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Deconstructing the Age-Prevalence Curve of Alcohol Dependence: Why “Maturing Out” is only a Small Piece of the Puzzle

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Abstract

Epidemiological studies have consistently demonstrated that heavy alcohol use and alcohol dependence (AD), tend to increase in adolescence and emerging adulthood and then show a large decline in the late 20s, a phenomenon called “maturing out”. This decline has been explained as an effect of “role incompatibility” in which involvement in new roles and activities interferes with a heavy drinking lifestyle. However, maturing out has been conceived mostly as a decrease in offset, with little attention paid to reductions in new onset or recurrence across decades of life. Moreover, although role incompatibility processes have been studied with young samples, little is known about the effect of life transitions (e.g., marriage, parenthood, changes in employment status) on AD later in life and whether similar effects are observed. Using longitudinal data from the National Epidemiologic Survey on Alcohol and Related Conditions (NESARC), a nationally representative epidemiologic survey, we examined the patterns of stability and change in AD across the lifespan and the differential effect of life transitions on AD across different age strata. Results showed that persistence of alcohol dependence tended to increase with age, although not dramatically, and that onset and recurrence tended to decrease with age. Moreover, the effects of

life transitions on the course of AD varied across the lifespan and were different for men and women. These results indicate that life transitions differentially affect the patterns of stability and change in younger versus older people, have a different impact for men and women, and highlight the need to consider the unique aspects of each stage of adult development on the course of AD.

Keywords

maturing out; alcohol dependence; life transitions; course

Perhaps the most significant finding in the epidemiology of alcohol use and of alcohol dependence (AD) is that the highest levels of excessive consumption and alcohol-related problems and dependence symptoms occur in the early twenties and decline rapidly in the third decade of life. In a meta-analysis of 27 longitudinal studies conducted in 10 different countries, Johnstone, Leino, Ager, Ferrer, & Fillmore (1996) found a rapid increase in drinking before 20 years, followed by a significant decrease and a relative stabilization after age 30. Similarly, Kerr, Fillmore, & Bostrom (2002) analyzed three national longitudinal studies, examining the patterns of stability or change associated with different subgroups and methodologies. They found that drinking patterns are more stable for adult rather than youthful samples, for those who are abstainers or moderate drinkers compared to heavy drinkers, and over short follow-up intervals. Recent data from the National Epidemiologic Survey on Alcohol and Related Conditions (NESARC) show differences in alcohol use patterns by age cohort as measured by frequency and quantity per drinking occasion (Chan, Neighbors, Gilson, Larimer, & Marlatt, 2007). After an increase in frequency and quantity that peaks around age 21 (the legal drinking age), the decrease in quantity is linear, whereas the general decline in frequency is interrupted by a gradual increase between ages 35 and 54. Moreover, an interaction between age and sex was found for quantity of use, with men showing a sharper decrease than women (Chan et al., 2007).

In contrast to data on alcohol consumption, data on course of AD are scarce. Grant (1997) reported cross-sectional data from the National Longitudinal Alcohol Epidemiologic Survey (NLAES) showing that younger participants (age 18-24) had a greater prevalence of AD than older subjects. Similarly, Hasin, Stinson, Ogburn, & Grant (2007) found that the prevalence of AD in the last twelve months decreased sharply during the third decade of life in cross-sectional analyses. Although these findings are limited to cross-sectional data, their consistency with longitudinal data on alcohol consumption suggests that a similar phenomenon underlies both decreases in drinking measures and AD.

In summary, it is reasonably well established that in the United States the consumption of alcohol and the prevalence of AD, tend to peak in early adolescence and young adulthood and then decrease abruptly in the third decade of life. This phenomenon has been called *maturing out* (Winick, 1962; see also, Dawson, Grant, Stinson, & Chou, 2006; Jochman & Fromme, 2009; Littlefield, Sher & Wood, 2009; Miller-Tutzauer, Leonard & Windle, 1991). The term “maturing out” is mostly used as a descriptive term although it suggests that processes involved in maturation might explain the decrease in prevalence. Indeed, it has been proposed that maturing out is the result of people entering into adult roles (e.g., getting married, becoming parent, entering the labor market) that are incompatible with drinking (Bachman, Wadsworth, O’Malley, Johnston, & Schulenberg, 1997; Bogart, Collins, Ellickson, Martino, & Klein, 2005; Leonard & Rothbard, 1999; Miller-Tutzauer et al., 1991; Yamaguchi & Kandel, 1985). We note, however, that it appears that normative decreases in traits related to negative emotionality and impulsivity also may contribute to “maturing out” of alcohol problems (Littlefield et al., 2009, 2010a). While most of the literature on “maturing out” has focused on alcohol use, similar effects have been documented for AD

(Chilcoat & Breslau, 1996; Gotham, Sher, & Wood, 2003). Thus, both external constraints (i.e., role incompatibilities) and increasing psychosocial maturity as indexed by decreases in emotionality and increases in conscientiousness appear to exert inhibiting effects on drinking and alcohol-related problems.

