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Young Adult Social Development as a Mediator of Alcohol Use Disorder Symptoms From Age 21 to 30

Rick Kosterman,
Social Development Research Group, School of Social Work, University of Washington

Karl G. Hill,
Social Development Research Group, School of Social Work, University of Washington

Jungeun Olivia Lee,
Social Development Research Group, School of Social Work, University of Washington

Meredith C. Meacham,
Division of Global Public Health, School of Medicine, University of California, San Diego

Robert D. Abbott,
Educational Psychology, University of Washington

Richard F. Catalano, and
Social Development Research Group, School of Social Work, University of Washington

J. David Hawkins
Social Development Research Group, School of Social Work, University of Washington

Abstract

Little research has examined social development in the young adult years relative to childhood and adolescence. This study tested the hypothesized pathways of the social development model (SDM) in young adulthood for predicting symptoms of alcohol use disorder (AUD) and positive functioning at age 30. A longitudinal panel study originally drawn from Seattle, Washington, elementary schools was examined. The sample included 808 participants with high retention and was gender balanced and ethnically diverse. Analyses focused on ages 21, 27, and 30. SDM constructs were assessed with self-reports of past-year behavior and combined multiple life domains. AUD symptoms corresponding to DSM-IV criteria were assessed using the Diagnostic Interview Schedule. Positive functioning combined measures of constructive engagement in work and school, civic engagement, physical exercise, and lack of depressive symptoms. The study found that AUD symptoms were moderately stable from age 21 to 30; however, developmental pathways hypothesized by the SDM at age 27 played a significant role in partially mediating this association. Alcohol-specific factors were key mediating mechanisms, whereas prosocial factors played little role. Conversely, prosocial factors had an important role in predicting positive functioning at age 30, while there were no significant pathways involving alcohol-specific factors. Findings suggest that age 27 is not too late for interventions targeting adult social development to help diminish alcohol use disorder symptoms by age 30. Alcohol-specific factors such as reducing

Correspondence should be addressed to Rick Kosterman, Social Development Research Group, University of Washington, 9725, 3rd Avenue N. E., Suite 401, Seattle, WA 98115; rickk@uw.edu; office 206-543-4546.

perceived opportunities or rewards for heavy alcohol use or challenging beliefs accepting of drunkenness are likely to be key ingredients of effective adult interventions.

Keywords

young adulthood; social development; alcohol use disorder; positive functioning

Young adulthood from age 21 to the early 30s is an important developmental period for alcohol use. For most young adults, consumption of alcohol peaks in the early 20s and then declines as many complete college and/or begin families and careers (Bachman et al., 2002; Chassin, Fora, & King, 2004; Flora & Chassin, 2005; Maggs & Schulenberg, 2004). For some, however, their alcohol use increases or remains high through the 20s, often exhibiting symptoms of abuse or dependence (Chassin et al., 2004; Flora & Chassin, 2005; Warner, White, & Johnson, 2007). Understanding the factors that predict alcohol use disorder (AUD) symptoms during this period is of particular importance because significant changes in alcohol consumption subsequent to the 20s are relatively rare. Johnstone et al. (1996) reported in their meta-analysis of 29 studies including more than 50,000 participants that, while change in alcohol consumption was substantial prior to age 30, subsequent years were characterized by moderate, nonsignificant, and statistically uniform declines.

Several studies have examined childhood and adolescent risk and protective factors for alcohol problems in early adulthood. Predictors have been identified across the major domains of life, including community (e.g., neighborhood disorganization), family (e.g., poor parental monitoring), school (e.g., lack of commitment), and individual or peer (e.g., positive alcohol expectancies, peer alcohol use) (Chassin, Pitts, & Prost, 2002; Flory, Lynam, Milich, Leukefeld, & Clayton, 2004; Guo, Hawkins, Hill, & Abbott, 2001; Hawkins, Catalano, & Miller, 1992). However, relatively little research has focused on predictors in the 20s of either concurrent or subsequent alcohol use problems. Exceptions include a study by Hicks et al. (2010) that examined a community-based sample of 530 men. They found that a persistent course of AUD to age 29 was associated with more antisocial peers, fewer prosocial peers, and higher rates of alcohol use by a romantic partner in young adulthood. Other studies have examined the importance of young adult social roles, finding marriage to be consistently protective against heavy alcohol use, but more mixed effects for other factors such as parenthood, college attendance, and job-related factors (Bachman et al., 2002; Bachman, Wadsworth, O'Malley, Johnston, & Schulenberg, 1997; Duncan, Wilkerson, & England, 2006; Eitle, Taylor, & Eitle, 2010; Paschall, Bersamin, & Flewelling, 2005; Schulenberg & Maggs, 2002). Reflecting the array of these and other possible young adult roles, Arnett (2000, 2005) has argued that the late teens to mid 20s is a distinct developmental stage characterized by unusually high levels of change and exploration that portend unique risks. Some recent reviews of development and alcohol use during this period are consistent with this perspective, noting exceptional social and cognitive vulnerabilities and wide variations in living and work situations (S. A. Brown et al., 2008; Carter, Brandon, & Goldman, 2010). A better understanding of these risks, their distinctive role beyond prior risk behaviors, and their specific contribution to AUD symptoms in the 30s is needed (Aseltine & Gore, 2005; Eitle et al., 2010).

