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Antisocial Peer Affiliation and Externalizing Disorders in the Transition From Adolescence to Young Adulthood: Selection Versus Socialization Effects

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Prior research has demonstrated both socialization and selection effects for the relationship between antisocial peer affiliation and externalizing problems in adolescence. Less research has evaluated such effects postadolescence. In this study, a cross-lagged panel analysis was used to evaluate the extent of *socialization* (i.e., the effect of antisocial peer affiliation on subsequent externalizing disorders) and *selection* (i.e., the effect of externalizing disorders on subsequent antisocial peer affiliation) in the prospective relationships between antisocial peer affiliation and externalizing disorders from adolescence through young adulthood. Data from a community sample of 2,769 individuals (52% female) with assessments at ages 17, 20, 24, and 29 were used. Analyses with a latent externalizing measure (estimated using clinical symptom counts of nicotine dependence, alcohol use disorder, illicit drug use disorder, and adult antisocial behavior) and self-reported antisocial peer affiliation revealed significantly stronger socialization effects from age 17 to 20, followed by significantly stronger *selection* effects from age 20 to 24 and 24 to 29. To better understand the impact of college experience, moderation by college status was evaluated at each developmental transition. Results were generally consistent for those who were in or were not in college. Results suggest selection effects are more important in later developmental periods than earlier periods, particularly in relation to an overall liability toward externalizing disorders, likely due to more freedom in peer selection postadolescence.

Keywords: adolescence, antisocial peers, externalizing disorders, peer relationships, young adulthood

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Externalizing behavior refers to undersocialized behavior broadly and is often characterized as acting out or acting aggressively. Particularly problematic externalizing behavior generally refers to engaging in delinquent or illegal activity, such as adolescent substance use, as well as more disordered psychopathology, such as meeting a clinical threshold for childhood disruptive disorders or substance use disorders (Iacono, Malone, & McGue, 2008). Research has long shown a relationship between affiliation to deviant peers and adolescent externalizing problems (Brendgen, Vitaro, & Bukowski, 2000; Dishion, Véronneau, & Myers, 2010; Gardner & Steinberg, 2005), including substance use and substance use disorders (Curran, Stice, & Chassin, 1997; Dishion & Owen, 2002). Recent research has shown peer affiliation and externalizing problems are related through both processes of selection as well as socialization (Monahan, Steinberg, & Cauffman, 2009; Walden, McGue, Iacono, Burt, & Elkins, 2004; Samek, Keyes, Iacono, & McGue, 2013). Selection refers to how an adolescent's current externalizing behavior predicts subsequent antisocial peer affiliation. Socialization refers to how an adolescent's current affiliation to antisocial peers predicts subsequent externalizing behavior (Andrews, Tildesley, Hops, & Li, 2002; Monahan et al., 2009; Curran et al., 1997).

Whereas cross-sectional research is unable to tease apart these effects, a growing number of longitudinal studies have demonstrated reciprocity (i.e., both socialization and selection effects) in the relationship between deviant peer affiliation and adolescent externalizing behaviors (Curran et al., 1997; Kendler et al., 2007; van Ryzin, Fosco, & Dishion, 2012; van Ryzin & Dishion, 2014). For example, using latent growth curve modeling, Curran et al. (1997) found that both the initial status of peer alcohol use predicted subsequent increase of adolescent alcohol use and the initial status of adolescent alcohol use predicted subsequent increase of peer alcohol use, evidencing reciprocal selection and socialization

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influences. Other research has shown that after accounting for earlier deviant peer affiliation, deviant peer affiliation at ages 16–17 mediated the association between substance use at age 14 and subsequent substance dependence at age 19 (van Ryzin & Dishion, 2014), evidencing a socialization influence above and beyond the effect of a selection influence in mid-to-late adolescence.

On the other hand, limited research has systematically evaluated the relationship between antisocial peer affiliation and externalizing problems postadolescence. One exception is a study conducted by Monahan et al. (2009), in which socialization and selection were evaluated in the transition from adolescence into young adulthood (age 14 to 22). Using a cross-lagged, structural equation analysis, these authors showed that while selection and socialization effects were both evident from ages 14 to 15 (such that both peer affiliation predicted subsequent antisocial behavior and antisocial behavior predicted subsequent peer affiliation), selection effects were not evident past age 16; only socialization effects were evident from ages 16 through 20. From ages 20 to 22, there was no evidence to support either socialization or selection. This overall pattern of results suggests selection may be less relevant past middle adolescence and that the influence of antisocial peer affiliation may be less relevant to the development of young adult antisocial behavior compared to adolescent antisocial behavior.

These results are somewhat at odds with developmental theory suggesting selection effects should become stronger in the transition of adolescence into young adulthood, as individuals have more freedom in selecting environments of their own choosing the older they become (e.g., see Scarr & McCartney, 1983). Monahan and colleagues (2009) argued that their findings demonstrate that peer influences may operate more in line with a socialization hypothesis in late adolescence because individuals have generally already sorted and established themselves into peer groups by this time period (thus there may be limited change in antisocial peer affiliation after middle adolescence). Additionally, Monahan et al. noted that peer influences (both selection and socialization) are likely less salient in early adulthood because individuals are more mature and thus more resistant to peer pressure as a result of this maturity.

On the other hand, the transition into young adulthood is often accompanied by a marked increase in binge drinking and other risky behavior (Esser et al., 2014; Schulenberg & Maggs, 2002) particularly among college students (Substance Abuse & Mental Health Services Administration (SAMHSA), 2014; Slutske, 2005; Schulenberg & Patrick, 2012; White, Labouvie, & Papadaratsakis, 2005). Thus, it seems somewhat odd that peer influences would not be salient in the development of antisocial behavior at this time. It is important to note that the Monahan et al. (2009) sample included individuals who were adjudicated for serious crime and the sample was predominately male. Thus, it is unclear if results will hold up in population-based studies, when increases in antisocial behavior are normative in the transition to young adulthood, and whether results are consistent for both males and females. Also, Monahan et al. evaluated this developmental process through age 22; it is unclear if this pattern continues into later young adulthood (i.e., through age 30 when antisocial behavior and problematic substance use decreases rapidly (Loeber et al., 2012; Moffitt, 1993; SAMHSA, 2014) or whether socialization or selection may again become relevant to explaining the link between deviant peer affiliation and externalizing disorder development after age 22.

Following prior developmental theory predicting a shift from more passive to active environment selection based on unique genetically influenced traits and interests (Scarr & McCartney, 1983), we expect to see more influence of socialization than selection in adolescence and more influence of selection than socialization in early and later young adulthood. This is consistent with studies demonstrating that individuals often become more resistant to antisocial peers over time (Albert, Chein, & Steinberg, 2013; Gardner & Steinberg, 2005; Sumter, Bokhorst, Steinberg, & Westenberg, 2009) and fits with developmental theory suggesting that people are more likely to actively select environments as they reach adulthood, in part because of increasing freedom to do so (Scarr & McCartney, 1983).

