Time-specific Associations and Developmental Trajectories of Co-occurring Substance Use and Disordered Eating among Adolescent Girls

by

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ABSTRACT

Despite the established co-prevalence of substance use (SU) and disordered eating (DE), few longitudinal studies have sought to examine their shared development. Findings have been inconsistent within the extant literature. This may be attributable in part to several methodological aspects, including overlooking distinct psychopharmacological properties of common substances of abuse, examining only between-person relations, and failing to account for shared risk factors. The current study sought to address these gaps by applying latent curve models with structured residuals (LCM-SR) to a preexisting, national sample of adolescent girls followed into adulthood, Add Health. In Aim 1, between-person effects examined the simultaneous development of alcohol, tobacco, and marijuana use and DE behaviors in substance-specific models. In Aim 2, bivariate latent curve models were expanded to account for within-person effects (LCM-SR) in order to examine the potentially bidirectional, prospective relationship between use of a specific substance and DE. Lastly, models accounted for shared developmental risk factors. Findings of the current study demonstrate preliminary evidence of substance-specific effects with DE emerging in adolescence. Across model-building steps, DE engagement in early adolescence was significantly associated with growth in tobacco use and marginally associated with marijuana use. Appetitive side-effects of both substances may link use with DE behaviors and enhance instrumental use for weight control. Significant associations did not emerge between alcohol and DE, and results of the conditional model indicate this co-occurrence is best explained by third variable mechanisms. Implications for prevention are discussed.

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INTRODUCTION

Adolescence is a period of marked elevations in psychopathology. The British Child and Adolescent Mental Health Survey (BCAMHS), the largest epidemiological study to cover the transition from childhood to adolescence, showed that rates of DSM-IV disorders steadily rose from 8.6% among those 8- to 10-years old to 9.6% at 11 and 12 to 12.2% at 13- to 15-years old (Green, McGinnity, Meltzer, Ford, & Goodman, 2005). In a review, the average rate of any adolescent psychiatric disorder was estimated as 21.8%, with drug abuse or dependence being the most common (12.1%) (Costello, Copeland, & Angold, 2011). Early psychiatric diagnosis is an early marker of psychiatric disorders in adulthood. In one prospective longitudinal study, 73.9% of those with an adult psychiatric diagnosis received a diagnosis before 18-years old and 50% before the age of 15 (Kim-Cohen et al., 2003). Among adolescent girls alone, 18% experienced the emergence of a mental health disturbance during adolescence (Moffitt, Caspi, Rutter, & Silva, 2001). Given such epidemiological findings, understanding etiology and course of psychopathology in adolescence is critical for early detection as well as prevention and intervention efforts designed to reduce the overall burden of mental illness on public health.

Additionally, comorbid diagnoses emerge during this period and continue to impact individuals well into adulthood. In one study, more than three-fourths (79%) of adults with a lifetime psychiatric diagnosis met criteria for more than one disorder (Kessler et al., 1994). Rates of comorbid diagnoses among adolescent girls surpass rates of a single diagnosis (Angold, Costello, & Erkanli, 1999; Kessler et al., 1994) and

continue to increase with age (Tolan & Henry, 1996). In a prospective study of adolescent girls 13- to 18-years old, elevations in depressive symptoms, disordered eating (DE), and substance use (SU) were significantly and positively associated with elevations in at least one other domain. Moreover, growth in one symptom domain was significantly associated with growth in others (Measelle, Stice, & Hogansen, 2006). The increasing rates of psychiatric disorders in adolescence have been primarily attributed to the surge in SUDs (SUD) and EDs (ED) during this time (Costello et al., 2011), two diagnoses whose development seem to be coupled among young women.

Characteristic of the broader literature, peak onset of SU and DE occurs in adolescence (Costello et al., 2011). Onset of SU in early to mid-adolescence is predictive of a stable and escalating course of substance abuse among girls (Chassin, Pitts, & Prost, 2002; Nagin & Tremblay, 2001). Girls also show evidence of addiction symptoms and suffer consequences sooner than their male peers (Piazza, Vrbka, & Yeager, 1989; The National Center on Addiction and Substance Abuse at Columbia University, 2003). SU trajectories are associated with other forms of psychopathology, including DE (Angold et al., 1999). Indeed, substance misuse among young girls has been linked to negative body image and engagement in DE behaviors (Parkes, Saewyc, Cox, & MacKay, 2008; Schinke, Fang, & Cole, 2008; Wolfe & Maisto, 2000). In a large sample of adolescents from American high schools, tobacco use, binge drinking, and cocaine use were positively associated with the number of DE behaviors endorsed (Eichen, Conner, Daly, & Fauber, 2012). Specifically, increases in use of alcohol, tobacco, and illicit drugs have

been observed among girls who engage in purging behaviors (Pisetsky, May Chao, Dierker, May, & Striegel-Moore, 2008).

This co-occurrence is found in clinical as well as normative samples. An estimated 37.8% of adolescent girls with an ED also meet criteria for comorbid SUD, and 12.4% with a SUD meet criteria for an ED (Stice, Presnell, & Bearman, 2001). Among 11,103 adolescents across American high schools, binge drinking positively predicted the number of DE behaviors endorsed (Eichen et al., 2012). Moreover, bulimic behaviors were significantly linked to negative consequences from drinking or marijuana use as well as binge drinking in community and university samples of young adult women (Piran & Robinson, 2011). Given the well-documented overlap across adolescence and adulthood (e.g., Corcos et al., 2001; Dansky, Brewerton, & Kilpatrick, 2000; Eichen et al., 2012), understanding developmental processes and time-specific associations between DE and SU is critical for prevention and intervention efforts in adolescence. Thus, the primary focus of this project is to address several limitations in the literature to better understand causal relations between DE and SU among females during the critical transition from adolescence to young adulthood.

To date, much of the literature on co-occurring SUD and ED is descriptive and heavily relies on documenting co-prevalence. While this knowledge is important for informing clinical interventions and informing research inquires, it fails to address *why* these two syndromes often intersect. Information regarding the nature of this comorbid relation would be beneficial in moving the field beyond co-prevalence and informing theory for the purposes of scientific investigation and clinical intervention.

Although there has been much speculation about the nature of relations between DE and SU, the lack of well-designed empirical investigations and longitudinal studies makes it difficult to arrive at any firm conclusions regarding why and how this comorbidity emerges. The literature broadly offers two hypotheses to clarify and explain this relation. The first suggests that these syndromes may emerge from a shared etiology or common psychopathological predisposition. While many factors have been proposed as potential underlying mechanisms explaining this comorbidity (e.g., familial and peer influence, genetic risk, exposure to adverse events), the current study focuses on the impact of early sexual development as well as internalizing and externalizing symptoms in an attempt to account for both the importance of developmental timing and transitions and broad domains of psychological and behavioral risk. In contrast to the shared risk factors hypothesis, researchers have proposed a causal relation where having problems in one area increases one's vulnerability for developing problems in the other area. Although DE can emerge as a consequence of SU (e.g., Camp, Klesges, & Relyea, 1993; French, Popovici, & Maclean, 2009), the majority of prior studies have suggested that the potentially causal relation is based on the initial presence of DE contributing to SU.

Shared Etiology Through Common Developmental Risk Factors

Early Sexual Maturation

Early sexual maturation is both cross-sectionally and prospectively predictive of SU (e.g., Tschann et al., 1994) and DE for girls (e.g., Le Grange et al., 2014). For instance, early-maturing girls were found to have a lifetime history of substance abuse at twice the rate experienced by on-time or late-maturing girls (Graber, Lewinsohn, Seeley,

& Brooks-Gunn, 1997). In addition, early-maturing girls with more depressive symptoms, showed lower levels of self-esteem, higher rates of current and past tobacco use, and were more likely to have lifetime history of EDs compared to on-time girls (Graber et al., 1997; Graber, Seeley, Brooks-Gunn, & Lewinsohn, 2004). Thus, early sexual maturation may potentially account for relations between SU and DE.

The early-maturation hypothesis proposes that early sexual maturity short-circuits developmental transitions psychologically and physiologically, thereby increasing the discrepancy between a young girl's developing, womanly figure and the ultra-thin societal body ideal for women. This deviation from the ideal body type increases risk for DE and increases exposure to SU through affiliation with older, deviant peers (e.g., Brooks-Gunn, Petersen, & Eichorn, 1985; Caspi & Moffitt, 1991). Research has found that early sexual maturation is associated with higher BMIs at various ages across adolescence, and that increased BMI is linked to increased body dissatisfaction and dieting behaviors (Striegel-Moore et al., 2001). Cross-sectional (e.g., Patton et al., 2004) and longitudinal research (e.g., Dick, Rose, Viken, & Kaprio, 2000; Tschann et al., 1994) similarly supports early sexual maturation as a risk factor for SU.

As dieting behaviors increase with age, older female peers engage in more "fat talk" (negative talk about one's own body; Nichter & Phillips, 2003) and discussion of dieting methods. Exposure to this talk among peers thereby promotes greater pressure to be thin (Nichter & Phillips, 2003; Stice, 1994). For example, one study found that 93% of college women engaged in fat talk with their friends (Salk & Engeln-Maddox, 2011). This peer-generated fat talk has been described as socially contagious (Salk & Engeln-

Maddox, 2012). These self-statements may be related to body dissatisfaction, (e.g., "I feel so fat") or specific dieting behaviors and motives (e.g., "I really want a thigh gap, so I've been upping my mileage on runs and am trying this new diet") (Stice, Rohde, & Shaw, 2013).

Moreover, dieting methods may be indirectly promoted for young girls through modeling of behaviors (Bandura, 1969; Bandura, Ross, & Ross, 1963). Research has shown this to be true in mother-daughter dyads (e.g., Attie & Brooks-Gunn, 1989; Hill & Pallin, 1997). Achieving sexual maturation early may increase the likelihood of a young adolescent girl being older than her biological age. Moreover, it may increase her exposure to older, deviant peers who promote and model dieting behaviors directly and indirectly while also increasing conformity motives for and access to SU. In tandem, early sexual maturation may serve as a causal mechanism in the development of both syndromal domains.

It is also possible that off-time developmental events, such as early sexual maturation, may increase emotional distress that then mediates the relation between early sexual maturation and risky health behaviors (Brooks-Gunn et al., 1985; Caspi & Moffitt, 1991). Early maturing girls experience dysynchrony in hormonal, physical, psychological, and social processes. This, in turn, may increase vulnerability to environmental stress, leading to an increase in risky health behaviors such as extreme dieting and SU (e.g., Dawes et al., 2000; Magnusson, Stattin, & Allen, 1985). For example, traditional models of how early sexual maturation leads to DE have focused on the role of body dissatisfaction. The increase in body image distress may be

consequential to an off-time developmental transition, which then contributes to the dual-pathway model of ED (Stice, 1994) and subsequent initiation of SU.

Internalizing Symptoms

Adolescent girls are particularly vulnerable to internalizing symptoms (e.g., low mood and anxiety) during puberty due to hormonal changes (Angold, Costello, & Worthman, 1998) and shifting social contexts and expectations (e.g., Brooks-Gunn & Warren, 1989). Elevations in internalizing symptoms are predictive of increases in DE and SU (e.g., Measelle et al., 2006). For instance, adolescent girls with higher levels of negative affect are more likely to negatively evaluate their own body and engage in upward comparisons with others' bodies, contributing to low self-esteem and body dissatisfaction (Cash & Fleming, 2002). In accordance with the dual pathway model of ED (Stice, 1994), early internalization of the thin ideal may promote negative affect and increased body dissatisfaction, thereby increasing the likelihood of engaging in dieting behaviors. These dieting attempts are ultimately unsuccessful (Korkeila, Rissanen, Kaprio, Sorensen, & Koskenvuo, 1999; Mann et al., 2007; Wadden, Foster, Letizia, & Mullen, 1990) and result in further increases in body dissatisfaction and negative affect. Moreover, these unsuccessful attempts promote experimentation with more extreme dieting behaviors (e.g., self-induced vomiting, diuretic misuse, laxative misuse) (Huon, 1994; Lowe & Caputo, 1991; Neumark-Sztainer, Story, Resnick, Garwick, & Blum, 1995; Neumark-Sztainer, Wall, Haines, Story, & Eisenberg, 2007). A recent meta-analysis of 42 longitudinal studies showed bidirectional risk for depression and ED diagnosis and developmental effects, such that the effect of depression on eating

pathology was strongest among younger samples (Puccio, Fuller-Tyszkiewicz, Ong, & Krug, 2016). Indeed, the presence of depression symptoms as early as 5th grade has been shown to predict developmental trajectories of compensatory behaviors over a three-year period (Davis, Guller, & Smith, 2016).

Negative affect is also an important risk factor for SU, particularly among girls. Research on gender differences indicates that negative affect significantly predicts SU in girls over models primarily emphasizing impulsivity and externalizing, which have garnered more support among boys (Maclean, Paradise, & Cauce, 1999). Further, low levels of positive affect prospectively predict tobacco, alcohol, and marijuana use (Colder & Chassin, 1999; Wills, Sandy, Yaeger, Cleary, & Shinar, 2001). Other studies suggest that negative affect is significantly associated with and predictive of cigarette smoking, alcohol use, and marijuana use among college students over and above measures of stress (e.g., Hussong, Jones, Stein, Baucom, & Boeding, 2011; Magid, Colder, Stroud, Nichter, & Nichter, 2009) and cross-sectionally predicts SU across drug type (Colder & Chassin, 1999; Patton et al., 2004; Verdejo-García, Bechara, Recknor, & Pérez-García, 2007). A recent review investigating an internalizing pathway for SU found that depression symptoms were most consistently predictive of SU composite scores among youth (Hussong, Ennett, Cox, & Haroon, 2017).

Taken together, the self-medication model has been applied to the SU-ED relationship due to the high prevalence of depression among those engaging in SU (e.g., Kilpatrick et al., 2003; Schwinn, Thom, Schinke, & Hopkins, 2015) and DE (e.g., Thew, Gregory, Roberts, & Rimes, 2017). Originally specific to SU, the model posits that

individuals may turn to substances for relief of painful affective states (Khantzian, 1995). It has since been expanded to include comorbid ED, conceptualizing SU as a way of coping with amplified negative affective consequence of DE behaviors (Harrop & Marlatt, 2010; Wolfe & Maisto, 2000). Thus, adolescent girls with elevated depressive symptomatology may be at increased risk for using either SU or DE as a coping mechanism.

A similar rationale underlies the tension reduction model (Kalodner, Delucia, & Ursprung, 1989), which proposes that SU is secondary to onset and escalation of anxiety symptoms in order to provide temporary relief from this mood state. One study found that alcohol dependence, cannabis dependence, and tobacco use were associated with 4.4, 4.3, and 2.4 times greater chance of meeting criteria for anxiety disorders (Degenhardt, Hall, & Lynskey, 2001). Similarly, lifetime prevalence of at least one comorbid anxiety disorder ranges from 23% to 75% among those with an ED (Swinbourne & Touyz, 2007). Onset of anxiety commonly precedes that of EDs and has been posited as a causal factor in DE development (e.g., Kaye, Bulik, Thornton, Barbarich, & Masters, 2004). As bulimia is characterized by fluctuations in tension, anxiety, and guilt in accordance with the binge/purge cycle (Mitchell & Laine, 1985), individuals engaging in such DE behaviors may have high levels of anxiety that predate an ED and experience momentary escalations in anxiety after engaging in a DE behavior (Wolfe & Maisto, 2000).

These theories suggest that internalizing symptoms may increase the onset of both SU and DE. First, individuals with DE who report higher levels of internalizing symptoms may be at greater risk for SU than individuals with DE who report lower levels

of internalizing symptoms. Second, substance-using girls who have elevated internalizing symptoms may be at higher risk for DE than substance-using girls with low levels of internalizing symptoms. Third, escalation in internalizing symptoms as a result of either syndrome may lead to initiation or escalation of the other as a means of coping. As such, internalizing symptoms may confound the causal and unique relation between DE and SU.

Externalizing Symptoms

Lastly, it has been theorized that those with co-occurring SU and DE share similar personality traits characteristic of externalizing problems. Those with prolonged and problematic SU share a pattern of erratic behavior oscillating between the extremes of restraint and disinhibition with women who engage in bingeing and purging behaviors (Vitousek & Manke, 1994). The developmental literature robustly supports an externalizing pathway to SU. Both cross-sectional and longitudinal studies consistently report prediction of not only early experimentation but also severity of use (e.g., Carlson, Johnson, & Jacobs, 2010; Tarter, Kirisci, Feske, & Vanyukov, 2007; Verdejo-García, Bechara, Recknor, & Pérez-García, 2006). In a sample from the Minnesota Twin Family study, presence of an externalizing disorder (i.e., conduct disorder, oppositional defiant disorder, and attention deficit/hyperactivity disorder) at 11-years old predicted use of tobacco, alcohol, and marijuana 3 years later (Carlson, Johnson, & Jacobs 2010). Even symptoms, such as impulsivity, independent of externalizing disorder diagnoses, are predictive of the rate of progression in substance use, diagnosis of a SUD by early adulthood, greater drinking- and/or drug-related consequences, and difficulty abstaining

from use (Dishion, Andrews, & Crosby, 1995; Kirisci, Tarter, Mezzich, & Vanyukov, 2007; Tarter et al., 2007; Verdejo-García et al., 2006).

In contrast to the SU literature, studies examining externalizing pathways for ED have largely focused on impulsivity. Numerous studies demonstrate a clear link between engagement in binge/purging behaviors and impulsivity (e.g., Waxman, 2009). Overall, personality studies have found women engaging in bingeing and purging behaviors to be more compulsive, impulsive, and neurotic than those who engage in restriction only (Vitousek & Manke, 1994). Women who engage in purging behaviors show higher levels of impulsivity than women engaging in both bingeing and purging and women who do not engage in purging behaviors (Favaro et al., 2004), suggesting that impulsivity may act as a specific indicator of risk for purging behaviors. Moreover, impulsivity measures have consistently predicted the onset of purging behaviors in longitudinal samples (Wonderlich, Connolly, & Stice, 2004).

Research considering the role of externalizing symptoms beyond impulsivity in the development of ED is limited and murky. Too often studies claim to examine externalizing symptoms yet rely on measures of substance use as indicators (e.g., Hopwood, Ansell, Fehon, & Grilo, 2010; Mitchell, Wolf, Reardon, & Miller, 2014; Slane, Burt, & Klump, 2010). Among studies using broader measures, such as the Child Behavior Checklist and Youth Self-Report, which incorporate items assessing aggression and delinquency, externalizing symptoms emerged as one of the strongest risk factors for a subset of young girls (e.g., Adambegan et al., 2011; Stice, Rohde, Gau, & Shaw, 2012; ter Bogt, van Dorsselaer, Monshouwer, Verdurmen, Engels, & Vollebergh, 2006). In a

study of 83 sister pairs, for example, young girls who later developed a bulimic disorder had significantly higher externalizing symptoms (i.e., aggressive and delinquent behavior) than their unaffected sisters (Adambegan et al., 2011). Thus, there is evidence to support externalizing symptoms among adolescent girls as a risk factor for DE, but studies using more nuanced measures are needed.

