Examining Stress-Related Pathways of Substance Use Among Sexual Minority Women

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Abstract

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Alcohol and drug abuse among sexual minority women (SMW) is an increasingly recognized public health concern in the United States. Across a multitude of nationally-representative samples, SMW populations report higher rates of substance use and disorder across the lifespan. SMW also report greater levels of adversity in adolescence (Friedman et al., 2011; Hughes & Eliason, 2002). Although minority stress models suggest that higher levels of adversity may explain mental health disparity among sexual minority populations (Hatzenbuehler, 2009; Meyer, 2003), few studies have longitudinally examined the impact of adversity among SMW in predicting substance use and disorder in later life. Moreover, none have examined psychological mediators such as emotion dysregulation that have previously explained greater internalizing disorder among SMW (e.g., Hatzenbuehler et al., 2008), and none have examined moderators of this developmental pathway.

The goal of this study was to examine the effect of adversity on co-developing emotion dysregulation and substance use among sexual minority and non-minority youth, and address moderators characterizing risk and protection for developing use. Data were drawn from 2,278 heterosexual and 173 sexual minority women who participated in the Pittsburgh Girls Study. The Pittsburgh Girls Study is a large, diverse sample of inner-city girls followed prospectively from age 5 to age 21, and contains a large longitudinal sample of SMW. Specifically, I tested whether greater adversity and rumination among sexual minority girls account for higher levels of substance use through young adulthood, and examined whether social support from peers and parents within adolescence buffers this proposed risk pathway. Missing data analysis, latent growth curve modeling, and multi-group structural equation modeling with structural invariance testing were used to test 1) whether sexual minority status predicted individual differences in level and change in rumination and substance use from adolescence through young adulthood; 2) whether rumination and adversity were direct and serial mediators explaining the relation between sexual minority status and developing substance use; and 3) whether relations between adversity, rumination, and substance use were moderated by sexual minority status and social support.

Results suggested that SMW reported greater marijuana use at age 20, though were no different from heterosexual peers in the level and change in rumination or alcohol use through young adulthood. Across sexual orientation groups, social stress predicted greater rumination in late adolescence and less decline through young adulthood, as well as greater marijuana use by age 20. Finally, I found evidence of indirect effects of sexual minority status on rumination and young adult marijuana use through social stress: though not a serial mediation process, SMW reported greater social stress in adolescence, which in turn predicted greater adolescent rumination, less decline in rumination, and greater marijuana use in young adulthood.

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Chapter 1. Introduction

1.1. Substance Use among Sexual Minority Women as a Public Health Concern

National surveys indicate that approximately 4 million women in the United States identify as lesbian or bisexual, comprising 3.4% of the entire adult U.S. population (Gates, 2011). In spite of this, research focus on psychopathology among sexual minority women (SMW) has received limited national interest to date, attributed largely to a history of institutional stigmatization as well as the declassification of homosexuality as a disorder itself occurring relatively recently within the past 40 years (Dean et al., 2000). As public health interest in lesbian, gay, and bisexual (LGB) psychopathology continues to grow, it has become increasingly evident that multiple forms of psychopathology, including substance use and disorder (SU/D), are particularly elevated among SMW (Meyer, 2003). However, funded studies investigating LGB substance use remains relatively limited to date; although only 0.1% of all NIH-funded studies have focused on LGB health, nearly 80% of these projects target HIV/AIDS research, with relatively minimal focus on illicit drug use (30.9%) or alcohol use (16.4%). Moreover, the overwhelming majority of LGB-related projects (86.1%) have focused on sexual minority men, with only 13.5% dedicated to SMW health (Coulter, Kenst, Bowen, Scout, & Scout, 2014). Study of SU/D among SMW is therefore a relatively novel and unexplored topic.

The majority of the LGB population does not meet criteria for substance-related disorder, though substantial evidence indicates that rates are elevated relative to heterosexual populations. In the United States, for instance, national surveys indicated that 16.1% of gay or lesbian and 19.5% of bisexual individuals meet DSM-IV criteria for alcohol dependence in the past year; 0.6% and 1.1% met criteria for marijuana dependence; and 3.2% and 5.1% met criteria for other drug dependence (McCabe, Hughes, & Bostwick, 2009), with these figures generally showing historical decline over time (Hughes & Eliason, 2002). Nonetheless, large and nationally representative surveys (e.g., McCabe et al., 2009) indicate that disparities between heterosexual and non-heterosexual populations may be more pronounced among lesbians than gay men (Marshal et al., 2008; McCabe & Hughes, 2005; Meyer, 2003) and may be especially prominent among bisexual women (Vrangalova & Savin-Williams, 2014). For instance, meta-analytic reviews (Meyer, 2003) and large, nationally-representative samples (Cochran, Ackerman, Mays, & Ross, 2004) suggest that homosexual men are approximately 1.5 times more likely and homosexual women 3.5 times more likely to have a lifetime occurrence of a substance use disorder relative to heterosexual populations, and that homosexual men have a 2.5-fold greater likelihood of at least one past-year symptom of dysfunctional drug use, with a nearly fourfold increase for women. Relative to heterosexual women (HW), lesbian women are also between two and four times as likely to report alcohol dependence; approximately eleven times as likely to report marijuana dependence; and are as high as twelve times as likely to report other drug dependence, with comparable figures among bisexual women (Marshal et al., 2008; McCabe & Hughes, 2005; Meyer, 2003).

Accumulating evidence suggests that adolescence and young adulthood may be particularly critical periods for the development of elevated SU/D among SMW. Across sexual orientation populations, substance use is typically first initiated by late adolescence (Eaton et al., 2010), with over half (54.1%) of Americans initiating some form of substance use by age 18 and over a third (37.4%) of high school seniors reporting past-month drinking (SAMHSA, 2014). Young adulthood (age 18-25) is also a developmental period during which problem and heavy use are at a lifetime peak (Chassin, Ritter, Trim, & King, 2003), and both early initiation and heavy use of substances through young adulthood are among the most robust predictors of lifecourse-persistent patterns of substance abuse and disorder across sexual orientations broadly (Chassin, Pitts, & Prost, 2002; McGue & Iacono, 2005) and among sexual minority populations specifically (Fish & Pasley, 2015; Needham, 2012) . Sociocultural factors such as the prevalence of bar culture among sexual minority communities (Green & Feinstein, 2012; Hughes & Eliason, 2002) may increase availability of substances and serve as sociocultural risk factors for substance-related disorder in adults; however, heightened substance use among LGB populations precedes the availability of these venues and first emerges in adolescence (Coker, Austin, & Schuster, 2010). Marshal and colleagues (2008), for instance, examined levels of substance use across 18 studies of LGB adolescents younger than age 18 and found that, similar to adult findings, the association between sexual orientation status and substance use was stronger among girls than boys and strongest among bisexuals, collectively indicating that LGB status conferred a 190% increase in the likelihood of initiating use across studies. Indeed, extant research indicates that SMW substance use disparities emerge during this developmental period (Needham, 2012) and either persist or increase (Marshal, Friedman, Stall, & Thompson, 2009) through young adulthood. Thus, this project addresses a robust yet largely unexplored public health concern by examining the development of elevated SU/D among SMW to inform how disparities emerge and persist through young adulthood.

1.2. The Role of Stress in Developing Substance Use among Sexual Minority Women

Sexual minority stress theories suggest that SMW may report higher rates of substance use disorder due to greater exposure to stressful life experiences (Hatzenbuehler, 2009; Meyer, 2003; Newcomb & Mustanski, 2010). Broadly, stress can be understood as adverse experiences with an environmental origin that are experienced as exceeding psychological or biological capacities (Grant et al., 2003) and have a damaging impact on an individual (Keyes, Hatzenbuehler, & Hasin, 2011). In the context of LGB stressors, these can range from events and conditions in the environment (termed distal stressors) to intrapersonal processes such as perceptions and appraisals that result from exposure to stigmatizing environments (termed proximal stressors). These include victimization, expectations of rejection, concealing one's sexual identity, internalized homophobia, and limited access to coping resources that are thought to induce stress and increase risk for both internalizing and substance-related psychopathology.

Given the predominant role of stress in sexual identity formation among LGB youth, minority stress theory may be extended to better understand risk in the early course of substance use among SMW. Initial sexual identity formation among LGB youth – that is, an internal recognition of one's own sexual orientation – begins approximately in early adolescence (between ages 8 and 11; Dubé & Savin-Williams, 1999) and is frequently characterized by numerous internal stressors prior to publically "coming out" (Carrion & Lock, 1997). These include confusion about one's identity, shame and fear regarding potential rejection from others, or denial of one's sexual identity that result in attempting to "pass" as heterosexual or failing to explore one's own sexuality. Although coming out can provide opportunity for navigating one's sexual identity with greater opportunities for social support (e.g., Wright & Perry, 2006), public identification of LGB status also confers greater risk of experiencing external stressors such as victimization and rejection from parents and peers (Legate, Ryan, & Weinstein, 2012). This is particularly salient in adolescence, during which sexuality-related victimization is at a peak across multiple contexts (Russell & Fish, 2015). In a meta-analysis, for instance, Friedman and colleagues (2011) found that sexual minority individuals were between 1.2 and 3.8 times more likely than heterosexual peers to have experienced physical or sexual abuse or experience victimization in a school environment. LGB youth are also more likely to get involved in fights (Faulkner & Cranston, 1998; Garofalo, Wolf, Kessel, Palfrey, & DuRant, 1998); skip school (Darwich, Hymel, & Waterhouse, 2012); be forced out of the home by caregivers due to their sexual identity (Cochran, Stewart, Ginzler, & Cauce, 2002; Fournier, 2009); and receive less acceptance and social support from caregivers than their same-age peers (Bouris et al., 2010; Ryan, Russell, Huebner, Diaz, & Sanchez, 2010), suggesting a breadth of exposure to external stressors spanning the home, peer, and school contexts. Together, these findings suggest that experiences of stressors within adolescence are heightened among LGB youth, and may serve an essential function in shaping substance use risk both within and beyond this developmental period.

Associations of social stress experiences with increased risk of SU/D are robust in both the general population (Grant et al., 2003) as well as among LGB groups (McCabe, Bostwick, Hughes, West, & Boyd, 2010). Recent national survey data, for instance, indicated that women who report two or more experiences of sexual abuse, physical abuse, or parental neglect prior to age 18 are between two and four times more likely to report alcohol dependence, drug abuse, and drug dependence relative to those reporting none (Hughes et al., 2010; McCabe et al., 2010). Among SMW, approximately 46% reporting multiple victimization experiences also report a past-year diagnosis of substance use disorder, a figure that is four times as high as SMW reporting no experiences of discrimination within the past year (McCabe et al., 2010). Moreover, meta-analytic findings suggest that higher levels of general social stress is posited as the strongest predictor of subsequent substance use among sexual minorities compared to other risk factors, including co-occurring internalizing and externalizing problems and reports of general distress (Goldbach, Tanner-Smith, Bagwell, & Dunlap, 2014). More recent research has also examined social stress as a prospective mediator of the relation between SMW status and substance use, indicating that these experiences in adolescence reduce the observed disparity in substance use outcomes (tobacco and alcohol use) between HW and SMW (Austin & Jun, 2008; McLaughlin, Hatzenbuehler, Xuan, &

Conron, 2012). Together, these findings indicate that social stress experiences predict greater SU/D, social stress is more frequent among SMW, and the association between sexual minority status and higher risk of SU/D may be explained by social stress-related processes that confer greater risk for SU/D.

1.3. Emotion Dysregulation as a Mechanism of Substance Use Risk

Current models of stress, adversity, and SU/D indicate that emotion dysregulation (ED) may be a principal mechanism linking SMW social stress in early life with development of SU/D, though research remains limited to date. ED has been conceptualized as difficulty in modulating emotions consciously or non-consciously in order to respond appropriately to demands in the environment (Aldao, Nolen-Hoeksema, & Schweizer, 2010). Although the precise definition of ED is an area of ongoing research (Eisenberg & Spinrad, 2004), current models suggest that ED involves poor emotional awareness and understanding of emotion; limited access to emotion coping resources and adaptive strategies; and engaging in maladaptive patterns of responding to emotion such as rumination (Gratz & Roemer, 2004), among other definitions. A meta-analysis, as well as subsequent research, suggested that these and related facets of ED are positively associated with SU/D (Aldao et al., 2010; Gross, 2007; Marlatt & Witkiewitz, 2004; Nolen-Hoeksema, Stice, Wade, & Bohon, 2007).

The link between stress and ED is an emerging topic in adversity research. Evidence indicates that stress experiences such as physical and sexual abuse are associated with ED in childhood (Kim & Cicchetti, 2010), and have been found to mediate relations between early stress and later psychopathology (Alink, Cicchetti, Kim, & Rogosch, 2009; Maughan & Cicchetti, 2002). Stress experiences such as abuse and victimization may affect neural circuitry involved in the experience of emotion that render individuals more reactive to later experiences and less able to engage in adaptive emotion regulation strategies (Sheridan & McLaughlin, 2014). Specifically, prolonged and/or acute activation of physiological stress systems in response to stress, such as the sympathetic nervous system and the hypothalamic-pituitary-adrenal cortical axis, may disrupt the longer-term structural integrity of brain regions implicated in affectivity as well as emotion regulation (Cohen, Kessler, & Gordon, 1995; Pechtel & Pizzagalli, 2011; Tops, Riese, Oldehinkel, Rijsdijk, & Ormel, 2008). The psychological impact of early social stress is therefore twofold. First, these experiences attenuate emotion regulation (Baumeister, Bratslavsky, Muraven, & Tice, 1998), decreasing the capacity to regulate effectively in response to environmental stressors. Second, this also creates vulnerability to the deleterious effects of subsequent stress due to increased reactivity to environmental stressors. The combination of these two processes may thus create a negative feedback loop (Chassin, Sher, Hussong, & Curran, 2013). In short, experiences of social stress may increase subsequent ED, rendering individuals more susceptible to subsequent stressors that in turn produces increases (or slower declines) in ED across adolescence and young adulthood. However, little work has explicitly tested whether social stress impacts change over time in ED.

Stress and its effect on growth in ED may in turn predict patterns of substance use behaviors through young adulthood. Though increases in substance use behaviors normatively increase through adolescence and peak in young adulthood (e.g., Grant et al., 2004), neurobiological theory suggests that individuals with histories of early stress may be particularly sensitized to the reinforcing properties of substance use within this period (Andersen & Teicher, 2009; Brady & Sinha, 2005; Enoch, 2011; Sapolsky, 2003). This is thought to result from stress-related increases in anhedonia and sensitization to substance-related cues (Chassin et al., 2013; Matthews & Robbins, 2003), as well as disruptions in developing brain regions associated with re-directing reward-driven behavior, such as the hippocampus and prefrontal cortex (Andersen & Teicher, 2008; Gogtay et al., 2006). The combination of greater anhedonia and diminished regulation of substance-motivated behavior may result in a "developmental cascade" in which greater sensitivity to the reinforcing effect of substance use progresses substance use more rapidly into heavier and disordered use over time (Andersen & Teicher, 2009). As such, both stress experiences and ED may characterize increased risk for a more rapid progression of SU/D from substance use initiation through young adulthood.

Evidence supporting ED as a mediator of LGB social stress and psychopathology is promising. In prospective studies of adolescents, McLaughlin, Herts, and colleagues (2009) found that ED mediated the relation between peer victimization and internalizing symptoms, and mediated the relation between stress and aggressive behaviors. Among LGB youth, Hatzenbuehler and colleagues (2008) found that sexual minority adolescents had higher ED than heterosexual peers, which in turn mediated the relation between minority status and internalizing symptoms. Related findings are reported in a daily diary of LGB adults (Hatzenbuehler, Nolen-Hoeksema, & Dovidio, 2009), in which rumination was a significant mediator in the daily relation between stigma-related stress on psychological distress. In the only study examining the mediating role of ED in sexual minority women, maladaptive coping strategies such as suppression and reactive coping mediated the relation between overt and perceived victimization and psychological distress (Szymanski & Henrichs-Beck, 2013).

These pathways may generalize to SU/D: vulnerable populations may develop SU/D over time by using substances to cope with distress (Hussong, Jones, Stein, Baucom, & Boeding, 2011), and some evidence has supported this theory among children of alcoholic parents (Chassin, Curran, Hussong, & Colder, 1996). This is particularly relevant in adolescence, during which individuals are especially sensitized to negative emotion (Silvers et al., 2012; Somerville, Jones, & Casey, 2010) relative to children and adults, yet are still maturing in their capacities to regulate emotional responses (Steinberg, 2010). Adolescent and young adult sensitivities may thus exacerbate pre-existing vulnerability to SU/D (Casey & Jones, 2010; Schulenberg & Maggs, 2002) that propel individuals toward greater SU/D through adulthood. However, only one cross-sectional study has addressed a related framework in the context of sexual minority social stress, reporting that negative affect mediated the effect of sexual minority-related victimization on substance use (Marshal et al., 2013).

Taken together, early stress predicts subsequent ED, and ED may be an indicator of mental health risk for sexual minority adolescents and adults. Moreover, ED may be a risk factor for later SU/D, particularly among SMW who have stress-related vulnerability in adolescence. ***Thus, the first aim of this proposal was to establish whether SM status was associated with level and change in ED through young adulthood, and whether this in turn was associated with developing substance use among SMW.*** However, no study to date has investigated whether social stress in youth affects growth in ED; whether social stress and ED impact trajectories of SU/D; and whether this developmental model is a mechanism explaining elevation in SU/D among SMW. ***Therefore, the second aim of this proposal was to then test social stress as a mechanism in these relations by examining whether the effect of social stress on ED mediates the relation between SM status and substance use.***

1.4. Social Support as a Moderator among Sexual Minority Women

In spite of recent appeals to address resilience within minority stress theory (Hatzenbuehler, 2009; Kwon, 2013; Riggle, Whitman, Olson, Rostosky, & Strong, 2008), research examining factors that moderate developmental pathways toward SMW SU/D remains lacking. A critical moderator proposed by a resilience model of LGB psychopathology (Kwon, 2013) is social support (Grant et al., 2006; House, Landis, & Umberson, 1988). Social support is thought to alter responses to stress by promoting effective coping strategies (Sheldon Cohen & Wills, 1985; Wethington & Kessler, 1986), as well as by buffering emotional and physiological reactivity to stress (Wills & Cleary, 1996). Although evidence of social support as a buffer is mixed for some forms of stress (Grant et al., 2006), studies examining social stress experiences such as violence (Ozer & Weinstein, 2004) and abuse (Luster & Small, 1997; Morrison & Clavenna-Valleroy, 1998) report significant protective effects among youth. Social support may be a particularly critical moderator for sexual minority mental health due to unique challenges posed by sexual identity formation. Because sexual minority status is both a concealable stigmatized identity (Quinn & Earnshaw, 2011) and an identity formed later in life relative to other minority identities (e.g., ethnic minorities; Meyer, 2003), sexual minorities may have less access to social support structures in adolescence due to stigma-related nondisclosure and isolation (Mak, Poon, Pun, & Cheung, 2007). As such, sexual minority individuals may be particularly vulnerable to social stress and ED when social support is low, while experiencing relatively greater buffering when social support is high.

To date, research examining social support as a buffer of substance use among SMW is limited, though findings reported in studies of LGB health more broadly provide support. Examined as a moderator, positive social support when disclosing sexual orientation reduced the effect of rejection experiences on substance use (Rosario, Schrimshaw, & Hunter, 2004); peer support buffered the relation between psychological distress and cigarette smoking (Rosario, Schrimshaw, & Hunter, 2011); and high sexuality-related support buffered the effect of sexuality stress on emotional distress (Doty, Willoughby, Lindahl, & Malik, 2010). In the direction of risk, maladaptive coping was associated with poor psychological well-being only when social support was also low (Griffin, Friend, Kaell, & Bennett, 2001). Taken together, social support theory and extant research in both the general population and LGB groups suggest that the impact of social stress and ED on SU/D may be moderated by social support among SMW. ***Thus, the third aim of this proposal is to explore whether the influence of adolescent emotion dysregulation and social stress on substance use is moderated by social support across heterosexual and sexual minority women.***

1.5. Aims and Hypotheses of Dissertation Study

The goal of this project is to address whether and how social stress experiences and ED among SMW explain change in substance use across development and greater substance use in young adulthood. Using data on 2,447 girls (8.3% SMW), I examined adolescent social stress and subsequent changes in ED as prospective indicators of substance use from late adolescence through young adulthood and explored how social support moderates these pathways. I examined these questions by addressing the following aims:

Aim 1: To examine the direct and indirect effects of sexual minority status and emotion dysregulation on the development of substance use.

I used parallel process latent growth curve modeling (PPGM; Cheong, MacKinnon, & Khoo, 2003) to assess the impact of sexual minority status and level and change in ED on the development of substance use through age 21. Before addressing substantive hypotheses, I specified unconditional latent growth curve models of ED and substance use to establish a model of linear change in each from age 17 to age 21. Then, I estimated a PPGM to determine whether age 17 ED and growth in ED through age 21 predicts age 21 substance use and growth in substance use through age 21. I then included sexual minority status as a predictor of these parallel processes to test whether level and change in both ED and substance use was greater among SMW. Finally, using the product of the coefficients approach to testing indirect effects (MacKinnon et al., 2007), I examined whether higher levels or more growth in substance use by age 21 among SMW was explained by level and change in ED.

Hypotheses for Aim 1: ED at age 17 and less age-related decline in ED will be associated with change in substance use from age 17 to 21 and higher levels of substance use by age 21. Sexual minority women will report higher age 17 ED and age 21 substance use, less age-related decline in ED, and more growth in substance use through age 21. Elevations in level and growth in substance use among SMW will be explained by level and growth in ED.

Aim 2: To test emotion dysregulation and social stress as mediators of the effect of SMW status on substance use.

I then built on the model proposed in Aim 1 to examine the influence of social stress among SMW on the development of ED and substance use through young adulthood. I used tests of serial mediation (Taylor, Mackinnon, & Tein, 2008) to determine whether social stress experiences explain the the relations specified in Aim 1.

Hypotheses for Aim 2: SM status will be associated with greater social stress at age 17. Social stress will predict higher ED at age 17, higher substance use by age 21, less decline in ED from age 17 to age 21, and steeper increases in substance use from age 17 to age 21. The observed relation between sexual minority status and substance use will be explained by the effect of adolescent social stress on level and changes in ED.

Aim 3: To explore whether the effects of adolescent emotion dysregulation and social stress on substance use is moderated by social support across heterosexual and sexual minority women.

Using the model specified in Aim 2, I aimed to test latent interactions of social support with ED intercept and adolescent social stress in predicting change in substance use and substance use at age 21 using the latent moderated structural equation (LMS; Klein & Moosbrugger, 2000) approach. Given the complexity of this model, I expected that this was unlikely to produce a reliable solution. Thus, if this model failed to converge, I planned to specify a simplified multi-group structural equation model using manifest variables at single time points. Treating SM status as a grouping variable, I aimed to examine the indirect effect of social stress at age 17 on substance use at age 21 through ED, social support, and interactions of social support with ED and social stress at age 18. I then aimed to use structural invariance testing (Vandenberg & Lance, 2000) to examine whether these indirect effects were stronger among SMW than HW (i.e., moderated mediation; Preacher, Rucker, & Hayes, 2007; Edwards & Lambert, 2007).

Hypothesis for Aim 3: High social support will buffer the effect of ED and adolescent social stress on substance use among both HW and SMW, and will enhance these effects when social support is low. The moderating effects of social support on ED and social stress will be stronger among SMW than HW.

1.6. Innovation and Public Health Impact

Although SU/D among SMW is an increasingly recognized public health disparity, there has been minimal research targeting this topic due to methodological challenges in the recruitment and longitudinal assessment of SMW. The proposed project addresses these limitations by conducting secondary data analysis on a longitudinal dataset spanning from adolescence (age 16) to young adulthood (age 21) with a large and diverse sexual minority and non-minority sample. Moreover, this project is one of few epidemiological studies of SU/D measuring co-developing psychological risk factors (e.g., ED) and is uniquely poised to address psychological mediation within minority stress theory. Thus, this study is an exceptionally rare opportunity for addressing developmental pathways toward SU/D among an at-risk group that will critically inform prevention and intervention of SU/D broadly, and particularly among SMW.

Chapter 2. Methodology

2.1. Research Design

This study used existing data from the Pittsburgh Girls Study (PGS), a large, longitudinal, community-based sample of inner-city girls designed to assess the development of conduct disorders, delinquency, and co-occurring problematic disorders from early childhood to young adulthood (Keenan, Hipwell, Chung, et al., 2010). The PGS is funded by the National Institute of Mental Health (R01 MH56630; Rolf Loeber, PI), the National Institute on Drug Abuse (R01 DA012237; Rolf Loeber & Tammy Chung, PIs), as well as funding from the FISA foundation and the Falk Fund. At the first time point, four age cohorts were recruited and ranged from ages of 5 and 8, and data were collected annually through age 21 in the oldest cohort. I conducted secondary data analysis using 10 waves of data, examining sexual minority status (age 16) and social support (age 17) in late adolescence, adversity experiences in childhood (age 12) through late adolescence (age 17), and levels and growth in ED and substance use from late adolescence (age 17) through young adulthood (age 21). A total of 2,447 girls were in the sample at the initial time point, and 144 girls reported sexual minority status at age 16. Approximately half the girls were African American (52%), 41% were European American, and the remaining girls were described as multiracial or a separate race. Low-income neighborhoods were oversampled with approximately 40% receiving public aid at wave 1. Approximately 47% of parents in the sample reported 12 or fewer years of education. Retention rates for the study were high across annual time points, ranging from 85.4% to 97.2%. Annual interviews were conducted in the home and were completed by trained interviewers using a laptop computer. The current study included measures of sexual orientation, childhood and adolescent social stress, ED, social support, and substance use. Nearly all measures were adapted from previously existing measures used in prior studies.

2.2. Method and Measures

Sexual Minority Status. Reports of sexual orientation were assessed using the following question: “Do you consider yourself to be (a) Heterosexual or straight; (b) Gay or lesbian; or (c) bisexual?”, asked annually beginning at age 16. Given the relatively small number of lesbian (n = 31) and bisexual (n = 113) respondents, these groups were combined to form a single SMW group (e.g., Marshal et al., 2013).

Social stress. Social stress was measured as a composite index derived from four domains measured through age 17: physical/sexual abuse, peer victimization, discriminatory experiences, and parental abuse.

Physical or sexual abuse within the past year was measured using a single dichotomous item developed by the PGS research staff. Participants were coded as “yes” on this measure if they responded affirmative to a question of whether they experienced physical or sexual abuse in the past year during an annual in-person interview conducted by PGS staff (Keenan et al., 2014). This measure was collected annually from age 12 to age 17. Due to the relative dearth of reported abuse in the sample for any given wave of data collection (between 3.7%, n = 82 at age 12 and 1.1%, n = 22 at age 17), I combined these measures across waves to reflect whether participants reported any abuse history prior to age 17 (9.2%, n = 207).

Reports of victimization by peers were measured using the Peer Experience Questionnaire (PEQ; Vernberg, Jacobs, & Hershberger, 1999) at age 17. This 9-item scale includes items measuring victimization by verbal aggression, confrontive physical aggression, and ostracism or relational aggression, rated on a scale ranging from never to a few times per week. Reliability for this scale in the PGS is high (α = .82; Keenan, Hipwell, & Feng, 2010).

Parental abuse was assessed using the child-report version of the 19-item Conflict Tactics Scale – Parent/Child Version (CTS, Murray Straus, 1979) which measures the level of child-directed harsh parenting practices from the parent. Subscales of this measure include nonviolent discipline, psychological aggression, and physical assault. Straus and colleagues (Straus, 1998) report adequate discriminant and construct validity on all subscales, and adequate reliability coefficients are reported in the PGS (α = .73-.75; Olino, Stepp, Keenan, Loeber, & Hipwell, 2014).

Discrimination at age 17 was measured using the Everyday Discrimination Scale (EDS; Williams & Yu, 1997). The EDS is a 9-item measure of chronic and routine experiences of unfair treatment, such as being treated with less respect than others. The EDS is computed as a sum, and reliability for this scale is high (α = .88; Williams & Yu, 1997).

Emotion Dysregulation. Annual measures of ED from age 17 to age 21 were collected using the Difficulties in Emotion Regulation Scale (DERS; Gratz & Roemer, 2004) and the rumination subscale of the Perfectionism Inventory (Hill, Huelsman, & Furr, 2004). The DERS is designed to assess clinically-relevant difficulties in emotion regulation. DERS subscales available in the present study are the awareness and understanding of emotions subscale (6 items; α = .80) and the emotion regulation strategies subscale (8 items, α = .88; Gratz & Roemer, 2004). The 7-item rumination subscale from the Perfectionism Inventory measures intrusive worrying about past errors, imperfect performance, or future mistakes, and has adequate reliability (α = .87; Hill et al., 2004). Administration of the DERS began in the final two waves of data collection and was therefore collected on two of the four study cohorts. Administration of the rumination subscale was completed across all study cohorts.

Substance Use. Nicotine, alcohol, and illegal substance use were assessed annually from age 17 to age 21 using the Nicotine, Alcohol and Drug Substance Use scale (NADU), adapted from an instrument of the Rutgers Health and Human Services Project (Pandina, Labouvie, & White, 1984). This scale contains items measuring use frequency of each substance within the past year, assessed on an 8-point scale ranging from 0 ('I did not within the past year) to 7 ('every day or more than once a day').

Social Support. Social support at age 17 was measured using the Inventory of Parent and Peer Attachment (IPPA; Armsden & Greenberg, 1987). The IPPA is a 75 item scale measuring the quality of adolescents' relationships with their close friends, mother, and father. Reliability for the IPPA subscales is high, ranging from .73 to .9l (Armsden & Greenberg, 1987). Social support ratings of mother, father, and peers were combined as a single measure in the Pittsburgh Girls Study.

Chapter 3: Data Analysis Plan

3.1. Handling Missing Data

Data were collected using an accelerated cohort design, which is one of several planned missingness designs (Graham, Taylor, Olchowski, & Cumsille, 2006; Little & Rhemtulla, 2013; Mistler & Enders, 2011). Specifically, four separate cohorts were followed over the course of the study, and each were followed for a maximum of four annual assessments. This resulted in missingness patterns ranging from 14.20% missing (age 18 rumination) to 62.60% missing (age 20 alcohol use) in the current study. Patterns of missingness for key study variables are provided in Table 2.

I considered two methods used to handle missingness in the data (Enders, 2010): full information maximum likelihood (FIML) and multiple imputation (MI). FIML is a method that derives parameter estimates using only the full observed data, such that missing observations are "skipped" when computing a maximum likelihood solution. FIML makes the assumption that observed data are continuous and normally distributed; if this assumption holds and data are not systematically missing, ignoring missingness will not result in bias in the parameter estimates, and will be less biased and more efficient than other ad hoc missing data techniques such as pair-wise and list-wise deletion (Enders, 2001).

In contrast, MI uses a regression-based approach to estimate missing values observed in the data. Multiple imputation proceeded in three steps: imputation, analysis, and pooling. In the imputation stage, each incomplete variable is regressed on all other variables with observed data, which produces a set of parameters that best approximates the values of missingness given the data. The parameters produced by this model are then used to generate predicted values for missing observations in the dataset, with error, and this process is repeated for all missing cells in the dataset. This is conducted repeatedly and iteratively using a Bayesian approach (Markov Chains; Li, 1988) in which a new set of estimates is generated using the previous set as a prior to "update" the parameter values used in imputation. This aids in reducing bias in each parameter due to error in estimating any single set of parameters. After some number of iterations of this procedure (called a burn-in phase), sets of parameter values will ideally stabilize around the true sample parameters, and the parameter estimates are considered stationary. Thereafter, the imputation process continues generating complete data, though versions of the complete data are saved intermittently across iterations for use in analysis. This is repeated until a large number of datasets have been generated and saved (typically 20 or more), which are then used in analysis. During the analysis phase, researchers run their models of interest across all imputed datasets, and store the parameter estimates and standard errors produced by each model. Finally, in the pooling phase, these results are combined to produce a single set of results.