On the other hand, peers may have unique effects on substance use and related externalizing behavior for college students in particular. It has long been demonstrated that college students use more substances (particularly alcohol) relative to their non-college-attending peers (SAMHSA, 2014; Schulenberg & Patrick, 2012; Schulenberg & Maggs, 2002; Slutske. 2005; White et al., 2005). College students are also often exposed to new peers in classrooms, on campus, and/or in conjunction with new living arrangements (e.g., dorm or off-campus apartment). Combined with the notion that college students in particular are more embedded in a culture of excessive drinking or "partying" (e.g., proximity to bars, marketing of drink specials toward college students, frequency of Greek and/or other parties, etc.), and that they are often exposed to new peers (on campus, in the classroom, and possibly in new living arrangements), we hypothesized that antisocial peer socialization processes may be more relevant to the development of externalizing problems and substance use for those in college compared to those not in college.

Taken together, we aimed to extend prior research on the topic of peer influences on substance use and externalizing problems by utilizing a cross-lagged panel design to test the prospective relationships between antisocial peer affiliation and externalizing disorders (nicotine dependence, alcohol use disorder, illicit drug use disorder, and adult antisocial behavior clinical symptom counts) at ages 17, 20, 24, and 29. We also evaluated differences in how college enrollment impacts the relationship between deviant peer affiliation and the development of externalizing disorders in adolescence through young adulthood. Guided by prior developmental theory (Scarr & McCartney, 1983), we expected to see stronger evidence for socialization effects earlier in time (i.e., age 17 to 20) followed by stronger effects for selection later in time (i.e., age 20 to 24 and age 24 to 29). As college experiences include a transition into new environments and new peer affiliations, we also hypothesized that socialization effects may be more apparent for those enrolled in college versus not enrolled in college.

Method

Participants

Data from the Minnesota Twin and Family Study (MTFS) were used. MTFS is a cohort-sequential, longitudinal study designed to evaluate the etiology of substance use and related externalizing behaviors (see Iacono, Carlson, Taylor, Elkins, & McGue, 1999

for a complete overview). Two cohorts of twins and their parents were included: the younger cohort, first assessed at age 11, and the older cohort, first assessed at age 17. Twins and parents were recruited from publically available birth certificates (years born 1972–1984); 90% of twin families were located and ~80% of those located and meeting study eligibility criteria participated. To be eligible, families had to live within a day's drive to the university laboratory, and children could not have a mental or physical handicap that impaired participation at assessments. Follow-up assessments were completed through age 29 for both cohorts, resulting in overlapping assessments of the younger and older cohort at ages 17, 20, 24, and 29. Participation rates across follow-up assessments ranged from 88–93% (more details provided in the Missing Data section, below).

In total, this sample includes 2,769 individuals (52% female) from 1,382 twin pairs (65% monozygotic, 35% dizygotic pairs, including 5 triplets), using data from the age 17 (*M* age = 17.8, *SD* = .69), age 20 (*M* age = 21.1, *SD* = .82), age 24 (*M* age = 25.0, *SD* = .90), and age 29 (*M* age = 29.4, *SD* = .67) assessments. Consistent with state demographics in the relevant birth years, nearly all participants were of European ancestry (96%), however, there was considerable diversity in other aspects, including socioeconomic status. The highest education completed for the majority of parents was a high school diploma or equivalent (63.5% for fathers, and 62.6% for mothers); 28.5% of fathers and 25.1% of mothers earned at least a BA/BS degree. At the intake assessment (assessment years ranged from 1990–1996), the median household income was $45,001 to $50,000. Twenty-five percent of families earned ≤$40,000 and 25% of families earned ≥ $60,001; 7% earned ≤ $20,000, meeting federal poverty guidelines for a family of four in the relevant data collection years (U.S. Census Bureau, 1996). Although the majority of rearing parents (79.3%) were married at the original intake assessment, 16.7% were divorced, 2.7% were never married and 1.3% were separated. The MTFS also included families from both rural (40%) and urban (60%) communities (Legrand, Keyes, McGue, Iacono, & Krueger, 2008), as well as approximately equivalent numbers of males (48%) and females (52%). As outlined below, externalizing disorders and antisocial peer affiliation were obtained in a consistent manner within each age-assessment period.

Measures

Externalizing problems. Clinical symptom counts of nicotine dependence, alcohol use disorder, illicit drug use disorder, and adult antisocial behavior were assessed at ages 17, 20, 24, and 29. SUDs are broadly characterized by social impairment and distress (e.g., desire/unable to cut down, continued use despite problems, marked tolerance). *DSM–III–R* nicotine dependence symptom counts range from 0 to 7. Alcohol and illicit drug use disorder included a best estimate of both abuse and dependence symptom counts for a total range of 0 to 10 *DSM–III–R* symptoms. Adult antisocial behavior is characterized by reckless disregard for others; meeting a threshold of both adult antisocial behavior in adulthood and conduct disorder prior to age 15 is required to meet the diagnosis for Antisocial Personality Disorder (American Psychological Association, 1987, 2013). Here, we are using the adult antisocial behavior symptom counts alone (e.g., irritable and aggressive), with a total range of 0 to 10 *DSM–III–R* symptoms at each assessment.

All substance use disorder symptoms were assessed using the Substance Abuse Module of the Composite International Diagnostic Interview (Robins et al., 1988). Adult antisocial behavior symptoms were assessed using an interview comparable to the Structured Clinical Interview for *DSM–III–R* Axis II (Spitzer, Williams, & Gibbon, 1987). Clinical symptoms were reviewed by at least two individuals with advanced training and consensus by both individuals was necessary to assign symptoms. Kappa coefficients indexing diagnostic reliability were > .90 for all substance use disorders and .79 for adult antisocial behavior.

Antisocial peer affiliation. The Friends Self-Report Survey (Burt, McGue, & Iacono, 2009; Walden et al., 2004) was used to assess antisocial peer affiliation at each time point. Participants rated their friendships on a scale of 1 (*All of my friends are like that*) to 4 (*None of my friends are like that*). A 7- to 9-item scale was used to measure affiliation with antisocial peers at each assessment (e.g., "My friends use drugs," "My friends get into trouble with the police"); items were adapted, dropped, or added based on developmental relevance. At age 29, two items were dropped ("My friends drink alcohol or beer," My friends smoke cigarettes or chew tobacco") as having peers that occasionally drink or smoke in later adulthood may not be indicative of antisocial behavior (e.g., getting into trouble with police). A sensitivity analysis showed that scales that excluded versus included these two items at the earlier assessments were almost perfectly correlated (*r*s ranged from .96 to .97, all *p*s < .001). Follow-up analyses also revealed essentially identical results using scales from earlier assessments that excluded versus included these two items.

Median scale internal consistency reliability (α) for antisocial peer affiliation was .88 for 17 year-olds, .83 for 20 year-olds, .83 for 24 year-olds, and .78 for 29 year-olds. Scales were highly correlated across time (*r*s ranged from .48 to .67, also see Table 1), demonstrating moderate stability. To put antisocial peer measures on roughly the same metric as the estimated externalizing disorder latent factors (*M* = 0, *SD* = 1), the antisocial peer scales were standardized (within-assessment) prior to analysis. This is especially useful for purposes of comparison of unstandardized coefficients in cross-lagged panel analyses, described further below.

