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Sarah E. Malone

University of Nebraska-Lincoln, malone.sarah89@gmail.com

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ADOLESCENT DEPRESSIVE SYMPTOMS AND SUBSTANCE USE:
THE MEDIATING INFLUENCE OF HEALTH SERVICE UTILIZATION

by

Sarah E. Malone

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ADOLESCENT DEPRESSIVE SYMPTOMS AND SUBSTANCE USE:
THE MEDIATING INFLUENCE OF HEALTH SERVICE UTILIZATION

Sarah E. Malone, M.A.

University of Nebraska, 2013

Advisor: Kimberly A. Tyler

A large number of American adolescents suffer from depression and the consequences have been shown to be detrimental to their well-being. Adolescent substance use is also an increasing social problem due to the high usage rates and negative lifelong consequences for users. This paper explores the relationships between victimization, substance use, psychological health service utilization, and depressive symptoms in a sample of 4,757 adolescents. Using two waves of data from the National Longitudinal Study of Adolescent Health (Add Health), the results revealed a positive relationship between victimization and adolescent depressive symptoms, even after controlling for several demographic variables and previous depressive symptoms. However, victimization was only moderately associated with depressive symptoms, indirectly through cigarette or marijuana use. Moreover, psychological health service utilization partially mediated the significant association between adolescent substance use (cigarette or marijuana) and adolescent depressive symptoms. In conclusion, adolescents who experience higher levels of victimization may be more likely to use cigarettes or marijuana, which is positively associated with utilizing psychological health services, thereby elevating the risk of adolescent depressive symptoms. Intervention to reduce adolescent substance use may reduce vulnerability to adolescent depressive symptoms.

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TABLE OF CONTENTS

Chapter 1: Introduction.....	1
Chapter 2: Literature Review.....	3
Chapter 3: Theoretical Framework.....	17
Chapter 3: Methods.....	20
Chapter 4: Results.....	28
Chapter 5: Discussion and Conclusion.....	35
References.....	43

LIST OF MULTIMEDIA OBJECTS

Figure 1 Conceptual Prospective Model: Wave I substance use as mediator.....	26
Figure 2 Conceptual Prospective Model: Wave I psychological service utilization as mediator.....	27
Figure 3 Conceptual Prospective Model: three-path mediational model.....	28
Table 1 Descriptive Statistics.....	28
Table 2 Correlation Matrix of Study Variables.....	29
Table 3 OLS Regression Analyses of WI Cigarette Use on WII Depressive Symptoms.....	30
Table 4 OLS Regression Analyses of WI Binge Drinking on WII Depressive Symptoms.....	32
Table 5 OLS Regression Analyses of WI Marijuana Use on WII Depressive Symptoms.....	34

INTRODUCTION

According to the National Survey on Drug Use and Health, 1.9 million, or 8% of adolescents aged 12 to 17 years experienced a major depressive episode (MDE) during 2010 (Substance Abuse and Mental Health Services Administration [SAMHSA] 2012). Among the major psychiatric disorders, depression is one of the most common to occur and frequently begins during adolescence (Hankin 2006). Depression has been shown to greatly impair adolescents in numerous ways, such as by increasing suicide attempts (Tandon and Solomon 2009) and increasing the likelihood of substance use (Goldstein et al. 2009). Adolescent depression may continue to negatively affect their mental and physical health well into adulthood (Adam 2009; Anderson et al. 2006; Gotlib and Hammen 2009). Clearly, a significant number of American adolescents suffer from depression and the consequences can be detrimental to their well-being.

Adolescent substance use is also a growing social problem due to the high usage rates and negative lifelong consequences for users (Johnston et al. 2011; SAMHSA 2012; Van Hasselt et al. 2005). Rates of cigarette use, binge drinking, and marijuana use are relatively high for today's youth (Johnston et al. 2011). For example, at least one out of every two high school seniors reports binge drinking at least once in their lifetime (Johnston et al. 2011). Likewise, marijuana was found to be the most widely used illegal drug among adolescents in 2008 (Farhat, Simons-Morton and Luk 2011). Overall, these high rates of substance use are a cause for concern given their known negative outcomes including lower academic achievement and goals, being less likely to receive a high school diploma or go to college, and being less likely to be employed than adolescents who do not use substances (Bogart et al. 2007). It has also been suggested that substance

use in adolescence increases the risk for alcohol and other drug dependence in adulthood (Brook et al. 2002; Grant et al. 2006; McCambridge, McAlaney and Rowe 2011).

Not only do numerous adolescents suffer from high rates of depression and substance use, but these two have been found to co-occur (Townsend et al. 2006). For instance, depression and alcohol use (Poulin et al. 2005) and depression and smoking have been found to co-occur and are main causes of mental and physical illness (Audrain-McGovern, Rodriguez and Kassel 2009). Additionally, those with dual disorders experience higher rates of relapse than those with only one disorder and they also have unique challenges when it comes to treatment (Ramo, Myers and Brown 2010; Townsend et al. 2006:475). In 2010, adolescents aged 12 to 17 with MDE had higher rates of daily cigarette use, heavy alcohol use, and co-occurring disorders than those without MDE (SAMHSA 2012). In an effort to determine what causes adolescent depression, some researchers have examined both distal (e.g., neighborhood disadvantage, victimization) and proximal (e.g., substance use) risk factors (Boardman et al. 2001; Howard et al. 2002; Latkin and Curry 2003; Latkin et al. 2007). More often than not, research finds that adolescent substance use is a significant factor in predicting adolescent depressive symptoms (Brook et al. 2002; Degenhardt et al. 2003; Poulin et al. 2005; Swendsen and Merikangas 2000; Vida et al. 2009; Wade and Pevalin 2005).

Because both depression and substance use are common among adolescents and the two frequently co-occur, utilization of health services becomes essential in order to treat these conditions. In 2010, just over one third of adolescents with MDE received professional treatment for their depression (SAMSHA 2012). Likewise, between 2003 and 2006, of those who could have benefited from services, only about 7% of adolescents

received alcohol treatment and approximately 10% received drug treatment (Sterling et al. 2010). Many adolescents, however, often go untreated for these conditions and thus suffer from both short-term and long-term consequences, such as increasing severity of the conditions and more difficulty being treated in the future (Mertens et al. 2007; Parthasarathy and Weisner 2006; Sterling et al. 2004; Wu and Ringwalt 2006).

Although researchers have investigated the relationship between mental health and substance use (Audrain-McGovern et al. 2012; Chinet et al. 2006; Clark, Ringwalt and Shamblen 2011; Curry et al. 2012; Goldstein et al. 2009; Poulin et al. 2005; Silberg et al. 2003) and the relationship between health service utilization and substance use and/or mental health (Curry et al. 2012; Drake et al. 1996; Lu and McGuire 2002; Townsend et al. 2006), there is a paucity of research that has examined psychological health service utilization as a mediator between substance use and depressive symptoms among adolescents. Given this gap in the literature, the current study will use the National Longitudinal Study of Adolescent Health (Add Health) to address this issue in order to develop a more complete picture of the effectiveness of utilizing psychological health services in treating adolescents with depressive symptoms and substance use issues.

LITERATURE REVIEW

Adolescent Depressive Symptoms

It is estimated that one out of every eight teenagers has clinical depression (Mental Health America 2010, as cited in Taylor 2011) and this varies by gender. That is, 4.4% of male adolescents and 11.8% of female adolescents aged 12 to 17 experienced

MDE during 2010 (SAMHSA 2012). MDE appears to increase with age: in 2010, 3.3% of 12 year olds, 7.9% of 14 year olds, and 10.3% of 17 year olds had MDE (SAMHSA 2012). These high rates of depression are disconcerting given that adolescent depression is one of the most influential factors in predicting suicide attempts and ideation (Tandon and Solomon 2009). Also concerning is the positive association that has been found between adolescent depression and adulthood depression (Fergusson and Woodward 2002; Fombonne et al. 2001; Gotlib and Hammen 2009). Furthermore, many other negative adult health outcomes are significantly related to adolescent depression (Anderson et al. 2006; Fergusson and Woodward 2002; Gotlib and Hammen 2009). Numerous studies have found that delinquents experience significantly higher rates of depressed feelings than non-delinquents (Hagan and Foster 2003; Moffitt et al. 2001). Such studies have found this relationship persists throughout adolescence and into adulthood (Overbeek et al. 2001). Unfortunately, adolescents suffering from depression are found to be less responsive to antidepressant medications than adults with depression, making treatment of adolescent depression more challenging (Andersen and Teicher 2008).