Further, we are aware of no studies that have sought to organize risk and protective factors in the 20s into a broad model of social development in young adulthood. Tests of models incorporating diverse social predictors across multiple life domains are needed to better understand the mechanisms by which the social environment affects alcohol abuse and dependence. Because predictors may be interrelated in a number of ways, theoretical specification and model testing is particularly important for identifying meaningful sequences of effects and mediated processes (Bursik & Grasmick, 1996). By illuminating these processes, well-tested models are indispensable for improving preventive intervention efforts designed to disrupt processes leading to risk for disorder, or to promote processes that enhance protection (Catalano, Hill, Haggerty, Fleming, & Hawkins, 2010; Catalano et al., 2005). The present investigation addresses these research needs by examining the utility of the social development model in young adulthood for predicting AUD symptoms at age 30, accounting for AUD symptoms at age 21.

The Social Development Model

The social development model (SDM) is a developmental theory that specifies etiological pathways to antisocial and prosocial outcomes (Catalano & Hawkins, 1996; Catalano et al., 2005; Hawkins & Weis, 1985). It integrates features of social learning theory (Bandura, 1977), social control theory (Hirschi, 1969), and differential association theory (Sutherland & Cressey, 1970), and has demonstrated good fit across studies and samples predicting diverse problem outcomes to age 21. Catalano et al. (1996) found that the SDM predicted a composite measure of substance use at ages 17 and 18 and that the hypothesized pathways of the model were largely consistent with that data. In a study of minority youth, Choi et al. (2005) also found support for SDM pathways predicting substance use across ethnic groups aged 10 to 14. A test by Lonczak et al. (2001) found confirmation of SDM hypotheses in the prediction of alcohol misuse at age 16. Other studies have found support for the SDM in predicting violent behavior (Choi et al., 2005; Huang, Kosterman, Catalano, Hawkins, & Abbott, 2001) and composite measures of problem behavior (E. C. Brown et al., 2005; Catalano, Oxford, Harachi, Abbott, & Haggerty, 1999; Catalano et al., 2005; Fleming, Catalano, Oxford, & Harachi, 2002; Kosterman, Haggerty, Spoth, & Redmond, 2004) in childhood and adolescence. The SDM provides a promising framework for investigating young adult problems because it incorporates empirically determined risk and protective factors across multiple life domains. The focus of the SDM on *social* influences may be especially apt for this period during which the alcohol-related norms and behaviors of peers and social networks have been found to be particularly important, both for both college and non-college young adults (Lau-Barraco & Collins, 2011; Overbeek et al., 2011; Talbott, Moore, & Usdan, 2012; van Schoor, Bot, & Engels, 2008; White, Fleming, Kim, Catalano, & McMorris, 2008). Here we extend the SDM into the mid 20s in order to investigate hypothesized social developmental processes in young adulthood and their relationship to later AUD symptoms.

As shown in Figure 1, the SDM identifies specific features of the social environment affecting behavior. The model hypothesizes that social development is influenced by important social structural factors, including external social constraints (social norms, rules, and laws) and one’s position in the social structure (socioeconomic status, race, gender), and

by individual characteristics (such as temperament differences that affect responses to the environment). Additionally, the SDM accounts for effects of behavior in the prior developmental period on subsequent social development. Within this context, the proximal social developmental environment is defined by perceived opportunities for involvement in both prosocial interactions and antisocial interactions, by one's actual involvement and his or her skills to perform in these pro- or antisocial interactions, and by the perceived rewards or costs one experiences as a consequence of involvement with prosocial or antisocial others. When opportunities lead to involvement, and that involvement is skillful and rewarded, a social bond of emotional attachment and commitment develops to the individual or group with whom one is interacting, whether prosocial or antisocial. Bonding, in turn, creates motivation in the individual to adopt and conform to the norms and values of the individual or group to whom the individual feels bonded, whether involved in prosocial or antisocial activities.

In sum, the SDM hypothesizes both a "prosocial pathway" leading to positive functioning and an "antisocial pathway" leading to problem behaviors such as substance abuse. Moreover, by focusing on opportunities, involvement, rewards-costs, and bonding, the pathways incorporate experiences across different life domains and can account for diverse life experiences. For example, if one young adult lives with roommates and spends most of his or her time with peers, and another lives with a spouse and children spending most of his or her time with family, the model will capture the opportunity and reward structures of each environment. In this way, the model is sensitive to the variety of life courses in young adulthood.

In testing the pathways hypothesized by the SDM, the present study investigated the relative strength of an alcohol-specific antisocial pathway in predicting AUD symptoms. Specifically, analyses examined an antisocial pathway that included perceived opportunities with partners, friends, and others who used alcohol heavily; involvement with heavy alcohol users; perceived rewards and costs for alcohol use; bonding with heavy alcohol users; and beliefs approving of regular drinking and getting drunk. To assess the relative importance of the alcohol-specific environment, the SDM specifies the inclusion of prosocial factors as well. The alcohol-specific pathway was therefore compared with a corresponding prosocial pathway that represented prosocial developmental influences (e.g., perceived opportunities with others who engaged in a variety of prosocial behaviors such as volunteering, health-promoting activities, etc.). Note that the modeled hypotheses included direct prediction of AUD symptoms from perceived alcohol rewards and costs, and from bonding with alcohol-using others, but no direct prediction from the corresponding prosocial constructs. These additional antisocial paths are specified by the SDM based on related theories (Hirschi, 1969) and empirical findings (Agnew, 1991) suggesting that perceived rewards (or low costs) for antisocial behavior, or bonding to others engaged in the behavior, can directly increase the behavior without the mediating influence of beliefs. For prosocial behaviors, it is hypothesized that one's belief in prosocial values is a necessary mechanism (Catalano & Hawkins, 1996).

