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Historical variation in young adult binge drinking trajectories and its link to historical variation in social roles and minimum legal drinking age

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Abstract

This study examines historical variation in age 18–26 binge drinking trajectories, focusing on differences in both level of use and rates of change (growth) across cohorts of young adults over three decades. As part of the national Monitoring the Future Study, over 64,000 youths from the high school classes of 1976–2004 were surveyed at biennial intervals between ages 18 and 26. We found that, relative to past cohorts, recent cohorts both enter the age 18–26 age band engaging in *lower* levels and exit the age 18–26 age band engaging in *higher* levels of binge drinking. The reason for this reversal is that, relative to past cohorts, binge drinking among recent cohorts accelerates more quickly across ages 18–22 and decelerates more slowly across ages 22–26. Moreover, we found that historical increases in minimum legal drinking age account for a portion of the historical decline in age 18 level, while historical variation in social role acquisition (e.g., marriage, parenthood, and employment) accounts for a portion of the historical acceleration in age 18–22 growth. We also found that historical variation in the age 18–22 and age 22–26 growth rates was strongly and positively connected, suggesting common mechanism(s) underlie historical variation of both growth rates. Findings were generally consistent across gender and indicate that historical time is an important source of individual differences in young adult binge drinking trajectories. Beyond binge drinking, historical time may also inform the developmental course of other young adult risk behaviors, highlighting the interplay of epidemiology and etiology.

Keywords

Transition to adulthood; Binge drinking; Historical variation; trajectories; social role acquisition; minimum legal drinking age

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Both sociologists (e.g., Bynner, 2005; Settersten & Ray, 2010) and psychologists (e.g., Arnett, 2000) recognize that the transition to adulthood has changed such that it is now, on average, more protracted, more individualized, and less linear than it was in the past. Moreover, across the last three decades the rate of college attendance has increased, while adult roles and responsibilities, such as residential independence, marriage, parenthood, and the transition to full-time employment, have all been delayed (Settersten & Ray, 2010). This pervasive historical shift raises questions about how the course of health and well-being during this transition has changed in concert with these shifts in social role changes. Drawing from a developmental-contextual perspective, and using multi-cohort panel data, we examine how the trajectories of an important index of health and well-being across the transition to adulthood, binge drinking (i.e., consuming five or more drinks in a row), have changed across the last three decades.

Existing research based on national US samples indicates that mean levels of binge drinking typically follows an inverted-U shaped pattern across the ages of 18 to 26: increasing across the ages of 18 to 22 and then, after peaking around the age of 22, decreasing through the age of 26, after which levels of binge drinking begin to stabilize (Chen & Kandel, 1995; Johnston, O'Malley, Bachman, & Schulenberg, 2013). Recent binge drinking prevalence rates follow this same inverted-U shaped pattern. Specifically, prevalence rates across ages 18, 22, and 26 respectively are 35%, 51%, and 44% for males, and 23%, 35%, and 27% for females (Johnston et al., 2013). Importantly, although average levels of binge drinking are higher during the transition to adulthood than any other period of the lifespan (Chen & Kandel, 1995; Johnston et al., 2013), for most transitioning adults this elevated pattern of use does not extend further into adulthood due to the average trajectory of decreasing use across ages 22 to 26. Therefore, given the individual and societal costs – including economic, physical, and psychological - associated with life-long patterns of binge drinking (Rehm & Monteiro, 2005; Sturm, 2002), this pattern of declining use across ages 22–26 constitutes an important juncture of the lifespan.

Recent evidence indicates that these normative trajectories of binge drinking are also given to variation across historical time (i.e., historical variation). Specifically, Jager, Schulenberg, O'Malley, and Bachman (2013) found that between 1976 and 2007, adolescent (age 18) binge drinking decreased substantially; however, because the age 18–22 growth rate increased sharply during this period, binge drinking among 22-year-olds was largely stable historically; thus, age 18–22 binge drinking accelerated at a faster rate among recent cohorts. Although this is an important first step, it begs the question as to what has happened in recent cohorts between ages 22 to 26, when it is typical for binge drinking to decrease. Thus, to provide a more complete account of historical variation in young adult binge drinking trajectories, the present study focuses on the full 18–26 age band and, in doing so, extends the Jager et al. analyses in three key ways. For our first aim we examine historical variation in age 22–26 binge drinking. For our second aim we examine the extent to which historical variation in age 18–22 growth is connected with historical variation in age 22–26 growth. As

we describe in more detail below, we pursue this second aim because it will help clarify the extent to which the mechanisms that underlie historical variation in age 18–22 growth overlap with the mechanisms that underlie historical variation in age 22–26 growth. Finally, for our third aim we examine whether historical changes in both (a) the frequency and timing of social role acquisition (full-time college attendance, full-time employment, marriage, parenthood, and residential independence) and (b) minimum legal drinking age (MLDA) help explain historical variation in age 18–22 and age 22–26 binge drinking, including the extent of their connection to one another – as we examine in our second aim. To achieve these aims, we use national panel data from 28 consecutive cohorts of high-school seniors (graduating classes from 1976 to 2003) from the Monitoring the Future project (MTF; Johnston et al., 2013). Finally, we examine these aims separately for each gender. We do so due to known gender differences in both young adult binge drinking (e.g., males report higher levels of use as well as larger individual increases in use; Chen & Jacobson, 2012; Needham, 2007) and social role acquisition patterns (e.g., males on average transition into parenthood and marriage at an older age and are more likely to transition into full-time employment; Staff et al., 2010).

Documenting Historical Trends in Age 22–26 Binge Drinking

Building off of Jager et al. (2013), who found that the age 18–22 binge drinking growth rate accelerated across historical time, for our first aim we examine how, if at all, the age 22–26 binge drinking growth rate has varied across historical cohort over past three decades (Aim 1; Figure 1a). The answer to this question is important for at least two reasons. First, because clarifying how and why binge drinking varies across individuals is central to our understanding of binge drinking's etiology, documenting whether the typical rate of decline across age 22–26 varies depending upon the historical period within which individuals are embedded is essential to our understanding of both the etiology and prevention of problematic alcohol use. Second, it is important to document historical variation in the trajectories up through age 26 because by this age most young adults have transitioned into multiple adult roles (e.g., marriage, parenthood, full-time employment, and/or residential independence), and therefore hold many of the initial responsibilities of adulthood (Maggs, Jager, Patrick, & Schulenberg, 2013).

Age 18–22 and 22–26 Historical Trends: Overlapping Mechanisms?