In sum, early sexual maturation, internalizing, and externalizing symptoms are common risk factors for the emergence of both SU and DE. Efforts to identify potential causal relations between SU and DE, therefore, must account for these possible shared risk factors if they are to provide definitive evidence. It is possible that these risk factors differentially relate to SU and DE over time, as these risk factors themselves change over time. An extensive literature has demonstrated that internalizing symptoms emerge and become more prevalent over the course adolescence. Conversely, externalizing symptoms tend to peak during adolescence with self-regulatory abilities improving in young adulthood as the prefrontal cortex develops (Steinberg, 2008; Steinberg et al., 2008). Among adolescent girls, depression and DE demonstrate a linear pattern of increase with SU increases exponentially during this time period (Measelle et al., 2006). If SU and DE are related to these shared risk factors, it is possible these syndromes will be most strongly related in adolescence given the developmental trajectories of these risk factors.

Exploring a Causal Etiology: Substance-Specific Effects

In contrast to the hypotheses of a shared etiology, it is possible that the relation between SU and DE is causal. In addition to failing to control for shared risk factors, a major limitation of prior work is the tendency to combine substances when examining

relations between DE and SU. This approach assumes that those who use alcohol, tobacco, marijuana, and other illicit drugs are a homogenous group (Dansky et al., 2000). It also fails to consider differences in pharmacological properties and motives for use across substances that may uniquely relate to DE behaviors. As such, this methodological approach may have contributed to inconsistencies in longitudinal findings regarding the pathogenesis of co-occurring SU and DE. The following section outlines the importance of examining potential substance-specific relations between use of various substances (e.g., tobacco, alcohol, and marijuana) and DE.

Tobacco Use

A core component that drives DE is exaggerated beliefs regarding food, calories, and weight-loss strategies (Garner, Garfinkel, & Bemis, 1982). It is widely believed that cigarettes have appetite-suppressing properties, (Gonseth, Jacot-Sadowski, Diethelm, Barras, & Cornuz, 2012) and onset of tobacco use has been shown to be associated with and a consequence of elevated body dissatisfaction, drive for thinness, and eating pathology among adolescent girls (e.g., Stice & Shaw, 2002). Given that onset of use co-occurs with the progression of dieting practices in adolescence (Camp et al., 1993; French, Perry, Leon, & Fulkerson, 1994; Klesges, Elliott, & Robinson, 1997), body dissatisfaction and early dieting attempts may make tobacco use more appealing for young girls due to the well-popularized appetitive effects and its availability among adolescents (Chiolero, Faeh, Paccaud, & Cornuz, 2008). Moreover, dieting girls may use cigarettes to cope with sensations of hunger. For instance, 55% of adult women reported smoking more when dieting and 54% smoked to suppress hunger (White, 2012). Thus,

restriction behaviors may prompt smoking initiation due to expectancies that tobacco will increase the ability to withstand hunger while dieting.

Expectations of appetite suppression may be further reinforced through tobacco use, as the physiological effects of tobacco use include the suppression of appetite. A recent review of the archived documents from 6 major US and UK tobacco companies evidenced that appetite-suppressant molecules, tartaric acid and 2-acetylpyridine, were added to cigarettes and marketed as weight-control agents (Gonseth et al., 2012). While the exact mechanism is unclear, increased metabolic rate, increased energy expenditure, and decreased caloric absorption have been proposed (Chiolero et al., 2008; Perkins, Sexton, Dimarco, & Fonte, 1994). Tobacco also may produce modest, transient anorexic effects (Perkins et al., 1994; White, McKee, & O'Malley, 2007), whereby hunger and appetite are not suppressed but still result in less caloric consumption. For example, one study found that nicotine did not alter sensations of hunger over a two-hour period, but it resulted in less caloric intake among both smokers and nonsmokers (Jessen, Buemann, Toubro, Skovgaard, & Astrup, 2005). Such findings have been used to explain why smokers are lower weight than nonsmokers as well as why quit attempts are linked with increased weight (e.g., Albanes, Jones, Micozzi, & Mattson, 1987).

These appetitive effects are well-known, and weight-related motivation for its use is commonly cited (e.g., Camp et al., 1993; French et al., 1994; Klesges et al., 1997; White, 2012). Weight-related motivation for smoking also distinguishes between experimental and regular smokers (Camp et al., 1993; French et al., 1994). Smoking initiation among adolescent girls has been found to be more frequent among those who

are overweight, trying to lose weight, or who see themselves as more overweight than their female counterparts without these characteristics (Cawley, Markowitz, & Tauras, 2004). Among adolescent girls (Klesges et al., 1997) and college women (Copeland & Carney, 2003), smoking rate and frequency are associated with weight concerns and the perception of tobacco as a weight-control aid. Such findings are in line with the sociocultural model of EDs (Stice, 1998), which proposes that those farthest from the thin ideal, or who perceive themselves to be, are more likely to have increased body dissatisfaction and motivation to engage in weight-control behaviors. Thus, initiation of tobacco use may be a consequence of pre-existing DE attitudes (e.g., body dissatisfaction, desire to lose weight) and behaviors (e.g., early dieting attempts or binge eating episodes).

The association between smoking and weight loss may also be moderated by duration and quantity of tobacco use. Tobacco's anorectic effects have been shown to be more pronounced with light- to moderate-levels of smoking, which is characteristic of tobacco use in adolescence (e.g., Griffin, Botvin, Doyle, Diaz, & Epstein, 1999). Such metabolic and appetitive effects may be recognized by light and moderate smokers with pre-existing body image concerns, thereby promoting heavier tobacco use in order to maintain or intensify the experienced physiological effects. As such, the association between tobacco use and DE may change across adolescence and into adulthood.

Although moderate smoking may aid in weight control, heavy smokers (i.e., 20 or more cigarettes a day) often have greater body mass indices (BMI) than light to moderate smokers and nonsmokers (e.g., Basterra-Gortari et al., 2010; Shimokata, Muller, &

Andres, 1989). In an 8-year follow-up of 55,000 women, nonsmokers had less weight gain than those who initiated smoking or were long-term smokers, and heavier smokers gained more weight than light smokers (Colditz et al., 1992). This may be due to heavier smokers adopting other unhealthy behaviors, such as limited physical activity, limited intake of fruits and vegetables, and higher consumption of alcohol. Unfortunately, heavy long-term smokers have a difficult time quitting and may perceive that quitting will cause further weight gain.

Increased weight and discrepancy from the thin ideal among heavy tobacco users may exacerbate DE attitudes and promote incorporation of extreme dieting behaviors such as fasting, use of diet pills, and purging (i.e., self-induced vomiting, misuse of diuretics and laxatives). Indeed, individuals with bulimia nervosa are more likely to smoke than normal controls (White et al., 2007) and psychiatric controls with affective and anxiety disorders (Welch & Fairburn, 1998). A recent meta-analysis of 31 studies found that tobacco use was more common among people with bulimia nervosa (life-time OR = 2.165) and binge eating disorder (life-time OR = 1.792) but not those with anorexia nervosa (life-time OR = 0.927) when compared to non-tobacco users (Solmi et al., 2016). Among those with EDs, tobacco use was more common among those with binge eating disorder (life-time prevalence = 47.73%) and bulimia nervosa (life-time prevalence = 39.4%) than those with anorexia nervosa (life-time prevalence = 30.8%).

Alcohol Use

When considering alcohol, women may drink in order to cope with negative side effects of eating pathology. As the most common consequences of early dieting are

increased body dissatisfaction and negative affect (Stice, 1994, 1998), alcohol may be used to cope with negative self-evaluation and depressed mood, which emerge as consequences of failed dieting attempts. Girls who initiate dieting behaviors and progress in eating pathology also report social anxiety due to their evaluation of their physical appearance (Bulik, Sullivan, Fear, & Joyce, 1997; Godart, Flament, Perdereau, & Jeammet, 2002; Wonderlich-Tierney & Vander Wal, 2010). Girls may initiate alcohol use to cope with anxiety heightened in social settings (e.g., parties). Purging behaviors, in particular, are predictive of initiation and escalation of alcohol use among adolescent and adult women (Piran & Robinson, 2006; Vidot, Messiah, Prado, & Hlaing, 2016).

Although there is a stronger empirical basis for expecting DE to predict later substance use, individual drinking occasions may be associated with increased DE behaviors. Heavy binge drinking may lead to increased consumption of food during or after drinking episodes to "sober up" or cope with negative physical side-effects, such as fatigue. This increased consumption of food may cause guilt regarding overall caloric intake associated with the drinking episode and promote a period of restriction, similar to the known sequence of behaviors characteristic of bulimia nervosa (Stice, 1994, 1998). In this way, a binge drinking episode may inadvertently initiate the binge-purge cycle wherein individuals eat more than they usually would, experience acute shame over caloric intake, and restrict to compensate for this period of consumption. Over time, heavy alcohol use may alter body shape and weight due to increased caloric content both through overeating while drunk and the caloric content of alcohol itself. In line with the sociocultural model of ED, a girl or woman who moves farther away from the idealized

female form (i.e., the thin ideal) is more likely to experience increased body dissatisfaction and adopt extreme weight-loss behaviors, which may include use of diet pills, induced vomiting, and misuse of laxatives (e.g., Stice, 1994).

A growing body of research has begun to describe and understand the phenomena of "drunkorexia." This practice is characterized as the purposeful manipulation of dietary intake prior to a drinking episode either to enhance effects of alcohol or to compensate for caloric intake from alcohol (e.g., Barry & Piazza-Gardner, 2017; Eisenberg & Fitz, 2014). Fasting prior to substance use quickens and exacerbates its physiological effects (Puhl et al., 2011; Shumsky, Shultz, Tonkiss, & Galler, 1997; Wellman, Nation, & Davis, 2007). Indeed, animal models have found that food deprivation, a state achieved through pathological dieting in humans, is associated with subsequent binges and increased drug-seeking and drug-taking behaviors (Puhl et al., 2011; Shumsky et al., 1997; Wellman et al., 2007).

Though fasting prior to binge drinking to enhance substantive effects is distinct from ED attitudes (Roosen & Mills, 2015), the practice of restricting food intake prior to drinking episodes may evolve into a consistent pattern of DE engagement. Restriction prior to drinking may lead to increased consumption of food during or after the drinking event, and those who restrict prior to drinking for substantive effects may engage in heavier drinking, thereby increasing caloric consumption. This possibility is supported by research showing that the association between weight control motives for "drunkorexia" was strongest among women with heavy (compared to light) drinking behavior (Eisenberg & Fitz, 2014). Taken together, these findings suggest that girls who initiate

heavy drinking episodes may grow discontent with changes in body shape and weight. Over time, girls who initially engage in drunkorexic practices motivated by a desire to enhance alcohol's effects may begin engaging in such behaviors for the sake of weight control, contributing to DE.

Marijuana Use

The physiological properties of marijuana and its metabolic effects due to endocannabinoids (Sansone & Sansone, 2014) also may promote an association between DE behaviors and marijuana use. Endocannabinoids are critical mediators for metabolic processes and comprise two receptor types, cannabinoid-1 receptors (CB1) and cannabinoid-2 receptors (CB2) (Akbas, Gasteyger, Sjödin, Astrup, & Larsen, 2009; Kirkham, 2008). CB1 receptors are found in the hypothalamus, stomach, and intestinal tissues (Sansone & Sansone, 2014). As such, agonism of these receptors is thought to stimulate appetite, enjoyment of food, and promote the deposition of food into fat (Sansone & Sansone, 2014). The acute effects of marijuana increase appetite, leading many to experience the "munchies" and engage in increased food intake. Thus, it is possible that marijuana use leads to acute overeating and subsequent remorse or guilt following this event. Similar to alcohol, acute effects of marijuana may predispose individuals to misuse food. Overindulgence while high may lead to next day restriction to compensate for caloric intake. Individuals may seek more extreme compensatory behaviors over time, having associated increased appetite with marijuana use.

At the same time, some research shows that chronic marijuana use has the opposite effects on appetite due to down-regulation of CB1 receptors (Sansone &

Sansone, 2014). Epidemiological studies have consistently found long-term marijuana users to have lower rates of obesity and body mass relative to those with less extensive use and non-users (Hayatbakhsh et al., 2010; Le Strat & Le Foll, 2011; Meier et al., 2016). Therefore, the relationship between DE and marijuana use may change over time. More longitudinal research is needed to clarify acute and chronic effects.

Despite marijuana's acute and chronic effects on appetite and shared risk factors with DE, research on these co-occurring syndromes is limited to cross-sectional studies. Findings from the Youth Risk Behavior Survey found that 60.1% of adolescents between 12- and 18-years old who reported SU also engaged in at least one DE behavior (Vidot et al., 2016). Moreover, adolescents who reported current marijuana use had significantly higher odds (adjusted odds ration = 2.2) of DE behaviors compared to non-using peers (Vidot et al., 2016). Negative self-esteem ($r = 0.11$, $p < .001$) and body image ($r = 0.10$, $p < 0.01$) specifically were found to be associated with marijuana use in a nationwide sample of adolescent girls (Schwinn, Schinke, Hopkins, & Thom, 2016). To date, there are no longitudinal studies that have investigated prospective relations between DE and marijuana use. Such research investigating unique temporal effects of marijuana use on DE (and vice versa) is needed.

Conceptual and Methodological Limitations of Casual Models in Previous Studies

The overwhelming majority of past research has assumed a unidirectional relationship between DE and SU with most examining elevations in DE as a predictor of systematic increases in SU. In nearly all studies, univariate logistic regression analyses have been used to predict onset of one disorder from symptoms or diagnosis of the other

(Franko et al., 2005, 2008; Jacobi et al., 2011; Johnson, Cohen, Kotler, Kasen, & Brook, 2002; Strober, Freeman, Bower, & Rigali, 1996; Zaider, Johnson, & Cockell, 2002). One study, for instance, found that 10% of young women seeking treatment for an ED reported onset of an alcohol use disorder (Franko et al., 2005), and 8.5% reported onset of a drug use disorder 9 years later (Franko et al., 2008). Another study reported that 18.9% of adolescent girls seeking treatment for an ED developed an SU diagnosis (11.6% abuse, 7.4% dependence) 10 years later (Strober et al., 1996). More recent research has considered the development of an ED diagnosis following early SU with largely null findings (Johnson et al., 2002; Zaider et al., 2002). More longitudinal investigations are needed to clarify potential causal relations between SU and DE and the direction of effects. Studies with population-based samples are particularly needed given the nearly exclusive focus on clinical samples in prior work.

In addition to focusing on clinical samples, prior studies have failed to address the possibility that SU and DE have reciprocal, causal relations (Baker et al., 2013). Previous data analytic approaches do not allow for estimates of simultaneous change. Thus, the simultaneous development of SU and DE and their potential bidirectional effects remain unexamined. Recent advances in longitudinal growth modeling allow for such estimates to be examined. Given inconsistent findings in the literature regarding the direction of effects and advances in quantitative approaches, novel investigations of the simultaneous development of these syndromes should be conducted.

Disaggregating Between- and Within-Person Effects

In addition to the lack of studies on potential reciprocal relations between DE and SU, prior research has been limited by a focus on between-person effects. Prior studies, therefore, tell us that the "kind of people" who use substances also engage in certain patterns of DE, or that the "kind of people" who increase their SU simultaneously increase their DE. What they do not tell us is whether increases in SU are associated with increases in DE within the same individual. Studies of within-person effects are necessary to draw such conclusions, and these approaches have the added benefit of allowing for identification of potential developmentally specific effects at the individual level. That is to say, SU may be more strongly related to increases in DE within the same person at particular points in development. As an example of the importance of studying within-person effects, qualitative findings suggest that a small number of women may develop an increased drive for thinness and desire for weight control after receiving compliments on weight loss due to tobacco use (Sirles, 2009). Recent literature on weight-motivated drinking behaviors (e.g., "drunkorexia") also provide reason to believe that periods of heavy drinking may contribute to periods of greater DE behaviors at the within-subjects level. Thus, it is critical to disaggregate group- and individual-level effects to further our understanding of the interplay between these two syndromes.

Optimal Modeling of Change over Time

Myriad approaches have been used to model change in a given variable over time. Repeated-measures ANOVA, which models change in a single variable over time, has long been a popular approach. However, this approach is limited in that it cannot assess

how individuals differ in change or how multiple constructs relate over time. Growth models, such as latent curve models (LCM), allow for more nuanced modeling of change between and within individuals over time. A bivariate growth model captures change in two constructs over time and estimates simultaneous growth, though it only allows for between-subjects analyses and inferences (Grimm, Ram, & Estabrook, 2016). Other growth modeling approaches, such as the time-varying covariate model, allow for the inclusion of individual-level variables that vary over time (i.e., DE and SU) (Grimm et al., 2016). Yet, they do not allow for examination of differences between groups. Moreover, these models cannot estimate how two variables like SU and DE may influence one another in a reciprocal manner over time. An approach that allows for the modeling of multiple growth processes over time and has the ability to predict growth from both individual-level, time-varying variables (like SU and DE) *and* group-level variables is needed to answer questions regarding the potential bidirectional nature of relations across important developmental transitions.

The current project seeks to extend the literature through its application of latent curve models with structured residuals (LCM-SR; Curran et al., 2014) in a preexisting sample of adolescent girls followed into adulthood. Unlike other data analytic approaches, LCM-SR conceptualizes residuals of each construct as time-specific estimates of deviations of observed measures from an underlying trajectory, therein allowing for modeling of within-person effects. The structure imposed on each set of residuals can be used to capture prospective and bidirectional associations. For example, the deviation of the observed DE score at baseline from an individual's underlying DE

trajectory can be used to predict the deviation of the observed DE from the underlying DE trajectory at time 1 (autoregression) as well as the same person's deviation of the observed SU score from her underlying SU trajectory at time 2. In essence, subtle fluctuations in symptoms within an individual can be modeled in meaningful ways and provide greater insight into the coupling of SU and DE. The unconditional LCM-SR also can be expanded to include important shared risk factors as predictors of change, allowing for inferences about group differences. Further, these models allow for the investigation of potential time-specific effects. Collectively, these analyses aimed to facilitate the development of more precise developmental theories regarding SU/DE comorbidity.