Finally, to better understand the role of the SDM across different young adult behaviors, we tested the extent to which both of these pathways predicted AUD symptoms at age 30 as

well as their prediction of young adult positive functioning included together in a two-outcome model. This model enabled comparison of the predictive influence of an alcohol-specific antisocial pathway and a corresponding prosocial pathway for an alcohol disorder outcome contrasted with a positive functioning outcome in young adulthood. These analyses were important for helping to understand general versus specific effects of pathways specified by the SDM on concurrent antisocial and prosocial outcomes.

Study Aims

We sought to address three main aims. First, we aimed to model social development in a sample of young adults using the SDM, including a prosocial, protective pathway and an antisocial, risk pathway specific to alcohol use. Modeled constructs and pathways conformed to the developmental processes hypothesized by the SDM. The second aim was to test the young adult SDM for predicting symptoms of alcohol use disorder at age 30. We hypothesized that the young adult mechanisms specified by the SDM would play a significant role in AUD symptoms at age 30, even after accounting for prior AUD symptoms at age 21. Further, we hypothesized that the antisocial pathway, specific to alcohol use, would play the largest role given the consistency of component indicators with the AUD outcome. Finally, we aimed to compare SDM constructs predicting AUD symptoms with those predicting positive adult functioning at age 30. In order to understand the specific effects of both the prosocial and antisocial pathways, we examined their effects in a two-outcome model adding a prosocial outcome. We hypothesized larger effects of the prosocial pathway on positive functioning in young adulthood because of consistency of pathway indicators (positive involvement in work, school, community, etc.) with components of positive functioning, in line with our hypothesis for the antisocial pathway and AUD symptoms.

Methods

Sample

The Seattle Social Development Project (SSDP) is a longitudinal study examining a broad range of behavior and health outcomes. The original study population in 1985 included all fifth-grade students in 18 Seattle, Washington, elementary schools that overrepresented students drawn from high-crime neighborhoods (N = 1,053). From this population, 808 students (77%) consented to participate in the longitudinal study and constituted the SSDP sample. Data for analyses reported here were collected in the spring of 1996, when most participants were 21 years old (median age was 20.83 years, M = 20.73, SD = .56), and again at ages 27 and 30 years. Retention of the original fifth-grade sample averaged 94% across these three waves among those still living (18 participants were deceased by age 30). The sample was 51% male, 47% European American, 26% African American, 22% Asian American, and 5% Native American. As children, 52% of participants were eligible for the federal school lunch/breakfast program at some point in the fifth, sixth, or seventh grade, based on low-income status. Median household income at age 30 (2005) was $47,500; 16% of households reported annual income below $19,000 (the approximate U.S. Census poverty threshold for a four-person household in 2005). At age 30, most participants were unmarried (60%), fully employed (89%), and had completed at least some college (65%).

Participants were interviewed in person, including those who moved outside Seattle, and received monetary compensation (e.g., $90 in 2005) for completing the survey which lasted 2 to 3 hours. Responses to close-ended items were entered into a laptop computer by the interviewer. All phases of the study were approved by the Human Subjects Review Committee at the University of Washington.

Measures

Social development model—Table 1 provides a summary of SDM measures used in the study. Other examples of measures are reported in Brown et al. (2005), Catalano et al. (1996; 1999; 2005), Fleming et al. (2002), Huang et al. (2001), and Kosterman et al. (2004). Measures were developed or added over the 20-year period of the longitudinal study and were validated in the prior reports. The model tested here focused on the key developmental constructs along the prosocial and antisocial (alcohol-specific) pathways. Given the analytic complexity of the model and our focus on the developmental pathways, social structural factors exogenous to these pathways were not included with the exception of gender. All SDM constructs were assessed with self-reports of past-year behavior at age 27. For each construct, multiple items similar to those shown illustratively in Table 1 were assessed across multiple life domains (spouse/partner, dating, peers, housemates, work/school, community). Measures were standardized and combined into mean scales. Note that some measures related to involvement, rewards-costs, and bonding required a *valence* adjustment. For example, for measures of involvement with partner it was necessary to account for the partner's engagement in prosocial activities (on the prosocial pathway) and the partner's heavy alcohol use (on the antisocial pathway). This was accomplished by using an algorithm wherein involvement measures were weighted by the partner's level of engagement in prosocial activities or heavy alcohol use, respectively (Hawkins, Doueck, & Lishner, 1988; Kosterman et al., 2011)y. For all measures, higher scores indicate more of the construct as labeled.

Alcohol use disorder symptoms—Alcohol use disorder diagnostic criteria corresponding to the DSM-IV (American Psychiatric Association, 1994) were assessed at ages 21 and 30 using the Diagnostic Interview Schedule (DIS) (Robins et al., 1999) (e.g., increased tolerance, withdrawal symptoms, failure to fulfill major role obligations). The number of abuse and dependence symptoms met were summed, consistent with King et al. (2012). The resulting measures were coded to a range of 0 to 4, because fewer than 8% of participants exceeded four symptoms, and then log transformed to address skewness (reliability α = .74 and .81 at ages 21 and 30, respectively). Approximately 48% and 30% reported one or more AUD symptoms at ages 21 and 30, respectively, with means of 1.26 and 0.79 symptoms, respectively.

