychological need for nicotine. It has been suggested that adolescents are more likely to continue smoking after the experimental stage due to the positive effect that nicotine causes on their reward system in their brains (Lydon et al., 2014; Smith, McDonald, Bergstrom, Ehlinger, & Brielmaier, 2015). The age of onset of smoking plays a role in how likely someone is to become dependent (Reidpath et al., 2014). The younger adolescents are when they start smoking, the more likely they will become addicted and the harder it is for them to quit (Walker & Loprinzi, 2014). Adolescents experience dependence symptoms quickly and also at very low levels of consumption (Kandel, Hu, Griesler, & Schaffran, 2007). DiFranza et al. (2007) showed that only within days to weeks of the onset of occasional smoking adolescents report feeling symptoms of nicotine dependence, such as strong cravings and restlessness.

Cigarette smoking continues to be one of the most preventable causes of premature death in the U.S. (World Health Organization, 2017). Approximately, 480,000 people die every year in the U.S. as a result of a smoking-related disease (DHHS, 2014). In 2016, approximately 15.5 % (37.8 million) of adults in the U.S. were smokers (Jamal et al., 2018). The vast majority of adult smokers began smoking during adolescence (Institute of Medicine, 2015). Specifically, nine of
ten adults who are cigarette smokers tried their first cigarette by age 18. Data from the National Youth Tobacco Surveys (NYTS) showed that in 2018 approximately 7.2% of middle school and 20% of high school students in the U.S. report the use of a tobacco product in the past 30 days (CDC, 2019).

The prevalence of regular cigarette smoking has declined over time. In 1997, 36% of high school students smoked at least one cigarette a month, which is usually defined as “a smoker” (DHHS, 2014), whereas recent data show that 8% of high school students were current cigarette smokers in 2018 (CDC, 2019). Although it is clear that cigarette smoking has declined in popularity these numbers continue to be troublesome. Particularly because it is likely that these numbers are underestimated, given that adolescents tend to underreport smoking behaviors (e.g., the number of cigarettes used in the past five days and ever smoking) (Bunnell et al., 2015). Therefore, while it is less than the 36% from 1997, rates are probably higher than the 8% reported.

Singh et al. (2016), in a study investigating tobacco use among middle and high school students in a national sample, found that overall tobacco use has not declined among adolescents; rather it has evolved to smoking different types of products. For example, 16% of high school students reported the use of electronic cigarettes (e-cigarettes), which have become the preferred tobacco product for this population (Singh et al., 2016). The increase in this type of product is due mostly to adolescents having the perception that e-cigarettes are safer than traditional cigarettes (Dobbs, Hammig, & Henry, 2017). Although it is true that they contain fewer toxins than regular cigarettes (Goniewicz et al., 2014), there is no evidence showing that they are completely harm free (Dobbs et al., 2017).
An additional concern is the fact that adolescents who use e-cigarettes have increased intention to smoke regular cigarettes (Bunnell et al., 2015). Not only do they have greater intention, they also continue to subsequent regular cigarettes contrary to the popular belief that e-cigarettes were intended to stop smoking (Rennie, Bazillier-Bruneau, & Rouëssé, 2016; Schneider & Diehl, 2016; Soneji et al., 2017). In a prospective study to investigate whether non-cigarette tobacco use during adolescence is related to regular cigarette one year later, Watkins, Glantz, and Chaffee (2018) found that the odds of past 30-day cigarette use one year later were approximately twice as high among baseline ever users of e-cigarettes. The likelihood of cigarette smoking was even greater for adolescents who tried more than one type of tobacco product at age 14 (baseline).

**Smoking Behaviors: Different Constructs**

Researchers have studied multiple components of smoking behaviors, such as smoking initiation or onset, smoking status, and tobacco dependence. Different operational definitions of smoking behaviors makes comparison among studies difficult (Park & June, 2006). One of the measures used more frequently is smoking initiation because once an adolescent initiates the behavior it could easily escalate to regular smoking (Reidpath et al., 2014; Sargent, Gabrielli, Budney, Soneji, & Wills, 2017). However, the multiple definitions of smoking initiation have been a factor complicating the understanding of what predicts smoking initiation (Wellman et al., 2016) and how smoking initiation is a risk factor for regular smoking due to inconsistencies in the operational definition (Reidpath et al., 2014).

In some studies, smoking initiation is measured by smoking a whole cigarette; in others by smoking part of a cigarette, or just having a puff (Sargent et al., 2017). These concepts represent different situations or stages. For example, smoking a whole cigarette is estimated to
occur 2.5 months after first puff, while daily smoking at 23 months (Gervais, 2006). Wellman et al., (2016) argued that these different stages of smoking may have different predictors. Therefore, it is important to be careful and cognizant of the definitions used in studies because different conclusions can be drawn from similar data (Reidpath et al., 2014).

Reidpath et al. (2014) found a significant association between smoking initiation (defined as smoking a whole cigarette) with subsequent regular smoking in a sample of adolescents from the U.S. Smoking a whole cigarette represented higher risk to subsequent regular smoking in both males and females. Similarly, Sargent et al. (2017) found in a recent study that even smoking just a few puffs predicted subsequent cigarette smoking. The operational definition of smoking and the type of smoking behavior used also has an influence when studying the relationship with other factors. For example, Weiss, Palmer, Chou, Mouttapa, and Johnson (2008) in a study with adolescents, found that being a "lifetime smoker," defined as smoking at least one cigarette, was associated with depressive symptoms; however, being a "recent smoker," defined as number of cigarettes smoked in the past 30-days was not significantly associated with depressive symptoms. Although there are a variety of smoking behaviors (e.g., smoking a whole cigarette, smoking once in a month, etc.), the trajectory of becoming a regular smoker is similar for those adults who begin smoking when adolescents and continue to become regular smokers.

**Cigarette Smoking Trajectory**

Typically, the developmental trajectory of smoking for those individuals who become regular smokers occurs in a four-stage process (Beach, Gerrard, Gibbons, Brody, & Robert, 2016). These stages are variable in length and not all individuals who engage in smoking at some point continue to regular smoking (Reidpath et al., 2014) or develop nicotine dependency (Bernat et al., 2008). The first stage of the trajectory is the initial exposure or smoking initiation.
It is at this stage when adolescents try their first cigarette, usually between the ages of 12 to 18 years old. Following this initial exposure is the experimental smoking stage. This stage is characterized by intermittent, contextually dependent, and irregular periods of smoking. Then, it progresses to regular smoking, which is recurrent in specific contexts, and finally, it may end in nicotine dependence.

It has been suggested that if preventions and interventions are to be effective, they need to be done during the first two stages when smoking behaviors are not a biological need as it is in the last stage of the smoking trajectory (Beach et al., 2016). Once an individual reaches the last stage, smoking becomes driven by a physiologic and psychological need for nicotine. At this stage termed “dependent smoking,” smoking behaviors become stably engrained. Most smokers go through multiple cycles of quitting and relapsing (Caponetto, Keller, Bruno, & Polosa, 2013). Smoking rises to a peak during the period of 18 to 24 years old and remains stable after that. However, for adolescents, it is more difficult to quit smoking than for adults (Bachmann, Znoj, & Brodbeck, 2012; Walker & Loprinzi, 2014). On the one hand, there are stronger social influences and values of peers’ perceptions about them. On the other hand, the adolescent brain allows them to continue enjoying smoking given that nicotine impacts their reward system causing more positive effects (Lydon et al., 2014; Smith et al., 2015).

The earlier adolescents begin smoking, the easier it is to become addicted to nicotine (Kendler, Myers, Damaj, & Chen, 2013; Park, Weaver, & Romer, 2009). Kendler et al., (2013) studied twin adults and found that those individuals who started to smoke regularly at earlier ages, controlling for genetics and environmental factors, were more likely to be nicotine dependent in adulthood. In addition, independent of age, a one year decrease in the age of smoking initiation is associated with greater odds of smoking regularly in female and male
adolescents (Reidpath et al., 2014). Therefore, avoiding smoking initiation during adolescence would be the ideal scenario to break the possible trajectory to future dependence (Bachmann et al., 2012).

**Reasons for Smoking Onset and Regular Smoking**

Adolescents initiate smoking for many reasons, such as curiosity, sensation seeking, influence from peers, influence from parents, among many others (Haas & Schaefer, 2014; Mays et al., 2014; Wellman et al., 2016). A substantial body of research has found a broad range of factors associated with smoking initiation. In a systematic review, Wellman et al. (2016) found a high number of conceptually different predictors of smoking in adolescents younger than 18 years old. Lower SES, poor academic performance, higher risk taking, smoking among family members, and smoking in films were the stronger factors associated with increased risk of smoking onset in adolescents (Wellman et al., 2016). Some of these factors differ across ages, which is essential information for prevention and intervention programming at different grade levels. For example, O’Loughlin et al. (2017) studied a set of possible predictors of smoking onset and found that friend smoking was a strong predictor of smoking initiation for younger adolescents (5th grade) and depressive symptoms a stronger predictor factor for early (5th grade) and middle adolescents (7th grade).

Progression to regular smoking is influenced by similar, but also different factors. Park et al. (2009) explored the transition from experimental smoking at baseline to daily smoking one year later using data from the National Longitudinal Study of Adolescent Health. They found that “greater number of friends who smoke,” higher marijuana and alcohol use, and lower academic performance were significant predictors of daily smoking. In a similar study, this time studying the predictors of a five-year follow-up after baseline, initiating smoking at an earlier
age, being from White Caucasian non-Hispanic race/ethnicity, high levels of risk-taking tendency, and marijuana use were found to be significant predictors of daily smoking in young adults (Park et al., 2010). Self-concept is another factor that plays a prominent role specific to smoking escalation (Hertel & Mermelstein, 2012). Hertel and colleagues found that when adolescents see smoking as part of their identity, they tend to progress from experimental smoking to regular smoking.

Some researchers have posited that smoking serves a purpose that may differ by individuals. For example, to feel in control or independence, and to calm down after stressful situations (Fidler & West, 2009; O’Loughlin et al., 2009). According to studies about motivational pathways to smoking, adolescents initiate smoking because of the social environment, but those who continue smoking or move to have the “smoker status” do it for more personal motives (Baker, Brandon, & Chassin, 2004). In conclusion, there are a variety of factors associated with smoking behavior and it is not possible to control for all these factors at the same time. Although most adolescents do not progress to regular smoking, the environmental situations are the perfect scenario for those who are already at-risk for smoking (e.g., lower SES, living with a parent who smokes, and having cigarettes available if wanted in the neighborhood).

**Dangers of Smoking: Impacts on Health and Schooling**

Any type of tobacco product is dangerous for adolescents (CDC, 2019). Not only does nicotine impact adolescents’ health (Dube et al., 2013), but also smoking has been related to low academic achievement (Morin, Rodriguez, Fallu, Maïano, & Janosz, 2012), and less likelihood to attend college (Maralani, 2014). Regular cigarette smoking is particularly dangerous given the numerous known carcinogens and toxins present in tobacco smoke (Talhout et al., 2011).
Smoking and health. Cigarette smoking is related to many health concerns, mostly during adulthood. It is known that for the major chronic diseases caused by smoking (e.g., coronary artery disease and chronic obstructive coronary disease), the risk progresses by the years of smoking because it is time-dependent (DHHS, 2014). While the risk is greater if smoking continues through adulthood, some health issues are seen earlier in adolescence. Hublet et al., (2007) found that adolescents who smoke were more likely to develop asthma than those who never smoked. In a meticulous report from the U.S. Surgeon General, a causal relationship was reported between active smoking and both reduced lung function and impaired lung growth during childhood and adolescence (DHHS, 2014). Adolescents who ever smoked report a greater number of physical and mentally unhealthy days and fair or poor healthy days in general, in comparison to adolescents who never smoked (Dube et al., 2013). It is true that most adolescents are not going to experience these health issues for smoking sporadically; however, their overall health may be impacted (Dube et al., 2013). Also, if they progress to regular smoking and continue smoking through adulthood, health problems are likely to be present.

Smoking and schooling. A relationship has been found between adolescent smoking and schooling. Although there has been some research studying this relationship, there is no central focus to establish a clear portrait of how schooling is affected by smoking, or vice-versa. Morin et al., (2012) examined different students’ profiles regarding their academic achievement and its relationship with smoking initiation. As expected, significant differences were found among students (from 7th to 10th grade) identified as high achievers, of which only 7% started to smoke, and the low achievers, of which 49% initiated smoking. These results show that adolescents who do not do well in school are more likely to initiate smoking. In a systematic review of longitudinal studies, Wellman et al. (2016) found that smoking onset was inversely related to
poor academic performance in youth 17 years old or younger in 10 out of 12 studies included in the review. Higher grade point average (GPA) is a protective factor for the transition from experimental smoking to daily smoking (Park et al., 2010). Students who do not finish high school have the highest probability of starting to smoke regularly (Maralani, 2014). In addition, adolescents who smoke by the age of 16 are less likely to complete a college degree than those who never smoked (Maralani, 2014).

**Measurement of Smoking**

Cigarette smoking is studied by information obtained through self-report or biological measures. Self-report is usually the most common method used in clinical settings and for research purposes. However, researchers and physicians have been concerned regarding the validity of self-report to detect smoking, especially in adolescents (Lantini et al., 2015).