Given the paucity of SU prevention programs targeting young girls, it is important to understand unique factors that lead to increased use in adolescence and early adulthood. Findings related to each hypothesis will have implications for the development and adaptation of universal, selective, and indicated prevention programs for SU and DE. Disaggregating between- and within-level effects may inform the timing and targets of prevention efforts. If results suggest that DE precedes SU, then selective programs targeting known risk factors for DE (e.g., idealizing a thin body shape, body dissatisfaction) in addition to universal targets of prevention should naturally delay onset of SU. Moreover, results may inform indicated prevention programs by targeting expectancies associated with SU (e.g., appetite suppressant effects of tobacco or coping strategies for negative affect) in young girls who engage in early dieting. If results suggest that the co-occurrence is largely accounted for by common factors, like negative

affect and poor self-regulation, then universal prevention programs targeting these shared risk factors should be effective at reducing incidence and delaying onset of both DE and SU. In addition, findings might indicate that selective prevention programs are needed for early maturing girls who are at elevated risk for DE and SU.

The Current Study

Despite research suggesting that SU and DE are highly comorbid, have similar trajectories in women (e.g., Chassin, Presson, Sherman, & Edwards, 1990; Copeland, Rooke, & Swift, 2013; Hingson, Zha, & Weitzman, 2009; Slane, Klump, McGue, & Iacono, 2014; Stice, Marti, & Rohde, 2013; Van Son, Van Hoeken, Bartelds, Van Furth, & Hoek, 2006), and share important risk factors, only one study to date has investigated their simultaneous trajectories in adolescence (Measelle et al., 2006). Measelle and colleagues estimated longitudinal growth models among adolescent girls between the ages of 13 and 18. Results of multivariate analyses revealed that initial elevations in depressive symptoms predicted increases in eating pathology and substance abuse. Moreover, initial elevations in eating pathology predicted future increases in substance abuse symptoms. More research is needed to replicate these finding and investigate within- as well as between-person effects in order to understand nuances in the course and covariation of these syndromes. It is also important to determine if longitudinal relations between DE and SU are similar or different across different substances of abuse, and adolescent co-occurrence of these symptom domains may be better investigated across development using behavioral indices (i.e., quantity and frequency of use) rather than diagnostic symptoms (i.e., problems relating to substance abuse). Finally, it is

important to determine if within-subject relations between DE and SU are stable or change systematically across development. Answers to these questions may ultimately inform effective prevention and treatment approaches.

The current study aims to address these gaps by investigating the co-occurrence and covariation of tobacco, alcohol, and marijuana use and DE behaviors from adolescence to adulthood. First, the simultaneous development of DE and each substance will be examined during the transition to adulthood at the between-person level. Second, time-specific effects will be examined at the within-person level as a function of time. Collectively, these models will allow for the identification of between- and within-person effects across age and time, which may strengthen as individuals progress through adolescence. Third, the study will examine the strength of the prospective relations at both the between- and within-person levels, controlling for shared developmental risk factors including early sexual maturation and internalizing and externalizing symptoms. The analyses will test the following hypotheses **(Table 1)**:

1. **Tobacco.** At the between-person level, initial levels of tobacco use are expected to be positively correlated with DE symptoms. Rate of change (slope) in tobacco use is expected to be positively correlated with rate of change in DE at the between-person level, suggesting simultaneous growth over time. Given previous literature on DE and tobacco use, initial DE symptoms are expected to covary with change (slope) in tobacco use when examining between- and within-person level effects. Moreover, fluctuations in individual's DE scores are expected to prospectively predict fluctuations in tobacco use over and above an individual's

underlying trajectory. This predictive relationship is expected to persist even when shared risk factors are accounted for, due to the unique anorectic properties associated with tobacco use.

2. **Alcohol.** At the between-level, the initial level (intercept) and rate of change (slope) in alcohol use are expected to be positively correlated with the initial level (intercept) and rate of change (slope) in DE. Initial DE symptoms (intercept) are expected to covary with change (slope) in alcohol use, and initial levels (intercept) of alcohol use are expected to covary change (slope) in DE. Once shared risk factors are accounted for at the between-level, only change in alcohol use is expected to be associated with initial levels and change in DE, given its caloric content and the associated weight changes over time. At the within-person level, reciprocal relations are expected to emerge in light of literature on weight changes due to heavy episodic drinking as well as "drunkorexia" and self-medication models of substance use. It is further hypothesized that these relations will remain though weaken in magnitude when controlling for shared risk factors.
3. **Marijuana.** Initial levels (intercept) of DE are expected to be positively related to initial levels (intercept) and rates of change (slope) in marijuana use across adolescence, in line with the directional effects reported in the literature. Evidence of reciprocal relations are expected to emerge at the within-person level. These proposed between- and within-level effects are expected to remain, though weaken, once accounting for shared risk factors.

METHOD

Procedure

The proposed project used data from the National Longitudinal Study of Adolescent to Adult Health (Add Health; Harris, 2009). Add Health is a longitudinal study of a nationally-representative sample of adolescents between 7th and 12th grade. High schools were selected based on size, school type, census region, level of urbanization, and percent White students. Of the 80 selected high schools, 52 agreed, and 28 replacement schools were reselected to match the initial school sample. Participating high schools and their respective feeder schools provided researchers with a roster of all enrolled students for recruitment into the at-home survey. The core sample oversampled for various racial groups (i.e., African Americans from well-educated families as well as those who identify as Chinese, Cuban, and Puerto Rican), students who reported physical disabilities, and pairs of siblings. School rosters allowed for recruitment of the core in-school sample of 12,105 adolescents. In the full in-home interview sample, 88.6% of the participants from Wave I completed the interview again at Wave II. More than three-fourths (77.6%) of Wave I participants completed the Wave III interview.

Public-use data from Waves I through III was used in the proposed study. The public-use data includes only those who participated in the in-home interview. The core in-home interview sample at Wave I consisted of 6,504 respondents. The Add Health sample made publicly available is one-third of the full Add Health sample. It consists of one-half of the core sample and one-half of the oversample of African American adolescents with a parent who had a college degree, chosen at random. Wave II excluded

participants who were in the 12th grade at Wave I and those not in the genetic sample of sibling pairs as well as those who were only in the disability sample at Wave I. Wave II added an additional 65 adolescents who were in the sample of sibling pairs at Wave I but had not been interviewed. A total of 4,834 adolescents who participated at Wave I were re-interviewed at Wave II. Wave III consisted of 4,882 respondents from the core sample at Wave I. Unfortunately, information regarding the percentage of Wave I participants from the public-use data who were eligible to complete Wave II is unavailable. Given the sampling procedures, it was possible to estimate retention from Wave I to Wave III; 78% of the female participants from Wave I completed the Wave III interview. In sum, the sample consisted of adolescents between 7th and 12th grades at Wave I, 8th through 12th grades at Wave II, and young adults 18- to 27- years old at Wave III.

Participants

Only data collected during in-home interviews from female participants were utilized due to the higher prevalence of DE among girls and worse treatment outcomes among women with comorbid ED and substance use disorders (Hudson, Hiripi, Pope, Kessler, & Kessler, 2007; Swanson, Crow, Le Grange, Swendsen, & Merikangas, 2011; Woodside et al., 2001). The resulting sample consists of 3,356 adolescent girls at Wave I, 2,519 at Wave II, and 2,629 at Wave III. Overall, the resulting sample for this study was diverse in race and age. The majority of the sample identified as Non-Hispanic (88.5%) and White (65.9%). A quarter (25%) of the sample identified as African American. The remaining participants identified as Asian/Pacific Islander (3.9%) or American Indian/Native American (4%) with an additional 6.8% identifying as "other." Participants

were allowed to identify as belonging to more than one race so percentages total greater than one-hundred percent. At the first measurement occasion, the sample ranged from 12- to 21-years of age with the majority of participants falling between the ages of 15- and 18-years old (M_{age} = 15.86, standard deviation (SD) = 1.77).

Measures

Primary outcome measures. Descriptive statistics for all variables are shown in Table 3. Table 4 displays the correlations among all individual items of primary predictors as well as summary scores of covariates (described below).

Disordered eating. Four items were used to assess DE behaviors (dieting, use of diet pills, self-induced vomiting, and misuse of laxatives) over the last 7 days at each wave. Each item was assessed on a binary scale. A latent factor was created using two-parameter longitudinal item response theory models. Scaling of the latent factor was transformed by multiple of 10 in order to overcome convergence issues in modeling. Longitudinal measurement invariance in the discrimination and difficulty parameters was assessed to ensure that the construct retained the same meaning over time and that each of the four DE behaviors contributed meaningfully to the latent factor. (Results reported below). Partial scalar invariance was achieved for the latent factor. Factor loadings from the partial scalar invariance model were significant for all items across each wave and ranged from 0.50 to 0.70.

Tobacco use. A summed composite was created using the number of smoking days in the last month and the number of cigarettes smoked each smoking day. Number of cigarettes smoked per day underwent a Winsor transformation such that all responses

of 30 or more cigarettes per day (less than 0.5% of responses) were recoded as 30. Standardized scores were used to create a sum of use at each wave because a latent variable model would be unidentified with two items. Correlations among items ranged from 0.44 to 0.80 across waves ($p < 0.01$). Internal consistency for these items was adequate with a Cronbach's alpha of 0.74 at Wave I, 0.75 at Wave II, and 0.79 at Wave III.

Alcohol use. Alcohol use was captured by a latent factor of three items assessing frequency and quantity of drinking as well as the frequency of feeling drunk over the past 12 months. A factor analysis examined longitudinal measurement invariance to ensure that the construct retains the same meaning over time (results reported below). Quantity per drinking occasion at Waves I and II underwent a Winsor transformation such that all responses of 18 or more drinks were recoded as 18 to be consistent with responses at Wave III. Correlations among these items ranged from 0.14 to 0.78 ($p < 0.01$) across all waves. Internal consistency reliability was adequate given that there were only three items, with a Cronbach's alpha of 0.68 at Wave I, 0.67 at Wave II, and 0.70 at Wave III. Partial scalar invariance was achieved for the latent factor. Factor loadings from the partial scalar invariance model were significant for all items across each wave and ranged from 0.61 to 0.92.

Marijuana use. A single open-ended item assessed how many times participants used marijuana over the last 30 days. The item underwent a Winsor transformation such that all responses at or above 30 (less than 0.5% of responses) were recoded as equal to 30.

Covariates. The following measures were chosen based on previous literature identifying sexual maturation, internalizing, and externalizing symptoms as related to the onset and course of SU and DE in adolescent girls. Each measure was included in the models in order to elucidate unique relations between SU and DE.

Sexual maturation. Self-reported menarcheal age was used as an index of sexual maturation. This item was asked at Waves I through III. To reduce missing data, responses were pulled at each wave into a collapsed variable. Responses from the earliest wave available were taken for each participant. Ninety percent of the responses came from Wave I, 8.8% from Wave II, and 0.01% at Wave III. Age of menarche is significantly correlated with the onset of pubertal maturation (Marshall & Tanner, 1969; Taranger, Engström, Lichtenstein, & Svennberg□Redegren, 1976) and has been shown to be an accurate indicator (Graber et al., 1997) with high test-retest reliability (.67 to .79) up to 30 years later (Casey et al., 1991; Must et al., 2002). It has been widely used as a marker of pubertal maturation in previous research (Striegel-Moore et al., 2001).

Internalizing symptoms. Internalizing symptoms were composed of depression and anxiety symptoms. Nine items from the Center of Epidemiologic Studies Depression Scale Revised (CESD-R-10; Björgvinsson, Kertz, Bigda-Peyton, McCoy, & Aderka, 2013; Miller, Anton, & Townson, 2008; Radloff, 1977) were used to assess depressive symptoms at Waves I, II, and III. The CESD has been well-validated and widely used to capture depressive symptomatology in community samples of adolescents and adults, with high internal consistency ($\alpha = 0.72$; Björgvinsson et al., 2013). In line with the original measure, these items were summed to create a total score of depressive symptom

severity. Loss of appetite was not included from the original measure due to the theoretical association between changes in appetite and disordered eating. A confirmatory factor analysis was used to test the tau equivalence assumption that each item contributes an equal amount to the factor. Results of the CFA suggested that items varied in loadings with a single factor. McDonald's omega reliability coefficient was generated and suggested adequate internal consistency at each wave (ω_{WI} = .79; ω_{WII} = .78, ω_{WIII} = .82).

In addition, five anxiety symptoms were assessed. A count of symptoms at Wave I and Wave II was used and consisted of fearfulness, moodiness, frequent crying, difficulty relaxing, and difficulty falling or staying asleep. Careful selection of items was undertaken in the analytic approach to ensure that items used were distinct from items used to capture depression. A confirmatory factor analysis was used to test the Tau equivalence assumption that each item contributes an equal amount to the factor. Results of the CFA suggested that items varied in loadings with a single factor. As such, McDonald's omega reliability coefficient was generated at each wave (ω_{W1} = .62; ω_{W2} = .54). These items have been used in previous research as an index of anxiety with adequate internal consistency reliability (α = 0.72; Sullins, 2016).

***Externalizing symptoms*.** A composite of externalizing symptoms was created from delinquency and hyperactivity/impulsivity items. Seven delinquency items at Wave I and II captured deviant behaviors, including lying to parents, acting rowdy in public, running away from home, using a car without permission, stealing <$50, stealing >$50, shoplifting, damaging property, vandalizing, and burglarizing. Only four of these original

seven items were available at Wave III. For the sake of consistency across waves, only these items were used to create a count of delinquent behaviors at Wave III. All items were coded as dichotomous and summed to create a count variable at each wave (Wave I $\alpha = .71$; Wave II $\alpha = .69$; Wave III $\alpha = .59$).

Eight items representing hyperactivity and impulsivity at Wave III retrospectively assessed impulsivity between the ages of 5 and 12. Each question had the preface, "Which answer best describes your behavior when you were that age? When you were between 5 and 12…" The response choices included never or rarely, sometimes, often, and very often. Items included: 1. You blurted out answers before the questions had been completed; 2. You felt "on the go" or "driven by a motor;" 3. You felt restless; 4. You fidgeted with your hands or feet or squirmed in your seat; 5. You had difficulty awaiting your turn; 6. You had difficulty doing fun things quietly; 7. You talked too much; and 8. You left your seat in the classroom or in other situations when being seated was expected. As items had high internal consistency ($\alpha = .80$), a mean was used to capture hyperactivity/impulsivity.

Data Analytic Plan

All analyses were carried out in M*plus* 8 v.1.6 (Muthén & Muthén, 2012-2018) using full maximum likelihood estimation, which applies robust full information methods to handle all missingness. Prior to conducting the main analyses, distributions of all variables were examined for non-normality and outliers. Appropriate transformation techniques were used for non-normally distributed variables. Measurement invariance testing procedures also were undertaken to ensure that both latent factors for DE and

alcohol use were appropriate for use in longitudinal analyses. Though the Add Health dataset consists of three levels (school, participant, and time), school information is not available in the public-use data. As such, all structural equation models consisted of only two levels (i.e., participant and time).

As the sample was heterogeneous in age at each measurement occasion, data were organized by measurement occasion but were age centered at the youngest age (i.e., 12-years old) available at Wave 1. The TSCORES command in conjunction with TYPE = RANDOM was used in M*plus* to account for individually-varying times of observation for the outcomes and to estimate growth models with a random slope (Muthén & Muthén, 2012-2018, p. 131). However, use of this approach limits model fit statistics available. Non-traditional fit statistics, Akaike Information Criteria (AIC) and Bayesian Information Criteria (BIC), are used to describe model fit.

Aim 1 Analyses. In order to accurately disaggregate between group- and individual-level effects, a model building approach using likelihood ratio tests first outlined by Curran et al. (2014) was taken. Models using centered age at each measurement occasion allowed for the optimal modeling of developmental effects. As a first step, univariate models that optimally fit growth in each construct were established and used to identify optimal change, testing of autoregressions among residuals, and testing of equality constraints of auto-regressions.

Second, bivariate models estimated simultaneous change in DE and use of specific substances across adolescence (conceptual model shown in **Figure 1**). Covariation between intercepts and slopes within and across constructs were examined to

assess group-level associations in symptomatology at age 12 as well as change across adolescence. Univariate and bivariate models allowed for intercept and slope parameters to vary randomly across person (Curran & Bauer, 2011). Residuals were conceptualized as deviations from individuals' expected scores while accounting for the underlying trajectory in each construct, therein capturing within-person processes. In order to accomplish this goal, the LCM-SR imposes a structure among the residuals rather than estimating unstructured correlations, as is done in a standard LCM. The LCM-SR regresses the residual at a given time point on the point of the residual of a previous time point across two constructs. Residual variances at Wave I are, therefore, freely estimated since they are not a function of other predictors. Residuals at Waves II and III are equated over time to account for the fact that these estimates are conditioned on a prior estimate, Wave I. In this way, the LCM-SR covariance matrix is dependent on the underlying latent curve factors as well as earlier residuals within- and across-construct (Curran et al., 2014, pg. 13).

Aim 2 Analyses. Building upon the bivariate models in Aim 1, models in Aim 2 added cross-lagged, reciprocal paths. Cross-lagged paths among residuals were added to estimate prospective relations between DE and each type of substance use at the within-person level. Model building first constrained one direction of the reciprocal effects to be zero (e.g., DE on tobacco use) while freely estimating the opposite direct of effects (e.g., tobacco use on DE). This model allowed the directional effect of interest to differ across time lags. This model was then compared to a model that constrained the lagged paths to be equal across time using a likelihood ratio test. The reverse direction of effects was

tested in the same way in order to unambiguously examine each direction of effect (e.g., examining the effects of DE on tobacco use while holding the effect of tobacco use on DE at zero). Finally, a model estimated bidirectional effects simultaneously. Between-person effects were only examined from the final, best-fitting model that included simultaneous estimation of bidirectional within-person effects (see **Figure 2**).

Aim 3 Analyses. Bivariate models outlined in Aims 1 and 2 were then expanded to include time-varying (i.e., internalizing and externalizing symptoms) and time-invariant (i.e., ethnicity, race, and sexual maturation) predictors of intercepts and slopes of SU and DE as well as residual scores within each time-point. The time-invariant covariates were regressed on latent growth factors for each construct, whereas time-varying covariates were regressed on time-specific, observed score residuals (see **Figure 3**). As such, this aim tested whether the strength of the co-occurring relations diminished once shared risk factors were considered.

RESULTS

Measurement Invariance across Time

Disordered Eating:

The extent to which an item factor model measuring disordered eating (with four observed items) exhibited measurement invariance and structural invariance across measurement occasions was examined. Two-parameter logistic item response models were used to ensure not only invariance across time but also that each of the four DE items contributed meaningfully to an item factor model, given low endorsement rates of purging behaviors **(Table 3)**. Weighted least squares maximum likelihood (WLSMV) estimation and theta parameterization were used to estimate all models. Nested model comparisons were conducted using the DIFFTEST procedure.

A configural invariance model was initially specified in which a single factor was estimated at each time-point. The factor variance was fixed to 1, and the factor mean was fixed to 0 across time such that all item factor loadings (one per item) and thresholds were freely estimated. The residual variances were not uniquely identified in the configural model and were constrained to 1. As shown in **Table 5**, the configural invariance model had good fit (χ^2 (51) = 62.76, $p > 0.05$). The analysis proceeded by applying parameter constraints in successive models to examine potential decreases in fit resulting from measurement or structural invariance across time.