Positive functioning—A global measure of positive functioning was created for age 21 and for age 30 by combining constructive engagement (total hours per week spent in work, school, and homework), civic engagement (hours per month spent in community groups and volunteering), physical exercise (minutes per week of at least moderate intensity exercise), and lack of depressive symptoms (taking the inverse of the sum of disorder criteria assessed with the DIS) (Hawkins, Kosterman, Catalano, Hill, & Abbott, 2005, 2008; Kosterman et

al., 2005; 2011). Physical exercise at the prior time point was assessed at age 24 because necessary items were not available at age 21. All other prior measures were assessed at age 21 (we refer to the timing of this measure as "age 21," having noted the exception for physical exercise). Component measures were log transformed and standardized (to reduce skewness and create compatible metrics), then combined into mean scales (α = .81 and .87 at ages 21 and 30, respectively).

Analyses

Analyses were conducted using Mplus 6.1 (Muthén & Muthén, 1998–2010), a program that computes full-information maximum likelihood (FIML) estimates with incomplete data. Maximum likelihood missing data estimation yields less biased parameter estimates than traditional methods such as pairwise or listwise deletion of cases (Schafer & Graham, 2002). The comparative fit index (CFI) (Bentler, 1990) and root mean square error of approximation (RMSEA) (Browne & Cudeck, 1993) are reported for overall model fit. Standardized path coefficients are presented throughout.

Part of the sample was exposed to a multicomponent preventive intervention in the elementary grades, consisting of teacher training, parenting classes, and social competence training for children (Hawkins et al., 2005, 2008). Control participants were interviewed but had no intervention exposure. Consistent with prior analyses that have shown few differences in the covariance structures of the intervention and control groups (Catalano et al., 1996; Herrenkohl, Kosterman, Hawkins, & Mason, 2009; Huang et al., 2001; Kosterman et al., 2005; McCarty et al., 2009), analyses shown here were based on the full sample after examining possible differences in the covariances corresponding to the models examined for this report, comparing the control group and intervention conditions combined. We conducted a multiple-group structural equation model test comparing a model where covariances were constrained to be equal across the control (n = 220) and intervention groups (n = 588) to a model in which these parameters were freely estimated (Muthén & Muthén, 1998–2010). This test showed no significant reduction in the overall fit of the constrained model ($\Delta\chi^2(17) = 7.65$, p = .97), suggesting no substantial group differences in the relationships of interest in this report and supporting single-group analyses.

Results

Zero-order correlations among study variables are shown in Table 2. As a first step, a measured variable model was tested predicting AUD symptoms with pathways hypothesized by the SDM. This model was consistent with the theoretical illustration in Figure 1 but included only gender and prior AUD symptoms as exogenous variables and AUD symptoms at age 30 as the outcome. The residuals of all corresponding prosocial and alcohol-specific constructs except opportunities were significantly correlated and these covariances were included in the model. Overall, this initial model fit the data poorly: $\chi^2(66) = 1398.14$, CFI = .84, RMSEA =.16.

The initial model indicated that the opportunities, involvement, rewards-costs, and bonding measures were closely linked in both the prosocial and alcohol-specific pathways. The magnitude of the standardized pathways connecting these variables ranged from .80 to .91,

suggesting that broad latent factors may be appropriate to represent prosocial socialization (capturing the common variation in prosocial opportunities, involvement, rewards-costs, and bonding) and alcohol-specific socialization (capturing the common variance in the respective alcohol-specific constructs). To model these constructs, we next examined the latent variable framework shown in Figure 2. Latent factors of "prosocial socialization" and "alcohol-use socialization" were each indicated by the respective opportunities, involvement, rewards-costs, and bonding scales. "Belief in the moral order" (indicated by beliefs in honesty, the work ethic, and civic obligations), "belief in pro-alcohol values" (indicated by approval of getting drunk regularly, occasionally, and drinking daily), and "skills for interaction" (indicated by interpersonal competence, consequential thinking, and refusal skills) were also modeled as latent factors. All factor loadings were positive and statistically significant ($p < .001$ for each) and the measurement model fit the data adequately: $\chi^2(145) = 798.68$, CFI = .93, RMSEA =.08. As in the measured variable model, significantly correlated residuals of indicator variables and model factors were included but, for clarity, are not shown. Fit of this model was adequate and considerably improved over the measured variable model: $\chi^2(151) = 723.60$, CFI = .94, RMSEA =.07.

The model in Figure 2 indicates that the structural relations among SDM constructs at age 27 were consistent with hypotheses and highly significant, with the exception of the path from skills to alcohol-use socialization. The model also shows stability in AUD symptoms with a significant direct link from age 21 to 30 ($\beta = .27$, $p < .001$), but that adult social development in the intervening years still played an important role in partially mediating the zero-order association (which, in addition to gender, diminished the zero-order coefficient from .37). The total indirect effect of prior AUD symptoms at age 21 on age 30 AUD symptoms through SDM constructs was significant at $p < .001$ ($\beta = .06$). The key mediated pathways were through alcohol-use socialization and belief in pro-alcohol values. The prosocial pathway was indirectly predicted by prior AUD symptoms through skills for interaction, but prosocial factors did not significantly predict AUD symptoms at age 30. Gender had significant associations with both pathways: males experienced less prosocial socialization and more alcohol-use socialization compared to females. We tested all other possible direct paths from each latent construct to AUD symptoms at age 30 but none were significant.