College status. College status was measured using items from the Social Adjustment Interview at ages 20, 24, and 29. Participants were asked to report on their educational experiences. The highest level of college educational attainment was computed at each assessment in adulthood (1 = *less than high school/GED*, 2 = *high school diploma or GED*, 3 = *some college/business certificate/technical degree*, 4 = *college degree*, 5 = *at least some professional or graduate school*). For example, at age 29, 1.5% of the sample reported earning less than a high school diploma or GED as the highest level of education completed, 10.3% reported a high school diploma/GED, 37.0% reported completing at least some college, 26.6% had completed a college degree and 14.6% reported some professional/graduate school (10% were missing this information due to nonparticipation or failure to complete the associated interview or questions at the age 29 assessment).

Because we were interested in whether socialization versus selection paths were moderated by college status, due to the new peers and friends typically encountered upon college entrance, we examined the effect of having initiated college during the time that

Table 1
Correlations Among Raw Study Variables for Males (n = 1,333) and Females (n = 1,436)

Variable	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20
1. Age 17 NIC	—	.48	.47	.54	.44	.58	.19	.23	.30	.24	.57	.16	.23	.28	.30	.53	.17	.16	.23	.34
2. Age 17 ALC	.53	—	.55	.58	.37	.31	.31	.23	.25	.23	.35	.21	.22	.35	.25	.30	.20	.18	.20	.27
3. Age 17 DRG	.52	.57	—	.55	.35	.20	.20	.32	.24	.19	.30	.17	.25	.31	.23	.26	.19	.28	.23	.27
4. Age 17 AAB	.39	.40	.45	—	.51	.36	.27	.27	.38	.32	.40	.23	.27	.44	.34	.36	.18	.18	.31	.35
5. Age 17 Antisocial peers	.52	.54	.52	.63	—	.38	.27	.25	.38	.55	.41	.21	.22	.29	.47	.37	.17	.15	.23	.41
6. Age 20 NIC	.53	.35	.32	.23	.45	—	.29	.31	.36	.35	.68	.25	.23	.23	.34	.59	.20	.19	.24	.35
7. Age 20 ALC	.28	.48	.26	.23	.42	.42	—	.35	.38	.31	.28	.33	.21	.23	.24	.23	.19	.09	.19	.26
8. Age 20 DRG	.35	.38	.48	.24	.44	.44	.46	—	.50	.28	.31	.26	.48	.29	.25	.27	.24	.29	.26	.29
9. Age 20 AAB	.33	.37	.30	.25	.46	.42	.51	.49	—	.38	.39	.31	.34	.43	.34	.35	.25	.28	.35	.35
10. Age 20 Antisocial peers	.32	.37	.31	.26	.58	.42	.50	.41	.47	—	.32	.27	.27	.35	.59	.26	.20	.21	.29	.51
11. Age 24 NIC	.46	.30	.25	.18	.35	.69	.30	.30	.33	.29	—	.33	.30	.36	.38	.75	.31	.26	.29	.39
12. Age 24 ALC	.26	.39	.22	.18	.37	.30	.49	.28	.36	.36	.35	—	.33	.42	.35	.29	.37	.21	.24	.28
13. Age 24 DRG	.31	.34	.39	.21	.36	.32	.32	.57	.35	.32	.33	.33	—	.41	.33	.25	.25	.51	.31	.31
14. Age 24 AAB	.34	.40	.31	.25	.45	.34	.42	.37	.50	.39	.35	.45	.48	—	.37	.32	.34	.33	.52	.40
15. Age 24 Antisocial peers	.28	.33	.28	.22	.55	.37	.38	.29	.40	.70	.34	.43	.34	.42	—	.35	.23	.22	.25	.63
16. Age 29 NIC	.41	.24	.22	.16	.31	.64	.26	.29	.33	.30	.68	.27	.30	.30	.30	—	.32	.26	.32	.40
17. Age 29 ALC	.23	.28	.16	.14	.26	.30	.40	.32	.34	.30	.24	.45	.25	.32	.31	.32	—	.33	.36	.29
18. Age 29 DRG	.29	.26	.31	.17	.23	.30	.31	.51	.33	.30	.22	.25	.55	.30	.31	.30	.42	—	.46	.29
19. Age 29 AAB	.29	.30	.25	.23	.34	.34	.35	.33	.46	.36	.26	.34	.34	.49	.38	.32	.50	.46	—	.37
20. Age 29 Antisocial peers	.29	.35	.30	.24	.48	.30	.40	.33	.38	.60	.27	.37	.31	.38	.64	.29	.34	.33	.40	—

Note. NIC = Nicotine Dependence Symptom Count; ALC = Alcohol Use Disorder Symptom Count; DRG = Illicit Drug Use Disorder Symptom Count; AAB = Adult Antisocial Behavior Symptom Count. Correlations for males are shown below the diagonal, correlations for females are shown above the diagonal. All variables were significant at $p < .05$.

preceded each assessment. Thus, at age 20, just over half the sample reported at least some college experience (n = 1,614, 58% of sample). At age 24, only 280 reported having new college experience since age 20 (i.e., had participated in college for the first time after the age 20 assessment, 10% of sample). Following this same line of logic, at age 29, only those who reported being in college for the first time at age 29 were considered to have a college status; n = 56 met this criteria at age 29 (2% of sample).

Missing Data

Little data were missing for externalizing disorders or reports of antisocial peer affiliation across time (missing cases for externalizing disorders at age 17: 5.5%, at age 20: 11.5%, at age 24: 10%, at age 29: 9.8%; missing cases for antisocial peer affiliation at age 17: 15.9%, at age 20: 15.6%, at age 24: 17.4%, at age 29: 12.7%). This was due to nonparticipation at each assessment (6.9–11.5% across assessments) or failure to complete self-report surveys (2.9–9.0% across assessments). To evaluate attrition effects, we compared mean differences in symptoms of externalizing disorders at age 17 for those who did or did not complete adult assessments. Those who participated in adult assessments had slightly fewer externalizing symptoms at age 17 than those who did not participate, but the effect sizes were small with average Cohen's ds = −0.21, −0.16, and −0.03 at ages 20, 24, and 29, respectively, indicating little evidence of attrition effects.

Analysis Plan

Our primary goal was to examine selection and socialization effects through the use of prospective data analytic methods, specifically, a cross-lagged panel design (for an overview, see Berrington, Smith, & Sturgis, 2006; Kenny & Harackiewicz, 1979). Preliminary analyses were conducted to compare cross-lagged results across clinical symptom counts of nicotine dependence, alcohol use disorder, illicit drug use disorder, and adult antisocial behavior. Based on the similarity of results and high correlations between symptom count variables (see Table 1), we then estimated latent factors of externalizing disorders at ages 17, 20, 24, and 29 using clinical symptom counts of nicotine dependence, alcohol and illicit drug use disorder, and antisocial behavior and evaluated a cross-lagged structural equation model (see Figure 1). In all cross-lagged analyses, the prospective relationship between antisocial peer affiliation and externalizing disorders at ages 17, 20, 24, and 29 was tested, after accounting for autoregressive effects and residual correlations at each time point. All analyses were tested using Mplus, 7.2 (Muthén & Muthén, 1998–2014), using full information maximum likelihood and the maximum likelihood with robust standard errors (MLR) estimator to deal with non-normal distribution of externalizing disorder variables. The CLUSTER specification was used to account for nonindependence of (twin) cases.