Of these two approaches, multiple imputation was a more optimal approach than FIML for several reasons. First, FIML estimation is considered inefficient, meaning that the standard errors produced by this procedure will be disproportionately large relative to using maximum likelihood with complete data and will consequently reduce power to detect true effects (Enders, 2017). Second, FIML assumes that the variables within the model are multivariate normally-distributed; although FIML and multiple imputation will yield nearly identical estimates and standard errors in the case that this assumption is held (Collins, Schafer, & Kam, 2001; Gelman et al., 2014; Schafer, 2003), this is not the case in my model of interest. Given that my data include variables that are categorical, ordinal, or (as a set) a mixture of distributions (for instance, sexual minority status is categorical and my substance use outcomes are ordinal), using FIML may result in bias to both my estimates and standard errors. Third, I proposed a multi-group structural equation model in Aim 3 of my analyses, which requires a fully-observed grouping variable for estimation. Given 390 observations in my grouping variable (Sexual minority status at age 16) was missing on this measure, this would reduce my observed sample size by 16% when using FIML, consequently reducing my power. Finally, although FIML's application of maximum likelihood estimation makes handling missing values relatively easy, I would not be able to use more rigorous estimation procedures, such as diagonally weighted least squares (DWLS), that rely on complete datasets and are asymptotically distribution-free (that is, no distributional form of the variables is assumed). This is crucial given the skewed and ordinal-distributed nature of my substance use variables, which frequently cause non-normality and heteroscedasticity in residuals when using unweighted estimators (Flora & Curran, 2004). Therefore, at the outset, I aimed to draw substantive conclusions from my results using a multiple imputation approach.

Despite these advantages, multiple imputation only produces valid results if the imputation model is specified in a sensible manner. For instance, researchers must consider and properly specify the distributions of included variables during imputation, consider the functional form of the outcome variable(s), include all relevant predictors during the imputation phase, and provide diagnostic evidence to help ensure that estimates are reliable (Nguyen, Carlin, & Lee, 2017; Sterne et al., 2009). Although I addressed most of these limitations my using an advanced approach to imputation (Enders, 2017; Enders, Mistler, & Keller, 2016), I was unable to estimate my imputations in an optimal manner (described in the next section), and the few diagnostic approaches available did not provide definitive guidance. Therefore, I conducted all analyses using both multiple imputation and FIML approaches as a means of confirming and replicating the effects I observed in my data. Where my solutions were inconsistent across approaches, I note them throughout my results and provide justification for one approach or the other given my observed data.

3.1.1. Imputation Procedure

I imputed datasets using the Blimp software package (Enders, 2017; Enders, Mistler, & Keller, 2016). Although other imputation software and packages are available (e.g., MICE, van Buuren & Groothuis-Oudshoorn, 2011), Blimp provides functionality for imputing data with more complicated distributional forms, such as nominal or ordinal data, and accommodates imputation for multilevel data. In the present context, race and minority status were specified as nominal variables, and I aimed to estimate my models treating substance use as ordered categorical variables (e.g., Agresti, 2012, though see caveats later). All other measures were imputed as continuous variables. Although my data were multilevel (i.e., observations clustered within people), the time interval between assessments was equivalent across participants (i.e., annual assessments). When the intervals between assessment occasions is identical for all participants, modelling the data as a two-level multilevel structure is computationally equivalent to a single-level latent growth curve model in a structural equation modelling framework (Chou, Bentler, & Pentz, 1998; Hox & Stoel, 2005; Mehta & West, 2000); therefore, I imputed my data using a single-level design because this imposed no constraints in specifying the functional form of growth in the model and simplified the imputation procedure considerably (Enders, 2017). Finally, to facilitate the inclusion of tests for moderation by social support, I used the transform-then-impute approach for interaction terms detailed by von Hippel (2009) and Enders, Baraldi, & Cham (2014). Namely, I computed raw interaction terms between rumination and social support as well as social support and social stress prior to imputation, and included these in the imputation procedure to ensure these effects would not be biased toward zero in the analysis phase (Enders, 2010; Enders & Gottschall, 2011; Graham, 2009; Schafer, 1997).

I diagnosed my imputation results in three ways. My first and primary method was by conducting imputations on two Markov chains simultaneously in order to compute a potential scale reduction factor (PSR; Gelman & Rubin, 1992) as a means diagnosing the convergence of the estimation procedure. PSRs assess convergence by comparing the estimated between-chains and within-chain variances for each model parameter in order to determine whether both chains are producing similar means and variances for a given parameter (i.e., a posterior distribution). In essence, the PSR factor reflects a ratio of a given parameter estimate's between and within variance across the chains. Letting $\hat{\theta}_m$ be the parameter mean for the *m*th chain and $\hat{\theta}$ the mean of the parameter across chains, the between-chain variance is computed as follows:

$$B = \frac{N}{M-1}\sum_{m=1}^{M}(\hat{\theta}_m - \hat{\theta})^2,$$

where M is the number of chains created (here, 2) and *N* is the chain length.

Letting $\hat{\sigma}^2{}_m$ be the parameter variance of the *m*th chain, the within-chain variance is computed as

$$W = \frac{1}{M}\sum_{m=1}^{M}\hat{\sigma}^2{}_m.$$

Given quantities *B* and *W*, the pooled variance $\hat{V}$ is computed as

$$\hat{V} = \frac{N-1}{N}W + \frac{M+1}{MN}B,$$

and the PSR is defined as the ratio of $\hat{V}$ to W, or

$$PSR = \frac{\hat{V}}{W}.$$

That is, if there is as much total variance as within-chain variance, there is no between-chain variance (i.e., this ratio is 1) and the variance for a given parameter have converged on the same posterior distribution for both chains. PSRs are computed during the burn-in phase after every 100 imputations for each chain. Blimp computes PSR values for all parameters in the model, including the coefficient estimates, the residual variance, and the threshold parameters for ordinal measures (if applicable), and provides users with the maximum PSR value of the variables imputed. Values between 1.05 and 1.10 or lower are considered acceptable (Enders, 2017).

I took two additional steps to ensure reliability of my MI results. Namely, I created density plots of the observed versus imputed values of my key study variables to check whether they had an appropriate distributional form (Azur, Stuart, Frangakis, & Leaf, 2011). Marked discrepancies between density functions were indications of bias and/or inefficiency produced in my imputed variables. As a final check of my parameter estimates, I also estimated all of my models using multiple imputation, FIML, and strictly the observed data to ensure that my parameters were sensible in my results. I expected that my estimates would be generally similar across all approaches, with some bias toward zero when using a FIML estimator for prediction of my substance use outcomes. Also, given prior literature, I suspected that standard errors will be largest and estimates most biased in the observed data; I also expected FIML may produce smaller (albeit biased) standard errors relative to multiple imputation with DWLS given FIML will apply less stringent corrections to the residual error terms (Enders, 2001). Where marked discrepancies were observed, I examined by observed data and selected the solution that provided the most sensible fit.

3.1.2. Imputation results

I imputed a total of 20 datasets. I conducted imputations using the Gibbs sampler imputation algorithm, and I included both study variables and auxiliary variables with complete data (e.g., cohort and race) or near-complete data (e.g., social support and harsh parenting at age 16) to improve the accuracy and efficiency of the imputation procedure. I estimated 20,000 iterations prior to saving the first set of imputed values (i.e., the burn-in rate) to increase confidence that my estimates were stationary. I specified the between-imputation interval to 10,000, meaning that once the burn-in phase ended, imputed datasets were saved after every 10,000 iterations.

I explored a variety of combinations of my key study variables in order to arrive at an imputation solution that converged given my data. However, I ultimately needed to trim a set of key study variables in order to achieve this because the inclusion of such variables resulted in non-positive definite solutions. Namely, I was unable to include alcohol and marijuana use at age 21, likely due to the very high proportion of missingness (78.3%). Including emotion regulation strategies and emotional awareness in any time point also produced non-positive definite solutions for the same reason, as missingness for these study variables ranged from 55.70% missing (age 19) to 80% missing (age 17 and age 21). Though suboptimal, I therefore continued with my analyses using alcohol and marijuana use from ages 18 to age 20, and proceeded using rumination from age 17 to age 20 as my sole indicator of emotion regulation.

Using this set of variables, I was able to estimate my imputed datasets treating substance use as an ordered categorical variable. However, with this specification, my fixed effects and threshold parameters were well above the recommended range (Max PSR = 5.247), suggesting that the imputation solution was unreliable (Figure 1a). I then explored a specification assuming my substance use variables were normally-distributed in order to achieve a reliable solution. This specification was suboptimal because the imputation procedure would produce imputed values of my substance use variables on a ratio scale, which prevents me from estimating an ordered categorical model for these outcomes (though I was still able to specify abuse and sexual minority status as categorical). Moreover, this introduced the potential for bias in my estimates because the imputed values produced could be unlikely or even nonsensical given the data (for instance, negative values and impossible values in-between ordered categories; Sterne et al, 2009). However, using this approach would also simplify my imputation procedure given I was no longer estimating threshold parameters, and would increase the chances of producing a more reliable set of datasets. I therefore imputed a new set of datasets with a continuous normal specification for my substance use variables. My maximum PSR value at the final iteration of this imputation approach was 1.004, which was well within the acceptable range (see also Figure 1b). However, I noted some discrepancies between observed and imputed values in my substance use variables (e.g., Figure 2), such that a number of observations were below the observed data. This was especially true for alcohol use at age 20, which I expected was because missingness constituted a larger proportion of data at that assessments relative to other periods (62.6% missing). All other imputed variables appeared similar to the observed data (see Figures 3 and 4). I therefore proceeded using the results of my normal specification, bearing in mind that some of my estimates in my alcohol use variables may have considerable bias toward zero.

3.1.3. Analysis and Pooling Procedure

All analyses were conducted in R (R Core Team, 2017) using the lavaan package for structural equation models (Rossell, 2012). Because I had sets of imputed datasets, I was able to estimate structural equation models using diagonally weighted least squares (DWLS) estimation, which provides more accurate parameter estimation and is more robust to variable type (e.g., continuous versus ordered or non-ordered categories) and non-normality compared to standard maximum likelihood estimation (Mîndrilă, 2010). DWLS is also more optimal than weighted least squares (WLS), as it better accommodates small sample sizes, large models, and skewed and ordinal data (Schumacker & Beyerlein, 2009; Rhemtulla, Brosseau-Laird, & Savalei, 2012). In essence, this approach accommodates models that violate the assumption of homoscedasticity in the residuals by weighting individual cases based on their error variance in a model. For instance, an observation with small error variance is weighted heavily in a model since it provides more information to the model parameters, whereas an observation with large error variance is given relatively less weight. It was critical to use this approach given the ordinal and non-normally distributed nature of my observed substance use variables. Specifically, the fact that these data were truncated at zero with positive skew will result in residuals that are non-normal and heteroscedastic using a standard maximum likelihood estimator. This will in turn bias my standard errors and corresponding confidence intervals (Aiken, Mistler, Coxe, & West, 2015).

I conducted all analyses in parallel across imputed datasets using an R function I created in-house. The function estimates a given structural equation model across a set of datasets and pools the parameter estimates, standard errors, and fit indices produced by these models. I pooled parameter estimates by computing arithmetic means of these values across datasets (e.g., Baraldi & Enders, 2010), and I used Rubin's rules (Rubin, 1987) to compute the standard errors for each pooled parameter estimate. Pooling of the standard errors proceeded as follows (e.g., Marshal, Altman, Holder, & Royster, 2009). First, letting $\widehat{U}_m$ be the parameter estimate for the *m*th dataset, I computed the average within-imputation variance of a particular parameter, $\overline{U}$, as

$$\overline{U} = \frac{1}{m}\sum_{i=1}^{m} \widehat{U}_m.$$

Then, I computed the between-imputation variance, B, as

$$B = \frac{1}{m-1}\sum_{i=1}^{m}(\widehat{U}_m - \overline{U})^2.$$

Then, I computed the total pooled variance, T, as

$$T = \overline{U} + \left(1 + \frac{1}{m}\right)B.$$

Finally, I took the square root of this value to determine the standard error for a given parameter estimate.

3.2. Principal component Analysis

I combined my social stress measures using principal components analysis (PCA; Smith, 2002). PCA is a factor analytic technique aimed at reducing dimensions of variance among a set of variables. Given k variables, PCA produces a set of k components, and each component represents a single dimension of variance (termed an eigenvector) that explains some proportion of the total variance shared among the variables (represented by an eigenvalue). Whereas other factor analytic approaches such as confirmatory factor analysis aim to establish an adequate measurement model for one or more latent variables thought to produce a set of observed variables, the goal of PCA is instead to produce a set of optimally-weighted index variables (termed components) that minimizes the loss of information provided by the variances of the observed variables (e.g., Espejo et al., 2007). Given that my social stress measures represented disparate aspects of adverse experiences, this approach was a more optimal method of combining my social stress variables compared to other common approaches, such as derived mean or sum scores, because each measure of social stress may contribute differentially to a latent dimension of social stress. All PCA analyses were conducted using the FactoMineR (Lê, Josse, & Husson, 2008) and missMDA (Josse & Husson, 2016) packages in R in conjunction with my in-house analysis and pooling functions.

I conducted PCAs using abuse, peer victimization, harsh parenting, and everyday discrimination measured at age 17, and I computed these in parallel in the observed dataset and across all imputed datasets. I evaluated my results as follows. First, I examined the eigenvalues for each eigenvector, as well as the proportion of variance each eigenvector explained in the total shared variance among predictors. Ideally, a PCA will produce one eigenvector that is considerably larger than the others (termed the principal component) that explains the majority of the shared variance in my variables. Then, I examined the correlations of each of my variables with each eigenvector to determine whether any single variable was unrepresentative (or conversely, solely representative) of a given eigenvector. If a variable did not contribute to the principal component, I then removed it from analyses and repeated the above steps. Given an optimal solution based on the criteria above, I then used the standardized principal component(s) as manifest indicator(s) of social stress in my sample. The PCA results were nearly identical across datasets, including in the observed data (e.g., Tables 5 and 6); therefore, in the subsequent sections, I report my PCA results below using mean averages across imputed datasets.

Principal component Analysis Results

I began my analyses by including all four social stress measures (harsh punishment, peer victimization, everyday discrimination, and abuse through age 17). Eigenvalues for each eigenvector are provided as a scree plot in figure 5a. The largest eigenvalue for a given eigenvector was 1.710, which constituted 42.64% of the total shared variance among the predictors. Whereas harsh parenting (r =.531), peer victimization (r = .805) and every discrimination (r = .832) were correlated strongly and positively with this eigenvector, abuse correlated relatively modestly (r = .292). In contrast, the next-highest eigenvector had an eigenvalue of 0.973 and compromised 24.33% of the total variance, was very strongly correlated with abuse (r = .923), and correlated minimally with the remaining predictors (see Table 5 and Figure 6a). These results suggested that harsh punishment, peer victimization, and everyday discrimination may be adequately summarized along a single dimension of variance, yet abuse may constitute a separate dimension of adverse life experiences.

Given these results, I estimated a separate PCA using only harsh punishment, peer victimization, and everyday discrimination at age 17 (that is, removing abuse). Figure 5b provides a scree plot for the eigenvalues generated by this analysis. The eigenvector with the largest eigenvalue was nearly as large as my four-measure PCA above (1.669) and constituted 55.63% of the shared variance among the predictors; moreover, similar to the solution generated when abuse was included, all three predictors correlated strongly and positively with this eigenvector (harsh parenting correlated with this dimension at .548, peer victimization at .816, and everyday discrimination at .838). This suggested that the largest eigenvector adequately described variation in each predictor with little to no loss in each variable's correlation with this component when abuse is removed. Although the second-highest eigenvector correlated strongly with harsh punishment (r = .834), this vector only captured 28.53% of the total shared variance, and was negatively correlated with peer victimization (r = -.332) and everyday discrimination (r = -.220). Given that harsh punishment was also moderately correlated with the first eigenvector and I had no sensible reasoning for what this eigenvector might represent, I selected only the principal component from this solution for use in my analyses.

Taken together, my PCA results suggested that harsh punishment, peer victimization, and everyday discrimination were adequately summarized by a single principal component. Results also suggested that abuse was minimally correlated with this principal component, and may instead represent a distinct social stress dimension. As such, in all subsequent analyses, I specified models where my abuse measure and my standardized principal component represented by harsh punishment, peer victimization, and everyday discrimination were treated as distinct predictors in my models.

3.3. Testing Primary Hypotheses

3.3.1. Parallel Process Growth Curve Modeling

To address my hypotheses for Aims 1 and 2 of the current project, I used parallel process growth curve modelling (PPGM; Singer & Willett, 2003; Cheong, MacKinnon, & Khoo, 2003; Preacher, Wichman, MacCallum, & Briggs, 2008) to examine the direct and indirect effects of sexual minority status and social stress on co-occurring level and change in rumination and substance use. I tested these models in a series of steps. First, I estimated unconditional latent growth curve models for both my rumination and substance use variables to determine whether a linear specification of growth adequately described whether and how each changed over time. Second, I estimated a PPGM regressing my slope and intercept at age 20 for substance use variables on the slope and intercept at age 17 for rumination to determine whether level and change in rumination was associated with level and change in substance use. That is, factor loadings for my growth rate factors were specified as [1 1 0] for substance use outcomes and [0 1 1 1] for rumination. Third, I included sexual minority status at age 16 as an exogenous predictor to establish whether there were differences across sexual orientation groups in the level and growth in rumination and substance use; at this step, I also tested the indirect effects of sexual minority status on substance use through both the slope and intercept of rumination. Finally, to test for serial mediation of these effects by social stress, I added both social stress measures (abuse and my stress composite) at age 17 as a mediator between sexual minority status and each of my latent growth parameters. In models at this step, I set my intercept of rumination to age 18 (i.e., growth rate factor loadings set to [1 0 1 1]) to ensure that my indirect effects reflected an appropriate temporal sequencing (that is, social stress at age 17 predicting rumination at age 18). I used diagonally weighted least squares (DWLS) for all of my multiple imputation models, and used maximum likelihood with robust standard errors (MLR) when using a FIML approach. In all models where sexual minority status was included, I included race as a covariate to control for confounding effects of racial minority identity. I provide my final conceptual model in Figure 7.

I evaluated model fit using the adjusted chi-square difference test, where a non-significant result was an indication of adequate model fit. When a chi-square test was significant for a given model, I supplemented this test with a number of alternative fit indices indices (Chen, 2007; Cheung & Rensvold, 2002; Meade et al., 2008), including the comparative fit index (CFI), the Tucker-Lewis index (TLI), the root mean square error of approximation (RMSEA), and the standardized root-mean square residual (SRMR). Recommended cutoffs for these fit indices were CFI values greater than 0.95, TLI values greater than 0.95, RMSEA values less than 0.06, and SRMR values less than 0.08 (Chen, 2007; Hu & Bentler, 1999; Yu, 2002). When evaluating multiple imputation models, I report the worst fits among my datasets (i.e., lowest observed CFI and TLI values and highest observed RMSEA and and SRMR values).

3.3.2. Mediation

To test my two-path and three-path (Taylor, Mackinnon, & Tein, 2008) mediated effects, I used the product-of-the-coefficients method (Mackinnon et al., 2002), in which the mediated effect is estimated by multiplying path coefficients together. Because the distributions of these product terms are non-normal, the statistical significance of this term cannot be determined by dividing the estimate by the standard error of this term. Therefore, I used the distribution of the product approach (Mackinnon et al., 2007) to compute an asymmetric confidence interval for each two-path and three-path mediated effect. These analyses were carried out using the Rmediation package (Tofighi & Mackinnon, 2011). Due to the large number of mediation tests I conducted, I used a more stringent 99% confidence interval estimate to correct for the inflated Type I Error rate. I treated significance at the 95% level as marginal evidence of an effect.

3.3.3. Moderation

I tested moderation by social support and sexual minority status in my Aim 3 analyses in a multi-group structural equation modeling (SEM) framework. Although I aimed extend my final model from Aims 1 and 2 to examine moderation, I expected this model would be difficult to estimate. If this proved to be the case, I aimed to test a simplified model across groups, provided in Figure 8. I used nested model comparisons (Raftery, 1995) as well as structural invariance testing (Cheung & Rensvold, 2002; Schmitt & Kuljanin, 2008; Vandenberg, Vandenberg, Lance, & Lance, 2016) to determine support for specific moderation hypotheses. For all model fit comparisons using multiple imputation results, I used pooled fit measures when conducting analyses.

I conducted analyses in a series of steps. First, I established a baseline model by estimating a SEM omitting my grouping variable (SM status) and any interaction terms (rumination*social support and social stress*social support); ideally, this would be identical to the final model I specified in my Aim 2 analyses with the SM status predictor removed. Using this model, I then included my social support interaction terms sequentially and compared likelihood-based fit indicators for nested models to determine support for moderation. I used likelihood ratio tests and changes in Bayesian Information Criteria (BIC) to assess improvement in fit. Namely, significance of the likelihood ratio tests in the direction of improvement and BIC improvements of 6 or greater (Raftery, 1995) provided justification for including each interaction term. If the interaction term(s) did not significantly improve model fit, I omitted them from the model in all subsequent analyses in favor of model parsimony.

I then proceeded to test for moderation of path coefficients and indirect effects by SM status using structural invariance testing following guidelines by Wang and Wang (2012). I conducted invariance tests in a series of steps. First, I tested whether the structure of this model fit well for both SMW and HW by estimating an identical SEM for both groups in a multi-group SEM. Model parameters were estimated freely across groups at this step to ensure that any non-invariance observed in subsequent steps was not a product of poor fit of the baseline model for a particular group (from measurement invariance testing literature, this is also termed configural invariance; Cheung & Rensvold, 2002; Schmitt & Kuljanin, 2008; Vandenberg et al., 2016); if model fit did not worsen relative to my baseline model, this indicated that the same structure of my model was appropriate for both groups and I could continue with invariance testing. To examine differences in individual path coefficients and indirect effects across SM groups, I then tested for partial invariance by fixing parameters of interest to equivalence across groups and examining change in model fit. Significant worsening of model fit via reductions in chi-squared difference tests was an indicator that a particular parameter differed among sexual minority versus heterosexual women (i.e., moderation of a coefficient by sexual minority status). Given these analyses were exploratory and I conducted a large number of these tests (12 total), I used a more stringent 99% confidence interval estimate for all tests of moderation by sexual minority status to correct for the inflated Type I Error rate, and treated significance at the 95% level as marginal evidence of an effect. As a final step, I then conducted a sequence of omnibus structural invariance tests of all path coefficients, intercepts, and residuals to determine whether and how structural parameters differed across sexual minority groups. These tests helped determine which parameters should remain freely estimated before describing results for each group.

Although I used a DWLS estimator for all multiple imputation models in aims 2 and 3 analyses, I used the MLR estimator for testing moderation hypotheses. This is because the DWLS estimator is least-squares based and does not use maximum likelihood-based estimation to produce parameter estimates; therefore, log-likelihood values and all fit measures that rely on this measure (i.e., AIC and BIC) are not produced in these models. Once I arrived at a final model, I re-estimated my model using the DWLS estimator to best accommodate heteroscedasticity and non-normality in my residual terms as a product of using categorical variables (described earlier). I cross-referenced these values with those obtained using MLR, as well as with those obtained using FIML and the observed data, to ensure this made no substantive changes to my results (they did not).

Chapter 4. Results

4.1. Assessing Growth in Rumination, Alcohol Use, and Marijuana Use

Results for all unconditional growth models are summarized in Table 10, and detailed below.

4.1.1. Rumination

Across all imputed datasets, my unconditional rumination growth models fit well (See table 7). Namely, though chi-square tests were significant in 18 of my 20 models, no fit indicator dropped below adequate fit across imputed datasets (the worst fits observed were RMSEA=0.053, CFI=0.989, TLI=0.99, SRMR=.010). I found no serious differences in my estimated parameters using the MI versus FIML approach (e.g., see Figure 9), and therefore proceeded with interpreting my MI-derived results.

The estimated level of rumination at age 17 was 2.577 (95% CI = 2.530, 2.624), with rumination ranging from 1 to 5 in the observed data. The significance of the fixed slope term indicated that rumination increased by .045 units each year from age 17 to age 20 (95% CI = 0.004, 0.085, ß = 0.291). A significant variance component of the intercept (ψ_{11} = 0.62, 95% CI = [0.546, 0.691]) indicated that approximately 68% of women (within 1 SD of the mean) reported rumination scores between 1.790 and 3.363. Moreover, there was variation in the degree to which rumination changed through age 20 (ψ_{22} = 0.024, 95% CI = 0.002, 0.045), as well as nonzero covariance between the intercept and slope of rumination (ψ_{21} = -0.043, 95% CI = [-0.078, -0.007], r = -0.355): those with higher levels of rumination at age 17 exhibited less growth through age 20. I plotted the growth parameter at 1 SD above the mean, at the mean, and 1 SD above the mean, as well as predicted values according to low, mean, and high slope values based on computations of the correlation between intercept and slope (see Figure 10). These findings indicated that at average levels of rumination at age 17, women increased by 0.169 units in rumination per year, that women with relatively higher levels of rumination reported almost no change over time (-0.026 units per year), and that those who were lower in rumination tended to increase more quickly over time (0.36 units per year).

4.1.2. Alcohol Use

In my multiple imputation model, although my CFI and TLI fit indicators suggested excellent fit for all datasets, 8 out of my 20 datasets yielded a RMSEA value above the suggested cutoff, and the RMSEA estimates produced for my models were markedly more variable relative to my rumination and marijuana use models (see Table 8). Moreover, I noticed a marked discrepancy in the estimate of my alcohol use slope when comparing my MI results to my FIML results (see Figure 11). Specifically, the estimate of the slope of alcohol use using my multiple imputation approach was significant and negative (ψ_{22} = -0.122, 95% CI = [-0.216, -0.028], ß = -0.212), whereas this value was significant and positive using my FIML approach (described below). Given the observed means (e.g., Table 3) showed a moderate pattern of increasing use over time (at least among heterosexual women) and that these values corresponded well with my FIML estimate, I used the results of my FIML alcohol use model, which demonstrated excellent fit ($\chi^2(1)$ = 3.611, p = 0.057). I proceeded to use my FIML solution in all subsequent alcohol use analyses.

The fixed slope term indicated that alcohol use increased by 0.169 units per year from age 18 to age 20 (95% CI = [0.111, 0.227], ß = 0.260), though there was substantial variation in the estimated individual growth trajectories (ψ_{22} = 0.423, 95% CI = [0.228, 0.618]); women one standard deviation below the mean of the average slope declined in their use by 0.481 units per year, whereas women one standard deviation above the mean increased by 0.819 units per year.

At age 18, the estimated level of use was 2.586 (95% CI = 2.510, 2.661). Given the ordered categories for the alcohol use item, this suggested that use at age 18 was between "(2) less than 5 drinks per year" and "(3) more than 5 drinks per year, but less than once a month". A significant variance component of the intercept at age 18 (ψ_{11} = 1.381, 95% CI = [1.066, 1.696]) indicated that approximately 68% of women (within 1 SD of the mean) reported alcohol use scores between 1.410 and 3.761 at this age. There was also nonzero and negative covariance between the intercept and slope of alcohol use (ψ_{21} = -.229, 95% CI = [-.433, -.025], ß = -.300), suggesting that those who drank at higher levels use at age 18 exhibited less steep increases in use through age 20. Figure 12 depicts this effect: those who were heavier drinkers at age 18 maintained their level of drinking (-0.026 unit change per year), those at mean levels increased modestly (0.169 unit change per year), and those who drank less tended to increase their drinking more each year (0.364 unit change per year). Although those who were low in use at age 18 were "catching up" to those who started at higher levels of use, those who began drinking at high levels continued reporting the highest levels of drinking by age 20. I then changed my intercept to reflect the level of alcohol use at age 20; at this age, the estimated level of use was 2.92 (95% CI = [2.83, 3.01]), with significant variation in use at this age (ψ_{11} = 2.157, 95% CI = [1.788, 2.525]), where approximately 68% of women reported alcohol use scores between 1.451 and 4.389 at age 20. Taken together, these findings suggest that, on average, women were drinking initiated by age 18, increased their use through age 20, and exhibited more between-person variability in their use at age 20 compared to age 18.

4.1.3. Marijuana Use

Across all imputed datasets, no chi-square test statistic was significant, indicating that the unconditional model exhibited excellent fit (See table 9). I found no marked differences in my estimated parameters using the MI versus FIML approach (e.g., see Figure 13), and therefore report the MI results below.

The fixed slope term indicated that marijuana use increased by 0.177 units from age 18 to age 20 ([95% CI = 0.047, 0.306], ß = 0.246), though there was substantial variation in the degree to which marijuana use changed through age 20 (ψ_{22} = 0.515, 95% CI = 0.175, 0.854); women one standard deviation below the mean of the average slope declined in their use by 0.541 units, whereas women one standard deviation above the mean increased by 0.894 units per year. At age 18, the estimated level of use was 2.060 (95% CI = 1.971, 2.149), indicating approximately "less than 5" instances of marijuana use per year. A significant variance component of the intercept (ψ_{11} = 2.787, 95% CI = [2.183, 3.391]) indicated that approximately 68% of women reported marijuana use scores between 0.391 and 3.729 at age 18. There was no significant correlation between the slope and age 18 intercept (ψ_{21} = -0.271, 95% CI = [-0.662, .120], *r* = -0.223; see also Figure 14). At age 20, the estimated level of use was 2.4131 (95% CI = 2.201, 2.625) with significant variance in this intercept value (ψ_{11} = 3.762, 95% CI = [3.117, 4.406]): approximately 68% of women (within 1 SD of the mean) reported marijuana use scores between 0.474 and 4.353 at age 20.

4.2. Predicting Growth in Rumination and Substance Use from Sexual Minority Status

4.2.1. Co-Development of Rumination, Alcohol Use, and Marijuana Use

I next specified my parallel process growth curve model examining the relation between level and change in rumination and level and change in alcohol and marijuana use through age 20. The FIML model specified for rumination and alcohol use demonstrated excellent fit ($\chi^2(14)$= 18.362, p = .191). My parallel process model for rumination and marijuana use also fit well for all datasets: though chi-square tests were significant in all but two of my models, worst fit values were

RMSEA = 0.033, CFI =0.991, TLI = .987, and SRMR = 0.025 across imputed datasets (see Table 12).

Results are summarized in Table 11 and Figure 15 for alcohol use and Table 13 and Figure 16 for marijuana use. Rumination at age 17 was modestly associated with age 20 alcohol use (*b* = 0.157, 95% CI = [0.003, 0.311], ß = 0.081) and age 20 marijuana use (*b* = 0.264, 95% CI = [0.083, 0.446], ß = 0.107), though no other effects were significant. Rumination at age 17 was unrelated to growth in alcohol use (*b* = 0.014, 95% CI = [-0.090, 0.118], ß = 0.016) or growth in marijuana use (*b* = -0.008, 95% CI = [-0.097, 0.081], ß = -0.009). Change in rumination was not associated with level (*b* = 1.258, 95% CI = [-1.667, 4.184], ß = 0.100) or change (*b* = 1.239, 95% CI = [-1.143, 3.620], ß = 0.223) in alcohol use, nor marijuana use level (*b* = 2.804, 95% CI = [-0.559, 6.166], ß = 0.217) or change in marijuana use (*b* = 0.273, 95% CI = [-1.293, 1.841], ß =0.062).

4.2.2. Sexual Minority Status as an Exogenous Predictor

I then regressed each of my PPGM models above on sexual minority status. I included race as a covariate throughout. Model fit remained excellent for my alcohol use model ($\chi^2(24)$=32.36, p = 0.078). Though chi-squared tests were significant in all but two marijuana use models, remaining fit indicators suggested excellent fit with the inclusion of SM status: worst fits across datasets were RMSEA of 0.029, CFI of 0.990, TLI of 0.996, and SRMR of 0.022 (see Table 15).

Results for my alcohol use model are summarized in Table 14 and Figure 17. I found no differences between sexual minority groups in the level and change in rumination or alcohol use. Sexual minority status at age 16 was not associated with level of rumination at age 17 (*b* = 0.084, 95% CI = [-0.079, 0.247], ß = 0.029), and was also unrelated to growth in rumination through age 20 (*b* = 0.027, 95% CI = [-0.045, 0.099], ß =0.132). There were also no observed differences between sexual minority groups in the level of alcohol use at age 20 (*b* = 0.043, 95% CI = -0.852, 0.939), ß = 0.007), nor were there differences in the growth of alcohol use over time (*b* = -0.193, 95% CI = [-0.982, 0.595], ß = -0.076). Tests of indirect effects were also non-significant: there was no evidence of an indirect effect of sexual minority status on age 20 alcohol use through the slope (*ab* = 0.157, 99% CI = [-1.562, 2.446]) or age 17 level (*ab* = 0.011, 99% CI = [-0.023, 0.065]) of rumination, nor was there evidence of indirect effects on alcohol use growth (slope: *ab* = -0.001 99% CI = [-0.012, 0.007], intercept: *ab* = -0.003, 99% CI = [-0.029, 0.015]). White women reported higher alcohol use at age 20 (*b* = 0.939, 95% CI = [0.475, 1.404], ß = 0.314), though no other effects of racial identity were significant.

