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Role of ADHD in the Co-occurrence between Heavy Alcohol Use and Depression Trajectories in Adulthood

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Abstract

Background: Attention-Deficit/Hyperactivity Disorder (ADHD) is associated with greater heavy alcohol use and depressive symptoms in adulthood. Yet, few studies have investigated whether childhood ADHD predicts an increased association between heavy drinking and depression in adulthood when this co-occurrence becomes more common. We examined associations among heavy alcohol use and depression longitudinally from ages 21 to 29 and whether these associations differed for those with or without childhood ADHD, as well as for those with or without persistent ADHD in adulthood.

Methods: Data were from the Pittsburgh ADHD Longitudinal Study (PALS), a prospective cohort of children diagnosed with ADHD and demographically similar individuals without ADHD histories. ADHD symptoms in adulthood were self- and parent-reported; depressive symptoms and frequency of drinking five or more drinks in a single drinking occasion were self-reported and measured at five time-points from ages 21–29. Depression and alcohol use were modeled in a multiple-group, parallel process longitudinal growth model.

Results: The slopes of heavy alcohol use and depression were significantly and positively associated from ages 25–29 but not at the younger ages. Although the strength of these associations did not differ by group (with or without ADHD, childhood or adulthood), the slopes of depression and heavy drinking at the older ages were highly variable and individuals with ADHD showed significantly faster growth in depression from ages 25–29.

Conclusions: Due to the strengthening association between heavy drinking and depression for adults in their late 20s, and increasing depression for adults with ADHD histories, individuals with ADHD may be at greater risk for co-occurring depression and binge drinking. Negative reinforcement-related alcohol use may strengthen as these individuals age toward the fourth decade of life. More rigorous testing of this possibility is warranted.

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Keywords

ADHD; Depression; Heavy Alcohol Use; Comorbidity; Adulthood

Attention-Deficit/Hyperactivity Disorder (ADHD) is a childhood neurodevelopmental disorder characterized by impulsivity, hyperactivity, and inattentiveness (Association 2013). Children with ADHD are at risk for poorer outcomes than children without ADHD across multiple domains of adulthood functioning (e.g., Barkley et al. 2006). Among these deleterious outcomes are greater heavy alcohol use and symptoms of depression (Meinzer et al. 2016; Molina et al. 2007). Importantly, a large body of research in the general population has documented a high rate of co-occurrence between various alcohol outcomes and depressive symptoms, particularly in adulthood (e.g., Grant and Harford 1995; Swendsen and Merikangas 2000). Yet, few studies at any age have examined whether heavy alcohol use and depressive symptoms are more strongly associated in individuals with versus without ADHD and no studies to our knowledge have done so from a longitudinal perspective (see Molina and Pelham 2014) for a review; Biederman et al. 2006). It is critical to understand whether individuals with ADHD are at greater risk for this distressing and impairing form of co-occurrence in adulthood to guide intervention for this already at-risk group (Brière et al. 2014).

ADHD, Heavy Alcohol Use, and Depressive Symptoms

A large body of work suggests that individuals with ADHD are more vulnerable to developing problematic alcohol use, as well as problematic use of other substances such as nicotine and marijuana, when compared to individuals without ADHD (Charach et al. 2011; Lee et al. 2011; Molina and Pelham 2003; Rooney et al. 2015; Walther et al. 2015). The link between ADHD and heavy alcohol use may be explained by several different mechanisms. For instance, the impulsivity and hyperactivity central to ADHD diagnoses have been conceptualized as part of a broader construct often called behavioral disinhibition (Iacono et al., 1999), which is a trait posited to underlie risk for alcohol use disorder (Rooney et al. 2015; Tarter et al. 2004; Zucker et al. 2011). High levels of behavioral disinhibition may predispose individuals with ADHD to follow a particularly risky developmental pathway, wherein comorbid conduct disorder and affiliation with deviant, substance-using peers create risk for problematic alcohol consumption (Sher, Grekin, & Williams, 2005). This particular pathway may prove even riskier for those with ADHD because of their heightened susceptibility to social processes that promote drinking (Belendiuk et al. 2016).

An alternative, yet little studied, mechanistic pathway to heavy drinking for individuals with ADHD is through vulnerability to depression. A link between ADHD and depression has primarily been conceptualized as a reaction to ADHD-related impairments in educational, occupational, and interpersonal functioning (Semeijn et al. 2015). Coupled with prior research revealing that individuals with ADHD show poorer coping skills and greater impulsivity in the context of negative emotions (Pedersen et al. 2016; Young 2005), individuals with ADHD may be at increased likelihood of drinking excessively to cope with

symptoms of depression. If so, there may be a heightened co-occurrence between heavy drinking and depressive symptoms.

Excessive alcohol consumption may also increase negative affect at a greater rate for individuals with ADHD, which may heighten heavy drinking and depression co-occurrence for this group. Due to their ADHD symptoms, adults with a history of ADHD are already more prone to experiencing negative life events (e.g., academic underachievement, financial and interpersonal problems) that are independent of drinking-related consequences (e.g., Barkley et al. 2006; Kuriyan et al. 2013). Thus, experiencing additional stressful life events that are due to heavy drinking, which is a well-established phenomenon (Brennan et al. 1999; Hart and Fazaa 2004), may be particularly problematic for adults with an ADHD history. Moreover, those with ADHD may perceive negative life events, such as those emanating from heavy drinking, more negatively than individuals without ADHD (King et al. 2017). Because individuals who perceive life events more negatively have been shown to be at risk for depression (Kuiper et al. 1986), individuals with ADHD may be particularly prone to becoming depressed as a result of negative drinking-related life events. Finally, it is important to note that alcohol withdrawal, which includes symptoms of negative affectivity, increases in adulthood relative to adolescence (Chung et al. 2002); adulthood is therefore a particularly salient period to observe drinking-induced depression. In sum, those with ADHD may be particularly susceptible to depression due to drinking during adulthood, but this hypothesis has yet to be tested.