The more historical trends in age 18–22 growth move in concert with historical trends in age 22–26 growth, the more historical trends across these two growth periods are likely attributable to the same underlying cause(s) and, therefore, can be understood as a singular phenomenon that warrant a singular research and, ultimately, policy response (as opposed to distinct responses for each growth period). Therefore, for our second aim we examine the extent to which historical variation in age 18–22 growth is related to historical variation in age 22–26 growth (Aim 2; Figure 1b). We do so using multi-level growth modeling that includes high-school cohort as the nesting variable. Put more concretely, we examine the cohort-level correlation between age 18–22 growth and age 22–26 growth (i.e., among the 28 high-school cohorts examined here, we examine whether cohort differences in age 18–22 growth are correlated with cohort differences in age 22–26 growth). While doing so, in

addition to the cohort-level correlation's strength, we also pay attention to its direction, as it suggests *how* the mechanisms responsible for historical variation in age 18–22 growth overlap with the mechanisms responsible for historical variation in age 22–26 growth.

For example, a cohort-level correlation between the age 18–22 and 22–26 growth rates that is at or near zero (i.e., non-significant) would indicate that the rate of a cohort's acceleration between the ages of 18–22 is unrelated to the rate of its deceleration between the ages of 22–26. Because this pattern would indicate that cohort differences in age 18–22 growth are unrelated to cohort differences in age 22–26 growth, it would suggest the absence of common mechanisms as well as the uniqueness of the two periods of development in regard to historical changes. Meanwhile, a positive cohort-level correlation would indicate that historical trends in age 18–22 growth move in concert with historical trends in age 22–26 growth. More specifically, a positive cohort level correlation would indicate that the faster a cohort's binge drinking increases across ages 18–22 (i.e., the more a cohort's age 18–22 growth is above the across-cohort mean) the slower a cohort's binge drinking decreases across the ages of 22–26 (the more a cohort's age 22–26 growth is above the across-cohort mean). This pattern would suggest overlapping, complementary mechanisms (i.e., mechanisms that underlie sharper increases across ages 18–22 also underlie weaker decreases across ages 22–26), as well as historical-developmental linkages between these two age periods – i.e., that the two periods experience historical change in similar ways. Finally, a negative cohort-level correlation would indicate that historical trends in age 18–22 growth run counter to historical trends in age 22–26 growth (i.e., the faster a cohort's binge drinking increases across ages 18–22, the faster a cohort's binge drinking decreases across the age of 22–26). While such a pattern would also suggest overlapping mechanisms (as well as historical-developmental linkages across the two age groups), it would suggest that the effect of the mechanism(s) are reversed – as opposed to complementary – across the two growth periods (i.e., mechanisms that underlie sharper increases across ages of 18–22 also underlie sharper decreases across ages 22–26).

Linking Historical Variation in Binge Drinking Trajectories to the Chronosystem

Moving beyond whether the same mechanisms underlie historical variation in both age 18–22 and age 22–26 growth (i.e., the focus of Aim 2), we now focus on identifying potential mechanisms for historical variation in both age 18–22 growth and age 22–26 growth. According to Bronfenbrenner's ecological model of human development (1977; 1994), the chronosystem is the most distal context within one's ecology and encompasses sociohistorical variation in society-level factors, such as opportunity structures, life-course options, and public policies. Here we focus on two components of the chronosystem that may help explain historical variation in young adult binge drinking trajectories: (a) historical variation in the frequency and timing of social role acquisition and (b) historical variation in minimum legal drinking age (MLDA).

Although young adults both recent and past have navigated what Settersten (2007) refers to as the “Big 5” young adult social roles (Big5; marriage, parenthood, education, residential status, and employment), due to historical changes in labor force structures and employment

options (i.e., chronosystem effects), the frequency and timing for most of these roles have varied across historical time (Arnett, 2000; Settersten, 2007). Moreover, in every case these historical trends have resulted in the increased occurrence among young adults of social roles that are associated with *more* binge drinking (i.e., attending college full-time, not working full-time, remaining single, and remaining childless; Bachman, Wadsworth, O'Malley, Johnston, & Schulenberg, 1997). Consequently, because the social roles associated with more binge drinking are more commonly held among today's young adults, this historical variation in the Big5 social roles may underlie, either in part or in full, historical variation in young adult binge drinking trajectories. Independent of historical variation in the Big5 social roles, changes in MLDA may also be associated with historical variation in young adult binge drinking trajectories. Due to changes in federal policy (i.e., chronosystem effects), between 1985 and 1987 thirty-one states (including the district of Columbia) increased their MLDA age from either age 18, 19, or 20 to age 21 (Hedlund, Ulmer, & Preusser, 2001; Wagenaar & Toomey, 2002). Following these changes, alcohol consumption decreased among the age cohorts that used to be of legal drinking age prior to the law change (O'Malley & Wagenaar, 1991; Wagenaar & Toomey, 2002). However, here we examine whether changes in MLDA altered growth of binge drinking, a matter that remains unclear. That is, although it is clear that MLDA contributed to reduced drinking based on analyses of point estimates, it is unknown the extent to which it also contributed to or altered trajectories of binge drinking across this critical transition to adulthood. Therefore, for our third aim we examine whether controlling for historical variation in the rates of Big5 social roles and in MLDA dampens historical variation in both age 18–22 growth and age 22–26 growth (Aim 3a; Figure 1c). In order to identify the unique and combined effects of the Big5 social roles and MLDA we examine their effects in a step-wise fashion.

Moreover, to the extent that historical variation in either the Big5 social roles or MLDA are a common mechanism underlying historical variation in both age 18–22 and age 22–26 growth, controlling for their historical variation should attenuate or weaken the cohort-level correlation between age 18–22 growth and age 22–26 growth. Therefore, for our third aim we also examine whether controlling for historical variation in the rates of Big5 social roles and in attenuates the cohort-level association between age 18–22 growth age 22–26 growth (Aim 3b; Figure 1d)

Methods

Respondents and Procedure

MTF is an ongoing national study of the epidemiology and etiology of drug use among adolescents and adults (Johnston et al., 2013). Beginning in 1975, large nationally representative samples of approximately 16,000 12th graders were drawn from about 135 public and private schools each year (Johnston et al., 2013). Beginning with the class of 1976, approximately 2,400 respondents were randomly selected for biennial follow-up from each cohort through mail surveys, with one random half being surveyed one year after high school and the other random half being surveyed two years after high school; each half was followed biennially thereafter (the two random halves of each cohort are combined in these analyses). Respondents who reported illicit drug use at baseline were oversampled for

follow-up. The national sample was predominantly White (72.8%), evenly split by gender (50.8% female), and, on average, had parents who completed some college.