Selection effects, referring to the effect of externalizing disorders on subsequent antisocial peer affiliation, are shown in bolded gray (see cross-path 1a, 2a, and 3a in Figure 1). Socialization effects, referring to the effect of antisocial peer affiliation on subsequent externalizing disorders, are shown in bolded black (see cross-path 1b, 2b, and 3b). To determine whether socialization effects were significantly stronger in magnitude in comparison to selection effects or vice versa, socialization and selection paths were constrained to be equivalent in three analyses (for each developmental transition; i.e., age 17–>20, age 20–>24, age 24–>29) and the Satorra-Bentler Chi-Square difference test (used in conjunction with the MLR estimator) was used to measure a significant decrement in model fit between the free and constrained models.

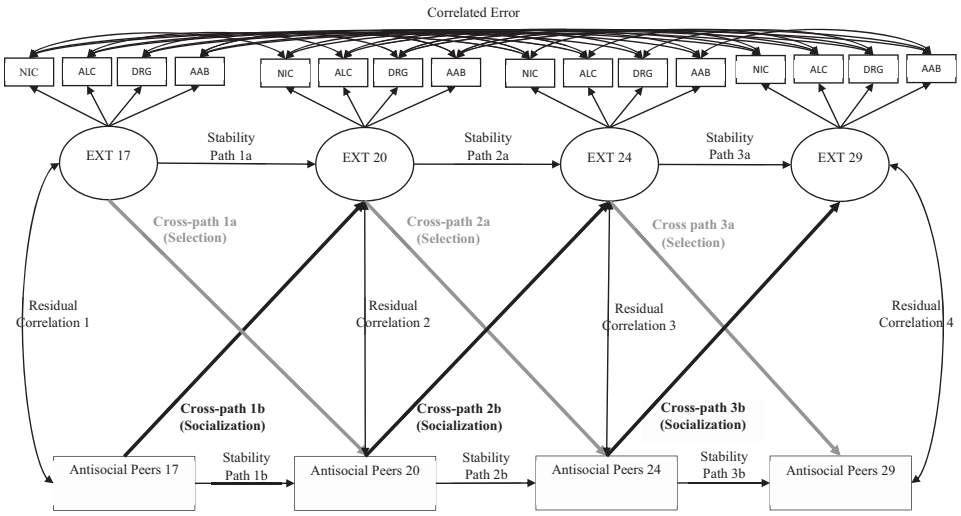

Figure 1. Study model depicting hypotheses. *Note.* This figure shows stability paths, cross-paths, and residual correlations associated with the study model. Circles represent latent factors, squares represent observed indicators. NIC = Nicotine Dependence Symptom Count; ALC = Alcohol Use Disorder Symptom Count; DRG = Illicit Drug Use Disorder Symptom Count; AAB = Adult Antisocial Behavior Symptom Count; EXT = externalizing disorder latent factor. Externalizing disorder indicators (e.g., NIC) were correlated across time (e.g., NIC 17 was correlated with NIC 20, NIC 24, and NIC 29; NIC 20 with NIC 24 and NIC 29; NIC 24 with NIC 29—i.e., correlated errors refer to the same variable across time). Cross-paths are shown in bold for clarity of presentation; cross-paths 1a, 2a, and 3a depict selection hypotheses (shown in bolded gray). Cross-paths 1b, 2b, and 3b depict socialization hypotheses (shown in bolded black).

A second aim was to test whether socialization and selection effects varied as a function of college enrollment status. To evaluate this, we tested the cross-lagged paths from the cross-lagged model (Figure 1) separately for those who had attended any college versus those who had not attended any college. Separate groupings were used for each developmental transition (college status at age 20 was used as a grouping variable for the analyses evaluating socialization vs. selection from age 17 to age 20, new college status at age 24 was used as a grouping variable for the analyses evaluating socialization vs. selection from age 20 to 24, and new college status at age 29 was used as a grouping variable for the analyses evaluating socialization vs. selection from for age 24 to 29). For example, if someone went to college for the first time at age 26, they would be grouped in the "no college experience" groups for the age 17 to age 20 analysis and the age 20 to age 24 analysis, but in the "new college experience" group for the age 24 to age 29 analysis. Significant differences in paths for those with any college versus no college were tested by constraining paths to be equivalent and using the Satorra-Bentler Chi-Square difference test to test for a significant decrement in model fit from the free and constrained models.

Results

Preliminary Analyses

Table 2 shows descriptive statistics on externalizing disorder symptom counts by sex. On average, males had higher mean symptom counts than females, across all externalizing disorders and across time (Cohen's *d* ranged from .13 to .67, *M* = .39). Table 1 shows correlations among all study variables for both males and females. In general, antisocial peer affiliation and externalizing disorders were significantly and moderately to substantially correlated both within and across time, for both males and females.

Table 2
Descriptive Statistics for DSM-IIR Clinical Symptoms for Males (n = 1,333) and Females (n = 1,436)

Variable	***M*** **(*SD*)** symptom count		Cohen's ***d***
	Males	Females	
NIC at age 17	.97 (1.83)	.75 (1.66)	.13
ALC at age 17	.86 (1.78)	.40 (1.24)	.30
DRG at age 17	.65 (1.88)	.29 (1.16)	.23
AAB at age 17	.99 (1.39)	.53 (1.00)	.38
NIC at age 20	1.43 (1.94)	.91 (1.64)	.29
ALC at age 20	1.70 (2.18)	.50 (1.26)	.67
DRG at age 20	.96 (2.05)	.36 (1.31)	.35
AAB at age 20	1.27 (1.28)	.55 (.80)	.67
NIC at age 24	1.32 (1.84)	.92 (1.62)	.23
ALC at age 24	1.56 (2.02)	.60 (1.42)	.55
DRG at age 24	.73 (1.80)	.32 (1.17)	.27
AAB at age 24	1.20 (1.12)	.65 (.85)	.55
NIC at age 29	1.24 (1.78)	.80 (1.53)	.27
ALC at age 29	1.03 (1.80)	.34 (1.09)	.44
DRG at age 29	.61 (1.73)	.22 (1.07)	.27
AAB at age 29	.99 (1.01)	.50 (.73)	.56

Note. NIC = Nicotine Dependence Symptom Count; ALC = Alcohol Use Disorder Symptom Count; DRG = Illicit Drug Use Disorder Symptom Count; AAB = Adult Antisocial Behavior Symptom Count; *M* = Mean; *SD* = Standard Deviation. Cohen's *d* of .2–.3 is considered a small, ~.5 a medium, and .8+ a large effect.

Cross-Lagged Panel Analyses

Figure 2 shows results of each individual externalizing disorder (results for nicotine dependence are shown in Panel A, results for alcohol use disorder are shown in Panel B, results for illicit drug use disorder in Panel C, and results for adult antisocial behavior are shown in Panel D). The stability of antisocial peer affiliation was moderate and stable across time (βs ranged from .59 to .65). The stability of externalizing disorders was smaller in magnitude from ages 17 to 20 (βs ranged from .15 to .46) but stronger and more stable from ages 20 to 24 (βs ranged from .43 to .69) and ages 24 to 29 (βs ranged from .41 to .70).