Although those with ADHD may show stronger *causal* links between depression and heavy drinking, a tighter association between these two problematic outcomes could also be due to the fact that ADHD increases the occurrence of shared risk factors that simultaneously heighten risk for both. For instance, because ADHD increases the risk for adulthood life stressors (Barkley et al. 2006; Kuriyan et al. 2013), this group could show tighter links between depression and heavy drinking simply because life stress is a shared risk factor for both (Boden et al. 2014; Kuiper et al. 1986). Similarly, some traits known to be core deficits or elevated in those with ADHD, such as low conscientiousness and negative urgency (Nigg et al. 2004; Pedersen et al. 2016), could simultaneously increase risk for both heavy alcohol use and depression (Cyders and Smith 2008; Smith et al. 2013).

Differences in Childhood ADHD versus Adulthood ADHD Symptom Persistence

The strength of the co-occurrence between heavy alcohol use and depressive symptoms could depend on the extent to which ADHD symptoms persist into adulthood. Indeed, one previous study found that young adults with persistent ADHD had higher rates of mood disorder, neuroticism, and risky behavior when compared to young adults with desistent ADHD symptoms or who were in a nonADHD comparison group (Hechtman et al. 2016). Adulthood functioning is also generally poorer when ADHD symptoms persist into adulthood (Barkley et al. 2008; Hechtman et al. 2016). Thus, we examined whether the association between heavy alcohol use and depressive symptoms differed for those with or without childhood ADHD, as well for those with or without persistence of ADHD

symptoms into adulthood. This allowed a test of whether it is the continuation of ADHD symptoms into adulthood that affects co-occurrence between heavy drinking and depression.

The Current Study

The first purpose of the current study was to test the association between longitudinal trajectories of heavy alcohol use and depression in adulthood. We specifically chose to model heavy drinking because it is an indicator of problematic alcohol use that has more variability than alcohol problems or dependence (Esser et al. 2014), and it is the more proximal alcohol behavior that might result from depression when compared to alcohol problems (i.e., consumption of increasing amounts of alcohol in direct response to negative affect yet not always resulting in easily measured, reported, or perceived problems). Our second purpose was to test the hypothesis that trajectories of depression symptoms and heavy alcohol consumption would be more strongly associated in adults with, versus without, ADHD histories. The contribution of ADHD symptom persistence to this association was also examined. Data were from the Pittsburgh ADHD Longitudinal Study (PALS), which has followed children diagnosed with ADHD through their 20s alongside a demographically similar group without an ADHD history; this study allowed our hypotheses to be tested using prospectively gathered longitudinal data spanning the ages of 21 to 29. Using longitudinal growth modeling, we tested associations between heavy drinking and depressive symptoms at age 21, when heavy drinking is at peak levels (Kerr et al. 2009), and change over time to age 29, when negative affect-mediated drinking may become increasingly relevant (Chung et al. 2002; Nicolai et al. 2012) and especially for individuals with ADHD (Molina and Pelham, 2014). We also tested whether these associations differed by childhood ADHD and by persistence of ADHD in adulthood.

Materials and Methods

Participants

ADHD participants.—The ADHD group participants in the PALS were recruited for longitudinal study based on their diagnosis of ADHD in childhood at the time of participation in an 8 week ADHD Summer Treatment Program (Pelham et al., 1996). From 1987–1996, the PALS children and adolescents (M_{age} = 9.4 years, SD = 2.27 years, range = 5.0–16.92) were diagnosed with ADHD using DSM-III-R or DSM-IV ADHD criteria at the ADD Clinic within Western Psychiatric Institute and Clinic. A Ph.D. level clinician administered standardized semi-structured diagnostic interviews to parents, and parents and teachers completed standardized DSM-III-R and DSM-IV symptom rating scales (DBD; Pelham et al. 1992). The structured diagnostic interview included DSM-III-R or DSM-IV criteria for ADHD, Oppositional Defiant Disorder (ODD), and Conduct Disorder (CD), and supplementary questions assessing situational and severity factors and other comorbidities (instrument available through co-author W.E.P). Diagnoses of ADHD, ODD, and CD were made if the number of symptoms passed the threshold for a DSM-based diagnosis. All ratings were independently reviewed by two Ph.D. level clinicians. Disagreements were reconciled by a third clinician's review of the file, and the majority decision was used. Exclusionary criteria included a full-scale IQ < 80, a history of pervasive developmental

disorder, schizophrenia, or other psychotic or organic mental disorders, and/or seizures or other neurological problems.

The first follow-up occurred an average of 8.26 years (SD = 2.72) after the initial diagnosis was made. Enrollment occurred on a rolling basis between 1999 and 2003 with annual interviews followed by age-specific interviews in adulthood. Of those eligible for the initial PALS follow-up (n = 516), 70.5% participated (n = 364). ADHD participants had slightly lower average CD symptom ratings compared to those who did not participate (participants M = .43, non-participants M = .53, Cohen's d = .30). No differences between these groups were found for other demographic, diagnostic, and related symptomatology variables.

nonADHD participants.—Participants without ADHD were recruited into PALS on a rolling basis during the same time that the ADHD participants were recruited into the follow-up study (see Molina et al., 2012 for details). This ensured demographic similarity among the groups (i.e., age within one year, race, sex, highest parental education). Participants were excluded if they met any exclusion criteria listed previously for the ADHD group. The ADHD group participants (n = 364) and nonADHD comparison participants (n = 240) did not differ significantly on age, sex, ethnicity/racial minority status, or highest parental education. Those included in the comparison group were interviewed on the same assessment schedule as the ADHD group.

Subsample for the current study.—PALS participants were interviewed annually through age 23, then moved to an age-based interviewing schedule with target ages of 25, 27, 30, and every 5 years thereafter. Each age-based assessment included a window surrounding the target; for example, 30-year old assessments were conducted between the ages of 29–31 with an average of 29.6. For the purposes of the analyses described herein we analyzed data from participants at the ages of 21, 23, 25, 27, and 29 (the latter is referred to as age 29 instead of 30 in this paper due participants' average age of 29.6). The selection of these ages allowed us to examine growth in depression and heavy alcohol use in early adulthood and model data at equal age intervals. All participants provided data at least once between ages 21 and 29. 167 participants reported on heavy alcohol use at all five of these ages, 220 at four ages, 105 at three ages, 49 at two ages, and 33 at one age. Similarly, 119 participants reported on depression at all five ages, 182 at four ages, 110 at three ages, 81 at two ages, and 60 at one age. Some of these participants did not provide ADHD symptom persistence data, so analyses considering ADHD symptom persistence included 272 probands and 192 comparison participants (see below for details on measurement). Included versus excluded participants did not differ in sex (χ^2 = 1.50, p = 0.22) or race (χ^2 = 1.92, p = 0.17).