Levels of adolescent depression and depressive symptoms have been found to vary by both gender and race. Research has found robust differences in levels of depressive symptoms by gender, with female adolescents more affected than male adolescents (Galambos, Leadbeater and Barker 2004). Females are twice as likely to suffer from depression as males (Poulin et al. 2005; Taylor 2011). Regarding racial differences, studies have found conflicting results in rates of adolescent depressive symptoms (Tandon and Solomon 2009). For instance, one study found that African

American students in 7th to 12th grade had higher levels of depressive symptoms than that of White, Hispanic, and Asian students (Wight et al. 2005). In contrast, others have found that African American students experienced depressive symptoms less often than White, Hispanic, and Asian middle school and high school students (Schraedley, Gotlib and Hayward 1999). Not only do many adolescents experience depression, but many of them also report substance use and the two have been found to be correlated (Chinet et al. 2006; Van Hasselt et al. 2005).

Adolescent Substance Use

Adolescents have relatively high prevalence rates of substance use (Johnston et al. 2011). Substance use among adolescents involves the use of any type of drug, ranging from cigarettes and alcohol to illegal drugs, such as marijuana and cocaine. In terms of alcohol, 16% of 8th grade students, 37% of 10th graders, and 54% of 12th graders reported binge drinking, having at least 5 or more drinks in one setting, one or more times in their lifetime in 2010 (Johnston et al. 2011). Marijuana was the most used illicit drug in 2010 with 17% of 8th graders, 33% of 10th graders, and 44% of 12th graders reporting usage at least once (Johnston et al. 2011). Adolescents also report high rates of cigarette usage: 20% of 8th graders and 42% of 12th graders have smoked a cigarette at least once in their lifetime (Johnston et al. 2011). In terms of past month drug use among 12 to 17 year olds in 2010, 23% reported binge drinking, 10.7% reported smoking cigarettes, and 7.4% reported smoking marijuana (SAMHSA 2011).

Substance use appears to be increasing in terms of frequency and associated risks, such as smoking marijuana daily and binge drinking (Chinet et al. 2006). In general,

adolescent substance use has long-term negative social, behavioral and economic consequences (Bogart et al. 2007). The earlier adolescents begin using drugs, the greater their risk for future substance use problems (Chinet et al. 2006). For example, Green and Ritter (2000) demonstrated that adults who used marijuana during adolescence were more likely to use marijuana during adulthood. Adolescent alcohol and substance use disorders are also linked with many negative consequences, such as risky sexual behavior, trouble with the law, and suicide attempts (Curry et al. 2012). Research has also found that most persistent substance-using adolescents are also persistent delinquents (Loeber, Stouthamer-Loeber and White 1999). Likewise, students who plan on attending college report lower rates of illicit drug use in high school than students who do not plan on attending college (Johnston et al. 2011).

Like rates of depression, levels of substance use have been found to vary by both gender and race. Regarding gender, males generally report using all types of substances more frequently compared to their female counterparts (Johnston et al. 2011). Similar to findings on racial differences in adolescent depressive symptoms, some discrepancies in the research exist when examining substance use. Using the Add Health, Watt and Rogers (2007) reported that White adolescents used alcohol more frequently than their African American counterparts, but rates of heavy drinking and other drug use were similar for both groups. In contrast, other studies have found that White adolescents were more likely to have higher rates of binge drinking, cigarette use, and other drug use than African American adolescents (Johnson 2004). Additionally, though Brown, Miller and Clayton concluded that marijuana and alcohol use levels were similar for African American and White adolescents, they found that cigarette use was much higher among

Whites (2004). Finally, Johnston et al. (2011) reported that African Americans had lower drug use rates in 2010 compared to White and Hispanic students. Hence, the existing research demonstrates that adolescent substance use is prevalent and varies by both gender and race. Not only do adolescents suffer from high rates of depression and substance use, but these two issues are frequently found to co-occur (Townsend et al. 2006). The following section examines the co-occurrence of adolescent depressive symptoms and substance use.

Co-Occurrence

The association between adolescent depression and substance use has been well established (Chinet et al. 2006; Clark, Ringwalt and Shamblen 2011; Curry et al. 2012; Goldstein et al. 2009; Poulin et al. 2005; Silberg et al. 2003; Wu et al. 2008). Depressed adolescents have been found to use substances at almost twice the rate as non-depressed adolescents (Goldstein et al. 2009). Chinet and colleagues (2006) found that a decrease in substance use coincided with a decrease in depressive symptoms whereas stable rates of substance use were associated with stable levels in depressive symptoms. Looking at specific substances, research has found an association between marijuana use and adolescent depression (Poulin et al. 2005) and a strong relationship between adolescent depression and smoking cigarettes (Audrain-McGovern et al. 2009; Audrain-McGovern et al. 2012). For example, higher levels of depressive symptoms during mid- to late adolescence (ages 14-18) predicted higher odds of smoking cigarettes by 23% (Audrain-McGovern et al. 2012).

The strong association between depression and substance use discussed above illustrates the phenomenon of co-occurring disorders. Unfortunately, it is more common for drug using adolescents to experience co-occurring disorders than to only have one issue (Chan et al. 2009). In fact, the most frequent type of mental health issue to co-occur with substance use is depression (Chan, Dennis and Funk 2008; Clark et al. 1997). Co-occurrence of depression and substance-related problems is high, with rates of depression ranging from 20-30% in adolescents who use substances (Chinet et al. 2006). A recent study concluded that adolescents with co-occurring disorders of depression and drug abuse are likely to experience negative outcomes in early adulthood, such as early parenthood and non-completion of high school (Vida et al. 2009). The same study concluded that mental health issues in early adulthood often continue to worsen for those who had substance abuse problems and co-occurring disorders during their adolescent years. Additionally, adolescents who suffer from co-occurring delinquency, such as substance abuse, and depressive symptoms suffer from negative outcomes far more severe than those experienced by adolescents with only one or neither of the two conditions (Kofler et al. 2011).

Research demonstrates a consistent gender difference in the relationship between depression and substance use among adolescents (Poulin et al. 2005). That is, not only do girls tend to have higher levels of depression than boys (Chinet et al. 2006; Galambos, Leadbeater and Barker 2004; Luk et al. 2010; Poulin et al. 2005), but the link between depression and substance use tends to be stronger for female adolescents than male adolescents (Luk et al. 2010). Additionally, females are at a higher risk of co-occurring disorders of depressive symptoms and substance use than males (Needham 2007, as cited

in Poulin et al. 2005; Sihvola et al. 2008). Males, however, tend to have higher rates of substance use disorders than females (Poulin et al. 2005). Other studies on depression and substance use by gender have found varying results, depending on the type of substance used. For example, one study found that depression leads to smoking, alcohol use, and other drug use for female adolescents but not male adolescents (Silberg et al. 2003). Similarly, both cigarette smoking and alcohol use have been found to be predictors of higher depressive symptoms in adolescent females, but not in adolescent males (Poulin et al. 2005). However, marijuana use is a predictor of higher depressive symptoms for both male and female adolescents. In contrast, another study found that marijuana use predicted depression among females but not males, even after controlling for early depressive symptoms and alcohol use (Patton et al. 2002). Though the results were consistent in both cross-sectional and longitudinal studies, the findings are mixed in terms of whether gender differences exist in the association between depression and marijuana, and also between depression and smoking cigarettes (Acierno et al. 2000; Galambos et al. 2004; Poulin et al. 2005).