Equality of the unstandardized item factor loadings across time was examined to assess metric invariance. The factor variance was fixed to 1 at Wave I and freely estimated at Waves II and III. The factor mean at each wave was fixed to 0 for

identification. As shown in **Table 5**, the metric invariance model had good raw fit (χ^2 (57) = 66.45, $p > 0.05$). Results of a DIFFTEST analysis showed that the metric invariance model did not fit significantly worse than the configural invariance model, $\Delta\chi^2(6) = 7.46$, $p = 0.28$. That is to say, each disordered eating item was related to the latent factor equivalently across time. Thus, the structure of the latent factor was roughly equivalent at each time point.

Equality of the unstandardized item thresholds across time was then examined to assess scalar invariance. The factor mean was fixed at 0 with factor variance constrained at 1 at the first time point and freely estimated at Waves II and III. All item factor loadings and item thresholds were constrained to be equal across waves for corresponding items. The full scalar invariance model for disordered eating fit significantly worse than the metric invariance model, $\Delta\chi^2$ (6) = 45.89, $p < 0.01$.

Modification indices suggested that the threshold for use of diet pills at Wave III, relative to Waves I and II, was the largest source of misfit and should be freed at this measurement occasion. After freeing the threshold for use of diet pills at Wave III, the partial scalar invariance model did not fit significantly worse than the metric invariance model, $\Delta\chi^2$ (5) = 0.58, $p = 0.99$. The fact that partial scalar invariance, or "strong invariance," held indicates that dieting, self-induced vomiting, and laxative misuse have the same expected response at the same absolute threshold across measurement occasions. That is to say, the observed differences in the proportion of responses in each disordered eating behavior was due to factor mean differences only and not attributable to a change in the meaning of the construct over time. In contrast, the threshold for use of

diet pills was lower at Wave III than at previous waves, indicating that young adult women between 18- and 27-years old had a greater probability of using diet pills than at previous time points. This may be due policies that prohibit the sale of over-the-counter diet pills to anyone under 18-years old.

In conclusion, these analyses demonstrated partial scalar measurement invariance across waves. The relations of the items to the latent factor were equivalent across measurement occasion. That is, the inclusion of purging behaviors captured meaningful information despite low prevalence, and the latent factor for DE was not primarily driven by dieting behaviors relative to the other DE behaviors. However, use of diet pills was significantly less difficult for young women between 18- and 27-years old compared to earlier age bands. These results are in accordance with van de Schoot, Lugtig, and Hox (2012), which supports using a factor with at least two indicator means constrained to be equal over time to accurately compare factor means across time. As such, model parameters and estimates from the final model with partial scalar invariance were saved and used in all analyses presented below.

Alcohol Use:

Similar to disordered eating, the extent to which an item factor model measuring alcohol use (with three observed items) exhibited measurement invariance across measurement occasions was examined. Since all alcohol items were continuous, maximum likelihood estimation with robust standard errors (MLR) was used in all models. Given the large sample size and significant chi-square index for the configural invariance model, it was determined that the chi-square was a less reliable statistical test

of model fit (Cheung & Rensvold, 2002). Therefore, nested model comparisons were conducted comparing RMSEA, CFI, and SRMR estimates for each model rather than the traditional chi-square test. Recommendations by Cheung and Rensvold (2002) propose that differences between nested models that exceed 0.015 and 0.010 for RMSEA and CFI serve as indicators of worse fit across all three levels of testing. Cheung and Rensvold (2002) further suggested different values of SRMR be used for different levels of testing. More specifically, a change of 0.030 should be used for testing loading invariance and 0.010 be used when testing intercept and residual invariance given SRMR's differential sensitivity across invariance tests.

A configural invariance model was initially specified in which a single factor was estimated simultaneously at each wave. The factor variance was fixed to 1, and the factor mean was fixed to 0 across time such that all item factor loadings (one per item) and means were estimated. The configural invariance model had good fit (RMSEA = .017, CFI =.997, SRMR = .013) when judged against the cut-offs suggested by Hu and Bentler (1999) (**Table 6**). The analysis proceeded by applying parameter constraints in successive models to examine potential decreases in fit resulting from constraints across time.

Metric invariance was assessed through the equality of the unstandardized item factor loadings across time. The factor variance was fixed to 1 at Wave I and freely estimated at Waves II and III. The factor mean at all waves was fixed to 0 for identification. All factor loadings were constrained to be equal across time with item means estimated. As shown in **Table 6**, the metric invariance model also had good fit (RMSEA = .044, CFI = .977, SRMR = .045). Comparing CFI, RMSEA, and SRMR

showed that the metric invariance model fit meaningfully worse than the configural invariance model using benchmarks from Cheung and Rensvold (2002). Modification indices suggested that the mean for quantity of alcohol use at Wave III was the largest source of misfit and should be freed at this measurement occasion. This partial metric invariance model did not fit meaningfully worse than the configural model based on differences in fit indices (RMSEA = 0.016, CFI = 0.997, SRMR = 0.014).

Scalar invariance was assessed by building upon the partially invariant metric model via constraints to the item means across waves. The factor mean was fixed at 0 with factor variance constrained at 1 at the first wave and freely estimated at Waves II and III. All item factor loadings and item means were constrained to be equal across waves. The full scalar invariance model for alcohol use fit meaningfully worse than the partial metric invariance model (RMSEA = 0.040; CFI= 0.979; SRMR = 0.036). Modification indices suggested that the mean for frequency of alcohol use at Wave III was the largest source of misfit and should be freed at this measurement occasion. After freeing the mean for frequency of alcohol use at Wave III, the partial scalar invariance model did not fit the model meaningfully worse than the partial metric invariance model (RMSEA = 0.017, CFI = .996, SRMR = 0.015). Since partial scalar invariance, or "strong invariance" held, results indicate that quantity of alcohol use on drinking days and frequency of feeling intoxicated within the last 30 days had comparable expected responses across waves. That is to say, the observed differences in these alcohol use items were due to factor mean differences only and not changes in the meaning of the items. In contrast, frequency of alcohol use was higher at Wave III relative to previous

waves, indicating that young adult women between 18- and 27-years of age experienced more drinking days than at previous time points. This may be due the transition to college and/or independent living, having greater access to drinking environments (e.g., house parties, using fake IDs, attending events that do not check IDs), and reaching the legal drinking age.

These analyses showed that partial metric and partial scalar measurement invariance was obtained across waves. The relations of the items to the latent factor, alcohol use, were equivalent across measurement occasion, whereas frequency of drinking within the last 30 days was more prevalent for young women between 18- and 27-years old compared to earlier assessments. Nevertheless, these results are in accordance with recommendations by van de Schoot and colleagues (2012) for factor comparisons across time. Factor scores from the final model that demonstrated partial scalar invariance were saved and used in all analyses presented below.

Unconditional Univariate Models

Disordered Eating

As a first step, a random intercept model was estimated for DE. The model included only a mean and variance of the intercept factor and residual variances for each of the repeated measures that were allowed to vary over time (AIC = 47059.56, BIC = 47090.18). This model was extended to include a slope factor. Means and variances were estimated for the intercept and slope parameters along with the covariation between intercept and slope. Time-specific residual variances varied over time. The model terminated normally (AIC = 42184.96, BIC = 42233.91). A likelihood ratio test indicated

significant improvement in model fit relative to the intercept only model ($\Delta\chi^2$ (3) = 4880.62, $p < 0.001$). Mean and variance components were significant for both the intercept (M = -1.35, z = -7.85, $p < 0.001$; ψ = 35.29, z = 30.59, $p < 0.001$) and slope (M = 0.56, z = 63.92, $p < 0.001$; ψ = 0.06, z = 7.92, $p < 0.001$). Intercept and slope parameters significantly covaried (ψ = -0.76, z = -15.73, $p < 0.001$), indicating that those with greater DE pathology at age 12 exhibited less of an increase in DE over time. This model was further expanded to include an autoregressive structure among the residuals. The model significantly improved fit (AIC = 41988.27, BIC = 42043.34; $\Delta\chi^2$ (1) = 198.68, $p < 0.001$. Thus, the autoregressive residual structure was retained.

Tobacco Use

An intercept-only univariate model for tobacco use was first estimated (AIC = 67029.85, BIC = 67060.44). It was then expanded to include a slope parameter. Means and variances were estimated for intercept and slope parameters as well as covariation between the latent factors. Time-specific residuals were allowed to vary over time. The model reflected improved fit (AIC = 66695.59, BIC = 66756.77, $\Delta\chi^2$ (3) = 16.4, $p <$ 0.001). Examination of the intercept showed significant mean (M = 3.55, z = 5.70, $p <$ 0.001) and variance (ψ = 189.98, z = 21.75, $p < 0.001$) components. The same was true for the slope parameter (M = 0.78, z = 8.93, $p < 0.001$; ψ = 5.42, z = 15.31, $p < 0.001$). Significant covariation between intercept and slope (ψ = -17.80, z = -13.79, $p < 0.001$) suggested that those with higher levels of tobacco use at age 12 demonstrated the least growth in use over time. When this model was expanded to include an autoregressive component among the residuals, a formal test showed an improvement in model fit ($\Delta\chi^2$

(1) = 327.88, $p < 0.001$). Thus, the model with autoregressive paths among residuals was retained.

Alcohol Use

An intercept-only univariate model was initially fit to the observed alcohol scores (AIC = 22985.12, BIC = 23015.72). This model was expanded to include a slope parameter, while estimating means and variances for intercept and slope parameters as well as their covariation. Time-specific residuals were allowed to vary over time. This model improved fit when compared to the intercept only model (AIC = 22508.85, BIC = 22557.80, $\chi^2_\Delta(3) = 486.26$, $p < 0.001$). Examination of the intercept showed significant mean ($M = -0.21$, z = -5.80, $p < 0.001$) and variance ($\psi = 0.79$, z = 16.68, $p < 0.001$) components. Similar to that of DE, the slope parameter showed small but significant mean ($M = 0.07$, z = 15.41, $p < 0.001$) and variance ($\psi = 0.001$, z = 0.89, $p < 0.001$) components. Significant covariation between intercept and slope ($\psi = -0.03$, z = -4.022, $p < 0.001$) indicated that those who engaged in greater alcohol use at age 12 exhibited smaller increases in use over adolescence. This model was expanded to include the autoregressive structure among the residuals. A formal test showed this structure improved model fit ($\Delta\chi^2(1) = 269.10$, $p < 0.001$) and was, therefore, retained.

Marijuana Use

Lastly, univariate unconditional models for marijuana use were estimated. An intercept-only model converged normally (AIC = 48611.76, BIC= 48642.34) and was expanded to include a random slope parameter while estimating means and variances for both intercept and slope as well as the covariance between these parameters. Time-

specific residuals were allowed to vary over time. This model significantly improved fit, (AIC = 48438.89, BIC = 48487.83, $\chi^2_\Delta(3) = 178.86$, $p < 0.001$). Results indicated significant mean and variance components for the intercept ($M = 0.35$, z = 1.42, p < 0.001; $\psi = 2.18$, z = 6.89, $p < 0.001$) and slope ($M = 0.15$, z = 2.93, $p < 0.01$; $\psi = 0.06$, z = 3.64, $p < 0.001$). Significant covariance among intercept and slope parameters (ψ = 0.33, z = 5.25, $p < 0.001$) signified that, at the average age of 12-years old, those who most frequently used marijuana demonstrated the greatest growth in use across the transition to young adulthood. Continuing with the model building approach, an autoregressive structure among the residuals was added to the model (AIC = 48409.78, BIC = 48464.84). A likelihood ratio test indicated that the structure significantly improved model fit ($\Delta\chi^2(1) = 31.12$, $p < 0.001$), so it was retained. Though model fit improved, the mean component of the intercept was no longer significant ($M = 0.36$, z = 1.41, $p = 0.16$) once autoregressive paths were taken into account. Significant individual heterogeneity in initial frequency of marijuana use remained ($\psi = 1.23$, z = 2.65, $p <$ 0.01).

Unconditional Bivariate Models: Estimating Growth and Between-Person Associations

Tobacco Use

Univariate growth models for tobacco and DE were combined into an unconditional bivariate model (Curran et al., 2014). The intercept and slope for each construct were allowed to covary within and across constructs. Time-specific residuals also were allowed to covary between tobacco use and DE, and an equality constraint was

used for Waves II and III. The autoregressive component among the residuals of tobacco use and DE was retained. The unconditional bivariate model converged normally (AIC = 108980.78, BIC = 109127.63). Parameter estimates are presented in **Table 7**. Results showed significant variability in the intercept (ψ = 183.38, z = 19.13, $p < .001$) and slope (ψ = 5.42, z = 14.87, $p < 0.001$) of tobacco use as well as the intercept (ψ = 36.22, z = 29.30, $p < .001$) and slope (ψ = 0.04, z = 5.88, $p < .001$) of DE. Significant variability in intercepts and slopes indicated individual heterogeneity in tobacco use and DE behaviors at age 12 and change across adolescence. Mean estimates also were significant for the intercept (M_{tobacco} = 3.56, z = 5.69, $p < 0.001$; M_{DE} = -1.38, z = -8.07, $p < 0.001$) and slope (M_{tobacco} = 0.77, z = 8.79, $p < 0.001$; M_{DE} = 0.55, z = 62.07, $p < 0.001$) of both constructs.

Significant covariation was detected within and across constructs. Initial level of tobacco use negatively covaried with trajectories of use (ψ = -17.49, z = -13.14, $p <$ .001). Those who reported greater initial use showed less growth in use across adolescence. Similarly, elevated initial DE scores were associated with less growth in DE over time (ψ = -0.93, z = -13.60, $p < .001$). Significant covariation also emerged between the intercept of DE and the slope of tobacco use (ψ = 0.71, z = 2.52, p = 0.01) but not its intercept (ψ = 0.26, z = 0.14, p = 0.89). Higher initial levels of DE were associated with greater increases in tobacco use over time. Covariation between the initial level of tobacco use and the slope of DE was marginally significant (ψ = -0.19, z = -1.75, p = 0.08), indicating those with greater tobacco use in early adolescence demonstrated less of an increase in DE over time. Covariation between slopes was not significant (ψ = 0.00, z = 0.18, p = 0.98).

In summary, analyses provided partial support for hypotheses that initial levels (intercepts) and rate of change (slopes) in DE would positively covary with initial levels and rate of change in tobacco use (**Table 1**). Initial level of DE and change in tobacco use demonstrated significant covariation in the hypothesized direction. However, engagement in tobacco use at Wave I was related to marginally less growth in DE. Engagement in DE at Wave I was not significantly related to concurrent engagement in tobacco use, and rates of change for the two constructs were not significantly correlated.

Alcohol Use

The process for the tobacco and DE model was repeated for alcohol and DE, wherein univariate models were combined into a bivariate unconditional model. Intercept and slope factors for each construct were allowed to covary within and across constructs. Time-specific residuals were allowed to covary between alcohol use and DE, with an equality constraint equating covariation for Waves II and III. The autoregressive components for each construct were retained from the univariate models.

This model converged without issue (AIC = 64370.84, BIC = 64517.69). Parameter estimates are presented in **Table 7**. Results indicated significant mean and variance components for intercepts ($M_{alcohol}$ = -0.22, z = -5.88, $p < 0.001$; M_{DE} = -1.39, z = -8.09, $p < 0.001$) and slopes ($M_{alcohol}$ = 0.07, z = 14.98, $p < 0.001$; M_{DE} = 0.56, z = 62.03, $p < 0.001$). In addition, the intercept and slope of alcohol use showed significant covariation (ψ = -0.02, z = -2.78, $p < 0.01$), such that those who used alcohol the least at age 12 showed the greatest growth across adolescence. The intercept parameter of alcohol also significantly covaried with the intercept (ψ = 0.40, z = 2.79, $p < 0.01$) and

slope (ψ = -0.03, z = -3.23, p = 0.001) of DE. Those who endorsed less alcohol use also endorsed less DE in early adolescence, but these individuals also exhibited the greatest growth in DE across adolescence. Neither initial DE scores (ψ = 0.03, z = 1.56, p = 0.12) nor change in DE (ψ = 0.001, z = 0.95, p = 0.34) significantly covaried with change in alcohol use.

These findings provide relatively limited support for hypothesized effects. Significant positive associations were expected across latent factors (described in **Table 1**). Only initial levels of DE and alcohol use showed a significant positive association. Additionally, initial level of alcohol use was negatively, rather than positively, related to change in DE. Neither initial engagement nor change in DE was significantly related to change in alcohol use.

Marijuana Use

Lastly, simultaneous growth in DE and marijuana use was examined. The model converged normally (AIC = 90393.34, BIC = 90540.20). Parameter estimates are presented in **Table 7**. The mean for the intercept parameter of marijuana use remained nonsignificant (M = 0.36, z = 1.38, p = 0.17), with significant individual heterogeneity (ψ = 1.31, z = 2.82, p < 0.01). Mean and variance components were significant for the slope of marijuana use (M = 0.15, z = 2.83, p < 0.01; ψ = 0.07, z = 3.81, p < 0.001) and for both latent parameters of DE (intercept: M = -1.39, z = -8.13, p < 0.001; ψ = 36.19, z = 29.42, p < 0.001; slope: M = 0.56, z = 62.26, p < 0.001; ψ = 0.04, z = 6.13, p < 0.001).

Within constructs, intercepts and slopes were significantly associated ($\psi_{\text{Marijuana}}$ = 0.25, z = 3.64, p < 0.001; ψ_{DE} = -0.94, z = -13.76, p < 0.001). Adolescent girls with

higher levels of marijuana use at age 12 showed the greatest growth in use over time, whereas those who engaged in greater DE behaviors showed the least growth in DE behaviors. Across constructs, only significant covariation between initial DE scores and change in marijuana use ($\psi = 0.31$, z = 2.77, $p < 0.01$) was evident. Those who engaged in more DE behaviors in early adolescence showed the greatest growth in marijuana use over time. A reciprocal relationship was not observed, as covariation between the intercept of marijuana use and slope of DE did not reach statistical significance ($\psi = -0.02$, z = -0.54, $p = 0.59$). Significant covariation did not emerge between slope parameters ($\psi = -0.01$, z = -1.07, $p = 0.28$) or intercepts ($\psi = -0.64$, z = -1.04, $p = 0.30$) across constructs.

Overall, findings provided limited support for the proposed hypotheses **(Table 1**). Significant covariation was expected among latent growth parameters across constructs in the positive direction. However, only the association between initial level of DE and change in marijuana use reached statistical significance. At this stage in model-building, there were no other significant associations between DE and marijuana use intercepts and slopes.