Procedure.—Post-baccalaureate research staff interviewed participants and parents in ADD program offices. Questionnaires were completed privately by participants and their parents. Informed consent was obtained and participants were assured confidentiality of disclosed material except in cases of imminent harm or danger to self or others (reinforced with a DHHS Certificate of Confidentiality). Mail and telephone were used to gather information in the cases where distance prevented office visits.

Measures

Depression.—The Center for Epidemiologic Studies Depression Scale (CES-D; Radloff 1977) is a self-reported 20-item questionnaire that assesses frequency of depression symptomatology (0 = rarely or none of the time to 3 = most or all of the time). Total scores were calculated as the sum of item responses and ranged from 0 to 60, with higher scores indicating greater symptoms of depression. Sum scores were used because no missing data at the item level was present. The CES-D has high internal consistency in both general (.85) and clinical (.90) populations and evidence of construct validity (Radloff 1977). From the ages of 21–29, descriptive statistics showed the following ranges: M = 11.34–12.15, SD = 7.44–9.60, Cronbach's Alpha=0.78–0.90.

Heavy Alcohol Use.—Participants reported their frequency of heavy episodic drinking ("In the past 12 months, how often did you drink 5 or more drinks when you were drinking?") on a 12-point response scale (e.g., 0 = not at all, 3 = 8–11 times; 11 = several times a day). This item was from the Substance Use Questionnaire (SUQ; Molina and Pelham, 2003), adapted from the Health Interview Questionnaire (Jessor et al. 1981) and the National Household Survey of Drug Abuse interview. From the ages of 21–29, descriptive statistics showed the following ranges: M = 2.37–3.25, SD = 2.53–2.87, Range=0–11.

Adulthood ADHD Symptom Persistence.—The Adult ADHD Rating Scale was administered to participants and their parents at selected follow-up visits (provided by R. Barkley before publication; Barkley 2011). Eighteen items [0 = never to 3 = very often] corresponding to the *DSM–IV–TR* symptoms of ADHD (Barkley et al. 2008; Kessler et al. 2010) were averaged to compute an ADHD symptom severity score. We calculated an ADHD symptom persistence variable based on prior work with this sample (Sibley et al. 2012). ADHD group participants who scored 2 standard deviations above the nonADHD group means, at any assessment after age 25, on the basis of parent- or self-reported scores, were coded as ADHD symptom persistent (1). All others, including those who were symptom-desistent by adulthood, were coded as 0.

Delinquency.—Participants and parents completed the Self-Reported Delinquency Questionnaire (SRD; Elliott et al. 1985) at age 21, which assessed whether the participant had ever engaged in 35 different delinquent acts (e.g., Have you ever snatched someone's purse or wallet?; Elliott et al. 1985). These items were summed. Given the tendency for underreporting of delinquent behavior by individuals with ADHD, the highest report (participant or parent) at the item level was used to create the total delinquency score (Sibley et al. 2010) (M = 1.79, SD = 2.61, Range=0–32).

Demographics.—Participants self-reported their race and sex.

Data Analytic Plan

Hypotheses were tested using latent growth curve modeling in Mplus version 7.2 (Muthén and Muthén 2015). Full information maximum likelihood was used to accommodate missing data and the maximum likelihood estimator was used. We used a model building approach,

described below, to arrive at a final model that examined whether associations between heavy alcohol use and depression symptoms differed by ADHD.

We examined graphical displays of average- and individual-level trajectories of heavy alcohol use and depression and fit linear, quadratic, and piecewise linear unconditional growth curve models to determine the optimal functional form of growth for each variable separately. Piecewise linear models capture nonlinearity by modeling two or more linear slope factors that are joined by a transition point or "knot." As recommended (i.e., Flora 2008), we selected the age at which to place the knot by graphically exploring the data to locate qualitative differences in patterns of change, given that there were no theoretically- or practically-based transitions in our data, such as the start of an intervention (Flora 2008). A knot of age 25 was selected because graphical displays showed that, after age 25, the depression slope began to decrease more steeply, whereas the heavy alcohol use slope began to decrease less steeply (see Figure 1). This knot also allowed for each "piece" (linear growth factor) to include three indicators, necessary for model identification (see Figure 2). The depression and heavy alcohol use indicators at ages 21–29 were set to 0, 1, 2, 2, and 2, respectively, for the first growth factor piece (ages 21–25) and were set to 0, 0, 0, 1, and 2, respectively, for the second growth factor piece (ages 25–29).

Second, the optimal models were combined to test the fit of unconditional parallel process models of heavy alcohol use and depression together and to evaluate the growth factors on average for the entire sample. All slope and intercept factors were allowed to covary. Next, mean-centered time-invariant covariates were added (sex, race, and age 21 delinquency) and the parallel process model was grouped by: 1) childhood ADHD vs. nonADHD or, 2) adulthood ADHD symptom persistence vs. no adulthood ADHD persistence (combining together ADHD group participants coded as ADHD desistent with those in the nonADHD group). Using the model building procedures described by Bollen & Curran (2006), we estimated the multiple group models in the following fashion. We started with a model where the effects of covariates on the growth parameters were constrained to be equal across groups, but all other parameters were freely estimated. This was the equivalent of excluding covariate-by-group interactions on growth parameters. We did so at the outset in an effort to reduce the number of tests performed, especially given that we did not have a priori hypotheses about covariate-by-ADHD interactions. In fact, many of the possible interaction effects make little sense from an a priori perspective (e.g., demographic variables interacting with ADHD symptom persistence at the ages of 25–29 to predict variables occurring in the past, such as the alcohol intercept at age 21).

From this initial model, we then constrained parameters to be equal across groups in the following order: means of intercept and slope factors, variances of intercept and slope factors, covariances among intercept and slope factors, and residual variances and covariances of indicators. Chi-square difference tests examined whether model constraints resulted in significantly worse fit. For a non-significant chi-square test, the equality constraints were retained. For a significant chi-square test, we identified sources of model misfit by examining modification indices > 3.8 (corresponding to p=0.05). These parameters were released one at a time until no other parameters reached this modification index

threshold. In all models, fit was determined using conventional fit statistics (e.g., chi-square, RMSEA, and CFI).