Many depressed adolescents who use substances may continue their usage in an attempt to cope with their depressive symptoms (Clark et al. 2011). For adolescents suffering from co-occurrence, though resorting to substance use may temporarily relieve depressive symptoms this decision is generally a harmful choice. For instance, research has found that the use of cigarettes and hard drugs during adolescence leads to lower adult life satisfaction and, thus, lower mental health (Bogart et al. 2007). Similarly, those who use marijuana as a coping strategy are more depressed than those who do not use marijuana in an effort to cope with problems (Green and Ritter 2000). Research suggests

that coping strategies are usually unsuccessful and even harmful when dealing with life stress (Bogart et al. 2007; Mattlin, Wethington and Kessler 1990) and adolescent substance use does not improve well-being over time. Additionally, many studies hypothesize a causal order between adolescent depression and substance use (Degenhardt et al. 2003; Poulin et al. 2005; Swendsen and Merikangas 2000; Vida et al. 2009; Wade and Pevalin 2005).

Causal Order

Because of the harmful consequences of adolescent substance use, researchers often question the causal order of the relationship: does substance use cause depressive symptoms or do depressive symptoms cause substance use? While an extensive amount of research examines depressive symptoms as an outcome of substance use, some researchers argue that depression causes adolescent substance use (Dembo et al. 1992; Stogner and Gibson 2011). For example, Clark and colleagues (2011) investigated whether adolescents use substances as a way to self-medicate in order to cope when experiencing depressed mood. Adolescent depressed mood and levels of substance use were measured in sixth, seventh, and eighth grade in order to see if substance use increased over time (Clark et al. 2011). The study concluded that students who had high levels of depressed mood in sixth grade had more dramatic increases in substance use over time than students who started with low levels of depressed mood. In this study, it appears that adolescent depression occurred prior to substance use.

On the other hand, many other studies have found that substance use and other delinquent behavior occur before any depressive symptoms are present (Biederman et al.

1995; Degenhardt et al. 2003; Poulin et al. 2005; Ritakallio et al. 2005; Swendsen and Merikangas 2000; Vida et al. 2009; Wade and Pevalin 2005; Wiesner and Kim 2006; Wolff and Ollendick 2006) and find evidence against self-medicating and coping hypotheses (Degenhardt, Hall and Lynskey 2003). For instance, in their longitudinal study Brook et al. (2002) found that early alcohol use, marijuana use, and overall drug use predicted later major depressive disorder. Similarly, Vida et al. found that adolescents who abuse drugs have much higher rates of depression in early adulthood (by age 25) than adolescents without drug or mental health problems (2009). These findings suggest that early substance use and/or abuse may cause depression because adolescents without drug problems had lower rates of depression in early adulthood than adolescents with substance use/abuse issues (Brook et al. 2002; Vida et al. 2009). As such, existing studies yield mixed results regarding the causal order between depressive symptoms and substance use among adolescents.

Thus, when conducting research, certain methodological steps must be taken in order to correctly establish causal order between depression and substance use. There are three criteria for establishing causality. The first criterion is to have a significant correlation between the two focal variables. As existing literature reveals, the association between adolescent depression and substance use is significant (Chinet et al. 2006; Clark, Ringwalt and Shamblen 2011; Curry et al. 2012; Deykin, Levy and Wells 1987; Goldstein et al. 2009; Poulin et al. 2005; Silberg et al. 2003). But because an association alone does not indicate causality, the second criterion, temporal order, must be clear to establish the causal order of the focal variables. In cases when the initial cause of depression is identified (as a factor other than substance use), depression appears to occur

before substance use. For instance, a recent study found that, among female subjects, depression was positively related to and occurred before substance use (Luk, Wang and Simons-Morton 2010). Luk et al. suggested that female victimization can cause higher depressive symptoms which lead to higher levels of substance use. Thus, the causal order that the study proposed suggests that substance use is an outcome of depression.

In contrast, an extensive body of research establishes the temporal order that adolescent substance use occurs before depressive symptoms. For example, adolescent cigarette smoking has been found to precede depression and, thus, increase the likelihood of depression (Wade and Pevalin 2005) and drug dependence (Brown et al. 1996). Similarly, alcoholism has been found to significantly predict depression (Swendsen and Merikangas 2000). Marijuana use at an early age is related to higher levels of depression later in life (Green and Ritter 2000). Indeed, a review of a number of studies found that early-onset, regular use, and heavy use of marijuana independently increased the risk of later depression but that depression did not increase marijuana use (Degenhardt et al. 2003). Furthermore, Poulin and colleagues (2005) investigated gender differences and found that alcohol use, cigarette smoking, and cannabis use all independently predicted higher levels of female adolescent depressive symptoms whereas cannabis use was found to be the only predictor of male depressive symptoms. Thus, the causal order of the aforementioned studies suggests that depression is an outcome of substance use and not vice versa. Clearly, existing studies have yielded inconsistent findings regarding the causal order between adolescent substance use and adolescent depressive symptoms. However, because an extensive body of existing research demonstrates the predictive effect of adolescent substance use on depression, the causal order of the focal relationship

for the current project will follow this model. That is, adolescent substance use precedes adolescent depressive symptoms.

After confirming the first two criteria of causality, the last but most crucial step is to account for other variables that may be responsible for the focal relationship. This third criterion in establishing causal order is accomplished by accounting for possible spurious variables. One way of achieving this is to control for the effects of the independent variable at the time that the dependent variable is measured. In the case where adolescent substance use occurs before depressive symptoms, it would be necessary to control for adolescent depressive symptoms at the time when adolescent substance use is measured. Another way of accounting for spuriousness is to include mediating variables in the conceptual model. It is necessary to consider whether mediating effects are responsible for the relationship between substance use and depressive symptoms. For instance, Bogart and colleagues found support for the mediating effects of health. That is, smoking cigarettes as an adolescent leads to poor health which, in turn, leads to lower life satisfaction (2007). This finding demonstrates that substance use does not fully predict poor well-being but, rather, other variables mediate the focal relationship. While many studies have examined the effect of substance use on adolescent depression, there are other factors, such as health service utilization, that may mediate the focal relationship and is the topic to which I now turn.

Health Services

Because adolescent substance use is often a prominent factor in predicting depressive symptoms (Brook et al. 2002; Degenhardt et al. 2003; Poulin et al. 2005;

Swendsen and Merikangas 2000; Vida et al. 2009; Wade and Pevalin 2005), the role of health services becomes an important topic of discussion. In 2010, approximately 40% of females with MDE were receiving professional treatment for their depression compared to 32% of males with MDE (SAMSHA 2012). Almost half of adolescents (47.6%) who received specialty mental health services (inpatient or outpatient care) reported receiving the services because they felt depressed and 30.5% because they had problems with family or home (SAMHSA 2012). As mentioned earlier, only a small percentage of adolescents suffering from depression or drug problems actually receive the treatment they need (SAMSHA 2012; Sterling et al. 2010). Many adolescents often go untreated for these conditions and are faced with many negative consequences (Mertens et al. 2007; Parthasarathy and Weisner 2006; Sterling et al. 2004; Wu and Ringwalt 2006). Thus, the utilization of psychological health services is crucial in treating adolescents with depression and/or substance use issues. Because adolescents are found to be less responsive to antidepressant medications than adults with depression (Andersen and Teicher 2008), psychological health service utilization becomes even more necessary for younger populations.

Health services, such as drug prevention programs, have become more popular over the years but researchers continue to evaluate program effectiveness. Lu and McGuire (2002) focused on the effectiveness of an outpatient substance abuse program and found that the treatment decreased drug use frequency and increased abstinence, especially for those who use drugs more heavily. These types of programs, however, focus on treating substance use only. In fact, a significant percentage of adolescents with co-occurring conditions do not receive treatment for their mental health issues while

receiving treatment for their substance use disorder (Bukstein et al. 2005; Jaycox, Morral and Juvonen 2003; Sterling and Weisner 2005).

Adolescents with co-occurring disorders require special assistance. The most common method of treating those with dual disorders is to offer two separate treatments – one for mental health and one for substance abuse (Townsend et al. 2006). Some research findings provide support for separate treatments. For instance, Curry and colleagues (2012) found that adolescents who received and responded positively to short-term treatment for major depressive disorder (MDD) had lower rates of future substance use disorders (SUD). However, offering two separate treatments for those with co-occurring disorders has often been found ineffective (Townsend et al. 2006).