Unconditional Bivariate LCM-SR Models: Estimating Growth and Prospective Within-Person Associations

Tobacco Use:

The unconditional bivariate model was expanded to examine prospective reciprocal relations among the residuals of tobacco use and DE. This model allowed the intercepts and slopes for tobacco use to covary within and across constructs. Time-

specific residuals were allowed to covary between tobacco use and DE with an equality constraint added for Waves II and III. The autoregressive relations among the residuals of tobacco use and DE were retained and expanded to examine reciprocal relations across constructs. In line with a model building approach, the regression of the residuals of one construct on another were tested, while the reciprocal regressions were estimated at zero (Curran et al., 2014).

First, the effect of tobacco residuals on DE residuals were freely estimated, while the effect of DE residuals on tobacco residuals was held at zero (AIC = 108982.43, BIC = 109141.51). Model fit did not significantly improve relative to the unconditional bivariate model, ($\Delta\chi^2$ (2) = 2.36, p = 0.31); relations among residuals were retained for model building due to theoretical importance. Results indicated that the within-person effect of tobacco use at 12-years-old on DE one year later was marginally significant (β = -0.03, z = -2.01, p = 0.05). That is, individuals with less tobacco use at Wave I reported greater DE at Wave II. However, this effect did not persist between Waves II and III. The lagged paths then were constrained to be equal over time. This restriction did not degrade model fit ($\Delta\chi^2$ (1) =-2.18, $p > 0.05$). As such, the model constraining lagged paths was retained. The lagged effect of tobacco at Waves I and II on DE at Waves II and III was not significant (β = 0.002, z = 0.34, p = 0.73).

Next, the within-person effect of earlier DE residuals on later tobacco residuals was freely estimated, while holding the effect of tobacco use residuals on later DE residuals at zero. This model terminated normally (AIC = 108508.80, BIC = 108667.88). The addition of these paths improved model fit ($\Delta\chi^2$ (2) = 475.98, $p < 0.001$). However,

neither the lagged path from DE at Wave I to tobacco use at Wave II (β = -127.52, z = -0.79, p = 0.43) nor that from DE at Wave II to tobacco use at Wave III was significant (β = 0.22, z = 1.39, p = 0.16). The lagged paths then were constrained to be equivalent over time (AIC = 108982.06, BIC = 109135.02). The within-person effects of prior DE on later tobacco use were not significant (β = 0.09, z = 0.67, p = 0.50). Relative to the unconstrained model, constraining the lagged paths led to a significant decrement in model fit ($\Delta\chi^2$ (1) = -475.26, p < 0.001). Thus, the freely estimated lagged paths were retained.

Finally, both sets of within-person regressions were combined into a single model (AIC = 64170.36, BIC = 64341.70) (see **Table 8** and **Figure 4**). Model fit was improved relative to the unconditional bivariate LCM model ($\Delta\chi^2$ (3) = 475.39, p < 0.001). Within constructs, intercepts were significantly related to slopes for DE (ψ = -0.94, z = -18.95, p < 0.001) and tobacco (ψ = 2.66, z = 1.31, p < 0.05), respectively. Initial tobacco use was not significantly related to initial level or rate of change in DE. In contrast, initial DE scores were significantly and positively related to rate of change in tobacco use (ψ = 0.67, z = 2.23, p < 0.05). Rates of change across constructs were not significantly related. At the within-person level, effects of DE on tobacco use were not significant ($\beta_{\text{Wave I}}$ = -61.12, z = -1.04, p = 0.30; $\beta_{\text{Wave II}}$ = 0.13, z = 0.79, p = 0.43), nor were the effects of tobacco residuals on DE residuals (β = -0.01, z = -1.37, p = 0.17).

The hypothesis proposing a within-person, directional effect of prior DE predicting later tobacco use was not supported (see **Table 1**). Significant relations did not emerge at the within-person level in either direction. Between-person effects also

changed in the context of the fully disaggregated LCM-SR model, such that the association between the intercept of tobacco use and the slope of DE was no longer significant. Nevertheless, the association between early DE engagement and an escalating course of tobacco use remained. This between-person association provides support for the hypothesis that DE indicates risk for increasing tobacco use, though this was not echoed at the within-person level.

Alcohol Use:

The established bivariate model examining alcohol use and DE across adolescence was expanded to investigate prospective relations across constructs at the within-person level. The same procedures used to estimate LCM-SR for tobacco use were repeated.

As a first step, the effect of prior DE residuals on later alcohol residuals were freely estimated, as the reciprocal direction of effects were held at zero. The model terminated normally (AIC = 64168.72, BIC = 64327.81) and showed significant improvement over the unconditional bivariate model ($\Delta\chi^2$ (2) = 206.12, $p < 0.001$). The regression of alcohol residuals at Wave II on DE residuals at Wave I (β = -6.37, z = -1.01, p = 0.31) and alcohol residuals at Wave III on DE residuals at Wave II (β = -0.002, z = -0.20, p = 0.84) were non-significant. The overall effect remained non-significant (β = -0.01, z = -0.95, p = 0.35), even when an equality constraint was added to these paths. A likelihood ratio test indicated that constraining these paths to equivalence across time lead to a significant decrement in model fit relative to when the parameters were freely

estimated ($\Delta\chi^2$ (1) = -204.90, $p < 0.001$). Therefore, the freely estimated model was retained.

The effects of alcohol residuals on DE residuals were then freely estimated over time, while the effect of DE residuals on alcohol residuals was held at zero. The model terminated normally (AIC = 25186.22, BIC = 25320.84) and improved model fit ($\Delta\chi^2$ (2) = 6.74, $p < 0.05$) relative to the unconditional bivariate LCM. Results showed that earlier alcohol use residuals significantly predicted subsequent DE residuals, though effects differed across adolescence. Greater alcohol use at Wave I was associated with greater increases in DE from Wave I to Wave II (β = 0.59, z = 3.67, $p < 0.001$) but did not persist between Waves II and III (β = -0.11, z = -1.10, $p = 0.27$). The imposition that these regressions were equal over time led to a significant decrement in model fit ($\Delta\chi^2$ (1) = -5.30, $p < 0.001$), so the freely estimated model was retained.

Lastly, both sets of regressions were combined into a single model **(see Table 8 and Figure 5)**. The overall addition of the structured residuals and cross-lagged paths reproduced the observed data (AIC = 64170.36, BIC = 64341.70) and improved model fit ($\Delta\chi^2$ (4) = 208.48, $p < 0.001$). Covariation within DE latent curve parameters remained significant (ψ = -0.95, z = -19.07, $p < 0.001$), though covariation between the alcohol intercept and slope was not (ψ = 0.01, z = 0.79, $p = 0.49$). Across construct, greater initial DE engagement was marginally associated with greater initial alcohol use (ψ = 0.49, z = 1.74, $p = 0.08$). No other estimates approached significance. At the individual-level, cross-lagged, within-person effects were non-significant (see **Table 8**).

Thus, findings of the LCM-SR model showed limited support for significant associations between DE and alcohol use. At the between-person level, results indicated a marginally significant association wherein the kind of adolescent girl who has elevated DE in early adolescence may also have greater concurrent alcohol use. The significance of the between-person effects in the bivariate LCM model largely disappeared after accounting for within-person effects. Contrary to *a priori* hypotheses, bidirectional relations at the within-person level from the LCM-SR model were not statistically significant.

Marijuana Use:

The bivariate LCM for DE and marijuana use was expanded to disaggregate within-person effects at the level of the residuals. Model procedures as described for tobacco and alcohol use were repeated.

The effect of marijuana residuals on DE residuals were freely estimated, and the reciprocal regressions were held at zero. This model terminated normally (AIC = 90341.44, BIC = 90500.54). Model fit did not improve relative to the bivariate model (χ^2_{Δ} (2) = 1.52, $p > 0.05$). The cross-lagged paths from marijuana residuals to DE residuals were not significant at either time-point ($\beta_{\text{Wave I}} = -0.05$, z = -1.39, $p = 0.17$; $\beta_{\text{Wave II}} = -0.004$, z = -0.18, $p = 0.85$). A likelihood ratio test indicated that constraining the paths to be equal across time did not degrade model fit ($\Delta\chi^2$ (1) = 0.34, $p > 0.05$), and the paths remained nonsignificant ($\beta = -0.01$, z = -1.21, $p = 0.23$). The constrained model was retained for model building.

The effect of DE residuals on marijuana residuals then were tested. The model terminated normally (AIC = 90341.44, BIC = 90500.54), with improved model fit (χ^2_Δ (2) = 55.8, $p < 0.001$). The effects of DE residuals on later marijuana use residuals were not statistically significant ($\beta_{\text{Wave I}}$ = -11.69, z = -1.23, p = 0.22; $\beta_{\text{Wave II}}$ = -0.01, z = -0.21, p = 0.83). The constraint that the magnitude of the effect was equal over time was then tested. This restriction degraded model fit ($\Delta\chi^2$ (1) = -55.46, $p < 0.001$), so the freely estimated model was retained.

As a final step, reciprocal within-person regressions were estimated simultaneously (see **Table 8** and **Figure 6**). The model terminated normally (AIC = 90340.26, BIC = 90505.47) and improved model fit relative to the bivariate LCM ($\Delta\chi^2$ (3) = 59.08, $p < 0.001$) At the between-person level, initial use of marijuana was marginally related to change in use over time (ψ = 0.12, z = 1.74, p = 0.08), such that those with greater use in early adolescence also demonstrated the greatest growth in use over time. Initial DE scores were significantly and inversely related to change in DE over time (ψ = -0.94, z = -13.69, $p < 0.001$). With respect to cross-construct relations, initial DE scores and changes in marijuana use were significantly and positively associated (ψ = 0.30, z = 2.17, $p < 0.05$). Adolescents who reported more DE at Wave I showed greater increases in marijuana use over time. No other significant associations emerged among latent curve parameters. Within-person effects were non-significant. Findings, therefore, did not provide support for the hypothesized bidirectional relationships between marijuana use and DE behaviors at the individual level (**Table 1**), though between-person

associations suggest early DE may be a risk factor for escalating marijuana use across adolescence.

Conditional Bivariate LCM-SR Models: Associations and Growth in the Context of Covariates

Between-person and within-person associations over time were reexamined with the inclusion of time-invariant (i.e., ethnicity/race, menarche, and hyperactivity/impulsivity) and time-varying (i.e., depression, anxiety, and delinquency) covariates. The goal of these analyses was to examine whether relations between DE pathology and specific substances remained after theoretically important covariates were taken into account (**Tables 9 – 13).** In other words, analyses sought to examine whether relations between DE and use of specific substances persisted over and above developmental considerations and internalizing and externalizing symptoms. Though Curran and Bauer (2011) recommend disaggregating between- and within-person effects of time-varying covariates used in longitudinal growth models, this could not be meaningfully accomplished in the current dataset due to missingness. Therefore, all time-varying covariates were added at measurement occasions for which data was available and did not disaggregate between- and within-person effects.

Tobacco Use

After establishing the optimal within-person model, the final step was to examine the effect of time-invariant and time-varying covariates on the three latent growth factors and their residuals (see **Tables 10** and **13**). Ethnicity, race, menarche, and a retrospective measure of hyperactivity were treated as time-invariant covariates and used to predict

intercepts of tobacco use and DE as well as the slope of tobacco use. Measures of depression, anxiety, and delinquency were included as time-varying covariates predicting residual variance in DE and tobacco use at each wave. The model terminated normally (AIC = 75885.88, BIC = 76261.94).

With respect to demographic variables, significant main effects of ethnicity and race on DE and tobacco use latent curve parameters emerged (**Table 13)**. Comparisons showed that Non-Hispanic and Hispanic adolescent girls experienced comparable levels of tobacco use early in adolescence (β = -1.29, z = -1.08, p = 0.28), though Non-Hispanic teens experienced marginally greater growth in use over time (β = -0.39, z = -1.78, p = 0.07). Examination of racial differences revealed only significant differences between Black and White adolescent girls. White adolescent girls reported significantly higher levels of tobacco use (β = -4.00, z = -2.72, p = 0.01) and DE (β= -1.32, z = -3.68, $p <$ 0.001) at age 12 as well as greater increases in use (β = -0.59, z = -3.06, $p < 0.01$). However, Black and White adolescent girls showed comparable changes in DE over time (β = 0.02, z = 1.30, p = 0.18). Significant differences did not emerge between Asian and Native American relative to White adolescents.

With respect to physical development, achieving menarche earlier in adolescence significantly predicted greater DE pathology at age 12 (β = -0.53, z = -5.12, $p < 0.001$) but not initial severity of tobacco use (β = -0.41, z = -1.60, p = 0.11). Main effects of menarche on slopes of tobacco use and DE were not significant.

Retrospective reports of hyperactivity/impulsivity significantly and positively predicted DE pathology at age 12 (β = 0.82, z = 3.26, p = 0.001) and change in DE across

adolescence (β = -0.03, z = -2.45, p = 0.01). Those who reported greater hyperactivity/impulsivity symptoms had greater DE pathology at age 12 but showed smaller increases in DE over time. In contrast, hyperactivity/impulsivity did not significantly predict initial level of tobacco use but did significantly predict the slope (β = 0.47, z = 4.58, p < 0.001), such that more impulsive adolescent girls showed greater increases in tobacco use over time.

Time-specific effects of time-varying covariates were examined at each wave **(Table 13)**. Higher concurrent depression scores were associated with higher tobacco use residuals at Wave I (β = 0.22, z = 4.49, p < 0.001) and II (β = 0.21, z = 4.01, p < 0.001) but not at Wave III (β = -0.01, z = -0.20, p = 0.84). Higher depression scores significantly predicted higher concurrent DE residuals at Wave II (β = 0.04, z = 2.88, p < 0.01) and Wave III (β = 0.04, z = 4.02, p < 0.001) but not Wave I. Concurrent anxiety scores only significantly predicted DE pathology at Wave II (β = 0.07, z = 2.11, p < 0.05), with greater DE pathology among those with higher anxiety, and anxiety scores did not significantly predict tobacco use at any time point. Concurrent delinquency scores were significantly and positively associated with tobacco use at each time point (β_{WaveI} = 0.65, z = 6.74, p < 0.001; β_{WaveII} = 1.08, z = 10.11, p < 0.001; β_{WaveIII} = 1.07, z = 4.40, p < 0.001) but not with DE pathology.

With inclusion of the covariates, the pattern of associations between DE and tobacco use did not change from results of the unconditional LCM-SR model **(Table 9)**. Within constructs, initial level of DE remained significantly and inversely associated with rate of change in DE (ψ = -1.01, z = -15.53, p < 0.001). Latent curve parameters for

tobacco use were not significantly related. Initial DE behaviors were significantly and positively related with change in tobacco use over time (ψ = 1.07, z = 2.11, p = 0.04) but not with initial level of tobacco use. In addition, prospective cross-lagged effects at the within-person level remained non-significant **(Table 10)**.

Taken together, these findings suggest that associations between tobacco use and DE are evident at the group-level but not at the individual-level. These findings were not in line with proposed hypotheses, which posited that directional effects of DE predicting later tobacco use would emerge at both levels of effect and persist in the context of risk factors due to its appetitive effects (**Table 1**). Rather, findings regarding individual-level effects indicated that depression and delinquency may play a larger role in explaining the co-occurrence than weight-related motives among those with DE attitudes and behaviors, though DE remained a risk factor for escalating tobacco use at the group level.

Alcohol Use

The procedures used to estimate the conditional bivariate LCM-SR model for tobacco use were replicated here. Main effects of the time-invariant covariates on the DE intercept and slope and main effects of the time-varying covariates on residual DE scores at each wave were previously reported and presented in **Table 14**. Only main effects of covariates on alcohol outcomes are presented below.

The model terminated normally (AIC = 71826.76, BIC = 72208.46). Significant findings emerged for a number of covariates **(Table 14)**. With respect to demographic variables, there was a significant main effect of race on alcohol use parameters. Dummy-coded comparisons revealed that identifying as White, compared to Black, predicted

higher initial levels of alcohol use (β = -0.19, z = -2.90, $p < 0.01$). Additionally, White adolescent girls experienced significantly greater increases in use over time relative to their Black (β = -0.04, z = -3.91, $p < 0.001$) and Asian same-sex peers (β = -0.06, z = -2.57, $p < 0.05$). Though ethnic identity did not significantly predict differences in initial levels alcohol use, identifying as Non-Hispanic significantly predicted greater increases in use (β = -0.03, z = -2.36, $p < 0.05$) relative to Hispanic peers.

With respect to physical development, earlier age of menarche significantly predicted greater alcohol use at age 12 (β = -0.04, z = -2.17, $p < 0.001$) and greater growth in use across adolescence (β = 0.01, z = 3.03, $p < 0.01$). A marginally significant main effect of hyperactivity/impulsivity emerged for change in alcohol use only (β = 0.01, z = 1.73, $p = 0.08$), with higher levels of hyperactivity/impulsivity associated with greater alcohol use.

Time-specific effects of time-varying covariates on residuals of alcohol use were examined at each wave. Delinquency significantly and positively predicted concurrent alcohol at all waves (β_{WaveI} = 0.14, z = 20.08, $p < 0.001$; β_{WaveII} = 0.18, z = 21.17, $p < 0.001$; $\beta_{WaveIII}$ = 0.15, z = 8.32, $p < 0.001$). Contemporaneous depression significantly and positively predicted alcohol use at all waves (β_{WaveI} = 0.03, z = 8.85, $p < 0.001$; β_{WaveII} = 0.03, z = 6.66, $p < 0.001$; $\beta_{WaveIII}$ = 0.02, z = 4.44, $p < 0.001$). Concurrent anxiety scores did not predict alcohol use at either wave (β_{WaveI} = -0.01, z = -0.90, $p = 0.41$; β_{WaveII} = -0.01, z = -1.01, $p = 0.28$).

Covariation among latent growth parameters changed between previous models and the final unconditional LCM-SR **(Table 9)**. Only covariation between DE latent

curve parameters remained significant (ψ = -111.87 z = -17.09, $p < 0.001$), and the once marginal association between initial levels of DE and alcohol use became non-significant (ψ = 2.32., z = 0.85, $p = 0.39$). All non-significant estimates are reported in **Table 9**. Within-person effects in both directions remained non-significant (**Table 11)**, as in previous models. Results of the conditional LCM-SR model did not support the hypothesis that bidirectional effects would be present, though weakened, in the context of important risk factors. Thus, any co-occurrence between DE and drinking behavior appears to be accounted for by a number of risk factors, including race, timing of sexual maturation, and internalizing and externalizing symptoms.

Marijuana Use

Procedures from the previous conditional bivariate LCM-SR models were replicated. Main effects of the time-invariant covariates on the DE intercept and slope and main effects of the time-varying covariates on residual DE scores at each wave were previously reported and presented in **Table 15**. Only main effects of covariates on marijuana outcomes are presented below.