**Self-report.** There are many reasons for the popularity of self-report as the most common method to study smoking: it is cost-effective, non-invasive, and accessible to researchers. The procedures may vary a little bit, as well as the questions asked. Usually, participants are asked to complete questionnaires, while other times interviewers ask participants questions related to smoking. Although self-report is usually accurate in epidemiological studies with adults (Benowitz, Bernert, Carabello, Holiday, & Wang, 2009), it is inaccurate with samples of adolescents when compared to objective biological measures (Carabello, Giovino, & Pechacek, 2004; Kandel et al., 2007).

Sometimes adolescents deny ever smoking cigarettes, other times they under-report the frequency of smoking and number of cigarettes smoked (Kandel et al., 2006; Lantini et al., 2015). Although it is less common, occasionally adolescents over-report the use of cigarettes to fit in with a group and impress peers (Lantini et al., 2015; Stein et al., 2002). Under-reporting
cigarette use is what happens more often. Adolescents are more likely to deny smoking cigarettes or under-reporting the number of cigarettes for several reasons, such as social stigma (Caraballo et al., 2004), fear of reprimand or punishment given that smoking is illegal for minors (Lantini et al., 2015). Also, adolescents even recognize that the use of questionnaires is not the best way to obtain valid measures. In a study with a sample of high school students who identified as smokers, only 37% of participants agreed that questionnaires were a good method to provide honest responses regarding smoking (Adams, Parkinson, Sanson-Fisher, & Walsh, 2008).

Some reasons for the limitations in the use of self-report when studying smoking in adolescents result from the way the data are collected. Griesler, Kandel, Schaffran, Hu, & Davies, (2008) studied discrepancies in self-report among adolescents in common data collection settings. They found that adolescents report smoking more often when questionnaires were completed in schools than in their homes. Other factors that affect the accuracy of answers in self-report are the procedure used (e.g., self-completion of questionnaires vs. interview format) (Brener, Billy, & Grady, 2003), the race/ethnicity of the participants (Lantini et al., 2015), and the desire to please the interviewer (Stein et al., 2002).

Although there are many situations and conditions that influence adolescents in providing honest responses, it is impossible to control all these variables. Adams et al. (2008) suggested that a way to improve self-report validity is using the bogus pipeline method (BPL), which consists of informing students that they will follow-up with biological validation of their smoking behaviors. The rationale is that adolescents will provide more honest answers because they know that they will be discovered lying. The researchers did an experiment to prove the BPL by dividing students in different conditions and found that adolescents in the BPL
condition had higher odds of reporting weekly and monthly smoking (Adams et al., 2008). Although threatening students with the validation of smoking status through biological measures seems to enhance the accuracy of self-report, the ideal is to actually validate smoking behaviors by using biological measures of smoking, which are more objective.

**Biological measures of cigarette use.** Detection of cigarette smoking has been possible thanks to biomarkers, which are characteristics that can be measured as indicators of biological processes (Atkinson et al., 2001). Two types of biomarkers are typically used to identify active smokers: exhaled carbon monoxide (CO) and serum cotinine (Florescu et al., 2009). Measuring CO levels is convenient because it is easy to perform and non-expensive. However, it only detects if a person has smoked within the past four to five hours. The second method is more complex; cotinine levels can be measured by studying an individuals’ blood, urine, or saliva. If an individual has smoked, changes in cotinine levels are expected because cotinine is the major metabolite of nicotine (Benowitz et al., 2009). This method is considered as the gold standard method for smoking detection. However, it presents some limitations: it also has a short life of 20 hours, which means that it can only detect if an individual has been smoking within the 20 hours before the sample is obtained (Benowitz et al., 2009). Another critique that researchers have about these biomarkers is that they are not possible to reflect smoking history. This represents a problem because some individuals could stop smoking for a short period of time when aware that smoking will be measured. In this case, those individuals will appear as “non-smokers”, which is erroneous.

Although CO and cotinine levels represent objective measures to study cigarette smoking, they are not ideal in studying smoking in adolescents (Andersen et al., 2017). Most adolescents do not smoke regularly (Sargent et al., 2017); the ones who smoke, tend to do it
intermittently as is characterized by the experimental stage of smoking (Bernat et al., 2008). Therefore, it was essential to develop a method to confirm smoking status objectively.

A new biomarker for smoking has been developed, which is sensitive to light exposure to nicotine (Philibert, Beach, & Brody, 2012). This could detect smoking in adolescents even if a small number of cigarettes have been smoked. This method consists of studying changes that happen in a particular region of a gene. More specifically, at the cg05575921, a CpG locus in the aryl hydrocarbon receptor repressor (AHRR) gene. Smoking (or inhaling tobacco through combustion), results in the production of polyaromatic hydrocarbons whose inhalation results in decreases of DNA methylation in key regions of AHRR. DNA methylation refers to a process in which a DNA segment can be modified altering its function or expression without changing its sequence (Jin, Li, & Robertson, 2011). These changes have been found to be a sensitive marker of smoking history (Philibert, et al., 2012; Philibert, Beach, Lei, & Brody, 2013). The sensitivity (correctly detecting individuals who smoked) and specificity (correctly detecting those who do not smoked) of the tool has been examined by Dr. Philibert and his colleagues for several years (Andersen, Dogan, Beach, & Philibert, 2015; Philibert et al., 2015; Philibert et al., 2018).

Because DNA methylation changes are relatively slow to initiate and slow to revert; once someone smokes, it causes a reversion of the DNA methylation process that can last for months, even years (Philibert et al., 2016). Studies involving participants with prolonged history of smoking, it was found that methylation was dependent of smoking status (Philibert, 2010). This biomarker has been found to be sensitive to even light exposure to tobacco (Philibert et al., 2012). In a study involving African American young adults (19 years old) with different smoking status (e.g., non-smokers, smoke less than five cigarettes, etc.), Philibert et al., (2012) showed that this methodology detected smoking consumption even when less than five packs of
cigarettes have been consumed. It is important to note that the test quantifies an individual exposure to conventional cigarettes or cigars only (Besingi & Johansson, 2013; Philibert et al., 2015). Non-combustionable forms of tobacco or nicotine absorption ingestion do not cause any change in the gene studied. Although more research is needed to confirm that other types of smoking do not alter the methylation process.

In summary, with this method of using a biomarker it is possible to infer cigarette smoking status. Some implications and positive benefits of this biomarker are that it will be possible to detect those adolescents who are smoking that were undetected in other studies because of “lack of honesty” and/or the differences in definitions used in other studies for "smoking or tobacco use" (e.g., smoking 10 or more cigarettes or cigarettes use in the past 30 days). The effectiveness of this biomarker of smoking has been established recently in the literature. The use of the AHRR biomarker has been seen as an effective tool to measure tobacco use in populations with different demographic characteristics (e.g., racial and ethnic groups), smoking histories (e.g., heavier smokers and nascent smokers), and rates of false-negative self-report of smoking behavior (e.g., adolescents and nicotine dependents) (Andersen et al., 2017). It has also been considered as a promising clinical tool to use in smoking prevention efforts in adolescents (Beach, Gerrard, Gibbons, Brody, & Philibert, 2015). For example, Beach and colleagues (2015) suggested a clinical care model, in which pediatricians use the biomarker as a tool to target adolescents who will benefit of smoking cessation intervention. The next section in this literature review will cover the variable of depression and depressive symptoms.

**Depression and Depressive Symptoms in Adolescents**

Adolescent depression is a problem that affects many young people in the U.S. (National Institutes of Mental Health [NIMH], 2019). Depression is defined as a cluster of symptoms
usually associated with impairment in daily life functioning. A major depressive episode (MDE) is a period of at least two weeks in which an individual experiences depressed mood, loss of interest or pleasure in activities, plus at least four more symptoms as described in the last edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-5) (American Psychiatry Association, 2013). Some of these symptoms include feelings of worthlessness or hopelessness, irritability, constant fatigue, sleep problems, changes in appetite or change in weight, lack of feelings of worthiness, and excessive guilt. In adolescents, the core symptoms include mood changes (e.g., low mood or irritability) or loss of enjoyment which lasts at least two weeks.

The onset of depressive episodes frequently occurs during middle to late adolescence (Kesler, Berglund, Demler, Jin, & Walters, 2005). Although current diagnostic criteria in the DSM-5 and ICD-10 for major depression are the same for adolescents and adults, adolescents may express depressive symptoms in a slightly different way based on developmental differences in cognitive, emotional, biological, and social competences (Kovacs, Obrosky & Sherrill, 2003; Weiss & Garber, 2003). Adolescents tend to express their feelings with more irritability, anger, show psychomotor agitation or retardation, and recurrent thoughts of death, differing from adults who usually express more sadness (NIMH, 2016).

The prevalence of depression has increased considerably among adolescents (Goodwin et al., 2017; Mojtabai, Olfson, & Han, 2016; Weinberger et al., 2017). In 2005, 8.7% of adolescents ages 12 to 17 experience at least one MDE within a 12 month period, whereas in 2014 the number increased to 11.3% (Mojtabai et al., 2016). More recent data show that approximately 3.1 million adolescents (12.8%) reported at least one MDE in 2016 (NIMH, 2019). In addition, approximately, 2.2 million adolescents (9%) had at least one major depressive episode with severe impairment. The prevalence of adolescent depression varies by
age, gender, race, and ethnicity (Mojtabai et al., 2016). Depression is more common in adolescents who have gone through puberty than in those who are at the pre-puberty stage (Hankin et al., 2015). More girls than boys experience symptoms of depression and report a depressive episode in their lives (NIMH, 2019). Girls usually experience depressive symptoms at younger ages in comparison to boys. Regarding race, biracial adolescents have the highest rates of MDE (15.6%), followed by White non-Hispanic (13%) and Hispanic (12%) adolescents (Mojtabai et al., 2016).

**Impact of Depression in Adolescents**

The negative consequences of adolescent depression are multiple. Depression is related to distress and often impairment in daily functioning (APA, 2013). Depression has been related to poor academic performance (Derdikman-Eiron et al., 2011) and poor social well-being, which refers to negative perceptions of relationships with peers (Verboom, Sijtsema, Verhulst, Penninx, & Ormel, 2014). Depression has high comorbidity with anxiety disorders (Cummings, Caporino, & Kendall, 2014). It is worrisome that many adolescents with depression go under treated (Mojtabai et al., 2016). Even more serious, adolescents who experience depression are at higher risk of committing suicide or having suicide thoughts (Twenge, Joiner, Rogers, & Martin, 2018).

Even though some adolescents may not meet full diagnostic criteria for a MDE, they may still experience some symptoms that are impactful. An area addressing the symptoms that has obtained attention in the past decade is the impact of subthreshold depression (SD) in children and adolescents. SD refers to the presence of depressive symptoms that is below the threshold for major depressive disorder (Rivas Rodríguez, Nuevo, Chatterji, & Ayuso-Mateos, 2012). SD is quite common among adolescents (Wesseihoeft, Sørensen, Heiervang, & Bilenberg, 2013). Adolescents with SD also experience impairment in their daily functioning similar to those with
MDE, such as high rates of suicidal ideation and problems at school (Carrellas, Biederman, & Uchida, 2017; Keenan-Miller, Hammen, & Brennan, 2007). Also, SD increases the risk of having a MDE (Shankman et al., 2009). Therefore, the presence of depressive symptoms should be taken seriously, not only because the impairments they generate, but also because they are related to many other problems that impact youth.

**Depressive Symptoms and Social Cognitions of Smoking**

Little is known about possible associations between the presence of depressive symptoms and perceptions within the context of health risk behaviors. Some researchers have investigated the susceptibility to smoking and its relationship to other factors. For example, Pierce et al. (1996) show the validity of the *susceptibility to smoking index* as a predictor of smoking in adolescents. This index measures an individuals’ intention to smoke and their self-efficacy to refuse cigarettes in the future. A study conducted with adolescents (ages 12 to 17) from Malaysia revealed that students with depressive symptoms were more susceptible to smoking in comparison to those who did not have high levels of depressive symptoms (Lim, Chong, Khoo, & Kaur, 2014). In this study, susceptibility to smoke referred to adolescents who answered “yes” to any of these questions: a) Do you think you will smoke a cigarette in the next year? b) If one of your best friends were to offer you a cigarette, would you smoke? This is one of the few studies that has added the variable of depressive symptoms as a factor when studying the intentions to smoke. Another study conducted with young adolescents (mean age of 14) in the US, showed that a specific depressive symptom was associated with susceptibility to smoke. Stone, Audrain-McGovern, and Leventhal (2017) reported that among never-smokers, anhedonia, which was defined as diminished capacity to experience pleasure, was associated
with more curiosity about smoking, willingness to smoke, and intention. All of those factors predicted smoking initiation.

The Relationship between Smoking and Depression in Adolescents

The relationship between depression and smoking in adolescents is complex. The way the relationship has been studied varies based on distinct methodologies used, which is further complicated by a variety of distinct population characteristics that have been studied (e.g., younger vs. older adolescents, girls vs. boys, and clinical vs. non-clinical samples) (Beal et al., 2014; Wilkinson et al., 2016). Researchers have been investigating the mechanisms and the nuances of this association among adults and adolescents. Some researchers have studied directionality and temporal precedence of the relationship (Audrain-McGovern et al., 2009; Wilkinson et al., 2016), while others explored factors that moderate and mediate the relationship (Chaiton et al., 2015; Maslowsky & Schulenberg, 2013).