In the present study we focused on the first 5 waves of the MTF panel data. Respondents were, on average, 18 years old at Wave 1, 19 to 20 years old at Wave 2, 21 to 22 years old at Wave 3, 23 to 24 years old at Wave 4, and 25 to 26 years old at Wave 5. For the purposes of this study, analyses were limited to those respondents who were in 12th grade between 1976 and 2003, and who provided data at one or more waves ($N = 64,833$). After adjusting for oversampling of drug users the weighted $N = 51,826$. Data extend until 2010/2011, when the most recent cohort of high-school seniors (i.e., the 2003 cohort) was aged 25/26. Wave to wave retention rates between Waves 1 and 5 were 69.70%, 80.31%, 81.21%, and 80.97%, respectively. The retention rate between Waves 1 and 5 was 57.07%. Attrition analyses indicated that compared to those lost, those retained through Wave 5 were more likely to be female, to be white, to live in non-urban areas, to report a higher high school GPA, to have more educated, affluent parents who are married, and to report lower senior year substance use, including binge drinking. These analyses also indicated that attrition rates were slightly higher among recent cohorts. Though statistically significant, the effects of attrition were small in magnitude (i.e., $R^2 = .03$ for high school GPA and race, $R^2 = .02$ for gender, and $R^2 < .005$ for all other variables, including binge drinking). In order to maximize the data, we include all possible cases, and adjust for effects of attrition, we used Full Information Maximum Likelihood estimation (FIML; Arbuckle, 1996), a missing data algorithm available within Mplus (Muthén & Muthén, 1998–2013). Using the auxiliary command within Mplus (Muthén & Muthén, 1998–2013), auxiliary variables related to missingness were included in the analyses.

Measures

Binge drinking—A single, self-report ordinal item was used to assess occasions of binge drinking, operationalized as 5 or more drinks in a row in the past two weeks. Item responses included 1 (*never*), 2 (*once*), 3 (*twice*), 4 (*3–5 times*), 5 (*6–9 times*), and 6 (*10 or more times*).

Cohort—The measure *cohort year* ranged from 0 to 27 and was based on the year that respondents were seniors in high school (i.e., 1976 seniors = 0, 1977 seniors = 1, and so forth). Using the variable *cohort year* and the *ORPOL* function within SAS/IML (SAS Institute Inc., 2011), we generated orthogonal polynomials ranging from the first (linear), second (quadratic), and third (cubic) degree. Collectively, these three measures were used to examine linear, quadratic, and cubic trends in the effect of cohort year on young adult binge drinking.

Social roles—At each wave we dichotomized, when available, indicators of parental status, marital status, full-time college status, residential status, and full-time employment status. All indicators were self-report and, unless noted otherwise, were available at each wave. For *marital status*, all those who were married were coded as 1 and all those who were single, divorced, separated, widowed, or engaged were coded as 0. For *parental status*, all those who had one or more children were coded as 1 and all those with no children were

coded as 0. For *full-time 4-year college status*, those who were attending a 4-year college full-time were coded as 1 and all others, including those attending two-year colleges (i.e., community colleges and vocational schools) and those attending any type of college part-time, were coded as 0. Data for college status were available for Waves 2 through 5. We focused on those attending a 4-year college full time for three reasons. First, the relation between college attendance and individual increases in alcohol use is strongest among those enrolled in 4-year colleges (Douglas et al., 1997) and those attending college full-time (Bachman et al., 1997). Second, attending a two-year college or attending college part-time versus attending a 4-year college full-time does not provide as many opportunities (both in terms of academics and social interactions) for exploration and experimentation (Davies & Casey, 1999). For example, relative to those attending a 4-year college full-time, those attending college part-time or attending a two-year college full-time are also more likely to work full-time, to drop out of school, and to be “nontraditional” students with their own families (Bailey, Leinbach, Scott, Alfonso, Kienzl, & Kennedy, 2003; Kane & Rouse, 1999). Third, Jager et al., 2013, which the present study builds upon, also focused on those attending a 4-year college full-time. For *residential status*, all those who lived with their parents were coded as 0 and those living independent of their parents were coded as 1. For *full-time employment status*, as defined by the US Census (2012), all those who worked 35 or more hours per week were coded as 1 and all others (i.e., those who worked less than 35 hours per week and those who did not work) were coded as 0. Data for full-time employment were available for Waves 2 through 5.

Minimum legal drinking age (MLDA)—*Minimum legal drinking age* was based on self-reported state residency at Waves 1, 2, 3, 4, and 5 as well as data on state-level changes in minimum drinking age compiled by others (Hedlund et al., 2001; Wagenaar & Toomey, 2002). Based on these data, we determined each respondent’s state’s minimum legal drinking age at Waves 1, 2, 3, 4 and 5. Where possible, we allowed for changes in state residency across waves. However, prior to 1987 state residency was asked at Wave 1 only. Therefore, for data collected prior to 1987, state residency at Waves 2, 3, 4, and 5 was based on state residency at Wave 1.

Results

Unless specified otherwise, all analyses were conducted with Mplus Version 7 (Muthén & Muthén, 1998–2013). The fit of each model tested was very good (in every case, CFI was greater than .98 and RMSEA was less than .02; McDonald & Ringo Ho, 2002); thus, given space constraints, fit indices for each model are not presented. Because substance use typically shows a positively skewed distribution, all analyses utilize a maximum likelihood estimator that is robust to non-normality.

Preliminary Analyses

As expected, among the aggregate sample (all cohorts combined) mean levels of binge drinking followed an inverted-U shaped pattern across ages 18 to 26. Specifically, mean levels across ages 18, 21/22, and 25/26, respectively, were 2.00, 2.23, and 2.01 for males, and 1.52, 1.62, and 1.43 for females. The same was true for prevalence rates, which across

ages 18, 21/22, and 25/26, respectively, were 42%, 53%, and 45% for males, and 25%, 31%, and 24% for females. Also as expected, across cohort year minimum legal drinking age increased (results not tabled), and the rates of experiencing social roles varied and, in every case the extent of variation was moderated by age (results not tabled). Specifically, the rate of full-time 4-year college attendance increased across cohort year and historical increases were more evident among the lower end of the 18–26 age band. The marriage rate, parenthood rate, rate of full-time employment, and rate of living with parents all decreased across cohort year, with decreases being more evident among the lower end of the 18–26 age band.