The model converged normally (AIC = 63594.04, BIC = 63970.13). Significant main effects of demographic variables and menarche on marijuana use latent curve parameters did not emerge **(Table 15)**. However, retrospective reports of hyperactivity/impulsivity significantly predicted growth in marijuana use across adolescence ($\beta = 0.09$, z = 2.16, $p < 0.05$). That is, individuals higher in hyperactivity/impulsivity reported greater increases in marijuana use across adolescence.

Time-specific effects of time-varying covariates on concurrent marijuana use and DE residuals were examined at each wave **(Table 15)**. Delinquency scores significantly predicted marijuana use at each time point ($\beta_{WaveI} = 0.33$, z = 8.62, $p < 0.001$; $\beta_{WaveII} = 0.49$, z = 11.94, $p < 0.001$; $\beta_{WaveIII} = 1.02$, z = 13.45, $p < 0.001$) with greater delinquency associated with more marijuana use. The concurrent effects of depression on marijuana residuals were time-specific and emerged as significant only at Waves I and II ($\beta_{WaveI} = 0.04$, z = 2.99, $p < 0.01$; $\beta_{WaveII} = 0.06$, z = 2.44, $p < 0.05$, $\beta_{WaveIII} = 0.05$, z = 1.39, $p = 0.17$), with greater depression associated with more marijuana use. The effect of concurrent anxiety on marijuana was only significant at Wave II ($\beta_{WaveII} = -0.13$, z = -2.00, $p = 0.05$), with greater anxiety associated with less frequent marijuana use.

Between- and within-person effects were largely consistent with the results of the unconditional LCM-SR model. Across constructs, covariation between initial DE and growth in marijuana use, which was present in earlier models, became only marginally significant ($\psi = 0.29$, z = 0.17, $p = 0.09$) in the conditional model. Within constructs, the significant association between initial DE scores and rate of change in DE over time ($\psi = -1.01$, z = -15.32, $p < 0.05$) persisted. No other significant associations emerged at the between-person level **(Table 9)**. Moreover, the conditional model did not provide evidence of within-person lagged effect paths. This replicated earlier findings of the LCM-SR **(Table 12)**. Overall, these findings offer limited support for the hypothesized results (**Table 1**). Group-level associations suggest early DE may act as a risk factor for greater growth in marijuana use, which is only partially accounted for by shared risk

factors. Individual-level effects did not emerge at any stage of model building, indicating that co-occurrence at this level of effect may due to third variable mechanisms.

DISCUSSION

It is well-established that SU and DE co-occur across development, yet the precise nature of their temporal associations and coupling has remained a topic of debate. The broader literature has predominately supported the theory that early DE behaviors and attitudes precede the initiation of SU (e.g., Measelle et al., 2006) based predominately on between-subject study designs. Although useful in understanding trends at the group level, such analytic approaches do not address the fundamental question of temporal precedence *within* individuals over time. Previous analytic approaches have rarely tested potential bidirectional effects and have collapsed across substances, thereby overlooking distinct psychopharmacological properties that may be uniquely related to DE.

The current study sought to expand the literature through the application of an appropriate analytic approach (i.e., LCM-SR) using longitudinal data during the transition from adolescence into early adulthood. Substances commonly used in adolescence (i.e., tobacco, alcohol, and marijuana) were of particular interest given the developmental timing and their distinct appetitive properties. Important contributions of this work include: (1) disaggregating between- and within-person effects in the context of developmental trajectories for DE and SU for the purpose of understanding both stable and dynamic components of this comorbidity; (2) testing bidirectional effects between DE and SU in substance-specific models; and (3) examining the extent to which a unique relationship persists after accounting for shared etiologic risk factors (i.e., ethnic and racial identity, early sexual maturation, and internalizing and externalizing symptoms).

Tobacco Use

Though some effects changed across model-building stages, findings consistently showed a directional effect of early DE behaviors on tobacco use during adolescence (Cawley et al., 2004). At the initial phase (bivariate LCM), greater initial levels of either DE were associated with growth in tobacco use, while those with greater initial level of tobacco use showed less growth in DE behaviors. When within-person effects were added to the model, the significant inverse association between initial levels of tobacco use and growth in DE engagement did not persist. However, initial DE engagement continued to be positively associated with growth in tobacco use, accounting for within person effects, and this effect persisted when controlling for important theoretical risk factors. Findings of the conditional model support the *a priori* hypothesis that early DE engagement serves as a risk factor escalation of tobacco use at the between-person level. This is in accordance with the bulk of previous literature, reflecting the known appetite-suppressing effects of nicotine (Chiolero et al., 2008; Gonseth et al., 2012).

Results of the conditional model underscored a link between early DE behaviors and an increased propensity for externalizing symptoms. Greater hyperactivity/impulsivity prior to 12-years-old significantly predicted more elevated DE engagement and greater growth in tobacco use across adolescents. Hyperactivity/impulsivity are often found to correlate with novelty seeking (e.g., Zuckerman, Kuhlman, Joireman, Teta, & Kraft, 1993). Girls who demonstrate greater hyperactivity/impulsivity in childhood may be at risk for early engagement in DE as well as experimentation with and progression to regular use of tobacco use (Fuemmeler,

Kollins, & McClernon, 2007). Interestingly, greater hyperactivity/impulsivity was associated with less growth in DE engagement across adolescence in the current study. This is contrary to literature suggesting that more extreme DE behaviors, such as use of diet pills, self-induced vomiting, and misuse of laxatives, emerge among those with greater impulsivity (Waxman, 2009). One explanation for this may be due to measurement. Items used to capture hyperactivity/impulsivity may reflect relations with hyperactivity more accurately than with impulsivity. Future research should continue to investigate the role of externalizing symptoms with more precise measurement.

A priori hypotheses positing within-person effects were not supported in the current study. Only one within-person effect emerged: greater Wave I tobacco use associated with less growth in DE engagement from Wave I to Wave II. This effect was in the opposite direction of predictions, and it did not persist when reciprocal within-person effects were modeled. Given the concurrent prediction by depression for both DE and tobacco use, within-person effects may be linked through increasing internalizing symptoms across adolescence. The link between DE behaviors and increased negative affect has been well-established (Stice, 1994). DE may increase the propensity for depressive symptoms, thereby increasing initiation or escalation of tobacco use for either regulation of appetite or negative affect. Alternatively, a lack of within-person findings may be due to limited variability at this level during the adolescent period coupled with unstructured time lags. The fluctuations between DE and tobacco use may be best captured with more discrete measure of time. The lag between Waves II and III spanning

5 years may have masked the potentially nuanced relations between DE engagement and tobacco use at the within-person level.

Alcohol Use

Results across models for alcohol use were also inconsistent with hypotheses. In this case, the resulting pattern of effects suggested that the co-occurrence of alcohol use and DE is completely explained by the presence of theoretical risk factors. Initial associations at the between-person level suggested that adolescent girls with elevated DE engagement at Wave I also engaged in greater concurrent alcohol use, and those with elevated initial alcohol use demonstrated the least growth in DE across adolescence. Although the positive correlation between the intercepts of DE and alcohol use remained marginally significant with the addition of within-person effects, this association was not evident after controlling for shared risk factors. Thus, findings did not provide support for *a priori* hypotheses proposing bidirectional relationships at either the between- or within-person level.

Rather, results indicated that comorbidity between alcohol use and DE may arise due to shared risk. Early menarche significantly increased risk for greater initial DE engagement across substantive models and showed a substance-specific effect for alcohol use. Most notably, early menarche predicted greater initial alcohol use and an escalating course of use across adolescence. This is consistent with prior work showing that early sexual maturation is associated with increased body dissatisfaction, dieting behaviors, and substance use (Dick et al., 2000; Striegel-Moore et al., 2001; Tschann et al., 1994). The mechanism through which early menarche promotes this comorbidity remains

unclear. One possibility is that girls who reach sexual maturation earlier than their peers may gravitate towards older or more deviant peer groups (Marceau & Jackson, 2017). In this way, early menarche may serve as a proxy for peer norms or facilitate social learning via modeling of behaviors.

Alternatively, off-time developmental transition may exacerbate vulnerability to negative affect due to physiological, psychological, and social transitions (Brooks-Gunn et al., 1985; Caspi & Moffitt, 1991). Though temporal precedence cannot be fully established in the current study due to its design, there is some support for this pathway, given the robust prediction of elevated DE and alcohol use by concurrent elevations in depression symptoms. Research utilizing more nuanced study designs is needed to further understand how pubertal timing and tempo confer risk for this comorbidity.

Marijuana Use

Lastly, substance specific models estimating marijuana use and DE failed to show support for anticipated bidirectional effects. Nevertheless, results highlighted the robust association of initial DE engagement with rate of change in marijuana use over time, similar to the results for tobacco use. This effect persisted over and above the inclusion of theoretical risk factors for both constructs, though the strength of the association weakened to marginal significance in the final model. The robust association across constructs may be in part due to the metabolic effects of marijuana. Whereas acute intoxication from marijuana may promote increased caloric consumption with bouts of "munchies," chronic use has been found to reduce appetite and be associated with lower

body weight (Meier et al., 2016). The current study adds to the scant literature on this co-occurrence, moving beyond cross-sectional study designs.

Despite findings at the between-person level, results did not show support for coupling of these behaviors at the within-person level. The lack of support for a within-person relation may suggest that adolescent girls most suspectable to experimenting with weight-loss aids are unaware of the appetitive effects of marijuana use. As marijuana use continues to become legalized in the US, the awareness of its long-term side effects may increase along with instrumental use. Alternatively, the measurement of marijuana use (single item) and length between measurement occasions may have hindered our ability to detect effects at this level. Future research should continue to investigate potential within-person prospective associations and a potential bidirectional relation using more robust measures of marijuana use and shorter time lags.

Extensive research has shown that externalizing symptoms are robustly associated with marijuana use (Pedersen, Rømer Thomsen, Pedersen, & Hesse, 2017; Pedersen et al., 2018), have a dose-response relationship, and precede experimentation with marijuana. Indeed, a recent study found that associations between marijuana use and internalizing/externalizing symptoms were consistently stronger among adolescent girls than their male peers in a large sample of adolescents between 12- and 17-years old (Girgis, Pringsheim, Williams, Shafiq, & Patten, 2020). Adolescent girls were more than 5 times more likely to use marijuana if experiencing concurrent externalizing symptoms and 2 times more likely if experiencing internalizing symptoms (Girgis et al., 2020). The current study's findings echo these findings, as hyperactivity/impulsivity predicted early

DE engagement as well as growth in marijuana use. Therefore, early onset DE may reflect a propensity for externalizing symptoms through which marijuana use is exacerbated.

Implications Across Substantive Models

Taken together, the results across substance specific models have two important implications for our understanding of the common co-occurrence of DE and SU. First, the current study suggests there are substance-specific effects with respect to relations between DE and commonly used substances. Even within the adolescence, a developmental period characterized by risk-taking and experimentation (Steinberg, 2008), relations between DE and tobacco and/or marijuana emerged as distinct from the relation between DE and alcohol. Prior work collapsing across substances may be missing important motives and pathways for comorbidity. Moreover, research failing to show effects of body acceptance programs on reducing overall risk for SU may be overlooking specificity within this comorbidity. Thus, future research should continue to consider differential effects when investigating this comorbidity.

Second, although relations between specific substances and DE are unique, the broader co-occurrence of DE and SU may be largely due to shared underlying mechanisms. Previous literature has suggested multiple shared indicators of risk rather than a single cause, posing great difficulty for treatment and prevention efforts. Given the support for between-person findings but lack of evidence for within-person effects (at least for tobacco and marijuana), the current study suggests that there may be a core set of risk factors that are the underlying culprit for the co-occurrence of DE and SU.

Relevant to this possibility, Barlow, Allen, and Choate (2004) proposed an underlying Negative Affect Syndrome to explain why most emotional disorders have considerable co-occurrence as well as share an interacting set of vulnerabilities. A decade later, Caspi and colleagues (2014) proposed an even broader proclivity towards psychopathology connecting all comorbid presentations. Little *p*, a latent factor capturing one General Psychopathology dimension, was found to underly all internalizing, externalizing, and thought disorders and served as a theoretical solution to the common barrier of conceptualizing comorbidity in nosology. An underlying negative affect syndrome or a broader proclivity towards psychopathology may best explain the shared risk of comorbidity for DE and SU at both the syndromal and diagnostic levels. Future research should consider examining how negative affect syndrome and little p may relate to the development of DE and SU across this critical period of risk.

Implications for Prevention Efforts

Prevention efforts should consider shared risk as well as distinct relations in program development. The current study underlines the theoretical importance of utilizing a transdiagnostic approach for reducing core dysfunction in universal prevention programs. Results highlight concurrent associations of both externalizing and internalizing symptoms with both onset and development of both DE and SU among teen girls. The Universal Protocol for Transdiagnostic Treatment of Emotional Disorders is one example of a treatment protocol that has recently been adapted and piloted as a universal prevention program to target common dysfunction across internalizing and externalizing problems with promising preliminary results (Ishikawa et al., 2019).

Prevention programs may consider further expanding transdiagnostic approaches by utilizing mobile technology to remind adolescents of alternative coping skills (e.g., Conrod, Castellanos-Ryan, & Strang, 2010). Between-person associations also suggest the importance of addressing core components of DE pathology in selective and indicated prevention programs for SU among teen girls. For example, programs may consider targeting an internalization of and desire to obtain a body ideal, internalized weight stigma, and body dissatisfaction in addition to universal targets when preventing tobacco or marijuana use.

Between-person findings also inform *who* prevention efforts should target. For example, girls who achieved menarche earlier than their peers had elevated levels of DE and alcohol use. Since elevated DE behaviors were related to escalating tobacco and marijuana use across development, early maturing girls who report elevated body dissatisfaction or desire to lose weight may be most at risk. Programming may be adapted to include brief psychoeducation on healthy and regular eating in the context of physical changes associated with adolescence as well as provide information on signs and symptoms of problematic dieting practices to parents during routine pediatric appointments.

The adaptation of brief intervention efforts within a pediatric setting may be particularly important when considering *who* is most at risk and *where* these families are most likely to seek psychological consultation. Between-person comparisons of racial/ethnic groups largely mimic the broader literature with comparable endorsement of SU and DE among Hispanics and Non-Hispanic adolescents, comparable endorsement

among Asian, Native American, and White adolescents, and less endorsement among Black adolescent girls (e.g., Bruening & Perez, 2019; Evans-Polce, Vasilenko, & Lanza, 2015; McCabe et al., 2007; Neumark-Sztainer et al., 2002). Despite being at comparable risk, minority girls and women are less likely to receive appropriate mental health treatment when indicated compared to White peers. For example, Hispanic and Black women are most likely to seek psychological consultation via their primary care physician but are less likely to be properly assessed by a doctor, even when acknowledging eating and weight concerns, or to receive a referral for further evaluation or care (Becker, Franko, Speck, & Herzog, 2003; Priester et al., 2016; Reyes-Rodríguez, Ramírez, Davis, Patrice, & Bulik, 2013). Prevention and early intervention efforts for this comorbidity, therefore, may consider the benefit of adapting early screening and intervention within the pediatric setting.

Limitations

Although the current investigation addressed specific methodological problems of prior work, it is not without its own limitations. First, analyses examined each substance individually, so they did not control for use of other substances (e.g., analyses for tobacco use did not control for alcohol and marijuana use). Failing to collapse across substances or control for polysubstance use may be problematic due to increased experimentation and risk-taking during this developmental period. Nevertheless, one would expect etiologic risk factors, particularly indices of impulsivity and delinquency, to mitigate this limitation, as these risk factors tend to be common to all substances of abuse. Nonetheless, future research should consider comparing analyses that collapse across

substances with substance specific models that control for use of other substances, as these findings would expand our understanding of unique relations between DE and use of specific substances.

Second, the current study was limited in its ability to analyze longitudinal changes in the primary outcomes due to the study design. The length of time-lags between assessments may impact the study's power in detecting bidirectional relations. A year, and particularly 5 years, may simply be too long of a period over which to identify reciprocal within-person relations. Studies that conduct more frequent assessments during critical developmental periods may have greater utility for identifying such effects.

Third, the current study was limited in its measurement of the primary outcomes of interest. Substances were operationalized using largely comparable but not identical items. Two and three items were used across waves to capture tobacco and alcohol use respectively. In contrast, only frequency of marijuana use was consistently available across all waves. Although the current study reflects measurement issues in the broader literature (e.g., lack of agreed upon methods for assessing quantity of marijuana use within an occasion), it should be noted that a more robust measurement of marijuana use may be better suited to capturing associations with DE. In addition, assessment of binge eating was only introduced into the study at the final wave (Wave III) and, therefore, could not be included as an index of DE. Prior research has shown robust associations between binge eating and tobacco, alcohol, and marijuana use (e.g., Piran & Robinson, 2011; White et al., 2007). The results of the current study may under-estimate the strength of associations between DE behaviors and substance use.

Fourth, findings are limited in their ability to generalize to current trends in SU and DE due to the age of the dataset. The burden of relevance is one shared by all longitudinal datasets and is inherent in its design. The author is not aware of prior work identifying cohort effects in ***relations*** between use of specific substances and disordered eating, despite recent increases in marijuana use and decreases in tobacco use. *If cohort effects occur in future work*, it is critical to understand how these behaviors are related prior to policy and social changes to serve as a baseline for comparison. This may be particularly relevant to relations between DE and marijuana use given many recent changes to laws regarding both medical and recreational marijuana in the United States.

Fifth, although we were able to compare across racial and ethnic groups, nuanced examination was beyond the scope of the current study. Thus, the current study serves as a launching point for future discussion and consideration regarding how etiology and pathogenesis may differ across racial/ethnic groups. Readers are cautioned against interpreting significant racial comparisons as suggesting a particular racial/ethnic group as "protected" against SU, DE, or their comorbidity. Studies examining how to adapt programs appropriately, to increase acceptability among diverse groups, and to enhance accessibility given cultural preferences for psychological consultation are critical.

Future work would also benefit from consideration of the broader impact of the environment on DE and SU development. Socioeconomic status and neighborhood characteristics (e.g., "wet" versus "dry" environments; food deserts and food swamps; community exposure to traumatic events) may contribute more meaningfully to the prediction of either outcome than belonging to a particular racial/ethnic group (Osorio,

Corradini, & Williams, 2013; Ryabov, 2020), though these are intertwined due to systematic racism. Lab-based studies have shown the impact of environmental as well as social contexts on individual responses to substances and food (e.g., Corbin, Hartman, Bruening, & Fromme, 2020; Cummings, Gearhardt, Ray, Choi, & Tomiyama, 2020). Moreover, findings suggest that these influences may have a greater effect on adolescent girls. In study of over 25,000 high school students, alcohol outlets ("wet environments") and perceived availability of other substances in an adolescent's neighborhood predicted substance use among girls, but not boys (Milam, Johnson, Furr-Holden, & Bradshaw, 2016). Future research should consider the impact of these factors on the development of DE and substance use comorbidity, particularly in identifying targets within the physical or social environment that may be the focus of prevention and policy work.