There is comorbidity between depression and smoking (Conway et al., 2017; Polednak, 2014; Weinberger et al., 2016). Richardson, He, Curry, and Merikangas (2012) explored the association between cigarette smoking and major depression/dysthymia in a sample of 1,884 adolescents from the U.S. ranging from ages 12 to 19. The results showed that rates of depression episodes (using a structured diagnostic interview based on the DSM-IV) were increased in adolescents that had ever smoked cigarettes. Similar results were reported by Redner, White, Harder, and Higgins (2014), who found that adolescents who met criteria for a diagnosis of a major depressive disorder have increased odds for current cigarette smoking.

In a study with a much bigger sample size from all 50 states, Polednak (2014) examined differences in prevalence rates of current cigarette smoking (i.e., smoking at least one cigarette in the past 30 days) and daily smoking (i.e., 30 cigarettes in a month) among adolescents with and
without a past-year major depressive episode in the U.S. from 2004 to 2010. Results showed that although cigarettes smoking declined among adolescents, the prevalence of current cigarette smoking in 2010 continued to be twice as high for those who experienced a depressive episode in the past year. Similarly, Goodwin et al., (2017) studied changes in the prevalence of depression among current, former, and never smokers in the U.S. population from 2005 to 2013 examining age, gender, and income in a large sample of 496,805 participants. The results were consistent with what Polednak and colleagues found; although depression increased in all groups (i.e., current, former, and never smokers), a marked increase was seen among adolescent current smokers (ages 12 to 17). More specifically, there was an increase from 16% in 2005 to 22% in 2013 for those who experienced depression and were current smokers. These studies suggest a co-segregation of the two syndromes.

The high prevalence of smoking among individuals with depression is not only seen in cases of major depressive disorders, but also among individuals who experience high numbers of depressive symptoms or subthreshold depression (SD) (Skrove, Romundstad, & Indredavik, 2013). Most of the studies investigating the relationship between smoking and depression use questionnaires to measure depressive symptoms and not diagnostics interviews (Audrain-McGovern et al., 2009; O’Loughlin et al., 2017; Prinstein & La Greca, 2009; Wilkinson et al., 2016). There has been great interest in investigating whether smoking has an etiologic role in depression onset or if it is the opposite, that depression leads to smoking. Time specific of the association can only be established through prospective longitudinal studies (Caruana, Roman, Hernández-Sánchez, & Solli, 2015; Chaiton, Cohen, O’Loughlin, & Rehm, 2009). Typically, the relationship between depression and smoking has been explained from four perspectives or
pathways: 1) depression occurs first and leads to smoking, 2) smoking goes first and it leads to depression, 3) the association is bidirectional, and 4) there is no relationship.

**Depression Predicts Smoking**

Many studies have shown that presenting depressive symptoms during adolescence predicts future smoking (Clark et al., 2011; Maslowsky, Schulenberg, & Zucker, 2014; Weinstein & Mermelstein, 2013). The underlying mechanism of this pathway is not completely clear, but researchers have explained it using the self-medication hypothesis (Khantzian, 1997), which posits that people smoke to alleviate the symptoms of depression. According to this hypothesis, nicotine and the act of smoking work as antidepressant reinforcing cigarette use (Brody, 2006). Lawrence, Zubrick, Mitrou, and Sawyer (2010) showed that adolescents with depression were more likely to initiate smoking, progress to daily smoking, and smoke more heavily.

Prospective longitudinal studies have shown that depressive symptoms predict smoking onset during adolescence (Clark et al., 2011; Moon et al., 2010; Weinstein & Mermelstein, 2013). These observations are seen among students of different ages and sexes. In a study including middle and high school students, the results showed that adolescents who presented higher depressive symptoms at baseline were more likely to initiate smoking one year later (Moon et al., 2010). Similarly, Clark et al. (2011) found that depressive symptoms predicted cigarette use in early adolescents; depressive symptoms at sixth grade predict smoking two years in the eighth grade. More recent studies have shown similar results. For example, O’Loughlin et al. (2017) reported that depressive symptoms were risk factors for smoking initiation in early (11 and 12 years old) and middle adolescence (13 and 14 years old).
Depressive symptoms lead to smoking progression (i.e., from experimentation to regular smoking) and daily smoking (i.e., smoking at least one cigarette daily within a 30 day period) (Audrain-McGovern et al., 2009; Fröjd et al., 2013; Maslowsky et al., 2014). Fleming, Mason, Mazza, Abbott, and Catalano (2008) found that higher levels of depressive symptoms of students in 8th grade were positively associated with higher frequency of cigarette use in girls in 11th grade. According to Hooshmand, Willoughby, and Good (2012), students with higher depressive symptoms in 9th grade reported faster increases in cigarette use across all grades in high school. Similarly, Fröjd et al. (2013) showed that girls with depression at age 15 were twice as likely to smoke daily than girls without depression. Maslowsky et al. (2014) also showed that depressive symptomatology at the 8th grade was associated with higher numbers of cigarettes used in 12th grade. The results of these studies are consistent with older studies supporting the self-medication hypothesis, in which depression leads to smoking (Brook, Schuster, & Zhang, 2004; Covey & Tam, 1990; Escobedo, Reddy, & Giovino, 1998; Patton et al., 1998).

Although studies have shown evidence of that pathway, some researchers argue that the self-medication hypothesis does not have enough evidence to be supported (Lembke, 2012). Therefore, the fact that depression leads to smoking must be explained by other reasons than to alleviate the symptoms. Niemelä et al. (2009) showed that experiencing depressive symptoms at a very early age (e.g., 8 years old) is associated with increased smoking at age 18. In this case other factors outside the self-medication hypothesis may explain this relationship. Another study was conducted with children who were 10 to 12 years old at baseline and were followed for six years. In this study, the researchers found that higher levels of depressive symptoms during childhood predicted higher number of cigarette used during adolescence (Prinstein & La Greca, 2009). Intercorrelations among variables were found to be a better explanation of the
relationship. In addition, other factors may account for the association, such as expectations of smoking rewards (Audrain-McGovern, 2012) and interaction of conduct problems (Maslowsky & Schulenberg, 2013).

**Smoking Predicts Depression**

The literature has also provided support for the other pathway: smoking leads to depression. This directionality is often explained by the notion that the nicotine and other chemical components in cigarettes interfere with neurochemical processes in the brain and environmental factors (Lydon et al., 2014). Fewer studies support this pathway in comparison to depression leading to smoking (Fluharty et al., 2017).

In a longitudinal study that followed adolescents from grades 7th to 12th, Steuber & Danner (2006) found that adolescents who reported current smoking or previous smoking were more likely to experience depression. Furthermore, those adolescents who were regular smokers showed the highest levels of depression. Beal et al. (2014) found that the higher the number of cigarettes used, the higher the depression scores. In another longitudinal study, Meng and D’Arcy (2014) studied risks for depression by different factors. In the youngest group, including individuals from 12 to 24 years old, smoking regularly was associated with increased risk of having episodes of major depression 12 years later. These results are consisted with older studies that suggested that previous and current smoking leads to depression (Choi, Patten, Gillin, Kaplan, & Pierce, 1997; Goodman & Capitman, 2000; Hanna, Yi, Dufour, & Whitmore, 2001). A possible reason for the small numbers of studies showing that smoking leads to depression might be that most studies in which smoking predicted depression also showed bidirectional association between depression and smoking.
Bidirectional Association

There is evidence supporting bidirectional association between depressive symptoms and smoking. Chaiton et al. (2009) explored 15 longitudinal studies with non-clinical samples of adolescents to investigate the temporal ordering of smoking onset and depression. They found evidence supporting bidirectional association among the variables; but they also conclude that studies that used clinical measures of depression were more likely to find a stronger effect of depression predicting smoking. Audrain-McGovern et al., (2009) claimed to conduct the first empirical study that shows a bidirectional association between depressive symptoms and smoking in adolescents. By conducting a prospective study with a sample of 1093 adolescents, starting at age 14 and ended at 18, they found a bidirectional association between numbers of cigarettes smoked and depressive symptoms. More specifically, higher depressive symptoms at age 14 predicted smoking progression at 18 years old; while smoking progression predicted a deceleration of depression symptoms from ages 14 to 18.

Similar results have been found in other studies. For example, Tjora et al., (2014) investigated the relationship with both cross-sectional and longitudinal analysis in a sample of 924 participants that were followed from adolescence (13 years old) to adulthood (30 years old). The results varied based on the type of analysis conducted. In cross-sectional analysis, in all except one wave, daily smoking was associated with depression. However, in the final longitudinal model, significant results were found only during adolescence. For example, early smoking predicted early depression (wave one to wave two) and vice-versa, early depression predicted early smoking. In a study with high frequency cigarette use, Wilkinson et al., (2016) obtained results supporting a bidirectional association in females only: depressive symptoms
associated with later increase of cigarette use and smoking associated with later increase in depressive symptoms from adolescence to adulthood.

**No Relationship between Cigarette Smoking and Depressive Symptoms**

There is a smaller number of studies that have not found significant relationships between depressive symptoms and smoking in adolescents. Many of those studies included a large number of factors to see whether they predicted smoking behaviors. In a controlled study with 59 adolescents, half of them identified at-risk for substance abuse (Leff et al., 2003). Leff and colleagues (2003) found that depression was not a significant predictor of smoking initiation. Some studies have found no significant results in the relationship in the opposite direction. Hooshmand et al. (2012), conducted a longitudinal study with a large sample of high school students from Canada. The participants, who were in their freshman year at the beginning of the study, were followed each year until the senior year of high school. The results showed that smoking did not predict changes in depression in high school students. However, those with higher depressive symptoms when freshman showed greater smoking consumption. In a more recent study, O’loughlin (2017) found that depressive symptoms predicted smoking initiation in early and middle adolescence; however, the association was non-significant by late adolescence (17 years old).

**Gender Differences in the Relationship between Depression and Smoking**

While depression is more common in girls than boys (Essau, Lewinsohn, Seeley, & Sasagawa, 2010), cigarette smoking is more common in males than females (CDC, 2019). There is evidence to support that depressive symptoms predicts smoking more in girls than boys (Fleming et al., 2008; Fröjd et al., 2013). Fleming et al., (2013) found a positive relationship between depressive symptoms and the number of cigarettes used in females only. Whitbeck, Yu,
McChargue, and Crawford (2009) found similar results. In a sample of 743 indigenous adolescents from Northern Midwest and Canada, Whitbeck and colleagues found that depressed females reported higher rates of smoking compared to depressed males at the third wave when participants were in the range of 12 to 15 years old.

Fröjd et al. (2013) also found that the association varied by gender. A concurrent association between depression and smoking was detected among girls and boys, both at age 15 and at age 17. However, depression at age 15 emerged as a risk factor for smoking at age 17 among girls but not among boys. In a study with 951 adolescents followed from 8th to 11th grades, Wilkinson et al., (2016) found a bidirectional relationship between smoking and depressive symptoms for females only. In contrast, Weiss et al. (2008), in a study with Chinese adolescents, found no gender differences in the relationship; depressive symptoms were associated with higher risk of lifetime smoking for both girls and boys. Similar results were found by Weinstein and Mermelstein (2013), boys with higher depressive symptoms at baseline were more likely to be stable smokers overtime, while this pattern was not observed among females. In addition, only boys were more likely to be smokers than no smokers if they experienced depressive symptoms at baseline. It is important to note that others studies have found no differences in the association by gender (Andrea H. Weinberger et al., 2016; Weiss et al., 2008).

**Conclusion**

The literature regarding the association between cigarette smoking and depression is inconclusive. Mixed results have been found in both adults and adolescents. Although it is clear that smoking has decreased, it represents a public health problem given the multiple dangers it portrays. While some researchers report that cigarette smoking occurs first and leads to
depression, others argue that having depression makes adolescents more vulnerable to smoking. Only prospective studies following individuals since childhood through adulthood can explore the directionality of the association. However, many authors have suggested exploring other mechanisms of the association in different populations and with different methods.

**Aims and Hypotheses**

The purpose of the current study was to explore cigarette smoking and depression from multiple dimensions. Data were collected from a longitudinal study funded by the National Institutes of Health (NIH), which aimed to understand health risk behaviors and cigarette smoking in a sample of adolescents from Iowa. Based on a review of the current literature, the following aims of the study were outlined:

1. To determine whether there is a relationship between cigarette smoking and depressive symptoms in a sample of sophomore high school students.
   a. Hypothesis: A relationship exists between cigarette smoking and depressive symptoms in sophomore high school students after adjusting for sex, race, and household income.

2. To examine whether smoking during the sophomore year of high school predicts depressive symptoms a year later (in the junior year).
   a. Hypothesis: Cigarette smoking during the sophomore year will be predictive of severity in depressive symptoms during the junior year after controlling for sex, race, and household income.

3. To examine whether depressive symptoms during the sophomore year of high school predicts cigarette smoking in the junior year.
a. Hypothesis: Depressive symptoms will predict cigarette smoking in the junior year after controlling for sex, household income, and depressive symptoms.