The Relative Importance of Onset, Recurrence, and Persistence on Maturing Out

The focus on “maturing out” has created a lens whereby the dramatic decrease in AD prevalence is assumed to reflect differences in persistence (that is, lower rates of persistence during young adulthood than later in life; see Dawson et al., 2006; Jochman & Fromme, 2009) over the lifespan. Putting aside the issue of period and cohort effects (e.g., Kerr, Greenfield, Bond, Ye, & Rehm, 2004; Karlamangla, Zhou, Reuben, Greendale, & Moore, 2006) and differential mortality, such age-graded changes in prevalence could result from multiple components and not just persistence. Specifically, such a decrease could result from fewer new onsets, and less recurrence of a past AD, as well as through increased persistence¹. In support of this, Hasin et al. (2007), using NESARC data reported that the onset of dependence steadily increased from early adolescence until age 19, after which it decreased. In a similar vein, Li, Hewitt, and Grant (2004) reported NESARC data showing that the likelihood of first meeting criteria for DSM-IV AD peaks around ages 18-19. Similarly, Kessler et al. (2005) reported that the median age of onset in the National Comorbidity Survey Replication (NCS-R) was 23 for AD. In addition, Dawson, Goldstein, & Grant (2007) reported that 5.1% of participants in full remission of dependence at Wave 1 of NESARC had a recurrence of dependence between Waves 1 and 2, and that age was negatively associated with recurrence. With regard to persistence, Grant (1997) showed that in the NLAES study the youngest cohort had the greatest prevalence of past-12-month alcohol dependence among the subsample of participants with dependence prior to the past year (i.e., the greatest prevalence of persistence) and Dawson et al. (2005), using NESARC data, reported that abstinent recovery (but not non-abstinent recovery) increased with age. However, Kalaydjian et al. (2009) reported that rates of remission from dependence were higher among younger individuals (i.e., lower rates of persistence) in the NCS-R sample. Thus in terms of understanding the dramatic decline in prevalence of AD during the third decade of life, we need to consider the relative importance of multiple components that determine developmental changes in the overall prevalence of AD. Additionally, assuming that these other components are relevant for understanding developmental changes in prevalence, it raises the important question of whether role incompatibility and other processes thought to explain “maturing out” also relate to onset and recurrence processes.

Does Role Incompatibility Occur over the Entire Lifespan?

Although role incompatibility is assumed to explain much of the decreases in drinking and AD that occur during young adulthood, it is not clear whether it may also be linked to changes in drinking in older individuals as well. In a meta-analysis of twelve longitudinal studies, Temple et al. (1991) found that never marrying was significantly related to increased alcohol consumption in younger (18-39 years) but not older men. Also, chronic unemployment was associated with a decrease in alcohol consumption in older men and younger women. Finally, getting married was related to a decrease whereas marital dissolution was related to an increase in alcohol use, but only in younger women. In a longitudinal study conducted in the Netherlands, Neve, Lemmens, & Drop (2000) found that

¹However, as noted above, there is a distinction between maturing out of alcohol use and maturing out of alcohol dependence. Thus, it is possible that changes in persistence of heavy drinking are associated with changes in persistence, onset, and recurrence of alcohol dependence.

a transition into a more structured everyday life –as reflected by becoming married, parent and employed- was negatively related to level of drinking, frequency of heavy drinking and drinking problems only in younger (under the age of 44) participants. Interestingly, in older participants (age 45 and older) the association was not significant for level of drinking but positive for frequency of heavy drinking and drinking problems. These findings suggest that the relation between life transitions and alcohol involvement varies as a function of life stage.

Little is known about the effect of life transitions on the course of AD. We were able to identify just one study, using longitudinal data over 3.5 years from members of a health maintenance organization, aged 21-30 years, showing that the rates of onset of alcohol abuse or dependence, as defined by the DSM-III-R (American Psychiatric Association, 1987) were higher among participants who were stably single during the follow-up interval, and that the rates of persistence in disorder were lower among participants who became married between the two waves (Chilcoat & Breslau, 1996). However, this study was not informative with respect to life course issues since age was included only as a covariate, and interactions of life transitions with age were not computed. Moreover, the focus was on onset and persistence of disorder, while recurrence was not examined. The current study is intended to fill this major gap in the research literature by assessing the effect of life transitions on all components of overall prevalence decrease (i.e., onset, persistence, and recurrence; see Method section) in AD (as defined by the DSM-IV; American Psychiatric Association, 1994), and across a wide range of age groups.

The Current Study

Drawing upon data from the NESARC, the current study aimed to characterize age-related changes in the prevalence of AD. In order to understand the relative contribution of onset, recurrence, and persistence components to this phenomenon of changing prevalence over the life course, we decompose past year prevalence into fractions associated with each of these components. We then examine the moderation of onset, persistence and recurrence by time-varying factors (i.e., life transitions) as a function of adult development in order to examine: (1) whether these vary by age, and, (2) whether they apply to onset, persistence, and recurrence components. These types of analyses are foundational for understanding the natural course of AD in a developmental context and bear on the question of whether there are distinct subtypes of AD defined by developmental course (e.g., Zucker, 1987, 1994).

Method

Participants

Secondary data analysis was performed using the two waves of the National Epidemiologic Survey on Alcohol and Related Conditions (NESARC; Grant, Moore, & Kaplan, 2003; Grant & Kaplan, 2005) which was funded by the National Institute of Alcoholism and Alcohol Abuse (NIAAA) and conducted by the U.S. Census Bureau. The data are a representative sample of civilians 18 years and older of the United States population. The survey oversampled Blacks and Hispanics and young adults between 18 and 24 years. An initial wave of face-to-face interviews was conducted during 2001-2002 and includes 43,093 respondents. A follow-up second wave of face-to-face interviews was performed during 2004-2005 and contains 34,653 of the same respondents. The dataset is weighted to approximate the general population of the United States. The sample at Wave 2 had a mean age of 48 ($SE=0.17$) years, and was composed of 52% ($SE=0.34$) of women, 71% White/non-Hispanic, 11% African-American, 12% Hispanic, 4% Asian, and 2% American Indian.

Measures

Alcohol Dependence—Alcohol dependence (AD) was assessed by the Alcohol Use Disorders and Associated Disabilities Interview Schedule-DSM-IV Version (AUDADIS-IV; Grant, Dawson, & Hasin, 2001). The AUDADIS-IV follows DSM-IV criteria for diagnosis of AD (at least three of seven dependence criteria). AD was assessed in both past 12-month, and prior to past 12 month time frames. The same criteria had to be met in order to be classified as prior-to-past-year dependence, provided that symptoms clustered within the same year. A number of studies have provided evidence of the reliability of the AD diagnoses as measured by the AUDADIS-IV (e.g., Grant, Harford, Dawson, Chou & Pickering, 1995; Grant et al., 2003).

