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Original article

Intergenerational Continuity in Substance Abuse: Does Offspring's Friendship Network Make a Difference?

Kimberly L. Henry, Ph.D.[a,*], Celia J. Fulco[b], Della V. Agbeke[b], and Anastasia M. Ratcliff[c]

[a] Colorado State University, Department of Psychology and Colorado School of Public Health, Fort Collins, Colorado
[b] Colorado State University, Department of Psychology, Fort Collins, Colorado
[c] Colorado State University, Colorado School of Public Health, Fort Collins, Colorado

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A B S T R A C T

Purpose: A parental history of substance abuse is a key risk factor for offspring's substance abuse. Identification of factors that may mitigate this effect is prerequisite to promoting resilience. In this study, we consider the substance use of peers in an adolescent's friendship network as a potential moderator of intergenerational continuity in substance abuse.
Methods: Prospective, longitudinal data from the Rochester Youth Development Study and the Rochester Intergenerational Study for 246 father–child dyads and 167 mother–child dyads were utilized. Ordinal generalized estimating equations were specified to examine the moderating role of friend's substance use in the relationship between parental substance use disorder and child's substance abuse between the ages of 13 and 17.
Results: Father's substance use disorder was associated with an increased risk of substance abuse by his child. Moreover, the harmful effect of paternal substance abuse on child's abuse of substances was apparent only when some or most of the child's friends used substances. Maternal substance use disorder was extremely rare in the sample and was not found to be associated with child's substance abuse, irrespective of the substance use of friends.
Conclusions: The intergenerational transmission of risk for substance abuse between father and child was mitigated when children were not exposed to friends who use substances, and exacerbated when children had substantial exposure to substance-using friends. Preventing the child's association with substance-using peers may be particularly important for children with this type of familial risk.

IMPLICATIONS AND CONTRIBUTION

The negative impact of a paternal history of substance abuse on children's uptake and escalation of substance use is heightened when the child associates with substance-using peers. The promotion of prosocial peer groups may be particularly salient for children with a paternal family history of substance abuse.

A parental history of substance abuse is a key risk factor for children's early onset of substances, as well as the escalation of use throughout adolescence [1,2]. There are numerous mechanisms that account for this observed continuity in substance abuse, including genetics, prenatal exposure, parental modeling, family norms and sanctions, family management, parent–child relationships, the familial context, and the neighborhood context [3]. Given the prevalence [4,5] and serious public health consequences [6] of substance abuse, examination of risk and resilience processes associated with the intergenerational continuity of substance abuse is of critical importance and may identify important strategies for breaking the cycle of substance abuse in vulnerable families.

While there is reasonably strong evidence of intergenerational continuity in substance abuse, including the mechanisms that

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* Address correspondence to: Kimberly L. Henry, Ph.D., Colorado State University, Department of Psychology and Colorado School of Public Health, 220 Behavioral Sciences Building, 410 W Pitkin St, Fort Collins, CO 80523.
E-mail address: kim.henry@colostate.edu (K.L. Henry).

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explain it, comparatively less work is available to identify potential moderators of continuity. That is, variables that may either heighten or mitigate the harmful influence that a parental history of a substance use disorder may have on substance abuse among offspring. The identification of factors capable of breaking the cycle of substance abuse in families is prerequisite to the development of effective intervention strategies. In this study, we consider a child's friendship network as a potential moderator of intergenerational continuity in substance abuse.

Friendships during adolescence furnish the primary social context for the onset and escalation of substance use and abuse. Through interactions with peers and friends, young people seek to integrate with their group's behavioral norms, regulations, and routines [6,7]. They learn what members of their group value and may begin to adopt the attitudes and behaviors of group members [8], whether these values, attitudes, and behaviors are prosocial or problematic. Coinciding with increases in autonomy during adolescence, adolescent friendship groups also tend to spend time together in unsupervised or risky settings, where drugs are more readily available and where sanctions against use are less likely to be present [9]. Moreover, the most oft-cited motivation for substance use in this developmental period is to have fun with friends [10], and some evidence suggests that engagement in risky behaviors with friends serves to enhance friendship bonds. Adolescents may view this cobenefit of substance use as particularly advantageous [11]. It is also important to recognize that adolescents commonly seek out other adolescents who share values and interests similar to their own. As such, homophily in substance use between friends exists due to both influence and selection processes [12]. Irrespective of how friendship networks form and evolve, it is clear that one of the strongest predictors of an adolescent's own use of substances is the use of substances by his or her close friends [13]. When an adolescent's close friends use substances, he or she is far more likely to also use.