Results for my marijuana use model are summarized in Table 17 and Figure 18. Though sexual minority status was unrelated to alcohol use, SM women reported greater marijuana use at age 20 by a factor of about one category (*b* = 0.904, 95% CI = [0.258, 1.552], ß = 0.119). However, no other effects were significant. As with my alcohol use model, my marijuana use model suggested that sexual minority status was not associated with age 17 rumination level (*b* = 0.064, 95% CI = [-0.092, 0.219], ß = 0.021) or change in rumination over time (*b* = 0.026, 95% CI = [-0.081, 0.133], ß = 0.041). Status was also unrelated to growth in marijuana use over time (*b* = -0.054, 95% CI = [-0.420, 0.312], ß = -0.019). Tests of indirect effects were also non-significant. There was no evidence of an indirect effect of sexual minority status on age 20 marijuana use through the slope (*ab* = 0.069, 95% CI = [-0.437, 0.700]) or age 17 level (*ab* = -0.016, 95% CI = [-0.040, 0.086]) of rumination, nor was there evidence of indirect effects on marijuana use growth (slope: *ab* = 0.007, 95% CI = [-0.159, 0208], intercept: *ab* = -0.000, 95% CI = [-0.017, 0.015]). No effects of racial identity were significant.

4.2.3. Interim Summary

Rumination, alcohol use, and marijuana use increased over time, with those reporting low levels of rumination and alcohol use increasing more rapidly from late adolescence through early adulthood, and those with high levels maintaining steady (and higher) levels through adulthood (Table 10). Although there were significant individual differences in the levels and rates of change of rumination, alcohol use, and marijuana use, sexual minority status only explained individual differences in marijuana use by age 20: assuming a ratio scale of my marijuana use variable, lesbian and bisexual women reported nearly one category higher in marijuana use by age 20 relative to heterosexual women, plus or minus half a category. Finally, inconsistent with my hypotheses, level and change in rumination were not intervening processes explaining the relation between sexual minority status and level and change in alcohol or marijuana use.

4.3. Examining Adversity as a Mediator

To address my Aim 2 hypotheses, I estimated my parallel process growth curve model treating sexual minority status as my exogenous predictor of both measures of adversity (social stress composite and abuse history), the slope and intercept (at age 18) of rumination, and the slope and intercept (at age 20) of my substance use measures. This model was an excellent fit for my alcohol use outcome ($\chi^2(29) = 40.18$, $p = 0.081$). Model fit was also excellent across all datasets in my marijuana use model (see Table 19). RMSEA was .027 and SRMR was 0.019 at highest, and CFI was 0.990 and TLI was 0.979 at lowest despite significant chi-square tests in all but two models.

Results are summarized in Table 18 and Figure 19 for alcohol use and Table 20 and Figure 20 for marijuana use.

4.3.1. Direct Effects of Social Stress and Abuse

Sexual Minority Status and Rumination

In my alcohol use model, sexual minority status was associated with a 0.517 standard deviation increase in social stress at age 17 (95% CI = [.305, .730], ß = .132), as well as a modest yet significant association with abuse history (*b* = 0.095, ß = 0.087, 95% CI = [0.034, 0.155]). Social stress was associated with higher rumination at age 18 (*b* = 0.240, 95% CI = [0.203, 0.277], ß = 0.327) and slower increases in rumination over time (*b* = -0.025, 95% CI = [-0.042, -0.007], ß = -0.204). Abuse history did not predict greater age 18 rumination (*b* = -0.031, 95% CI = [-0.149, 0.086], ß = -0.012) or change in rumination (*b* = 0.028, 95% CI = [-0.031, 0.089], ß = 0.066).

In my marijuana use models, relations between social stress, sexual minority status, and rumination were consistent with those I observed in my alcohol use model. I found that sexual minority status was associated with a 0.562 standard deviation increase in social stress at age 17 (95% CI = [0.310, 0.814], ß = .144), and was again modestly associated with abuse history (b=0.093, 95% CI = [0.030, 0.155], ß = .085). Social stress was also associated with both higher age 18 rumination (*b* = 0.272, 95% CI = [0.232, 0.312], ß =0.365) and slower increases in rumination over time (*b* = -0.029, 95% CI = [-0.055, -.004], ß = -0.196). Abuse history was unrelated to age 18 rumination (*b* = -0.046, 95% CI = [-0.147, 0.054], ß = 0.017) and growth in rumination over time (*b* = 0.013, 95% CI = [-0.065, 0.090], ß = 0.023).

Substance use

Social stress was unrelated to alcohol use at age 20 (*b* = 0.087, 95 CI = [-0.034, 0.208], ß = 0.059) or change in alcohol use over time (*b* = -0.039, 95 CI = [-0.117, 0.040], ß = -0.060). Abuse was unrelated to age 20 alcohol use (*b* = 0.154, 95 CI = [-0.188, 0.497], ß = -0.029) or growth in alcohol use (*b* = 0.059, 95 CI = [-0.165, 0.283], ß = 0.025).

Though unrelated to alcohol use, social stress was associated with greater marijuana use by age 20 (*b* = 0.253, 95% CI = [0.056, 0.450], ß = 0.131). No other effects were significant: social stress did not predict marijuana use growth (*b* = -0.073, 95% CI = [-0.179, 0.033], ß = -0.102), and abuse history was unrelated to age 20 marijuana use (*b* = 0.395, 95% CI = [-0.105, 0.895], ß = 0.057) or marijuana use growth (*b* = 0.054, 95% CI = [-0.248, 0.356], ß = 0.022).

4.3.2. Two-Path Indirect Effects

For both alcohol use and marijuana use models, there was evidence of indirect effects of SM status on both level and change in rumination through my social stress measure. In my alcohol use model, results suggested an indirect effect of SM status on age 18 rumination (*ab* = 0.124, 99% CI = [0.056, 0.200]) and change in rumination (*ab* = -0.013, 99% CI = [-0.030, -0.001]) through age 17 social stress. Estimates were nearly identical for level (*ab* = 0.153, 99% CI = [0.062, 0.23]) and change (*ab* = -0.017, 99% CI = [-0.042, 0.002]) in rumination in my marijuana use model (though note the coefficient fell short of significance at the 99% level for the rumination change indirect effect).

There was marginal evidence of an indirect effect of SM status on marijuana use through my stress composite. Indirect effects of SM status on marijuana use at age 20 through both age 17 social stress (*ab* = 0.142, 99% CI = [-0.003, 0.345]) and abuse history (*ab* = 0.037, 99% CI = [-0.025, 0.128]) fell short of significance at the 99% confidence level for my multiple imputation solution. However, the effect of SM status on age 20 marijuana use through my stress composite was of similar magnitude and significant at the 99% confidence level in my FIML model (*ab* = 0.123, 99% CI = [0.008, 0.281]), and was significant at the 95% level of confidence in my multiple imputation model (95% CI = [0.028, 0.287]). There was no evidence of these indirect effects for alcohol use (rumination intercept: *ab* = -0.003, 99% CI = [-0.022, 0.0134], rumination slope: *ab* =

0.002, 99% CI = [-0.005, 0.012]) or marijuana use (rumination intercept: *ab* = -0.004, 99% CI = [-0.021, 0.009], rumination slope: *ab* = 0.001, 99% CI = [-0.010, 0.013]) when using the abuse history variable.

No other indirect effects were significant. There was no evidence of an indirect effect of SM status on marijuana use growth through social stress (*ab* = -0.041, 99% CI = [-0.127, 0.030]) or abuse history (*ab* = 0.005, 99% CI = [-0.032, 0.046]). There was also no indication of an indirect effect of sexual minority status on alcohol use growth (*ab* = -0.020, 99% CI = [-0.075, 0.029]) or alcohol use at age 20 (*ab* = 0.045, 99% CI = -0.029,0.134]) through social stress, nor were there indirect effects through abuse (*ab* = 0.005, 99% CI = [-0.022, 0.037] and *ab* = 0.015, 99% CI = [-0.026, 0.064], respectively).

4.3.3. Serial Indirect Effects

There was no evidence of serial mediation of the effect of SM status on age 20 alcohol use through abuse or social stress and age 18 rumination (social stress: *abc* = 0.017, 99% CI = [-0.008, 0.050]; abuse: *abc* = 0.000, 99% CI = [0.004, 0.003]) or change in rumination (social stress: *abc* = -0.017, 99% CI = [-0.090, 0.036]; abuse: *abc* = 0.004, 95% CI = [-0.014, 0.035]). Similarly, there was no evidence of serial mediation of alcohol use growth through abuse or social stress and age 18 rumination (social stress: *abc* = 0.004, 99% CI = [-0.013, 0.023], abuse: *abc* = 0.000, 99% CI = [-0.002, 0.001]) or change in rumination (social stress: *abc* = -0.013, 99% CI = [-0.067, 0.027], abuse: *abc* = .003, 99% CI = [-0.011, 0.025]).

I also found no evidence of serial mediation for marijuana use. There was no evidence of serial mediation of SM status on marijuana use at age 20 through abuse or social stress and age 18 rumination (social stress: *abc* = 9.020, 99% CI = [-0.008, 0.056], abuse: *abc* = -0.001, 99% CI = [-0.004, 0.001]) or change in rumination (social stress: *abc* = -0.044, 99% CI = [-0.173, 0.026], abuse: *abc* = 0.003, 99% CI = [-0.030, 0.043]), nor was there evidence of serial mediation of marijuana use growth through abuse or social stress and age 18 rumination (social stress: *abc* = 0.004, 99% CI = [-0.012, 0.022], abuse: *abc* = 0.000, 99% CI = [-0.001, 0.001]) or change in rumination (social stress: *abc* = -0.003, 99% CI = [-0.046, 0.036], abuse: *abc* = 0.000, 99% CI = [-0.009, 0.011]).

4.3.4. Interim Summary

Consistent with prior literature, lesbian and bisexual women reported greater social stress and a higher likelihood of abuse history relative to heterosexual women. Social stress was also associated with higher rumination by age 20 as well as slower increases in rumination across time (though abuse history was not). Of note, the effect of social stress on slower increases in rumination may strictly be an artifact of social stress predicting higher rumination at age 17. As seen in Figure 10 depicting the slope-intercept correlation, higher rumination at age 17 "carried forward" through age 20 while those low at age 17 "caught up" over time; thus, social stress predicted high rumination at age 17, which in turn stayed high (i.e., increased less) over time.

Although higher levels of social stress predicted more marijuana use by age 20 (but not growth), neither social stress nor abuse history were associated with level and change in alcohol use. Moreover, social stress was an intervening variable in the relation between sexual minority status and both level and change in rumination; sexual minority status predicted greater social stress through age 17, which in turn predicted more rumination at age 18 and less decline through age 20 (though see note on the change effect in the preceding paragraph). There was some (albeit inconsistent) evidence that the social stress composite was an intervening variable between sexual minority status and higher age 20 marijuana use. Notably, although my social stress measures were key intervening variables in the relations between sexual minority status and both rumination and marijuana use separately, I found no evidence that social stress and rumination serially mediated the relation between status and substance use. That is, my hypothesis that heightened substance use among lesbian and bisexual women was explained by stress-related increases in emotion dysregulation was not supported.

4.4. Assessing Moderation by Sexual Minority Status and Social Support

4.4.1. Model Selection

I was unable to estimate PPGMs described above using a multi-group model due to problems with convergence for both my alcohol use and marijuana use outcomes. As such, I instead estimated a simplified path model in which I predicted rank-order change in my substance use outcomes over time. I estimated these models with the following specifications: I regressed substance use at age 20 on both my abuse and social stress measures at age 17, controlling for substance use at age 18; 2.) I regressed age 18 rumination and social support on abuse and social stress at age 17, controlling for age 17 rumination and social support; and 3.) I regressed substance use at age 20 on rumination and social support at age 18, controlling for age 18 substance use. Though estimates were notably different in my alcohol use model for the observed data among sexual minority women, all other estimates were consistent across approaches (see Figures 21 and 22): fit measures, parameter estimates, and standard errors were mostly identical across approaches and differences did not affect substantive conclusions. Given I had a priori justification for using a multiple imputation approach, I report results from my multiply imputed models below.

For alcohol use models, chi-square tests were significant in 6 of 20 datasets, and the worst fit measures across imputed datasets remained in the acceptable range (RMSEA=0.033, CFI=0.993, TLI=.976, SRMR = .015), indicating adequate model fit. For marijuana use models, the worst fit measures across imputed datasets remained in the acceptable range (RMSEA=0.053, CFI=0.988, TLI=.958, SRMR = .007) despite significant chi-square tests in all but one of my marijuana use models, again indicating adequate model fit.

4.4.2. Moderation by Social Support

Next, I included the interaction between age 18 rumination and social support. However, model fit decreased with the inclusion of these terms for both alcohol use ($\Delta\bar{L}_{df=5}$ = 6630.453, p > .05, $\Delta\overline{BIC}$ = -13299.92) and marijuana use ($\Delta\bar{L}_{df=5}$ = 6632.669, p > .05, $\Delta\overline{BIC}$ = -13304.35), indicating no evidence of interaction between rumination and social support. The same was true when adding the interaction between my stress composite at age 18 and social support (alcohol use: $\Delta\bar{L}_{df=5}$ = 6785.452, p > .05, ΔBIC= -13609.92), marijuana use: ($\Delta\bar{L}_{df=5}$ = 6787.852, p > .05, $\Delta\overline{BIC}$ = -13614.72), as well as the interaction between abuse and social support (alcohol use: $\Delta\bar{L}_{df=5}$ = 3678.152, p > .05, $\Delta\overline{BIC}$ = -7395.318), marijuana use: $\Delta\bar{L}_{df=5}$ = 3678.029, p > .05, $\Delta\overline{BIC}$ =-7395.071). I therefore determined that there was no evidence of moderation of rumination or adversity by social support, and I omitted all social support interactions from the model moving forward.

4.4.3. Moderation by Sexual Minority Status

I then proceeded to specify a multi-group model with sexual orientation as a grouping variable. These models fit well for both my substance use outcomes: the worst fit measures in my alcohol and marijuana use models remained within the acceptable range despite significance of chi-square tests in the majority of datasets (Alcohol: RMSEA = 0.054, CFI = 0.968, TLI = 0.949, SRMR = 0.24; Marijuana: RMSEA = 0.033, CFI = 0.989, TLI = 0.983, SRMR = 0.020; see also Tables 21 and 22). Further, specifying multi-group models did not worsen fit relative to my ungrouped models (alcohol use: $\Delta\overline{\chi^2}$(18)= 15.272, p = 0.643), marijuana use: $\Delta\overline{\chi^2}$(18)= 11.55, p = 0.869), suggesting my path structure was appropriate for both SMW and HW (i.e., configural invariance was met). This ensured that evidence of non-invariance of my model parameters would not be due to differential fit of my path structure between groups.

Although a multi-group specification provided optimal fit, I found almost no evidence that sexual minority status moderated any of my direct and indirect path coefficients of interest for both alcohol and marijuana use (See Table 23). Therefore, I constrained all path parameters to equivalence and conducted a sequence of structural invariance tests to assess differences across groups in other structural parameters of my model. My results indicated that although invariance held when constraining all path coefficients to equivalence (Alcohol: $\Delta\chi^2(13)= 13.513$, $p = 0.409$; Marijuana: $\Delta\chi^2(13)= 10.199$, p = 0.678), I found evidence of non-invariance of the intercepts (Alcohol: $\Delta\chi^2(3)= 27.896$, $p < 0.001$; Marijuana: $\Delta\chi^2(3)= 23.532$, $p < 0.001$) and residuals (Alcohol: $\Delta\chi^2(3)= 11.162$, $p = 0.011$; Marijuana: $\Delta\chi^2(5)= 31.299$, $p < 0.001$) across groups. I therefore estimated a final model for both substance use outcomes in which all path coefficients were fixed to equivalence across groups, and estimated the intercepts and residuals freely in both groups. I report significant results of these models below, and all results are summarized in Table 24.

4.4.4. Direct and Indirect Effects

Social stress at age 17 was modestly associated with increases in rumination (Alcohol: b = .087, 95% CI = [0.019, 0.155], ß = 0.082; Marijuana: b = 0.094, 95% CI = [0.027, 0.160], ß = 0.087) and decreases in social support (Alcohol: b = -0.481, 95% CI = [-0.749, -0.214], ß = -0.133; Marijuana: b = -0.513, 95% CI = [-0.783, -0.243], ß = -0.142) from age 17 to age 18. However, no other relations were significant (see Table 24).

4.4.5. Interim Summary

There was no evidence that social support moderated the effects of rumination and social stress on substance use. The results of my structural invariance tests suggested that, as a whole, the parameters in my model were best approximated when estimating parameters separately for sexual minority and heterosexual groups; however, invariance testing of model parameters indicated that this was driven by differences in the intercept and residual variance parameters, and not by differences in the magnitude of path coefficients. Put differently, there was no evidence that the effects of social stress, rumination, and social support on rank-order change in substance use was different for sexual minority versus heterosexual women. Finally, there was no evidence of indirect effects of social stress on substance use through social support or rumination.

Chapter 5. Discussion

Disparities in the rates of substance-related disorder between sexual minority and non-minority populations may originate from the greater frequency, intensity, and chronicity of adverse life experiences, such that minority status functions as an imprecise proxy for stress (Hatzenbuehler, 2009). In particular, experiences of sexual minority stressors may increase social and emotional risk factors that induce vulnerability to developing substance use behaviors. However, few studies to date have tested mechanisms linking stress experiences with the development of substance use from adolescence through young adulthood. In the current study, I examined whether and how stress-related mechanisms of LGB psychopathology – including experiences of peer victimization, discrimination, harsh parenting, abuse, and greater emotion dysregulation – explained relations between sexual minority status and alcohol and marijuana use in a diverse sample of sexual minority and non-minority women. Treating sexual minority status as both a predictor and moderator, I tested direct and indirect effects of adverse life experiences and rumination on level and growth in alcohol and marijuana use from age 17 to age 20, and further explored social support as a moderator and mediator of these relations.

Results suggested a number of key longitudinal findings. First, sexual minority status explained individual differences in marijuana use by age 20; however, sexual minority women were otherwise no different from their heterosexual peers in levels of rumination at age 17 and growth in rumination through young adulthood, nor did they report steeper increases in marijuana or alcohol use or higher levels of alcohol use by age 20. Second, consistent with previous findings, sexual minority women experienced higher levels of social stress and abuse than heterosexual women by late adolescence. Third, these social stress experiences in turn predicted greater levels of rumination through age 20, as well as greater marijuana use by age 20.

5.1. Co-development in Rumination and Substance Use

Prior studies have shown that substance use increases normatively from adolescence through young adulthood, during which problem use is at a lifetime peak (e.g., Chassin, Ritter, Trim, & King, 2003). Studies have also suggested that adolescence is marked by greater higher sensitization to negative emotion (Silvers et al., 2012; Somerville, Jones, & Casey, 2010) coupled with still-developing capacities in regulating emotions (Steinberg, 2010), and prior cross-sectional research has shown an association between emotion dysregulation and substance use (Aldao, Nolen-Hoeksema, & Schweizer 2010). However, no prior studies have examined whether change in emotion regulation predicts change in substance use in the period in which each are co-developing, and whether these associations persist when examined longitudinally.

In the current study, I tested whether variation in adolescent rumination and change through young adulthood predicts change in substance use and level of use by age 20. I found that both rumination and substance use increased from adolescence through young adulthood, though women exhibited marked variation in the level and growth of both through young adulthood. Additionally, this study longitudinally replicates and extends prior meta-analytic findings from seven studies suggesting a moderate cross-sectional association between rumination and substance use (r = 0.21, 95% CI = [0.11, 0.31]; Aldao, Nolen-Hoeksema, & Schweizer, 2010), such that rumination was associated with levels of substance use three years later in an adolescent sample. Though I found relatively more modest relations for alcohol (β = 0.08) and marijuana use (β = 0.11), these may have been attenuated relative to meta-analytic findings due to the longitudinal nature of analyses. Moreover, I found no relations between growth factors, yet future research may be better powered to detect associations across a more protracted time course of development.

5.2. Sexual Minority Status Predicting Rumination and Substance Use Growth Processes

Prior studies have suggested that sexual minority women increase more rapidly in some forms of substance use (for instance, alcohol use, marijuana use, and problem drinking) from adolescence through young adulthood (Marshal et al, 2012; Marshal et al, 2009; Dermody et al., 2014). The current study sought to extend these findings to examine whether sexual minority status was associated with co-developing rumination across the same time period, and test whether level and growth in rumination explained faster increases in substance use. However, inconsistent with these prior findings, I found little evidence that sexual minority women differed from their heterosexual peers in their level and growth in alcohol use, and found only that sexual minority women reported more marijuana use by age 20. Similarly, I found no evidence that sexual minority women were different in levels and change in rumination from adolescence through young adulthood.

Relative to prior research, the current study examined a fewer number of annual assessments, and examined associations in a relatively earlier developmental period. For instance, three previous studies (Marshal et al., 2012; Marshal et al., 2009; Dermody et al., 2014) found steeper increases in substance use from ages 15 to as old as age 29 among LGB participants, with one study (Dermody et al., 2014) adding that growth rates were faster among LGB youth through age 22 but were no different through age 29; it may be that the present study was underpowered to detect such differences in rates of change given its three-year window of study. Moreover, in contrast to these studies, no women in my sample were of legal drinking age. This is critical given cultural factors influencing the availability of alcohol, such as patronage of gay bars and nightclubs in young adult samples, may play a role in distinguishing growth rates between sexual orientation groups (e.g., Greenwood et al., 2001; Trocki & Drabble, 2008). Future work should examine whether growth rates between sexual orientation groups differ both prior to and after age 21 to better quantify growth rates across groups.

5.3. Mediation by Social Stress and Abuse

Prior studies have found that emotion dysregulation was a mediator between sexual minority status and internalizing symptoms (Hatzenbuehler et al., 2008) and stigma-related stress and psycholgoical distress among sexual minority populations (Hatzenbuehler, Nolen-Hoeksema, & Dovidio, 2009; Szymanski & Henrichs-Beck, 2013). The current study was the first to examine stress as the intervening process between prior sexual minority identification and later emotion dysregulation, finding that greater social stress among sexual minroity women explained individual differences in both level and change in rumination over time. Moreover, consistent with one prior study reporting similar longitudinal findings (Dermody et al., 2016), I found trending evidence that social stress also explained the relation bewteeen sexual minority status and hightened marijuana use by young adulthood.

However, contrary to my hypotheses, I did not find that these pathways were serially mediated: the effect of stress on substance use among sexual minority women was not a product of how stress affected rumination. This suggests that minority stress may increase risk of substance use through other mediation processes. Drawing from theory in the general population (Hussong, 2011) as well as other proposed psychological mediators in sexual minority stress theory (e.g., Hatzenbuehler, 2009; Pachankis, 2015), these may include increased coping motives for substance use, stress-related changes in positive reinforcement sensitivity, and affiliation with substance-permissive peers and contexts. Two prior studies (Huebner, Thoma, and Neilands 2015; Dermody et al., 2016) have tested whether affiliation with deviant peers expalined the relation between victimization and substance use among LGB youth. Though the former (Huebner, Thoma, and

Neilands, 2015) found cross-sectional evidence of this pathway, the latter (Dermody et al., 2016) was unable to replicate this effect in a longitudinal sample. Future researach aimed at explaining elevated substance use risk among LGB youth should test these additional mediating processes. It may be that risk is best characterized by a confluence of these intervening factors that shape risk of disorder across development.

5.4. Strengths of the Current Study

The current study adds to the literature in a number of ways. First, existing longitudinal studies testing the mediating role of emotion dysregulation only examined internalizing disorder among LGB adults as an outcome. Given that sexual minority stress theory states that emotion dysregulation may serve as a mediator for both internalizing and substance-related disorder, this study was critical in extending prior work to establish whether emotion dysregulation was a transdiagnostic indicator of multiple forms of LGB psychopathology. Concretely, though I did not find evidence that the mediating role of emotion dysregulation generalized to substance use, there remained an intervening effect of social stress directly in increasing risk for marijuana use.

Second, and relatedly, this was the first longitudinal study to examine stress and emotion dysregulation as serial mediators of the relation between sexual minority status and substance use. To my knowledge, only one prior cross-sectional study (Marshal, 2013) has tested a related serially-mediated pathway. All other mediation studies have tested two-path mediation treating either LGB stress or LGB status as exogenous predictors of LGB psychopathology, yet none have tested whether heightened stressors and emotion dysregulation sequentially mediate the relation between sexual minority status and substance use. This approach allowed me to establish more precisely whether and how stressors increase risk for substance use among sexual minority women; results suggested that stressors may increase risk for marijuana use for sexual minority women, and that stress among SM women increased level and change in rumination, yet these stress-related pathways of risk are distinct from each other.

Third, although all but one (Needham, 2012) prior study testing growth in LGB substance use estimated models treating outcomes as continuous, the current study used more rigorous methods of handling missing data and estimation to more optimally characterize true relations in my data. In particular, I applied recent advances in multiple imputation in tandem with utilizing diagonally-weighted least squares estimation for complete data to most optimally accommodate binary and ordinal variables, which has been shown in prior literature to reduce bias and inefficiency relative to standard maximum likelihood-based estimation.

Fourth, no prior studies have tested moderators of mediated pathways toward LGB psychopathology. This was an essential focus given that addressing risk and resilience factors acknowledges and aims to better understand heterogeneity in the experiences of sexual minority individuals (Hatzenbuehler, 2009; Kwon, 2013; Riggle, Whitman, Olson, Rostosky, & Strong, 2008). Although I found no evidence that that social support was a moderator of substance use risk among sexual minority women, future research should continue to address both within- and between-group risk indicators of LGB psychopathology.

5.5. Limitations and Future Directions

Due to sample size concerns, I was unable to test my hypotheses treating lesbian and bisexual women as separate groups. However, evidence suggests significant heterogeneity in substance use behaviors across sexual minority subgroups, and understanding the proposed mechanisms among LGB subgroups is an area of future research. For instance, prevalence rates of alcohol and other drug use behaviors among LGB sub-populations indicate that bisexuality in particular may be the highest-risk sexual minority group, with multiple studies suggesting that both bisexual identity and behavior increased risk for alcohol and drug use problems (Green & Feinstein, 2012). Further evidence is needed to determine whether and how each of the pathways proposed in the current review shape substance use experiences within each of these sexual minority subgroups.

Further, there were a two methodological limitations that arose in my data largely due to missingness in a number of key variables. For instance, the omission of two additional indicators of emotion regulation (emotion regulation strategies and awareness) prevented me from testing and determining a more comprehensive, content-valid latent construct of emotion dysregulation. Additionally, I noted discrepancies in the slope parameter of my alcohol use models when comparing FIML and multiple imputation results. These discrepancies could be for several reasons. First, the imputation solution generated by Blimp may have estimated missing values that were smaller than the observed data due to the assumption of normality in this measure, thus biasing the estimates toward zero in the imputation case. Second, and relatedly, there was considerable missingness at age 20 of my alcohol use variable (over 60%), which may have caused considerable difficulty in attaining a reliable estimate of a slope parameter. Third, I had to omit both age 17 and age 21 alcohol use from analysis due to difficulties in attaining a converging solution. This, compounded with the issues above, may have limited my capacity to estimate a reliable growth trajectory due to range restriction across time.

Finally, a careful consideration of how and when minority status is quantified is essential in addressing these questions in future research. The vast majority of studies (as well as the current study) used a binary indicator sexual identity (e.g., endorsing “not 100% heterosexual”) at a single time point, yet different methods of quantifying status may suggest differing results (e.g., Marshal et al., 2012; Needham, 2012). For instance, one prior study (Talley et al., 2010) tested changes in substance through adulthood using three separate measures (identity, attraction, and behavior) of orientation reported at either the initial or the final time point of the study. The authors found that although general increases in substance use behaviors were evident among sexual minority adults, sexual identity tended to be a more reliable indicator of higher and sustained levels of substance use behaviors compared to attraction and behavior, and measuring identity at either the first or final time point altered how cannabis and alcohol use changed over time. Indeed, some research has suggested that coming out earlier in development may confer positive health benefits in some contexts but may be health risk factors in others (e.g., D'Augelli, 2006; Legate et al., 2012); it is possible that the age of coming out may have a significant impact on both the experiences of minority stress and substance use risk, yet more research is needed to address this question.

5.8. Conclusions

The current project was the first longitudinal study of LGB youth examining the effects of stress on changes in rumination and substance use across sexual orientation populations. No prior LGB studies to date has tested emotion regulation as a mediator of substance use, and none have addressed these questions while addressing moderators of these pathways. In general, I found largely no differences across sexual orientation groups in level and change in rumination and substance use through young adulthood, with the exception of heightened marijuana use among sexual minority women. There was also no evidence that rumination explained elevations in substance use among sexual minority women in tests of two-path and serial mediation. Instead, social stress was a critical intervening variable explaining more marijuana use and rumination among sexual minority women. These findings provide greater clarity on identifying whether and how experiences of stress increase risk for developing psychopathology among LGB youth. The null direct findings of this study also echo the point the that LGB youth are a largely resilient population in spite of greater adversity experienced across multiple domains.

Understanding the role of stress in explaining LGB substance use and related psychopathology is essential in informing intervention efforts for LGB youth struggling with mental health concerns. The current study showed that experiencing greater adversity among LGB communities may have downstream effects on higher and persisting difficulties in emotion regulation, and separately, on increasing risk for greater drug use in young adulthood. These findings highlight that policy and treatment-level interventions aimed at reducing and managing experiences of victimization may serve to reduce internalizing psychopathology through reducing emotion dysregulation, and may have long-term protective effects on reducing drug use directly. Treatments that explicitly target emotion dysregulation and interpersonal difficulties, such as Cognitive Behavioral Therapy and Dialectical Behavior Therapy, may also be exceptionally well-suited treatment approaches for victimized sexual minority youth. Overall, the current findings highlight the crucial need to expand current sexual minority stress theories and research of LGB substance use to better understand mechanisms of risk (and protection) among LGB youth.

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Table 1. Study measures.

Construct(s)	**Measure (Cronbach's Alpha)**	**Age**
Sexual Minority Status	Single sexual orientation item	16 - 21
Physical/Sexual Abuse	Official police records and abuse questionnaire	12 - 17
Peer Victimization	9-item Peer Experiences Questionnaire (α = .82)	17
Parental Abuse	19-item Conflict Tactics Scale – Parent/Child Version (α = .73-.75)	17
Discrimination	9-item Everyday Discrimination Scale (α = .88)	17 - 21
Awareness and Understanding of Emotions	6 items from the Difficulties in Emotion Regulation Scale (α = .80)	17 - 21
Emotion Regulation Strategies	8 items from the Difficulties in Emotion Regulation Scale (α = .88)	17 - 21
Rumination	7 items from the Perfectionism Inventory (α = .87)	17 - 21
Substance-related disorders	Substance use modules of the Composite International Diagnostic Interview	17 - 21
Substance Use Past Year Frequency	Single items from the Nicotine, Alcohol, and Drug Substance Use Scale	17 - 21
Parent and Peer Support	75 items from the Inventory of Parent and Peer Attachment (α = .73 - .91)	11 - 21

Table 2. Percent observed in key study variables by assessment period.

Measure	Age 16	Age 17	Age 18	Age 19	Age 20	Age 21
Rumination	.	**83.90%**	**85.80%**	**66.10%**	**43.30%**	21.70%
Emotion Regulation Strategies	.	<u>*20.00%*</u>	<u>*42.40%*</u>	<u>*44.30%*</u>	<u>*43.30%*</u>	<u>*21.70%*</u>
Emotional Awareness	.	<u>*20.00%*</u>	<u>*42.40%*</u>	<u>*44.30%*</u>	<u>*43.30%*</u>	<u>*21.70%*</u>
Past Year Alcohol Use	.	<u>*16.00%*</u>	**44.80%**	**51.10%**	**37.40%**	<u>*21.70%*</u>
Past Year Marijuana Use	.	**83.80%**	**85.70%**	**66.10%**	**43.30%**	<u>*21.70%*</u>
Parent and Peer Attachment	**84.50%**	**83.80%**	65.00%	43.20%	21.50%	.
Everyday Discrimination	.	**62.80%**	85.90%	66.10%	43.30%	21.80%
Harsh Parenting	**83.60%**	**81.50%**	.	.	.	.
Peer Victimization	**84.60%**	**83.90%**	65.50%	43.60%	21.70%	.
Physical/Sexual Abuse (Yes/No)	***85.50%***	***84.80%***	.	.	.	.
Sexual Minority Status	**84.10%**	83.40%	85.40%	65.80%	43.00%	21.70%

Note. Bolded values indicate variables included in the final imputation model, and underlined and italicized variables were attempted, but caused non-convergence in the model if they were included. Complete cohort and race variables were also included in the model as auxiliary measures.