4. To explore if there is a relationship between depressive symptoms and social cognitions that are antecedents of smoking in the PWM.

   a. Hypothesis: Depressive symptoms are associated with the prototypes and willingness of smoking
CHAPTER III

METHODS

The current study is based on data that were collected from a longitudinal study funded by the National Institutes of Health (NIH). The Healthy Iowans Study was conducted at the University of Iowa and at the University of Connecticut by the principal investigators: Robert Philibert (M.D., Ph.D.) and Meg Gerrard (Ph.D.), respectively. The main purpose of the longitudinal study was to improve the understanding of the trajectory of smoking behaviors in adolescents who are at risk for smoking.

Participants

Participants included 448 high school sophomores and one of their parents or legal guardians who were recruited for the longitudinal study between the period of March 2015 to March 2018. These participants were drawn from seven high schools in the eastern Iowa area. Students from a diverse range of genders, racial, and ethnic backgrounds were included in the study. To be part of the study, students had to meet two eligibility requirements: 1) being a sophomore (enrolled in the 10th grade of high school) and 2) having a friend who smokes. The rationale for this selection criteria was based on the purpose of the longitudinal study. Most adult smokers begin smoking during adolescence (Institute of Medicine, 2015). Therefore, if one was interested in improving the prevention of smoking, it makes sense to study the trajectory of smoking in adolescents. The rationale for the second inclusion criterion was straightforward. It is well established that having a peer who smokes is a major risk factor for beginning smoking (Fujimoto & Valente, 2012; Haas & Schaefer, 2014). Therefore, by examining adolescents who have friends that smoke, the researchers were more likely to identify those students that were at higher risk for smoking. Thus, the researchers could be able to learn about the environmental
factors around those at-risk students. In the current study, the sample consisted of 438 participants. Two of the students withdrawn after signing the consent forms. The other eight participants were removed from the analyses for not having available data in one or more of the main variables (i.e., cigarette smoking and/or depressive symptoms). Demographic information included in the analyses were: sex (female or male as observed by the interviewer), race/ethnicity (Caucasian/Non-Hispanic origin and others), household annual income (a range of six categories that included salary from both parents or guardians; the midpoint was used in the analyses).

**Procedures**

Permission was granted by the principal investigators of the longitudinal study for the use of the data for this dissertation. Data were collected under a protocol approved by the University of Iowa Human Subjects Institutional Review Board. The principal investigators obtained permission from the school leadership board at each district to have access to the publically available names, addresses and phone numbers of each high school sophomore in that district (seven high schools from eastern Iowa). A letter was sent to their homes and follow-up phone calls were made to recruit participants for the study. Once the students voluntarily decided to participate, research assistants with appropriate training scheduled in-person meetings at the students preferred locations, which usually were the students’ homes or public libraries.

During the initial visit, a consent form was explained to the students and to one of their parents or guardians. All participants who were a minor provided written assent and their parents provided written consent. As part of the longitudinal study, participants agreed to be contacted five times over a period of two years. Participants who turned 18 years of age during the duration of the study provided their own written consent. Participants received a monetary compensation of $300 during the two years that they were enrolled in the longitudinal study.
The distribution was the following: $100 during the intake visit, $10 for a phone call at six months of enrollment, $80 for a visit in-person after one year, $10 for a phone call at 18 months, and $100 for a visit in person after two years.

For the current study, only data from the initial visit and the one-year follow-up visit were used. The data collection for the main study began in March 2015 and will continue through the summer of 2019. During the initial visit students were asked to complete the following activities: a) an interview, b) a blood draw, and c) questionnaires. In the same initial visit, one of their parents or guardians completed a short interview about substance use and demographic information. During the one-year follow-up visit, students completed questionnaires and had their blood drawn. This study utilized demographic information and factors included as covariates that were generated from the interview, the questionnaire provided information about depressive symptoms, and blood samples were used to test for smoking.

Once the data were collected, the interview and questionnaire responses were double entered by research assistants into a secure database. The data was inspected and cleaned to ensure accuracy. The blood drawn was processed and analyzed by Dr. Philibert and his team at the Psychiatric Genetics Laboratory at the University of Iowa. The methodology used to obtain the biomarker followed the procedures detailed in another study (see Philibert et al., 2013 for more details). During the initial and final visits, students completed questionnaires and blood draws. The interview, which included questions about demographic information and psychiatric disorders, was only completed during the initial and final visit.

**Research Assistants' Training**

The research assistants received ongoing training, as needed, by a research specialist in the following areas: recruitment strategies, use of structured interviews, and referral procedures
for suicidality risk. This training process was informal and cumulative based on the needs of the team and lasted for several months; the number of training is unknown, given that all the research assistants did not join the team at the same time. The training in recruitment strategies consisted of learning how to make phone calls to increase the number of participants (e.g., knowing what the best time of the day was to call possible participants, developing a script, and completing detailed records about each phone call attempt). Training regarding structured interviews consisted of getting familiar with the questions and practicing the administration of the interview, which require the interviewer to be skilled on skipping questions and/or sections based on the responses.

Regarding procedures to follow about suicidality risk, a child psychiatrist who was assigned to work with the longitudinal study, provided guidelines to follow when a student reported suicidal thoughts. The guidelines consisted of asking participants a series of questions and then informing the child psychiatrists, who would follow up with the participants and their parents. In addition, bi-weekly team meetings were conducted to discuss the study's progress. Most of the research assistants received formal training on phlebotomy and were certified phlebotomists before their involvement with the study.

**Instruments**

**Quantitative Measure of Cigarette Consumption**

Smoking was measured using a biomarker that represents a quantitative measure of cigarette consumption. This biomarker was studied through specialized blood analyses. According to Atkinson et al., (2001) a biomarker is defined as “a characteristic that is objectively measured and evaluated as an indicator of normal biological processes, pathogenic processes, or pharmacologic responses to a therapeutic intervention.” In this case, the biomarker studied was
an indicator of a biological process that was altered after smoking (Philibert et al., 2012). This is an innovative epigenetic tool to measure smoking initiation and changes in its trajectory that is not yet accessible to the clinical/medical community or the general population. This instrument provides a number that represents the probability of daily smoking. The smallest the number, the higher the probability of daily smoking. Values < 82 are suggestive of daily smoking. For more details, review the “Biological measures of cigarette use” section in Chapter 2.

**Patient Health Questionnaire-9 (PHQ-9)**

Depressive symptoms were measured through the PHQ-9. The PHQ-9 is a nine-item self-report version from the Primary Care Evaluation of Mental Disorders (PRIME-MD). This instrument was developed by Robert L. Spitzer, Janet B.W. Williams, Kurt Kroenke, and colleagues in 1998 with a grant from Pfizer US Pharmaceutical (Kroenke, Spitzer, & Williams, 2001). The nine items cover the following experiences within a period of the past two weeks: pleasure, feeling down, sleep disruption, energy levels, appetite, feeling a failure, trouble concentrating, speaking slowly or being fidgety and having negative thoughts concerning suicide or self-harm (Williams, 2014). Each item of the PHQ-9 requires a response on a 4-point scale, ranging from 0 (never) to 3 (nearly every day). Responses are recorded and added for a total score ranging from 0 to 27. Higher scores indicate increased likelihood of meeting criteria for current major depressive disorder (Kroenke et al., 2001). However, by itself, this instrument alone cannot be used to diagnose depression as a clinical disorder. The PHQ-9 is only another tool that medical doctors and psychologists can use together with other clinical techniques to assess the presence of depression. The PHQ-9 is a widely used self-report measure of depressive symptom severity in clinical and research settings (Williams, 2014). Some of
the practical benefits of using this instrument are that it is free, easily accessible online, brief, and easy to understand and score (Richardson et al., 2010; Williams, 2014).

The psychometric properties of the PHQ-9 have been studied in a variety of populations, including adolescents in several countries (Andreas & Brunborg, 2017; Ganguly et al., 2013; Tsai et al., 2014). Kroenke et al., (2001), evaluated the psychometrics properties of the PHQ-9 in a sample of 6,000 patients from eight primary care clinics and seven obstetric-gynecologist care clinics. The internal reliability based on Cronbach’s α equaled 0.89 in the primary care clinic study and 0.86 in the obstetric-gynecologist study. The researchers found that a total score of ≥10 had a sensitivity (detecting depression in individuals who really have depression) of 88% and a specificity (detecting those who do not have depression) of 88% for current major depression (Kroenke et al., 2001).

In a sample of 442 adolescents from the U.S. ranging from 13 to 17 years old, a cutoff score of 11 on the PHQ-9 was found to give 89.5% sensitivity and 77.5% specificity for meeting criteria for major depression on the DSM-IV (Richardson et al., 2010). The same group of researchers found that in a receiver operating characteristic curve (ROC curve), the total score in the PHQ-9 had an area under the curve of 0.88. Andreas and Brunborg (2017) used a sample of 846 adolescents from Norway to see whether the PHQ-9 was as good as other instruments to measure depression. A confirmatory factor analysis showed that the PHQ-9 measures a unidimensional theoretical construct for boys and girls; they reported RMSEA = 0.073, CFI = 0.93. They also found Cronbach's alpha, for boys of 0.81 and for girls of 0.88. In terms of convergent validity, specific risk factors for depression, such as lack of close friendship and history of suicide attempts in the Strengths and Difficulties Questionnaire (SDQ), were significantly associated with higher total depressive symptom in the PHQ-9 (Andreas &
Brunborg, 2017). For each one-point increase in the SDQ Emotional Problems scores, there was a significant increase of 1.17 point in boys and 1.33 point in girls in the PHQ-9 scores. They reported standardized regression coefficients of $\beta = 0.54$ for boys and $\beta = 0.63$ for girls, $p < 0.001$.

**Cigarette Smoking Questionnaire**

Social cognitions of smoking (i.e., prototype and willingness) were measured using a questionnaire that was developed by the principal investigators of the main longitudinal study. This questionnaire includes eight questions (three about willingness to smoke cigarettes and five about perceptions of smokers or “prototypes”). In the questions about willingness, participants were asked the following “Suppose you were with a group of kids and there were some cigarettes that you could have if you wanted. How willing would you be to do the following things: 1) take a puff, 2) take one and smoke it, 3) take some cigarettes for you for later?” and they had three options: 1) not at all willing, 2) kind of willing, and 3) very willing. In the aim number four, each item or question was treated independently. Then, a composite of “willingness” was calculated based on the average of the three items. To measure the internal consistency of the willingness composite, a Cronbach’s alpha was calculated. The Cronbach’s $\alpha$ of the composite of willingness was .83.

The five questions about prototypes began with the following statement: “A number of young people your age smoke. I want to know what you think about them. Take a moment and think about the type of kid your age who smokes. I am not thinking about anyone in particular, just your image of kids who smoke.” Then, participants were asked “Type of kids your age who smoke cigarettes: 1) How popular are they? 2) How smart are they? 3) How cool are they? 4) How attractive are they? 5) How boring are they? (this item was reversed). The participants had four
options: 1) not at all, 2) a little bit, 3) kind of, and 4) very. The composite of prototypes had a Cronbach’s α of .74.

**The Semi-Structured Assessment for the Genetics of Alcoholism for Adolescents**

The Semi-Structured Assessment for the Genetics of Alcoholism for Adolescents (CSSAGA-A-IV) was used in the current study to measure self-reported smoking and to obtain demographic characteristics of participants. Self-reported of smoking data was collected by asking participants to answer the question “Have you ever smoked a full cigarette?” Only two answers were available: yes or no. Demographic data obtained from this instrument included sex and race/ethnicity. Household income data was obtained as part of brief interview conducted to one of the participants’ parents or legal guardians.

The CSSAGA-A-IV is a semi-structured interview developed specifically for the Collaborative Study on the Genetics of Alcoholism (COGA) (Washington University School of Medicine, 1998) (Begleiter et al., 1995). COGA is a multisite and multidisciplinary project with the main objective of studying the familial transmission of alcoholism. The University of Iowa was one of the nine academic centers where data collection, analysis, and storage took place (Ohannessian et al., 2005).

The C-SSAGA-A-IV is a modified version of the SSAGA, which was designed to use with adults. The development of the SSAGA interviews was supported by an NIH grant from the National Institute on Alcohol Abuse and Alcoholism (NIAAA). The C-SSAGA-A-IV has gone through four phases of changes and it can be used with adolescents ages 13 to 17. The SSAGA interviews were designed to assess the social, physical, and psychological
manifestations of alcohol abuse, substance use or dependence, and other psychiatric disorders, such as depression and antisocial personality disorder (Kuperman, Schlosser, Lidral, & Reich, 1999). It follows a structure that facilitates diagnostic criteria from the DSM-IV. It also covers general demographic information and medical history. The C-SSAGA-A has been used in several studies with adolescents (Ohannessian et al., 2005; Sartor et al., 2013). The C-SSAGA-A-IV was modified to be used in the larger longitudinal study; sections and/or items that were not relevant for the study were deleted by the principal investigators.

