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Research Article

Is It Important to Prevent Early Exposure to Drugs and Alcohol Among Adolescents?

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ABSTRACT—*Exposure to alcohol and illicit drugs during early adolescence has been associated with poor outcomes in adulthood. However, many adolescents with exposure to these substances also have a history of conduct problems, which raises the question of whether early exposure to alcohol and drugs leads to poor outcomes only for those adolescents who are already at risk. In a 30-year prospective study, we tested whether there was evidence that early substance exposure can be a causal factor for adolescents' future lives. After propensity-score matching, early-exposed adolescents remained at an increased risk for a number of poor outcomes. Approximately 50% of adolescents exposed to alcohol and illicit drugs prior to age 15 had no conduct-problem history, yet were still at an increased risk for adult substance dependence, herpes infection, early pregnancy, and crime. Efforts to reduce or delay early substance exposure may prevent a wide range of adult health problems and should not be restricted to adolescents who are already at risk.*

Many adolescents experiment with drugs and alcohol, and parents, teachers, and policymakers want to know the consequences of adolescent substance use. Parents, in particular, worry that their adolescents who use drugs or alcohol may suffer long-term consequences, such as a dependence on drugs; be drawn into risky sexual behaviors; contract sexually transmitted diseases (STDs); fall behind in school; or get a criminal record. Research has demonstrated that exposure to illicit drugs and alcohol prior to age 15 statistically predicts substance disorders in adulthood (Grant & Dawson, 1997; Hingson, Heeren, & Winter, 2006). Exposure to these substances during adolescence has also been linked to involvement in risky sexual behaviors and STDs (Stueve & O'Donnell, 2005), early pregnancy (Ellickson, Tucker, & Klein, 2003), low educational attainment (King, Meehan, Trim, & Chassin, 2006), and crime (Elliott, Huizinga, & Menard, 1989). On the basis of this evidence, the U.S. Surgeon General's office has issued a call to action to stop underage drinking (U.S. Department of Health and Human Services, 2007), placing a special emphasis on risks to youth who report drinking before the age of 15. However, nagging doubts remain regarding whether substance use affects adolescents' later lives causally.

Critics of this recent policy stance allege that early exposure to alcohol and drugs per se is not the cause of problems in adolescents' later lives (Peele, 2007). Indeed, research has repeatedly shown that adolescents who use substances in early adolescence are also more likely than other adolescents to have a childhood history of conduct problems, which itself predicts the same adult outcomes (Moffitt, 2006). A recent review concluded that adolescents at risk for developing substance-use disorders are not "just normal adolescents who happen to be experimenting with substances but, in many cases, are youths with other . . . problems" (Armstrong & Costello, 2002, p. 1235). Thus, parents, teachers, and policymakers are caught between mixed messages: On the one hand, they are told that exposure to drugs and alcohol prior to age 15 is harmful for adolescents and should be prevented, and on the other hand, they are told that it is normal for adolescents to try these substances and that the majority will not become addicts or ruin their lives.

It is not practical or feasible to administer alcohol and drugs to young adolescents in a randomized control trial in order to test

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the causal effect of substance use on adolescents' future lives. However, analytic strategies are now available to quantify "treatment" effects in observational studies when randomized control trials are not a viable option (D'Agostino & D'Agostino, 2007; Stuart & Green, 2008). Such propensity-score methods are designed to re-create the desirable features of experimental designs by creating balance between exposed ("treatment") and nonexposed ("control") groups that have formed naturally over the course of an observational study (Haviland, Nagin, & Rosenbaum, 2007).

Here we report the results of applying propensity-score matching (Rosenbaum & Rubin, 1985) in a 30-year longitudinal study to address a question of significant public-health importance that cannot be studied using randomized control trials. Specifically, we asked whether there is evidence that early exposure to illicit substances is a causal factor in adolescents' future lives ("are drugs bad for kids?"), or whether adolescents with a developmental history of conduct problems are simply more likely than other adolescents to be exposed to alcohol and illicit substances and to experience poor adult outcomes ("do bad kids do drugs?"). At present, the extent to which a history of conduct problems in childhood has confounded the association between early substance exposure and adult outcomes is unclear. Researchers have called for prospective longitudinal designs that are able to adequately account for childhood conduct problems (Hingson et al., 2006); however, to date, only a handful of studies have met this criterion.

In the present study, adolescents' developmental history of conduct problems was assessed prospectively by multiple informants. Assessments were made when the study members were ages 7, 9, 11, and 13. Early substance exposure was defined as frequent exposure to illicit substances prior to age 15. Adolescents were then followed prospectively into adulthood to assess the influence of early substance exposure on their adult lives. We estimated two types of treatment effects to test whether early substance exposure influenced adolescent's adult lives. First, we estimated the effect of early substance exposure on adolescents' adult outcomes across the entire birth cohort. Propensity-score matching was applied to adjust treatment-effect estimates for nonrandom assignment to the treatment condition (in this case, defined as exposure to illicit substances prior to age 15, which we term early exposure) and to facilitate causal inference by ensuring that adolescents with early exposure were balanced relative to those with no early exposure on key background factors that might otherwise confound the results.