Results

Optimal functional form of growth curves.

See Figure 1 for the means of heavy alcohol use and depression from ages 21–29 by childhood ADHD diagnosis. See Table 1 for fit statistics of the growth curves tested. The linear heavy alcohol use and depression models did not fit the data as well as the quadratic or piecewise linear models. Although the quadratic models had slightly better fit than the piecewise models, the piecewise models still had very good fit to the data and provided a more parsimonious approach to capturing nonlinearity and interpretation (Flora 2008). Therefore, the depression and heavy alcohol use piecewise linear growth models with knots at age 25 were selected for further analysis.

Growth model across all participants.

The unconditional parallel process model fit the data well ($\chi^2 = 64.27(35)$, $p = 0.002$, CFI=0.98, RMSEA = 0.04). See Tables 2 and 3 for parameter estimates.

Depression.—On average, depression levels at age 21 were significantly different from zero and showed significant individual differences (see Table 2). On average, the rates of change in depression were not significantly different from zero during ages 21–25 or ages 25–29, but there were significant individual differences in the rates of change (Table 2). This reflects that, on average, individuals were not changing in depression over these ages, but there was meaningful individual change over time reflected in the significant variance. Higher levels of depression at age 21 were associated with faster declines in depression from ages 21–25 (see Table 3). Faster declines in depression from ages 21–25 were associated with slower declines in depression from ages 25–29 (Table 3). No other depression parameters significantly covaried.

Heavy alcohol use.—On average, heavy alcohol use levels at age 21 were significantly different from zero and showed significant individual differences (see Table 2). On average, the rates of change in heavy alcohol use declined from ages 21–25 and ages 25–29, and there were significant individual differences in the rates of change (Table 2). Higher levels of heavy alcohol use at age 21 were associated with faster declines in heavy alcohol use from ages 21–25 (see Table 3). No other heavy alcohol use parameters significantly covaried with one another.

Associations across depression and heavy alcohol use.—A positive covariance between the depression and heavy alcohol use slopes from ages 25–29 suggested that individuals showing faster increases in depression also showed faster increases in heavy alcohol use at these ages (see Table 3). Non-significant covariances included those between: heavy alcohol use and depression intercepts, heavy alcohol use intercept and depression slopes at both ages, depression intercept and heavy alcohol use slopes at both ages, age 21–

25 heavy alcohol use and depression slopes, and age 21–25 alcohol slope and age 25–29 depression slope (Table 3).

Growth model comparing childhood ADHD to no childhood ADHD.

The multiple group conditional parallel process model fit the data well (χ^2 = 192.78(135), p< 0.001, CFI=0.96, RMSEA = 0.04). See Table 2 and Figure 2 for parameter estimates. Childhood ADHD groups differed on several depression parameters. On average, participants with childhood ADHD showed significantly higher mean levels of and greater individual differences in levels of depression at age 21 than those without ADHD (see Table 2). Participants with childhood ADHD also showed significantly steeper negative slopes in age 21–25 depression, as well as fewer individual differences in age 21–25 depression slopes, than those without ADHD (although note that for neither group were the slopes significantly different from zero; Table 2). In contrast, participants with childhood ADHD showed significantly steeper positive slopes in age 25–29 depression, as well as greater individual differences in age 25–29 depression slopes, than those without ADHD (note that this slope was only significantly different from zero for the nonADHD group; Table 2). Childhood ADHD groups also differed on several heavy alcohol use parameters. On average, participants with childhood ADHD showed significantly lower levels of heavy alcohol use at age 21 and steeper negative slopes in heavy alcohol use from ages 21–25 (see Table 2). The residual variances and covariances of indicator variables were also significantly different across groups and were therefore allowed to vary. No other parameters differed across groups.

Note that one covariance that was not previously significant in the single group parallel process model became significant in only this model, which showed that greater levels of heavy alcohol use at age 21 were significantly associated with faster declines in heavy alcohol use from age 25–29 (invariant across ADHD vs. nonADHD groups; see Figure 2). In addition, the covariance between the age 21–25 and 25–29 depression slopes was previously significant in the full sample (single group) parallel process model but became non-significant in this model, and this non-significant effect was invariant across ADHD vs. nonADHD groups (Figure 2).

Growth model comparing adulthood ADHD persistence to no adulthood ADHD persistence.

The multiple group conditional parallel process model fit the data well (χ^2 = 190.30(131), p < 0.001, CFI=0.96, RMSEA = 0.04; n =470). See Tables 2 and 3 for parameter estimates. Results were identical to those from the childhood ADHD model except higher levels of heavy alcohol use at age 21 were no longer associated with faster declines in ages 25–29 heavy alcohol use in this model (across all participants; Table 3).

Summary of associations.

The only cross-disorder covariance that remained significant across all models, but did not differ by childhood or adulthood persistent ADHD, was the positive covariance between ages 25–29 heavy alcohol use and depression slopes. In addition, across all models, the depression intercept was associated with the age 21–25 depression slope, and the alcohol

intercept was associated with the age 21–25 alcohol slope, and these associations did not differ by childhood or adulthood persistent ADHD.

Post-hoc analyses.

To better understand the age-specificity found in the association between depression and heavy alcohol use (i.e., significant only from ages 25–29), we conducted post-hoc analyses to test whether the strength of the association between depression and alcohol slopes from ages 25–29 significantly differed from the depression-heavy drinking association from ages 21–25. The association between the slopes in the late 20s was significantly stronger than the parallel association in the early 20s in the childhood ADHD model (Wald $\chi^2 = 4.44(1)$, $p = 0.04$), but was only marginally significantly stronger in the adulthood ADHD model where the sample size was lower (Wald $\chi^2 = 2.89(1)$, $p = 0.09$).