Historically, programs failed to treat those with dual disorders of depression and substance use, and the few that have been established are incomplete and ineffective in treating both disorders (Drake et al. 1996). Integrated care that includes treatment for both mental health and substance abuse in one program has proved most beneficial to those with co-occurring disorders; unfortunately, not all treatment programs offer integrated care (Drake et al. 2004; Ducharme, Knudsen and Roman 2006). Chinet and colleagues (2006) have suggested that adolescents who are being treated for depression should be automatically screened for substance use problems because, many times, adolescents with substance-related issues do not request help. The same suggestion can be made for adolescents being treated for substance use issues because research has illustrated the low percentages of adolescents with mental health issues who seek treatment (Bukstein et al. 2005; Jaycox et al. 2003; Sterling and Weisner 2005). These findings highlight the need for more productive and complete treatment programs for

those suffering from co-occurring disorders. Because research has found a positive association between adolescent substance use and service utilization (Casanueva et al. 2011; Yanos, Czaja and Widom 2010) and a negative association between health care service utilization and adolescent depression (Dickerson et al. 2012), it is likely that there is an indirect effect of substance use on depression through health service utilization.

Though health service utilization may play a significant role in the relationship between adolescent depressive symptoms and substance use, other factors such as experiencing victimization may also be important. That is, the neighborhood in which one is raised is likely to increase the risk of witnessing victimization or being a victim of violence. These experiences of victimization may have a significant influence on future adolescent depression well before health service utilization or substance use occurs. For example, growing up in a neighborhood with high rates of crime may increase one's risk of victimization which could impact an adolescent's substance use and depressive symptoms. The following section looks at research that has examined the effects of one's neighborhood on substance use and health outcomes.

Neighborhood and Victimization

When exploring factors that influence adolescent depressive symptoms and well-being, some studies consider the role of neighborhood disadvantage (Aneshensel and Sucoff 1996; Gutman, McLoyd and Tokoyawa 2005). In one longitudinal study Latkin and Curry (2003) examined the effects of neighborhood disadvantage on depressive symptoms among a community sample of 818 adults, most of whom were current or former substance users. Their results revealed a positive association between perceived

neighborhood disadvantage and levels of depressive symptoms. Typical characteristics of neighborhood disadvantage are experiencing personal victimization and/or witnessing violent victimization (Martin, Sigda and Kupersmidt 1998; Reed et al. 2009). For instance, Howard and colleagues (2002) found that witnessing violence or experiencing violent victimization were associated with negative adolescent psychosocial symptoms of distress, such as difficulties with concentration and feelings of being unloved and afraid. Because research has also found an association between neighborhood disadvantage and substance use (Boardman et al. 2001; Latkin et al. 2007) and between substance use and depressive symptoms (Brook et al. 2002; Degenhardt et al. 2003; Poulin et al. 2005; Swendsen and Merikangas 2000; Vida et al. 2009; Wade and Pevalin 2005), it is likely that there is an indirect effect. That is, neighborhood context (e.g., presence of drug dealers, high crime rates) place individuals at greater risk for victimization which leads to substance use (Boardman et al. 2001), thereby increasing one's depressive symptoms. Because I am unaware of any studies that have empirically tested this model to date, the current study will contribute to the existing literature on adolescent depressive symptoms by examining the mediating role of substance use between victimization and depressive symptoms.

THEORETICAL FRAMEWORK

Background

Adolescent substance use is an important risk factor when examining adolescent depressive symptoms. However, studies that have tested the association between adolescent depression and substance use generally do not identify a particular theory to

explain the relationship. One possible reason for this may be because a causal relationship could not be developed due to the fact that each variable is found to have predictive power over the other (Chinet et al. 2006). Another possible reason for omitting a theory may be due to page limitations within a journal article (Steuber and Danner 2006). Thus, some researchers do not utilize theoretical explanations for their findings and, instead, simply state that substance use and depression co-occur and, thus, are not predictive of one another. However, not attempting to develop a causal order leaves many questions unanswered especially given the fact that mounds of research have made statements about a causal order (Degenhardt et al. 2003; Poulin et al. 2005; Swendsen and Merikangas 2000; Vida et al. 2009; Wade and Pevalin 2005). Thus, when conducting empirical research, it is beneficial to offer theoretical explanations for the cause of each phenomenon while also controlling for co-occurrence, which is possible when using longitudinal data (Brook et al. 2002).

Stress Process

The stress process model (Pearlin et al. 1981; Pearlin 1989) provides a useful framework for analyzing the relationship between various stressors and health outcomes. More specifically, it approaches deviance and health from a life course perspective which is valuable when considering the longitudinal effects of adolescent substance use on depressive symptoms (Pearlin et al. 2005; Siennick 2007). According to the stress process, when an individual experiences social stress, such as neighborhood disadvantage, there is an increased risk of negative mental health outcomes (Wickrama and Baltimore 2010). The model has been used to explain the significant association

between distal risk factors (or primary stressors, which come first in people's experiences), such as victimization and proximal risk factors (or secondary stressors, which are a result of primary stressors; Pearlin, 1989), such as substance use (Boardman et al. 2001; Latkin et al. 2007) and the significant relationship between both distal and proximal risk factors and mental health outcomes (Latkin and Curry 2003).

The current study utilizes a stress process model to understand how primary stressors (i.e., victimization) and second stressors (i.e., substance use) are related to adolescent depressive symptoms. Researchers have observed instances of chronic stressful conditions and found that they do in fact increase the risk of substance use and depression (Clark et al. 2011; Robles et al. 2005). Similarly, research has found that adolescent marijuana use increases the likelihood of other illicit drug use and depression in later adolescence and young adulthood (Fergusson, Horwood and Swain-Campbell 2002). Marijuana use has psychological consequences that increase depression and start the long course of negative outcomes such as anti-conventional conduct (Kandel et al. 1986; Kandel, Yamaguchi and Chen 1992). Furthermore, the stress process model emphasizes the role of protective factors in mediating the direct effect of stressors on mental health outcomes (Pearlin 1989). Applied to the current study, psychological health service utilization will act as a protective factor by mediating the direct effect of substance use, the secondary stressor, on adolescent depressive symptoms. Thus, in the present research model, higher levels of victimization are expected to be associated with adolescent substance use which, in turn, will be positively related to psychological health service utilization, thereby causing lower levels of depressive symptoms.

HYPOTHESES

Based on the above literature review research findings and the stress process theory, several mediation models of adolescent depressive symptoms were proposed and tested. Each mediator was tested in separate single-mediator models and was then tested together in a three-path mediational model (Preacher and Hayes 2008). The research hypotheses are as follows:

Hypothesis #1 – Higher levels of victimization will be associated with higher levels of depressive symptoms.

Hypothesis #2 – Substance use (i.e., cigarette use, binge drinking, and marijuana use, independent from one another) will mediate the relationship between victimization and depressive symptoms. That is, higher levels of victimization will be positively associated with substance use which will lead to higher levels of depressive symptoms.

Hypothesis #3 – Psychological health service utilization will mediate the relationship between substance use and depressive symptoms. More specifically, substance use will be positively related to psychological health service utilization which will lead to *lower* levels of depressive symptoms.

METHODS

Sampling Procedure

Data from the National Longitudinal Study of Adolescent Health (Add Health) will be used to examine the effect of substance use on adolescent depressive symptoms. Add Health is a nationally representative, longitudinal study with four waves of data collection to date. The data were collected through cluster sampling of schools with unequal probability. Within the clusters, the sample was stratified by grade and sex and

within the stratified clusters of schools, 80 high schools and 52 middle schools from the U.S. were selected for the sample (Harris et al. 2009).

According to the Add Health website, the sample is nationally representative in regards to “region of country, urbanicity, school size, school type, and ethnicity” due to stratification and systematic sampling in the design of the study (Harris et al. 2009). From each school within the sample, the administrators filled out questionnaires and students were chosen for participation (Chantala and Tabor 1999). The current study will use Wave I and Wave II of the dataset. The first wave was collected in-school from 1994 to 1995 when the students were in grades 7-12. In-home interviews were also conducted in 1995 for Wave I. Wave II data was collected in 1996 when the students were in grades 8-12 (Harris et al. 2009).