In order to characterize the composition of past-12-month AD prevalence on the basis of prior course, three groups were created from participants who had a past-12-month AD diagnosis at Wave 2 ($n = 1,433$). Individuals with no lifetime AD diagnosis at Wave 1 constituted the New Onset group ($n = 767$); those with a past-twelve-months AD diagnosis at Wave 1 constituted the Persistence group ($n = 422$); and participants who had a lifetime AD diagnosis but no diagnosis in the past 12 months at Wave 1 constituted the Recurrence group ($n = 244$). We note that our operational definition of Persistence includes individuals with both continuous AD and episodic AD. That is, an individual could have a period of remission in the follow-up period that did not extend to the year just prior to follow-up.

While the procedures above describe how the various fractions (e.g., new onset cases, persistent cases, and recurrent cases) of overall Wave 2 prevalence were estimated, it is important to highlight the fact that knowing the relative contribution of these outcomes to Wave 2 prevalence tells us little about what the “rates” of each of these are. That is because the number of individuals “at risk” for each of these statuses is different than the total number of participants at Wave 2. For instance, only those individuals with no lifetime history of AD are at risk for new onset. Similarly, only those individuals with a baseline past-12-month diagnosis are “at risk” for persistence. Thus we could get, say, a prevalence of Wave 2 persistence AD of 20% of participants via a number of different rates of persistence: a Wave 1 prevalence of 40% and a persistence rate of .5, a Wave 1 prevalence rate of 20% and a persistence rate of 1.0, etc. Thus, in order to characterize developmental changes in each of these processes, we also estimated rates of onset, recurrence, and persistence based on the number of individuals at risk for that outcome at Wave 1.

Life Transitions—Direct measures of change in marriage and parenthood status between waves were obtained at Wave 2, from variables indicating change in marital status, and having any children or any adopted/foster/step children since the last interview. Change in marital status was defined broadly, including participants who reported starting or stopping living with someone as if married². With regard to employment status, measures of change were constructed using data from both waves. Specifically, employment status was coded as “became employed” if an individual reported being unemployed or retired at Wave 1 and being employed at Wave 2; employment status was coded as “became unemployed” if the participant reported being employed at Wave 1 and unemployed or retired at Wave 2³; and it was coded as “remained stable” if it remained the same at Wave 2.

In order to determine the predictors of course in AD across the lifetime, three categories of life transitions were considered: (i) change in parenthood status, coded as remained stable or became a parent; (ii) change in marital status, coded as became married, remained stable or

²Additional analyses excluding changes in “living with someone as if married” were conducted, yielding similar results to those reported here.

³Unfortunately, there were not sufficient retirements to study that phenomenon separately from losing a job.

separated/divorced/widowed; and (iii) change in employment status, coded as became unemployed, remained stable or became employed.⁴

Other variables—Sex, age, and ethnicity as measured in Wave 1 were included in the analyses. Age was categorized into seven groups for the analysis of prevalence, and into two groups for the interactions with life transitions (see Results section). Ethnicity was coded as White or any other ethnic group. Age at first drinking (i.e., “age when started drinking, not counting small tastes or sips”) was included only as a descriptive variable to characterize the transition groups.

Analysis

The NESARC utilized a complex survey sampling design. These analyses used SUDAAN in order to adjust for the sampling weights in the calculation of standard errors of parameter estimates (statistical package version 10; Research Triangle Institute, 2004). SUDAAN’s PROC CROSSTAB was used for the estimation of prevalences and PROC REGRESS and PROC RLOGISTIC were used for logistic regressions and for linear regressions (used for the comparison of groups with regard to age and age at first drinking; see Table 2). Weights used at Wave 2 also adjusted for attrition and Wave 1 lifetime psychiatric disorders. SUDAAN uses a Taylor linearization method for the computation of standard errors.

Results

Attrition was assessed through separate logistic regressions conducted for each variable (including demographic variables and AD diagnoses), in order to determine the difference between participants who were retained in Wave 2 and those who were not retained. The results are presented in Table 1. Retained participants were significantly more likely than non-retained participants to be females, older, White or American Indian, and to have a prior-to-past-12-months diagnosis of AD. However, all analyses were also conducted using multiple imputation for missing data at Wave 2, yielding similar results to those reported here (the few exceptions to this are explicitly mentioned below)⁵.

Figure 1 shows a marked decrease in the prevalence of past-12-month AD (as measured at Wave 2) across the lifespan. The values on the Y-axis correspond to the percentage diagnosed at Wave 2 in a given age bracket; the values on the bottom correspond to the number of participants in that age bracket at Wave 2. Note that in order to highlight the third decade of life, we created three age strata (21-23, 24-27, and 28-32) to emphasize distinct phases of this rapidly changing life stage (note the strata were created based on ages reported at Wave 1, plus three years, so the somewhat unusual age groupings presented in the Figure correspond to approximations [given that the interval between waves is not the same for all participants] of ages at follow-up). As shown in Figure 1, the relative contribution of persistence to the AD prevalence at each age remains relatively stable (29% at age 21-23, 27% at age 63+), whereas the relative contribution of new onset decreases (from 66% to 58%) and the relative contribution of recurrence increases (from 5% to 15%).

A slightly different picture emerges when looking at each component separately among their respective populations “at risk” for that outcome. Figure 2 depicts the prevalences of the

⁴Note that our coding includes participants who are married, single or divorced at both waves as “stable” in marital status, and those who are employed or unemployed at both waves as “stable” in employment status. Although this introduces heterogeneity in the stable categories, we could not distinguish these groups due to the sparseness of the data. See discussion section for more comments on this issue.

⁵Note that missing data are not limited to Wave 2 reports of alcohol dependence. Another source of missing data is due to missing life transition information. Thus, the *N*s reported in logistic regressions involving life transitions differ from those reported when describing the AD groups.

three AD groups across the lifespan. For these figures, values on the bottom indicate the number of participants at risk for diagnosis within a given age strata; the Y-axis again corresponds to percentage diagnosed at Wave 2. Separate logistic regressions were conducted for each AD group (that is, for each subpopulation “at risk” for persistence, new onset, and recurrence, as defined at Wave 1) with age group as independent variable, Wave 2 diagnosis status as dependent variable and sex and race as control variable covariates. These analyses showed that persistence (Wald $F = 2.28$, 6 df , $p = .047$) exhibits an increasing trend with age (that is not significant in the imputation analyses), whereas onset (Wald $F = 44.22$, 6 df , $p < .0001$) and recurrence (Wald $F = 6.64$, 6 df , $p < .0001$) decrease significantly with age. Thus, most of the decrease in prevalence of AD across the lifespan appears to be attributable to less onset (i.e., decreasing incidence) and recurrence.