Baseline Model

We first examined the age 18–26 binge drinking trajectory in the aggregate sample. Using the *Baseline Piece-Wise Growth Model* in Figure 2, we estimated the five following intercepts and growth factors: (1) age 18 level, (2) age 18–21/22 growth rate, (3) age 21/22 level, (4) age 21/22–25/26 growth rate, and (5) age 25/26 level. Using piece-wise growth modeling (Li, Duncan, Duncan, & Hops. 2011), we broke age 18–26 growth into two linear growth pieces: (1) Age 18–21/22 (hereafter referred to as age 18–22) and (2) Age 21/22–25/26 (hereafter referred to as age 22–26). We estimated age 21/22 level and age 25/26 level by centering the intercept on age 21/22 (hereafter referred to as age 22) and 25/26 (hereafter referred to as age 26), respectively. Generally, all binge drinking estimates were of the expected magnitudes (See “Factor Means”, Table 1). For both genders, whereas the age 18–22 binge drinking growth rate was positive, the age 22–26 binge drinking growth rate was negative. As a result, the model-based level of binge drinking was highest at age 22. Next, by including cohort year as a grouping variable in the Baseline Piece-Wise Growth Model, we estimated the intercepts and growth factors for each of the 28 individual cohort years. For both males and females, each of these estimates (i.e., age 18 level, age 18–22 growth rate, age 22 level, age 22–26 growth rate, and age 26 level) is plotted by cohort year (Figure 3) to provide a visual indication of historical variation in each.

Aim 1: Magnitude and Scope of Historical Variation of Age 22–26 Binge Drinking

To examine historical variation in binge drinking, we built on the Baseline Piece-Wise Growth Model in Figure 2 and included the three cohort year polynomials (linear, quadratic, and cubic) as exogenous predictors of each binge drinking intercept and growth factor (*Cohort Year Piece-Wise Growth Model*, Figure 2). For the linear, quadratic, and cubic polynomial predictors respectively, the result is an estimate of the linear, quadratic, and cubic change in each binge drinking intercept and growth factor for each annual increase in cohort year. Standardized results are presented in the second through fourth columns of Table 1 (labeled as “Unadjusted”). Because this analytical approach is analogous to fitting a polynomial regression or “trend” line through the estimate-plots in Figure 3, Figure 3 includes these “trend” lines as well.

Age 18–22 binge drinking—Building on the Jager et al. (2013) findings regarding linear historical trends in age 18–22 binge drinking, we include here an emphasis also on quadratic and cubic trends across cohorts. Consistent with Jager et al., the linear effect of cohort year on age 18 level of binge drinking was negative for both males (–.144) and females (–.099),

indicating that age 18 level declined across cohort year; however, declines in age 18 drinking across cohort year were less steep among recent cohorts, as evidenced by the positive quadratic and cubic effects of cohort year (See “Age 18 Trend Line” in Figure 3a for males and Figure 3b for Females). Also consistent with Jager et al, the linear effect of cohort year on age 18–22 growth rate in binge drinking was positive for both males (.138) and females (.150), indicating that the age 18–22 growth rate, which was positive on average (Table 1), accelerated (became more positive) across cohort year; however, increases across cohort year in the age 18–22 growth rate were the steepest among the middle cohorts, as evidenced by the positive quadratic effect and negative cubic effect of cohort year (See “Age 18–22 growth trend line” in Figure 3c for Males and Figure 3d for Females). The linear effect of cohort year on age 22 binge drinking was negative for males (–.023), indicating that age 22 level declined across cohort year. However, for females the linear effect of cohort year on age 22 level of binge drinking was positive (.042), indicating that age 22 level increased across cohort year. Because the quadratic effect of cohort year on age 22 binge drinking was negative for both genders, historical decreases in male age 22 binge drinking was less pronounced among recent cohorts and historical increases in female age 22 binge drinking were more pronounced among recent cohorts (See “Age 22 Trend line in Figure 3a for males and Figure 3b for females).

Age 22–26 binge drinking—The linear effect of cohort year on age 22–26 growth rate in binge drinking was positive for both males (.088) and females (.056), indicating that the age 22–26 growth rate, which was negative on average (Table 1), decelerated (became less negative) across cohort year. However, for both genders increases across cohort year in the age 22–26 growth rate were the least pronounced among the earliest cohorts, as evidenced by the positive quadratic effect of cohort year (See “Age 22–26 growth trend line” in Figure 3c for males and Figure 3d for females).

Because the rate of decrease across ages 22–26 slowed across cohort year, age 26 level of binge drinking increased across cohort year for both genders. For males the linear effect of cohort year on binge drinking switched from negative at age 22 (–.023) to positive at age 26 (.038), and for females the linear effect of cohort year on binge drinking was even more positive at age 26 (.084) than it was at age 22 (.042). Moreover, for both genders historical increases in age 26 binge drinking were more pronounced among more recent cohorts, as evidenced by the positive quadratic effect of cohort year on age 26 level (See “Age 26 Trend line” in Figure 3a for males and Figure 3b for females). For females, however, the pace at which age 26 binge drinking increased across cohort did slow slightly among the most recent cohorts, as evidenced by the negative cubic effect of cohort year on age 26 level.

Summary—For both genders, although recent cohorts enter the age 18–26 age band engaging in lower levels of binge drinking relative to past cohorts, they exit the age 18–26 age band engaging in higher levels of binge drinking relative to past cohorts. Moreover, the reason for this reversal is that relative to past cohorts, binge drinking among recent cohorts accelerates more quickly across ages 18–22 and decelerates more slowly across ages 22–26. To illustrate these trends we created three cohort groups by dividing the 28 cohorts examined here into roughly equal thirds: “Distant” (76–84 cohorts), “Middle” (85–93

cohorts), and “Recent” (94–03 cohorts). Using these three cohort groups, Figure 4 lists the age 18–26 trajectories separately by gender and cohort group. Regardless of gender, across the Distant, Middle, and Recent cohort groups, age 18 level decreases monotonically (i.e., age 18 level is lowest among the Recent group), whereas the rate of increase across ages 18–22 increases monotonically (i.e., the increase across ages 18–22 is the sharpest among the Recent Group), while the rate of decrease across ages 22–26 decreases monotonically (i.e., the decrease across ages 22–26 is the least pronounced among the Recent group). Finally, for both genders the age 26 level of binge drinking is highest among the Recent Group. Although linear trends were the most pronounced (as evidenced by the standardized effects of the linear trends), there were also some notable non-linear trends. Specifically, beginning roughly with the early 90s senior-year cohorts, the historical decline in age 18 level slowed sharply while the historical increases in both age 22 and 26 levels quickened (Figure 4). Meanwhile, beginning with the early 80s senior-year cohorts the acceleration in age 18–22 growth and the deceleration in age 22–26 growth both quickened (Figure 4).