Across each individual externalizing disorder, results showed that after accounting for the stability of traits and residual associations at each time point, there were stronger socialization than selection effects from ages 17 to 20. For example, the β corresponding to socialization path for nicotine dependence was small-to-moderate in magnitude (β = .24) and significant at $p < .001$, while the β corresponding to the selection path was estimated to be .00 and was not significantly different than zero at $p < .05$ (see Panel A of Figure 2). A similar pattern was found for alcohol use disorder (Panel B), illicit drug use disorder (Panel C) and adult antisocial behavior (Panel D). Following this, constraining the socialization and selection paths to be equivalent from age 17 to 20 for the separate nicotine, alcohol, drugs, and antisocial behavior models resulted in a significant decrement in model fit in all cases (nicotine: $\Delta\chi^2(1) = 37.80$, $p < .001$; alcohol: $\Delta\chi^2(1) = 30.65$, $p < .001$; drugs: $\Delta\chi^2(1) = 50.59$, $p < .001$; adult antisocial behavior: $\Delta\chi^2(1) = 68.47$, $p < .001$), suggesting socialization effects were significantly stronger than selection effects across the board.

On the other hand, and across each individual externalizing disorder, comparing socialization versus selection paths from ages 20 to 24 and age 24 to 29 showed more similarity and reciprocity in effects. For example, for age 24 to age 29 the β corresponding to socialization path for nicotine dependence was small in magnitude (β = .05) but significant at $p < .05$, as was the β corresponding to the selection path (β = .12, $p < .001$). Similar effects were demonstrated for age 24 to 29 (see Panel A of Figure 2). Nonetheless, for nicotine dependence, constraining the socialization and selection pathways to be equivalent from 20 to 24 resulted in a significant decrement of fit ($\Delta\chi^2(1) = 7.15$, $p = .007$), suggesting selection effects were significantly stronger than socialization effects from 20 to 24, as well (see Panel A of Figure 2 for details). However, constraining the socialization and selection pathways to be equivalent 24 to 29 did not result in a significant decrement of fit ($\Delta\chi^2(1) = .67$, $p = .41$, suggesting selection and socialization effects were equivalent in magnitude from 24 to 29 for nicotine dependence.

There was more evidence for reciprocity in socialization vs. selection effects for ages 20 to 24 and 24 to 29 for the other externalizing disorders. Across alcohol use disorder, illicit drug

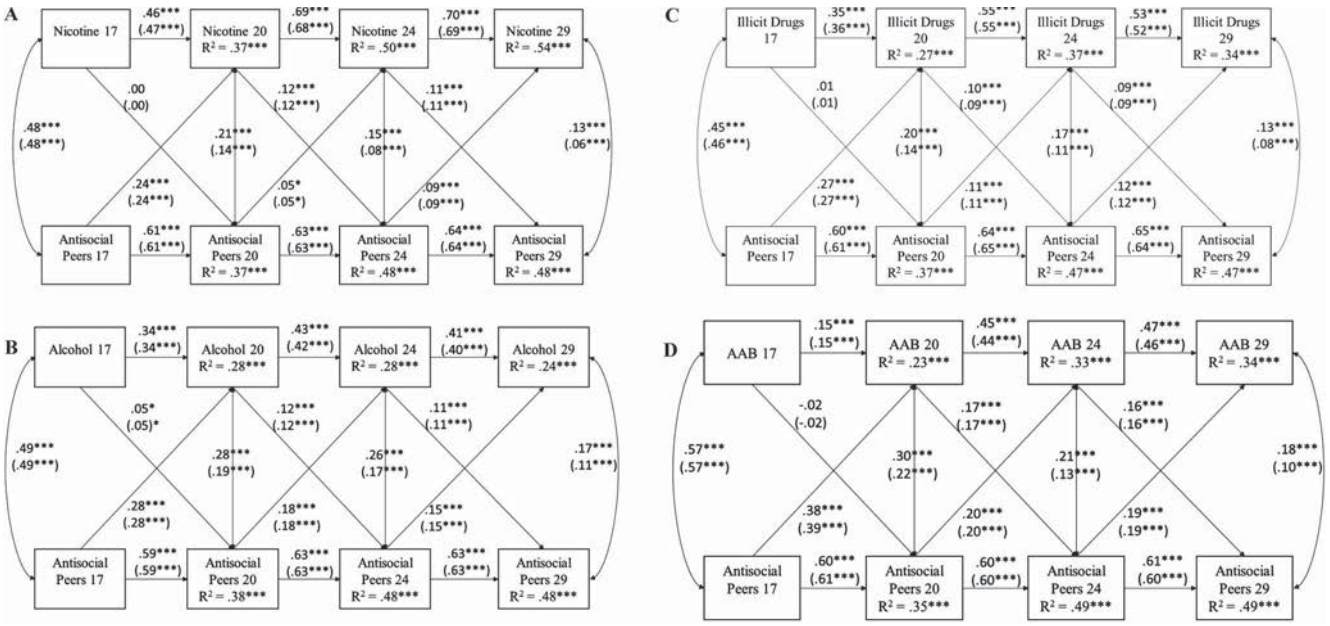

Figure 2. The prospective relationship between antisocial peer affiliation and each indicator of externalizing disorders from adolescence through young adulthood. *Note.* Standardized (unstandardized) coefficients are shown. Circles represent latent factors, squares represent observed indicators. *Selection* effects are paths that begin with each externalizing disorder and predict subsequent antisocial peer affiliation. *Socialization* paths are those that begin with antisocial peer affiliation and predict subsequent externalizing disorders (see Figure 1 for further notation). Panel A shows results for Nicotine Dependence (N = 2,732, fit statistics: $\chi^2(12) = 274.14$, MLR = 1.57, RMSEA = .089, 95% CI_{RMSEA} = .080, .099, CFI = .95, TLI = .89, SRMR = .054). Panel B shows results for Alcohol Use Disorder. Panel C shows results for Illicit Drug Use Disorder. Panel D shows results for Adult Antisocial Behavior. Significance of depicted coefficients is denoted by *** $p < .001$, ** $p < .01$, * $p < .05$, † $p < .10$.

use disorder, and adult antisocial behavior, constraining socialization and selection pathways to be equivalent from age 20 to 24 did not result in a significant decrement to model fit (alcohol: $\Delta\chi^2(1) = 2.62$, $p = .11$; illicit drug use disorder: $\Delta\chi^2(1) = .39$, $p = .53$; adult antisocial behavior: $\Delta\chi^2(1) = 1.16$, $p = .28$). The same was true for constraining socialization and selection paths to be equivalent from age 24 to 29 for each of these externalizing disorder symptom counts (alcohol: $\Delta\chi^2(1) = 1.51$, $p = .22$; illicit drug use disorder: $\Delta\chi^2(1) = .78$, $p = .38$; adult antisocial behavior: $\Delta\chi^2(1) = .54$, $p = .46$). This suggests equivalently strong selection and socialization effects involving antisocial peers and these alcohol use disorder, illicit drug use disorder, and adult antisocial behavior beyond age 20.