Role Transitions and Stability and Change of AD across Development

Having established that there are significant age effects on the course of prevalence and some of its components, we next analyzed the association between role transitions in the patterns of stability and change of AD across age in adulthood. We also created three referent groups which were not diagnosed at Wave 2, including those who had a past 12-month diagnosis at Wave 1 (offset), those who had a lifetime diagnosis but not in the 12 months prior to Wave 1 (non-recurrent), and those who had no lifetime diagnosis (consistent non-diagnosis). These referent groups provide a useful comparison between participants “at risk” who receive a diagnosis at Wave 2 versus those who do not.

Table 2 presents descriptive statistics for the AD groups and referent groups. Participants who exhibit offset of dependence across waves tend to be younger, whereas those who did not meet criteria for dependence in both waves are older. The consistent non-diagnosis group is also the one that started drinking at an older age, and included the lowest proportion of males, whereas the onset group included less White individuals. With regard to the variables of interest in the analysis of transitional life events presented below, the persistent group included a smaller proportion of married people at both waves, whereas the participants in the offset group are less likely to be parents at both waves. Moreover, the non-recurrent and consistent non-diagnosis groups included the highest percentages of marriage and parenthood at both waves. Finally, the percentage of employed people is smaller in the consistent non-diagnosis group at both waves.

In order to examine the way in which stability and change are differentially influenced by life transitions across the life span, we conducted logistic regression analyses including the interactions between the three life transitions with age. We also examined moderation by sex (with female as the reference group), as there may be gender differences in the associations between role transitions and AD (e.g., a more powerful effect of parenthood among women). The models predicted the likelihood of diagnosis at Wave 2, given no lifetime diagnosis (predicting new onset versus persistent non-diagnosis), a past 12-month diagnosis (predicting persistent versus offset), and prior lifetime but not past 12-month diagnosis (predicting recurrent versus non-recurrent) at Wave 1. For these analyses, age was categorized in two groups (21-37, and 38+ years) in order to have sufficient n in each cell when estimating the interaction terms, and the youngest age group (age 21-37) served as the reference group. Table 3 presents odds ratios and 95% confidence intervals for main effects and interactions in the AD groups. As can be seen, 3-way interactions involving parenthood and change in job were significant in predicting AD persistence (although only the interaction with parenthood was still significant using multiple imputation). The patterns of interactions (shown in Figure 3) suggest that becoming a parent is a protective factor against persistence in AD for older men (OR = 0.02 [CI = 0.00-0.28]; see Figure 3, top panel). Also, older men who got a new job between the two waves were less likely to get a persistent diagnosis of AD (OR = 0.03 [CI = 0.00-0.48]; see Figure 3, bottom panel). Moreover,

becoming a parent was associated with decreased ($OR = 0.39$ [$CI = 0.18-0.83$]) whereas getting a new job was associated with increased ($OR = 2.79$ [$CI = 1.19-6.55$]) rates of new onset regardless of the age or sex of the participants.

Alternative Definitions of Persistence and Recurrence

Our definition of persistence is based on participants continuing to meet full syndromal criteria at Wave 2. Thus, a participant could have one or two dependence symptoms due to heavy drinking (what some have called *diagnostic orphans*; see Pollock & Martin, 1999) at Wave 2 and fail to be classified as persistent. If this is the case, the use of an arbitrary symptom threshold defined by the DSM-IV might lead to misclassification of some participants as non-persistent due to measurement error. To examine the impact of this issue in our results, we reanalyzed the data including any participant who had AD at Wave 1 and at least one symptom of dependence at Wave 2. The prevalence of persistence increased in all age groups as expected, but the overall pattern of changes with age was very similar to the one reported here. However, the decrease in persistence at age 63+ was pronounced enough to make the age effect significant. Also, only the 3-way interaction involving change in job was significant.

In a related note, we decided to adopt a strict “time-sampling” approach to define persistence, as opposed to requiring AD diagnosis during the 3-year interval, based on two potential problems with the latter: i) problems of assessing the temporal parameters of distant measures as evidenced by “negative prevalence” of lifetime ADs (and other conditions) over retest periods as short as one year (e.g., Vandiver & Sher, 1991) and (ii) the fact that different participants could meet criteria for AD during the interval with dissimilar temporal patterns (i.e., diagnosis only at the beginning of the interval, diagnosis only at the end of the interval, continuous diagnosis during the 3 years). That is, although we recognize that requiring ostensible continuous diagnosis is a more stringent criterion for persistence than requiring a past-year diagnosis at follow-up, the former is not without problems. Moreover, this is further compromised by the vicissitudes of retrospection of more than one year. However, in the interest of evaluating alternative definitions of persistence, we reanalyzed the persistence data requiring a continuous diagnosis. The results showed no significant change with age (although the pattern was very similar to the one in Figure 2 for persistence), and only the interaction with parenthood remained significant using this definition of persistence.

Finally, recurrence could be defined in alternative ways and the definitional complexities are similar to those discussed for persistence (i.e., diagnostic orphans: endorsing only one or two symptoms at Wave 1, but still having problems with alcohol use). If this is the case, some participants could be misclassified as “recurrent” when a more accurate description of their course would be “persistence”. To examine the impact of this issue in our results, we reanalyzed the data using a more stringent definition of recurrence, requiring the absence of any dependence symptom in the last 12 months at Wave 1 (and a dependence diagnosis prior to past-12 months). The prevalence of recurrence by age had a very similar pattern to the one reported here. However, the interactions with life transitions could not be estimated due to a small sample size.