Table 3. Descriptive Statistics for Study Variables by Sexual Minority Status and Age.

		Lesbian and Bisexual Women (n = 144)						Heterosexual Women (n = 1913)					
	Age	N	Min	Max	Mean/%	SD	Skew	N	Min	Max	Mean	SD	Skew
Racial Identity (Black)	-	144	0	1	57%	-	-	1913	0	1	55%	-	-
Racial Identity (White)	-	144	0	1	33%	-	-	1913	0	1	40%	-	-
Racial Identity (Other)	-	144	0	1	10%	-	-	1913	0	1	5%	-	-
Abuse History (Yes/No)	-	144	0	1	19%	-	-	1913	0	1	8%	-	-
Harsh Parenting	17	126	1	11	3.75	2.39	0.84	1796	1	13	3.09	2.14	1.22
Peer Victimization	17	137	1	21	3.42	3.68	2.15	1837	1	22	2.10	2.21	3.34
Everyday Discrimination	17	110	1	18	6.17	4.35	0.77	1366	1	22	4.76	3.91	1.30
Social Support	17	137	2	19	14.58	4.05	-0.70	1834	1	19	16.10	3.65	-1.33
	18	95	1	19	14.21	4.51	-0.72	1370	1	19	16.51	3.38	-1.49
Rumination	17	136	1	5	2.65	1.04	-0.06	1836	1	5	2.60	1.06	0.00
	18	140	1	5	2.74	1.03	-0.27	1793	1	5	2.57	1.02	-0.01
	19	90	1	5	2.69	1.12	0.00	1376	1	5	2.61	1.04	-0.05
Rumination	20	55	1	5	2.85	0.95	-0.10	901	1	5	2.63	1.03	0.02
	21	27	1	5	2.78	1.12	-0.21	450	1	5	2.67	1.07	-0.09
Emotional Awareness	17	44	1	24	11.84	6.10	0.05	425	1	25	10.12	5.79	0.32
	18	80	6	27	16.65	5.73	-0.02	878	6	30	14.73	5.65	0.29
	19	63	1	20	10.75	5.65	-0.07	915	1	24	9.24	5.55	0.43
	20	55	1	22	10.45	5.80	0.14	901	1	25	9.13	5.56	0.44
	21	27	1	18	9.22	4.61	0.51	450	1	21	8.70	5.40	0.44
Emotion Regulation Strategies	17	44	1	23	9.20	5.83	0.47	425	1	26	7.85	5.11	1.00
	18	80	8	34	16.33	5.66	0.88	878	8	39	14.66	5.33	1.07
	19	63	1	24	9.37	5.31	0.66	915	1	28	8.01	5.47	1.07
	20	55	1	24	10.22	6.16	0.12	901	1	30	7.94	5.37	1.24
	21	27	1	21	8.93	5.60	0.46	450	1	27	7.78	5.31	1.11
Alcohol Use	17	44	1	6	3.09	1.48	0.65	335	1	6	2.72	1.08	1.19
	18	84	1	6	2.87	1.50	0.58	926	1	7	2.60	1.42	0.74
	19	71	1	7	3.00	1.66	0.53	1058	1	8	3.02	1.56	0.46
	20	47	1	7	3.23	1.82	0.40	779	1	7	3.09	1.62	0.34
	21	27	1	6	3.44	1.67	0.07	450	1	7	3.42	1.63	0.00
Marijuana Use	17	137	1	8	2.75	2.33	1.01	1836	1	8	1.72	1.66	2.49
	18	139	1	8	3.02	2.48	0.78	1792	1	8	1.95	1.86	2.01
	19	90	1	8	3.13	2.70	0.76	1376	1	8	2.16	2.05	1.71
	20	55	1	8	3.24	2.81	0.74	901	1	8	2.18	2.14	1.70
	21	27	1	8	3.56	2.67	0.33	450	1	8	2.00	1.92	1.96

Note: Min = minimum, Max = maximum, SD = Standard Deviation

Table 4. Correlations among Continuous Study Variables by Sexual Minority Status.

	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.	13.	14.	15.
1. Harsh Punishment	-	0.18	0.26	-0.45	-0.35	0.15	0.13	0.17	0.07	0.03	0.02	0.01	0.08	0.15	0.08
2. Peer Victimization	*0.19*	-	0.51	-0.14	-0.17	0.20	0.16	0.16	0.14	0.12	0.05	0.00	0.11	0.11	0.05
3. Discrimination	*0.31*	*0.54*	-	-0.30	-0.27	0.30	0.23	0.25	0.22	0.15	0.15	0.11	0.19	0.19	0.14
4. Age 17 Social Support	*-0.38*	*-0.08*	*-0.21*	-	0.57	-0.14	-0.12	-0.14	-0.11	-0.11	-0.05	-0.04	-0.09	-0.09	-0.05
5. Age 18 Social Support	*-0.35*	*-0.25*	*-0.40*	*0.65*	-	-0.13	-0.12	-0.12	-0.09	-0.09	-0.06	-0.01	-0.11	-0.13	-0.06
6. Age 17 Rumination	*0.29*	*0.27*	*0.43*	*-0.20*	*-0.30*	-	0.52	0.52	0.45	0.10	0.06	0.02	0.02	0.06	0.04
7. Age 18 Rumination	*0.24*	*0.17*	*0.36*	*-0.14*	*-0.24*	*0.49*	-	0.53	0.48	0.10	0.02	0.04	0.07	0.05	0.05
8. Age 19 Rumination	*0.24*	*0.11*	*0.24*	*-0.11*	*-0.29*	*0.57*	*0.53*	-	0.51	0.05	0.02	0.00	0.08	0.11	0.06
9. Age 20 Rumination	*0.35*	*0.23*	*0.14*	*-0.13*	*-0.10*	*0.42*	*0.58*	*0.51*	-	-0.02	-0.01	0.09	0.12	0.11	0.10
10. Age 18 Alcohol Use	*0.08*	*-0.02*	*0.07*	*-0.23*	*0.00*	*-0.08*	*0.02*	*-0.07*	*-0.30*	-	0.45	0.39	0.31	0.11	0.10
11. Age 19 Alcohol Use	*0.09*	*0.05*	*0.07*	*-0.23*	*-0.35*	*0.10*	*0.04*	*0.12*	*-0.36*	*0.33*	-	0.50	0.16	0.27	0.12
12. Age 20 Alcohol Use	*-0.05*	*-0.26*	*-0.04*	*-0.36*	*-0.10*	*-0.36*	*-0.14*	*-0.23*	*-0.30*	*0.27*	*0.35*	-	0.18	0.20	0.28
13. Age 18 Marijuana Use	*0.10*	*-0.04*	*0.12*	*-0.11*	*-0.03*	*0.00*	*0.00*	*0.02*	*-0.04*	*0.35*	*0.24*	*0.07*	-	0.60	0.47
14. Age 19 Marijuana Use	*-0.03*	*-0.03*	*0.01*	*0.03*	*0.03*	*0.05*	*-0.01*	*-0.01*	*0.06*	*0.24*	*0.42*	*0.13*	*0.51*	-	0.60
15. Age 20 Marijuana Use	*-0.14*	*-0.20*	*0.04*	*0.03*	*0.26*	*-0.10*	*-0.04*	*-0.33*	*-0.17*	*-0.14*	*0.20*	*0.33*	*0.59*	*0.53*	-

Note. Lower triangle of matrix (italicized) reflect correlations among sexual minority women, and upper triangle (non-italicized) reflect correlations among heterosexual women.

Table 5. Correlation of Social Stress Measures with Four-Factor Principal Components.

	Component 1 (mean eigenvalue = 1.710)				Component 2 (mean eigenvalue = 0.973)				Component 3 (mean eigenvalue = 0.475)			
	HP	PV	ED	Abuse	HP	PV	ED	Abuse	HP	PV	ED	Abuse
Obs. Data	0.544	0.798	0.847	0.324	-0.439	-0.016	-0.042	0.884	0.707	-0.413	-0.194	0.335
Imp. 1	0.513	0.806	0.829	0.311	-0.383	-0.071	-0.032	0.905	0.767	-0.343	-0.250	0.289
Imp. 2	0.527	0.806	0.837	0.311	-0.376	-0.049	-0.053	0.908	0.759	-0.368	-0.228	0.281
Imp. 3	0.548	0.802	0.832	0.284	-0.245	-0.088	-0.076	0.946	0.797	-0.363	-0.228	0.154
Imp. 4	0.517	0.809	0.828	0.300	-0.381	-0.053	-0.040	0.909	0.766	-0.338	-0.253	0.290
Imp. 5	0.526	0.794	0.830	0.290	-0.370	-0.053	-0.034	0.915	0.762	-0.378	-0.219	0.278
Imp. 6	0.519	0.806	0.827	0.276	-0.346	-0.031	-0.061	0.924	0.780	-0.344	-0.242	0.265
Imp. 7	0.535	0.802	0.832	0.295	-0.361	-0.054	-0.041	0.917	0.761	-0.366	-0.232	0.268
Imp. 8	0.532	0.815	0.839	0.280	-0.318	-0.054	-0.057	0.931	0.783	-0.346	-0.238	0.233
Imp. 9	0.532	0.803	0.829	0.286	-0.336	-0.047	-0.058	0.925	0.775	-0.359	-0.235	0.249
Imp. 10	0.525	0.795	0.837	0.335	-0.381	-0.095	-0.032	0.900	0.758	-0.385	-0.219	0.273
Imp. 11	0.544	0.807	0.831	0.269	-0.282	-0.050	-0.071	0.942	0.788	-0.353	-0.237	0.199
Imp. 12	0.534	0.806	0.830	0.283	-0.337	-0.045	-0.055	0.926	0.773	-0.353	-0.240	0.250
Imp. 13	0.530	0.806	0.832	0.272	-0.296	-0.045	-0.075	0.938	0.792	-0.355	-0.231	0.214
Imp. 14	0.532	0.814	0.835	0.277	-0.360	-0.035	-0.042	0.921	0.765	-0.341	-0.246	0.275
Imp. 15	0.508	0.821	0.839	0.284	-0.362	-0.054	-0.039	0.917	0.781	-0.328	-0.247	0.278
Imp. 16	0.530	0.800	0.830	0.304	-0.338	-0.064	-0.060	0.921	0.775	-0.366	-0.232	0.244
Imp. 17	0.524	0.811	0.830	0.296	-0.295	-0.070	-0.078	0.933	0.798	-0.336	-0.249	0.206
Imp. 18	0.566	0.790	0.827	0.289	-0.243	-0.103	-0.066	0.946	0.784	-0.383	-0.222	0.144
Imp. 19	0.531	0.817	0.829	0.259	-0.288	-0.042	-0.068	0.943	0.797	-0.321	-0.258	0.210
Imp. 20	0.529	0.802	0.827	0.298	-0.322	-0.061	-0.069	0.926	0.783	-0.354	-0.240	0.231
Mean	**0.531**	**0.805**	**0.832**	**0.292**	**-0.336**	**-0.056**	**-0.055**	**0.923**	**0.774**	**-0.357**	**-0.235**	**0.246**

Note. Obs. Data = Observed Data, Imp. = Imputation, HP = harsh punishment, ED = Everyday Discrimination, Abuse = Abuse History. Mean reflects the average correlation across imputations. Correlations with component 4 removed for parsimony.

Table 6. Correlation of Social Stress Measures with Three-Factor Principal Components.

	Component 1 (mean eigenvalue = 1.669)			Component 2 (mean eigenvalue = 0.856)			Component 3 (mean eigenvalue = 0.475)		
	HP	PV	ED	HP	PV	ED	HP	PV	ED
Obs. Data	0.540	0.834	0.841	0.838	-0.344	-0.148	0.077	0.428	-0.503
Imp. 1	0.535	0.820	0.834	0.844	-0.309	-0.238	0.036	0.482	-0.497
Imp. 2	0.549	0.817	0.844	0.833	-0.342	-0.211	0.066	0.465	-0.493
Imp. 3	0.562	0.813	0.840	0.825	-0.346	-0.217	0.066	0.469	-0.498
Imp. 4	0.539	0.820	0.835	0.842	-0.311	-0.238	0.037	0.481	-0.496
Imp. 5	0.547	0.805	0.834	0.834	-0.349	-0.210	0.072	0.479	-0.510
Imp. 6	0.537	0.813	0.835	0.842	-0.328	-0.222	0.054	0.481	-0.503
Imp. 7	0.557	0.813	0.837	0.829	-0.340	-0.221	0.061	0.474	-0.500
Imp. 8	0.549	0.824	0.845	0.834	-0.327	-0.224	0.051	0.463	-0.485
Imp. 9	0.551	0.811	0.836	0.833	-0.339	-0.219	0.061	0.476	-0.502
Imp. 10	0.549	0.814	0.841	0.833	-0.343	-0.212	0.067	0.470	-0.498
Imp. 11	0.559	0.814	0.839	0.827	-0.339	-0.222	0.060	0.471	-0.497
Imp. 12	0.553	0.814	0.837	0.832	-0.334	-0.224	0.056	0.475	-0.499
Imp. 13	0.546	0.813	0.840	0.835	-0.340	-0.213	0.065	0.472	-0.499
Imp. 14	0.552	0.822	0.840	0.833	-0.322	-0.232	0.046	0.470	-0.490
Imp. 15	0.526	0.831	0.845	0.849	-0.302	-0.232	0.035	0.468	-0.482
Imp. 16	0.549	0.811	0.837	0.833	-0.341	-0.216	0.064	0.475	-0.502
Imp. 17	0.541	0.821	0.839	0.840	-0.316	-0.231	0.043	0.474	-0.492
Imp. 18	0.581	0.802	0.833	0.810	-0.364	-0.215	0.078	0.473	-0.510
Imp. 19	0.545	0.823	0.837	0.838	-0.309	-0.241	0.034	0.476	-0.491
Imp. 20	0.547	0.812	0.835	0.835	-0.333	-0.223	0.057	0.479	-0.503
Mean	**0.548**	**0.816**	**0.838**	**0.834**	**-0.332**	**-0.220**	**0.056**	**0.472**	**-0.498**

Note. Obs. Data = Observed Data, Imp. = Imputation, HP = harsh punishment, ED = Everyday Discrimination, Abuse = Abuse History. Mean reflects the average correlation across imputations. Correlations with component 4 removed for parsimony.

Table 7. Fit Measures for Unconditional Rumination Growth Models.

	χ^2	*p*-value	CFI	TLI	RMSEA	RMSEA Lower CL	RMSEA Upper CL	SRMR
Imputation 1	23.407	0.000	0.994	0.993	0.039	0.024	0.055	0.027
Imputation 2	22.851	0.000	0.995	0.994	0.038	0.023	0.055	0.026
Imputation 3	29.298	0.000	0.993	0.991	0.045	0.030	0.061	0.030
Imputation 4	12.210	0.032	0.998	0.997	0.024	0.007	0.042	0.019
Imputation 5	20.580	0.001	0.995	0.994	0.036	0.021	0.052	0.025
Imputation 6	39.080	0.000	0.989	0.987	0.053	0.038	0.069	0.034
Imputation 7	13.164	0.022	0.998	0.997	0.026	0.009	0.043	0.020
Imputation 8	16.099	0.007	0.997	0.996	0.030	0.014	0.047	0.022
Imputation 9	16.687	0.005	0.996	0.996	0.031	0.015	0.048	0.022
Imputation 10	25.304	0.000	0.994	0.992	0.041	0.026	0.057	0.028
Imputation 11	25.176	0.000	0.994	0.993	0.041	0.026	0.057	0.027
Imputation 12	32.182	0.000	0.991	0.989	0.047	0.032	0.063	0.031
Imputation 13	13.968	0.016	0.997	0.997	0.027	0.011	0.044	0.021
Imputation 14	15.611	0.008	0.997	0.996	0.029	0.014	0.047	0.022
Imputation 15	3.105	0.684	1.000	1.001	0.000	0.000	0.022	0.010
Imputation 16	18.593	0.002	0.996	0.995	0.033	0.018	0.050	0.024
Imputation 17	13.941	0.016	0.997	0.997	0.027	0.011	0.044	0.021
Imputation 18	17.392	0.004	0.996	0.995	0.032	0.016	0.049	0.023
Imputation 19	15.627	0.008	0.997	0.996	0.029	0.014	0.047	0.022
Imputation 20	7.170	0.208	0.999	0.999	0.013	0.000	0.033	0.015
Imputation Mean	**19.072**	**-**	**0.996**	**0.995**	**0.032**	**0.017**	**0.049**	**0.023**
FIML	6.294	0.279	0.999	0.999	0.011	0.000	0.032	0.019
Observed Data	14.106	0.015	0.990	0.988	0.045	0.019	0.072	0.027

Note. CFI = Confirmatory Factor Index, TLI = Tucker-Lewis Index, RMSEA = Root Mean Squared Error of Approximation, SRMR = Standardized Root Mean Residual. RMSEA confidence limits are at the 90% level of confidence. Chi-squared values reflect scaled values for FIML and observed data models. Bolded rows indicate the selected model.

Table 8. Fit measures for Unconditional Alcohol Growth Models.

	χ^2	*p*-value	CFI	TLI	RMSEA	RMSEA Lower CL	RMSEA Upper CL	SRMR
Imputation 1	1.434	0.231	1.000	0.999	0.013	0.000	0.057	0.008
Imputation 2	2.129	0.145	0.999	0.998	0.021	0.000	0.063	0.010
Imputation 3	6.083	0.014	0.997	0.991	0.046	0.017	0.083	0.017
Imputation 4	14.909	0.000	0.992	0.977	0.075	0.045	0.111	0.026
Imputation 5	4.955	0.026	0.997	0.992	0.040	0.011	0.078	0.015
Imputation 6	4.646	0.031	0.998	0.993	0.039	0.009	0.077	0.015
Imputation 7	16.002	0.000	0.991	0.974	0.078	0.048	0.114	0.027
Imputation 8	3.894	0.048	0.998	0.995	0.034	0.002	0.073	0.013
Imputation 9	0.640	0.424	1.000	1.001	0.000	0.000	0.049	0.005
Imputation 10	7.118	0.008	0.996	0.989	0.050	0.021	0.087	0.018
Imputation 11	18.823	0.000	0.989	0.968	0.085	0.054	0.121	0.029
Imputation 12	21.735	0.000	0.987	0.960	0.092	0.061	0.128	0.031
Imputation 13	20.331	0.000	0.989	0.966	0.089	0.058	0.124	0.030
Imputation 14	0.594	0.441	1.000	1.001	0.000	0.000	0.049	0.005
Imputation 15	15.804	0.000	0.991	0.972	0.078	0.047	0.114	0.027
Imputation 16	10.996	0.001	0.994	0.981	0.064	0.034	0.100	0.022
Imputation 17	1.227	0.268	1.000	1.000	0.010	0.000	0.056	0.007
Imputation 18	0.001	0.981	1.000	1.002	0.000	0.000	0.000	0.000
Imputation 19	25.071	0.000	0.986	0.958	0.099	0.068	0.134	0.034
Imputation 20	2.469	0.116	0.999	0.997	0.025	0.000	0.065	0.011
Imputation Mean	8.943	-	0.995	0.986	0.047	0.024	0.084	0.018
FIML	**3.611**	**0.057**	**0.992**	**0.976**	**0.036**	**0.000**	**0.000**	**0.015**
Observed Data	7.675	0.006	0.931	0.793	0.170	0.000	0.000	0.037

Note. CFI = Confirmatory Factor Index, TLI = Tucker-Lewis Index, RMSEA = Root Mean Squared Error of Approximation, SRMR = Standardized Root Mean Residual. RMSEA confidence limits are at the 90% level of confidence. Chi-squared values reflect scaled values for FIML and observed data models. Bolded rows indicate the selected model.

Table 9. Fit measures for Unconditional Marijuana Use Growth Models.

	χ^2	*p*-value	CFI	TLI	RMSEA	RMSEA Lower CL	RMSEA Upper CL	SRMR
Imputation 1	0.524	0.469	1.000	1.001	0.000	0.000	0.048	0.005
Imputation 2	0.366	0.545	1.000	1.002	0.000	0.000	0.045	0.004
Imputation 3	0.850	0.357	1.000	1.000	0.000	0.000	0.052	0.006
Imputation 4	1.692	0.193	0.999	0.998	0.017	0.000	0.060	0.009
Imputation 5	0.164	0.685	1.000	1.002	0.000	0.000	0.040	0.003
Imputation 6	0.128	0.720	1.000	1.002	0.000	0.000	0.038	0.002
Imputation 7	0.039	0.843	1.000	1.003	0.000	0.000	0.031	0.001
Imputation 8	1.211	0.271	1.000	0.999	0.009	0.000	0.055	0.007
Imputation 9	1.346	0.246	1.000	0.999	0.012	0.000	0.057	0.008
Imputation 10	3.355	0.067	0.998	0.994	0.031	0.000	0.070	0.012
Imputation 11	0.307	0.579	1.000	1.002	0.000	0.000	0.044	0.004
Imputation 12	0.950	0.330	1.000	1.000	0.000	0.000	0.053	0.007
Imputation 13	0.640	0.424	1.000	1.001	0.000	0.000	0.049	0.005
Imputation 14	0.861	0.353	1.000	1.000	0.000	0.000	0.052	0.006
Imputation 15	1.959	0.162	0.999	0.998	0.020	0.000	0.062	0.009
Imputation 16	0.892	0.345	1.000	1.000	0.000	0.000	0.052	0.006
Imputation 17	4.138	0.042	0.997	0.992	0.036	0.006	0.074	0.014
Imputation 18	1.236	0.266	1.000	0.999	0.010	0.000	0.056	0.007
Imputation 19	2.684	0.101	0.999	0.996	0.026	0.000	0.066	0.011
Imputation 20	2.305	0.129	0.999	0.997	0.023	0.000	0.064	0.010
Imputation Mean	**1.282**	**-**	**1.000**	**0.999**	**0.009**	**0.000**	**0.053**	**0.007**
FIML	1.546	0.214	0.999	0.996	0.016	0.000	0.000	0.007
Observed Data	0.824	0.364	1.000	1.002	0.000	0.000	0.000	0.006

Note. CFI = Confirmatory Factor Index, TLI = Tucker-Lewis Index, RMSEA = Root Mean Squared Error of Approximation, SRMR = Standardized Root Mean Residual. RMSEA confidence limits are at the 90% level of confidence. Chi-squared values reflect scaled values for FIML and observed data models. Bolded rows indicate the selected model.

Table 10. Summary of Unconditional Growth Curve Models.

	Rumination				Alcohol Use				Marijuana Use			
	Est.	SE	95% CI	Std. Est.	Est.	SE	95% CI	Std. Est.	Est.	SE	95% CI	Std. Est.
Growth Parameters												
Intercept	2.577	0.024	[2.53, 2.62]	-	2.586	0.038	[2.51, 2.66]	-	2.060	0.045	[1.97, 2.15]	-
Slope	**0.045**	**0.021**	**[0.00, 0.09]**	**0.291**	**0.169**	**0.030**	**[0.11, 0.23]**	**0.260**	**0.177**	**0.066**	**[0.05, 0.31]**	**0.246**
Intercept Variance	**0.619**	**0.037**	**[0.55, 0.69]**	**1.000**	**1.381**	**0.161**	**[1.07, 1.70]**	**1.000**	**2.787**	**0.308**	**[2.18, 3.39]**	**1.000**
Slope Variance	**0.024**	**0.011**	**[0.00, 0.05]**	**1.000**	**0.423**	**0.100**	**[0.23, 0.62]**	**1.000**	**0.515**	**0.173**	**[0.18, 0.85]**	**1.000**
Slope-Intercept Covariance	**-0.043**	**0.018**	**[-0.08, -0.01]**	**-0.355**	**-0.229**	**0.104**	**[-0.43, -0.02]**	-0.300	-0.271	0.199	[-0.66, 0.12]	-0.223
Residual Variances												
Age 17	0.495	0.046	[0.41, 0.58]	0.445	-	-	-	-	-	-	-	-
Age 18	0.489	0.030	[0.43, 0.55]	0.468	0.639	0.160	[0.32, 0.95]	0.316	1.117	0.340	[0.45, 1.78]	0.286
Age 19	0.561	0.038	[0.49, 0.64]	0.509	1.157	0.075	[1.01, 1.30]	0.462	1.771	0.196	[1.39, 2.15]	0.391
Age 20	0.496	0.057	[0.38, 0.61]	0.463	0.574	0.170	[0.24, 0.91]	0.210	1.529	0.375	[0.79, 2.26]	0.289

Note. Fixed parameters are omitted for parsimony. Est. = Estimate, SE = Standard Error, 95% CI = 95% Confidence Interval, Std. Est. = Standardized Estimate. All models specified intercepts at the first time point. Bolded values indicate significance of parameters of interest at the 95% level of confidence.

Table 11. Parallel Process Growth Curve Model of Rumination and Alcohol Use.

	Estimate	SE	95% CI	Standardized Estimate
Intercepts				
Age 20 Alcohol Use	2.500	0.217	[2.07, 2.93]	-
Age 17 Rumination	2.599	0.021	[2.56, 2.64]	-
Growth Rates				
Alcohol Use Slope	0.117	0.151	[-0.18, 0.41]	0.180
Rumination Slope	**0.013**	**0.009**	**[-0.01, 0.03]**	**0.109**
Fixed Effects				
Age 17 Rumination → Age 20 Alcohol Use	**0.157**	**0.079**	**[0.00, 0.31]**	**0.081**
Rumination Change → Age 20 Alcohol Use	1.258	1.493	[-1.67, 4.18]	0.100
Age 17 Rumination → Alcohol Use Change	0.014	0.053	[-0.09, 0.12]	0.016
Rumination Change → Alcohol Use Change	1.239	1.215	[-1.14, 3.62]	0.223
Growth Factor Variances				
Alcohol Use Intercept	2.143	0.193	[1.76, 2.52]	0.988
Alcohol Use Slope	0.405	0.106	[0.20, 0.61]	0.952
Rumination Intercept	0.583	0.033	[0.52, 0.65]	-
Rumination Slope	0.014	0.008	[0.00, 0.03]	-
Growth Factor Covariances				
Alcohol Intercept ↔ Alcohol Slope	**0.607**	**0.127**	**[0.36, 0.86]**	**0.651**
Rumination Intercept ↔Rumination Slope	-0.026	0.014	[-0.05, 0.00]	-0.285
Residual Variances				
Age 18 Alcohol Use	0.643	0.160	[0.33, 0.96]	0.319
Age 19 Alcohol Use	1.157	0.075	[1.01, 1.3]	0.462
Age 20 Alcohol Use	0.564	0.170	[0.23, 0.90]	0.206
Age 17 Rumination	0.526	0.033	[0.46, 0.59]	0.474
Age 18 Rumination	0.504	0.021	[0.46, 0.54]	0.480
Age 19 Rumination	0.515	0.024	[0.47, 0.56]	0.490
Age 20 Rumination	0.515	0.043	[0.43, 0.60]	0.482

Note. Fixed parameters are omitted for parsimony. Chi-square = 19.050, CFI = .998, TLI = .996, RMSEA = .013, SRMR = .017. Effects of substantive interest that are significant at the α = .05 level are indicated in bold.

Table 12. Fit Measures for Parallel Process Growth Models of Rumination and Marijuana Use.

	χ^2	*p*-value	CFI	TLI	RMSEA	RMSEA Lower CL	RMSEA Upper CL	SRMR
Imputation 1	34.661	0.002	0.995	0.993	0.025	0.014	0.035	0.020
Imputation 2	33.622	0.002	0.996	0.994	0.024	0.014	0.034	0.020
Imputation 3	36.620	0.001	0.995	0.993	0.026	0.016	0.036	0.021
Imputation 4	28.069	0.014	0.997	0.995	0.020	0.009	0.031	0.018
Imputation 5	36.870	0.001	0.995	0.993	0.026	0.016	0.036	0.021
Imputation 6	52.243	0.000	0.991	0.987	0.033	0.024	0.043	0.025
Imputation 7	24.327	0.042	0.998	0.997	0.017	0.003	0.029	0.017
Imputation 8	27.603	0.016	0.997	0.995	0.020	0.008	0.031	0.018
Imputation 9	33.675	0.002	0.996	0.994	0.024	0.014	0.034	0.020
Imputation 10	38.318	0.000	0.994	0.992	0.027	0.017	0.037	0.021
Imputation 11	36.944	0.001	0.995	0.992	0.026	0.016	0.036	0.021
Imputation 12	49.406	0.000	0.992	0.988	0.032	0.023	0.042	0.024
Imputation 13	26.449	0.023	0.997	0.996	0.019	0.007	0.030	0.018
Imputation 14	25.363	0.031	0.997	0.996	0.018	0.005	0.029	0.017
Imputation 15	15.608	0.338	1.000	0.999	0.007	0.000	0.021	0.013
Imputation 16	27.022	0.019	0.997	0.996	0.020	0.008	0.030	0.018
Imputation 17	31.838	0.004	0.996	0.994	0.023	0.012	0.033	0.020
Imputation 18	35.486	0.001	0.995	0.992	0.025	0.015	0.035	0.021
Imputation 19	32.189	0.004	0.996	0.994	0.023	0.013	0.034	0.020
Imputation 20	17.919	0.211	0.999	0.999	0.011	0.000	0.024	0.015
Imputation Mean	**32.212**	**-**	**0.996**	**0.994**	**0.022**	**0.012**	**0.033**	**0.019**
FIML	22.621	0.067	0.996	0.994	0.017	0.000	0.028	0.016
Observed Data	33.838	0.002	0.986	0.979	0.039	0.023	0.056	0.021

Note. CFI = Confirmatory Factor Index, TLI = Tucker-Lewis Index, RMSEA = Root Mean Squared Error of Approximation, SRMR = Standardized Root Mean Residual. RMSEA confidence limits are at the 90% level of confidence. Chi-squared values reflect scaled values for FIML and observed data models. Bolded rows indicate the selected model.

Table 13. Parallel Process Growth Curve Model of Rumination and Marijuana Use.

	Estimate	SE	95% CI	Standardized Estimate
Intercepts				
Age 20 Marijuana Use	1.606	0.294	[1.03, 2.18]	
Age 17 Rumination	2.577	0.024	[2.53, 2.62]	
Growth Rates				
Marijuana Use Slope	0.183	0.144	[-0.1, 0.47]	0.255
Rumination Slope	0.045	0.021	[0.00, 0.09]	0.291
Fixed Effects				
Age 17 Rumination → Age 20 Marijuana Use	**0.264**	**0.093**	**[0.08, 0.45]**	**0.107**
Rumination Change → Age 20 Marijuana Use	2.804	1.715	[-0.56, 6.17]	0.217
Age 17 Rumination → Marijuana Use Change	-0.008	0.046	[-0.1, 0.08]	-0.009
Rumination Change → Marijuana Use Change	0.273	0.799	[-1.29, 1.84]	0.062
Growth Factor Variances				
Marijuana Use Intercept	3.592	0.361	[2.89, 4.3]	0.955
Marijuana Use Slope	0.510	0.173	[0.17, 0.85]	0.990
Rumination Intercept	0.619	0.037	[0.55, 0.69]	-
Rumination Slope	0.024	0.011	[0.00, 0.05]	-
Growth Factor Covariances				
Marijuana Intercept ↔ Marijuana Slope	**0.740**	**0.210**	**[0.33, 1.15]**	**0.547**
Rumination Intercept ↔Rumination Slope	-0.043	0.018	[-0.08, -0.01]	-0.355
Residual Variances				
Age 18 Marijuana Use	1.117	0.340	[0.45, 1.78]	0.286
Age 19 Marijuana Use	1.771	0.196	[1.39, 2.15]	0.391
Age 20 Marijuana Use	1.529	0.375	[0.79, 2.26]	0.289
Age 17 Rumination	0.495	0.046	[0.41, 0.58]	0.445
Age 18 Rumination	0.489	0.030	[0.43, 0.55]	0.468
Age 19 Rumination	0.561	0.038	[0.49, 0.64]	0.509
Age 20 Rumination	0.496	0.057	[0.38, 0.61]	0.463

Note. Fixed parameters are omitted for parsimony. Fit measures are provided in Table 13. Significant effects of substantive interest are indicated in bold ($\alpha = .05$).