Evidence from the behavioral genetics literature suggests that low-risk environments can protect adolescents who are genetically predisposed to substance abuse. Specifically, environments that discourage substance use or preclude opportunities for substance use have been found to mitigate the harmful impact of a genetic vulnerability [14–20]. For example, Legrand et al. [21] characterized the degree of environmental risk that boys were exposed to in preadolescence by considering negative peer models, school attitudes, mother–son relationships, religiosity, and engagement in extracurricular activities. They found that a family history of substance use and a high-risk environment each independently predicted substance use, and that a low risk environment provided a buffer against the negative effects of a family history of substance use. Likewise, Harden et al. identified a gene–environment interaction such that the positive correlation of a best friend's substance use and an adolescent's own use of substances was largest for those at highest genetic vulnerability for substance use. The results of these studies suggest that adolescents who do not associate with substance-using peers may not be at a heightened risk for substance use even if a family history of a substance disorder is present.

In the current study, we used prospective data from a longitudinal multigenerational panel study, the Rochester Intergenerational Study (RIGS), and consider father–child and mother–child dyads. For the father–child dyads, we consider both the father's influence and the other primary caregiver's (OCG's; typically the biological mother) influence. Our objective was to determine if intergenerational continuity in substance abuse is conditioned on the child's exposure to substance-using friends. We hypothesized that the harmful impact of parental substance use disorder would be exacerbated if members of the child's friendship network used substances.

Methods

Sample

The data for this study come from two longitudinal, companion studies. The original study, the Rochester Youth Development Study (RYDS), began in 1988 and the intergenerational extension, the RIGS, began in 1999. Detailed information about the designs of these studies is presented elsewhere [22]; only a brief summary is provided here.

The original RYDS sample of 1,000 adolescents (referred to as G2) is representative of the seventh and eighth grade public school population of Rochester, NY in 1988. Youth at high risk for antisocial behavior were overrepresented by oversampling males and residents in high-crime areas of the city. RYDS participants completed regular interviews in school or home every 6 months from 1988 to 1992 (Phase 1), annually from 1994 to 1996 (Phase 2), and biannually from 2003 to 2006 (Phase 3). In general, sample retention was good (>80% at Phase 3) and there is no evidence that attrition appreciably biased the sample [23].

Beginning in 1999, RIGS selected G2's oldest biological child, referred to as G3, and added new firstborns to the G3 sample in each subsequent year. G2, and G3's OCG for G2 fathers, completed annual interviews since the inception of RIGS (continuing until G3 turns/turned 18) and G3 completed annual interviews once he/she turned eight. For G2 fathers, the other caregiver is typically the child's biological mother (93%). To date, there are prospective, longitudinal data on 539 parent–child dyads. The present analysis utilizes data from 246 father–child dyads and 167 mother–child dyads, this constitutes all dyads with available data on parental disorder history and interview data from G3 for at least 1 year between the ages of 13 and 17. The University at Albany's Institutional Review Board approved all data collection procedures.

Measures

Child's substance abuse was measured via a series of self-report questions at each annual interview. Children indicated whether they had used alcohol and cannabis since the date of last interview. If affirmative, the child reported whether they had used alcohol and cannabis at least once a month during the past year. If the child indicated monthly use, then the child reported whether their use of alcohol and cannabis resulted in nine different problematic consequences of adolescent substance use. Using this question series, we created an ordinal measure of substance abuse at each age from age 13 to age 17, where 0 = **no use**; 1 = **rare user** (some use, but less than monthly); 2 = **regular user** (monthly use, but without consequences); and 3 = **problem user** (monthly use that resulted in harmful consequences/problems).

Lifetime substance abuse and dependence for G2 and OCG was measured between 2004 and 2011 using the Computerized Diagnostic Interview Schedule Version IV. [24] The Computerized Diagnostic Interview Schedule Version IV is based on the Diagnostic and Statistical Manual, Edition 4 [25] criteria for lifetime substance use, abuse, and dependence. Participants who met criteria for lifetime abuse or dependence (referred to in the results as a disorder) of either alcohol or cannabis were assigned a 1; those who did not