Conclusion

The current study sought to add to the literature by 1) examining the coupling of DE and SU among adolescent girls in substance-specific models and 2) disaggregating between- and within-person effects using prospective data from a nationally representative sample. Findings across models provide some evidence of differential effects by substance. Inherent in both tobacco and marijuana use are appetitive side-effects that may be instrumentally used to facilitate weight control, which may contribute to this distinct relationship. No significant effects between alcohol and DE emerged in final steps of model building. Overall, findings suggest distinct relations by substance may emerge even during adolescence, a period characterized by heightened risk-taking and experimentation. The pattern of substantive effects indicated that broad emotional

dysfunction in addition to specific aspects of DE pathology may provide critical targets to be addressed within prevention programs. Future research may continue to consider disaggregating level of effects with discrete, smaller time lags in order to further clarify changes in the coupling of these behaviors and to precisely inform the timing of prevention and intervention efforts.

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Table 1.

Summary of Substance-Specific Hypotheses across Three Aims of Project

Substance	Between-Person	Within-Person	Controlling for Shared Risk Factors
Tobacco	DE intercept and slope are positively associated with SU intercept and slope.	DE predicts SU	Associations and directional effects at each level remain.
Alcohol	DE intercept and slope are positively associated with SU intercept and slope.	Reciprocal effects	Only change in alcohol use is associated with initial scores and change in DE at the between-person level. Reciprocal effects remain at the within-person level but weaken in magnitude.
Marijuana	DE intercept and slope are positively associated with SU intercept and slope.	Reciprocal effects	Initial scores and change in marijuana use is associated with initial scores in DE the between-person level. Reciprocal effects remain but are weaker in magnitude at the within-person level.

Table 2.
Proposed Models and their Estimated Effect

	Analysis	Effect(s)
Aim 1	1. Estimate unconditional univariate models that account for underlying trajectory of each outcome 2. Estimate unconditional bivariate latent curve model (LCM)	Between
Aim 2	3. Expand unconditional bivariate LCMs to account for cross-lag paths among residuals controlling for the underlying trajectory of each outcome (LCM-SR)	Between & Within
Aim 3	4. Estimate conditional LCM-SRs accounting for covariates relevant to both constructs	Between & Within

Table 3.
Descriptive Statistics for Outcome Variables and Covariates

	Wave I				Wave II				Wave III			
	M/%	SD	Skew	Kurt	M/%	SD	Skew	Kurt	M/%	SD	Skew	Kurt
Dieting	21%				23%				27%			
Use of diet pills	1.2%				1.7%				5.2%			
Self-induced vomiting	0.3%				0.6%				0.3%			
Laxative misuse	0.3%				0.4%				0.3%			
Tobacco frequency	4.09	9.39	2.16	2.96	5.10	10.25	1.81	1.53	7.21	12.21	1.22	-0.41
Tobacco quantity	1.49	4.06	3.63	14.46	1.89	4.69	3.28	11.25	3.12	6.39	2.30	4.76
Alcohol frequency	0.97	1.35	1.41	1.26	0.99	1.41	1.39	1.11	1.87	1.61	0.36	-0.94
Alcohol quantity	2.02	3.65	2.72	7.93	2.29	4.06	2.43	5.82	2.60	3.07	2.26	7.14
Alcohol intoxication	0.53	1.09	2.34	5.16	0.57	1.14	2.29	4.93	0.86	1.21	1.49	1.63
Marijuana frequency	0.84	3.78	6.05	38.85	1.05	4.36	5.37	29.67	1.74	5.89	3.92	14.62
Sexual maturation[a]	12.19	1.90	0.05	1.04								
Anxiety	2.97	1.49	-0.26	-0.95	3.31	1.45	-0.54	-0.68				
Depression	6.29	4.22	0.83	0.61	7.38	4.23	0.81	0.50	7.27	3.66	1.48	2.91
Delinquency	1.77	1.80	1.29	1.65	1.56	1.67	1.41	2.32	.32	1.13	4.37	21.92
Hyperactivity/Impulsivity[b]									0.75	0.54	1.06	1.21

Note. Raw data is presented here for individual items of primary predictors and outcomes of interest. Dichotomous variables, which include cigarette use, dieting, use of diet pills, self-induced vomiting, and laxative misuse, are coded such that endorsement of the item was coded as 1. Frequencies are displayed for these items.

[a] Sexual maturation was conceptualized as age of menarche. This question was asked at all waves. Responses were pulled from each wave to reduce missing data. Ninety percent of the responses came from Wave I, 8.8% from Wave II, and 0.01% at Wave III.

[b] Hyperactivity/impulsivity items were available only at Wave III and assessed symptoms between the ages of 5- and 12-years old.

Table 4.
Zero-Order Correlations among Variables of Interest at Wave I

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
1. Dieting	-	.09*	.01	.03	**.04**	.03	.08*	.06*	.07*	.00	**-.04**	.10*	.10*	**.04**	.03
2. Use of diet pills		-	**.05**	.10*	**.04**	.03	.07*	.08*	.05*	.00	.01	.05*	.08*	.03	**.04**
3. Self-induced vomiting			-	.10*	.03	.00	**.04**	.02	.03	.00	.01	**.03**	.05*	.02	.03
4. Laxative misuse				-	-.02	-.02	**.04**	.03	.03	.00	-.01	.00	.02	.02	.00
5. Cigarettes frequency					-	.80*	.34*	.36*	.32*	.38*	-.02	.12*	.19*	.24*	.11*
6. Cigarettes quantity						-	.35*	.40*	.35*	.33*	**-.04**	.09*	.18*	.24*	.11*
7. Alcohol frequency							-	.65*	.76*	.35*	.02	.17*	.23*	.38*	**.05**
8. Alcohol quantity								-	.60*	.30*	.01	.14*	.21*	.33*	.10*
9. Alcohol intoxication									-	.40*	.03	.15*	.21*	.36*	.07*
10. Marijuana use										-	.01	.07*	.13*	.24*	.06*
11. Sexual maturation[a]											-	.01	-.02	.00	.02
12. Anxiety												-	.40*	.20*	.11*
13. Depression													-	.30*	.16*
14. Delinquency														-	.06*
15. Hyperactivity/impulsivity[b]															-

Note. Correlations significant at the 0.05 level (2-tailed) are in boldface. Correlations significant at the 0.01 level (2-level) are denoted by a single asterisk (*). Point-biserial correlations are shown among dichotomous and continuous variables

[a] Sexual maturation was conceptualized as age of menarche. This question was asked at all waves. Responses were pulled from each wave to reduce missing data. Ninety percent of the responses came from Wave I, 8.8% from Wave II, and 0.01% at Wave III.

[b] Hyperactivity/impulsivity were only available at Wave III and assessed symptoms between the ages of 5- and 12-years old. Associations between variables of interest at Wave I and hyperactivity/impulsivity at Wave III are shown here.

Table 5.
Disordered Eating Latent Factor Model Estimates for Item-Response Testing over Time

Model	χ^2	Df	$\Delta \chi^2$	Δdf	p-value
Configural	62.76	51	---	---	---
Metric	66.45	57	7.46	6	0.28
Full Scalar	97.04	63	45.89	6	0.01
Partial Scalar	65.55	62	0.58	5	0.99

Table 6.
Model Fit Indices for Measurement Invariance Testing of Alcohol Use Items

	RMSEA	CFI	SRMR
Configural	0.017	0.997	0.013
Metric	0.044	0.977	0.045
Partial Metric	**0.016**	**0.997**	**0.014**
Scalar	0.040	0.979	**0.036**
Partial Scalar	**0.017**	**0.996**	**0.015**

Note. Bolded estimates in the table indicate that the model fit index was not meaningful worse than the previous nested model. Cut-offs suggested by Cheung and Rensvold (2002) were used. Differences of 0.015 (RMSEA), 0.01(CFI), 0.030 (SRMR) for metric invariance tests and 0.015 (RMSEA), 0.01 (CFI), and 0.010 (SRMR) for scalar invariance tests.

Table 7.
Variance and Covariance Matrix for Intercepts and Slopes across Unconditional Bivariate LCM (Aim 1)

	1	2	3	4	5	6	7	8
1. DE Intercept	36.22***	-0.93***	0.26	0.71**	0.40**	0.03	-0.64	0.31**
2. DE Slope		0.04***	***-0.19***	>0.001	-0.03***	0.001	-0.02	-0.01
3. TU[a] Intercept			183.38***	-17.49***	--	--	--	--
4. TU[a] Slope				5.42***	--	--	--	--
5. AU[b] Intercept	36.25***	-0.94***			0.55***	-0.02**	--	--
6. AU[b] Slope		0.04***				0.001	--	--
7. MU[c] Intercept	36.19***	-0.94***					1.31**	0.25***
8. MU[c] Slope		0.04***						0.07***

Note: Unstandardized estimates of variance and covariance among latent curve parameters are reported here. As such, covariance estimates are not bounded between 0 and 1. Estimates among the parameters for DE changed across substance-specific models. Therefore, the variance and covariance estimates are reported in the rows corresponding to the substance-specific model.
[a]TU = Tobacco Use
[b]AU = Alcohol Use
[c]MU = Marijuana Use
*$p < 0.05$, **$p < 0.01$, ***$p < 0.001$

Table 8.
Variance and Covariance Matrix for Intercepts and Slopes across Unconditional Bivariate LCM-SR (Aim 2)

	1	2	3	4	5	6	7	8
1. DE Intercept	36.25***	-0.94***	3.05	0.36	***0.49***	0.02	-0.49	0.30*
2. DE Slope		0.04***	-0.19	0.01	-0.03	0.002	-0.01	-0.004
3. TU[a] Intercept			4.01	2.66*	--	--	--	--
4. TU[a] Slope				2.99***	--	--	--	--
5. AU[b] Intercept	36.28***	-0.95***			0.05	0.01	--	--
6. AU[b] Slope		0.04***				0.002	--	--
7. MU[c] Intercept	36.18***	-0.93***					0.20	***0.12***
8. MU[c] Slope		0.04***						0.14***

Note. Unstandardized estimates are reported here. As such, covariance estimates are not bounded between 0 and 1. Variance of the DE intercept parameter changed across substance-specific models. Therefore, the variance estimate is reported in the first row to indicate its corresponding model.
[a]TU = Tobacco Use
[b]AU = Alcohol Use
[c]MU = Marijuana Use
*$p < 0.05$, **$p < 0.01$, ***$p < 0.001$, marginal effects (p-value between 0.1 and 0.05) are bolded and italicized.

Table 9.
Variance and Covariance Matrix for Intercepts and Slopes across Conditional Bivariate LCM-SR (Aim 3)

	1	2	3	4	5	6	7	8
1. DE Intercept	36.98***	-1.01***	-3.75	1.07*	2.32	0.28	-0.70	***0.29***
2. DE Slope		0.05***	0.05	-0.03	-0.34	0.03	>0.000	-0.003
3. TU[a] Intercept			1.35	1.16	--	--	--	--
4. TU[a] Slope				3.29***	--	--	--	--
5. AU[b] Intercept	3739.67***	-111.87***			0.03	0.004	--	--
6. AU[b] Slope		8.37***				0.004*	--	--
7. MU[c] Intercept	36.93***	-1.01***					0.06	-0.05
8. MU[c] Slope		0.05***						0.20***

Note. Unstandardized estimates are reported here. As such, covariance estimates are not bounded between 0 and 1. Variance of the DE intercept parameter changed across substance-specific models. Therefore, the variance estimate is reported in the first row to indicate its corresponding model.
[a]TU = Tobacco Use
[b]AU = Alcohol Use
[c]MU = Marijuana Use
*$p < 0.05$, **$p < 0.01$, ***$p < 0.001$, marginal effects (p-value between 0.1 and 0.05) are bolded and italicized.

Table 10.

Mean, Regression Estimates, and Standard Errors across Tobacco Use Models

Between-Person Effects	Univariate Model M (SE)	Unconditional Bivariate LCM M (SE)	Unconditional Bivariate LCM-SR M (SE)	Conditional Bivariate LCM-SR M (SE)
DE intercept	-1.39 (0.17)***	-1.39 (0.17)***	-1.38 (0.17)***	-0.98 (0.29)***
DE slope	0.56 (0.01)***	0.56 (0.01)***	0.56 (0.01)***	0.54 (0.02)***
TU[a] intercept	2.96 (0.70)***	3.56 (0.63)***	2.95 (0.65)***	0.03 (0.89)
TU slope	0.83 (0.10)***	0.77 (0.09)***	0.82 (0.10)***	1.45 (0.14) ***
Within-Person Effects			B(SE)	B(SE)
DE Wave II on TU Wave I			-0.01 (0.01)	0.004 (0.01)
DE Wave III on TU Wave II			-0.01 (0.01)	0.004 (0.01)
TU Wave II on DE Wave I			-61.12 (59.03)	59.53 (68.62)
TU Wave III on DE Wave II			0.13 (0.17)	-0.05 (0.19)

Note. Results for the unconditional LCM and unconditional and conditional bivariate LCM-SRs are presented for models wherein the variance of the slope of DE was set to zero. Each of models terminated normally.

[a]TU = Tobacco Use

[b] Estimates are small due to limited residual variance in DE.

*$p < 0.05$, **$p < 0.01$, ***$p < 0.001$, marginal effects (p-value between 0.1 and 0.05) are bolded and italicized.

Table 11.

Mean, Regression Estimates, and Standard Errors across Alcohol Use Models

	Univariate Model	Unconditional Bivariate LCM	Unconditional Bivariate LCM-SR	Conditional Bivariate LCM-SR
Between-Person Effects	M (SE)	M (SE)	M (SE)	M (SE)
DE intercept	-1.39 (0.17)***	-1.39 (0.17)***	-1.39 (0.17)***	-10.24 (2.84)***
DE slope	0.56 (0.01)***	0.56 (0.01)***	0.56 (0.01)***	5.56 (0.20)***
AU[a] intercept	-0.23 (0.04)***	-0.22 (0.04)***	-0.23 (0.04)***	-0.74 (0.06)***
AU slope	0.07 (0.01)***	0.07 (0.004)***	0.07 (0.01)***	0.12 (0.01)***
Within-Person Effects			B(SE)	B(SE)
DE Wave II on AU Wave I			-0.05 (0.13)	-0.94 (1.78)
DE Wave III on AU Wave II			-0.17 (0.14)	-0.66 (1.79)
AU Wave II on DE Wave I			-4.31 (6.30)	-0.31 (0.43)
AU Wave III on DE Wave II			-0.01 (0.01)	0.0001 (0.001)

Note. Results for the unconditional and conditional bivariate LCM-SRs are presented for models wherein the variance of the slope of DE was set to zero. Each of models terminated normally.

[a]AU = Alcohol Use

*$p < 0.05$, **$p < 0.01$, ***$p < 0.001$, all effects with a p-value between 0.1 and 0.05 are bolded and italicized.

Table 12.
Mean, Regression Estimates, and Standard Errors across Marijuana Use Models

Between-Person Effects	Univariate Models M (SE)	Unconditional Bivariate LCM M (SE)	Unconditional Bivariate LCM-SR M (SE)	Conditional Bivariate LCM-SR M (SE)
DE intercept	-1.39 (0.17)***	-1.39 (0.17)***	-1.39 (0.17)***	4.83 (1.26)***
DE slope	0.56 (0.01)***	0.56 (0.01)***	0.56 (0.01)***	0.48 (0.06)***
MU[a] intercept	0.36 (0.26)	0.36 (0.26)	0.38 (0.27)	0.89 (1.19)
MU slope	0.15 (0.05)**	0.15 (0.05)**	0.14 (0.06)**	-0.06 (0.22)
Within-Person Effects			B(SE)	B(SE)
DE Wave II on MU Wave I			-0.02 (0.02)	-0.03 (0.03)
DE Wave III on MU Wave II			-0.02 (0.02)	-0.03 (0.03)
MU Wave II on DE Wave I			-11.69 (11.85)	-7.25 (8.43)
MU Wave III on DE Wave II			-0.04 (0.06)	0.003 (0.07)

Note. Results for the unconditional and conditional bivariate LCM-SRs are presented for models wherein the variance of the slope of DE was set to zero. Each of models terminated normally.
[a]MU = Marijuana Use
*$p < 0.05$, **$p < 0.01$, ***$p < 0.001$, all effects with a p-value between 0.1 and 0.05 are bolded and italicized.

Table 13.

Regression Estimates and Standard Errors of a Conditional Bivariate LCM-SR Model with DE and Tobacco Use

	DE intercept	DE slope	TU[a] intercept	TU[a] slope
TIC[b] Effects	B (SE)	B (SE)	B (SE)	B (SE)
Ethnicity[b]	-0.23 (0.48)	0.03 (0.02)	-1.29 (1.20)	***-0.39 (0.22)***
Black[b]	-1.32 (0.36)***	0.02 (0.02)	-4.00 (1.47)**	-0.59 (0.19)**
Asian[b]	-0.01 (0.72)	-0.04 (0.04)	-2.17 (2.15)	-0.46 (0.37)
Native American[b]	1.75 (1.26)	-0.04 (0.06)	-2.53 (7.68)	-0.75 (1.24)
Menarche	-0.53 (0.10)***	0.01 (0.01)	-0.40 (0.25)	-0.04 (0.04)
Hyperactivity/Impulsivity	0.82 (0.25)***	-0.03 (0.01)*	0.05 (0.56)	0.49 (0.11)***

	DE			TU[a]		
TVC[c] Effects	Wave I	Wave II	Wave III	Wave I	Wave II	Wave III
Delinquency	0.02 (0.02)	-0.03 (0.03)	0.02 (0.03)	0.65 (0.10)***	1.08 (0.11)***	1.07 (0.24)***
Depression	0.01 (0.01)	0.04 (0.01)**	0.04 (0.01)***	0.22 (0.05)***	0.21 (0.05)***	-0.01 (0.07)
Anxiety	0.01 (0.03)	0.07 (0.03)*		-0.02 (0.13)	0.03 (0.14)	

[a]TU = Tobacco Use

[b]Time-invariant covariates (TIC) included comparisons across ethnic and racial identities. Dummy codes were used such that Non-Hispanic and White identities were coded as 0. Identifying as Hispanic and/or Black, Asian, or Native American was coded as 1. Higher values for DE and tobacco use represent greater engagement in behaviors.

[c]Time-varying covariates (TVC) are reported such that each cell represents the regression parameter of concurrent syndromes. For example, the first row shows the regression parameter of DE at Wave I on Delinquency at Wave I, of DE at Wave II on Delinquency at Wave II, of DE at Wave III on Delinquency at Wave III, and so forth.

*$p < 0.05$, **$p < 0.01$, ***$p < 0.001$; marginal effects (p-value between 0.1 and 0.05) are bolded and italicized.

Table 14.