The association between the depression and heavy drinking slopes from ages 25–29 characterizes individuals whose depression and heavy drinking declined together over time, as well as individuals whose depression and heavy drinking increased together over time. Those with ADHD showed significantly faster growth in depression from ages 25–29; thus, those with ADHD may be more likely to be simultaneously *increasing* in drinking and depression as opposed to simultaneously *decreasing* in drinking and depression. We conducted post-hoc analyses to quantify this possibility that could not be detected by the currently employed latent variable multiple-group models. We identified categorical groups of childhood ADHD and age 29 depression using established clinical cut-offs (≥ 24 indicates severe depressive symptomatology) and compared them on age 29 heavy drinking. Results indicated that 0% (0/11) of nonADHD participants with severe depressive symptomatology reported weekly or more heavy drinking. In contrast, 26.7% (8/30) of those with childhood ADHD plus severe depressive symptomatology reported weekly or more heavy drinking, suggesting that those with ADHD are more likely to show high levels of both problems. The difference in heavy drinking between these groups was marginally significant ($\chi^2(1) = 3.64$, $p = 0.056$). A scatterplot of the associations between the depression and heavy drinking slopes from ages 25–29, showing a subgroup of ADHD group cases with relatively stronger positive associations, also complements these observations (see Supplementary Figure 1).

Discussion

The present investigation is one among few to examine the longitudinal associations between heavy alcohol use and depression solely in adulthood, as well as how these associations differ by research-quality childhood ADHD diagnoses and symptoms of ADHD persisting into adulthood. Thus, the findings from this study are uniquely informative about long-term risk for individuals with and without ADHD. We found that heavy alcohol use and depressive symptoms changed in concert in the late 20s (ages 25–29), but not in the early 20s (ages 21–25). A post-hoc analysis also showed that the association between the depression and alcohol slopes was significantly stronger for the older, versus younger, participants. Although the strength of this association did not differ significantly by childhood ADHD diagnosis or adulthood ADHD symptom persistence, the slopes of depression and heavy drinking for the older participants were highly variable and older

individuals with ADHD had significantly faster growth in depression. Thus, these findings and our post-hoc analyses provided some suggestion that those with ADHD may have increased vulnerability to co-occurring heavy drinking and depression as they age into their late 20s.

Covariation between Heavy Alcohol Use and Depressive Symptoms

We found that changes in depression and heavy drinking tracked together in the late 20s for all adults regardless of ADHD history (i.e., drinking and depression increased, decreased or stayed stable *together*). In contrast, we did not find an association between the slopes of heavy alcohol use and depression at younger ages (from ages 21–25). In fact, in the childhood ADHD model, we found that the depression-alcohol use association was significantly weaker in the early 20s than in the late 20s. Thus, heavy alcohol use and depressive symptoms may track together more strongly as individuals enter their late 20s.

The tighter linkage between depression and heavy drinking over time may be due to a shifting from positive to negative reinforcement-driven drinking. Indeed, the early 20s is a peak period of drinking in the U.S. during which celebratory and socially-motivated drinking is most typical (Schulenberg and Maggs 2002); this is consistent with our finding that depression and heavy drinking in the early 20s were not associated. Studies of individuals not selected for ADHD provides some support for this idea. Research on alcohol use by adolescents revealed only inconsistent associations between depression and drinking after controlling for externalizing problems (Hussong et al., 2017).

In contrast, the stronger link that we found between heavy drinking and depression in the late 20s might be explained by the greater and more stressful responsibilities that emerge in adulthood. Failing to attain adulthood roles (e.g., financial security, stable romantic relationships) or perceiving adulthood roles as particularly stressful (e.g., marital problems, occupational stress) may engender distress that independently influences both depression and heavy drinking, causes depression and subsequently leads to heavy drinking to cope with negative affect, or causes greater drinking, which leads to greater depression (Littlefield and Winograd 2013; Mirowsky and Ross 1992; Rosenthal et al. 2012). In line with our findings, researchers have documented a group whose alcohol use disorder onsets later in life and may partly function to relieve negative affect (i.e., type 1 alcoholism; Cloninger et al. 1996; Zucker, 1986). The strengthening of negative reinforcement addiction processes with age may also explain why we did not find an association between age 21 levels of depression and rates of change in heavy alcohol use through the 20s, or between age 21 levels of heavy alcohol use and rates of change in depression through the 20s.

Our initial hypothesis, that depression and alcohol use would be more strongly associated for the ADHD versus nonADHD group participants, was not supported in the main analyses (multiple group, latent growth curve models). However, it is important that the positive association between depression and alcohol use among the older participants characterizes individuals with strong parallel *decreases* as well as strong parallel *increases* in these constructs. Our data provide tentative indications that those with childhood or persistent adulthood ADHD are more likely to show parallel increases, as opposed to decreases, in these associations. Indeed, our data showed that those with ADHD have steeper increases in

depression than those without and that there were more heavy drinkers in the depressed ADHD versus nonADHD groups. Visual inspection of the scatterplot of the association between the depression and heavy drinking slopes from ages 25–29 also suggested an emergence of a stronger depression-alcohol link for the ADHD than nonADHD group.

Although beyond the scope of this study, our findings suggest that future research should examine whether there exist latent groups underlying these growth trajectories (i.e., growth mixture modeling), especially because depression and heavy alcohol use trajectories from ages 25–29 showed significant variability. Based on present results, we hypothesize that a group characterized by fast increases in heavy alcohol use and depression in the late 20s would be overrepresented by those with ADHD. If substantiated, results would suggest that clinicians should remain aware of the heightened risk for heavy drinking and depression co-occurrence in adult patients with ADHD, even during the late 20s when many individuals have already "matured out" of such behaviors. In addition, following these associations beyond age 30 may be important to detect continuation and strengthening of this process for some individuals.

Differences in Growth Parameters by ADHD

We found that participants with childhood or adulthood ADHD were less likely to report heavy alcohol use at age 21 and that their drinking frequency between ages 21 and 25 decreased more rapidly than for those without ADHD. Groups did not differ in their slopes of heavy drinking from ages 25–29. These are somewhat surprising findings given multiple reviews indicating increased risk for negative alcohol outcomes among children with ADHD (Charach et al. 2011; Lee et al. 2011). However, there are several reasons these findings may have occurred in our data. First, heavy drinking in this age range (i.e., 21–25) is closely tied to substance use in one's peer network (Andrews et al. 2002). Interestingly, individuals with ADHD may be protected from heavy drinking during emerging adulthood because of social impairments that lead to withdrawal or isolation from peers (Molina and Pelham 2014). Alternatively, lighter drinking reported by those with ADHD could be due to lower college attendance rates, which is typically linked with heavier alcohol consumption (Kuriyan et al. 2013; Pingault et al. 2011). Consideration of these alternative social and impairment-related mediational pathways to negative alcohol outcomes was beyond the scope of the current analyses but should be considered in future examinations of ADHD-related pathways to alcohol problems in adulthood.