Within the dataset, there is representation error. As for coverage error, adolescents that were home-schooled or in juvenile detention centers had zero probability of being selected in the survey sample. The only schools that were eligible during the stratified, random selection were those that included the 11th grade and had over 30 students enrolled (Harris et al. 2009). Furthermore, because there were a significant amount of clusters, the sampling error is most likely very low. However, there is evidence that there are school-level and student-level nonresponse error that should be adjusted for using weights (Tourangeau, Shin and the NORC 1999). Although nonresponse threatens accuracy of estimates and is especially present in longitudinal data, nonresponse bias for thirteen measures in the first three waves of Add Health has been found to be very low, around 1% (Brownstein et al. 2005). Because of the minor aforementioned representation

error, the Add Health data is generalizable to the whole population. Also, the large number of respondents supports the generalizability of the sample.

Missing Data

Because of the complex sampling design of Add Health, the data were weighted using Wave II weights and missing data were handled using listwise deletion. The original Wave I sample size included 6,504 observations. However, only cases with complete data in both waves and on each variable of interest were retained for the analyses. Potential bias due to missing cases with incomplete data were assessed by comparing the variables of interest of the observations with missing data with those cases with complete data ($n = 4,757$). Both χ^2 and t -tests were estimated to assess any potential bias. The results indicated five significant differences. Respondents who were African American ($\chi^2_{1df} = 14.34, p \leq .001$), older ($t(6499) = -33.51, p \leq .001$), smoked cigarettes at least one day in the past 30 days ($\chi^2_{1df} = 8.33, p \leq .001$), binge drank at least one day in the past 12 months ($\chi^2_{6df} = 35.52, p \leq .001$), and had higher levels of Wave I depressive symptoms ($t(6455) = -2.06, p \leq .05$) were more likely to be missing from the analyses. Given the loss of some higher risk respondents suggests that the results from the current study err on the conservative side. Finally, because only 4,834 of the original 6,504 Wave I respondents were included in the Wave II public-use data (a loss of 1,670 original respondents), the current analysis had a large percentage of missing cases (26.9%; $n = 1,747$).

Measures

Outcome variable

WII Depressive symptoms is the focal dependent variable and is conceptualized as the Feelings Scale in Wave II of the Add Health in-home questionnaire codebook. A 19-item modified Center for Epidemiological Studies (CES-D) scale was used to measure respondents' level of depressive symptoms. Often, researchers use the CES-D scale which measures depressive symptoms with strong validity and reliability (Radloff 1977). Originally, the CES-D scale included 20 items to measure depressive symptomatology (Waller et al. 2006). Wave II of Add Health modified the original CES-D scale by removing two items ("I had crying spells" and "My sleep was restless"), adding one item ("I felt that life was not worth living"), and rewording two other items (Lehrer et al. 2006b). The modified scale asks respondents about their overall emotional state during the past week, such as whether they could not shake off the blues, feeling depressed, feeling lonely, and feeling disliked by others (Radloff 1977). Item scores ranged from 0 (never or rarely) to 3 (most or all of the time). The items were then summed to create an overall scale of depressive symptoms, where a higher number represents more depressive symptoms. The CES-D is a highly valid and popular screening tool used extensively in studies on both adolescent and adult populations (Dierker et al. 2001; Goodman, Slap and Huang 2003; Warren, Harvey and Henderson 2010). Alpha reliability for this scale in the current study is .87, similar to other studies using the CES-D for adolescents (Garrison et al. 1991; Robert et al. 1990; Roberts, Lewinsohn and Seeley 1991).

Independent variables

Victimization at Wave I is a composite index of five items that taps exposure to both witnessing and personally experiencing violent victimization. Respondents were asked how often the following things happened during the past 12 months: You saw someone shoot or stab another person; Someone pulled a knife or gun on you; Someone cut or stabbed you; You got into a physical fight; and You were jumped. Response categories ranged from 0 (never) to 2 (more than once). The five items were summed to create a victimization scale (range 0-10). A higher score represents higher levels of victimization. Alpha reliability for this scale in the current study is .71.

Substance use included three items: tobacco, alcohol, and marijuana from Wave I. A skip pattern was used so respondents were only asked relevant questions. First, *cigarette use* was measured by asking respondents, “During the past 30 days, on how many days did you smoke cigarettes?” Response options ranged from 0 (no days) to 30 (thirty days). Due to skewness, the item was dichotomized so that 0 = no cigarette use in past 30 days and 1 = at least one day of cigarette use in past 30 days. Second, *binge drinking* was measured by asking respondents, “Over the past 12 months, on how many days did you drink five or more drinks in a row?” Response options ranged from 1 (every day or almost every day) to 7 (never). The response options were reverse coded so that a higher score represents higher levels of binge drinking. Third, *marijuana use* was measured by asking respondents, “During the past 30 days, how many times have you used marijuana?” Response options ranged from 0 (never) to 365 times. Due to skewness, this item was dichotomized so that 0 = no marijuana use in past 30 days and 1 = used marijuana at least one time in past 30 days. Any missing values within each of the three

substances were recoded as 0 (no use) to retain cases so results err on the conservative side.

Psychological health service utilization at Wave I is the focal mediating variable and is a single item indicator that asked respondents “In the past year, have you received psychological or emotional counseling?” Response categories were 0 (no) and 1 (yes). Any missing values were recoded as 0 (no) to retain cases so results err on the conservative side.

Control variables

Gender was coded such that 0 = male and 1 = female. *Respondent age* was calculated by subtracting respondent’s birth year from the year the study was conducted, and ranged from 12-21 years. *Respondent race* included four dichotomous variables: White, Black, Hispanic, and Other race. Finally, *depressive symptoms* is controlled for in Wave I and can be found in the Feelings Scale section of the codebook. The scale is comprised of a 19-item modified CES-D scale identical to that found in Wave II (Lehrer et al. 2006a) described above. Like the focal dependent variable, item scores ranged from 0 (never or rarely) to 3 (most or all of the time). The 19 items were summed to create an overall scale of depressive symptoms, where a higher number represents more depressive symptoms. Internal consistency reliability in the current study is .86 for Wave I CES-D.

Data Analyses

To evaluate the hypothesized relationships, ordinary least squares (OLS) regression was used as the main statistical analysis technique within the current study due to the continuous dependent variable, *WII depressive symptoms*. Using Stata, a statistical

software program, several mediation models were estimated. The conceptual figures for each mediation model are shown below in Figures 1 through 3. The first model analyzed the relationship between victimization, substance use, and depressive symptoms (see Figure 1). In order to investigate whether Wave I substance use mediated the influence of victimization on depressive symptoms, model 1 (Figure 1) estimated three single-mediator effects on this relationship: the mediating effect of Wave I cigarette use, Wave I binge drinking, and Wave I marijuana use. If there is a decrease in the direct effect of victimization on depressive symptoms when the mediator is added to the model, then there is evidence of mediation (MacKinnon 2008).

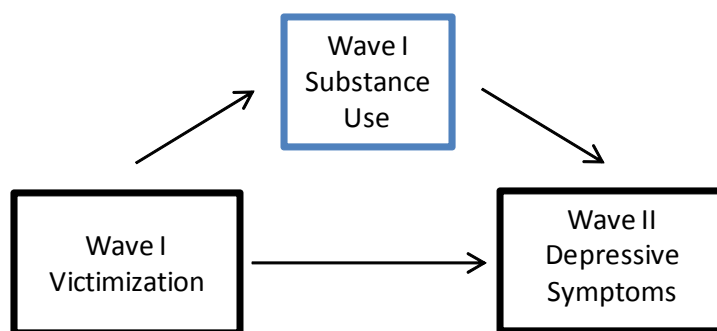


Figure 1: Conceptual Prospective Model: Wave I substance use as mediator

Next, the second mediation model investigated the relationship between substance use, psychological health service utilization, and depressive symptoms (see Figure 2). Because the influence of each substance on Wave II depressive symptoms was established in the first model described above, this focal relationship did not have to be re-run. Thus, the second model investigated the mediating effect of psychological health service utilization on the influence of each separate substance on Wave II depressive symptoms. Again, if there is a decrease in the direct effect of substance use on depressive

symptoms when the mediator is added to the model, then there is evidence of mediation (MacKinnon 2008).