**Data Analysis**

**Aim One**

The first aim was to determine whether there was a relationship between cigarette smoking and current depressive symptoms in a sample of sophomore high school students that had friends who smoke (n = 438). Two different measures of cigarette smoking were used: a) a quantitative measure of cigarette smoking suggestive of daily smoking (which was called the “biomarker” in the current study), and b) a self-reported answer to the question “have you smoked at least one cigarette in your lifetime?” To address this aim, the following analysis was conducted:

1) A hierarchical linear regression with depressive symptoms as the dependent variable and cigarette smoking (measured by the biomarker) and self-report of smoking as the main independent variables. The variables were entered in the following order; in the first step, demographic variables were entered as control variables, including sex (females or males), race (Caucasian/White or others), and household income (midpoints of the category). In the second step, cigarette smoking (measured by the biomarker) was entered; in the third step, self-report of smoking; and in the fourth
and final step, interaction terms between demographic variables and the main independent variables were entered (i.e., sex * cigarette smoking, race * cigarette smoking, income * cigarette smoking, sex * self-report, race * self-report, and income * self-report).

When the assumptions of multiple regression were evaluated, it was evident that some of the assumptions were violated, including linearity, outliers, normality, and homoscedasticity. To address these violations, the dependent variable (depressive symptoms) was transformed by a base 10 logarithmic transformation, given that the original distribution was strongly positively skewed. Once transformed, the regression analysis was run one more time; then, the data were examined to decide whether the transformation was successful. Transforming the dependent variable successfully addressed the violations. There was independence of residuals, as assessed by a Durbin-Watson statistic of 2.03. There was no strong evidence of multicollinearity, as assessed by tolerance values greater than 0.80. There were no studentized deleted residuals greater than 3 standard deviations, no leverage values greater than 0.2, and values for Cook's distance above 1. The assumption of normality was met, as assessed by a normal Q-Q plot. A visual inspection of a plot of studentized residuals versus unstandardized predicted values revealed that there was heteroscedasticity, even after transforming the dependent variable. Therefore, the results had to be interpreted with caution considering that differences in variance lower the precision of the coefficient estimates.

To address the presence of outliers, the four subjects with outliers were removed from the sample, then, the regression was conducted one more time. Given that there was no significant difference in the results (e.g., similar $R^2$ and $p$ values), the outliers were kept in the analysis; these outliers represented data from adolescents who had higher levels of depressive symptoms.
and those with lower methylation level (which were indicators of cigarette smoking). After evaluating that the assumptions of linear regressions were met, the variable cigarette smoking was reversed to facilitate the interpretation of results. In this way, higher values mean higher probability of daily smoking, as higher values of depressive symptoms meant more severity of symptoms. A simple linear transformation was conducted using the following formula: -1(original score) + 1.5.

**Aim Two**

The second aim was to examine whether smoking during or prior to the sophomore year of high school predicted depressive symptoms a year later, during their junior year of high school. Only participants that completed the study during the junior year of high school and had interpretable values of cigarette smoking (as measured by the biomarker) were included in the analysis (n = 264). From the 266 participants who completed the interview and blood draw during their junior year, only two did not have the necessary data from both variables. Some reasons for not having interpretable data included errors in the blood analyses procedures and errors from the research assistants when collecting the data. To address Aim Two, the following analysis was conducted:

1) A hierarchical multiple regression with depressive symptoms from the junior year of high school as the dependent variable and cigarette smoking from the sophomore year of high school (as measured by the biomarker) as the independent variable. In the first step, demographic variables (i.e., sex, race/ethnicity, and household income) were entered as control variables. In the second step, cigarette smoking (measured by the biomarker) was entered. In the third and final step, interaction terms between demographic variables and cigarette smoking from the sophomore year of high school
were entered (i.e., sex * cigarette smoking, race * cigarette smoking, income * cigarette smoking).

The assumptions of hierarchical multiple regression were examined revealing that the following assumptions were violated: linearity, outliers, normality, and homoscedasticity. To address these violations the dependent variable (depressive symptoms) was transformed using a logarithmic transformation, because the original data were strongly positive skewed. The transformation was successful. There was independence of residuals, as assessed by a Durbin-Watson statistic of 2.01. Homoscedasticity was supported, as assessed by visual inspection of a plot of studentized residuals versus unstandardized predicted values. There was no compelling evidence of multicollinearity, as assessed by tolerance values greater than 0.91. There were no studentized deleted residuals greater than 3 standard deviations, no leverage values greater than 0.2, and values for Cook's distance above 1. The assumption of normality was supported, as assessed by a normal Q-Q plot. To have all the variables in the same direction, the variable cigarette smoking was reversed using a simple linear transformation with the following formula: 

\[-1(\text{original score}) + 1.5.\]

**Aim Three**

The third aim was to examine whether symptoms of depression during the sophomore year predict cigarette smoking in the junior year. To address this aim, the following analysis was conducted:

1) A hierarchical linear regression with cigarette smoking from the junior year as the dependent variable entering demographic variables (i.e., sex, race/ethnicity, and household income) first as control variables, and then depressive symptoms from the sophomore year as the main independent variable. Interaction terms between
demographic variables and depressive symptoms from the sophomore year of high school were entered in the third step (i.e., sex * depressive symptoms, race * depressive symptoms, and income * depressive symptoms).

When the assumptions of hierarchical multiple regression were examined, the following assumptions were violated: linearity, normality, outliers, homoscedasticity. To address these violations, the dependent variable (cigarette smoking) was transformed using a logarithmic transformation, given that it was strongly negatively skewed. The transformation was successful. Independence of residuals was supported, as assessed by a Durbin-Watson statistic of 1.918. Linearity was supported by using a plot of studentized residuals against the predicted values. Homoscedasticity was supported, as assessed by visual inspection of a plot of studentized residuals versus unstandardized predicted values. There was no compelling evidence of multicollinearity, as assessed by tolerance values greater than 0.91. There were no studentized deleted residuals greater than 3 standard deviations, no leverage values greater than 0.2, and values for Cook's distance above 1. The assumption of normality was supported by the inspection of the normal Q-Q plot. There were five outliers in the data; they were kept in the analyses given that there was no significant difference in the results with and without the outliers (i.e., similar $R^2$ and $p$ values). Then, a simple linear transformation was done to the dependent variable, cigarette smoking, to facilitate interpretations of results. The following formula was used: $-1(\text{original score}) + 1.5$.

**Aim Four**

The fourth aim was to investigate whether a relationship existed between depressive symptoms and the following social cognitions: prototypes and willingness. To address this aim, the following analysis was conducted:
1) Spearman’s rank order correlations

**Additional Analysis**

According to studies using the PWM, prototypes and willingness to smoke are considered antecedents of smoking. An additional analysis was conducted to explore whether prototypes and willingness predicted cigarette smoking in this sample. The following analysis was conducted:

1) A hierarchical linear regression with cigarette smoking from the sophomore year of high school as the dependent variable. Demographic variables (i.e., sex, race/ethnicity, and household income) were added in the first step, the willingness composite in the second step, and the prototype composite in the third step.

After running the analysis, the assumptions of multiple regression were examined. To avoid violation of assumptions, the transformed variable of cigarette smoking was used as a dependent variable. Independence of residuals was supported, as assessed by a Durbin-Watson statistic of 1.81. Linearity was supported by a plot of studentized residuals against the predicted values. Homoscedasticity was supported by visual inspection of a plot of studentized residuals versus unstandardized predicted values. There was no compelling evidence of multicollinearity, as assessed by tolerance values greater than 0.83. There were no studentized deleted residuals greater than 3 standard deviations, no leverage values greater than 0.2, and values for Cook's distance above 1. The assumption of normality was supported by the normal Q-Q plot. There were five outliers in the data; they were kept in the analyses given that there was no significant difference in the results with and without the outliers (e.g., similar $R^2$ and $p$ values). Then, a simple linear transformation was done to the dependent variable, cigarette smoking, to facilitate interpretations of results. The following formula was used: $-1(\text{original score}) + 1.5$. 
CHAPTER IV

RESULTS

This chapter includes the results of the statistical analyses used to examine the study aims: 1) determine whether there was a relationship between cigarette smoking and depressive symptoms in a sample of sophomore high school students, 2) examine whether smoking during the sophomore year predicted depressive symptoms in the junior year, 3) examine whether symptoms of depression during the sophomore year predicted cigarette smoking in the junior year, and 4) investigate whether a relationship existed between depressive symptoms and social cognitions of smoking (i.e., prototypes and willingness). First, descriptive demographic data are presented followed by the findings of the four study aims. An alpha level of .05 was used in all the analyses. IBM SPSS Statistics 25.0 software (IBM Corporation Armonk, NY) was sued for all statistical analyses. Assumptions were checked prior to evaluating results to determine appropriateness of the chosen statistical analysis (described in Chapter III).

Demographic and Descriptive Data

There were 448 participants in total when the data were collected (when the participants were in the sophomore year of high school). Eight participants were removed from the sample during the data analysis process because of missing data from the main variables of the current study (i.e., depressive symptoms and cigarette smoking). A total of 440 participants (Mean age: 15.70; SD = 0.57) were included in the data analyses for the current study. There were 243 females (55.23 %) and 197 males (44.77 %). From this sample, 306 (69.55 %) of the participants were Caucasian/White and 134 (30.45 %) were from other racial and ethnic backgrounds, including Caucasian/Hispanics, Black/African Americans, Asians, Native Americans, Pacific Islanders and mixed racial backgrounds. Two of the participants did not
specify their racial/ethnic background. From the 440 participants, 40 % came from families with a yearly household gross income of $100,000 or more, as reported by their parents. More details of demographic data appear in Table A1 of the Appendix.

Although the variables of cigarette smoking and depressive symptoms were treated as continuous in the analyses, they were also categorized to describe the behaviors in the sample. During the sophomore year, 39 (8.86 %) of the adolescents were smokers, based on methylation levels of < 82 %, which is suggestive of daily smoking. In the junior year, the available data included 264 participants. From this group, 31 (11.74 %) had methylation levels suggestive of daily smoking. Regarding depressive symptoms, 46 (10.45 %) of adolescents in the sophomore year presented at least moderate depressive symptoms as indicated by total scores in the PHQ-9 of ≥ 11. In the junior year, 27 (10.23 %) had moderate to severe depressive symptoms.

Descriptive statistics of all the variables included in the study are detailed in Table A2.

Aim One

The first aim was to determine whether there was a relationship between cigarette smoking and depressive symptoms in a sample of sophomore high school students. To address this aim, a hierarchical multiple regression was conducted. Bivariate correlations among variables are reported in Table A3. Depressive symptoms were greater for females, non-Caucasian, and low-income individuals. Cigarette smoking was greater for non-Caucasian and low-income individuals; whereas self-reported cigarette smoking was greater for low-income individuals. Also, the greater reported self-reported smoking, the greater the depressive symptoms.

In the first step of the hierarchical regression, demographic variables were entered as control variables (i.e., sex, race/ethnicity, and household income). The demographic variables
significantly explained approximately four percent of the variance in depressive symptoms, $R^2 = .036$, $p = .001$. The statistically significant results for individual demographic variables within that block indicated that depressive symptoms were higher for females and lower income adolescents after controlling for the other demographic variables. In the second step, the main independent variable, cigarette smoking as measured by the biomarker, was entered in the model. Adding cigarette smoking to the regression model did not produce a significant change in the variance of depressive symptoms beyond the contribution of control variables, $\Delta R^2 = .001$, $F (1, 433) = .230$, $p = .63$. In the third step, a variable of self-report smoking was entered. The addition of self-report smoking to the model was statistically significant, $\Delta R^2 = .031$, $F (1, 432) = 14.54$, $p < .001$. It improved the model by 3.1% percent in explaining depressive symptoms. However, the effect size of the $R^2$ change was small (Cohen $f^2 = .03$). To examine whether the demographic variables had moderator effects in the main variables (i.e., cigarette smoking and self-report of smoking), interaction terms were added in the final step of the model. The result indicated that the interaction terms as a group were not statistically significant, $\Delta R^2 = .012$, $p = .470$, thereby providing no trustworthy evidence that relationships between depressive symptoms and cigarette smoking varied as a function of sex, race, or income. The result for the full regression model was statistically significant, $R^2 = .079$, $F (11, 4256) = 3.343$, $p < .001$; adjusted $R^2 = .056$. See Table A4 for full details on each regression model.