Despite our finding less frequent heavy drinking by those with versus without ADHD, previous findings from this sample and others have shown more alcohol-related *problems* and alcohol use disorder in adulthood associated with childhood ADHD (e.g., (Lee et al. 2011; Pedersen et al. 2016)). This suggests that even at comparable levels of heavy drinking, individuals with ADHD are more likely to experience negative consequences and dependence symptoms from alcohol use than those without. We specifically chose to model heavy drinking because it is an indicator of problematic alcohol use that has more variability than alcohol problems or dependence (Esser et al. 2014), and of the two variables, it is the more proximal alcohol behavior that might result from depression. Future work in this area would benefit from testing alternative models that allow consideration of additional alcohol

use indicators or progression from heavy drinking to alcohol use disorder as a function of worsening depression. Clarifying the unique mechanisms that may render individuals with ADHD more at risk for alcohol problems at levels of drinking that do not create problems for their nonADHD group peers could also yield new targets for alcohol treatment in this group.

Our results tell an interesting developmental story of how the course of depression differs between those with versus without ADHD. Compared to participants without ADHD, those with childhood and adulthood ADHD showed higher levels of depression at age 21, which is consistent with an earlier report from this sample showing greater depression at age 18 for those with childhood ADHD (Meinzer et al. 2016). Although Meinzer et al. (2016) did not find that childhood ADHD predicted different rates of decline in depression from ages 18–25, our results showed that individuals with ADHD more quickly declined in depression from ages 21–25; this discrepancy is likely due to the examination of different age windows, which will naturally capture differing rates of change. Our results specifically showed that depression *declined* on average from ages 21–25 for those with ADHD, whereas they *increased* on average for those without ADHD (these slopes were each marginally significant in the childhood ADHD model). These trends could reflect regression to the mean. An alternative explanation is that many comparison participants in this sample were experiencing the transition out of college and associated increases in distress and depression (Compas et al. 1986). However, this group also declined in depression rapidly from ages 25–29, consistent with age-normative declines in depression (Mirowsky and Ross 1992). Those in the ADHD group actually showed positive growth in depression from ages 25–29 (although not statistically significant). Impairment across multiple domains of adulthood functioning may be responsible for this result (e.g., Altszuler et al. 2016; Hechtman et al. 2016).

We found that grouping participants by childhood ADHD versus adulthood persistent ADHD produced very similar findings. The association between adulthood heavy alcohol use and depressive symptoms does not appear to differ for those whose ADHD symptoms persist into adulthood. This finding is somewhat surprising and important given the common clinical expectation that adulthood persistent ADHD symptoms should worsen multiple forms of impairment. Indeed, Hechtman et al. (2016) recently found that those with symptom-persistent ADHD had greater rates of mood disorders and risky behaviors than those with symptom-desistent ADHD or those without ADHD (Hechtman et al. 2016). Because adulthood ADHD symptom persistence did not drive a stronger relation between depression and heavy drinking in the late 20s, perhaps other factors are at play, such as family history vulnerability for these problems or ADHD-related impairments that remain present even among symptom-desistent individuals. We note that stronger differences by adulthood persistent ADHD may have emerged had we not included symptom-desistent individuals in our comparison group. Although we did not have an adequate sample size to conduct multiple group analyses using all three groups (i.e., no childhood ADHD, adult symptom-desistent, adult symptom-persistent), we conducted post-hoc analyses to examine whether findings would change after excluding symptom-desistent participants ($n = 84$) from the analysis (results available upon request). All findings remained identical to the

original model of adulthood ADHD persistence, suggesting that including symptom-desistent individuals in the comparison group did not attenuate group differences.

Finally, it is worthwhile to consider how revisions to the DSM would influence the diagnoses of PALS participants given this study's focus on comparisons by ADHD diagnosis. Two major changes to the ADHD diagnostic criteria in the DSM-5 included reducing, from six to five, the minimum number of symptoms needed to meet criteria for each symptom domain, as well as increasing the threshold for the first onset of symptoms from age 7 to 12. For PALS, these diagnostic changes could potentially result in some nonADHD participants being diagnosed with ADHD, given the more relaxed criteria. However, these revisions would not have resulted in an exclusion of participants from the PALS proband group.