Discussion

Rethinking the Dramatic Age-Gradient in the Prevalence Curve of Alcohol Disorders

The results found in this study document, as have others in this (Hasin et al., 2007) and other data sets (e.g., Grant, 1997), that there is a dramatic decrease in the 12-month prevalence of AD in emerging and young adulthood. However, analysis of patterns of stability and change

are not consistent with the notion that this is due primarily to dramatically higher rates of “maturing out” (narrowly understood as offset) or “natural recovery” in the third decade of life as opposed to later periods of adulthood. Although AD does tend to be less persistent in younger adults than older adults (i.e., AD persistence rates of 32.4% and 44.5% in ages 21-23 and ages 43-52, respectively) this difference is only a minor contributor to the notable age gradient in AD prevalence at this stage of life. Moreover, the persistence rate is relatively stable over the third decade of lifetime despite the fact that we are witnessing dramatic decreases in prevalence over this time frame. The rates of new onsets and recurrences drop as individuals age, so that we can view the rapidly decreasing prevalence rates as reflecting primarily decreases in processes generating these phenomena over the life course. This dramatic decrease observed in AD seems to be due primarily to a decrease in the rates of new onset (i.e., incidence), whereas the rates of offset (the opposite of persistence), that according to the role incompatibility explanation should peak before age thirty, are indeed relatively stable although decreasing somewhat. From this perspective, the notion of a “developmentally limited alcoholism” (e.g., Zucker, 1987, 1994) reflects not so much a different *species* of AD but rather the fact that AD is not highly stable at any age but the high hazard rates of emerging adulthood give the superficial appearance that early onset alcohol disorders are less stable than those later in life.

These results with regard to persistence are not consistent with retrospective cross-sectional findings reported by Grant (1997) in the cross-sectional data of the NLAES study, although this discrepancy may stem from differences in the definition of persistence. Grant reported a significant age-related decrease in AD persistence, defined as past-12-month dependence among those with AD prior to the past year, whereas we examined past-12-month dependence at Wave 2 among those who had a past-12-month diagnosis at Wave 1. However, using her definition of persistence, we were able to replicate those results with the Wave 1 NESARC data (results not shown). This implies that different definitions of persistence (prevalence of dependence among people with prior-to-past-12-month dependence versus prevalence of dependence among people with past-12-month dependence in a previous assessment) can lead to conflicting findings with regard to estimates of persistence as a function of age. A potential explanation for this difference is that the definition of persistence used by Grant introduces a difference among ages, such that younger individuals have less time to desist from their alcohol involvement than older individuals. Instead, our definition involves the same time frame regardless of age, so that both younger and older individuals have 3 years to desist. Although there is no single way to look at course that is far more correct than others, in our opinion the use of two waves of assessment to evaluate persistence is potentially more accurate for three related but different reasons: (1) it provides a common follow-up period that provides a clear frame for assessing persistence, (2) it does not invoke the concept of lifetime diagnosis which is known to show poor reliability (e.g., Vandiver & Sher, 1991), and (3) it involves recalling recent information and not a lifetime estimate of a diagnosis that can be subject to a differential recall bias depending on age (although our estimates of new onset and recurrence are subject to the same criticism).

Our results regarding new onset are consistent with previous cross-sectional (Hasin et al., 2007; Kessler et al., 2005) and longitudinal (Crum, Chan, Chen, Storr, & Anthony, 2005; Mattisson, Bogren, Horstmann, & Öjesjö, 2010) reports, showing that the onset of alcohol dependence tends to decrease with age. Although both the estimation procedure and the time frame of our data differ from other longitudinal reports, the results are strikingly similar. Using data from the Baltimore Epidemiologic Catchment Area, Crum et al. (2005) reported that the incidence of DSM-III-R AD during a 12.6 year follow-up decreased with age, from 52.7% at age 18-29 to 4.3% at age 65+. Similarly, Mattisson et al. (2010) reported that the incidence rate of AD decreased between ages 15-29 to 70+ among both males and females

in the periods 1947-1972 and the period 1972-1997 in the Lundby study conducted in Sweden. However, NESARC is the first study that provides longitudinal data on age-related decreases in new onsets at a national level.

The current findings are remarkably consistent with our earlier analyses of the course of bipolar disorders in NESARC (Cicero, Epler, & Sher, 2009). In bipolar disorder (BP), especially if diagnosed broadly, we see a similarly dramatic decrease in the past-12-month prevalence of BP in the third decade of life with rates of offset being roughly comparable over the life course. These findings suggest that the trends we are observing here are very general, perhaps also extending to much of the externalizing spectrum (Krueger et al., 2002; Krueger, Markon, Patrick, Benning, & Kramer, 2007). If this is the case, it suggests that we might want to view the age-prevalence curve as reflecting basic aspects of psychological maturation in the areas of negative affectivity and self-regulation that affect both behavioral maturation (successful attainment of normative roles) and mental health. Indeed, personality traits related to the externalizing spectrum, such as conscientiousness (Roberts, Jackson, Burger, & Trautwein, 2009; Roberts, Walton, & Viechtbauer, 2006), and to psychopathology in general, such as neuroticism (Roberts et al., 2006), show similar age gradients. We have previously shown that changes in alcohol problems are correlated with changes in personality traits associated with self-regulation and negative affectivity (Littlefield et al., 2009, 2010a, 2010b). Others have shown similar personality effects with respect to tobacco use (Welch & Poulton, 2009), drug use (Johnson, Hicks, McGue, & Iacono, 2007), and antisocial behavior (Blonigen, Littlefield, Hicks, & Sher, 2010; Johnson et al., 2007). These patterns suggest that in addition to age-related offset there is also age-related loss of vulnerability to new and recurring problems.