Table 14. Sexual Minority Status Predicting Rumination and Alcohol Use Growth Factors.

	Estimate	SE	95% CI	Standardized Estimate
Intercepts				
Age 20 Alcohol Use	2.059	0.221	[1.62, 2.49]	1.399
Age 17 Rumination	2.598	0.029	[2.54, 2.66]	3.402
Growth Rates				
Alcohol Use Slope	0.027	0.158	[-0.28, 0.34]	0.042
Rumination Slope	0.019	0.013	[-0.01, 0.04]	0.162
Fixed Effects				
Sexual Minority Status →Age 20 Alcohol Use	0.157	0.249	[-0.33, 0.65]	0.027
Sexual Minority Status → Alcohol Use Change	-0.071	0.153	[-0.37, 0.23]	-0.028
Sexual Minority Status →Age 17 Rumination	0.082	0.083	[-0.08, 0.24]	0.027
Sexual Minority Status → Rumination Change	0.029	0.037	[-0.04, 0.10]	0.062
Age 17 Rumination → Age 20 Alcohol Use	**0.182**	**0.077**	**[0.030, 0.33]**	**0.095**
Rumination Change → Age 20 Alcohol Use	1.410	1.492	[-1.51, 4.33]	0.113
Age 17 Rumination → Alcohol Use Change	0.019	0.053	[-0.08, 0.12]	0.022
Rumination Change → Alcohol Use Change	1.206	1.203	[-1.15, 3.56]	0.219
Growth Factor Variances				
Alcohol Use Intercept	1.964	0.191	[1.59, 2.34]	0.908
Alcohol Use Slope	0.393	0.103	[0.19, 0.60]	0.935
Rumination Intercept	0.583	0.033	[0.52, 0.65]	0.999
Rumination Slope	0.014	0.008	[0.00, 0.03]	0.989
Growth Factor Covariances				
Alcohol Intercept ↔ Alcohol Slope	0.565	0.124	[0.32, 0.81]	0.643
Rumination Intercept ↔Rumination Slope	-0.026	0.014	[-0.05, 0.00]	-0.288
Indirect Effects				
SM Status→Rumination Slope→Age 20 Alcohol Use	0.158	0.588	[-1.56, 2.45]	-
SM Status→Rumination Intercept→Age 20 Alcohol Use	0.011	0.015	[-0.02, 0.07]	-
SM Status→Rumination Slope→Alcohol Use Slope	-0.001	0.003	[-0.01, 0.01]	-
SM Status→Rumination Intercept→Alcohol Use Slope	-0.003	0.007	[-0.03, 0.02]	-

Note. Fixed parameters, covariate effects, and residual variances are omitted for parsimony. Chi-square = 30.55, CFI = .996, TLI = .993, RMSEA = .014, SRMR = .015. Effects of substantive interest that are significant at the $\alpha = .05$ level are indicated in bold.

Table 15. Fit measures for Sexual Minority Status Predicting Rumination and Marijuana Use Growth Factors.

	χ^2	p-value	CFI	TLI	RMSEA	RMSEA Lower CL	RMSEA Upper CL	SRMR
Imputation 1	53.280	0.000	0.994	0.988	0.023	0.015	0.031	0.018
Imputation 2	53.706	0.000	0.994	0.988	0.023	0.015	0.032	0.019
Imputation 3	56.828	0.000	0.993	0.987	0.025	0.017	0.033	0.019
Imputation 4	36.420	0.037	0.997	0.995	0.015	0.004	0.025	0.015
Imputation 5	50.248	0.001	0.994	0.989	0.022	0.014	0.030	0.018
Imputation 6	71.711	0.000	0.990	0.980	0.029	0.022	0.037	0.022
Imputation 7	41.535	0.010	0.996	0.992	0.018	0.009	0.027	0.017
Imputation 8	40.468	0.014	0.996	0.993	0.018	0.008	0.026	0.016
Imputation 9	58.283	0.000	0.993	0.986	0.025	0.017	0.033	0.020
Imputation 10	49.159	0.001	0.994	0.989	0.022	0.013	0.030	0.018
Imputation 11	58.109	0.000	0.993	0.986	0.025	0.017	0.033	0.020
Imputation 12	66.303	0.000	0.991	0.981	0.028	0.020	0.036	0.021
Imputation 13	38.689	0.021	0.997	0.994	0.017	0.006	0.026	0.016
Imputation 14	32.078	0.099	0.998	0.996	0.013	0.000	0.022	0.014
Imputation 15	34.057	0.064	0.998	0.995	0.014	0.000	0.023	0.015
Imputation 16	39.577	0.017	0.997	0.993	0.017	0.007	0.026	0.016
Imputation 17	51.332	0.001	0.994	0.989	0.022	0.014	0.031	0.018
Imputation 18	52.631	0.000	0.993	0.987	0.023	0.015	0.031	0.019
Imputation 19	42.966	0.007	0.996	0.992	0.019	0.010	0.027	0.017
Imputation 20	34.054	0.064	0.998	0.996	0.014	0.000	0.023	0.015
Imputation Mean	**48.072**	**-**	**0.995**	**0.990**	**0.021**	**0.011**	**0.029**	**0.018**
FIML	34.386	0.060	0.996	0.991	0.014	0.000	0.023	0.015
Observed Data	44.368	0.005	0.987	0.974	0.033	0.018	0.046	0.019

Note. CFI = Confirmatory Factor Index, TLI = Tucker-Lewis Index, RMSEA = Root Mean Squared Error of Approximation, SRMR = Standardized Root Mean Residual. RMSEA confidence limits are at the 90% level of confidence. Chi-squared values reflect scaled values for FIML and observed data models. Bolded rows indicate the selected model.

Table 17. Sexual Minority Status Predicting Rumination and Marijuana Use Growth Factors.

	Estimate	SE	95% CI	Standardized Estimate
Intercepts				
Age 20 Marijuana Use	1.673	0.309	[1.07, 2.28]	0.863
Age 17 Rumination	2.578	0.030	[2.52, 2.64]	3.278
Growth Rates				
Marijuana Use Slope	0.187	0.152	[-0.11, 0.49]	0.262
Rumination Slope	0.052	0.021	[0.01, 0.09]	0.341
Fixed Effects				
Sexual Minority Status →Age 20 Marijuana Use	**0.905**	**0.330**	**[0.26, 1.55]**	**0.119**
Sexual Minority Status → Marijuana Use Change	-0.054	0.187	[-0.42, 0.31]	-0.019
Sexual Minority Status →Age 17 Rumination	0.063	0.079	[-0.09, 0.22]	0.021
Sexual Minority Status → Rumination Change	0.026	0.055	[-0.08, 0.13]	0.041
Age 17 Rumination → Age 20 Marijuana Use	**0.248**	**0.093**	**[0.07, 0.43]**	**0.101**
Rumination Change → Age 20 Marijuana Use	2.680	1.705	[-0.66, 6.02]	0.207
Age 17 Rumination → Marijuana Use Change	-0.007	0.046	[-0.10, 0.08]	-0.008
Rumination Change → Marijuana Use Change	0.283	0.820	[-1.33, 1.89]	0.064
Growth Factor Variances				
Marijuana Use Intercept	3.521	0.358	[2.82, 4.22]	0.936
Marijuana Use Slope	0.507	0.173	[0.17, 0.85]	0.986
Rumination Intercept	0.618	0.037	[0.55, 0.69]	0.999
Rumination Slope	0.023	0.011	[0.00, 0.04]	0.984
Growth Factor Covariances				
Marijuana Intercept ↔ Marijuana Slope	0.740	0.211	[0.33, 1.15]	0.554
Rumination Intercept ↔Rumination Slope	-0.043	0.018	[-0.08, -0.01]	-0.358
Indirect Effects				
SM Status→Rumination Slope→Age 20 Marijuana Use	0.069	0.18	[-0.44, 0.70]	-
SM Status→Rumination Intercept→Age 20 Marijuana Use	0.016	0.022	[-0.04, 0.09]	-
SM Status→Rumination Slope→Marijuana Use Slope	0.007	0.052	[-0.16, 0.21]	-
SM Status→Rumination Intercept→Marijuana Use Slope	0.000	0.005	[-0.02, 0.02]	-

Note. Fixed parameters, covariate effects, and residual variances are omitted for parsimony. Fit Measures are provided in Table X. Effects of substantive interest that are significant at the α = .05 level are indicated in bold.

Table 18. Sexual Minority Status and Adversity Predicting Rumination and Alcohol Use Growth Factors.

	Estimate	SE	Confidence Interval	Standardized Estimate
Intercepts				
Age 20 Alcohol Use	2.157	0.223	[1.72, 2.59]	1.469
Age 17 Rumination	2.630	0.024	[2.58, 2.68]	3.577
Growth Rates				
Alcohol Use Slope	-0.017	0.149	[-0.31, 0.28]	-0.026
Rumination Slope	0.015	0.013	[-0.01, 0.04]	0.126
Fixed Effects of Sexual Minority Status				
Sexual Minority Status→Social Stress	**0.517**	**0.108**	**[0.30, 0.73]**	**0.132**
Sexual Minority Status→Abuse History	**0.095**	**0.031**	**[0.03, 0.16]**	**0.087**
Sexual Minority Status →Age 17 Rumination	-0.031	0.071	[-0.17, 0.11]	-0.011
Sexual Minority Status → Rumination Change	0.041	0.037	[-0.03, 0.11]	0.086
Sexual Minority Status →Age 20 Alcohol Use	0.106	0.258	[-0.40, 0.61]	0.018
Sexual Minority Status → Alcohol Use Change	-0.045	0.154	[-0.35, 0.26]	-0.018
Fixed Effects of Social Stress				
Social Stress→Age 18 Rumination	**0.240**	**0.019**	**[0.20, 0.28]**	**0.327**
Social Stress→Rumination Change	**-0.025**	**0.009**	**[-0.04, -0.01]**	**-0.204**
Social Stress→Age 20 Alcohol Use	0.087	0.062	[-0.03, 0.21]	0.059
Social Stress→Alcohol Use Change	-0.039	0.040	[-0.12, 0.04]	-0.060
Fixed Effects of Abuse History				
Abuse History→Age 18 Rumination	-0.031	0.060	[-0.15, 0.09]	-0.012
Abuse History→Rumination Change	0.029	0.031	[-0.03, 0.09]	0.066
Abuse History→Age 20 Alcohol Use	0.154	0.175	[-0.19, 0.50]	0.029
Abuse History→Alcohol Use Change	0.059	0.114	[-0.17, 0.28]	0.025
Fixed Effects of Rumination Growth Factors				
Age 17 Rumination → Age 20 Alcohol Use	0.140	0.080	[-0.02, 0.30]	0.070
Rumination Change → Age 20 Alcohol Use	1.306	1.414	[-1.47, 4.08]	0.109
Age 17 Rumination → Alcohol Use Change	0.034	0.051	[-0.07, 0.14]	0.039
Rumination Change → Alcohol Use Change	0.964	1.050	[-1.09, 3.02]	0.184
Growth Factor Variances				

Alcohol Use Intercept	1.947	0.192	[1.57, 2.32]	0.903
Alcohol Use Slope	0.390	0.102	[0.19, 0.59]	0.941
Rumination Intercept	0.483	0.020	[0.45, 0.52]	0.894
Rumination Slope	0.014	0.008	[0, 0.03]	0.947
Growth Factor Covariances				
Alcohol Intercept ↔ Alcohol Slope	0.562	0.124	[0.32, 0.80]	0.644
Rumination Intercept ↔Rumination Slope	-0.006	0.008	[-0.02, 0.01]	-0.071
Two-Path Indirect Effects				
SM Status→Social Stress→Age 18 Rumination	**0.124**	**0.028**	**[0.056, 0.200]**	-
SM Status→Abuse History→Age 18 Rumination	-0.003	0.006	[-0.022, 0.013]	-
SM Status→Social Stress→Rumination Change	**-0.013**	**0.005**	**[-0.030, -0.001]**	-
SM Status→Abuse History→Rumination Change	0.003	0.003	[-0.005, 0.013]	-
SM Status→Social Stress→Age 20 Alcohol Use	0.045	0.034	[-0.038, 0.145]	-
SM Status→Abuse History→Age 20 Alcohol Use	0.015	0.018	[-0.031, 0.071]	-
SM Status→Social Stress→Alcohol Use Change	-0.020	0.021	[-0.082, 0.035]	-
SM Status→Abuse History→Alcohol Use Change	0.006	0.012	[-0.026, 0.041]	-
Three-Path Effects				
SM Status→Social Stress→Age 18 Rumination→Age 20 Alcohol Use	0.017	0.011	[-0.008, 0.049]	-
SM Status→Abuse History→Age 18 Rumination→Age 20 Alcohol Use	0.000	0.001	[-0.004, 0.003]	-
SM Status→Social Stress→Rumination Change→Age 20 Alcohol Use	-0.017	0.021	[-0.091, 0.036]	-
SM Status→Abuse History→Rumination Change→Age 20 Alcohol Use	0.004	0.007	[-0.014, 0.035]	-
SM Status→Social Stress→Age 18 Rumination→Alcohol Use Change	0.004	0.007	[-0.013, 0.023]	-
SM Status→Abuse History→Age 18 Rumination→Alcohol Use Change	0.000	0.000	[-0.002, 0.001]	-
SM Status→Social Stress→Rumination Change→Alcohol Use Change	-0.013	0.016	[-0.067, 0.027]	-
SM Status→Abuse History→Rumination Change→Alcohol Use Change	0.003	0.005	[-0.011, 0.025]	-

Note. Fixed parameters, covariate effects, and residual variances are omitted for parsimony. Chi-square = 30.55, CFI = .996, TLI = .993, RMSEA = .014, SRMR = .015. Significant effects of substantive interest are indicated in bold. Confidence intervals reflect the 95% level for all direct effects and 99% level for indirect effects.

Table 19. Fit measures for Sexual Minority Status and Adversity Predicting Rumination and Marijuana Use Growth Factors.

	χ^2	*p*-value	CFI	TLI	RMSEA	RMSEA Lower CL	RMSEA Upper CL	SRMR
Imputation 1	63.964	0.000	0.994	0.986	0.022	0.015	0.030	0.017
Imputation 2	60.838	0.000	0.995	0.988	0.021	0.014	0.029	0.017
Imputation 3	60.186	0.001	0.995	0.988	0.021	0.013	0.028	0.017
Imputation 4	45.390	0.027	0.997	0.993	0.015	0.005	0.023	0.015
Imputation 5	56.568	0.002	0.995	0.989	0.020	0.012	0.027	0.016
Imputation 6	79.352	0.000	0.991	0.979	0.027	0.020	0.034	0.019
Imputation 7	48.126	0.014	0.997	0.992	0.016	0.007	0.024	0.015
Imputation 8	48.323	0.014	0.997	0.992	0.017	0.008	0.024	0.015
Imputation 9	72.235	0.000	0.993	0.983	0.025	0.018	0.032	0.019
Imputation 10	55.145	0.002	0.995	0.989	0.019	0.011	0.027	0.016
Imputation 11	62.871	0.000	0.994	0.986	0.022	0.014	0.029	0.017
Imputation 12	71.610	0.000	0.992	0.982	0.025	0.017	0.032	0.018
Imputation 13	45.319	0.027	0.997	0.994	0.015	0.005	0.023	0.015
Imputation 14	38.249	0.117	0.998	0.996	0.011	0.000	0.020	0.013
Imputation 15	43.496	0.041	0.997	0.994	0.014	0.003	0.023	0.014
Imputation 16	49.979	0.009	0.996	0.992	0.017	0.009	0.025	0.015
Imputation 17	56.188	0.002	0.995	0.989	0.020	0.012	0.027	0.016
Imputation 18	60.694	0.001	0.994	0.986	0.021	0.014	0.029	0.017
Imputation 19	54.959	0.003	0.995	0.990	0.019	0.011	0.027	0.016
Imputation 20	40.665	0.074	0.998	0.995	0.013	0.000	0.021	0.014
Imputation Mean	**55.708**	**-**	**0.995**	**0.989**	**0.019**	**0.010**	**0.027**	**0.016**
FIML	44.814	0.031	0.994	0.987	0.015	0.005	0.023	0.014
Observed Data	55.922	0.002	0.984	0.963	0.033	0.020	0.045	0.018

Note. CFI = Confirmatory Factor Index, TLI = Tucker-Lewis Index, RMSEA = Root Mean Squared Error of Approximation, SRMR = Standardized Root Mean Residual. RMSEA confidence limits are at the 90% level of confidence. Chi-squared values reflect scaled values for FIML and observed data models. Bolded rows indicate the selected model.

Table 20. Sexual Minority Status and Adversity Predicting Rumination and Marijuana Use Growth Factors.

	Estimate	SE	Confidence Interval	Standardized Estimate
Intercepts				
Age 20 Marijuana Use	1.943	0.318	[1.32, 2.57]	-
Age 17 Rumination	2.643	0.022	[2.60, 2.69]	-
Growth Rates				
Marijuana Use Slope	0.100	0.157	[-0.21, 0.41]	0.139
Rumination Slope	0.050	0.022	[0.01, 0.09]	0.328
Fixed Effects of Sexual Minority Status				
Sexual Minority Status→Social Stress	**0.562**	**0.128**	**[0.31, 0.81]**	**0.144**
Sexual Minority Status→Abuse History	**0.093**	**0.032**	**[0.03, 0.16]**	**0.085**
Sexual Minority Status→Age 17 Rumination	-0.059	0.064	[-0.18, 0.07]	-0.020
Sexual Minority Status→Rumination Change	0.041	0.056	[-0.07, 0.15]	0.067
Sexual Minority Status→Age 20 Marijuana Use	**0.730**	**0.345**	**[0.05, 1.41]**	**0.096**
Sexual Minority Status→Marijuana Use Change	-0.019	0.190	[-0.39, 0.35]	-0.006
Fixed Effects of Social Stress				
Social Stress→Age 18 Rumination	**0.272**	**0.020**	**[0.23, 0.31]**	**0.365**
Social Stress→Rumination Change	**-0.029**	**0.013**	**[-0.05, 0.00]**	**-0.196**
Social Stress→Age 20 Marijuana Use	**0.253**	**0.100**	**[0.06, 0.45]**	**0.253**
Social Stress→Marijuana Use Change	-0.073	0.054	[-0.18, 0.03]	-0.102
Fixed Effects of Abuse History				
Abuse History→Age 18 Rumination	-0.046	0.051	[-0.15, 0.05]	-0.017
Abuse History→Rumination Change	0.013	0.040	[-0.07, 0.09]	0.023
Abuse History→Age 20 Marijuana Use	0.395	0.255	[-0.10, 0.90]	0.057
Abuse History→Marijuana Use Change	0.054	0.154	[-0.25, 0.36]	0.022
Fixed Effects of Rumination Growth Factors				
Age 17 Rumination→Age 20 Marijuana Use	0.132	0.099	[-0.06, 0.33]	0.051
Rumination Change→Age 20 Marijuana Use	2.673	1.840	[-0.94, 6.28]	0.206
Age 17 Rumination→Marijuana Use Change	0.025	0.050	[-0.07, 0.12]	0.026
Rumination Change→Marijuana Use Change	0.222	0.828	[-1.40, 1.85]	0.052
Growth Factor Variances				
Marijuana Use Intercept	3.446	0.370	[2.72, 4.17]	0.916
Marijuana Use Slope	0.502	0.173	[0.16, 0.84]	0.975
Rumination Intercept	0.484	0.018	[0.45, 0.52]	0.869

Rumination Slope	0.022	0.011	[0.00, 0.04]	0.943
Growth Factor Covariances				
Marijuana Intercept ↔ Marijuana Slope	0.753	0.212	[0.34, 1.17]	0.573
Rumination Intercept ↔Rumination Slope	-0.012	0.010	[-0.03, 0.01]	-0.116
Two-Path Indirect Effects				
SM Status→Social Stress→Age 18 Rumination	**0.153**	**0.037**	**[0.062, 0.253]**	-
SM Status→Abuse History→Age 18 Rumination	-0.004	0.005	[-0.021, 0.009]	-
SM Status→Social Stress→Rumination Change	-0.017	0.008	[-0.042, 0.002]	-
SM Status→Abuse History→Rumination Change	0.001	0.004	[-0.010, 0.013]	-
SM Status→Social Stress→Age 20 Marijuana Use	0.142	0.066	[-0.003, 0.345]	-
SM Status→Abuse History→Age 20 Marijuana Use	0.037	0.028	[-0.025, 0.128]	-
SM Status→Social Stress→Marijuana Use Change	-0.041	0.033	[-0.138, 0.039]	-
SM Status→Abuse History→Marijuana Use Change	0.005	0.015	[-0.037, 0.051]	-
Three-Path Indirect Effects				
SM Status→Social Stress→Age 18 Rumination→Age 20 Marijuana Use	0.020	0.012	[-0.008, 0.056]	-
SM Status→Abuse History→Age 18 Rumination→Age 20 Marijuana Use	-0.001	0.001	[-0.004, 0.001]	-
SM Status→Social Stress→Rumination Change→Age 20 Marijuana Use	-0.045	0.036	[-0.173, 0.026]	-
SM Status→Abuse History→Rumination Change→Age 20 Marijuana Use	0.003	0.011	[-0.030, 0.043]	-
SM Status→Social Stress→Age 18 Rumination→Marijuana Use Change	0.004	0.006	[-0.012, 0.022]	-
SM Status→Abuse History→Age 18 Rumination→Marijuana Use Change	0.000	0.000	[-0.001, 0.001]	-
SM Status→Social Stress→Rumination Change→Marijuana Use Change	-0.003	0.013	[-0.046, 0.036]	-
SM Status→Abuse History→Rumination Change→Marijuana Use Change	0.000	0.003	[-0.009, 0.011]	-

Note. Fixed parameters, covariate effects, and residual variances are omitted for parsimony. Fit measures are provided in Table 19. Significant effects of substantive interest are indicated in bold. Confidence intervals reflect the 95% level for all direct effects and 99% level for indirect effects.

Table 21. Fit Measures for Aim 3 Multi-Group Structural Equation Model for Alcohol Use.

	χ^2	*p*-value	CFI	TLI	RMSEA	RMSEA Lower CL	RMSEA Upper CL	SRMR
Imputation 1	20.577	0.607	1.000	1.002	0.000	0.000	0.021	0.012
Imputation 2	15.121	0.890	1.000	1.006	0.000	0.000	0.011	0.010
Imputation 3	20.293	0.624	1.000	1.002	0.000	0.000	0.020	0.014
Imputation 4	21.385	0.558	1.000	1.001	0.000	0.000	0.022	0.014
Imputation 5	12.823	0.956	1.000	1.009	0.000	0.000	0.000	0.009
Imputation 6	17.219	0.798	1.000	1.005	0.000	0.000	0.016	0.011
Imputation 7	16.506	0.833	1.000	1.005	0.000	0.000	0.014	0.010
Imputation 8	21.081	0.576	1.000	1.002	0.000	0.000	0.021	0.011
Imputation 9	12.022	0.970	1.000	1.009	0.000	0.000	0.000	0.008
Imputation 10	21.749	0.535	1.000	1.001	0.000	0.000	0.022	0.014
Imputation 11	8.768	0.997	1.000	1.011	0.000	0.000	0.000	0.006
Imputation 12	24.424	0.381	0.999	0.999	0.007	0.000	0.025	0.014
Imputation 13	15.495	0.876	1.000	1.006	0.000	0.000	0.012	0.011
Imputation 14	11.111	0.982	1.000	1.009	0.000	0.000	0.000	0.007
Imputation 15	20.131	0.634	1.000	1.002	0.000	0.000	0.020	0.011
Imputation 16	27.859	0.221	0.998	0.996	0.013	0.000	0.028	0.016
Imputation 17	15.295	0.884	1.000	1.006	0.000	0.000	0.012	0.009
Imputation 18	20.982	0.582	1.000	1.002	0.000	0.000	0.021	0.014
Imputation 19	21.258	0.565	1.000	1.001	0.000	0.000	0.021	0.012
Imputation 20	15.705	0.868	1.000	1.006	0.000	0.000	0.013	0.011
Imputation Mean	**17.990**	**-**	**1.000**	**1.004**	**0.001**	**0.000**	**0.015**	**0.011**
FIML	5.047	0.410	1.000	1.000	0.002	0.000	0.028	0.009
Observed Data	5.025	0.413	1.000	1.000	0.004	0.000	0.086	0.014

Note. CFI = Confirmatory Factor Index, TLI = Tucker-Lewis Index, RMSEA = Root Mean Squared Error of Approximation, SRMR = Standardized Root Mean Residual. RMSEA confidence limits are at the 90% level of confidence. Chi-squared values reflect scaled values for FIML and observed data models. Bolded rows indicate the selected model.

Table 22. Fit Measures for Aim 3 Multi-Group Structural Equation Model for Marijuana Use.

Imputation	χ^2	p-value	CFI	TLI	RMSEA	RMSEA Lower CL	RMSEA Upper CL	SRMR
1	16.099	0.851	1.000	1.006	0.000	0.000	0.013	0.012
2	23.276	0.445	1.000	1.000	0.003	0.000	0.024	0.013
3	15.951	0.857	1.000	1.006	0.000	0.000	0.013	0.011
4	24.301	0.387	0.999	0.999	0.007	0.000	0.025	0.015
5	13.881	0.930	1.000	1.008	0.000	0.000	0.007	0.011
6	19.163	0.692	1.000	1.003	0.000	0.000	0.019	0.011
7	26.921	0.259	0.998	0.997	0.012	0.000	0.027	0.015
8	27.418	0.239	0.997	0.996	0.013	0.000	0.028	0.015
9	18.063	0.754	1.000	1.004	0.000	0.000	0.017	0.012
10	21.818	0.531	1.000	1.001	0.000	0.000	0.022	0.014
11	16.095	0.851	1.000	1.006	0.000	0.000	0.013	0.011
12	38.206	0.024	0.992	0.987	0.023	0.008	0.036	0.019
13	16.356	0.840	1.000	1.006	0.000	0.000	0.014	0.012
14	10.464	0.988	1.000	1.011	0.000	0.000	0.000	0.009
15	16.319	0.841	1.000	1.006	0.000	0.000	0.014	0.011
16	19.445	0.675	1.000	1.003	0.000	0.000	0.019	0.012
17	23.858	0.412	1.000	0.999	0.006	0.000	0.024	0.015
18	22.585	0.485	1.000	1.000	0.000	0.000	0.023	0.013
19	25.815	0.310	0.998	0.997	0.010	0.000	0.026	0.015
20	13.147	0.949	1.000	1.008	0.000	0.000	0.002	0.010
Imputation Mean	**20.459**	**-**	**0.999**	**1.002**	**0.004**	**0.000**	**0.018**	**0.013**
FIML	12.812	0.025	0.994	0.977	0.025	0.009	0.042	0.012
Observed Data	7.585	0.181	0.997	0.988	0.024	0.000	0.054	0.013

Note. CFI = Confirmatory Factor Index, TLI = Tucker-Lewis Index, RMSEA = Root Mean Squared Error of Approximation, SRMR = Standardized Root Mean Residual. RMSEA confidence limits are at the 90% level of confidence. Chi-squared values reflect scaled values for FIML and observed data models. Bolded rows indicate the selected model.

Table 23. Tests of Moderation by Sexual Minority Status.

Parameter Fixed Across Sexual Minority Groups	$\Delta\overline{\chi^2}$	Δdf	p-value
Alcohol Use			
Age 18 Rumination→Age 20 Alcohol Use	1.045	1	0.307
Age 17 Life Social Stress → Age 20 Alcohol Use	1.252	1	0.263
Abuse History → Age 20 Alcohol Use	0.477	1	0.490
Age 18 Social Support→Age 20 Alcohol Use	0.813	1	0.367
Age 17 Life Social Stress → Age 18 Rumination	0.161	1	0.688
Abuse → Age 18 Rumination	0.633	1	0.426
Age 17 Life Social Stress → Age 18 Social Support	0.370	1	0.543
Abuse History → Age 18 Social Support	0.838	1	0.360
Life Social Stress→Social Support→Alcohol Use	1.231	2	0.540
Abuse→Social Support→Alcohol Use	1.655	2	0.437
Life Social Stress→Rumination→Alcohol Use	0.873	2	0.646
Abuse→Rumination→Alcohol Use	1.647	2	0.439
Marijuana Use			
Age 18 Rumination→Age 20 Marijuana Use	0.223	1	0.636
Age 17 Life Social Stress→Age 20 Marijuana Use	2.015	1	0.156
Abuse History→Age 20 Marijuana Use	2.643	1	0.104
Age 18 Social Support→Age 20 Marijuana Use	1.290	1	0.256
Age 17 Life Social Stress→Age 18 Rumination	0.151	1	0.698
Abuse→Age 18 Rumination	0.585	1	0.444
Age 17 Life Social Stress→Age 18 Social Support	0.442	1	0.506
Abuse History→Age 18 Social Support	0.773	1	0.379
Life Social Stress→Social Support→Marijuana Use	1.754	2	0.416
Abuse→Social Support→Marijuana Use	2.065	2	0.356
Life Social Stress→Rumination→Marijuana Use	0.105	2	0.949
Abuse→Rumination→Marijuana Use	0.800	2	0.670

Note: $\Delta\overline{\chi^2}$ = change in chi-squared value when parameter is fixed versus estimated freely across across groups; Δdf = change in degrees of freedom.

Table 24. Aim 3 Multi-Group Structural Equation Model for Alcohol Use.