Regression Estimates and Standard Errors of a Conditional Bivariate LCM-SR Model with DE and Alcohol Use

	DE intercept	DE slope	AU intercept	AU slope
TIC[b] Effects	B (SE)	B (SE)	B (SE)	B (SE)
Ethnicity[b]	-2.49 (4.88)	0.32 (0.23)	-0.01 (0.08)	-0.03 (0.02)*
Black[b]	-13.63 (3.58)***	0.23 (0.17)	-0.19 (0.07)**	-0.04 (0.01)***
Asian[b]	-0.27 (7.26)	-0.34 (0.37)	0.12 (0.13)	-0.06 (0.02)**
Native American[b]	16.24 (12.46)	-0.37 (0.57)	-0.16 (0.18)	0.004 (0.03)
Menarche	-5.32 (1.03)***	0.08 (0.05)	-0.04 (0.02)*	0.01 (0.003)**
Hyperactivity/Impulsivity	7.97 (2.51)***	-0.30 (0.12)*	-0.01 (0.04)	***0.01 (0.01)***

	DE			AU[a]		
TVC[c] Effects	Wave I	Wave II	Wave III	Wave I	Wave II	Wave III
Delinquency	0.24 (0.24)	-0.28 (0.28)	0.14 (0.27)	0.14 (0.01)***	0.18 (0.01)***	0.15 (0.02)***
Depression	0.14 (0.12)	0.34 (0.12)**	0.33 (0.09)***	0.03 (0.004)***	0.03 (0.004)***	0.02 (0.01)***
Anxiety	0.14 (0.32)	0.78 (0.33)*		-0.01 (0.01)	-0.01 (0.01)	

[a]AU = Alcohol Use

[b]Time-invariant covariates (TIC) included comparisons across ethnic and racial identities. Dummy codes were used such that Non-Hispanic and White identities were coded as 0. Identifying as Hispanic and/or Black, Asian, or Native American was coded as 1. Higher values for DE and alcohol use represent greater engagement in behaviors.

[c]Time-varying covariates (TVC) are reported such that each cell represents the regression parameter of concurrent syndromes. For example, the first row shows the regression parameter of DE at Wave I on Delinquency at Wave I, of DE at Wave II on Delinquency at Wave II, of DE at Wave III on Delinquency at Wave III, and so forth.

*$p < 0.05$, **$p < 0.01$, ***$p < 0.001$; marginal effects (p-value between 0.1 and 0.05) are bolded and italicized.

Table 15.

Regression Estimates and Standard Errors of a Conditional Bivariate LCM-SR Model with DE and Marijuana Use

	DE intercept	DE slope	MU[a] intercept	MU[a] slope
TIC[b] Effects	B (SE)	B (SE)	B (SE)	B (SE)
Ethnicity[b]	-0.24 (0.49)	0.03 (0.02)	0.16 (0.33)	0.003 (0.08)
Black[b]	-1.34 (0.36)***	0.02 (0.02)	-0.36 (0.35)	-0.02 (0.06)
Asian[b]	-0.02 (0.73)	-0.03 (0.04)	-0.26 (0.92)	-0.10 (0.23)
Native American[b]	1.74 (1.25)	-0.04 (0.06)	-0.21 (1.09)	-0.12 (0.20)
Menarche	-0.53 (0.10)***	0.01 (0.01)	-0.09 (0.09)	0.02 (0.02)
Hyperactivity/Impulsivity	0.82 (0.25)***	-0.03 (0.01)*	-0.16 (0.22)	0.09 (0.04)*

	DE			MU[a]		
TVC[c] Effects	Wave I	Wave II	Wave III	Wave I	Wave II	Wave III
Delinquency	0.02 (0.02)	-0.03 (0.03)	0.04 (0.01)***	0.33 (0.04)***	0.49 (0.04)***	1.02 (0.08)***
Depression	0.02 (0.01)	0.04 (0.01)**	0.02 (0.03)	0.04 (0.02)**	0.06 (0.03)*	0.05 (0.03)
Anxiety	0.01 (0.03)	0.07 (0.03)*		-0.08 (0.05)	-0.13 (0.06)*	

[a]MU = Marijuana Use

[b]Time-invariant covariates (TIC) included comparisons across ethnic and racial identities. Dummy codes were used such that Non-Hispanic and White identities were coded as 0. Identifying as Hispanic and/or Black, Asian, or Native American was coded as 1. Higher values for DE and marijuana use represent greater engagement in behaviors.

[c]Time-varying covariates (TVC) are reported such that each cell represents the regression parameter of concurrent syndromes. For example, the first row shows the regression parameter of DE at Wave I on Delinquency at Wave I, of DE at Wave II on Delinquency at Wave II, of DE at Wave III on Delinquency at Wave III, and so forth.

*$p < 0.05$, **$p < 0.01$, ***$p < 0.001$; marginal effects (p-value between 0.1 and 0.05) are bolded and italicized.

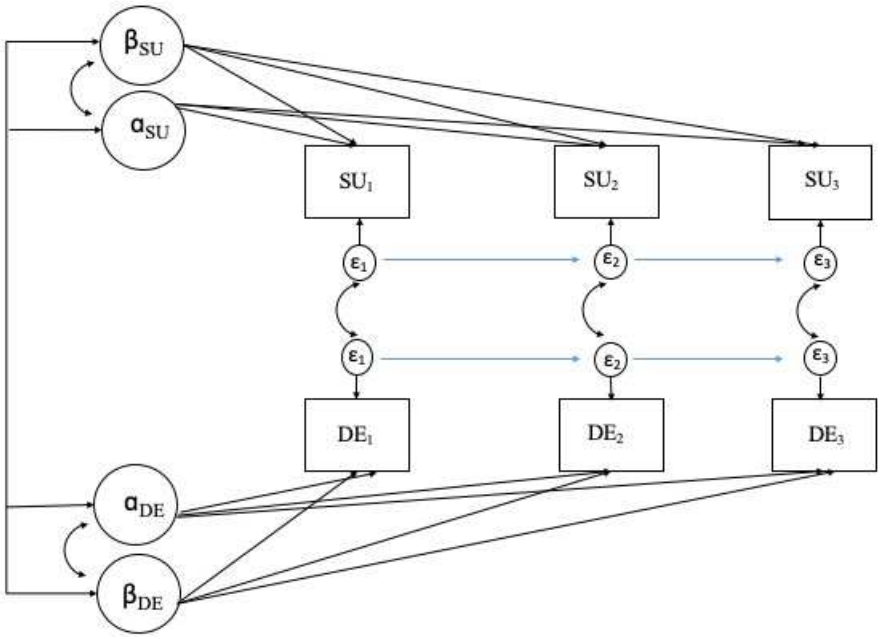

Figure 1. Conceptual Model of an Unconditional Bivariate LCM

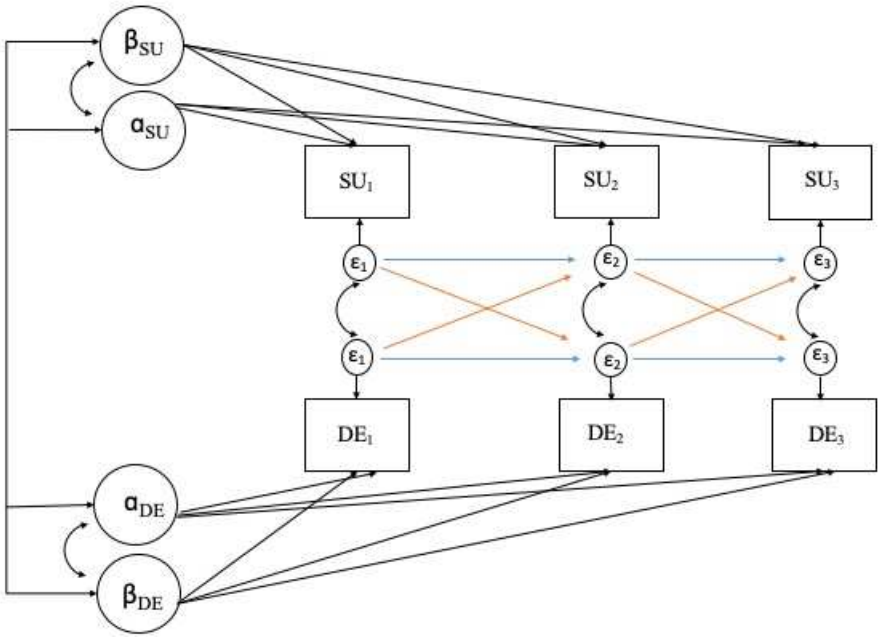

Figure 2. Conceptual Model of an Unconditional Bivariate LCM-SR

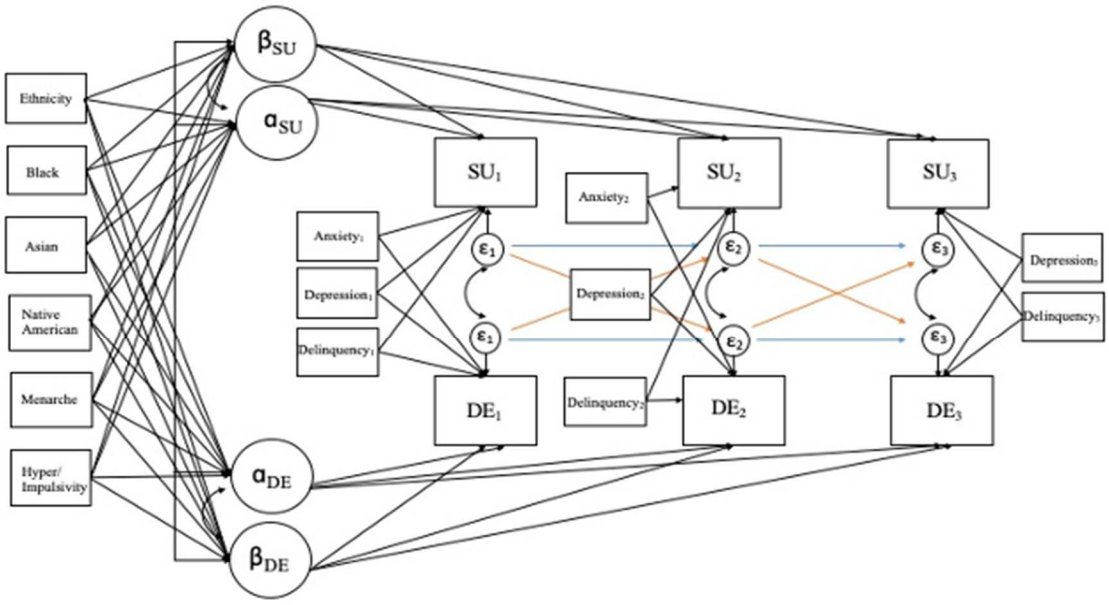

Figure 3. Conceptual Model of a Conditional Bivariate LCM-SR

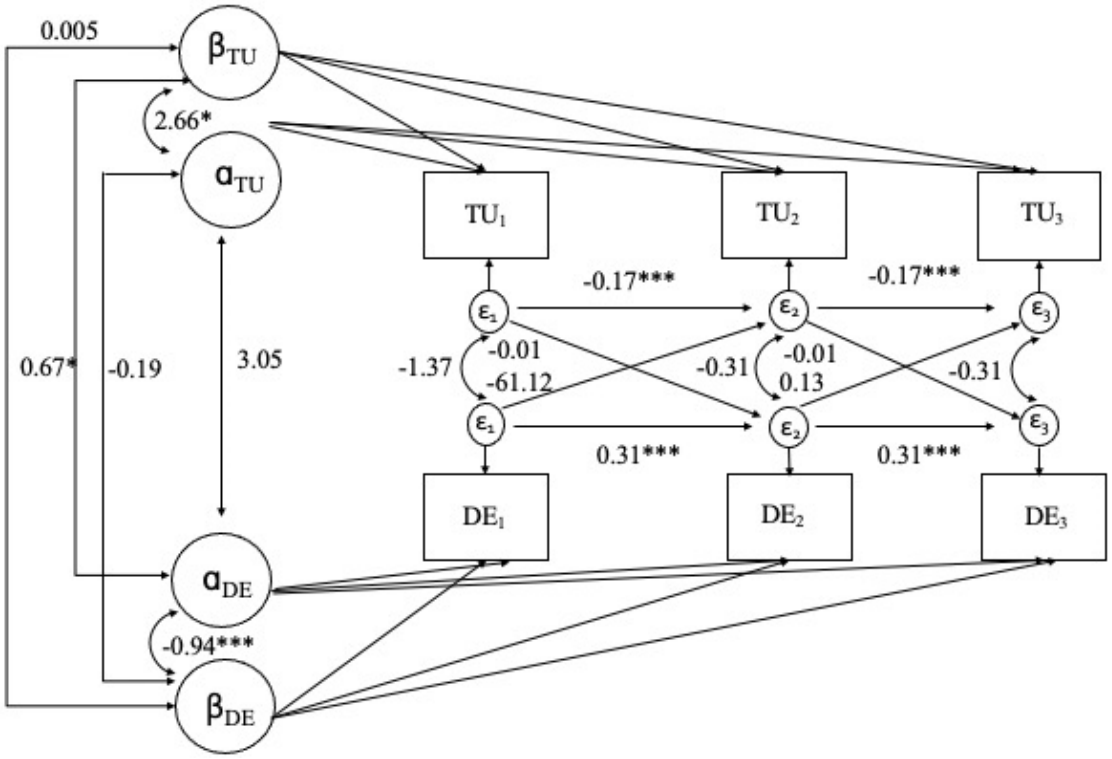

Figure 4. Unconditional Bivariate LCM-SR of Tobacco Use and Disordered Eating

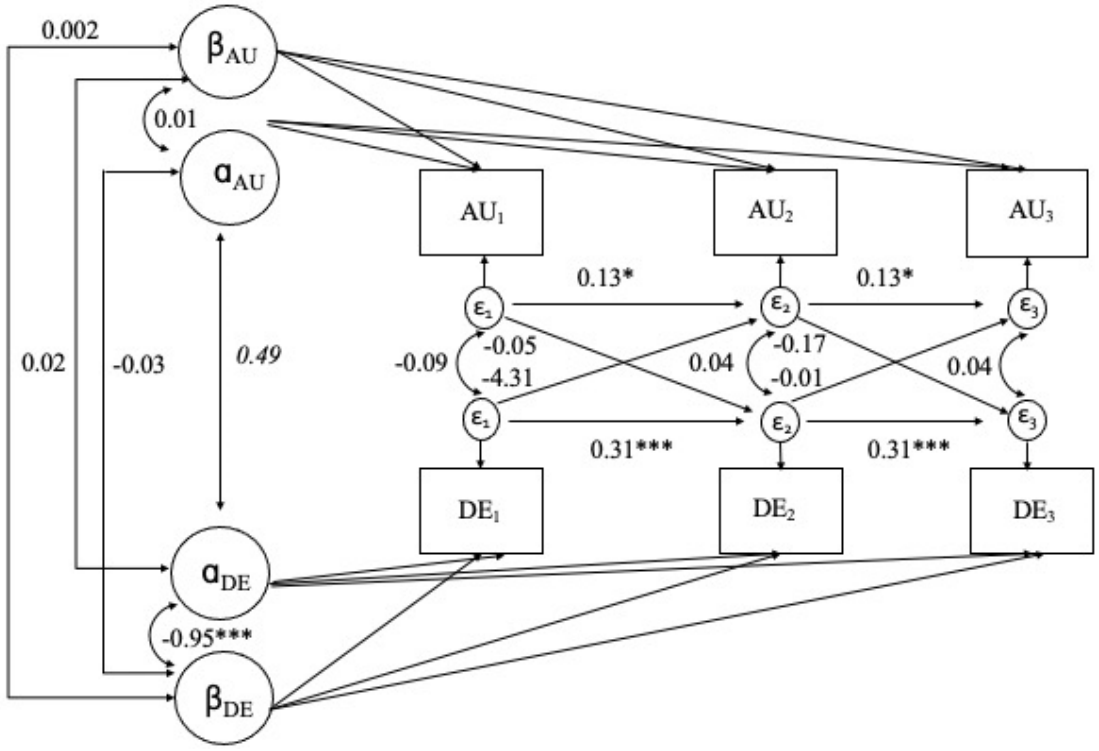

Figure 5. Unconditional Bivariate LCM-SR of Alcohol Use and Disordered Eating

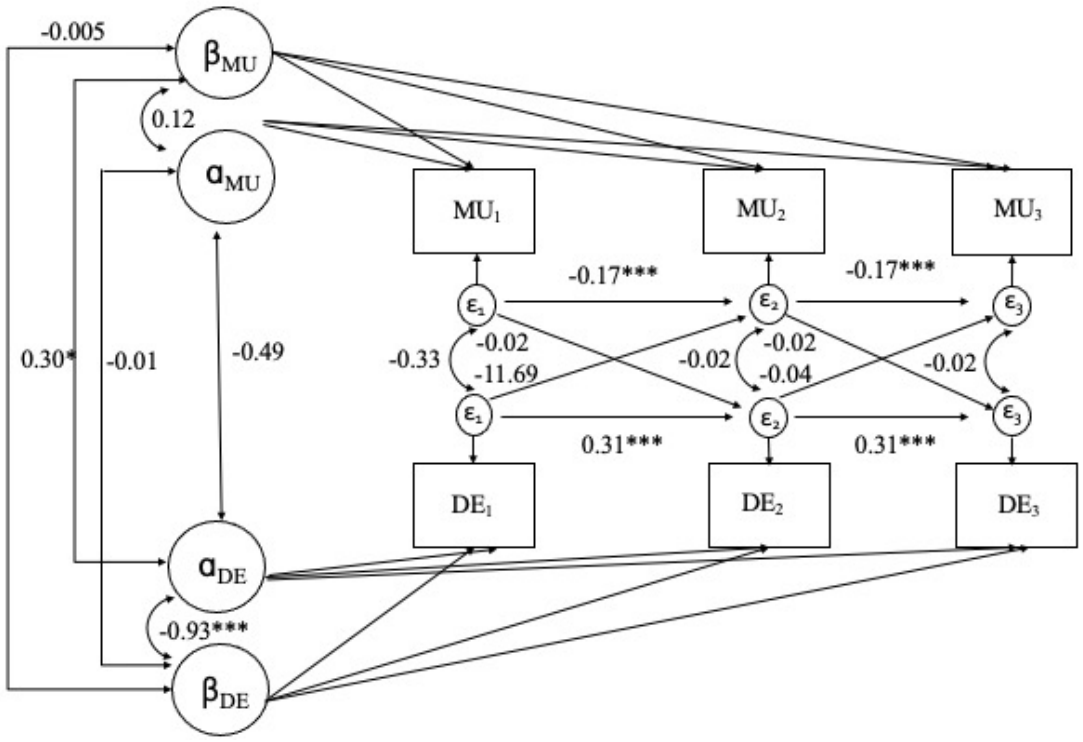

Figure 6. Unconditional Bivariate LCM-SR of Marijuana Use and Disordered Eating