Aim 2: Do age 18–22 growth and age 22–26 growth move in concert historically?

To examine across-growth factor cohort-level associations, we conducted multi-level modeling by returning to the baseline piece-wise growth model (Figure 2) and including cohort year as a cluster variable. For our purposes here, we focus on the cohort-level association between age 18–22 growth and age 22–26 growth. For both males (.915, $p < .001$) and females (.903, $p = .002$), at the cohort level growth in binge drinking across ages 18–22 was positively related to growth in binge drinking across ages 22–26. Thus, the higher a cohort was (relative to other cohorts) on the age 18–22 binge drinking growth factor, the higher it was (relative to other cohorts) on the age 22–26 growth factor. Moreover the strength of the associations (i.e., $r > .90$ for both genders) suggests strong similarity between age 18–22 historical variation and age 22–26 historical variation. The positive value of the cohort-level associations indicate that for both genders historical trends in age 18–22 growth move in concert with historical trends in age 22–26 growth and, thereby, suggest overlapping, complementary mechanisms (i.e., mechanisms that underlie sharper increases across ages 18–22 also underlie weaker decreases across ages 22–26).

Aim 3: Accounting for Historical Variation in Big5 social roles and MLDA

To this point all analyses have used observed measures of binge drinking that do not adjust for historical variation in either the Big5 social roles (i.e., parental status, marital status, full-time 4-year college status, residential status, and full-time employment) or MLDA. For our third aim we, instead, used measures of binge drinking that adjust for the effects of historical variation (i.e., across-cohort variation) in these factors. Using these adjusted measures of binge drinking, we then reran the Aim 1 and Aim 2 analyses.

We produced two sets of adjusted measures: one set that adjusts for the effects of the Big5 social roles and another set that adjusts for the effects of both the Big5 social roles and MLDA. To produce the measures of binge drinking that adjust for historical variation in the Big5 social roles we followed a three-step process separately for each gender. First, starting with the first wave, we centered each of the age-varying Big5 social roles on its wave-specific across-cohort mean (e.g., for females, we subtracted observed values of parental

status at Wave 1 from the female parental status across-cohort Wave 1 mean, which was .02 or 2%) and then we repeated this step for Waves 2 to 5. Second, using SAS 9.3 (SAS Institute Inc., 2011), we regressed a given wave's binge drinking measure on that wave's mean-centered social roles (e.g., we regressed male Wave 2 binge drinking on the following male mean-centered variables: Wave 2 parental status, Wave 2 marital status, Wave 2 full-time 4-year college status, Wave 2 residential status, and Wave 2 full-time employment) and outputted the predicted values. Third, to return each adjusted estimate to the original metric, we added together the predicted value and the intercept term from the regression that generated the predicted value (e.g., if a predicted value was $-.342$ and the intercept was 2.00 , then we added the two estimates together to reach 1.668). While incorporating MLDA in addition to the Big5 social roles, we followed the same three-step process to produce measures of binge drinking that adjust for historical variation in *both* Big5 social roles and MLDA. We used this three-step process for two reasons: (1) it effectively holds the rate of the Big-5 social roles, and when applicable MLDA, constant across cohort, while still allowing for the fact that, as our preliminary analyses indicated, historical variation in the Big-5 social roles and MLDA varies by age¹, and (2) it enabled us to conduct multi-level modeling while also holding variation in the Big5 social roles, and when applicable MLDA, constant across cohort².

Historical variation in age 18–26 binge drinking (Aim 3a)—Presented in Table 1 are the estimates for the linear, quadratic, and cubic effects of cohort year obtained when using the measures of binge drinking adjusted for historical variation in (a) the Big5 social roles (labeled as “Big5-Adjusted”) and (b) both the Big5 social roles and MLDA (labeled as “Big5/MLDA-Adjusted”) ³. To identify the effect of historical variation in Big5 social roles we calculated the difference between the Big5-Adjusted estimate and the Unadjusted estimate [(Big5-Adjusted) – (Unadjusted)] and then divided the difference by their pooled standard errors to obtain a z-score (B versus U, Table 1). To identify the effect of historical variation in MLDA we calculated the difference between the Big5/MLDA-Adjusted estimate and the Big5-Adjusted estimate [(Big5/MLDA-Adjusted) – (Big5-Adjusted)] and then divided the difference by their pooled standard errors to obtain a z-score (BM versus B, Table 1).

¹The procedure holds the rate of chronosystem factors (i.e., Big5 social roles and MLDA) constant across cohort because it produces the predicted value of binge drinking under the condition that all chronosystem factors match the across-cohort average (i.e., at each wave the procedure produces the predicted value of binge drinking under the condition that all chronosystem factors are equal to zero, and, by design, for each chronosystem factor the value of zero is the across-cohort average for that wave). In turn, because this procedure holds the rates of all chronosystem factors constant across all individuals, it necessarily also holds all chronosystem factors constant across all cohorts. This procedure allows for the fact that historical variation in chronosystem factors varies by age because all age-varying chronosystem factors are mean-centered on their wave-specific across-cohort means.

²Within multi-level modeling, in order for the model to be properly identified, the total number of model parameters cannot exceed the number of clusters (Muthén & Muthén, 1998–2009). For our purposes, the number of clusters is the number of cohorts, which is 28. Because our three-step approach added no additional parameters to our growth models, it enabled us to conduct multi-level modeling while also holding historical variation in chronosystem factors constant. This is in contrast to a more traditional approach (i.e., including the mean-centered chronosystem measures as exogenous predictors in a latent growth model), because including the mean-centered chronosystem factors as predictors in a latent growth model would lead to a total number of model parameters far exceeding the number of cohorts.

³The pattern of results, in terms of the unique and combined effects of Big5 and MLDA, did not vary depending on the order of the step-wise sequence (i.e., removing Big5 effects first and then removing both Big5 and MLDA effects, as we did here, versus removing MLDA effects first and then removing both MLDA and Big5 effects).

After adjusting for historical variation in the Big-5 social roles, the positive linear effect of cohort year on age 18–22 growth was significantly reduced for males (unadjusted = .140, Big5-Adjusted = .109) and females (unadjusted = .150; Big5-Adjusted = .092). In turn, this reduction in the pace of acceleration across ages 18–22 resulted in historical trends at ages 22 and 26 shifting downwards. For males, decreases in age 22 binge drinking across cohort were more pronounced (Unadjusted: $-.023$, Big5-Adjusted = $-.046$), while increases in age 26 binge drinking across cohort were less pronounced (Unadjusted = .042, Big-5-Adjusted = .019). For females, age 22 binge drinking no longer increased across cohort year (Unadjusted = .042; Big5-Adjusted = $-.008$), while increases in age 26 binge drinking across cohort were less pronounced (Unadjusted = .084, Big-5-Adjusted = .051). Thus, after controlling for historical variation in the Big5 social roles, the historical acceleration in age 18–22 growth in binge drinking was reduced, although still significant, for both genders, which in turn altered age 22 and age 26 trends.