**Aim Two**

The second aim was to examine whether smoking during the sophomore year of high school predicted depressive symptoms in the junior year of high school. Only 264 of the participants (58.10% females and 41.90% males) had all essential data at the time the current study was conducted. To address the second aim, a hierarchical linear regression was conducted.
entering the demographic variables as control variables in the first step (i.e., sex, race/ethnicity, and household income). Intercorrelations among variables are reported in Table A5. Significant correlations showed that the variable sex was positively correlated with depressive symptoms, whereas income was negatively associated with depressive symptoms. Depressive symptoms were greater for females and low-income adolescents. The results from the first model revealed that the demographic variables explained approximately five percent of the variance in depressive symptoms, $R^2 = .047$, $F (3, 261) = 4.29$, $p = .006$. The statistically significant results for individual demographic variables within that block indicated that depressive symptoms were higher for lower income adolescents after controlling for the other demographic variables. In the second step, cigarette smoking as measured by the biomarker, was entered into the model. The addition of cigarette smoking did not yield a significant change in the explained variance for depressive symptoms, $\Delta R^2 = .000$; $R^2 = .048$, $F (1, 260) = .134$, $p = .715$. The addition of the interaction terms to the model also was non-significant, $\Delta R^2 = .013$; $p = .302$; providing no trustworthy evidence that relationships between depressive symptoms the following year and cigarette smoking varied as a function of sex, race, or income. The full model of the hierarchical regression was statistically significant, $R^2 = .061$, $F (7, 257) = 2.383$, $p = .022$; adjusted $R^2 = .035$ ($\text{Cohen } f^2 = .03$). Overall, the results revealed negligible relationships between cigarette smoking during the sophomore year of high school and depressive symptoms in the junior year of high school. See Table A6 for full details on the regression model.
Aim Three

The third aim was to examine whether depressive symptoms during the sophomore year of high school predict cigarette smoking in the junior year of high school. To address this aim, a hierarchical linear regression was conducted entering the demographic variables in the first step (i.e., sex, race/ethnicity, and household income) and depressive symptoms from the sophomore year of high school in the second step. Intercorrelations among variables are presented in Table A7. The correlation results showed that depressive symptoms were greater in females, non-Caucasians, and low-income adolescents. Cigarette smoking also was greater for non-Caucasian and low-income adolescents. In the first step, the demographic variables explained 4.5% of the variance in cigarette smoking $R^2 = .045, F(3, 260) = 4.04, p = .008$. Income was the only variable within this block to uniquely predict cigarette smoking after controlling for the other demographic variables, with cigarette smoking higher for low-income adolescents. Adding depressive symptoms in the second step did not yield a significant change in the variance explained, $\Delta R^2 = .001, R^2 = .045, F(1, 259) = .230, p = .632$. The interaction terms were non-statistically significant, providing no trustworthy evidence that relationships between cigarette smoking and depressive symptoms varied as a function of sex, race, or income. The result of the full model was non-statistically significant, $R^2 = .051, F(7, 256) = 1.979, p = .058$; adjusted $R^2 = .03$. Details of the models are presented in Table A8. Overall the results provide no compelling evidence that depressive symptoms experienced in the sophomore year of high school predict cigarette smoking in the junior year of high school.
**Aim Four**

The fourth aim was to investigate whether a relationship existed between depressive symptoms and eight social cognitions from the PWM, which are considered antecedents of smoking (i.e., prototypes and willingness). To address this aim, Spearman’s Rank Order Correlations was computed between depressive symptoms and the eight willingness and prototype items. Pearson correlations were also computed; see Table A9. There was a statistically significant, weak positive correlation between depressive symptoms and the five prototypes of adolescents their age who smoke cigarettes: popular $r_s = .12, p = .008$; smart $r_s = .16, p < .001$; cool $r_s = .19, p < .001$; attractive $r_s = .23, p < .001$, and boring $r_s = -.11, p = .02$.

Having elevated depressive symptoms was associated with having positive attitudes toward adolescents who smoke. There was a statistically significant, weak positive correlation between depressive symptoms and willingness to engage in smoking behavior. Specifically, willingness to take a puff $r_s = .21, p < .001$; willingness to smoke one cigarette $r_s = .22, p < .001$; and willingness to take a cigarette for later $r_s = .16, p = .001$. Sophomore high school students with more elevated depressive symptoms expressed more willingness to engage in smoking behaviors.

**Additional Analyses: Willingness and Prototypes Predicting Cigarette Smoking**

Additional analyses were conducted to explore whether prototypes and willingness predicted cigarette smoking concurrently during the sophomore year of high school. A hierarchical linear regression was conducted entering the demographic variables in the first step (i.e., sex, race/ethnicity, and household income), the willingness composite in the second step, and the prototype composite in the third step. Intercorrelations among variables were reported in Table A10. Adolescents from lower income household expressed more willingness to engage in smoking behaviors. Females expressed more positive attitudes toward adolescent who smoke
In the first step, the demographic variables significantly explained six percent of the variance in cigarette smoking, $R^2 = .060$, $F (3, 426) = 9.03, p < .001$. The addition of the willingness composite was statistically significant, improving the model beyond the contribution of control variables, $\Delta R^2 = .026$, $R^2 = .086$, $F (1, 425) = 12.23, p = .001$. Expressing more willingness to engage in smoking behaviors predicted smoking concurrently. However, the predictive contribution had a small effect size Cohen’s $f^2 = .02$. In the third step, the prototypes composite was added. Adding the prototypes composite did not had a significant change in the variance of cigarette smoking, $\Delta R^2 = .002$, $R^2 = .088$, $F (1, 424) = .745, p = .38$. The full model was statistically significant, $R^2 = .088$, $F (5, 424) = 8.15, p < .001$; adjusted $R^2 = .07$. See Table A11 for full details on the regression model.

**Summary**

The results of Aim One revealed that there was not a significant relationship between cigarette smoking and depressive symptoms when smoking was measured with a biomarker suggestive of daily smoking. However, self-reports of smoking at least one full cigarette significantly predicted depressive symptoms. In Aim Two, cigarette smoking during or before the sophomore year of high school did not predict depressive symptoms a year later, when students were in their junior year of high school. Similarly, results from Aim Three showed that depressive symptoms reported in the sophomore year of high school did not predict cigarette smoking a year later in the junior year of high school. In Aim Four, a significant association was found between depressive symptoms and social cognitions. More willingness to engage in specific smoking behaviors were associated with more elevated depressive symptoms. When the willingness and prototypes were examined as composites, only willingness to engage in smoking behaviors predicted smoking in the sophomore year of high school.
CHAPTER V
DISCUSSION

Adolescence is the period where most future adult smokers tried their first cigarette (Bernat et al., 2008; DHHS, 2014). It is also the period in which a high number of individuals experience elevated depressive symptoms (Twenge et al., 2018; A. H. Weinberger et al., 2017). Although most adolescents do not become regular smokers, earlier initiation predicts subsequent regular smoking (Reidpath et al., 2014). Cigarette smoking is associated with a high number of health and social problems, including depression, which affects adolescents’ overall well-being, (Dube et al., 2013; Fluharty et al., 2017b).

Research on the relationship between adolescent cigarette smoking and depression has shown mixed results. While some researchers have found that these two constructs are related (Audrain-McGovern et al., 2009; Tjora et al., 2014; A. L. Wilkinson et al., 2016), others have found that the association does not exist in late adolescence (Arnold, Greco, Desmond, & Rotheram-Borus, 2014; O’Loughlin et al., 2009). Variations in the constructs studied and the methods used provides a possible explanation for the variation in findings. Also, differences have been found between cross-sectional and longitudinal studies (Tjora et al., 2014). Banzer et al. (2017) found a positive association between emotional problems, including depression, and cigarette smoking in cross-sectional analysis, whereas Audrain-McGovern et al. (2009) concluded that the association was bi-directional in a longitudinal study. Finally, studies with more advanced analysis, such as Mendelian randomization, provide no evidence of causality (Taylor et al., 2014).

In the current study, I sought to address some of the limitations present in the literature. For example, a shortcoming discussed in previous studies is the exclusive use of self-report to
study cigarette smoking (Moon et al., 2010; Thompson et al., 2015). Although it is easy and convenient, asking adolescents to report their cigarette use may provide misleading information. For example, adolescents often underreport cigarette use due to fear of punishment, social stigma, and mistrust of researchers (Dube et al., 2013; Lantini et al., 2015).

The main purpose of the current study was to explore whether a relationship exists between cigarette smoking and depressive symptoms for high school students from Iowa using a biological measure of cigarette smoking and a short measure of depressive symptoms. The relationship between the variables were examined in various ways: 1) concurrent cigarette smoking and depressive symptoms in the first aim, 2) cigarette smoking in the sophomore year predicting depressive symptoms a year later in the second aim, 3) depressive symptoms from the sophomore year predicting cigarette smoking a year later in the third aim, and 4) whether depressive symptoms were associated with willingness to smoke and perceptions about smokers using the PWM in the fourth aim.

In the next section, the results of the current study are discussed in relation to the most recent literature in the pertinent areas. The discussion is divided into two main sections: 1) the relationship between cigarette smoking and depressive symptoms from different perspectives, as studied in aims one to three and 2) the social cognitions and their association with depressive symptoms and smoking.

**Relationship between Cigarette Smoking and Depressive Symptoms**

The first aim of the study was to examine whether a relationship existed between cigarette smoking and depressive symptoms in a sample of sophomore high school students who had at least one friend who smoked. I hypothesized that cigarette smoking and depressive symptoms would be associated. However, the results did not support this hypothesis. After
conducting a hierarchical multiple regression to control for demographic variables, the results showed that cigarette smoking was not related to depressive symptoms. Neither of the two smoking variables included in the first aim (i.e., cigarette smoking, as measured by the biomarker and the self-report smoking), explained much variance in overall depressive symptoms. Although the full model with all variables considered provided statistically significant results, the $R^2$ change after adding cigarette smoking was non-significant. Adding self-report of smoking in the last step was statistically significant; however, the change had a small effect size (Cohen’s $f^2 = .03$). Therefore, the relationship between cigarette smoking and depressive symptoms as measured here is weak.

These findings differ to what has been found in most studies with cross-sectional analyses in which different smoking behaviors have been related to depressive symptoms (Lee et al., 2018; Moon et al., 2010; Tjora et al., 2014). For example, Tjora et al., (2014) found that daily smoking was associated with depressive symptoms in cross-sectional analyses in almost all waves of data from the study, starting during early adolescence when participants were 13 years old to adulthood when they were 30 years old. Not only has daily smoking been associated with depressive symptoms, but also occasional smoking. In a more recent study conducted in Korea, Lee et al. (2018) showed that adolescents ages 12 to 18 with depressive symptoms were more likely to occasionally smoke in comparison to those who did not show elevated symptoms of depression.

Other researchers have reporting results similar to what was found in the current study. For example, Weiss et al. (2008) found a non-significant relationship between smoking and depressive symptoms for a sample of Chinese adolescents in seventh and eleventh grades. There was no association between smoking within the past 30 days and depressive symptoms. Only
“lifetime smokers” (which they defined as smoking at least a puff in the past 30 days) was associated with elevated depressive symptoms. Weiss and colleagues attributed the disparity of results to the small number of participants that reported smoking within the past 30 days.

It is possible that the relationship between cigarette smoking and depressive symptoms mostly occurs in those adolescents that have early onset of smoking and those who are younger, as was reported in some studies (Arnold, Greco, Desmond, & Rotheram-Borus, 2014; O’Loughlin et al., 2009). Arnold et al. (2014) found that when adolescents were between the ages of 12 to 14, they were four times more likely to have depressive symptoms if they were considered smokers. However, the likelihood of depressive symptoms for those who smoked within a period of 30 days was non-significant by the time the adolescents were 17 years old. In the current study, the participants were, on average, 15 years old at the beginning of the study, and 16 years old when in their junior year of high school.

**Cigarette Smoking Predicting Depressive Symptoms**

The purpose of the second aim was to examine whether cigarette smoking during the sophomore year of high school predicted depressive symptoms in the junior year of high school. I hypothesized that cigarette smoking would predict higher levels of depressive symptoms during the junior year. However, the results did not support this hypothesis. Having stronger evidence of daily smoking did not predict elevated depressive symptoms a year later. Fewer studies have supported this pattern of smoking predicting depression in comparison to depressive symptoms predicting smoking. The finding is consistent with what was found by Hooshmand et al. (2012) who concluded that smoking (as measured by the number of cigarettes smoked) did not predict levels of depression in high school students. In contrast, Steuber and Danner (2006) showed a pattern of increases in depression at the onset of smoking among adolescents.
Cigarette smoking is often a social event; when adolescents smoke, they tend to do so with friends or acquaintances (Haas & Schaefer, 2014). It was surprising to see the small number of adolescents from this sample who smoked, considering that they all had at least one friend who did. Given that both events (smoking and presence of depressive symptoms) may occur for a brief period of time, being a nascent smoker or smoking in low frequency does not necessarily interfere with how they feel a year later.

**Depressive Symptoms Predicting Cigarette Smoking**

The third aim of this study was to investigate whether depressive symptoms reported in the sophomore year of high school predicted cigarette smoking a year later. Similar to what was found in the second aim, having elevated depressive symptoms did not predict smoking in the junior year of high school. This finding is the opposite of what other studies have found in adolescents across high school. For example, Moon et al. (2010) found that higher levels of depressive symptoms predicted regular smoking two years later. Similarly, Clark et al. (2011), with a sample of younger adolescents, reported that having a depressed mood in the 6th grade predicted using cigarettes at some point in their lifetime. Fröjd et al. (2013) found similar results showing that girls who have depressive symptoms at the age of 15 were more likely to smoke daily at the age of 17. In a more recent study, Lechner, Janssen, Kahler, Audrain-McGovern, & Leventhal (2017) found that having higher depressive symptoms in 9th grade increased the odds to cigarette smoking onset a year later. Researchers have pointed out that it may be due to the self-medication hypothesis or to the fact that adolescents with elevated depressive symptoms tend to engage in other health risk behaviors, such as driving while drunk (Testa & Steinberg, 2010).
Findings in Context

In contrast to the findings summarized above, having elevated depressive symptoms in the present study did not predict cigarette smoking a year later. In the same way, smoking during or before the sophomore year of high school did not predict high or low levels of depressive symptoms. The great majority of the adolescents in the current study did not smoke and did not have elevated depressive symptoms. Only 8.86% of the sophomores in high school showed indications of smoking in the biological measure and 13.41% of them had elevated depressive symptoms of ≥ 10. Similar to what the literature indicates, depressive symptoms were more elevated among females and non-Caucasian adolescents; and among those from low-income households.