	Heterosexual Women				Sexual Minority Women			
	Est.	SE	CI	Std. Est.	Est.	SE	CI	Std. Est.
Intercepts								
Social Stress	-0.040	0.021	[-0.08, 0.00]	-0.041	0.523	0.113	[0.30, 0.74]	0.405
Abuse History	0.078	0.006	[0.07, 0.09]	0.291	0.172	0.030	[0.11, 0.23]	0.456
Age 17 Social Support	16.078	0.081	[15.92, 16.24]	4.399	14.685	0.338	[14.02, 15.35]	3.570
Age 18 Social Support	8.361	0.697	[6.99, 9.73]	2.414	7.194	0.77	[5.68, 8.70]	1.734
Age 17 Rumination	2.606	0.024	[2.56, 2.65]	2.465	2.632	0.089	[2.46, 2.81]	2.561
Age 18 Rumination	1.462	0.145	[1.18, 1.75]	1.430	1.540	0.167	[1.21, 1.87]	1.512
Age 18 Alcohol Use	2.738	0.053	[2.63, 2.84]	1.918	2.995	0.145	[2.71, 3.28]	1.985
Age 20 Alcohol Use	0.955	0.512	[-0.05, 1.96]	0.539	0.726	0.518	[-0.29, 1.74]	0.390
Fixed Effects								
Abuse History→Age 18 Social Support	0.230[a]	0.355[a]	[-0.46, 0.93][a]	0.018[a]	0.231[a]	0.355[a]	[-0.46, 0.93][a]	0.018[a]
Social Stress→Age 18 Social Support	**-0.473**[a]	**0.137**[a]	**[-0.74, -0.20]**[a]	**-0.131**[a]	**-0.473**[a]	**0.137**[a]	**[-0.74, -0.20]**[a]	**-0.131**[a]
Age 17 Social Support→Age 18 Social Support	**0.514**[a]	**0.040**[a]	**[0.44, 0.59]**[a]	**0.542**[a]	**0.514**[a]	**0.040**[a]	**[0.44, 0.59]**[a]	**0.542**[a]
Age 17 Rumination→Age 18 Social Support	-0.044[a]	0.094[a]	[-0.23, 0.14][a]	-0.013[a]	-0.044[a]	0.094[a]	[-0.23, 0.14][a]	-0.013[a]
Abuse History→Age 20 Alcohol Use	0.325[a]	0.200[a]	[-0.07, 0.72][a]	0.049[a]	0.325[a]	0.200[a]	[-0.07, 0.72][a]	0.049[a]
Social Stress→Age 20 Alcohol Use	-0.014[a]	0.069[a]	[-0.15, 0.12][a]	-0.008[a]	-0.014[a]	0.069[a]	[-0.15, 0.12][a]	-0.008[a]
Age 18 Social Support→Age 20 Alcohol Use	-0.006[a]	0.023[a]	[-0.05, 0.04][a]	-0.012[a]	-0.006[a]	0.023[a]	[-0.05, 0.04][a]	-0.012[a]
Age 18 Alcohol Use→Age 20 Alcohol Use	**0.532**[a]	**0.061**[a]	**[0.41, 0.65]**[a]	**0.426**[a]	**0.532**[a]	**0.061**[a]	**[0.41, 0.65]**[a]	**0.426**[a]
Age 18 Rumination→Age 20 Alcohol Use	0.065[a]	0.054[a]	[-0.04, 0.17][a]	0.037[a]	0.065[a]	0.054[a]	[-0.04, 0.17][a]	0.037[a]
Abuse History→Age 18 Rumination	0.097[a]	0.086[a]	[-0.07, 0.26][a]	0.025[a]	0.097[a]	0.086[a]	[-0.07, 0.26][a]	0.025[a]
Social Stress→Age 18 Rumination	**0.089**[a]	**0.034**[a]	**[0.02, 0.16]**[a]	**0.084**[a]	**0.089**[a]	**0.034**[a]	**[0.02, 0.16]**[a]	**0.084**[a]
Age 17 Social Support→Age 18 Rumination	-0.006[a]	0.008[a]	[-0.02, 0.01][a]	-0.022[a]	-0.006[a]	0.008[a]	[-0.02, 0.01][a]	-0.022[a]
Age 17 Rumination→Age 18 Rumination	**0.468**[a]	**0.028**[a]	**[0.41, 0.52]**[a]	**0.484**[a]	**0.468**[a]	**0.028**[a]	**[0.41, 0.52]**[a]	**0.484**[a]
Covariances								

Age 17 Social Support ↔ Abuse History	-0.045	0.022	[-0.09, 0.00]	-0.046	-0.321	0.137	[-0.59, -0.05]	-0.206
Age 17 Rumination ↔ Abuse History	0.002	0.006	[-0.01, 0.01]	0.006	-0.015	0.025	[-0.06, 0.03]	-0.040
Social Stress ↔ Abuse History	0.030	0.007	[0.02, 0.04]	0.117	0.042	0.042	[-0.04, 0.12]	0.086
Social Stress ↔ Age 17 Social Support	-1.232	0.100	[-1.43, -1.04]	-0.351	-1.389	0.374	[-2.12, -0.66]	-0.261
Social Stress ↔ Age 17 Rumination	0.295	0.024	[0.25, 0.34]	0.291	0.481	0.109	[0.27, 0.69]	0.362
Age 17 Social Support ↔ Age 17 Rumination	-0.463	0.086	[-0.63, -0.29]	-0.120	-0.87	0.266	[-1.39, -0.35]	-0.206
Abuse History ↔ Age 18 Alcohol Use	0.004	0.013	[-0.02, 0.03]	0.010	0.028	0.048	[-0.07, 0.12]	0.050
Social Stress ↔ Age 18 Alcohol Use	0.199	0.047	[0.11, 0.29]	0.145	0.243	0.194	[-0.14, 0.62]	0.124
Age 17 Social Support ↔ Age 18 Alcohol Use	-0.602	0.186	[-0.97, -0.24]	-0.115	-1.467	0.551	[-2.55, -0.39]	-0.235
Age 17 Rumination ↔ Age 18 Alcohol Use	0.114	0.038	[0.04, 0.19]	0.075	-0.009	0.152	[-0.31, 0.29]	-0.006
Residual Variances								
Age 18 Social Support	7.642	0.643	[6.38, 8.90]	0.637	11.723	2.082	[7.64, 15.81]	0.680
Age 18 Rumination	0.763	0.036	[0.69, 0.83]	0.730	0.746	0.104	[0.54, 0.95]	0.718
Age 20 Alcohol Use	2.566	0.171	[2.23, 2.90]	0.809	2.77	0.442	[1.90, 3.64]	0.799
Indirect Effects								
Stress →A18 Rumination→A20 Alcohol Use	0.006[a]	0.008[a]	[-0.014, 0.039][a]	-	0.006[a]	0.008[a]	[-0.014, 0.039][a]	-
Abuse →A18 Rumination→A20 Alcohol Use	0.006[a]	0.006[a]	[-0.007, 0.024][a]	-	0.006[a]	0.006[a]	[-0.007, 0.024][a]	-
Stress →A18 Soc. Support→A20 Alcohol Use	0.003[a]	0.011[a]	[-0.029, 0.036][a]	-	0.003[a]	0.011[a]	[-0.029, 0.036][a]	-
Abuse →A18 Soc. Support →A20 Alcohol Use	-0.001[a]	0.010[a]	[-0.040, 0.031][a]	-	-0.001[a]	0.010[a]	[-0.040, 0.031][a]	-

Note. Fit Measures are provided in Table 21. Exogenous variable residual variances are omitted for parsimony. Fixed effects significant at the α = .05 level are indicated in bold.

[a] Parameters were fixed to equivalence across groups.

Table 25. Aim 3 Multi-Group Structural Equation Model for Marijuana Use.

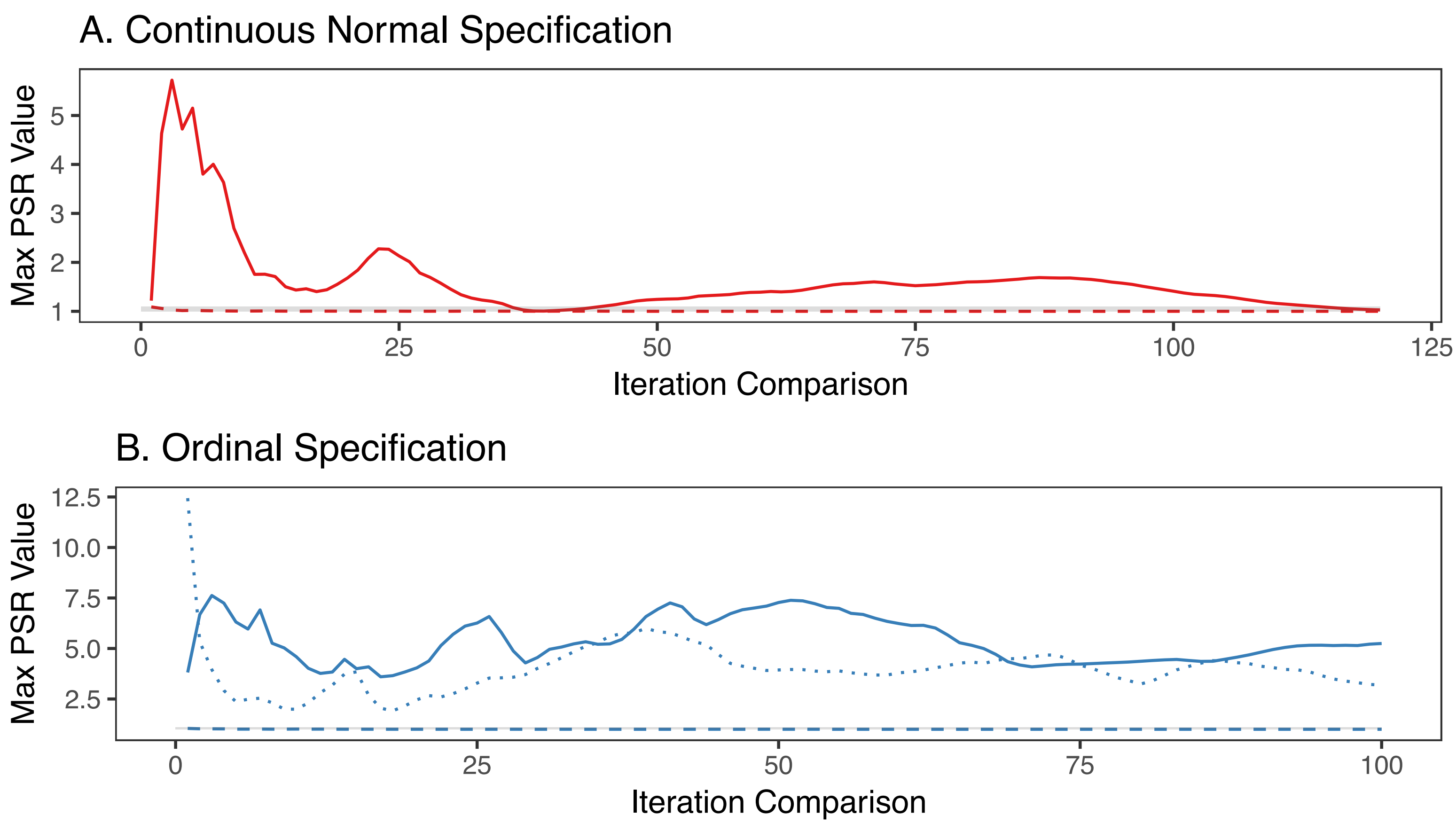
	Heterosexual Women				Sexual Minority Women			
	Est.	SE	CI	Std. Est.	Est.	SE	CI	Std. Est.
Intercepts								
Social Stress	-0.040	0.021	[-0.08, 0.00]	-0.041	0.523	0.113	[0.3, 0.74]	0.405
Abuse History	0.078	0.006	[0.07, 0.09]	0.290	0.172	0.030	[0.11, 0.23]	0.457
Age 17 Social Support	16.078	0.081	[15.92, 16.24]	4.395	14.685	0.338	[14.02, 15.35]	3.570
Age 18 Social Support	8.422	0.705	[7.04, 9.80]	2.431	7.268	0.774	[5.75, 8.79]	1.753
Age 17 Rumination	2.606	0.024	[2.56, 2.65]	2.464	2.632	0.089	[2.46, 2.81]	2.559
Age 18 Rumination	1.456	0.146	[1.17, 1.74]	1.424	1.530	0.168	[1.20, 1.86]	1.501
Age 18 Marijuana Use	1.980	0.044	[1.89, 2.07]	1.041	3.128	0.212	[2.71, 3.54]	1.261
Age 20 Marijuana Use	1.092	0.441	[0.23, 1.96]	0.484	1.480	0.510	[0.48, 2.48]	0.565
Fixed Effects								
Abuse History→Age 18 Social Support	0.203[a]	0.35[a]	[-0.49, 0.90][a]	0.016[a]	0.203[a]	0.35[a]	[-0.49, 0.90][a]	0.016[a]
Social Stress→Age 18 Social Support	**-0.508[a]**	**0.139[a]**	**[-0.78, -0.24][a]**	**-0.140[a]**	**-0.508[a]**	**0.139[a]**	**[-0.78, -0.24][a]**	**-0.140[a]**
Age 17 Social Support→Age 18 Social Support	**0.508[a]**	**0.040[a]**	**[0.43, 0.59][a]**	**0.536[a]**	**0.508[a]**	**0.040[a]**	**[0.43, 0.59][a]**	**0.536[a]**
Age 17 Rumination→Age 18 Social Support	-0.030[a]	0.094[a]	[-0.21, 0.15][a]	-0.009[a]	-0.030[a]	0.094[a]	[-0.21, 0.15][a]	-0.009[a]
Abuse History→Age 20 Marijuana Use	0.288[a]	0.274[a]	[-0.25, 0.82][a]	0.034[a]	0.288[a]	0.274[a]	[-0.25, 0.82][a]	0.034[a]
Social Stress→Age 20 Marijuana Use	-0.061[a]	0.093[a]	[-0.24, 0.12][a]	-0.026[a]	-0.061[a]	0.093[a]	[-0.24, 0.12][a]	-0.026[a]
Age 18 Social Support→Age 20 Marijuana Use	-0.004[a]	0.023[a]	[-0.05, 0.04][a]	-0.007[a]	-0.004[a]	0.023[a]	[-0.05, 0.04][a]	-0.007[a]
Age 18 Marijuana Use→Age 20 Marijuana Use	**0.578[a]**	**0.048[a]**	**[0.48, 0.67][a]**	**0.487[a]**	**0.578[a]**	**0.048[a]**	**[0.48, 0.67][a]**	**0.487[a]**
Age 18 Rumination→Age 20 Marijuana Use	0.059[a]	0.060[a]	[-0.06, 0.18][a]	0.027[a]	0.059[a]	0.060[a]	[-0.06, 0.18][a]	0.027[a]
Abuse History→Age 18 Rumination	0.107[a]	0.086[a]	[-0.06, 0.27][a]	0.028[a]	0.107[a]	0.086[a]	[-0.06, 0.27][a]	0.028[a]
Social Stress→Age 18 Rumination	**0.098[a]**	**0.034[a]**	**[0.03, 0.16][a]**	**0.092[a]**	**0.098[a]**	**0.034[a]**	**[0.03, 0.16][a]**	**0.092[a]**
Age 17 Social Support→Age 18 Rumination	-0.005[a]	0.008[a]	[-0.02, 0.01][a]	-0.018[a]	-0.005[a]	0.008[a]	[-0.02, 0.01][a]	-0.018[a]
Age 17 Rumination→Age 18 Rumination	**0.462[a]**	**0.028[a]**	**[0.41, 0.52][a]**	**0.478[a]**	**0.462[a]**	**0.028[a]**	**[0.41, 0.52][a]**	**0.478[a]**
Covariances								
Age 17 Social Support ↔ Abuse History	-0.044	0.022	[-0.09, 0.00]	-0.044	-0.307	0.136	[-0.57, -0.04]	-0.198
Age 17 Rumination ↔ Abuse History	0.001	0.006	[-0.01, 0.01]	0.004	-0.015	0.025	[-0.06, 0.03]	-0.039
Social Stress ↔ Abuse History	0.030	0.007	[0.02, 0.04]	0.117	0.042	0.042	[-0.04, 0.13]	0.086

Social Stress ↔ Age 17 Social Support	-1.225	0.099	[-1.42, -1.03]	-0.350	-1.387	0.374	[-2.12, -0.65]	-0.261
Social Stress ↔ Age 17 Rumination	0.294	0.024	[0.25, 0.34]	0.291	0.477	0.110	[0.26, 0.69]	0.359
Age 17 Social Support ↔ Age 17 Rumination	-0.469	0.086	[-0.64, -0.30]	-0.121	-0.871	0.267	[-1.39, -0.35]	-0.206
Abuse History ↔ Age 18 Marijuana Use	0.041	0.013	[0.02, 0.07]	0.081	-0.014	0.070	[-0.15, 0.12]	-0.015
Social Stress ↔ Age 18 Marijuana Use	0.377	0.048	[0.28, 0.47]	0.207	0.130	0.250	[-0.36, 0.62]	0.040
Age 17 Social Support ↔ Age 18 Marijuana Use	0.059	0.043	[-0.03, 0.14]	0.059	-0.593	0.685	[-1.94, 0.75]	-0.058
Age 17 Rumination ↔ Age 18 Marijuana Use	0.059	0.043	[-0.03, 0.14]	0.059	0.037	0.174	[-0.30, 0.38]	0.015
Residual Variances								
Age 18 Social Support	7.669	0.631	[6.43, 8.91]	0.639	11.706	2.080	[7.63, 15.78]	0.680
Age 18 Rumination	0.766	0.036	[0.70, 0.84]	0.732	0.747	0.104	[0.54, 0.95]	0.718
Age 20 Marijuana Use	3.870	0.254	[3.37, 4.37]	0.760	4.762	0.857	[3.08, 6.44]	0.695
Indirect Effects								
Stress→A18 Rumination→A20 Marijuana Use	0.006[a]	0.006[a]	[-0.010, 0.027][a]	-	0.006[a]	0.006[a]	[-0.010, 0.027][a]	-
Abuse→A18 Rumination→A20 Marijuana Use	0.006[a]	0.010[a]	[-0.016, 0.042][a]	-	0.006[a]	0.010[a]	[-0.016, 0.042][a]	-
Stress→A18 Soc. Support→A20 Marijuana Use	0.002[a]	0.012[a]	[-0.031, 0.037][a]	-	0.002[a]	0.012[a]	[-0.031, 0.037][a]	-
Abuse→A18 Soc. Support→A20 Marijuana Use	-0.001[a]	0.010[a]	[-0.037,0.031][a]	-	-0.001[a]	0.010[a]	[-0.037,0.031][a]	-

Note. Fit Measures are provided in Table 22. Exogenous variable residual variances are omitted for parsimony. Fixed effects significant at the $\alpha = .05$ level are indicated in bold.

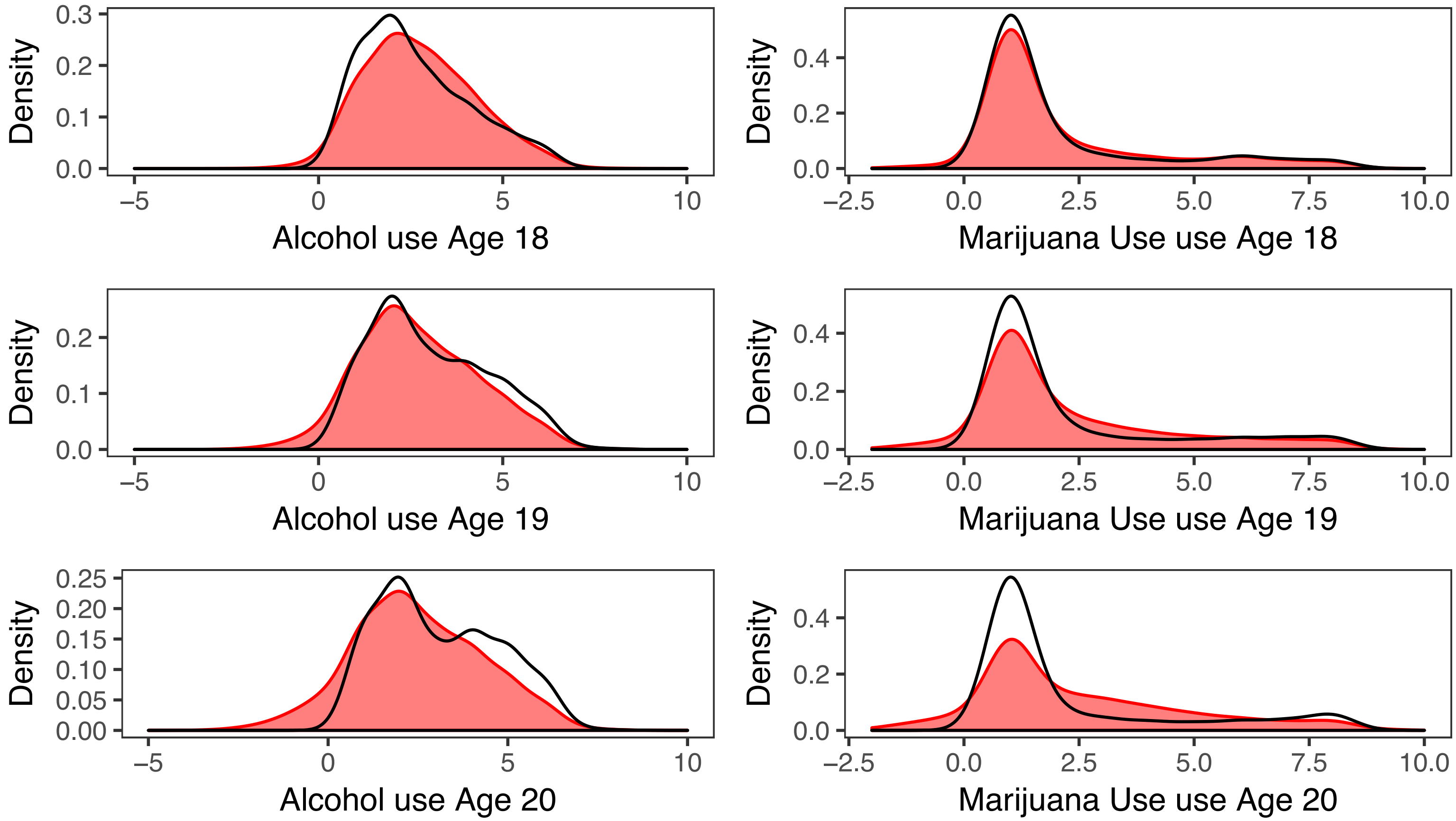
[a] Parameters were fixed to equivalence across groups.

Figure 1. Potential Scale Reduction Factors over Imputation Iteration Comparisons.



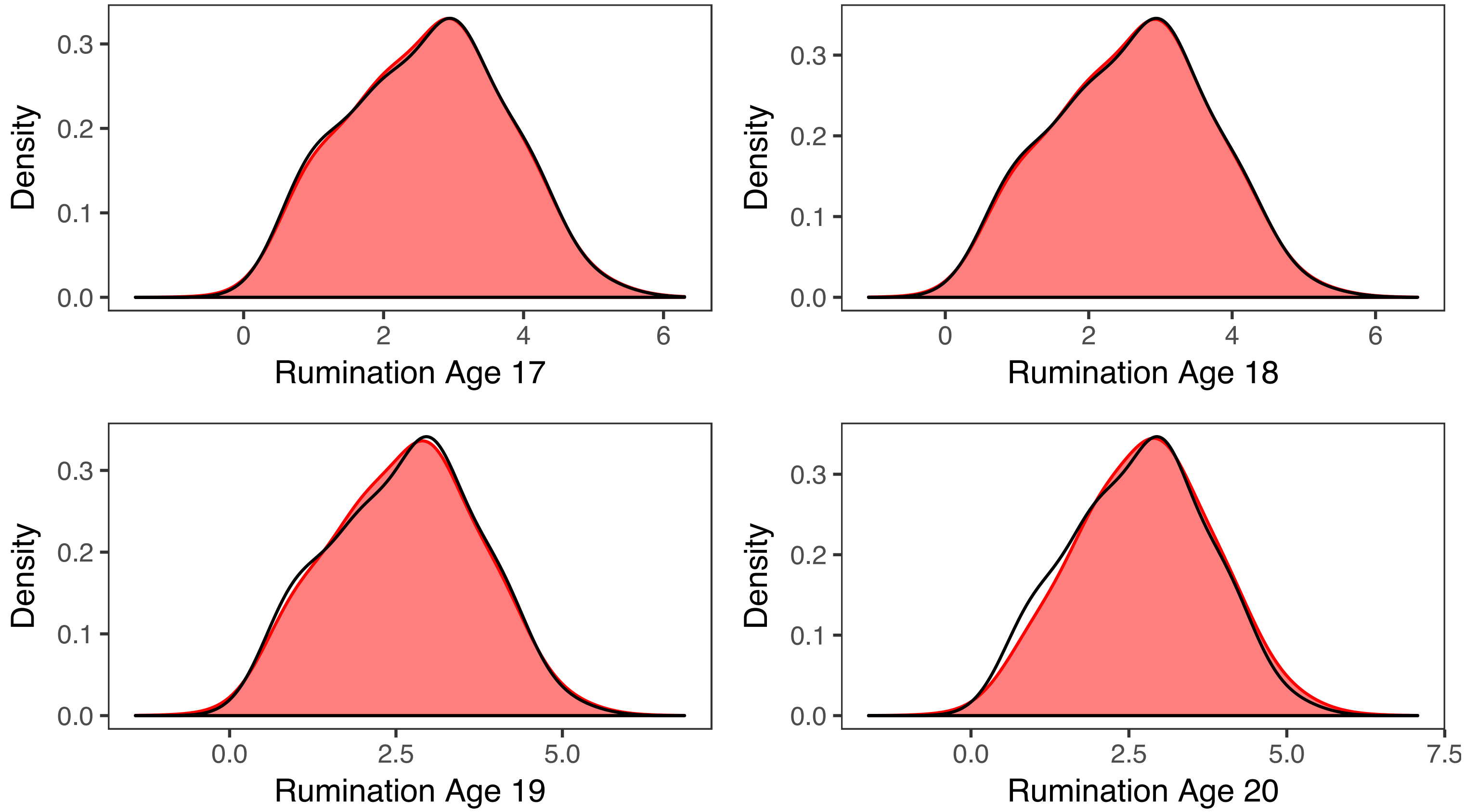
Note. Solid lines indicate fixed effect estimates, dashed lines indicate residual estimates, and (when applicable) dotted lines indicate threshold values.

Figure 2. Observed-versus-Imputed Density Plots of Substance Use Variables by Age.



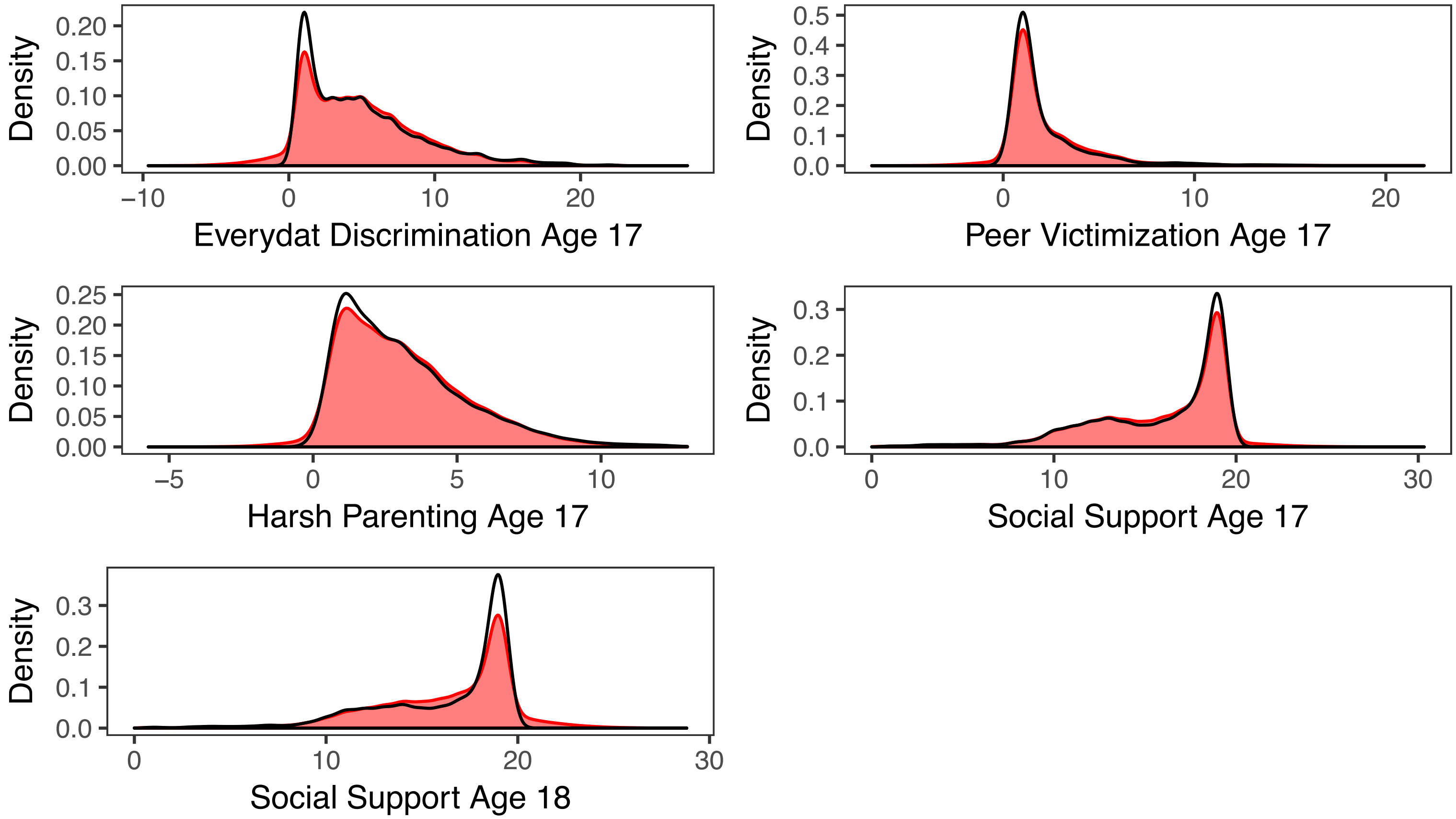
Note. Red shaded region indicates density values of imputed data, and black lines indicate values of observed data.

Figure 3. Observed-versus-Imputed Density Plots of Rumination by Age.



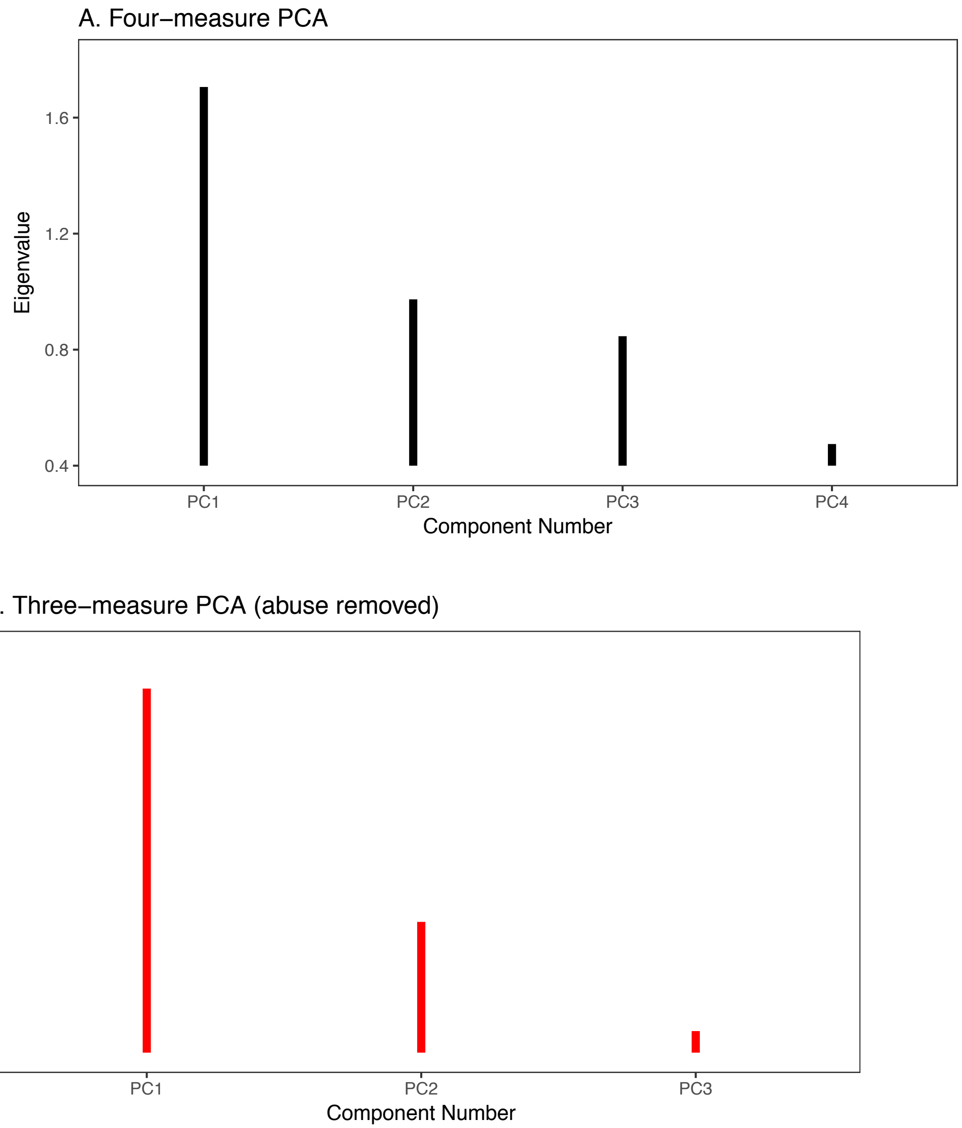
Note. Red shaded region indicates density values of imputed data, and black lines indicate values of observed data.

Figure 4. Observed-versus-Imputed Density Plots of Social Stress and Social Support Measures.