Conclusions

In sum, associations between depressive symptoms and heavy alcohol use in early adulthood tracked closely together in the late 20s, but not in the early 20s. This strengthening association appears to have modest implications for adults with an ADHD history and suggests that monitoring of depression and heavy drinking together may be important for this population. The late 20s may be a particularly salient period in which to study reciprocal relations and shared causes of depression and heavy alcohol use. Future research is needed to elucidate factors that differentiate individuals whose depression and alcohol use, including alcohol-related problems, grow increasingly inter-dependent beyond early adulthood.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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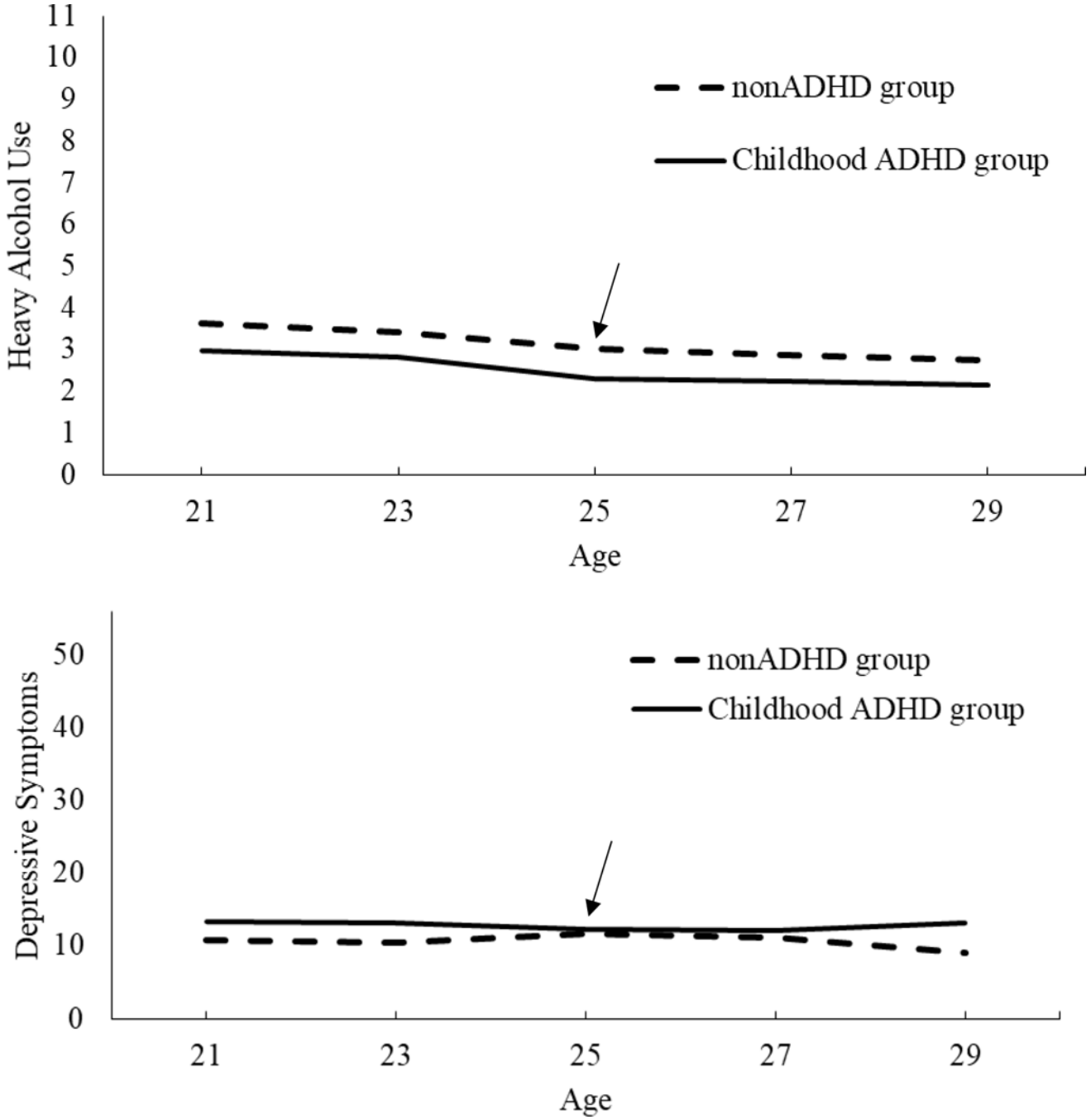

Figure 1.
Means of heavy alcohol use and depressive symptoms from ages 21–29 by childhood ADHD and non-ADHD groups. Arrows refer to the age at which the "knot" was placed.

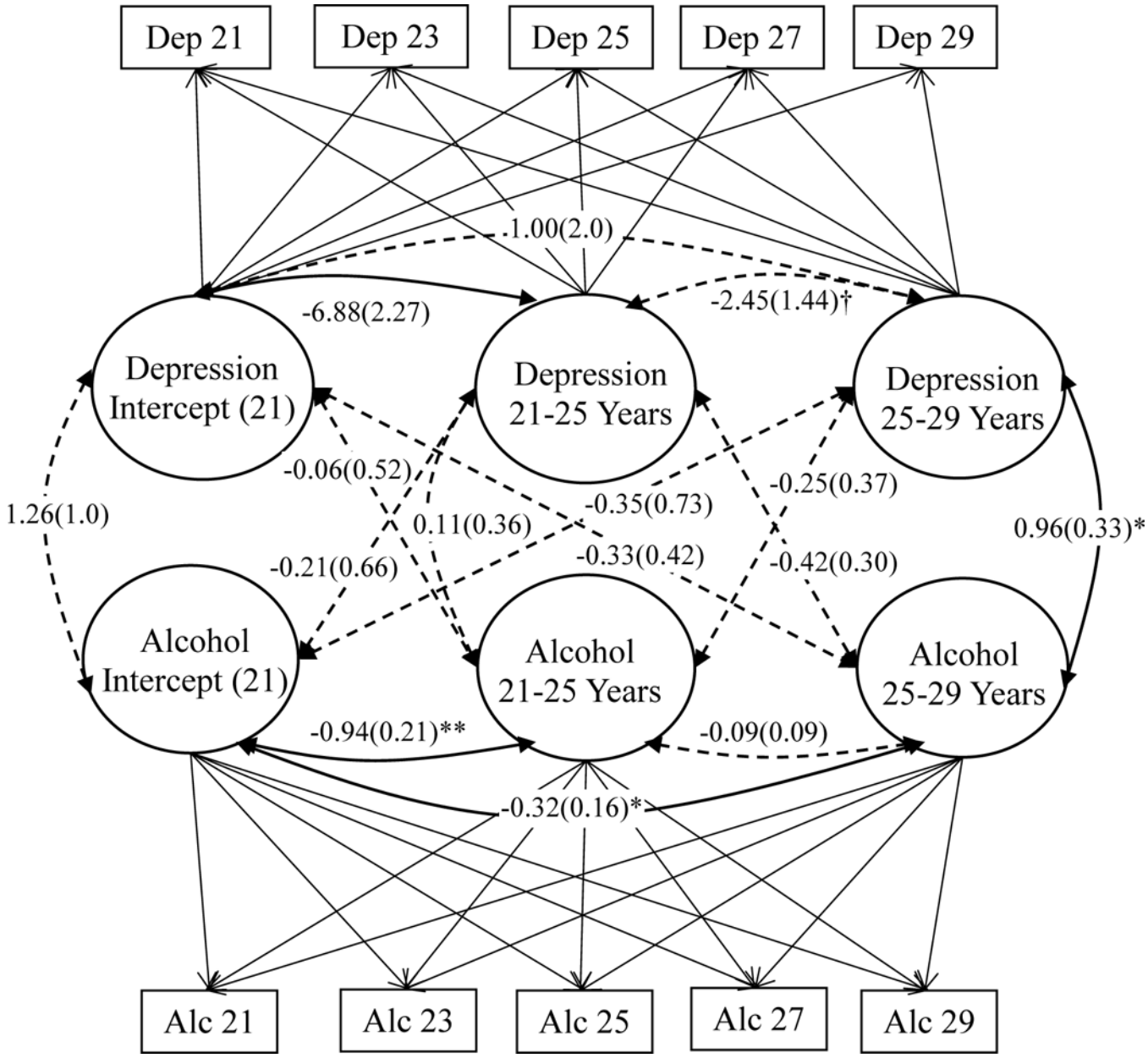

Figure 2.