It is crucial to examine these results considering the constructs studied and the methodology used. Depressive symptoms were measured by the PHQ-9, a short instrument used mostly in the medical field, to screen for depression. The participants selected the intensity of symptoms that they experienced within a period of the last two weeks. Most of the studies that found significant relationships between the constructs of interest used the Center for Epidemiologic Studies – Depression Scale (CES-D). For example, Arnold et al. (2014), Lechner et al. (2017), and Moon et al. (2010) all used this instrument. This CES-D may be measuring a slightly different aspect of depressive symptoms than what is measured in the PHQ-9. The PHQ-9 is a measure of the severity of depressive symptoms that are part of the diagnostic criteria in the DSM-IV, whereas the CES-D does not assess the diagnostic criteria of appetite, anhedonia, psychomotor agitation or retardation, guilt, or suicidality (Smarr & Keefer, 2011).

In the present study, cigarette smoking was measured by a biomarker that can detect whether an individual has smoked cigarettes within the period of one year. The number or value
obtained for each participant represented the probability of daily smoking. Operationalization of smoking behaviors and definitions used vary greatly across studies. For example, researchers in other studies have examined smoking onset (Lechner et al., 2017; Wellman et al., 2016), daily smoking (Berg et al., 2013; Lee et al., 2018), and other variants of occasional smoking, such as lifetime smoking and smoking within the last 30 days (Clark et al., 2011; Fröjd et al., 2013). Because there are no previous studies using the present measure of smoking behavior, it is challenging to compare the results with previous findings. For example, in Clark (2011), smoking one cigarette was coded as “smoker,” whereas in the current study, an individual who smoked a single cigarette would not show enough demethylation levels (or temporary changes in the DNA) to be identified as a smoker. Also, it is important to note that the biological measure used in the present study is time dependent, in that smoking cigarettes more than a year ago would not be detected with accuracy (Andersen et al., 2017; R. Philibert et al., 2016). However, because participants were around 15 years old at the beginning of the study, it seems doubtful that many of them were smokers before the age of 14.

Overall, cigarette smoking and depressive symptoms have been associated in most studies using self-reports of smoking including this study. However, the addition of self-reports of smoking only explained three percent of the variance in depressive symptoms beyond that explained by demographic measures. Moreover, it is possible that studies showing no association were never published. Another factor to consider is that most studies used larger sample sizes (e.g., with 1000 or more participants), thus increasing the chance of finding significant results. Although the sample used in the current study seemingly had reasonable power to detect important effects, the lack of variability in characteristics of interest likely undermined the possible detection of such effects.
Social Cognitions: Associations with Depressive Symptoms and Cigarette Smoking

The fourth aim was to investigate whether depressive symptoms were associated with the social cognitions: willingness and prototypes. Correlational results revealed that depressive symptoms were positive associated with the prototypes of smokers and their willingness to smoke. However, the strength of the relationships were weak with $r_s$ ranging from .13 to .24 between depressive symptoms and prototypes, and from .16 to .22 between depressive symptoms and willingness. These results cannot be compared to other studies, because this was the first study to investigate constructs from the PWM in relation to depressive symptoms. However, there are some studies in the literature that have used similar constructs, such as susceptibility of smoking. Susceptibility is somewhat like the willingness presented in the PWM. The measure of willingness in the PWM is an explicit indication of individuals’ willingness to try smoking, whereas the susceptibility can be more subtle (e.g., the absence of a commitment to not smoke or not explicitly saying “no” to the possibility of smoking) (Pierce et al., 1996). Studies with this construct have found that susceptibility of smoking is associated with depressive symptoms.

For example, Lim, Chong, Khoo, and Kaur (2014) found that among adolescents in Malaysia, those with elevated levels of depressive symptoms were more likely to be susceptible to smoking. Also, in a study conducted with young adolescents, Stone et al. (2017) found that higher levels of anhedonia predicted smoking initiation susceptibility in early adolescence who had never tried a cigarette. The susceptibility in Stone and colleagues’ study included a “willingness” question: “Would you try cigarettes if one of your best friends offered it to you?” and participants answered “yes” or “no.” This is slightly different to what was measured in the current study, which was the level of willingness to take a puff, smoke a whole cigarette or take some cigarettes for later. However, both studies included a social component in their statement.
of “willingness.” It is also possible that willingness is more strongly associated with a specific depressive symptom (e.g., anhedonia) than to the total of all the symptoms. In the current study, elevated depressive symptoms were associated with expressing more willingness to engage in smoking behaviors (i.e., take a puff, smoke a cigarette, take some to smoke later). The exact reasons for this association are unknown. However, there are several possibilities to explain this association. It may be possible that adolescents would like to escape momentarily from the discomfort or state of mind of having elevated depressive symptoms. Also, it may be that they perceive the social environment of smoking as attractive.

Given that depressive symptoms were associated with the social cognitions of the PWM, it was of interest to do additional analysis to investigate whether those social cognitions or schemas were also associated with cigarette smoking after controlling for demographic variables. The results showed that willingness provided a statistically significant incremental contribution to predicting smoking, but prototype did not. This finding implies that it is more important how willing students are to engage in smoking than perceptions of others their age engaging in that behavior. This is not surprising considering that in previous studies with the PWM willingness was a stronger predictor of smoking (Gerrard et al., 2005). It is possible that today, adolescents are not strongly influenced by perceptions of others’ behaviors.

Implications for Practice

Although the results of this study were not completely in line with expectations, they may still provide a valuable addition to the literature. The fact that there was a positive association between depressive symptoms and willingness to smoke is something of interest. This finding suggests that, given the circumstances in an unplanned social situation, adolescents with elevated depressive symptoms may be more willing to try a cigarette. Although trying a
cigarette does not mean that they are going to progress to be smokers, they may also be more willing to engage in other risky behaviors. The presence of depressive symptoms not only affects their daily life functioning but also the way adolescents perceive others who engage in smoking. Determining how this association occurs is beyond the scope of the study. However, the findings can be used to encourage parents and professionals who serve adolescents to monitor adolescents’ behaviors and provide support.

**Limitations**

There are several limitations to this study that must be considered. One key limitation is the lack of variability of among students in cigarette smoking and depressive symptoms. Most participants did not smoke and did not report depressive symptoms. Absence of these problems is something positive for the overall well-being of adolescents. Uncovering stronger relationships between depression and smoking would likely require inclusion of more individuals at higher levels of these constructs. In addition, the individuals in the current sample are unlikely to be representative of students across the U.S., thereby limiting the generalization of results. Most participants came from upper-middle class families (i.e., 40% with an income of $100,000 or more) and included only those adolescents who reported having a friend who smoked. Another limitation was that the original data did not meet all assumptions of multiple regression analysis. While in most of the analyses transforming the data was a practical solution, in others it did not solve the problems (i.e., heteroscedasticity in aim 1). Because heteroscedasticity tends to produce p-values that are smaller than would otherwise be the case, power is reduced somewhat.

There are also limitations regarding the methods used. The PHQ-9 is a screener of depression, and it measures the intensity of those symptoms. Although the total of those
symptoms correlates well with having a major depressive episode (Andreas & Brunborg, 2017), the busy life of adolescents today may lead to elevated scores that do not necessarily mean that they are depressed long term. Having elevated symptoms does not necessarily mean that the adolescent’s level of functionality is seriously impacted overall. Moreover, this study was not focused on specific depressive symptoms, such as anhedonia, which may have a stronger role in the relationship with cigarette smoking. Although not an inherent problem within the study itself, the novel methods used do not coincide with those from most other studies. For example, the method to obtain the biological measure of cigarette smoking was not considered in previous research. This measure is expensive requires an equipped laboratory, specialized software, and trained staff (e.g., laboratory personnel and phlebotomist). Finally, the current study does not reflect causal relationships and does not address the directionality debate of which comes first, smoking or depression.

**Suggestions for Future Research**

Future studies should include larger and more heterogeneous samples, to increase the chance of finding relationships among the variables considered. Also, having more diversity in the sample in terms of race/ethnicity and socioeconomic status could improve the generalizability of results. Researchers could also make comparisons of relationships between demographic groups rather than simply using those variables as statistical controls. I further recommend that researchers explore the association of willingness to smoke and depressive symptoms in greater depth. Does a specific symptom influence how willing adolescents are to smoke? Is there a difference between different levels of severity? These are some questions that can guide future studies.
Conclusions and Summary

The overriding finding from this study was failure to obtain evidence of a relationship between cigarette smoking and depressive symptoms when smoking was measured more objectively by a biomarker, suggestive of daily smoking. Only self-reports of smoking at least one full cigarette significantly predicted depressive symptoms in sophomore high school students. However, the small effect size indicates that the potential explanatory effect was small and therefore of limited utility. Cigarette smoking during or before the sophomore year of high school did not predict depressive symptoms a year later, when students were in their junior year of high school. When studied in the opposite direction, similar results were found: depressive symptoms reported in the sophomore year of high school did not predict cigarette smoking a year later in the junior year of high school. A significant association was found between depressive symptoms and social cognitions. Willingness to smoke was positively associated with depressive symptoms. Also, positive prototypes or images of smokers were associated with depressive symptoms.
APPENDIX

TABLES

Table A1.

*Demographic Variables*

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<thead>
<tr>
<th>Variable</th>
<th>Frequency</th>
<th>Percentage</th>
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</thead>
<tbody>
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<tr>
<td>Female</td>
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<td>Male</td>
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<tr>
<td>$1-9,999</td>
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Note. Data collected when participants were in the sophomore year of high school.
Table A2.

**Descriptive Data**

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<td></td>
</tr>
<tr>
<td>Depressive symptoms</td>
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<td>4.22</td>
</tr>
<tr>
<td>Cigarette smoking</td>
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<td>3.44</td>
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<tr>
<td><strong>Junior Year of high School</strong></td>
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<td>Depressive symptoms</td>
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<tr>
<td>Cigarette smoking</td>
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<tr>
<td>“take a puff”</td>
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<td>.36</td>
</tr>
<tr>
<td>“take 1 cigarette”</td>
<td>1.07</td>
<td>.29</td>
</tr>
<tr>
<td>“take some for later”</td>
<td>1.04</td>
<td>.22</td>
</tr>
<tr>
<td><strong>Prototype kids your age who smoke…</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>“Popular”</td>
<td>1.89</td>
<td>.84</td>
</tr>
<tr>
<td>“Smart”</td>
<td>1.86</td>
<td>.76</td>
</tr>
<tr>
<td>“Cool”</td>
<td>1.57</td>
<td>.79</td>
</tr>
<tr>
<td>“Attractive”</td>
<td>1.61</td>
<td>.78</td>
</tr>
<tr>
<td>“Boring”</td>
<td>2.23</td>
<td>.88</td>
</tr>
</tbody>
</table>

*Note. Data from the willingness and prototype variables were collected during the sophomore year of high school.*
### Bivariate Correlations of Variables in Aim 1

<table>
<thead>
<tr>
<th>Variable</th>
<th>Sex</th>
<th>Race</th>
<th>Income</th>
<th>Depressive symptoms</th>
<th>Cigarette smoking</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sex</td>
<td>-.00</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Race</td>
<td></td>
<td>-.01</td>
<td>.38*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Income</td>
<td></td>
<td></td>
<td>-.11*</td>
<td>-.14*</td>
<td></td>
</tr>
<tr>
<td>Depressive</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>symptoms</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cigarette smoking</td>
<td>.03</td>
<td>-.14*</td>
<td>-.23*</td>
<td>.06</td>
<td></td>
</tr>
<tr>
<td>Self-report smoking</td>
<td>.02</td>
<td>.01</td>
<td>-.14*</td>
<td>.19*</td>
<td>.20*</td>
</tr>
</tbody>
</table>

*Note.* Data collected when participants were in the sophomore year of high school. Variables coded as: Sex (1=females and 0=males); Race (1=Caucasian/White and 0=Other); Income (midpoints); Depressive symptoms (total); Cigarette smoking (biomarker; % of methylation); Self-report of smoking (1=yes and 0=no). *p < .05.
Table A4.