Finally, we sought to understand the specific effects of both SDM pathways across different young adult behaviors including alcohol use as well as prosocial outcomes. We examined a two-outcome model building on the prior model by adding positive functioning at age 30 and accounting for a parallel measure of positive functioning at age 21. Figure 3 shows mostly significant structural relations among SDM constructs similar in magnitude to the prior model, as well as significant stability in positive functioning from the early 20s to age 30 ($\beta = .32$, $p < .001$). A significant indirect effect through adult SDM constructs of prior positive functioning on functioning at age 30 was also found ($\beta = .05$, $p < .001$). This indirect effect was primarily through skills for interaction, prosocial socialization, and belief in the moral order. When a direct path from pro-alcohol beliefs to positive functioning was added to this model it was significant but *positive*; however, the zero-order correlation between these variables was −.15, suggesting that the positive path coefficient reflected a suppression effect (MacKinnon, Krull, & Lockwood, 2000). A direct path added from

alcohol-use socialization to positive functioning was not significant. These paths were not included in the final model. The two-outcome model, then, indicated mostly specific prediction of AUD symptoms from alcohol-specific social development factors, and specific prediction of positive functioning from prosocial factors. This model had acceptable fit with the data: $\chi^2(184) = 831.52$, CFI = .93, RMSEA =.07.

Discussion

This study sought to contribute to a better understanding of social development in the young adult years, a distinctive and consequential stage of development, but for which there is little research relative to childhood and adolescence (Arnett, 2000, 2005; Aseltine & Gore, 2005; Eitle et al., 2010). The social development model, a theory specifying etiological pathways to antisocial and prosocial outcomes, was largely supported by analyses at age 27. Prosocial and antisocial pathways including opportunities, involvement, rewards-costs, bonding, and beliefs indicated highly significant sequential links among these constructs as hypothesized by the SDM. The modeling of latent factors for “prosocial socialization” and “alcohol-use socialization” – to capture the common variance in respective SDM processes – provided acceptable overall fit of the model to the data. The hypothesized pathway from skills for interaction to alcohol-use socialization was not significant, suggesting that such skills (as measured broadly here) were not sufficient to diminish the influence of alcohol-specific socialization factors.

Modeling analyses found significant stability in AUD symptoms from age 21 to age 30. However, accounting for this stability, social development processes at age 27 in addition to gender played an important role in partially mediating the zero-order association, reducing the bivariate relationship by 27%. This finding suggests that regardless of AUD symptoms on entry into young adulthood, one’s social environment in the late 20s can significantly increase or decrease those symptoms by age 30. It is worth reiterating that the late 20s may represent an important window after which substantial changes in drinking behavior become relatively rare (Johnstone et al., 1996). Further, SDM pathways distinguishing between prosocial and antisocial mechanisms highlighted the importance of alcohol-specific influences on AUD symptoms. Alcohol-use socialization and belief in pro-alcohol values were key mediating mechanisms linking prior AUD symptoms with symptoms at age 30, whereas prosocial factors played little role. One implication of these results is that time spent in positive social environments and having prosocial beliefs may do little to counteract the effects of young adult socialization experiences involving heavy alcohol use and beliefs favorable to heavy alcohol use.

Although the prosocial pathway had little role in predicting AUD symptoms, it played an important role in predicting positive functioning at age 30. A significant indirect effect of prior positive functioning on later positive functioning was found over and above the direct effect. The significant mediated pathways were through skills, prosocial socialization, and belief in the moral order. One path, from pro-alcohol values to positive functioning, was significant but its sign was positive and opposite from the zero-order correlation. This suggested a suppression effect due to the correlation of this predictor with other predictors in the model, particularly prosocial beliefs. This path was omitted from the final model. There

were no other significant pathways indicating that alcohol-use socialization worked to diminish positive functioning. Together, these results suggest specific pathways of prediction in young adulthood such that the mechanisms influencing positive functioning – broadly defined as constructive engagement, civic engagement, physical exercise, and lack of depressive symptoms – were primarily prosocial; whereas the mechanisms influencing AUD symptoms were specific to alcohol.

While we hypothesized stronger content-specific effects (prosocial pathways predicting positive functioning and alcohol-specific pathways predicting AUD symptoms), we were nonetheless surprised that no crossover effects emerged. In prior tests of the SDM using similar methodology, prosocial beliefs were found to be protective against substance use and misuse, violent behavior, and broad measures of antisocial behavior in adolescence (E. C. Brown et al., 2005; Catalano et al., 1996; 2005; Choi et al., 2005; Huang et al., 2001; Lonczak et al., 2001). One possible explanation for finding no crossover effects at ages 27 and 30 is that young adults, compared with earlier ages, may have more opportunity to separate their social experiences so that associating with heavy alcohol users does not significantly disrupt those who were functioning well in other domains of life, and prosocial influences do not significantly deter those with alcohol problems. In contrast, among adolescents it may be more difficult to separate social experiences because one's peers (and often siblings) are more likely to live in the same neighborhoods, attend the same schools, and go to the same social gatherings so that their influence, whether prosocial or antisocial, is a more consistent presence. A question for future research may be to consider the extent to which young adults socialize with those with whom they work or attend school. If this speculation is correct, more crossover effects would be expected among those with more overlap in their peer groups and co-workers or schoolmates. Conversely, a continued lack of crossover effects may signal more psychological compartmentalization between alcohol use and positive functioning than is found at younger ages. Future research should also consider the role of specific life domains, and particularly how their influence may shift with age. For example, how does the relative influence of coworkers versus one's spouse or partner evolve over the life course, and how does this affect the pathways of the SDM?