It is important to note that both selection and socialization effects were varied in effect size across developmental transition and by disorder. Squaring the standardized path coefficient (β) and dividing by the total predicted variance accounted for (R^2) illustrates the percentage of explained variance accounted for by that pathway. Although large socialization effects from age 17 to 20 were found across the board (nicotine: antisocial peers at age 17 explained 15.5% of the predicted variance of nicotine dependence, 28% of the predicted variance in alcohol use disorder at age 20, 27% of the predicted variance in illicit drug use disorder at age 20, and 62.8% of the predicted variance in antisocial behavior at age 20), generally smaller socialization effects were found beyond age 20. For example, antisocial peers at age 17 explained <1% of the predicted variance in nicotine dependence at age 20, 11.6% of the predicted variance in alcohol use disorder at age 20, 3.3% of the predicted variance in illicit drug use disorder at age 20, and 12.1% of the predicted variance in adult antisocial behavior at age 20. This suggests that in line with expectations, socialization effects may be more relevant from age 17 to 20 compared to age 20 to 24 or 24 to 29 for each of the externalizing disorders.

Cross-Lagged Panel Results for the Latent Externalizing Disorder Factors

As results were similar across each facet of externalizing disorders, and externalizing disorders were highly correlated (as shown in Table 1), we estimated latent factors of externalizing disorders at ages 17, 20, 24, and 29. Figure 3 shows the cross-lagged panel results.

Following the results for each individual externalizing disorder facet, the stability of externalizing disorders from ages 17 to 20 was smaller in effect size (β = .44, $p < .001$) than the stability of externalizing disorders from ages 20 to 24 (β = .83, $p < .001$) and 20 to 29 (β = .82, $p < .001$). Comparatively, the stability of antisocial peer affiliation from ages 17 to 20 was somewhat larger in effect size (β = .58, $p < .001$) than the stability of externalizing disorders from 17 to 20 (reported above) and relatively stable from ages 20 to 24 (β = .52 $p < .001$) and ages 24 to 29 (β = .48, $p < .001$).

After accounting for the stability of externalizing disorders and antisocial peer affiliation over time and the residual correlations between externalizing disorders and antisocial peer at each time point, results showed there were stronger socialization than selection effects from age 17 to 20 (as the path from antisocial peer affiliation at age 17 to externalizing disorders at age 20 was significant and moderate in magnitude and the path from externalizing disorders at age 17 to antisocial peer affiliation at age 20 was not significantly different than zero). Conversely, there were generally stronger selection than socialization effects from age 20 to 24, and from ages 24 to 29 (as the paths from externalizing disorders to subsequent antisocial peer affiliation were significant and moderate in magnitude in both cases, but the paths from antisocial peer affiliation to subsequent externalizing disorders was not significant from zero in either case; see Figure 2 for details).

Constraining these paths to be equivalent resulted in a significant decrement of fit for each developmental transition, suggesting socialization effects were significantly stronger than selection effects from 17 to 20 ($\Delta\chi^2(1) = 41.07$, $p < .001$) and that selection effects were significantly stronger than socialization effects from 20 to 24 ($\Delta\chi^2(1) = 22.38$, $p < .001$) and 24 to 29 ($\Delta\chi^2(1) = 28.07$, $p < .001$). Follow-up tests showed results were consistent across males and females (see online supplementary materials, eFigures 1–2) and across younger and older MTFS cohorts (see online supplementary materials, eFigures 3–4). We also confirmed results were consistent when including family of-origin socioeconomic status as a covariate (see online supplementary materials, eFigures 5) or a moderator of these prospective relationships (see supplementary materials, eFigures 6).

Squaring the standardized path coefficient and dividing by the total predicted variance accounted for (obtained from Figure 1) illustrates the percentage of explained variance in each dependent variable account for by that pathway. Thus, antisocial peers at age 17 explained 25.8% of the predicted variance in externalizing disorders at age 20, a relatively large effect. Comparatively, externalizing disorders at age 17 explained less than 1% of the predicted variance in antisocial peers at age 20. Alternatively, antisocial peers at age 20 explained less than 1% of the predicted variance of externalizing disorders at age 24, whereas externalizing disorders at age 20 explained 13.5% of the predicted variance in antisocial peers at age 24. Similarly, antisocial peers at age 24 explained less than 1% of the predicted variance of the predicted variance of externalizing disorders at age 29, whereas externalizing disorders at age 24 predicted 18.4% of the total predicted variance of antisocial peers at age 29. Thus, there were relatively large socialization effects from age 17 to 20, followed by relatively moderate selection effects from ages 20 to 24 and 24 to 29.

Moderation by College Status

Table 3 shows results for the cross-lagged model by college status. At a descriptive level, socialization paths tended to be stronger in effect size for those with new college experience compared to those without new college experience for each developmental transition, including from ages 17 to 20 ($b_{college} = .39$ vs. $b_{no\ college} = .24$), ages 20 to 24 ($b_{college} = .12$ *vs.* $b_{no\ college} = .05$), and ages 24 to 29 ($b_{college} = .55$ *vs.* $b_{no\ college} = .01$). However, constraining these paths to be equivalent across college versus no college status did not result in a significant decrement of model fit for the age 17 to 20 transition ($\Delta\chi^2(1) = 1.02$, $p = .31$) or the age 20 to 24 transition ($\Delta\chi^2(1) = .92$, $p = .34$), suggesting socialization effects were similar in magnitude across college status during those developmental transitions. Only path coefficients from the age 24 to 29 transition were significantly different ($\Delta\chi^2(1) = 4.61$, $p = .03$). Thus, hypotheses were not supported by