Childhood ADHD Status Multiple Group Model of Piecewise Parallel Process Depression and Heavy Alcohol Use Latent Growth Curves. $^{**}p < 0.001$, $^{*}p < 0.05$, $\dagger p < 0.10$. Unstandardized coefficients are shown. Covariates include race, sex, and delinquency at age 21. Dashed lines indicate paths that were not statistically significant. Solid lines indicate paths that were statistically significant. Dep: Depression indicator. Alc: Heavy Alcohol Use indicator.

Table 1.

Fit Statistics of Competing Longitudinal Growth Models

	χ^2(df), *p*	CFI	RMSEA	AIC	BIC
Linear Depression	67.63(14), <0.001	0.866	0.083	12763.713	12789.595
Quadratic Depression	29.09(10), 0.001	0.952	0.059	12733.166	12776.301
Piecewise Depression	31.64(10), <0.001	0.946	0.063	12735.723	12778.858
Linear Heavy Alcohol Use	46.82(14), <0.001	0.969	0.064	9346.112	9372.228
Quadratic Heavy Alcohol Use	13.80(10), 0.18	0.996	0.026	9321.095	9364.621
Piecewise Heavy Alcohol Use	18.93(10), 0.04	0.992	0.039	9326.224	9369.751

Note. CFI: Comparative Fit Statistic. RMSEA: Root Mean Square Error of Approximation. AIC: Akaike Information Criteria. BIC: Bayesian Information Criteria.

Table 2.

Means and Variances of Intercept and Slope Factors for Full Sample Model, Childhood ADHD Multiple Group Model, and Adulthood ADHD Multiple Group Model

	Unconditional Full Sample Parallel Process Model (*n* =574)		**Multiple Group Conditional Model by Childhood ADHD (*n* =574)**		**Multiple Group Conditional Model by Adulthood ADHD (*n* =470)**	
	Means(*SE*)	**Variances(*SE*)**	**Means(*SE*)**	**Variances(*SE*)**	**Means(*SE*)**	**Variances(*SE*)**
Depression Intercept	12.12(0.38)**	38.96(5.0)**	ADHD:13.32(0.56)** N: 10.49(0.45)**	ADHD: 42.35(5.70)** N: 20.03(3.99)**	ADHD: 14.49(0.70)** N: 10.48(0.44)**	ADHD: 46.68(6.93)** N: 19.70(3.84)**
Depression 21–25 Slope	−0.05(0.24)	7.79(1.83)**	ADHD: −0.56(0.33)† N: 0.59(0.33)†	ADHD: 3.84(1.83)* N: 9.14(2.06)**	ADHD: −0.61(0.41) N: 0.32(0.31)	ADHD: 4.51(2.11)* N: 9.20(2.08)**
Depression 25–29 Slope	−0.34(0.26)	12.53(2.20)**	ADHD: 0.31(0.37) N: −1.10(0.33)**	ADHD: 12.49(2.72)** N: 8.39(2.17)**	ADHD: 0.71(0.47) N: −1.06(0.31)*	ADHD: 13.52(3.36)** N: 7.62(2.02)**
Alcohol Intercept	3.29(0.13)**	6.54(0.56)**	ADHD: 3.06(0.17)** N: 3.61(0.18)**	5.31(0.48)**	ADHD: 3.06(0.20)** N: 3.30(0.18)**	5.21(0.51)**
Alcohol 21–25 Slope	−0.30(0.06)**	0.77(0.14)**	ADHD: −0.32(0.08)** N: −0.27(0.09)*	0.72(0.14)**	ADHD: −0.40(0.10)** N: −0.23(0.09)**	0.69(0.14)**
Alcohol 25–29 Slope	−0.15(0.06)*	0.31(0.11)*	−0.16(0.05)**	0.31(0.10)*	−0.15(0.06)*	0.23(0.11)*

Notes.

** $p < 0.001$,

* $p < 0.05$,

† $p < 0.10$. Childhood ADHD and persistent adulthood ADHD were grouping factors in the latter two models. Parameter estimates that significantly differed by group are presented separately for “ADHD” (ADHD group) or “N” (nonADHD Group). When ADHD and nonADHD participants did not differ, the parameter estimates presented apply to the full sample for each model. Unstandardized coefficients are shown. The unconditional model did not include covariates. The conditional models included mean-centered time-invariant covariates of race, sex, and delinquency at age 21. Thus, the means of intercepts and slopes for the multiple-group models (but not for the full group model) represent values for participants with average levels of delinquency, and adjusted for the proportion of female and Caucasian participants in the sample.

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Table 3.

Covariances among Growth Parameters for the Full Sample Parallel Process Model (Unconditional Model) and the Multiple Group Adulthood ADHD Model (Conditional Model)

	Depression Intercept	Depression 21–25 Slope	Depression 25–29 Slope	Alcohol Intercept	Alcohol 21–25 Slope	Alcohol 25–29 Slope
Depression Intercept	--	−9.53(2.51)**	2.94(2.29)	1.05(1.20)	−0.60(0.60)	−0.08(0.48)
Depression 21–25 Slope	−7.77(2.38)*	--	−3.92(1.52)*	0.03(0.74)	0.24(0.38)	−0.60(0.31)*
Depression 25–29 Slope	1.07(1.98)	−2.16(1.47)	--	−0.75(0.82)	−0.17(0.39)	1.14(0.36)**
Alcohol Intercept	1.03(1.03)	−0.08(0.69)	−0.72(0.74)	--	−1.27(0.23)**	−0.28(0.17)
Alcohol 21–25 Slope	−0.05(0.53)	0.14(0.38)	−0.13(0.37)	−0.93(0.22)**	--	−0.10(0.09)
Alcohol 25–29 Slope	−0.19(0.43)	−0.43(0.31)	0.82(0.34)*	−0.24(0.16)	−0.09(0.09)	--

Notes.

** $p < 0.001$,

* $p < 0.05$,

† $p < 0.10$. Top half above the diagonal are covariances from the full sample unconditional parallel process model ($n = 574$). Bottom half below the diagonal are covariances from the multiple group model by adulthood ADHD ($n = 470$). Unstandardized coefficients are shown. Covariates include race, sex, and delinquency at age 21.