This study was limited to a sample originally drawn from students in urban public schools overrepresenting high-crime neighborhoods, likely placing them at higher risk than nationally representative samples. Consistent with tests of the SDM across other populations, we would expect higher risk to result in higher *rates* of problem outcomes like AUD, but not to significantly alter the *covariances* among predictors (E. C. Brown et al., 2005; Catalano et al., 2005; Choi et al., 2005; Fleming et al., 2002; Kosterman et al., 2004), and hence for the model tests reported here to have broad application. However, tests of the SDM to confirm the generalizability of findings across different populations remain important. The study was also limited to self-report measures, with the possibility of differential underreporting or overreporting of behaviors. However, we have administered private and confidential surveys with this sample over several years and such methods are likely to provide valid self-report data (Hindelang, Hirschi, & Weis, 1981). One measure (perceived alcohol opportunities) had moderate reliability of .66. Because this study sought to incorporate broad measures assessing diverse domains of life (peer, work, family, etc.),

there was some diminishment of internal consistency for this scale. All other reliability coefficients were greater than .78.

As noted, the initial measured variable model did not fit the data well. Tests of the SDM have consistently found measures of opportunities, involvement, rewards-costs, and bonding to be highly correlated, and that modeling them as separate constructs reduces model fit. While it is important for etiological understanding and intervention development to conceptualize and illustrate these socialization components in tested models, they tend to form one higher order factor empirically. This may suggest that the socialization processes by which young adults perceive opportunities, become involved with others, perceive rewards and costs of that involvement, and become bonded to others are highly intertwined, and that distinctions are difficult to observe in self-report surveys as were used in this study. The latent variable model tested here therefore incorporated these socialization processes, if not the successive links between constructs, and also better represented the covariance structure analytically, resulting in acceptable model fit.

This study reinforces the importance of testing etiological theories throughout the life course to better inform prevention efforts addressing risks across development. Tests of the SDM at age 27 suggested that this age is not too late for interventions targeting adult social development to help diminish AUD symptoms by age 30. Specifically, continuation of AUD symptoms from age 21 to age 30 was partially mediated through social developmental experiences, and those experiences can be addressed by preventive interventions to help disrupt existing symptoms. Alcohol-specific factors such as reducing perceived opportunities or rewards for heavy alcohol use or challenging beliefs accepting of drunkenness are likely to be key ingredients of effective adult interventions, according to our analyses. Interventions that improve prosocial environments and beliefs in young adulthood should increase positive functioning, but our findings suggest little expectation of crossover effects on AUD symptoms.

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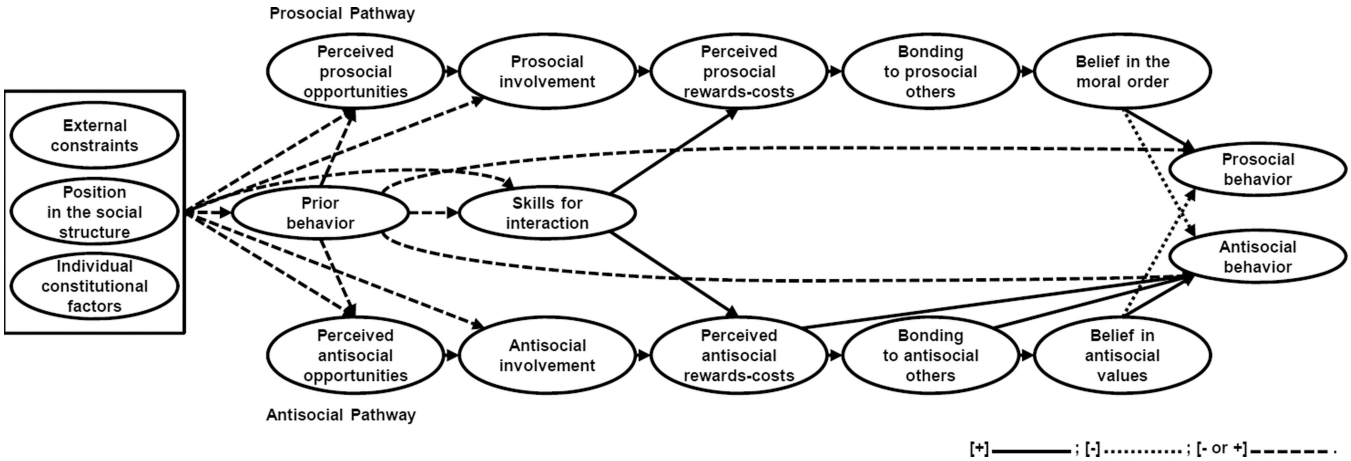

Figure 1.