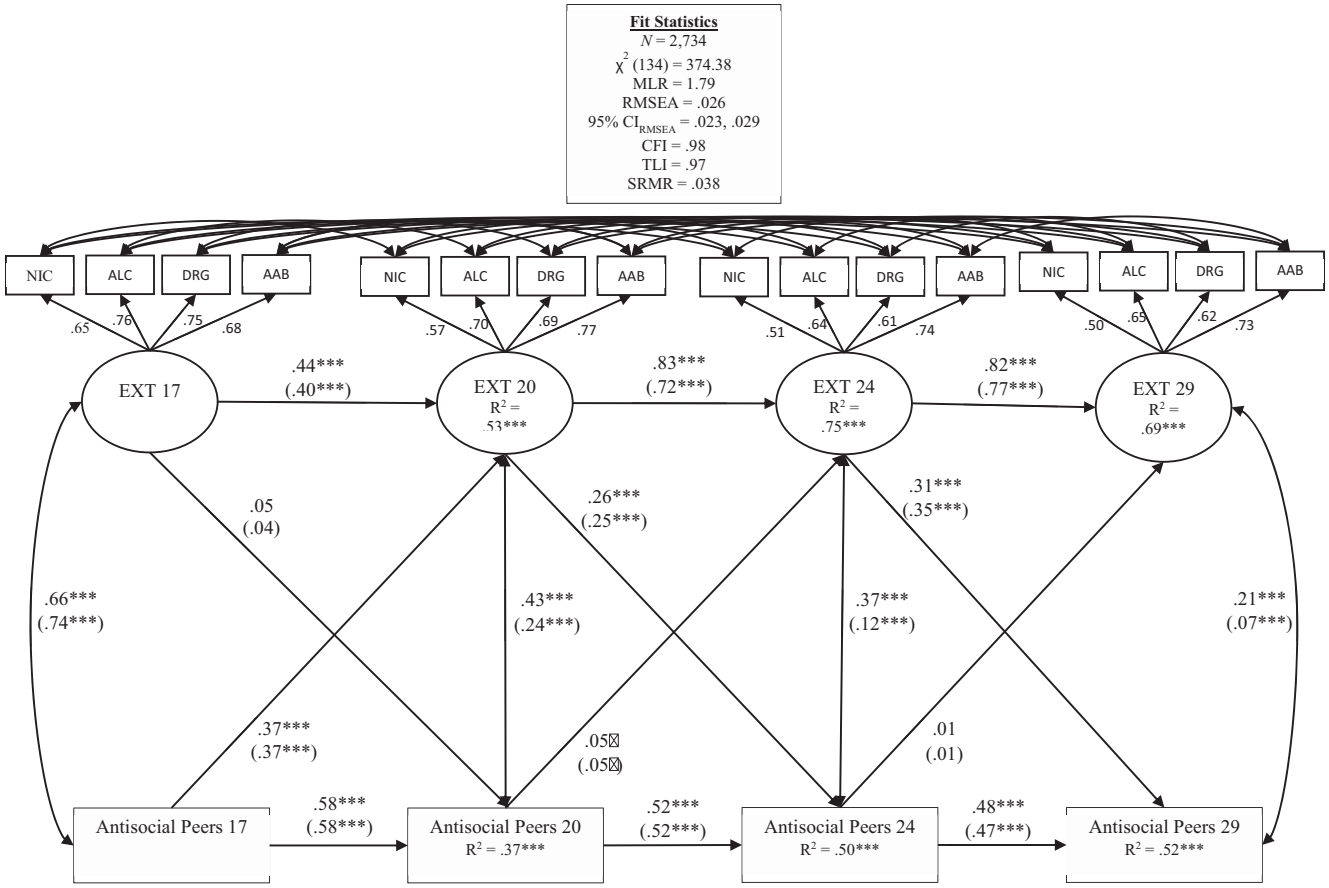

Figure 3. The prospective relationship between antisocial peer affiliation and externalizing disorders from adolescence through young adulthood. *Note.* Standardized (unstandardized) coefficients are shown (except for factor loadings, in which only standardized coefficients are shown for clarity of presentation). Circles represent latent factors, squares represent observed indicators. NIC = Nicotine Dependence Symptom Count; ALC = Alcohol Use Disorder Symptom Count; DRG = Illicit Drug Use Disorder Symptom Count; AAB = Adult Antisocial Behavior Symptom Count; EXT = externalizing disorder latent factor. Externalizing disorder indicators (e.g., NIC at age 17) were correlated within substance/category across time (e.g., NIC at age 17 was correlated with NIC at age 20, NIC at age 24, and NIC at age 29); coefficients not shown for clarity of presentation. *Selection* effects are paths that begin with externalizing disorders and predict subsequent antisocial peer affiliation. *Socialization* paths are those that begin with antisocial peer affiliation and predict subsequent externalizing disorders (see Figure 1 for further notation). Significance of depicted coefficients is denoted by $^{\dagger}\ p < .10$, $^{*}\ p < .05$, $^{**}\ p < .01$, $^{***}\ p < .001$.

except for the age 24 to 29 assessment, and even then caution should be warranted as only 56 people (2% of the sample) met criteria for the new college experience criteria post age 24 (suggesting it was a relatively rare occurrence).

Moreover, across developmental transitions and for both those with any college compared to those with no college, results showed that socialization effects tended to be stronger from age 17 to 20 ($b_{socialization}$ ranged from .24 to .29 vs. $b_{selection}$ ranged from .04 to .10), and that selection effects tended to be stronger from age 20 to 24 ($b_{selection}$ = .27 vs. $_{socialization}$ ranged from .05 to .10), and from 24 to 29 ($b_{selection}$ ranged from .36 to .61 vs. $b_{socialization}$ ranged from .01 to .55). Thus, regardless of college experience, results generally support stronger socialization than selection effects from ages 17 to 20 and stronger selection than socialization effects from ages 20 to 29. Follow-up analyses showed a similar pattern of results across each externalizing disorder facet, as well (see eTable 1 in the online supplementary materials for details).

Discussion

The purpose of this study was to evaluate the processes of socialization and selection as they apply to the associations between antisocial peer affiliation and externalizing problems in the transition from adolescence through young adulthood. We also evaluated whether these socialization and selection processes varied as a result of college experiences. As a reminder, *socialization* refers to how an adolescent's affiliation to antisocial peers predicts subsequent externalizing behavior. *Selection* refers to how an adolescent's externalizing behavior predicts subsequent antisocial

Table 3
Prospective Associations Between Externalizing Disorders and Antisocial Peer Affiliation by College Status

Path	Age 17 → Age 20		Age 20 → Age 24		Age 24 → Age 29	
	Any college (n = 1,614)	No college (n = 521)	New college (n = 280)	No college (n = 1,996)	New college (n = 56)	No college (n = 2,344)
Selection cross path: Externalizing disorders → Antisocial peers	.10 (.04)*	.04 (.05)	.27 (.06)***	.27 (.04)***	.61 (.41)$_{NS}$	.36 (.05)***
$\Delta\chi^2(1\ df)$	.78$_{NS}$		.01$_{NS}$		.47$_{NS}$	
Socialization cross path: Antisocial peers → Externalizing disorders	.39 (.05)***	.24 (.14)†	.12 (.08)	.05 (.03)	.55 (.28)*	.01 (.04)
$\Delta\chi^2(1\ df)$	1.02$_{NS}$		.92$_{NS}$		4.61*	

Note. Showing unstandardized coefficients (standard errors). Cross-path associations are shown for those who had newly attended college compared to those who had not newly attended college for each associated developmental transition (e.g., someone who had gone to college for the first time between the ages 20 and 24 assessments would be categorized in the new college group for the age 20 to 24 analysis, and in the no college group for the age 17 to 20 analysis, as well as the no college group for the age 24 to 29 analysis). To test for significant differences by college status grouping, paths were constrained to be equivalent and the Satorra-Bentler Chi-Square Difference Test ($\Delta\chi^2$) on 1 degree of freedom (*df*) was used to test for significant decrement in fit between free and constrained models. Significance is denoted by $^{***}p < .001$, $^{**}p < .01$, $^{*}p < .05$, $^{\dagger}p < .10$. Coefficients that are not significantly different from zero are denoted in gray and by $_{NS}$ for clarity.

peer affiliation (Andrews et al., 2002; Monahan et al., 2009; Curran et al., 1997).