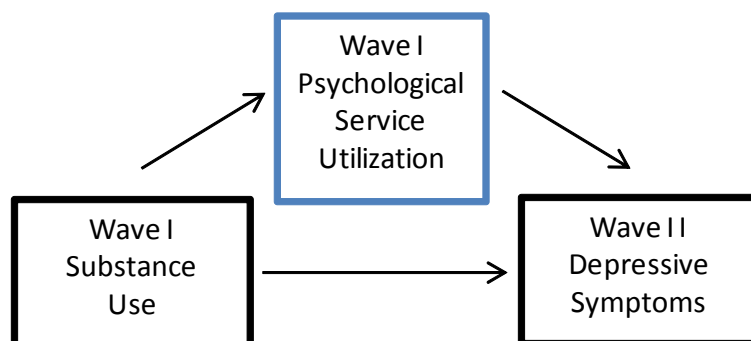


Figure 2: Conceptual Prospective Model: Wave I psychological service utilization as mediator

Finally, the third model tested the three-path mediational model (see Figure 3). In this case, both mediators, Wave I substance use and Wave I psychological health service utilization, were tested together in one model to investigate the total effect of the mediators on the relationship between victimization and depressive symptoms (Taylor, MacKinnon and Tein 2008). This final model essentially tested whether victimization is positively related to substance use, which in turn affects psychological health service utilization, and finally leads to depressive symptoms.

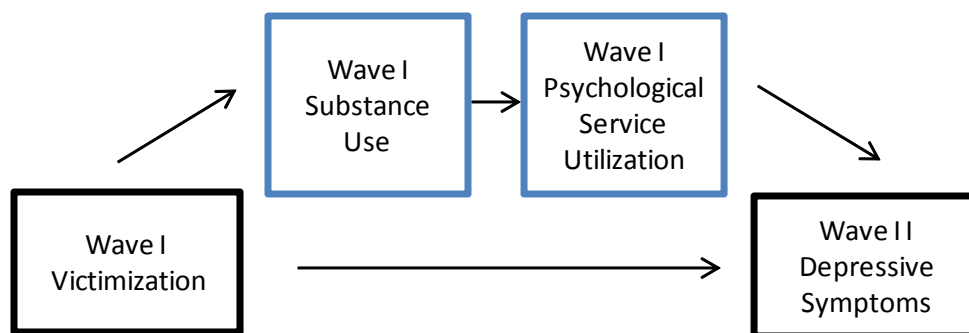


Figure 3: Conceptual Prospective Model: three-path mediational model

RESULTS

The descriptive statistics of the analyzed variables are shown in Table 1. The final sample (N=4,757) consists of approximately 52% female and 59% White participants whose average age was 15.63 years old. See Table 1 for means, standard deviations (*SD*), and range.

Table 1: Descriptive Statistics (N=4757)

Variable	Mean	SD	Min	Max
<i>WII Depressive Symptoms</i>	10.76	7.48	0	56
<i>WI Independent Variables</i>				
Cigarette Use	.2438		0	1
Binge Drinking	.5871	1.26	0	6
Marijuana Use	.1234		0	1
<i>WI Psychological Service Utilization</i>	.1246		0	1
<i>WI Controls</i>				
Female	.5228		0	1
Age	15.63	1.59	12	21
White	.5878		0	1
Black	.2317		0	1
Hispanic	.1154		0	1
Other	.0652		0	1
Depressive Symptoms	10.81	7.43	0	50
Victimization	.9382	1.60	0	10

Before running the regression models, bivariate correlations among the study variables were examined (see Table 2). Analyses at the bivariate level revealed high correlations between Wave II depressive symptoms and each independent variable at Wave I ($p \leq .001$). Also, Wave I cigarette use, binge drinking and marijuana use were significantly correlated with victimization and psychological health service utilization.

Table 2: Correlation Matrix of Study Variables

Variable	1	2	3	4	5	6	7	8	9	10
1. WII Depressive Symptoms	1.0									
2. WI Cigarette Use	.18***	1.0								
3. WI Binge Drinking	.12***	.40***	1.0							
4. WI Marijuana Use	.15***	.41***	.41***	1.0						
5. Victimization	.15***	.15***	.24***	.22***	1.0					
6. Psych. Service Utilization	.15***	.16***	.13***	.15***	.11***	1.0				
7. Female	.14***	-.02	-.10***	-.04	-.19***	.04	1.0			
8. Age	.10***	.16***	.22***	.13***	.09***	.02	-.06**	1.0		
9. Race	-.12***	.14***	.08***	.01***	-.15***	.06**	-.01	-.02	1.0	
10. WI Depressive Symptoms	.60***	.19***	.16***	.17***	.18***	.18***	.13***	.13***	-.13***	1.0

* $p \leq .05$, ** $p \leq .01$, *** $p \leq .001$

In order to investigate the effects of victimization, substance use, and psychological health service utilization on adolescent depressive symptoms, three regression analyses were estimated using weighted data from Add Health. OLS regression was used as the main statistical analysis technique for the continuous outcome variable of adolescent depressive symptoms. All models controlled for gender, age, race, and Wave I depression symptoms. Whites are the omitted racial group.

Table 3 examined the effect of Wave I cigarette use on adolescent depressive symptoms. Model 1 in Table 3 revealed that victimization was positively associated with adolescent depressive symptoms ($\beta = .048$; $p \leq .001$). That is, adolescents who

experienced higher levels of victimization reported higher depressive symptomology. This model explained 36% of the variance in the dependent variable. This relationship supports Hypothesis #1.

Table 3: OLS Regression Analyses of WI Cigarette Use on WII Depressive Symptoms

	Model 1		Model 2		Model 3	
	<i>B</i>	<i>SE</i>	<i>B</i>	<i>SE</i>	<i>B</i>	<i>SE</i>
<i>Controls</i>						
Female	.075 ***	.187	.074 ***	.186	.073 ***	.187
Age	.018	.064	.010	.067	.009	.067
Black	.030 *	.271	.041 **	.260	.044 **	.260
Hispanic	.061 ***	.335	.067 ***	.335	.068 ***	.337
Other	.021	.457	.024	.464	.025	.466
WI Depressive Symptoms	.563 ***	.017	.551 ***	.018	.546 ***	.018
Victimization	.048 ***	.059	.037 **	.060	.034 *	.061
<i>WI Cigarette Use</i>			.067 ***	.265	.064 ***	.266
<i>Psychological Service Utilization</i>					.033 *	.348
R-Squared	.362		.366		.367	

* $p \leq .05$, ** $p \leq .01$, *** $p \leq .001$

All models controlled for gender, age, race, and Wave I depression symptoms. Whites are the omitted racial group. N=4,757

Because this relationship was supported, possible mediators (i.e., cigarette use, binge drinking, marijuana use, and psychological health service utilization) were tested by investigating whether the independent variable was associated with depressive symptoms, whether the mediator was associated with the independent variable, and whether after including both the mediator and the independent variable in the model, the independent variable would still be associated with depressive symptoms (Baron and Kenny 1986). In order to test the significance of the indirect paths from the independent variable to the mediator, and from the mediator to the dependent variable, the joint

significance test was employed (Hines and Straus 2007). Compared to other tests of mediation, the joint significance test has the most conservative Type I error rate and best statistical power in testing mediational effects (MacKinnon et al. 2002). Also, the joint significance test can be used to test single-mediator models and has recently been adapted to test three-path mediational models (Taylor et al. 2008). Thus, the joint significance test was computed in Stata using the Sobel-Goodman Mediation test (Goodman 1960; MacKinnon et al. 2002; Sobel 1982) for each mediational model in the present study¹.

To calculate the percentage of the total effect that was mediated and the z-score, Stata used the following equations:

$$\textit{Proportion of Total Effect that is Mediated} = \frac{\beta_{\textit{indirect effect}}}{\beta_{\textit{total effect}}} = \frac{(\beta_{IV \textit{ before mediation}} - \beta_{IV \textit{ after mediation}})}{\beta_{IV \textit{ before mediation}}}; z = \frac{\beta_{\textit{indirect effect}}}{SE_{\textit{indirect effect}}}$$

The β of the independent variable before mediation is the unstandardized regression coefficient when the mediating variable is not included in the model. The β of independent variable after mediation is the unstandardized regression coefficient when the mediating variable is included in the model. The β of the indirect effect (numerator) is also the β that is produced for the Sobel-Goodman coefficients. To turn the proportion into a percentage, the proportion is multiplied by 100.