The social development model. Solid lines indicate positive associations; dotted lines indicate negative associations; dashed lines indicate positive or negative associations depending on the valence of specific behaviors or processes involved.

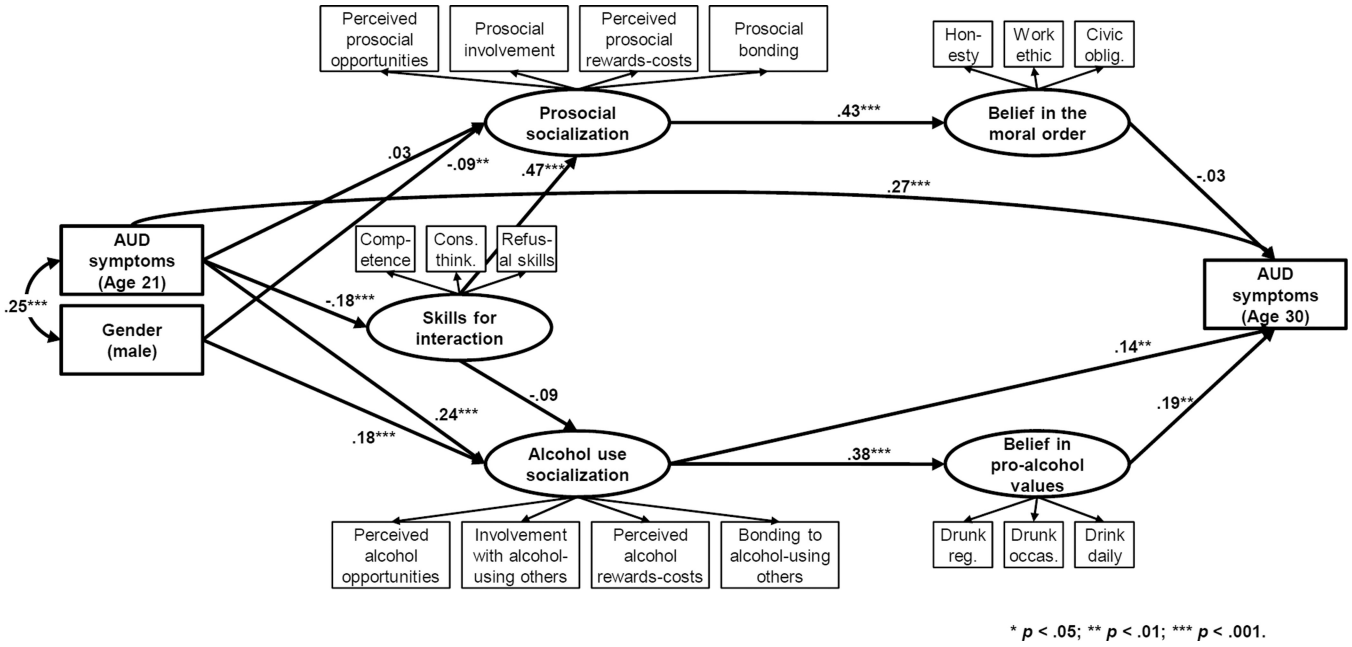

Figure 2.

Latent variable model predicting alcohol use disorder symptoms with pathways hypothesized by the social development model. Significant correlations between residuals of corresponding prosocial and alcohol constructs are included in the model but not shown for clarity. Measures are described in Table 1. χ^2 (151) = 723.60, CFI = .94, RMSEA =.07.

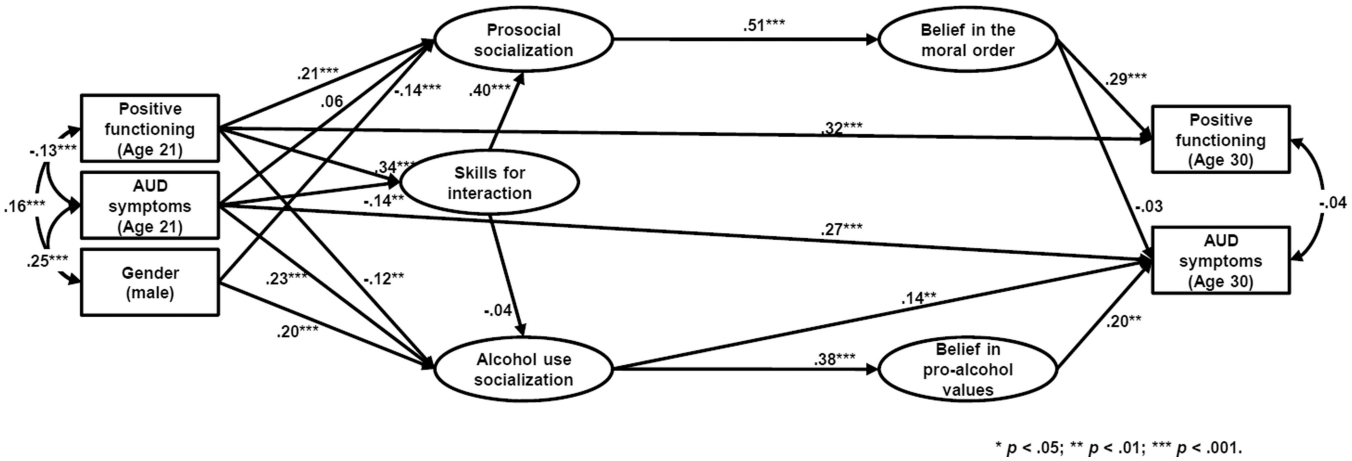

Figure 3.