Following previous research (Curran et al., 1997; Kendler et al., 2007; van Ryzin et al., 2012; van Ryzin & Dishion, 2014), after accounting for the stability of traits and correlated residual variance at each time point, we found evidence for socialization processes linking antisocial peer affiliation with externalizing disorders from age 17 to 20 and for selection processes linking externalizing disorders with antisocial peer affiliation from age 20 to 24 and 24 to 29. This was true for each of the externalizing disorder facets examined (nicotine dependence, alcohol use disorder, illicit drug use disorder, and adult antisocial behavior) and our estimated latent factor of externalizing disorders.

Contrary to our expectations that we would find significantly stronger selection than socialization effects postadolescence, we found evidence for essentially equivalent socialization and selection effects from ages 20 to 24 and 24 to 29 for alcohol use disorder, illicit drug use disorder, and adult antisocial behavior, specifically. However, and in line with our expectations, we showed evidence of significantly stronger selection than socialization effects from age 20 to 24 and 24 to 29 using a latent measurement of externalizing disorders. These results were consistent across males and females and across different levels of socioeconomic status. Moreover, this general pattern of results was consistent for those who were in or were not in college. In total, results support developmental theory, suggesting selection effects become more important in later than in earlier developmental periods due to more freedom in environmental/social-relationship selection postadolescence (Scarr & McCartney, 1983). However, this may be particular to a liability toward externalizing generally, rather than specific drug use and clinical antisocial behavior (see Iacono et al., 2008).

This follows other research from the Minnesota Twin and Family Study. Samek, Hicks, Keyes, McGue, and Iacono (in press) showed evidence of Gene × Environment interaction involving antisocial peer affiliation and a broad measure of externalizing disorders at age 17, but no such evidence at ages 20, 24, or 29. The broad externalizing measure included several substance use disorders as well as the adult antisocial behavior symptom count, similar to this study. Samek et al. showed the genetic influence on adolescent externalizing disorders was greater under the context of a greater degree of antisocial peer affiliation and smaller under the context of a lesser degree of antisocial peer affiliation in adolescence.

This generally supports a socialization hypothesis in that antisocial peers appear to activate genetic risk for externalizing problems—but only in adolescence. That is because no such genetic moderation was found for the cross-sectional associations at ages 20, 24, or 29, or the longitudinal associations between adolescent antisocial peer affiliation and adult externalizing disorders (Samek et al., in press). However, subsequent analyses by Samek and colleagues confirmed that there was unique evidence for genetic moderation for several substance use disorders rather than the broader measurement of externalizing problems. In combination, these studies suggest important socializing influence of adolescent antisocial peers that seem to dissipate over time, especially in regards to broad externalizing problems. On the other hand, these studies suggest some continued socializing influence of antisocial peers on individual (substance and behavior specific) externalizing problems.

The present study's findings are somewhat inconsistent with results reported by Monahan et al. (2009). Monahan and colleagues found evidence of both selection and socialization from 14 to 15, did not find evidence for selection past age 16, and did not find evidence for either selection or socialization from age 20 to 22. Potential reasons for the discrepancy in the Monahan et al. study and this one was that the Monahan et al. study included individuals who were adjudicated of serious crime and the sample was made up of predominately males. Using a large community sample of males and females, we have shown that selection appears to still be operating in a meaningful and developmental pattern through age 29.

It is important to note that in this study, the stability of externalizing disorders was smaller in effect size from ages 17 to 20 than from ages 20 to 24 and 20 to 29. This follows earlier research demonstrating marked instability from adolescence to early adulthood in antisocial behavior and substance use disorders (Loeber et al., 2012; Moffitt, 1993; Moffitt & Caspi, 2001), including marked increases substance use disorders in early adulthood relative to adolescence (SAMHSA, 2014). Comparatively, the stability of

antisocial peer affiliation from ages 17 to 20 was generally similar in effect size to the stability of antisocial peer affiliation from ages 20 to 24 and ages 24 to 29. This suggests that adolescents who affiliate with antisocial peers in adolescence are to some extent more likely to continue to affiliate with antisocial peers as they transition into early adulthood (i.e., age 20) and in later young adulthood (i.e., age 24 and 29). Nonetheless, even with relatively stable antisocial peers, our findings suggest the relative socializing influence of antisocial peers appears to decrease across time. This again follows studies a general marked desistence in antisocial behavior and substance use generally as people become older (Loeber et al., 2012; SAMHSA, 2014).

Future Directions

In addition to replicating findings in other community and "at-risk" samples, additional future directions include an analysis of other aspects of socialization and selection that may be relevant to externalizing disorders in young adulthood, such as romantic relationship experiences and romantic relationship affiliations. There is some evidence of this as time spent with friends and peer groups decreases and time spent with romantic partners increases in early adulthood (Reis, Lin, Bennett, & Nezlek, 1993). Following this, van Dulmen, Goncy, Haydon, and Collins (2008) indicated that romantic relationship security was more strongly and inversely related to externalizing problems in early adulthood (ages 23–24) relative to adolescence (age 16). This suggests that romantic partner characteristics, rather than peers, may be more relevant to adult externalizing disorders.

This study is not without limitations. As this sample was representative of Minnesota for the birth cohorts sampled, it is predominately White. Nonetheless, there was considerable diversity in family-of-origin socioeconomic status and the percentages of those that went to versus did not go to college. It is useful to reiterate that antisocial peer affiliation and externalizing disorders were evaluated at ages 17 and through 29; likely these constructs were impacted by earlier antisocial peer and externalizing problems (i.e., prior to age 17) not captured in the modeling used here. This is supported by prior research, which has evidenced both socialization and selection in earlier adolescent periods (e.g., Curran et al., 1997; van Ryzin & Dishion, 2014). Moreover, there is research to support the notion that peer influence may be even more important to adolescent development in early rather than middle or late adolescence (Albert et al., 2013; Gardner & Steinberg, 2005; Sumter et al., 2009). It will also be important for future research to take advantage of multiple measurements of antisocial peer affiliation to determine whether that may have impacted results. Finally, we were unable to evaluate for variation by type of college experience (e.g., some college experience vs. finished degree) as the majority of cases who met the new college experience criteria evaluated in this study had completed only "at least some college" (92–100% at ages 20, 24, and 29). It remains important for future studies to better explore this and determine whether there may be specific reasons to expect one college type to have a stronger socialization effect than another. There are also important strengths of this study, including the large sample size, inclusion of both males and females, and the use of clinical interviews to assess externalizing disorders, as well as the longitudinal design, and incorporation of prospective data analysis.

Conclusion

This study showed support for socialization as the predominant mechanism to explain the link between antisocial peer affiliation and a broad measure of externalizing disorders in the developmental transition from adolescence to early adulthood (i.e., age 17 to 20), but past this developmental transition, selection was more important in explaining the prospective links between antisocial peer affiliation and this broad measure of externalizing disorders (i.e., ages 20 to 24 and 24 to 29). Results were generally consistent across males and females and regardless of college status, suggesting these processes are not context-specific to college experience. On the other hand, there was more support for smaller socialization and selection influences beyond age 20 for each individual externalizing disorder (e.g., substance use disorder and adult antisocial behavior). More research is needed to evaluate the socialization versus selection impact of other mechanisms linked to externalizing disorders in adolescence and young adulthood, including romantic relationship affiliation and experiences.

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