Our findings call into question distinctions between adolescent-limited and more life-course persistent forms of disorder (Moffitt, 1993; Zucker, 1987). This is not to deny that some disorders are highly prevalent in adolescence and tend to remit. However, we believe one should be more reticent to conclude that this course implies a specific “species” or subtype of disorder. If offset rates of various disorders tend to be somewhat constant over the life course, perhaps we should think of “short duration” versus “chronic and episodic” courses with “short duration” not limited to adolescence (although likely more prevalent then). Findings from a recent taxometric analysis of antisocial behavior (Walters, 2010) suggest that the distinction between adolescence-limited and life-course persistent antisociality are likely more of degree than of kind and the current analyses suggest that a similar argument could be made with respect to AD.

Life Transitions and the Course of AD

Although the role incompatibility explanation suggests an effect of life transitions that occurs mostly during emerging adulthood, our analyses of the association between course and major life role transitions present a more complex picture. Significant associations involving life transitions were found as main effects as well as in interaction with sex and age, suggesting that life transitions play a role that is not limited to young individuals. In particular, both parenthood and change in employment status were associated with a new onset of AD between waves, an effect that was consistent across gender and age. This suggests that life transitions that relate to new onset are not limited to emerging adulthood but might also play a role later in life.

Moreover, when interactions were found, these revealed a larger association of role transitions (i.e., parenthood and change in job, although the latter was not significant in multiple imputation analyses) with persistence among older men, another unexpected finding according to the role incompatibility explanation.

The fact that changes in marriage status were not found to be associated with any of the patterns of stability and change was also unexpected in light of the broader literature. Both marriage (Miller-Tutzauer et al., 1991) and divorce (Bachman et al., 1997) have been reported to be associated with changes in alcohol use. With regard to AD, Chilcoat and Breslau (1996) found that rates of new onset and persistence of DSM-III-R abuse or dependence were influenced by marital status during a 3.5-year interval, and Dick et al. (2006) found evidence of a significant gene-environment interaction involving marital status in predicting AD, such that the influence of the *GABRA2* genotype on alcohol dependence is high among individuals who report a stable marriage and is not significant among those who are not married. However, this is the first report on this issue that is based on epidemiological data, and it indicates that the effect of marital status on the course of AD might not be as strong as suggested by previous studies.

In order to take a closer look at this issue, we examined the prevalence of AD at both waves by transition in marital status, in order to determine if there is a marriage effect in the overall sample (that is, not conditioned on AD group). This allowed for a further distinction in the stable group between participants who were married, single, or divorced at both waves. Results (not shown) suggest a pattern of selection and socialization effects that are consistent with the broader literature, in particular with results of the Monitoring the Future study reported by Bachman et al. (1997), in that preexisting differences (i.e., the effect of selection into roles) are important, but not as strong as the current status (i.e., the effect of role socialization) at each wave. Moreover, the effect of marriage transitions is consistent with expectations, showing that getting married is associated with a decrease, whereas getting divorced is associated with an increase in the percentage of AD (with the notable exception of an increase in AD among older participants who reported being single at wave 1 and married at wave 2). In sum, although an association with change in marital status can be found in the overall sample, no such association was found for persistence, onset or recurrence in AD. This might be due in part to the fact that we had to collapse different categories of stability (i.e., stable single, married, and divorced) into a very heterogeneous group.

Our findings regarding parenthood and employment status were more consistent with previous reports. Becoming a parent was protective against new onset of AD in the overall sample, and against persistence of AD among older males. Bachman et al. (1997) found that becoming pregnant was linked to a significant decrease in heavy drinking among women as well as their partners, although to a lesser extent. Similarly, we found that becoming employed was protective against AD persistence among older males. This is consistent with the notion that structured everyday life, as defined by Neve et al. (2000), has a salutary effect on alcohol involvement, but is not consistent with their finding that a higher level of structure is positively associated with heavy drinking and drinking problems among older individuals. Moreover, the fact that getting a job is associated with AD onset is not consistent with the positive effects of a more structured everyday life, but is consistent with a tension reduction perspective (according to which stress at work is related to an increase in alcohol use; Butler, Dodge, & Faurote, 2010), and with the increase of alcohol availability through the new work environment and increased financial resources (Gruenewald, Millar, & Treno, 1993; Treno, Grube, & Martin, 2003). The general pattern of findings we see is that role transitions are associated with the course of alcohol disorders throughout the life course but that the effects differ as a function of age, sex, and type of transition. This is not surprising in that depending upon when a transition occurs, it can either represent an age-normative transition or a non-normative transition. Additionally, the degree to which a given role transition presents structural role incompatibility (e.g., Yamaguchi & Kandel, 1985) with drinking will likely vary as a function of the stage of life when it occurs, the sex of the

person, and particulars of the nature of the transition (e.g., employment in a wet or dry working environment; Hodgins, Williams, & Munro, 2009; Roman & Blum, 2002).

Limitations

Although the large sample size of the NESARC presents a number of opportunities that permit, perhaps uniquely so, the types of analyses presented here, there are a number of limitations that need to be noted. First, all of these data are based on self-report and thus subject to a range of reporting biases. Also, although retention in the sample was excellent, approximately 80%, attrition could affect our findings with respect to course of disorder. However, the fact that the analyses using multiple imputation yield a very similar picture to the one found with listwise deletion suggests that the effect of attrition does not produce a strong bias in our findings. Additionally, although the NESARC has considerable information relevant to various role transitions, this study was not designed as a study of role transitions and various contextual measures of role functioning (e.g., dyadic adjustment; success in one's work role) and structure (e.g., whether or not parenthood was associated with offspring with special needs; the degree of job decision latitude in the workplace) are not resolved and so these analyses are relatively crude. Our measure of change in employment status is imperfect, as our coding could incorrectly classify a few individuals who were unemployed at Wave 1, employed during the period between Waves 1 and 2, and were unemployed again at Wave 2 (and vice versa). The effect of such coding errors would be to underestimate the effects of employment. Also, in our coding for change in marital status we did not differentiate between first marriage (more likely among younger participants) and being remarried (more likely among older participants). A similar issue occurs with parenthood, given that an individual who becomes a parent after age 35 is more likely to be a step-parent. Thus, our finding of an association between parenthood and new onset of AD must be interpreted considering that different types of parenthood occur at different ages. The dataset also lacks information about indices of psychosocial maturity such as traits related to negative emotionality and impulsivity, and hence the present study is limited to the examination of external constraints (i.e., role incompatibilities) on AD.