Accordingly, the mediating effect of each Wave I substance on this association was tested through separate single-mediator models. First, the Sobel-Goodman test demonstrated that victimization was predictive of cigarette use (not shown in table: $\beta = .891$; $p \leq .001$), which then predicted depressive symptoms (Model 2 in Table 3: $\beta =$

¹ Though both gender and protective factor interaction terms were tested in each of the models, neither variable was significant; therefore, were not included in the results.

.067; $p \leq .001$). The Sobel-Goodman test statistic for this mediational model revealed that 24.1% ($= \frac{.2252-.171}{.2252} * 100$) of the total effect was mediated by cigarette use ($Z = \frac{.2252-.171}{.0138} = 3.93$; $p \leq .001$). Models 1 and 2 in Table 3 demonstrated that there was a decrease in the direct effect of victimization on depressive symptoms when Wave I cigarette use was added to the model. This decrease in the direct effect is evidence of mediation (MacKinnon 2008).

Table 4: OLS Regression Analyses of WI Binge Drinking on WII Depressive Symptoms

	Model 1		Model 2		Model 3	
	B	SE	B	SE	B	SE
<i>Controls</i>						
Female	.075 ***	.187	.075 ***	.188	.074 ***	.190
Age	.018	.064	.015	.067	.016	.067
Black	.030 *	.271	.032 *	.271	.035 **	.269
Hispanic	.061 ***	.335	.061 ***	.337	.063 ***	.340
Other	.021	.457	.022	.458	.023	.461
WI Depressive Symptoms	.563 ***	.017	.562 ***	.017	.556 ***	.017
Victimization	.048 ***	.059	.045 **	.060	.042 **	.089
<i>WI Binge Drinking</i>			.014	.089	.011	.089
<i>Psychological Service Utilization</i>					.039 *	.345
R-Squared	.362		.362		.363	

* $p \leq .05$, ** $p \leq .01$, *** $p \leq .001$

All models controlled for gender, age, race, and Wave I depression symptoms. Whites are the omitted racial group.

N=4,757

Next, the process was repeated to test the mediating effect of Wave I binge drinking and the mediating effect of Wave I marijuana use on the relationship between victimization and depressive symptoms. Although the Sobel-Goodman test found that binge drinking did not mediate the relationship ($Z = \frac{.2252-.2102}{.0168} = .893$; $p > .05$; see Table 4 for β 's), the test for the mediating effect of marijuana use revealed that 16.2% (=

$\frac{.2252-.1888}{.2252} * 100$) of the total effect was mediated ($Z = \frac{.2252-.1888}{.01557} = 2.34$; $p \leq .05$).

Indeed, victimization was associated with marijuana use (not shown in table: $\beta = 1.29$; $p \leq .001$), which then predicted depressive symptoms (Model 2 in Table 5: $\beta = .039$; $p \leq .05$). Model 1 and 2 in Table 5 demonstrated the decrease in the direct effect of victimization on depressive symptoms when marijuana use was added to the model.

These results partially support Hypothesis #2 that both cigarette use as well as marijuana use partially mediated the association between victimization and depressive symptoms. Binge drinking, however, did not mediate this relationship.

Next, to test Hypothesis #3, the mediating effect of psychological health service utilization on the association between Wave I substance use (cigarette use; marijuana use) and Wave II depressive symptoms was tested through two single-mediator models.

According to the Sobel-Goodman test, cigarette use was positively associated with psychological health service utilization (not shown in table: $\beta = .827$; $p \leq .001$), which then predicted depressive symptoms (Model 3 in Table 3: $\beta = .033$; $p \leq .05$). That is,

5.2% ($= \frac{1.151-1.091}{1.151} * 100$) of the total effect was mediated by psychological health

service utilization ($Z = \frac{1.151-1.091}{.0302} = 1.99$; $p \leq .05$). Models 2 and 3 in Table 3

demonstrated that there was a decrease in the direct effect when psychological health service utilization was added to the model. Likewise, the Sobel-Goodman test found that marijuana use was positively related to psychological health service utilization (not shown in table: $\beta = .710$; $p \leq .001$), which then predicted depressive symptoms (Model 3

in Table 5: $\beta = .063$; $p \leq .05$). Thus, 9.4% ($= \frac{.8737-.7916}{.8737} * 100$) of the total effect was

mediated by psychological health service utilization ($Z = \frac{.8737-.7916}{.0398} = 2.06$; $p \leq .05$).

Model 2 and 3 in Table 5 showed that there was a decrease in the direct effect when psychological health service utilization was added to the model. Thus, Hypothesis #3 was partly supported.

Table 5: OLS Regression Analyses of WI Marijuana Use on WII Depressive Symptoms

	Model 1		Model 2		Model 3	
	<i>B</i>	<i>SE</i>	<i>B</i>	<i>SE</i>	<i>B</i>	<i>SE</i>
<i>Controls</i>						
Female	.075 ***	.187	.075 ***	.186	.074 ***	.184
Age	.018	.064	.014	.065	.015	.065
Black	.030 *	.271	.032 *	.268	.035 **	.268
Hispanic	.061 ***	.335	.061 ***	.335	.063 ***	.334
Other	.021	.457	.022	.458	.023	.456
WI Depressive Symptoms	.563 ***	.017	.558 ***	.018	.552 ***	.018
Victimization	.048 ***	.059	.040 **	.062	.037 *	.063
<i>WI Marijuana Use</i>			.039 *	.361	.036 *	.366
<i>Psychological Service Utilization</i>					.036 *	.346
R-Squared	.362		.363		.364	

* $p \leq .05$, ** $p \leq .01$, *** $p \leq .001$

All models controlled for gender, age, race, and Wave I depression symptoms. Whites are the omitted racial group.
N=4,757

Finally, in order to test multiple mediators at once, two three-path mediational models were tested (Taylor et al. 2008). Following statistical methods adapted from Preacher and Hayes (2008) for Stata, the proportion of the total effect that is mediated is calculated by dividing the total indirect effect of both mediators by the sum of the total indirect effect plus the total direct effect. The proportion is then multiplied by 100 to obtain the percentage of the total effect that is mediated. The first model included both Wave I cigarette use and Wave I psychological health service utilization as mediators on the focal relationship between Wave I victimization and Wave II depressive symptoms.

25.2% $\left(= \frac{.0834}{(.0834+.2475)} * 100 \right)$ of the total effect was mediated by cigarette use and psychological health service utilization (Stata FAQ 2013). Furthermore, the second model included both Wave I marijuana use and Wave I psychological health service utilization as mediators on the focal relationship between victimization and depressive symptoms. Indeed, 21.2% $\left(= \frac{.0702}{(.0702+.2607)} * 100 \right)$ of the total effect was mediated by these two mediators. These three-path mediational models essentially tested and supported the model that higher levels of victimization were positively related to substance use (cigarette use; marijuana use), which in turn positively influenced psychological health service utilization, thereby increasing levels of depressive symptoms.

DISCUSSION

The purpose of the current study was to explore the relationships between victimization, adolescent substance use, psychological health service utilization, and adolescent depressive symptoms. The results indicate that victimization is positively related to adolescent depressive symptoms and this relationship was partially mediated by adolescent substance use. Additionally, psychological health service utilization partially mediated the significant relationship between adolescent substance use (cigarette or marijuana) and adolescent depressive symptoms. Finally, the three-path mediational models including substance use and psychological health service utilization partially mediated the relationship between victimization and adolescent depressive symptoms.

The first analysis examining the effect of Wave I victimization on Wave II depressive symptoms revealed a positive association which indicates that adolescents

with higher levels of victimization experience greater depressive symptoms. This finding supports Hypothesis #1 and is also consistent with previous research which finds a positive association between neighborhood victimization and depression (Aneshensel and Sucoff 1996; Gutman et al. 2005; Howard et al. 2002). It is possible that experiencing or witnessing victimization can cause an adolescent to feel chronically unsafe instead of comfortable and secure. As such, feeling unsafe may lead to emotional distress and eventually increase levels of depressive symptoms among these exposed adolescents.