After also adjusting for historical variation in MLDA, the negative linear effect of cohort year on age 18 binge drinking growth was reduced for both males (Big5-Adjusted = $-.145$, Big5/MLDA-Adjusted = $-.098$) and females (Big5-Adjusted = $-.099$, Big5/MLDA-Adjusted = $-.074$). For males only, the positive linear effect of cohort year on age 18–22 growth was further reduced after adjusting for historical variation in both the Big5 social roles and MLDA (Big5-Adjusted = .109, Big5/MLDA-Adjusted = .076). Thus, independent of historical variation in Big5 social roles, after controlling for historical decreases in MLDA, historical decreases in age 18 binge drinking was reduced, although still significant, for both genders, and historical increases in age 18–22 growth was further reduced for males, although again still significant. Finally, in only one instance was the quadratic effect of cohort year altered – the male estimate for age 18 intercept was reduced to non-significance (.004).

To summarize, in terms of linear trends, historical variation in Big5 social roles was linked to historical increases in the age 18–22 growth rate, whereas historical variation in MLDA was linked to historical decreases in age 18 level (although it was also linked to historical increases in the male age 18–22 growth rate). After holding both Big5 social roles and MLDA constant across cohort, historical decreases in age 18 level, historical increases in age 18–22 growth, and historical increases in age 26 level were all noticeably reduced, though not altogether eliminated for both genders. Historical variation in Big5 social roles and MLDA did not account for historical variation in age 22–26 growth. Moreover, aside from historical trends in age 22 level for females, all historical trends remained significant after controlling for Big5 social roles and MLDA. Consequently, in most of the instances that historical variation in Big5 social roles and MLDA accounted for linear historical variation in binge drinking, it did not account for all of it.

Growth moving in tandem historically (Aim 3b)—As was found when using the unadjusted measures of binge drinking, the association between age 18–22 growth and age 22–26 growth was positive at the cohort-level when using both the Big5-Adjusted measures of binge drinking (males: .835, $p = .002$; females: .917, $p < .001$) and the Big5/MLDA-Adjusted measures of binge drinking (males: .684, $p = .008$; females: .946, $p < .001$). For both genders, these findings indicate that independent of historical variation in the Big5

social roles and MLDA, age 18–22 binge drinking growth and age 22–26 binge drinking growth moved in tandem historically.

Discussion

Here we portray the importance of taking an historical perspective on alcohol use etiology, showing that the normative, developmental course of substance use has shifted in very important ways over the past three decades. Consistent with the overall lengthening of the pathway from adolescence to adulthood, we found for both genders that the escalation of binge drinking, a stronghold of this time of life, has shifted forward, starting later and ending later in young people's lives. We found that legal and cultural shifts accounted for some of this change, with the MLDA laws accounting for a portion of the decline in age 18 level (as well as a portion of the acceleration in age 18–22 growth for males) and delays in social role acquisition accounting for a portion of the acceleration in age 18–22 growth. We also found that historical variation in the age 18–22 and age 22–26 growth rates was strongly and positively connected, suggesting that common mechanism(s) underlie historical variation of both growth rates as well as historical-developmental linkages between these two age periods – i.e., that the two periods experience historical change in similar ways. However, our findings also suggest that the factors examined (Big5 social roles and MLDA) here do not fully comprise the common mechanism(s). Finally, although levels and growth of young adult binge drinking are known to vary across gender (Chen & Jacobson, 2012; Needham, 2007), we found that, by and large, historical variation in young binge drinking does not vary across gender. Put another way, when it comes to young adult binge drinking, the effects of gender do not appear to be moderated by historical time (and vice-versa). Indeed, apart from the fact that changes in MLDA laws were associated with historical increases in the age 18–22 growth rate for males only, historical variation in young adult binge drinking, in terms of both patterns and correlates, was largely consistent across gender.

A Historical Shift in the Developmental Progression of Binge Drinking

Consistent with the notion that the transition to adulthood is now more protracted than it used to be (Arnett, 2000; Settersten & Ray, 2010), our findings show for both genders a historical shift in the normative, developmental progression of binge drinking toward a later start, faster increase, and slower decrease across the ages of 18–26. Specifically, relative to past cohorts, recent cohorts both enter the age 18–26 age band engaging in *lower* levels and exit the age 18–26 age band engaging in *higher* levels of binge drinking. The reason for this reversal is that, relative to past cohorts, binge drinking among recent cohorts accelerates more quickly across ages 18–22 and decelerates more slowly across ages 22–26.

Although these historical trends were evident across the entire historical span examined here (as evidenced by the linear effects of cohort year), across this historical span the pace of the trends varied somewhat (as evidenced by the non-linear effects of cohort year). For example, across all cohorts (i.e., the 1976–2003 cohorts) age 18 level declined for both genders (20% for males and 10% for females); however, declines were most evident across the 1976 and 1993 cohorts, as age 18 levels remained relatively flat across the 1993 and 2003 cohorts. In

contrast to age 18 trends, all other historical trends were more evident among recent cohorts. Across all cohorts the age 18–22 growth rate increased around 250% for males and switched from nearly zero to sharply positive for females; however, across the 1983 (ages 18–22 across 1983–87) and 2003 (ages 18–22 across 2003–07) cohorts, it increased around 450% for males and for females reversed direction from negative to sharply positive. Likewise, although for both genders the age 22–26 growth rate slowed (became less negative) across all cohorts (47% decline for males, 25% decline for females), across the 1983 (ages 22–26 across 1987–1991) and 2003 (ages 22–26 across 2007–2011) cohorts it slowed more markedly (65% decline for males, 55% decline for females). Similarly, although age 26 level increased across all cohorts for both genders (6% for males and 12% for females), it increased the most across the 1988 and 2003 cohorts (16% for males and 20% for females), which corresponds to age 26 binge drinking across 1996 and 2011.