Two-outcome latent variable model predicting alcohol use disorder symptoms and positive functioning with pathways hypothesized by the social development model. Significant correlations between residuals of corresponding prosocial and alcohol constructs are included in the model but not shown for clarity. χ^2 (184) = 831.52, CFI = .93, RMSEA =.07.

Table 1

Social Development Model Measures

Construct	Description	Number of items	Reliability α	Example
Skills for interaction	Interpersonal competence, consequential thinking, and refusal skills for antisocial opportunities	30	.90	How good are you at helping a close companion cope with family or roommate problems?
Perceived prosocial opportunities	Perceived opportunities for involvement with prosocial others or in prosocial settings	48	.80	To what extent are community centers available in your community to provide activities for people your age?
Prosocial involvement	Actual involvement with prosocial others or in prosocial settings	25	.80	How often do you interact or talk with your partner? [weighted by partner's engagement in prosocial activities]
Perceived prosocial rewards-costs	Perceived consequences for involvement with prosocial others or in prosocial settings	29	.87	I gain a sense of accomplishment from my job.
Prosocial bonding	Attachment and commitment to prosocial others and settings	23	.85	Continuing or completing my education is important to me.
Belief in the moral order	Prosocial values including honesty, work ethic, and civic obligations	15	.79	You can make it in life without having to cheat.
Perceived alcohol opportunities	Perceived opportunities for involvement with others who drink alcohol heavily	10	.66	Does your closest friend drink alcohol heavily?
Involvement with alcohol-using others	Actual involvement with others who drink alcohol heavily	11	.82	How often do you interact or talk with your coworkers? [weighted by coworkers' heavy alcohol use]
Perceived alcohol rewards-costs	Perceived rewards and costs for involvement with alcohol or with others who drink alcohol heavily	20	.84	Do you think that drinking alcohol makes people worry less?
Bonding to alcohol-using others	Attachment and commitment to others who drink alcohol heavily	7	.79	How close is your relationship with this friend? [weighted by friend's heavy alcohol use]
Belief in pro-alcohol values	Values accepting of heavy alcohol use (getting drunk regularly or occasionally, drinking daily)	3	.78	Do you think it is okay for adults to get drunk once or twice each weekend?

Table 2

Correlations Between Measures

Construct	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21
Measured variables																				
1. Gender (male)	.03	−.05	−.05	−.10*	−.11*	−.28*	.22*	.20*	.28*	.16*	.29*	.25*	.19*	.16*	.08*	.03	−.10*	−.39*	.22*	.34*
2. Skills for interaction		.29*	.31*	.30*	.33*	.31*	−.08*	.00	−.07*	.00	−.06	−.11*	−.07	.20*	.24*	--	--	--	--	--
3. Pro. opportunities			.81*	.85*	.86*	.26*	−.06	−.01	−.08*	−.05	−.11*	−.05	−.03	.31*	.35*	--	--	--	--	--
4. Pro. involvement				.86*	.84*	.26*	−.08*	.05	−.09*	−.04	−.12*	−.06	−.05	.29*	.36*	--	--	--	--	--
5. Pro. rewards-costs					.92*	.33*	−.13*	−.06	−.12*	−.07*	−.16*	−.08*	−.06	.31*	.39*	--	--	--	--	--
6. Pro. bonding						.33*	−.10*	−.02	−.10*	−.03	−.14*	−.09*	−.08*	.30*	.35*	--	--	--	--	--
7. Pro. beliefs							−.27*	−.25*	−.37*	−.24*	−.43*	−.22*	−.22*	.10*	.19*	--	--	--	--	--
8. Alc. opportunities								.88*	.80*	.83*	.32*	.30*	.31*	−.12*	−.13*	--	--	--	--	--
9. Alc. involvement									.80*	.86*	.29*	.25*	.28*	−.08*	−.08*	--	--	--	--	--
10. Alc. rewards-costs										.89*	.48*	.31*	.32*	−.12*	−.11*	--	--	--	--	--
11. Alc. bonding											.31*	.24*	.27*	−.13*	−.11*	--	--	--	--	--
12. Alc. beliefs												.29*	.31*	−.06	−.12*	--	--	--	--	--
13. AUD symps (age 21)													.37*	−.13*	−.05	−.18*	−.08*	−.29*	.29*	.33*
14. AUD symps (age 30)														−.06	−.10*	−.16*	−.06	−.30*	.31*	.36*
15. Pos. func. (age 21)															.37*	.37*	.32*	.15*	−.13*	−.09*
16. Pos. func. (age 30)																.35*	.39*	.27*	−.11*	−.15*
Latent variables																				
17. Skills for interaction																	.46*	.64*	−.10*	−.16*
18. Pro. socialization																		.47*	−.08*	−.19*
19. Pro. beliefs																			−.38*	−.65*
20. Alc. socialization																				.43*
21. Alc. beliefs																				

Notes. Pro. = Prosocial; Alc. = Alcohol; AUD symps = Alcohol use disorder symptoms; Pos. func. = Positive functioning.

*$p < .05$.