Results from the first model which tested a mediating effect revealed that both Wave I cigarette and marijuana use partially mediated the direct effect of Wave I victimization on Wave II depressive symptoms. In other words, adolescents who experience higher levels of victimization smoke cigarettes or marijuana at least once which in turn is associated with higher levels of depressive symptoms. This finding supports Hypothesis #2 and is generally supportive of prior research which finds a positive association between victimization and greater substance use (Lo, Kim, and Church 2008; Silverman et al. 2001) and a positive association between substance use and depressive symptoms (Brook et al. 2002; Degenhardt et al. 2003; Poulin et al. 2005; Swendsen and Merikangas 2000; Vida et al. 2009; Wade and Pevalin 2005). These findings may be explained by the fact that the stress associated with being personally victimized or witnessing victimization may encourage some adolescents to use substances such as cigarettes or marijuana as a way to cope with the stress. Consequently, substance use increases levels of depressive symptoms because of the many negative consequences of drug use. For example, substance use and other delinquent behavior have been shown to eliminate more pro-social behavioral opportunities (Laub, Nagin, and

Sampson 1998; Sampson and Laub 1997) which, in turn increase the likelihood of depression (Mirowsky and Ross 1992) and many other ensuing problems (Patterson and Yoerger 1993).

Contrary to what was hypothesized, Wave I binge drinking did not mediate the relationship between victimization and depressive symptoms because it was not predictive of Wave II depressive symptoms. One possible explanation may be due to the fact that adolescents who binge drink may find it more socially acceptable and are, thus, less likely to experience future depressive symptoms because the behavior is generally not viewed as deviant by society compared to other substances (Becker 1963). The lack of a significant finding may also be due to the manner in which binge drinking was coded. That is, while cigarette use and marijuana use were dichotomized binge drinking had seven response options. Because of the young age of many of the respondents, it is unlikely that the majority of them have binge drank on several occasions.

Model two which tested a second mediating effect revealed that psychological health service use partially mediated the effect of both Wave I cigarette and marijuana use on Wave II depressive symptoms. That is, adolescents who smoke cigarettes or marijuana at least once are more likely to utilize psychological health services, which, in turn leads to greater depressive symptoms. Because no studies to date have empirically tested this model before, there are no findings to which current study results can be compared. The current study findings only partly support Hypothesis #3. While substance use was correlated with psychological health service utilization, the positive effect between psychological health service utilization and depressive symptoms was not expected. One possible explanation may be that adolescents who utilize psychological

health services become more aware of their mood or feelings after talking with a counselor and, therefore, report higher levels of depressive symptoms than adolescents who do not utilize psychological health services. Another potential explanation is that adolescents who used health services in Wave I did so because they were experiencing depressive symptoms before accepting the services. These earlier depressive symptoms may then be the cause of higher levels of depressive symptoms in Wave II. Thus, it is possible that these earlier depressive symptoms, which cannot be controlled for using Add Health data, were the driving force for both Wave I psychological health service utilization and continued high levels of depressive symptoms in Wave II.

Finally, the third model, which tested the three-path mediational model revealed that adolescents who experience higher levels of victimization are more likely to report cigarette and marijuana use, which, in turn is correlated with utilization of psychological health services, thereby increasing depressive symptoms. Although prior research has not examined this specific model, one potential explanation for these findings is that stressors from both victimization and substance use lead some adolescents to seek more psychological help from health services but this does not decrease the level of future adolescent depressive symptoms.

Overall, the stress process model is a useful framework for understanding these findings. Consistent with the stress process model, the current study findings demonstrate a positive effect of victimization on later adolescent depressive symptoms which is consistent with previous research (Latkin and Curry 2003; Pearlin 1989; Pearlin et al. 2005). This finding also demonstrates how distal factors are important for understanding mental health outcomes (Latkin and Curry 2003). Though the significant mediating effect

of cigarette use and marijuana use on victimization and depressive symptoms is consistent with this theory, the mediating influence of psychological health service utilization on depressive symptoms did not act as a protective factor. That is, although psychological health service utilization mediated the direct effect of substance use on adolescent depressive symptoms it was in the opposite direction of what was hypothesized. Overall, the current findings are generally supportive of a stress process model in that both distal and proximal risk factors are important for explaining adolescent depressive symptoms and mediating influences also play a significant role in understanding mental health outcomes.

Limitations

As with all social research, the current study has some limitations that should be noted. First, there is a possibility of respondent biases in the data because all information was self-reported. For example, due to social desirability bias, some youth may have under-reported their use of substances. Second, although the relevance of both mediators in understanding adolescent depressive symptoms was demonstrated, mediation was only partial which suggests that other significant factors were not investigated. For example, research has demonstrated a significant association between family influences, such as child abuse, and later adolescent depression (Brown et al. 1999; Harkness, Bruce and Lumley 2006; Stuewig and McCloskey 2005). Because Add Health does not include assessments of child abuse until Wave III, however, the current study did not control for these family variables. Third, the temporal order of the Wave I measures is unknown because the Add Health survey questions do not indicate which occurred first if measured

in the same wave. Thus, it is possible that substance use preceded victimization or that psychological health service utilization preceded substance use, rather than vice versa. Because the timing is unknown, causation between the Wave I measures could not be certain. Finally, because health service utilization was limited to a single item it is possible that current results may have been different if other types of services were included. Though Add Health does include other services (e.g., family planning) they were either not relevant to the dependent variable in the current study or were not predictive of depressive symptoms.

Implications

Current study findings have important implications for preventing adolescent substance use and adolescent depressive symptoms. Research has shown that there is a visible rise in health problems among adolescents (Wickrama et al. 2009). It is not surprising, then, that adolescents have relatively high rates of substance use (Johnston et al. 2011), which is associated with later major depressive disorder (McGue and Iacono 2005; Vida et al. 2009). Thus, there is a need for more prevention programs for adolescents and especially those in disadvantaged neighborhoods and those who have witnessed or personally experienced victimization. Additionally, Clark and colleagues (2011) suggest the creation of more drug prevention programs that work to decrease the level of positive expectations of substance use by adolescents. Also, previous research with high school samples has focused on pinpointing social situations where substance use occurs and then using interactive videos as a way to challenge social norms by teaching young people about social norms, attitudes, and beliefs that are associated with

effective refusal skills (Duncan, Duncan, Beauchamp, Wells and Ary 2000). These interventions have been effective in changing personal self-efficacy and perceptions about social norms surrounding substance use (Duncan et al. 2000). Prevention programs that take place in school settings are likely to have a lot of success. As Gottfredson explains, schools are one of the best places to conduct prevention programs because a school is the “only institution that provides consistent access to millions of children throughout their important developmental years” (Petrosino 2003: 182). Creative programs such as the one outlined above are likely to have the most success and should be established in schools from kindergarten to high school in order to educate adolescents and to reformulate their norms regarding substance use.

Future studies should build upon findings from the current project in order to further our understanding of risk factors for adolescent depressive symptoms. One suggestion for future research is to investigate other possible mediators of the relationships between victimization, substance use, and adolescent depressive symptoms. Because mediator effects were only partial in the current study, there are other important factors that need to be investigated, such as child abuse variables. Similarly, because temporal order could not be established among some of the current measures, future studies should analyze three or more waves of Add Health to develop causal order. Finally, future research may wish to examine young people’s perceptions regarding why they or their friends use substances which may lead to better prevention strategies and programs in school, which subsequently may lower the negative mental health outcomes among adolescents.

CONCLUSION

The current study investigated the effect of victimization, substance use, and psychological health service utilization on depressive symptoms in a school sample of U.S. adolescents. The analyses added to the existing literature by examining the mediating influence of psychological health service utilization on the effect of substance use on depressive symptoms among adolescents. Overall, the findings indicate that adolescents who experience more victimization may be at increased risk of using cigarettes or marijuana. Additionally, using these substances is associated with young people's chances of utilizing psychological health services, which, in turn, results in higher levels of depressive symptoms approximately one year later. As such, these results demonstrate that both distal and proximal stressors are associated with negative health outcomes among a national sample of U.S. adolescents. A better understanding of adolescent stressors and possible protective factors could aid in developing future prevention strategies to decrease the risk of future depression and other poor health outcomes among this population.

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