The fact that age 26 binge drinking is higher among today's young adults may put them at elevated risk for alcohol-related problems. By the age of 26 most young adults, including today's young adults, have already transitioned into multiple adult roles (Maggs et al., 2012). Consequently, today's young adults, on account of their elevated binge drinking at age 26, are potentially at greater risk for the host of negative mental, physical, and social outcomes associated with binge drinking during adulthood (Corrao, Bagnardi, Zambon & Arico, 1999; Rehm & Monteiro, 2003; Sturm, 2002). Moreover, it is important to monitor these trends among future cohorts. After all, to the extent that these trends continue among future cohorts, future young adults will be launched into adulthood engaging in even higher levels of binge drinking. As we discuss in the next section, while historical variation in Big5 social roles and MLDA helped explain this historical shift in binge drinking trajectories, they largely only helped to explain linear trends in binge drinking trajectories, and only across the age 18–22 portion of the 18–26 age band.

Historical trends in Big5 and MLDA are linked to linear trends in age 18–22 binge drinking

In terms of linear trends, historical increases in the age 18–22 binge drinking growth rate were diminished after controlling for the social role changes – that is, relative to the past, today's young adults, are more likely to hold social roles associated with more binge drinking (going to a 4-year college full-time) and are more likely to delay the transition to social roles associated with less binge drinking (working full-time, getting married, having children, and living independently). Indeed, after controlling for historical variation in Big5 social roles, the positive linear effect of cohort year on the age 18–22 binge drinking growth rate was reduced by around 25% for males and 40% for females. These controls reduced the pace of age 18–22 acceleration which in turn led to reductions in the pace of linear increases in age 26 level (55% reduction for males, 40% reduction for females). Thus, consistent with an ecological model of human development (Bronfenbrenner, 1994), here we identified a range – from proximal to distal - of contextual influences on binge drinking. As demonstrated by existing research (Bachman et al., 1997; Galea, Nandi, & Vlahov, 2004), proximal contexts (e.g., school, family, work) influence young adult binge drinking, while, as demonstrated here, distal contexts (i.e., the chronosystem as represented here by broad

cultural changes regarding the transition to adulthood) inform which proximal contexts young adults are embedded in, and thereby influence young adult binge drinking.

Our findings also build on existing research examining the impact of changes in MLDA on young adult alcohol use, another aspect of the chronosystem. Like existing research (O'Malley & Wagenaar, 1991; Wagenaar & Toomey, 2002), we found that laws leading to increases in MLDA across the country were associated with historical declines in age 18 binge drinking for both genders, with declines proving more pronounced for males (O'Malley & Wagenaar, 1991). Specifically, controlling for historical variation in MLDA reduced historical declines in age 18 binge drinking by around 35% for males and 25% for females. Additionally, for both genders the positive quadratic effect of cohort year on age 18 binge drinking was no longer significant once MLDA laws were controlled, thus linking MLDA laws that were implemented in the mid-1980s with the sharp decline in age 18 binge drinking across the mid-1980s and early 1990s. These findings also suggest that the effects of historical increases in MLDA on age 18 binge drinking stabilized during the early 1990s – the historical point at which sharp declines in age 18 binge drinking abated. Unlike existing research, which focuses on point estimates of alcohol use, we also found that changes in MLDA were associated with changes in the trajectories of young adult binge drinking, but for males only, accounting for an additional 20% (over and above the 25% accounted for by Big5 social roles) in the male age 18–22 binge drinking growth rate. Thus, for males, although increasing MLDA lowered age 18 levels of binge drinking, an unintended correlate, if not result, of this policy change appears to be a sharper acceleration in binge drinking across the ages of 18 to 22. The fact that changes in MLDA are linked to a sharper decline in male age 18 binge drinking may help explain why changes in MLDA were only associated with a sharper acceleration in male age 18–22 binge drinking. That is, for females the relatively muted effect of changes in MLDA on age 18 level may, in turn, dampen any potential effect of changes in MLDA on subsequent (i.e., age 18–22) binge drinking.

Finally, our findings expand on those of Jager et al. (2013), who found that historical variation in social roles had only a modest impact on historical variation in age 18–22 binge drinking trajectories. Importantly, because Jager et al.'s primary focus was whether certain social roles moderate historical variation in binge drinking trajectories, they, unlike the present study, (a) focused on only a subset of the Big5 social roles and only across ages 18 to 22, (b) did not account for the fact that historical variation in social roles varies by age, and (c) did not attempt to disentangle the effects of MLDA from Big5 social roles. Thus, relative to Jager et al, the present study applies a more rigorous, long-term, and nuanced examination of historical variation in Big5 social roles and MLDA and, thereby, provides a more detailed and complete account of their effects.

Historical shift in binge drinking: Much remains to be explained

Although the chronosystem components examined here help explain historical variation in age 18 level and age 18–22 growth, and, by connection, age 26 level, they primarily only help explain linear trends, and even in these cases they only help explain a portion of these trends (i.e., although reduced, these linear trends remained significant after controlling for

Big5 social roles and MLDA). Meanwhile, the chronosystem components examined here largely failed to explain non-linear historical trends in age 18–22 growth and age 26 level, linear and non-linear trends in age 22–26 growth, and the cohort-level correlation between age 18–22 and age 22–26 growth.

Regarding historical trends in age 22–26 growth, because across-cohort variation in the chronosystem components examined here proved to be substantially higher among the age 18–22 portion of the 18–26 age band (e.g., historical variation in social roles and MLDA was far higher at age 19 than at age 26), controlling for it did not account for (a) much variance in age 22–26 growth or (b) much *common* variance between the age 18–22 and age 22–26 age bands. As a result, controlling for Big5 social roles and MLDA did not lead to reductions in historical trends for the age 22–26 growth rate or attenuate the cohort-level correlation between the age 18–22 and age 22–26 growth rates. Thus, although the strong, positive cohort-level correlation between the age 18–22 and age 22–26 growth rates suggests a historical-developmental linkage and that many of the same mechanism(s) underlie historical variation in each growth rate, our findings suggest that the chronosystem components examined here do not fully capture those common mechanisms.

The likely reason historical variation in Big5 social roles was primarily linked to linear historical trends in binge drinking is that across-cohort variation in the Big5 social roles was itself quite linear. As a result, controlling for it primarily impacted linear trends in binge drinking. In contrast, historical variation in MLDA, which was linked to both linear and non-linear trends in age 18 level, was itself non-linear (historical variation in MLDA was flat until the mid-80s). Left unexplained, however, are the non-linear trends in the age 18–22 and 22–26 growth rates (i.e., why both the acceleration in the 18–22 growth rate and deceleration in the 22–26 growth rate was more evident among recent cohorts) and age 26 level (i.e. why the age 26 level increased at a faster pace among recent cohorts).