**Hierarchical Regression of Control Variables and Cigarette Smoking Predicting Depressive Symptoms (Aim 1)**

| Model 1 |  |  |  |  |  |  |
|---------|---|---|---|---|---|
| Sex | .083* | .035 | .112* |  |  |  |
| Race | -.051 | .041 | -.064 |  |  |  |
| Income | -.001* | .000 | -.115* | .036* |  |  |

| Model 2 |  |  |  |  |  |  |
|---------|---|---|---|---|---|
| Sex | .082* | .035 | .112* |  |  |  |
| Race | -.050 | .041 | -.063 |  |  |  |
| Income | -.001* | .000 | -.110* |  |  |  |
| Cigarette Smoking | .002 | .005 | .023 | .036* |  | .001 |

| Model 3 |  |  |  |  |  |  |
|---------|---|---|---|---|---|
| Sex | .081* | .034 | .110* |  |  |  |
| Race | -.062 | .040 | -.077 |  |  |  |
| Income | -.001 | .000 | -.087 |  |  |  |
| Cigarette Smoking | -.001 | .005 | -.009 |  |  |  |
| Self-Report Smoking | .294* | .077 | .182* | .068* |  | .031* |

| Model 4 |  |  |  |  |  |  |
|---------|---|---|---|---|---|
| Sex | .311 | .916 | .421 |  |  |  |
| Race | 1.102 | .978 | 1.377 |  |  |  |
| Income | .009 | .010 | 1.177 |  |  |  |
| Cigarette Smoking | -.019 | .013 | -.174 |  |  |  |
| Self-Report Smoking | .302 | .171 | .187 |  |  |  |
| Sex * cigarette smoking | .003 | .011 | .305 |  |  |  |
| Race * cigarette smoking | .014 | .012 | 1.465 |  |  |  |
| Income * cigarette smoking | .000 | .000 | 1.292 |  |  |  |
| Sex * self-report | -.123 | .168 | -.059 |  |  |  |
| Race * self-report | -.051 | .202 | -.027 |  |  |  |
| Income * self-report | .002 | .002 | .088 | .079* |  | .012 |

*Note. Analysis for Aim 1 (n=438). Variables coded as: Sex (1=females and 0=males); Race (1=Caucasian/White; and 0=Other); Income (midpoints); Cigarette smoking (biomarker; % of methylation; Self-report of smoking (1=yes and 0=no); Depressive symptoms (total).

*p < .05.
Table A5.

*Bivariate Correlations of Variables in Aim 2*

<table>
<thead>
<tr>
<th>Variable</th>
<th>Sex</th>
<th>Race</th>
<th>Income</th>
<th>Cigarette Smoking</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sex</td>
<td>.04</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Race</td>
<td></td>
<td>.02</td>
<td>.29</td>
<td></td>
</tr>
<tr>
<td>Income</td>
<td>-.02</td>
<td></td>
<td></td>
<td>-.12</td>
</tr>
<tr>
<td>Cigarette smoking</td>
<td>.01</td>
<td>-.05</td>
<td>-.12</td>
<td></td>
</tr>
<tr>
<td>Depressive symptoms</td>
<td>.12*</td>
<td>-.04</td>
<td>-.18*</td>
<td>.05</td>
</tr>
</tbody>
</table>

*Note.* Data from sex, race, income, and cigarette smoking were collected when participants were in the sophomore year of high school. Data from depressive symptoms collected during the junior year of high school. Variables coded as: Sex (1=females and 0=males); Race (1=Caucasian/White and 0=Other); Income (midpoints); Cigarette smoking (biomarker; % of methylation); Depressive symptoms (total). *p < .05.
### Hierarchical Regression of Control Variables and Cigarette Smoking Predicting Depressive Symptoms a Year Later (Aim 2)

<table>
<thead>
<tr>
<th>Model 1</th>
<th>b</th>
<th>SE b</th>
<th>B</th>
<th>R²</th>
<th>ΔR²</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sex</td>
<td>.082</td>
<td>.043</td>
<td>.115</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Race</td>
<td>.003</td>
<td>.048</td>
<td>.004</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Income</td>
<td>-.001*</td>
<td>.000</td>
<td>-.183*</td>
<td>.047*</td>
<td></td>
</tr>
<tr>
<td>Cigarette Smoking</td>
<td>.002</td>
<td>.006</td>
<td>.022</td>
<td>.048*</td>
<td>.000</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Model 2</th>
<th>b</th>
<th>SE b</th>
<th>B</th>
<th>R²</th>
<th>ΔR²</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sex</td>
<td>.082</td>
<td>.043</td>
<td>.115</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Race</td>
<td>.004</td>
<td>.048</td>
<td>.005</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Income</td>
<td>-.001*</td>
<td>.000</td>
<td>-.180*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cigarette Smoking</td>
<td>.002</td>
<td>.006</td>
<td>.022</td>
<td>.048*</td>
<td>.000</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Model 3</th>
<th>b</th>
<th>SE b</th>
<th>B</th>
<th>R²</th>
<th>ΔR²</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sex</td>
<td>-1.932</td>
<td>1.153</td>
<td>-2.716</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Race</td>
<td>1.142</td>
<td>1.258</td>
<td>1.494</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Income</td>
<td>-.001</td>
<td>.000</td>
<td>-1.712</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cigarette Smoking</td>
<td>.016</td>
<td>.015</td>
<td>.159</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sex * cigarette smoking</td>
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<td>.014</td>
<td>-2.834</td>
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<td></td>
</tr>
<tr>
<td>Race * cigarette smoking</td>
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<td>.015</td>
<td>1.491</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Income * cigarette smoking</td>
<td>-.001</td>
<td>.000</td>
<td>-1.545</td>
<td>.061*</td>
<td>.013</td>
</tr>
</tbody>
</table>

*Note. Analysis for Aim 2 (n=264). Model 1 includes demographic variables as control variables. Variables coded as: Sex (1=females and 0=males); Race (1=Caucasian/White); Income (midpoints); Cigarette smoking (biomarker; % of methylation); Depressive symptoms (total). *p < .05.*
Table A7.

*Bivariate Correlations of Variables in Aim 3*

<table>
<thead>
<tr>
<th>Variable</th>
<th>Sex</th>
<th>Race</th>
<th>Income</th>
<th>Depressive symptoms</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sex</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Race</td>
<td>.05</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Income</td>
<td>-.02</td>
<td>.29*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Depressive symptoms</td>
<td>.11*</td>
<td>-09</td>
<td>-.21*</td>
<td></td>
</tr>
<tr>
<td>Cigarette smoking</td>
<td>-.01</td>
<td>-.12*</td>
<td>-.20*</td>
<td>.02</td>
</tr>
</tbody>
</table>

*Note.* Analysis for Aim 3 (n=264). Demographic and depressive symptoms data were collected when participants were in the sophomore year of high school. Data from cigarette smoking were collected during the junior year of high school. Variables coded as: Sex (1=females and 0=males); Race (1=Caucasian/White and 0=Other); Income (midpoints); Cigarette smoking (biomarker; % of methylation); Depressive symptoms (total).

*p < .05.*
Table A8.

Hierarchical Regression of Demographic Variables and Depressive Symptoms Predicting Cigarette Smoking a Year Later (Aim 3)

<table>
<thead>
<tr>
<th>Model 1</th>
<th>b</th>
<th>SE b</th>
<th>B</th>
<th>R²</th>
<th>∆R²</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sex</td>
<td>.004</td>
<td>.034</td>
<td>.007</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Race</td>
<td>.039</td>
<td>.038</td>
<td>.065</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Income</td>
<td>-.001*</td>
<td>.000</td>
<td>-.183</td>
<td>.045*</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Model 2</th>
<th>b</th>
<th>SE b</th>
<th>B</th>
<th>R²</th>
<th>∆R²</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sex</td>
<td>.006</td>
<td>.034</td>
<td>.010</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Race</td>
<td>-.040</td>
<td>.038</td>
<td>-.066</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Income</td>
<td>-.001*</td>
<td>.000</td>
<td>-.189*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Depressive symptoms</td>
<td>-.002</td>
<td>.004</td>
<td>-.030</td>
<td>.045*</td>
<td>.001</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Model 3</th>
<th>b</th>
<th>SE b</th>
<th>B</th>
<th>R²</th>
<th>∆R²</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sex</td>
<td>-.011</td>
<td>.050</td>
<td>-.019</td>
<td></td>
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</tr>
<tr>
<td>Race</td>
<td>-.091</td>
<td>.061</td>
<td>-.150</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Income</td>
<td>-.001</td>
<td>.000</td>
<td>-.122</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Depressive sx. * sex</td>
<td>.005</td>
<td>.009</td>
<td>.061</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Depressive sx. * race</td>
<td>.010</td>
<td>.010</td>
<td>.138</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Depressive sx. * income</td>
<td>-.001</td>
<td>.000</td>
<td>-.100</td>
<td>.051</td>
<td>.006</td>
</tr>
</tbody>
</table>

Note. Data from cigarette smoking were collected in the junior year of high school. All other variables collected during the sophomore year of high school. Model 1 includes demographic variables as control variables. Variables coded as: Sex (1=females and 0=males); Race (1=Caucasian/White); Income (midpoints); Cigarette smoking (biomarker; % of methylation); Depressive symptoms (Depressive sx.; total).

*p < .05.
Table A9.

Correlations of Depressive Symptoms and Social Cognitions (Aim 4)

<table>
<thead>
<tr>
<th>Variable</th>
<th>Depressive Symptoms</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Spearman correlations</td>
<td>Pearson correlations</td>
<td></td>
</tr>
<tr>
<td></td>
<td>($r_s$)</td>
<td>($r$)</td>
<td></td>
</tr>
<tr>
<td>Prototypes kids your age who smoke…</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>“Popular”</td>
<td>.12*</td>
<td>.13*</td>
<td></td>
</tr>
<tr>
<td>“Smart”</td>
<td>.16*</td>
<td>.19*</td>
<td></td>
</tr>
<tr>
<td>“Cool”</td>
<td>.19*</td>
<td>.15*</td>
<td></td>
</tr>
<tr>
<td>“Attractive”</td>
<td>.23*</td>
<td>.20*</td>
<td></td>
</tr>
<tr>
<td>“Boring”</td>
<td>-.11*</td>
<td>-.08</td>
<td></td>
</tr>
<tr>
<td>Willingness to:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>“take a puff”</td>
<td>.21*</td>
<td>.20*</td>
<td></td>
</tr>
<tr>
<td>“take 1 cigarette”</td>
<td>.22*</td>
<td>.23*</td>
<td></td>
</tr>
<tr>
<td>“take some for later”</td>
<td>.16*</td>
<td>.18*</td>
<td></td>
</tr>
</tbody>
</table>

Note. Analysis for Aim 1 (n=438). Data collected during the sophomore year of high school. Social cognitions refer to willingness to engage in smoking behaviors and prototypes or images of smokers. Depressive symptoms (total score). *$p < .05.$
Table A10.

*Bivariate Correlations of Demographic Variables, Willingness, Prototype, and Cigarette Smoking*

<table>
<thead>
<tr>
<th>Variable</th>
<th>Sex</th>
<th>Race</th>
<th>Income</th>
<th>Willingness</th>
<th>Prototype</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sex</td>
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<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Race</td>
<td></td>
<td>-.02</td>
<td>.38*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Income</td>
<td>-.01</td>
<td>.01</td>
<td>-.15*</td>
<td>.23*</td>
<td></td>
</tr>
<tr>
<td>Willingness</td>
<td></td>
<td></td>
<td></td>
<td>-.01</td>
<td></td>
</tr>
<tr>
<td>Prototype</td>
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<td>-.05</td>
<td>-.04</td>
<td>.24*</td>
<td>-.09*</td>
</tr>
<tr>
<td>Cigarette Smoking</td>
<td>.03</td>
<td>-.15*</td>
<td>-.23*</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Note.* Data collected when participants were in the sophomore year of high school. Variables coded as: Sex (1=females and 0=males); Race (1=Caucasian/White and 0=Other); Income (midpoints); Willingness (average of three; 1=not at all willing, 2=kind of willing, and 3=very willing); Cigarette smoking (biomarker; % of methylation). *p < .05.
Table A11.

*Hierarchical Regression of the Willingness and Prototype Composites Predicting Cigarette Smoking*

<table>
<thead>
<tr>
<th>Model</th>
<th>b</th>
<th>SE b</th>
<th>B</th>
<th>R²</th>
<th>ΔR²</th>
</tr>
</thead>
<tbody>
<tr>
<td>Model 1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sex</td>
<td>.007</td>
<td>.011</td>
<td>-.028</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Race</td>
<td>-.017</td>
<td>.013</td>
<td>-.066</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Income</td>
<td>-.001*</td>
<td>.000</td>
<td>-.209*</td>
<td>.60*</td>
<td></td>
</tr>
<tr>
<td>Model 2</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sex</td>
<td>.007</td>
<td>.011</td>
<td>-.030</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Race</td>
<td>-.020</td>
<td>.013</td>
<td>-.079</td>
<td></td>
<td></td>
</tr>
<tr>
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<td>.000</td>
<td>-.179*</td>
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<td>.165*</td>
<td>.086*</td>
<td>.026*</td>
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<td></td>
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<tr>
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<td>-.020</td>
<td>.013</td>
<td>-.077</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Income</td>
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<td>.000</td>
<td>-.180*</td>
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<tr>
<td>Willingness</td>
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<td>.155*</td>
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<td></td>
</tr>
<tr>
<td>Prototypes</td>
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<td>.013</td>
<td>.041</td>
<td>.088*</td>
<td>.002</td>
</tr>
</tbody>
</table>

*Note. Data collected when participants were in the sophomore year of high school. Variables coded as: Sex (1=females and 0=males); Race (1=Caucasian/White and 0=Other); Income (midpoints); Willingness (average of three items; 1=not at all willing, 2=kind of willing, and 3=very willing); Prototypes (average of five items; 1) not at all, 2) a little bit, 3) kind of, and 4) very).

*p < .05.*
REFERENCES


Maralani, V. (2014). Understanding the links between education and smoking. Social Science Research, 48, 20–34. https://doi.org/10.1016/j.ssresearch.2014.05.007