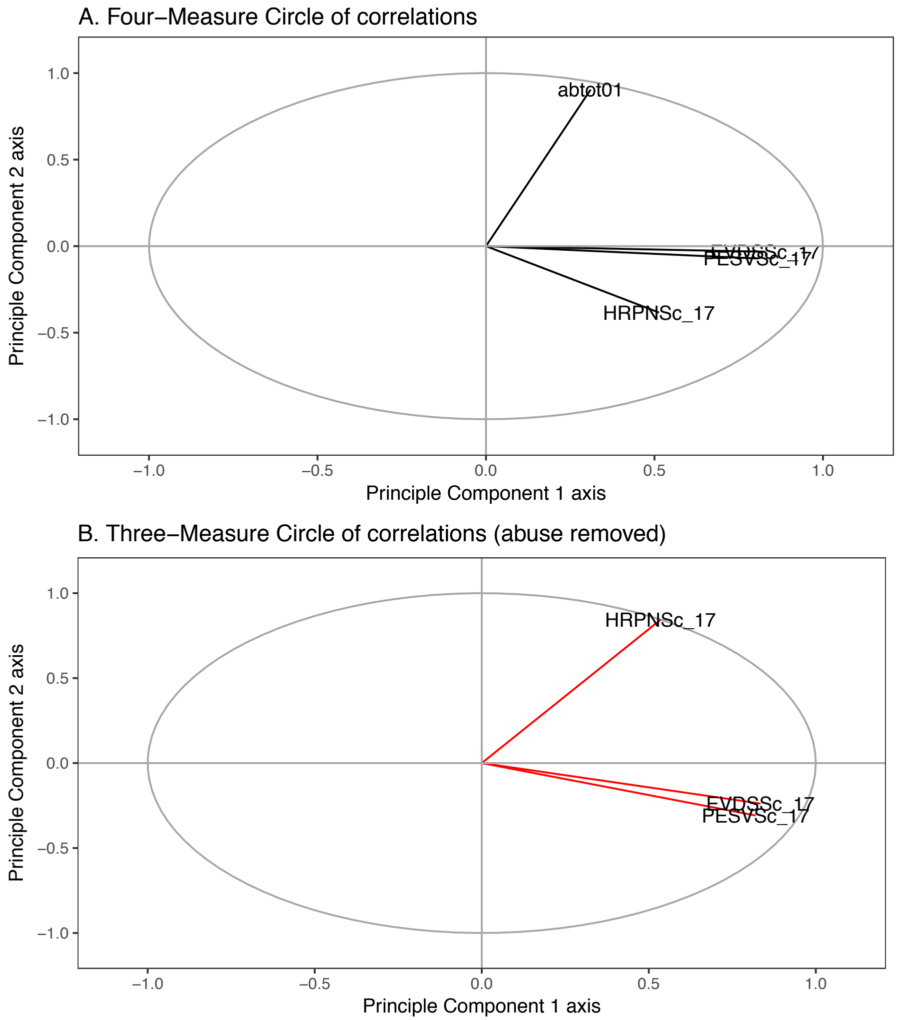
Note. Red shaded region indicates density values of imputed data, and black lines indicate values of observed data.

Figure 5. Principal components analysis scree plot.



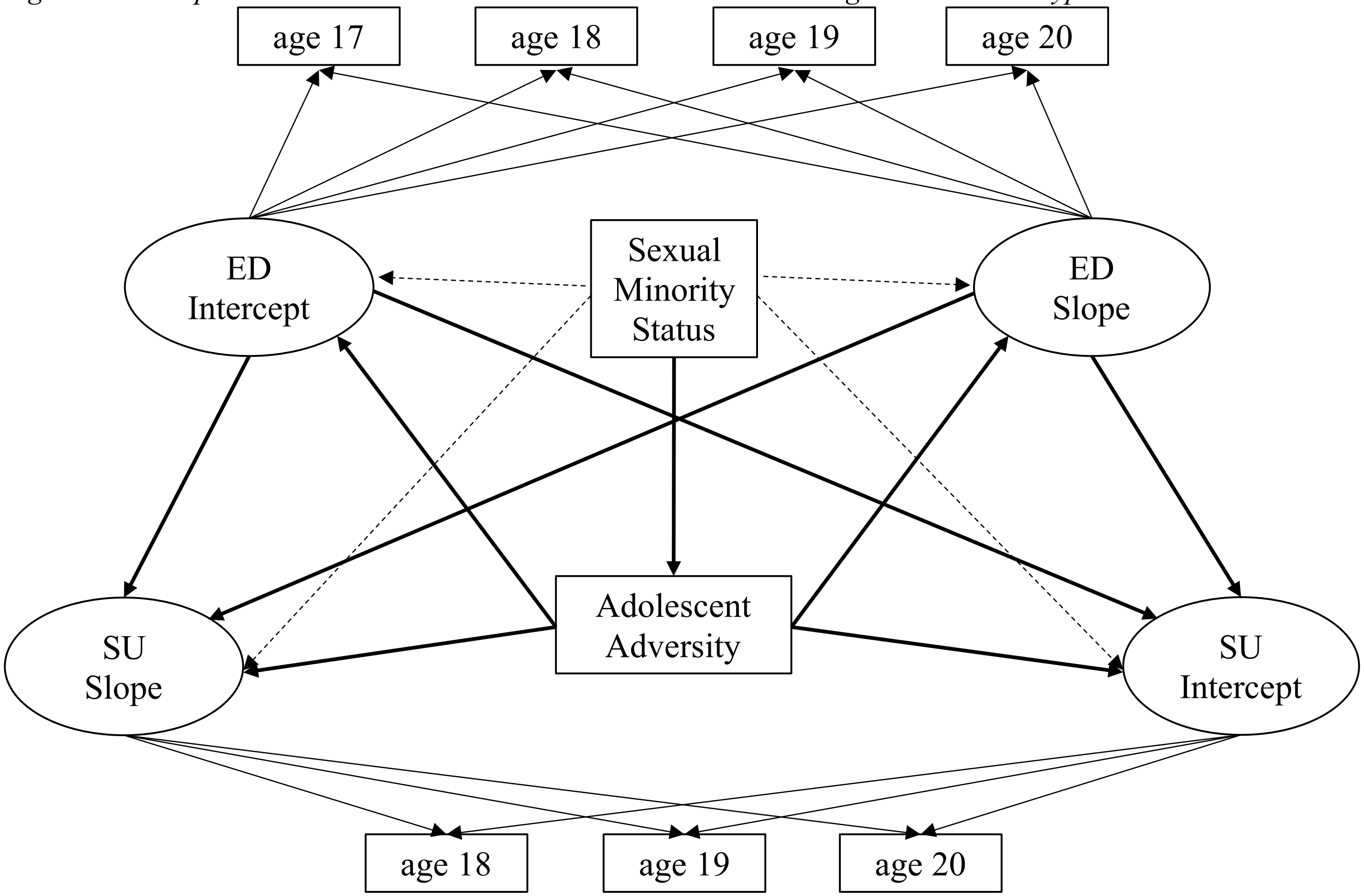
Note. PC = Principal component.

Figure 6. Circle of Correlations Plots.



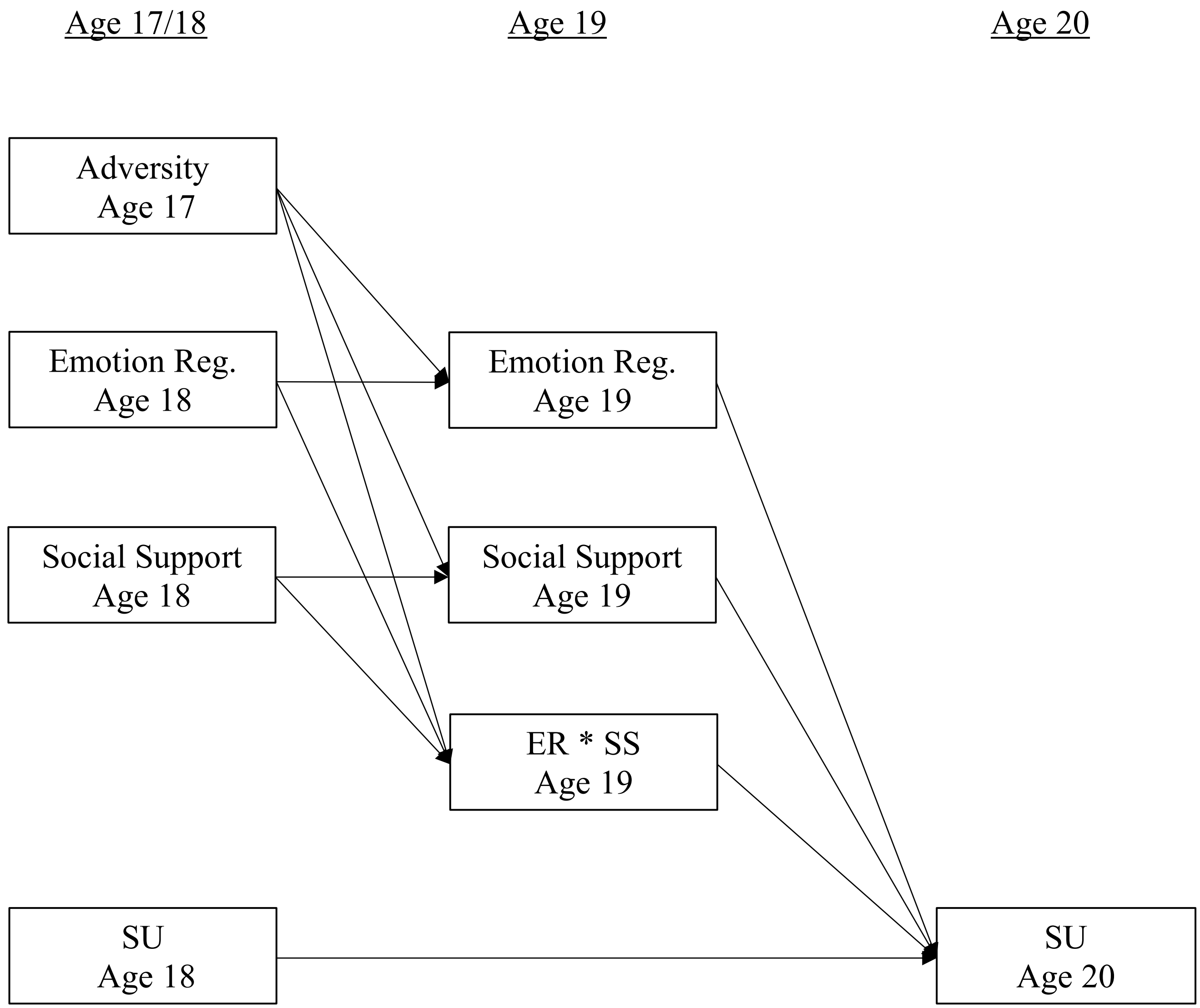
Note. HRPNSc_17 = Age 17 Harsh Parenting, EVDSSc_17=Age 17 Everyday Discrimination, PESVSc_17 = Age 17 Peer Victimization, abtot01 = Abuse History.

Figure 7. Conceptual Parallel Process Growth Curve Model addressing Aims 1 and 2 Hypotheses.



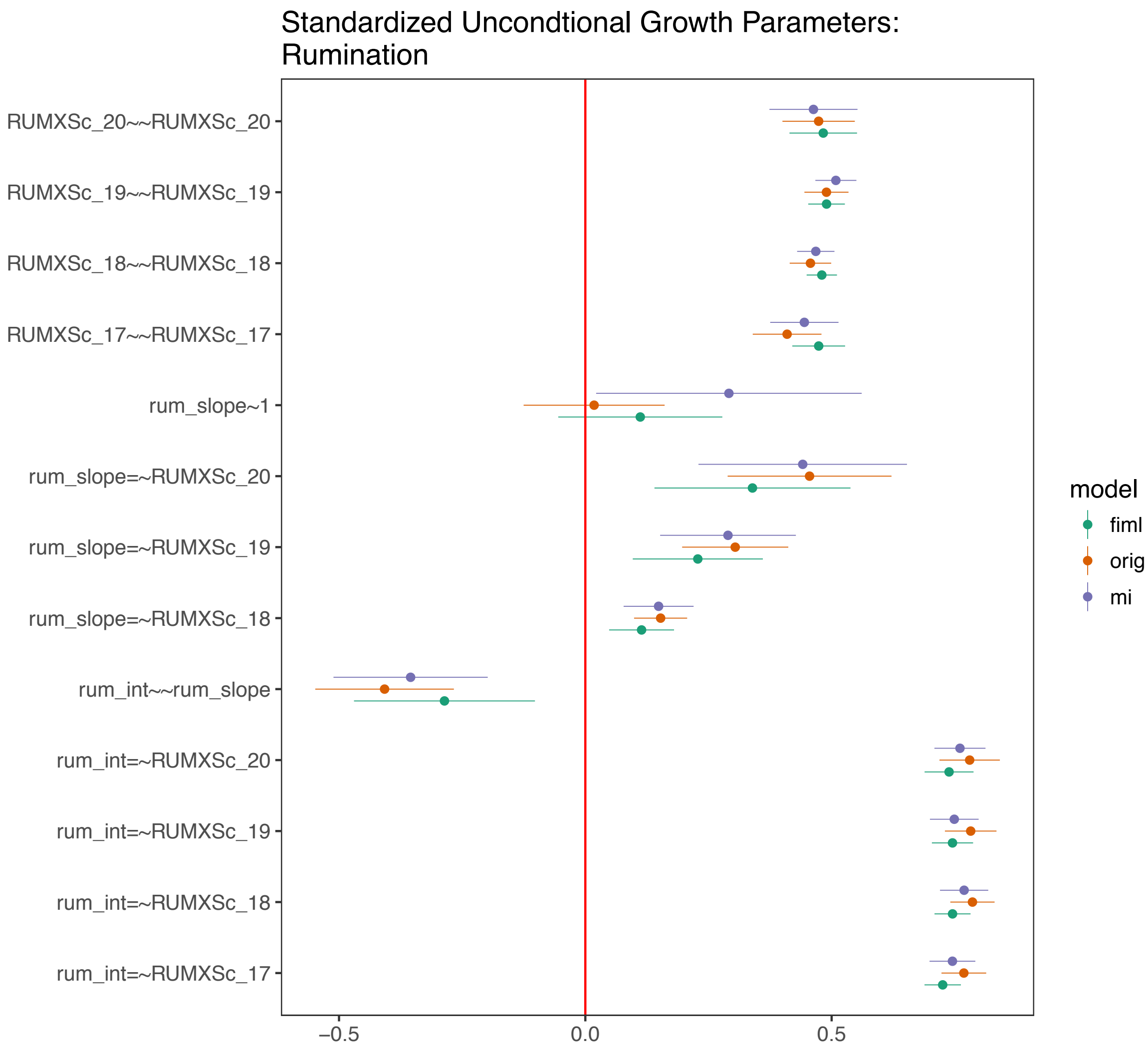
Note. ED = Emotion Dysregulation, SU = Substance Use. Race covariates omitted for parsimony. Dashed lines indicate mediated effects.

Figure 8. Conceptual Simplified Multi-Group Structural Equation Model addressing Aim 3 Hypotheses.



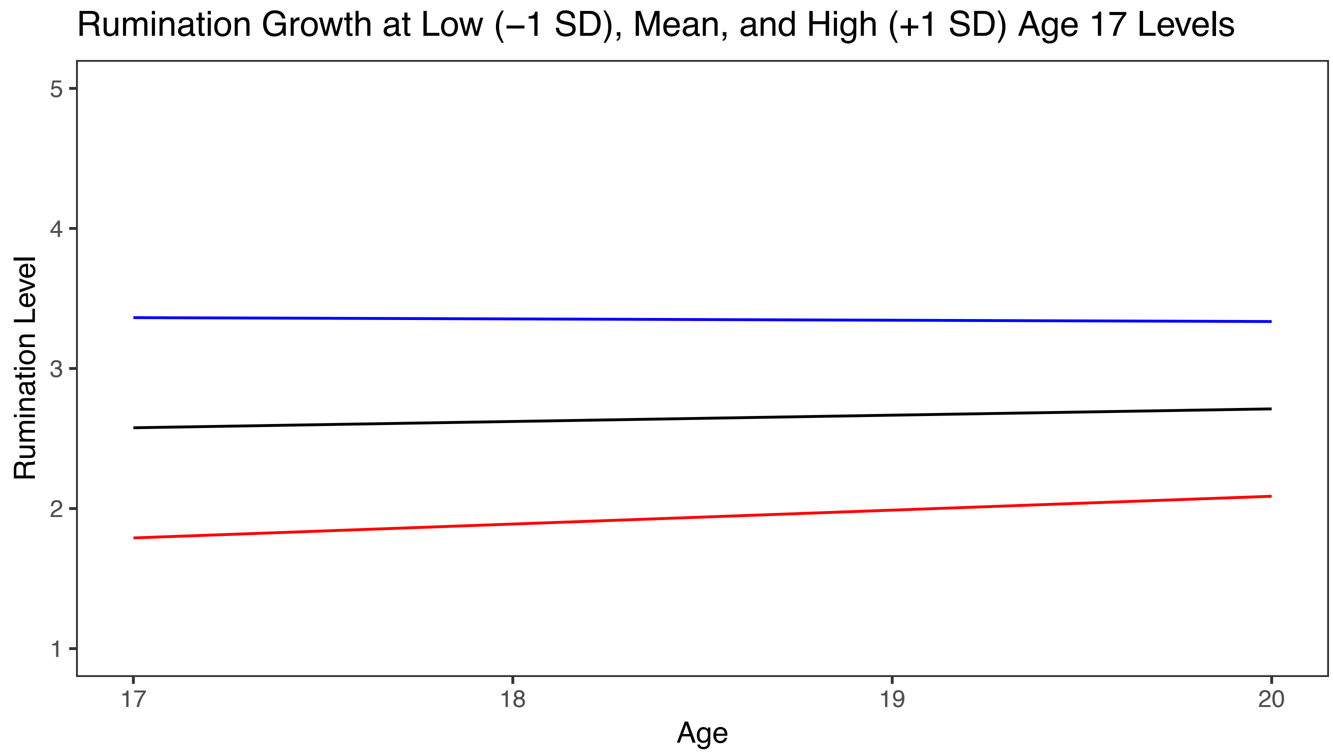
Note: Emotion Reg. = ER = Emotion Regulation, SU = Substance Use, SS = Social Support. This model is specified for both heterosexual and sexual minority women. Intercepts and residual variances and covariances are excluded from this diagram for clarity.

Figure 9. Rope Ladder Plot of Unconditional Growth Model Parameters for Rumination.



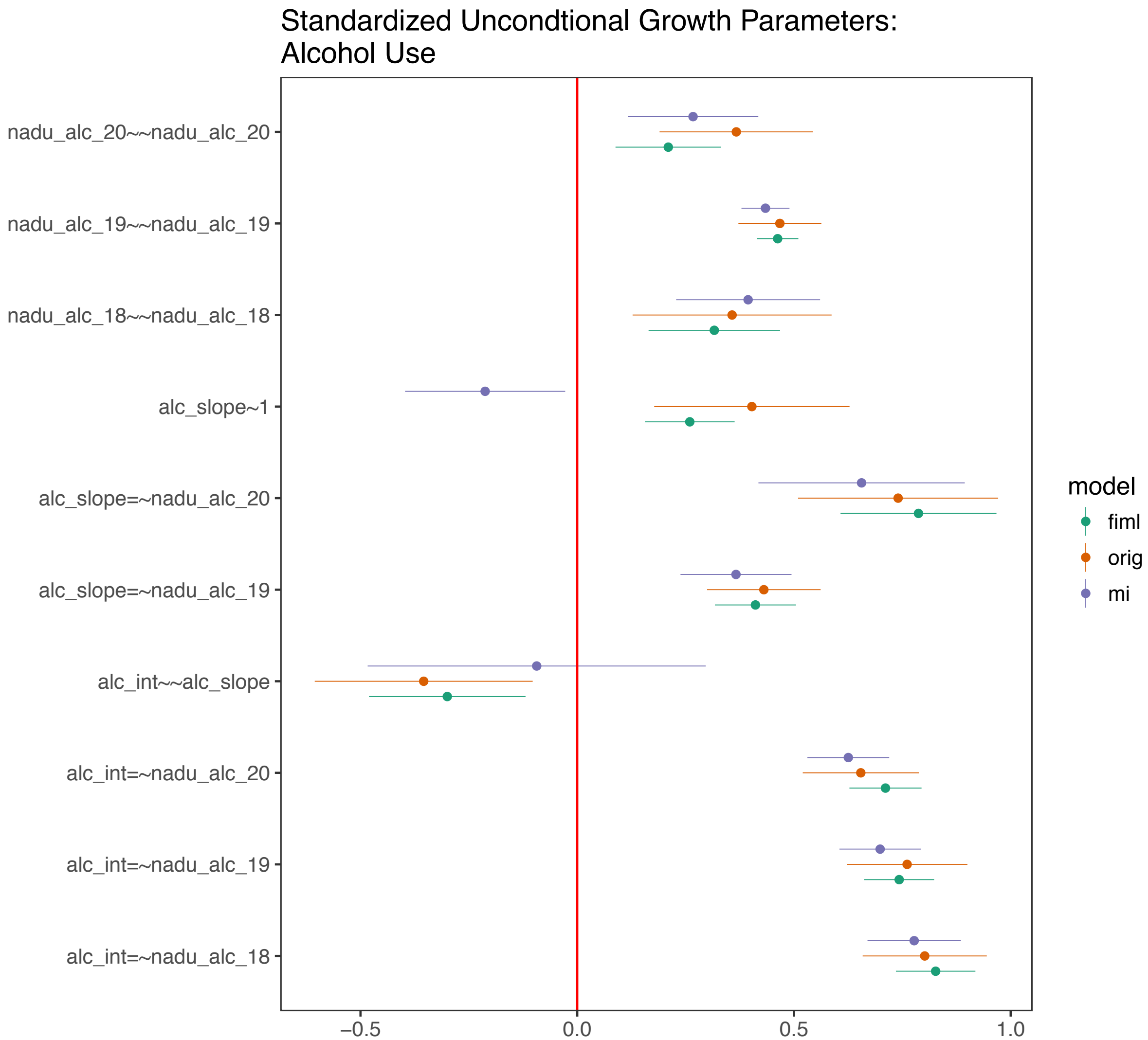
Note. FIML = Full Information Maximum Likelihood, Orig. = Original Data, MI = Multiple Imputation. Operators “~~” indicate variances and covariances, “~” indicate regressions, and “=~” indicate factor loadings.

Figure 10. Slope-Intercept Correlation of Rumination.



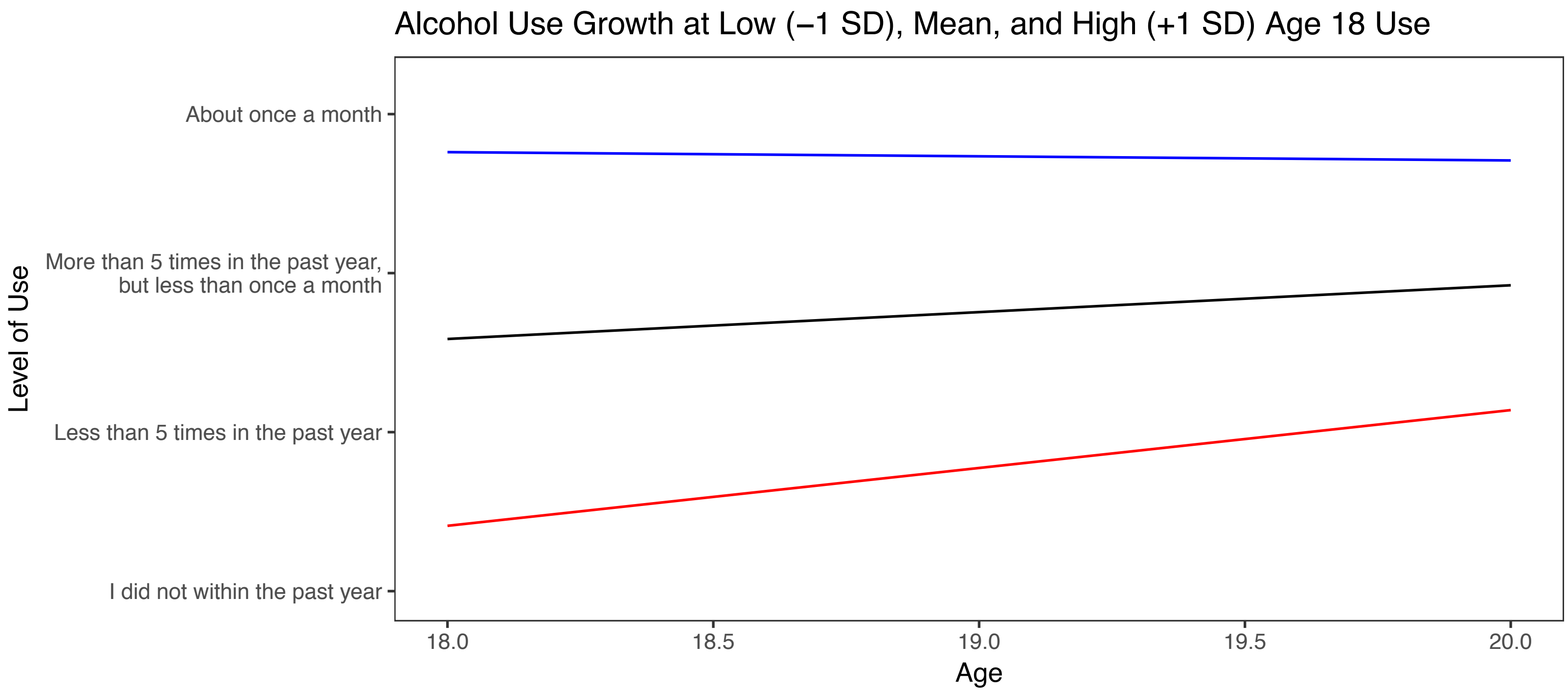
Note: Red line indicates low (-1 SD), black indicates mean, and blue indicates high (+1 SD) levels of Rumination at age 17.

Figure 11. Rope Ladder Plot of Unconditional Growth Model Parameters for Alcohol Use.



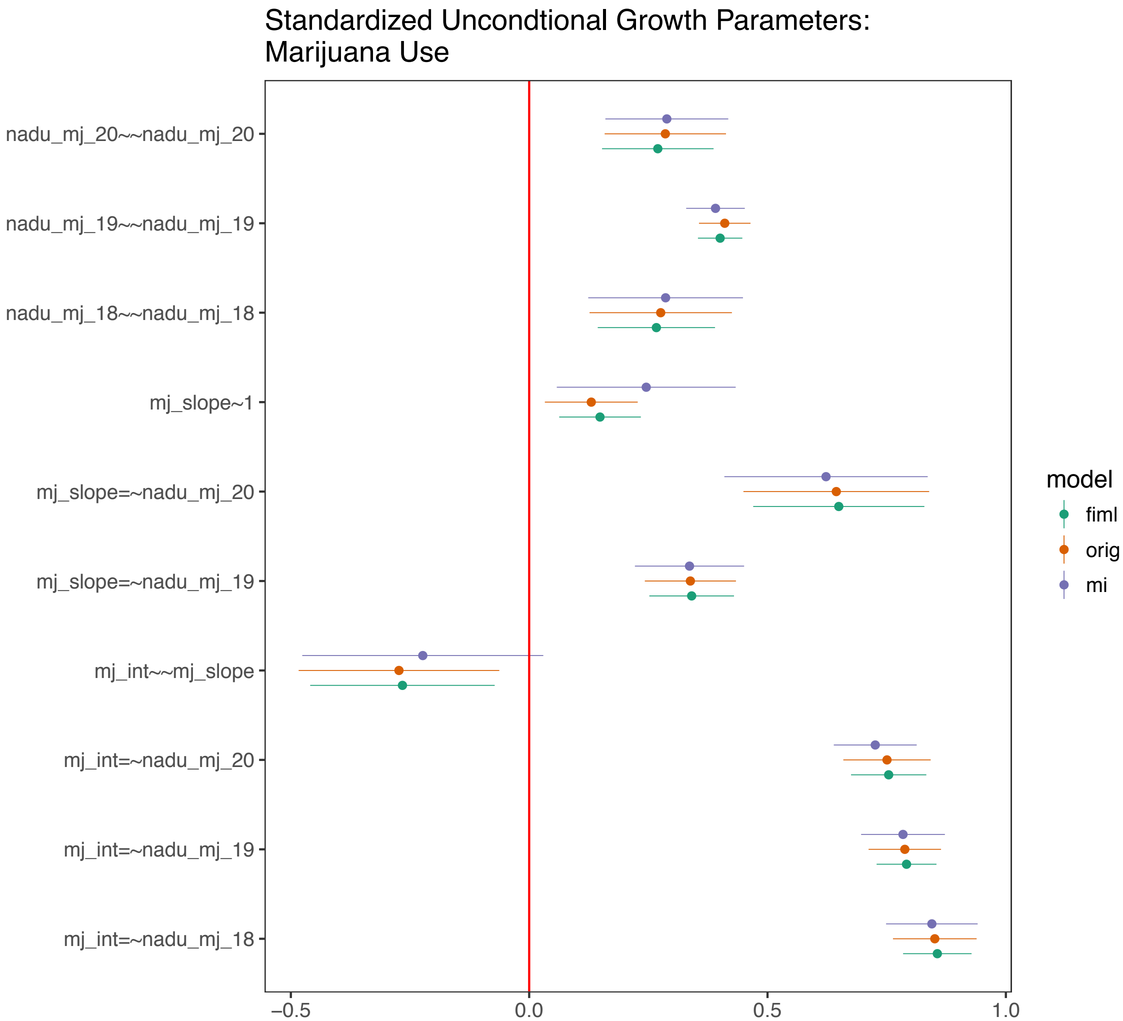
Note. FIML = Full Information Maximum Likelihood, Orig. = Original Data, MI = Multiple Imputation. Operators "~~" indicate variances and covariances, "~" indicate regressions, and "=~" indicate factor loadings.

Figure 12. Slope-Intercept Correlation of Alcohol Use.



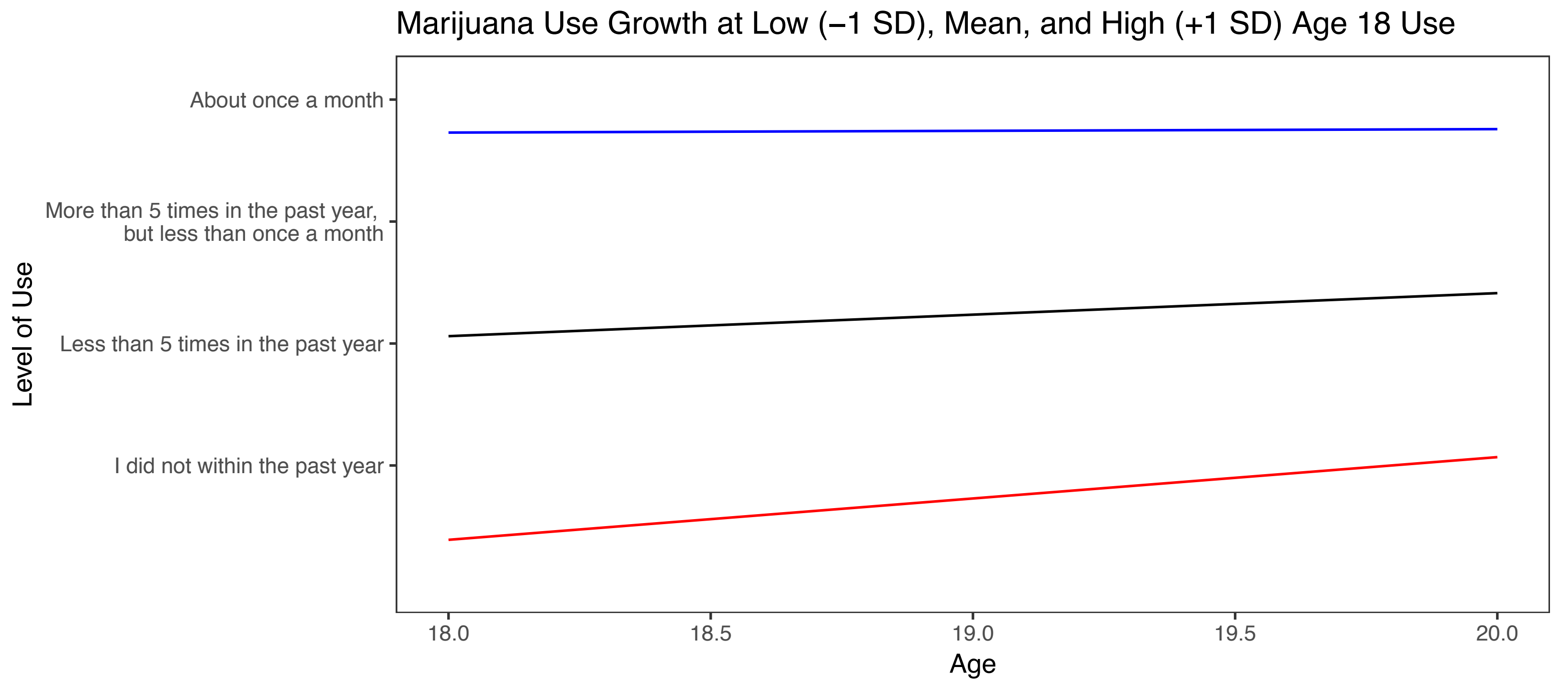
Note: Red line indicates low (-1 SD), black indicates mean, and blue indicates high (+1 SD) levels of alcohol use at age 18.