Therefore, beyond the chronosystem components examined here, future research should focus on other proximal contextual factors that are known to be associated with binge drinking and have potentially varied historically (in both linear and non-linear fashions). Although by no means an exhaustive list, the following are known to be associated with binge drinking: the perceived risk and availability of alcohol, parental attitudes as well as peer norms regarding alcohol use, and alcohol abuse prevention efforts, including school-based programs, media campaigns, etc. (Patrick, Schulenberg, Martz, Maggs, O'Malley, & Johnston, 2013; Tobler et al., 2000; Wakefield, Loken, & Hornik, 2010; Wood, Read, Mitchell, & Brand, 2004). It is possible that these contextual factors, as well as others, varied historically, and thereby account for (a) historical variation, both linear and non-linear, in binge drinking left unexplained by the chronosystem components examined here and (b) operate as mechanism(s) that underlie continuous historical trends across the 18–22 and 22–26 age bands.

Strengths and limitations

An important strength of this study derives from the use of consistent measures in national multicohort panel data spanning the transition to adulthood. We offer a view of binge

drinking as a moving target developmentally and historically that is typically unavailable in longitudinal data sets with one or few cohorts. This study also has important limitations. First, this study relies on self-report data, introducing the possibility of reporting bias. Second, given the way social roles were measured and operationalized, it is possible that some variation in social roles went undetected. For example, to reduce complexity and provide consistency, all social roles were dichotomized. Additionally, although unavoidable given the structure of the data, it is possible that social roles changed more than once between the study's two-year assessments. Third, although beyond the scope of this study, given the differences in alcohol consumption across both race/ethnicity and socio-economic status (Bachman et al., 1997), it is possible that the historical trends found here vary across these demographic groups. Future research should explore this possibility. Fourth, given the correlational nature of this study, whether the relation between historical variation in chronosystem factors and historical variation in binge drinking is causal is unclear, and if causal, the direction of effect is unknown. Fifth, the statistical approach we used to adjust for the effects of historical variation in Big5 social roles and MLDA on binge drinking did not allow us to isolate the unique effects of a given Big5 social role. Because testing the individual effects of each Big5 social role was not feasible in this study, we leave this topic to future research.

Conclusions and Future Directions

The present study, along with Jager et al. (2013), indicates that historical time or the chronosystem is an important source of systematic individual differences in young adult trajectories of binge drinking. By focusing on the dimension of historical time, we were able to both isolate important individual differences in the development of binge drinking and identify mechanisms (i.e., historical changes in Big5 social roles and MLDA) that partially underlie those differences. Consequently, although existing context-focused substance use research yields important insights about the etiology of substance use, because it largely ignores historical time while focusing on more proximal contexts (e.g., families, peers, schools), there is reason to believe it provides an incomplete account, and therefore limits our understanding of the role that social context plays in the development of binge drinking. Importantly, historical variation in the developmental landscape of the transition to adulthood may have altered normative developmental progression of other risky behaviors beyond binge drinking, which raises many possibilities for future research. More generally, our approach and findings highlight the interplay of etiology – typically the focus of developmental science – and epidemiology or demography. Indeed, interdisciplinary efforts require integrating perspectives as well as levels of analysis, and our study represents a concrete example that can help advance the science in terms of content and approach.

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Figure 1.
Conceptual models for aims 1, 2, 3a, and 3b

Figure 2.

Baseline and cohort year piece-wise growth models of binge drinking. Black portion of the model represents the *Baseline Piece-wise Growth Model* and the black and gray portions of the model combined represent the *Cohort Year Piece-wise Growth Model*. When necessary, factor loadings were linearly transformed to render ages 21/22 and 25/26 the intercepts; Doing so had no impact on model fit or the estimates of age 18–21/22 and age 21/22–25/26 growth.

Figure 3.
Historical trends in binge drinking (last two weeks), by gender and cohort year

Figure 4.
Binge drinking age 18–26 trajectory, by historical group and gender

Table 1

Linear, quadratic, and cubic effects of cohort year on binge drinking, unadjusted and adjusted, by gender

Factor means	Standardized effects of cohort year									Differences in effects of cohort year (z-scores)								
	Unadjusted (U)			Big5-Adjusted (B)			Big5/MLDA-Adjusted (BM)			U versus B			B versus BM					
	L	Q	C	L	Q	C	L	Q	C	L	Q	C	L	Q	C	L	Q	C
Males																		
Age 18 intercept	1.998**	-.144**	.023**	.029**	-.145**	.023**	.030**	-.098**	.004	.030**	.09	.01	-.17	-.532**	2.77**	.10		
Age 18–22 growth	.115**	.148**	.031*	-.016*	.109**	.034*	-.018*	.076**	.039*	-.016	2.79**	-.21	.32	3.76**	-.67	-.60		
Age 22 intercept	2.229**	-.023**	.031**	.006	-.046**	.034**	.005	-.036**	.026**	.008	2.68**	-.22	.16	-.125	1.00	-.40		
Age 22–26 growth	-.111**	.088**	.027*	-.018*	.092**	.024*	-.020*	.083**	.029*	-.018	.02	.21	.28	.59	-.39	-.19		
Age 26 intercept	2.006**	.042**	.045**	-.014	.019*	.046**	-.019*	.024*	.041**	-.013	2.60**	.02	.47	-.57	.54	-.62		
Females																		
Age 18 intercept	1.526**	-.101**	.019**	.034**	-.099**	.019**	.035**	-.074**	.011	.035**	-.20	-.01	-.07	-.214*	1.12	.00		
Age 18–22 growth	.049**	.154**	.026*	-.019*	.092**	.030*	-.020*	.086**	.031*	-.017	5.47**	-.24	.11	.56	.01	-.27		
Age 22 intercept	1.625**	.042**	.040**	.004	-.008	.044**	.004	.002	.037**	.007	6.53**	-.28	.07	-.124	.94	-.33		
Age 22–26 growth	-.100**	.056**	.023*	-.017	.069**	.021	-.019	.057**	.018	-.020	-.71	.38	-.39	.29	-.71	-.12		
Age 26 intercept	1.424**	.084**	.060**	-.019*	.051**	.062**	-.024**	.053**	.060**	-.022*	4.00**	.17	.44	-.16	.19	-.22		

Note.

* $p < .05$;

** $p < .01$.

L - linear; Q = quadratic; C = Cubic. MLDA = minimum legal drinking age. "Big5-Adjusted" estimates control for historical (cohort) variation in marital status, parental status, residential status, full-time 4-year college attendance, and full-time employment. "Big5/MLDA-Adjusted" estimates control for historical cohort variation in MLDA as well as marital status, parental status, residential status, full-time college attendance, and full-time employment.