Despite the fact that the NESARC surveyed almost 35,000 individuals on two occasions, the size of subsamples become quite small when considering participants who have a given diagnosis at baseline, at a certain age, of a specific gender, and experiencing a change in role status. Indeed, age is strongly related to role transitions like marriage, starting a family, and entering the workforce (i.e., these life transitions are common during young adulthood, but become less prevalent later in life), forcing us to use fairly wide age strata to characterize developmental stages, and preventing us from distinguishing between participants who remained employed and those who remained unemployed, as well as between participants who remained married and those who remained single (all of which were coded as "stable"). Additionally, since life transitions were assessed at the same time as transitions in AD (i.e., both types of transitions were determined through the comparison of status at wave 1 and wave 2) and individuals can select into these roles, our results should not be taken to imply a causal effect of life transitions on the pattern of stability and change in AD.

Given that the current paper was focused on AD diagnosis, we did not conduct analyses on heavy drinking. As noted in footnote 1, offset from heavy drinking might underlie changes in AD persistence, onset and recurrence. In that case, our results regarding AD onset and recurrence might still be due to "maturing out" as traditionally understood (i.e., as offset). However, it is also possible that heavy drinking shows a similar decomposition as the one reported here, with changes in new onset driving most of the change in the overall prevalence of heavy drinking. If this is the case, we would need to look at processes beyond alcohol use to account for changes in AD diagnosis.

Conclusions

In recent years it has become increasingly recognized that AD is largely a phenomenon of youth, and that individuals tend to mature out of this disorder (Sher & Gotham, 1999). However, it has not been widely recognized that most of the decrease in AD over the life course is attributable to the reduction of onset and recurrence processes in addition to the increase in persistence. Moreover, the rates of persistence are relatively constant over the course of adulthood. These findings present challenges to subtyping approaches that assume that some forms of disorder are developmentally limited to the extent that the period of development is adolescence. Indeed, our findings raise the possibility that AD is developmentally limited throughout the life course and that their courses vary as a function of maturation and role statuses. Moreover, given similar findings with other disorders, at least those in the externalizing spectrum, it seems likely that the general principles that are suggested by these data may be broadly applicable to other conditions and perhaps to personality as well. Specifically, it appears that late adolescence and emerging adulthood are associated with a high risk for the onset and recurrence of disorder but that offset from these problems occurs at a fairly constant rate over adulthood. Moreover, the same processes that lead to offset likely also lead to less onset and recurrence of problems so that by focusing on “maturing out”, narrowly understood as offset, we are missing a broader and potentially equally important phenomenon. We do not believe that this study’s findings necessarily cast doubt on all developmental taxonomies. However, the findings highlight the need to consider the larger developmental context (i.e., there are pathogenic and salutary influences on disorder throughout the life span) and expand our view to include factors that generate pathology as well as mitigate it throughout the life span. While we believe the field recognizes that many affected individuals “mature out” of problems, it is less recognized that maturation processes similarly inhibit the development and recurrence of problems. This is likely because problems that fail to occur are less obvious than ones that remit.

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Figure 1.

Relative contribution of the three AD groups to the overall AD prevalence across age groups. The Y-axis represents the percentage of participants diagnosed with past-12-month AD at Wave 2. Age groups were created based on age reported at Wave 1, so that the X-axis shows the approximated age at Wave 2. n = total number of participants within each age group.

Figure 2.

Prevalences of the three AD groups among participants at risk. Brackets represent 95% confidence intervals around the mean. The Y-axis represents the percentage of participants diagnosed with past-12-month AD at Wave 2. Age groups were created based on age reported at Wave 1, so that the X-axis shows the approximated age at Wave 2. In the upper panel (new onset) participants at risk are those with no lifetime history of AD at Wave 1. In the middle panel (persistence) participants at risk are those with past-12-month AD diagnosis at Wave 1. In the bottom panel (recurrence) participants at risk are those with prior-to-past-12-month, but no diagnosis in the past 12 months at Wave 1. n = number of participants at risk within each age group.

Figure 3.

Three-way interactions among age, sex, and life transitions. Brackets represent standard errors. Age groups were created based on age reported at Wave 1, so that the X-axis shows the approximated age at Wave 2. Parenthood = Had any children or any adopted/foster/step children from Wave 1 to Wave 2; No Parenthood = no change in parenthood from Wave 1 to Wave 2; Lose Job = Became unemployed or retired from Wave 1 to Wave 2; Stable = No change in employment status from Wave 1 to Wave 2; Get Job = Became employed from Wave 1 to Wave 2.

Table 1

Characteristics of Participants Retained at Wave 2

Variable	% Retained (SE) (N=28,482)
Females	82.7 (0.6) *
Males (r)	80.2 (0.7)
Age	
18-20 (r)	79.0 (1.3)
21-24	76.4 (1.5)
25-29	81.0 (1.2)
30-39	81.8 (0.9) *
40-49	85.4 (0.5) *
50-59	85.7 (0.6) *
60 Over	76.9 (0.6)
Race	
White (r)	83.6 (0.4)
Black	79.2 (0.8) *
American Indian	83.8 (1.7)
Asian	73.3 (1.7) *
Hispanic	73.4 (1.2) *
Alcohol Dependence	
No Diagnosis (r)	81.2 (0.6)
Current	79.1 (1.5)
Prior to Past 12	
Months	85.6 (0.8) *

Note. Age presented at baseline. (r) = group of reference for comparisons.

* significantly different from the group of reference.