Figure 13. Rope Ladder Plot of Unconditional Growth Model Parameters for Marijuana Use.



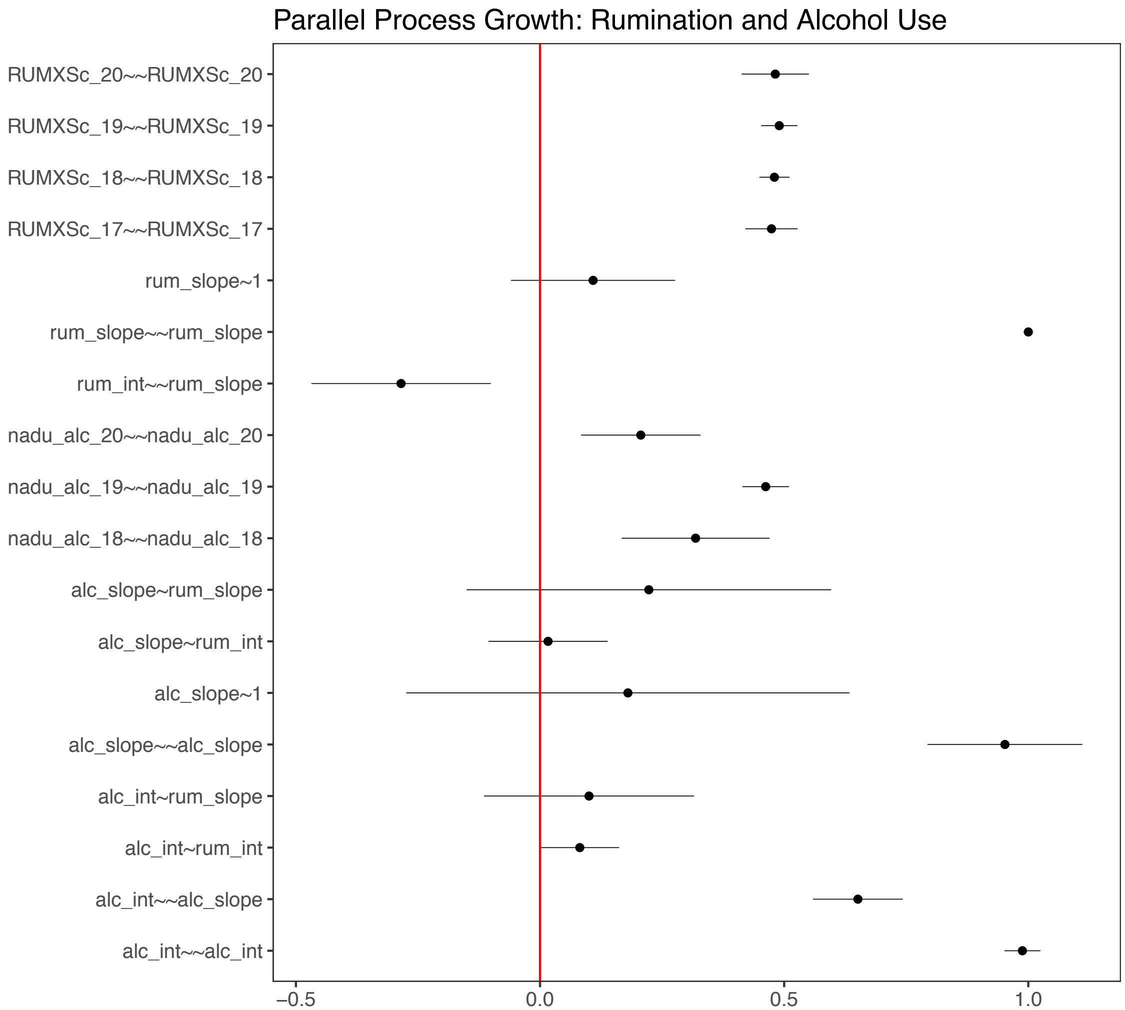
Note. FIML = Full Information Maximum Likelihood, Orig. = Original Data, MI = Multiple Imputation. Operators "~~" indicate variances and covariances, "~" indicate regressions, and "=~" indicate factor loadings.

Figure 14. Slope-Intercept Correlation of Marijuana Use.



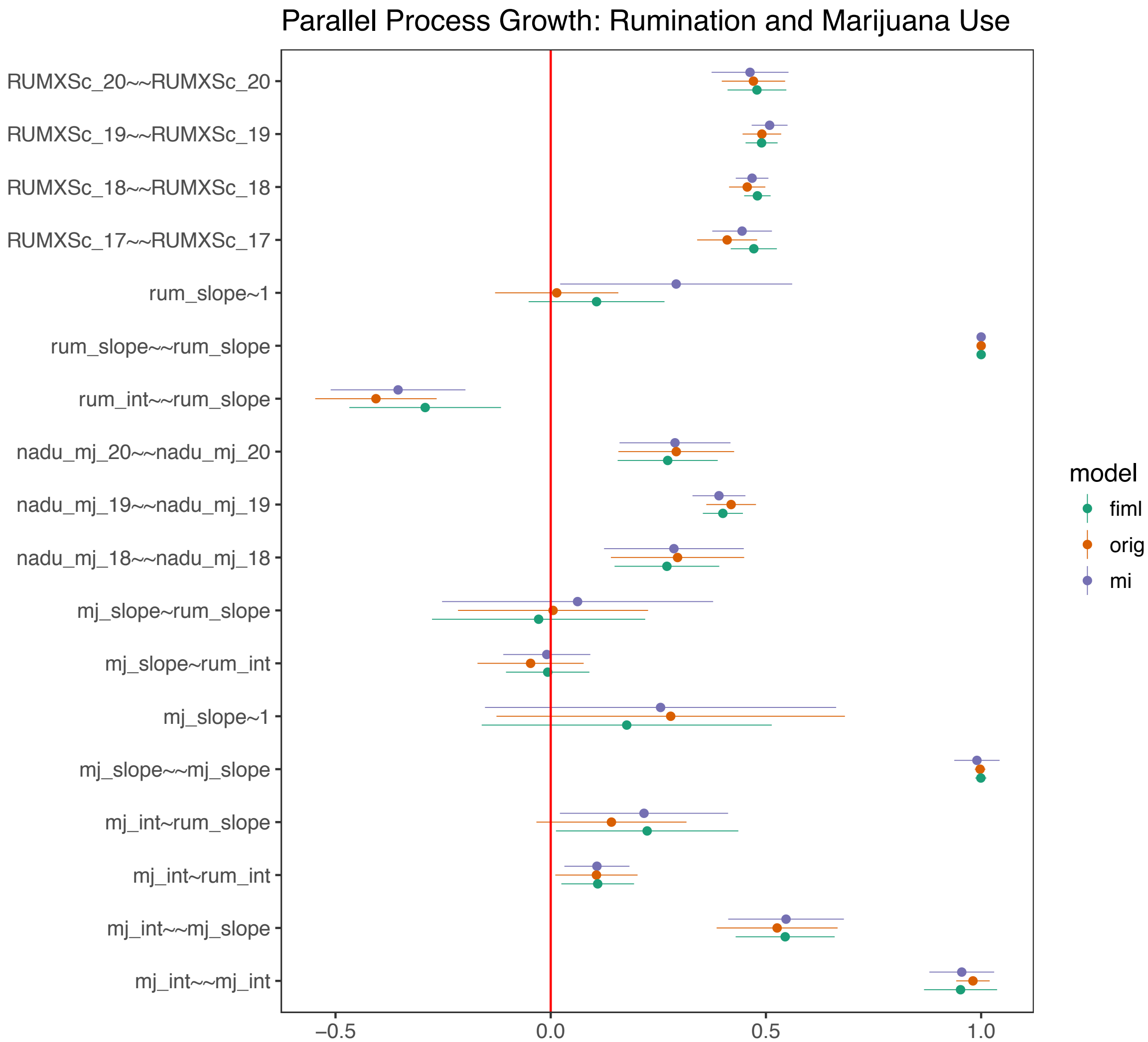
Note: Red line indicates low (-1 SD), black indicates mean, and blue indicates high (+1 SD) levels of marijuana use at age 18.

Figure 15. Rope Ladder Plot of Parallel Process Growth Model Parameters for Rumination and Alcohol Use.



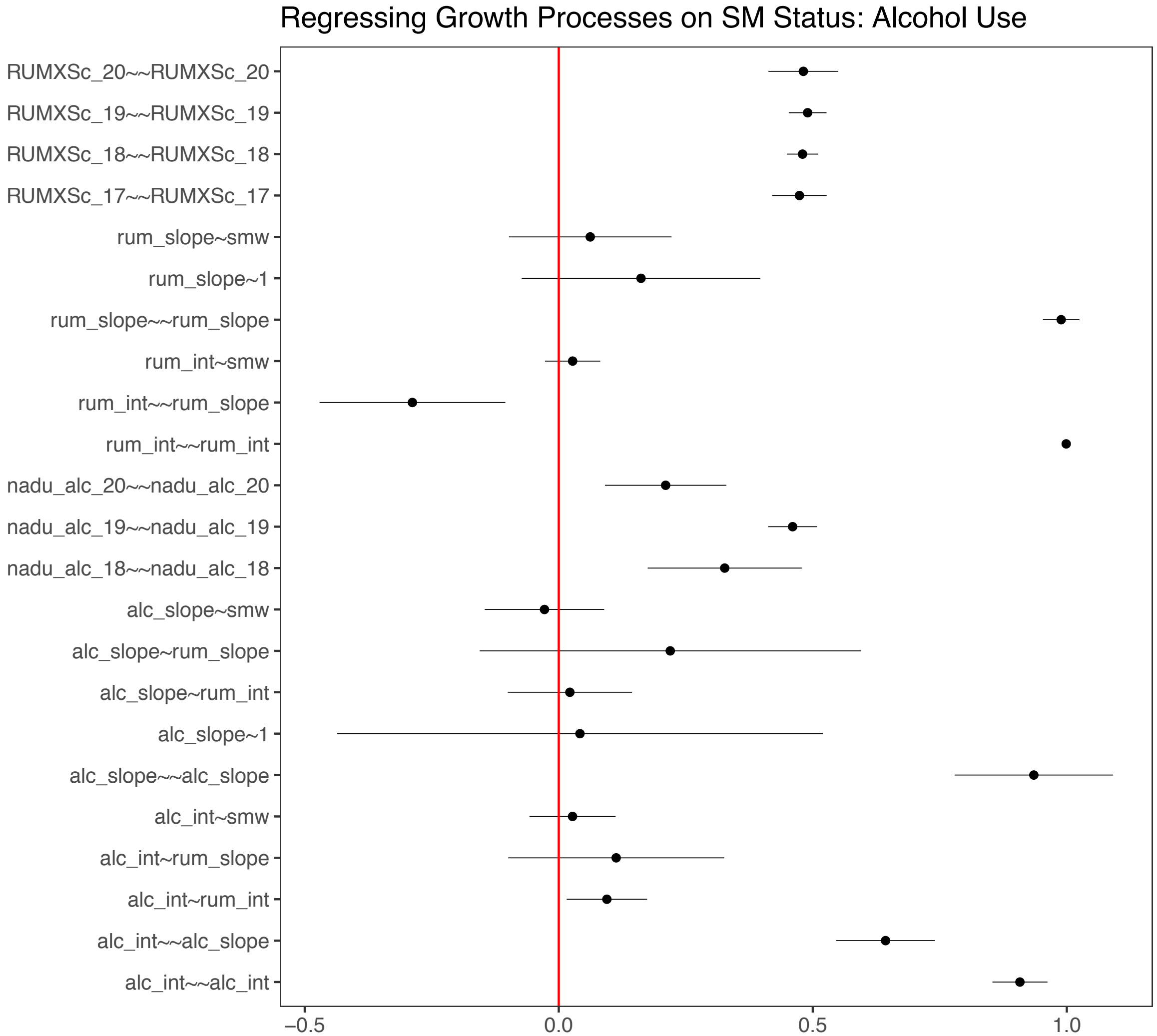
Note. Estimates reflect those derived from the full information maximum likelihood solution given convergence issues using other approaches. Operators "~~" indicate variances and covariances, "~" indicate regressions, and "=~" indicate factor loadings.

Figure 16. Rope Ladder Plot of Parallel Process Growth Model Parameters for Rumination and Marijuana Use.



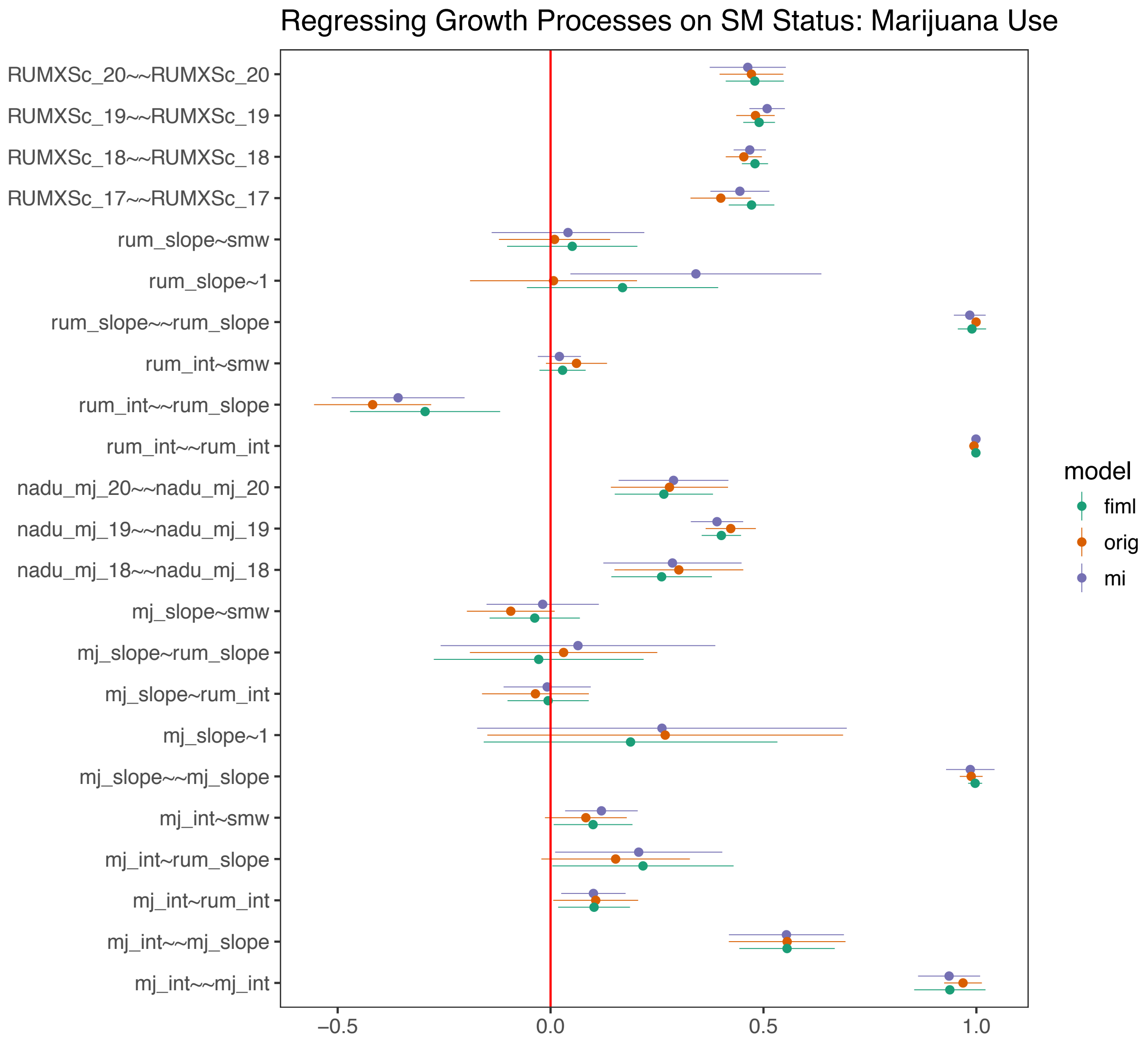
Note. FIML = Full Information Maximum Likelihood, Orig. = Original Data, MI = Multiple Imputation. Operators "~~" indicate variances and covariances, "~" indicate regressions, and "=~" indicate factor loadings.

Figure 17. Rope Ladder Plot of Model Estimates Regressing Alcohol Use and Rumination Growth on Sexual Minority Status.



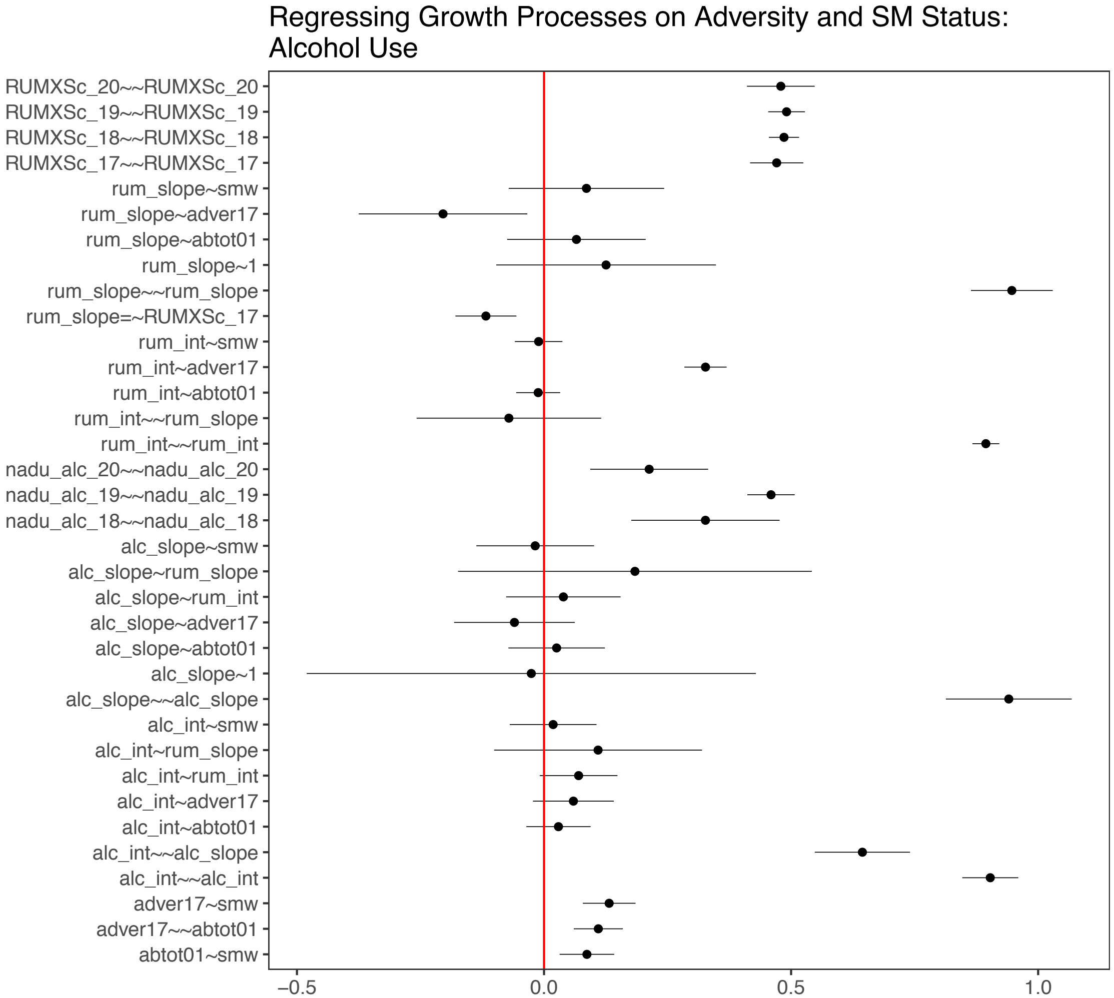
Note. Estimates reflect those derived from the full information maximum likelihood solution given convergence issues using other approaches. Operators "~~" indicate variances and covariances, "~" indicate regressions, and "=~" indicate factor loadings.

Figure 18. Rope Ladder Plot of Model Estimates Regressing Marijuana Use and Rumination Growth on Sexual Minority Status.



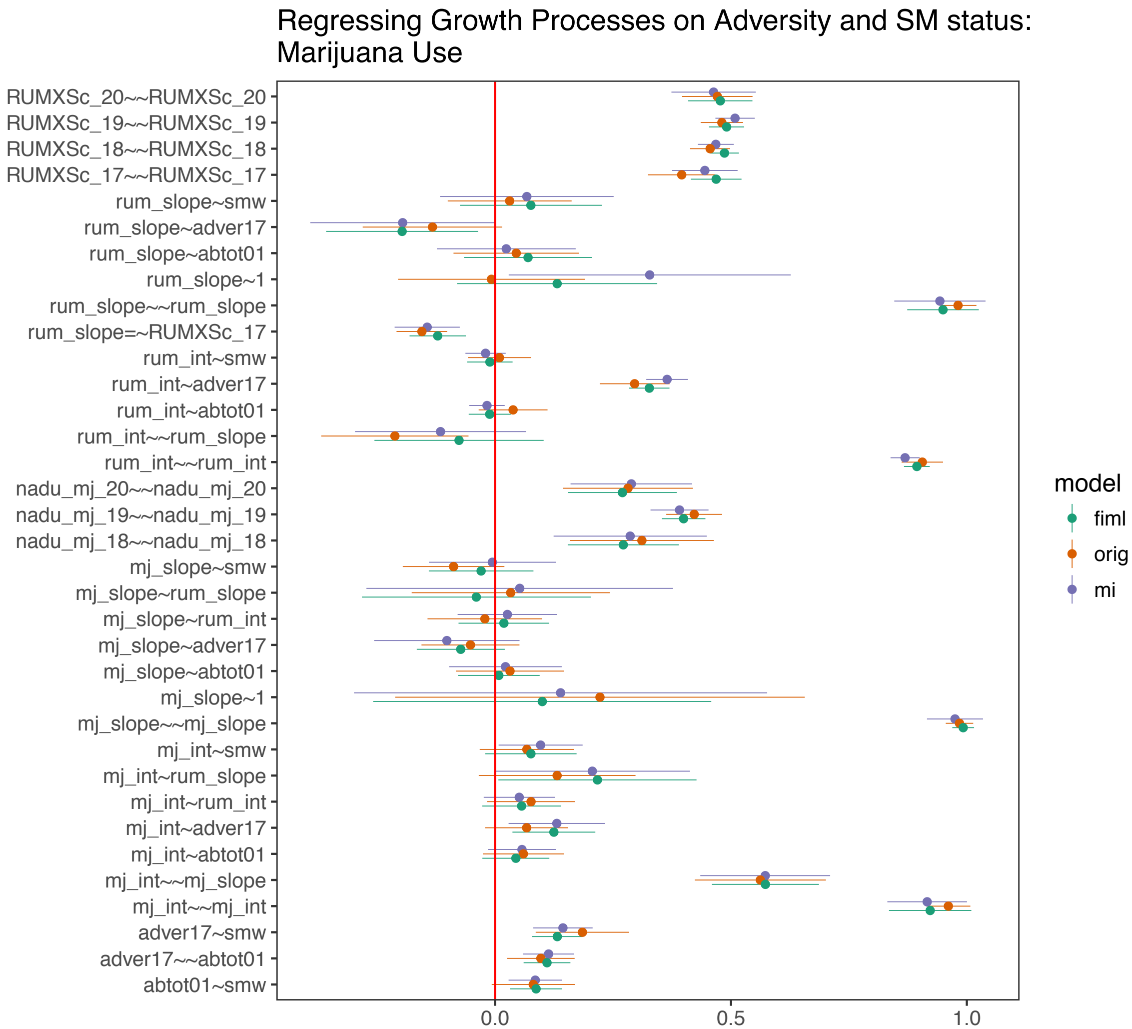
Note. FIML = Full Information Maximum Likelihood, Orig. = Original Data, MI = Multiple Imputation. Operators "~~" indicate variances and covariances, "~" indicate regressions, and "=~" indicate factor loadings.

Figure 19. Rope Ladder Plot of Alcohol Use Model Estimates Regressing Growth Parameters on SM Status and Adversity.



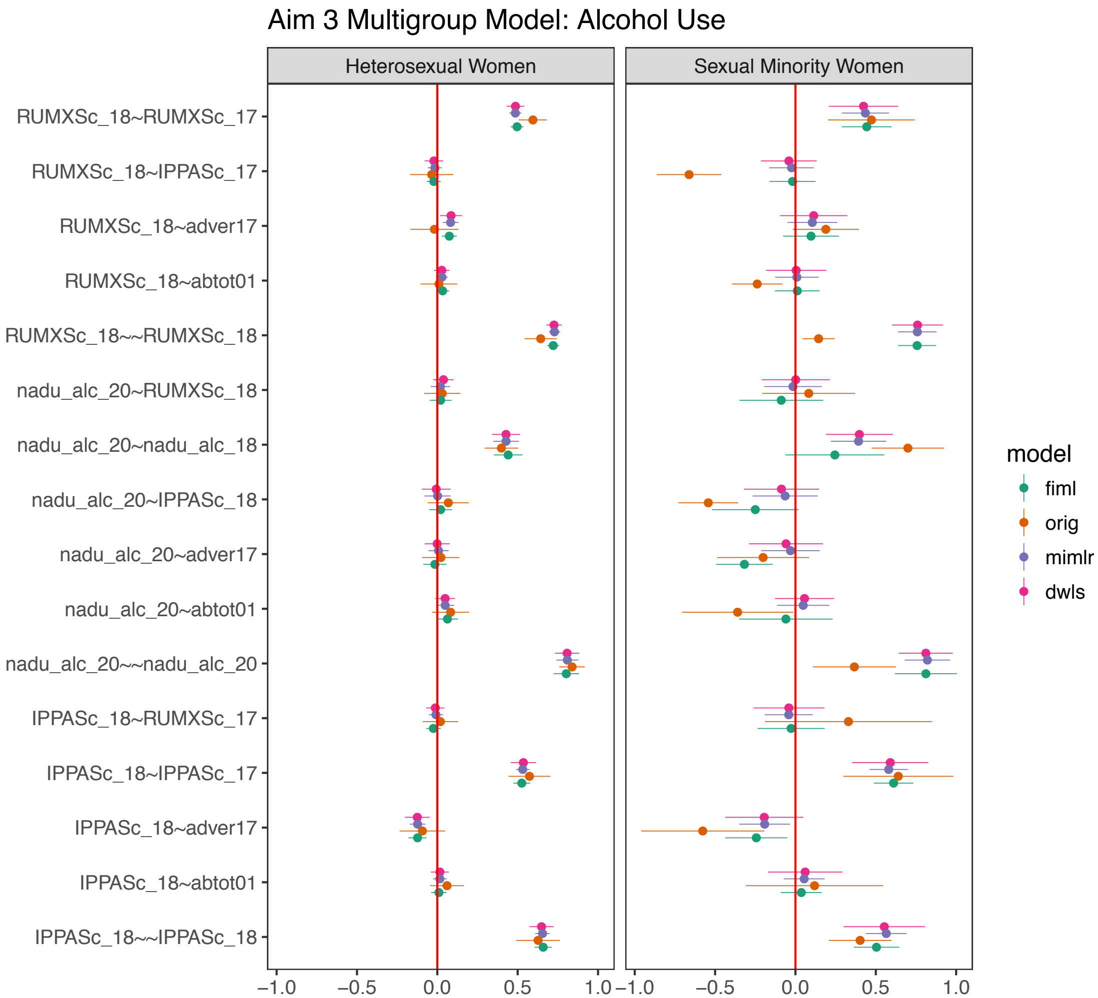
Note. Estimates reflect those derived from the full information maximum likelihood solution given convergence issues using other approaches. Operators "~~" indicate variances and covariances, "~" indicate regressions, and "=~" indicate factor loadings.

Figure 20. Rope Ladder Plot of Marijuana Use Model Estimates Regressing Growth Parameters on SM Status and Adversity.



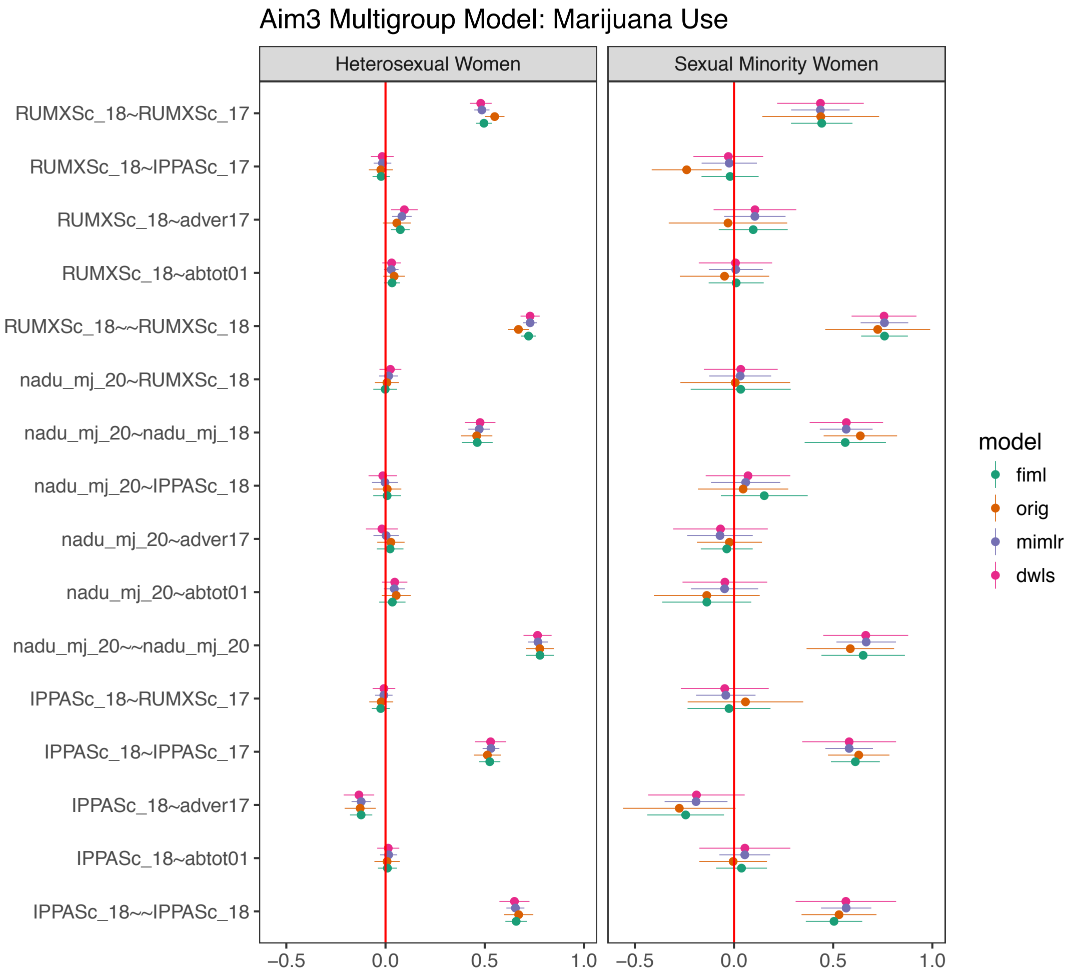
Note. FIML = Full Information Maximum Likelihood, Orig. = Original Data, MI = Multiple Imputation. Operators “~~” indicate variances and covariances, “~” indicate regressions, and “=~” indicate factor loadings.

Figure 21. Rope Ladder Plot of Aim 3 Multi-Group Structural Equation Models for Alcohol Use.



Note. FIML = Full Information Maximum Likelihood, Orig. = Original Data, MI = Multiple Imputation, MIMLR = Multiple Imputation using Maximum Likelihood with Robust Standard Errors. Operators “~~” indicate variances and covariances, “~” indicate regressions, and “=~” indicate factor loadings.

Figure 22. Rope Ladder Plot of Aim 3 Multi-Group Structural Equation Models for Marijuana Use.



Note. FIML = Full Information Maximum Likelihood, Orig. = Original Data, MI = Multiple Imputation, MIMLR = Multiple Imputation using Maximum Likelihood with Robust Standard Errors. Operators "~~" indicate variances and covariances, "~" indicate regressions, and "=~" indicate factor loadings.