Table 2

Characteristics of the AD Groups

Variable	Alcohol Dependence at W2			No Alcohol Dependence at W2		
	Persistence (N=422)	Onset (N=767)	Recurrence (N=244)	Past 12 MO AD W1 (N=750)	Lifetime but no 12 MO AD W1 (N=2541)	No Lifetime AD W1 (N=29929)
Age	32.8 (0.7) ^b	33.0 (0.6) ^b	36.6 (0.9) ^c	30.4 (0.5) ^a	41.8 (0.3) ^d	46.4 (0.2) ^e
Age First Drinking	16.9 (0.3) ^a	19.9 (0.5) ^b	16.7 (0.3) ^a	17.3 (0.2) ^a	17.1 (0.1) ^a	21.7 (0.1) ^c
% Male	72.4 (2.5) ^c	65.4 (2.0) ^b	71.6 (3.2) ^b	65.5 (1.9) ^b	65.5 (1.1) ^b	44.7 (0.4) ^a
% White	73.6 (3.3) ^{bc}	60.4 (2.9) ^a	78.6 (3.2) ^{cd}	67.8 (2.5) ^b	81.5 (1.3) ^d	70.2 (1.6) ^b
% Married W1	34.3 (2.9) ^a	42.9 (2.2) ^b	54.5 (3.9) ^c	35.3 (2.1) ^a	65.4 (1.1) ^d	64.7 (0.5) ^d
% Married W2	37.7 (3.1) ^a	41.5 (2.3) ^a	55.9 (4.4) ^b	42.2 (2.1) ^a	67.1 (1.0) ^c	65.1 (0.5) ^c
% Parent W1	49.8 (3.0) ^b	52.0 (2.1) ^b	59.4 (3.6) ^b	41.5 (2.2) ^a	73.3 (1.1) ^c	74.7 (0.5) ^c
% Parent W2	52.8 (2.9) ^a	55.2 (2.2) ^{ab}	62.5 (3.6) ^b	51.0 (2.2) ^a	77.3 (1.0) ^c	78.1 (0.5) ^c
% Employed W1	72.2 (3.0) ^b	72.8 (2.1) ^b	75.4 (3.8) ^b	74.4 (1.8) ^b	76.0 (1.0) ^b	64.5 (0.5) ^a
% Employed W2	72.2 (2.9) ^b	76.9 (1.7) ^{bc}	80.3 (2.8) ^{bc}	80.9 (1.5) ^c	74.8 (1.1) ^b	63.2 (0.4) ^a

Note. Values that share the same subscript letter are not statistically different. Age, age at first drinking, sex and race, were assessed at Wave 1.

Table 3

Summary of Analysis of Predictors of AD transitions

Variable	Persistence N 12 MO AD at BL = 993 n 12 MO AD at FU = 363		Onset N no LT AD at BL = 26079n 12 MO AD at FU = 666		Recurrence N LT no 12 MO AD at BL = 2497n 12 MO AD at FU = 221	
	OR	95% CI	OR	95% CI	OR	95% CI
Sex (Female ref)	1.20	0.67-2.15	1.87	1.29-2.73	1.00	0.52-1.93
Race (White ref)	0.76	0.54-1.08	1.23	0.99-1.52	1.02	0.65-1.60
Age (21-37 ref)	1.44	0.68-3.05	0.22	0.14-0.34	0.47	0.22-1.01
Parenthood (Not Parent ref)	0.22	0.06-0.82	0.39	0.18-0.83	0.19	0.03-1.40
Change Marital (Stable ref)						
Divorced	1.00	0.26-3.83	1.80	0.85-3.77	2.56	0.79-8.35
Married	1.01	0.34-3.02	1.49	0.74-3.03	1.34	0.49-3.67
Change in Job (Stable ref)						
Lost Job	3.18	0.81-12.49	0.56	0.19-1.65	1.94	0.34-11.20
New Job	0.84	0.19-3.72	2.79	1.19-6.55	0.51	0.06-4.34
Sex*Age	1.13	0.47-2.72	1.06	0.62-1.80	1.46	0.60-3.55
Sex*Parenthood	4.56	1.09-19.08	1.48	0.61-3.57	3.50	0.39-31.60
Sex* Change Marital						
Male Divorced	1.31	0.18-9.29	1.18	0.44-3.17	0.57	0.08-4.20
Male Married	0.65	0.18-2.34	0.63	0.26-1.55	1.63	0.43-6.22
Sex*Change in Job						
Male Lost Job	0.67	0.12-3.66	2.81	0.80-9.89	0.43	0.05-3.70
Male New Job	2.14	0.36-12.70	0.71	0.24-2.08	5.93	0.42-83.46
Age*Parenthood	9.56	1.14-79.78	0.55	0.11-2.86	10.64	0.55-204.78
Age*Change Marital						
38+ Divorced	0.64	0.08-5.25	0.83	0.27-2.50	0.70	0.09-5.59
38+ Married	2.60	0.51-13.20	1.12	0.39-3.17	1.37	0.29-6.51
Age*Change in Job						
38+ Lost Job	0.53	0.07-4.27	3.14	0.69-14.29	0.96	0.10-9.61
38+ New Job	1.90	0.21-17.59	1.23	0.29-5.15	0.55	0.03-10.29

Variable	Persistence N 12 MO AD at BL = 993 n 12 MO AD at FU = 363		Onset N no LT AD at BL = 2607n 12 MO AD at FU = 666		Recurrence N L T no 12 MO AD at BL = 2497n 12 MO AD at FU = 221	
	OR	95% CI	OR	95% CI	OR	95% CI
Sex*Age*Parenthood	0.02	0.00-0.28	2.81	0.41-19.12	0.08	0.00-2.06
Sex*Age*Change Marital						
Male 38+ Divorced	1.01	0.06-18.14	2.89	0.73-11.39	2.98	0.18-49.23
Male 38+ Married	1.95	0.23-16.68	2.89	0.63-13.26	0.22	0.03-1.93
Sex*Age*Change in Job						
Male 38+ Lost Job	2.15	0.19-24.55	0.33	0.06-1.85	1.02	0.06-16.16
Male 38+ New Job	0.03	0.00-0.48	0.90	0.16-5.25	1.26	0.04-35.95

Note. Sample sizes differ from those in Table 2 due to missing data in the role transition variables. MO = months; BL = baseline; FU = follow-up; LT = lifetime; OR = odds ratio; ref = reference group.