Second, we estimated *group-specific* treatment effects to test whether early substance exposure is similarly dangerous for all adolescents, or whether some adolescents' developmental history of conduct problems signals particular vulnerability to early substance exposure. Developmental trajectories of conduct problems were defined previously in this cohort using group-based trajectory modeling (Odgers et al., 2008); this work isolated a group of children (66% of the cohort) who followed a *no-conduct-problem trajectory* between the ages of 7 and 13. Adolescents on this trajectory had very few deficits in childhood and were less likely than their already-at-risk peers to experience early substance exposure and poor adult outcomes. The question for the majority of ordinary adolescents, therefore, was whether adolescents who had no history of conduct problems were protected from any untoward effects of early substance exposure. By contrast, the remaining 34% of the children in this cohort entered adolescence with a history of conduct problems; these adolescents were *more* likely (or had a higher propensity) than their normative peers to experience early substance exposure and poor adult outcomes. The question for this subgroup of already-at-risk adolescents, therefore, was not whether they would go on to experience poor adult outcomes, but rather whether early substance exposure placed an additive burden on their already-compromised future lives.

METHOD

Participants

Participants were members of the Dunedin Multidisciplinary Health and Development Study. The cohort of 1,037 children (52% male) was constituted at 3 years of age, when investigators enrolled 91% of children born consecutively between April 1972 and March 1973 in Dunedin, New Zealand. Cohort families represent the full range of socioeconomic status in New Zealand's South Island and were primarily White. Follow-up assessments were conducted, with informed consent, when the cohort members were 5, 7, 9, 11, 13, 15, 18, 21, 26, and 32 years of age; 96% of the living study members participated in the age-32 assessment, which took place in 2003 through 2005. Research ethics committees at the University of Otago, Duke University, and Maudsley Hospital approved this research.

Early Exposure and Conduct-Problem Measures

Early Substance Exposure

At ages 13 and 15, study members reported on their frequency of exposure to illicit substances during the past year. They reported whether they had sniffed glue, gasoline, or other inhalants; smoked cannabis; used any illegal drugs other than cannabis; bought or drunk alcoholic drinks; or drunk alcoholic drinks during school. Response options were 0, *never*; 1, *once or twice*; and 2, *multiple occasions*. In total, 11.2% of the study members were classified as being exposed to substances on multiple occasions at age 13, age 15, or both; hereafter, we refer to these study members as *early-exposed* adolescents. Alcohol was the substance most commonly used by the young adolescents in our cohort; 10% of study members were exposed to alcohol prior to age 15, 5% were exposed to cannabis, and fewer than 2% were exposed to inhalants or other drugs.

Conduct Problems

Self-, parent, and teacher reports were used to assess study members' conduct problems at ages 7, 9, 11, and 13 years. The

following six symptoms of conduct disorder, as defined by the fourth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM–IV; American Psychiatric Association, 1994), were assessed as being present or absent at each age: physical fighting, bullying other people, destroying property, telling lies, truancy, and stealing. A composite score, ranging from 0 to 6, was calculated to represent the number of different types of conduct-problem behaviors each study member had engaged in during the past year. As noted earlier, prior group-based trajectory modeling isolated young adolescents following a no-conduct-problem trajectory between the ages of 7 and 13 (65.7% of the cohort; 74.1% of females and 57.9% of males). This group represented a "pure," or super-healthy, group in that the average number of conduct-problem symptoms at each age was below 1.

Background Covariates

Psychometric properties of all background covariates in this study have been reported in detail elsewhere (Moffitt, Caspi, Rutter, & Silva, 2001), and all estimates of their reliability exceeded .70. Background covariates were assessed prior to age 13, unless otherwise stated.

Family history of alcohol and drug disorders was assessed in 2003 through 2006 as part of the Dunedin Family Heath History Study (DFHHS; Odgers et al., 2007). That study used the Family History Screen (Weissman et al., 2000) to collect psychiatric-history data about each study member's biological parents, grandparents, and siblings older than 10 years old. Ratings were obtained from multiple informants. A family-liability score was computed for each study member. This score indexed the proportion of family members, across three generations, with an alcohol or drug disorder.

Criminal conviction of a parent was also assessed through the DFHHS; 25% of study members had at least one parent with a criminal conviction.

Socioeconomic status (SES) was measured as the higher of the father's or mother's occupation, as rated on a 6-point scale for New Zealand (Elley & Irving, 1976); 21% of the families were classified as low SES.

Maltreatment was measured using the following indicators: rejecting mother-child interactions (as observed by staff members), parental reports of harsh discipline, two or more changes in primary caregiver, and retrospective self-reports of injurious physical abuse or unwanted sexual contact. A study member was considered to be maltreated if he or she had two or more indicators of maltreatment (Caspi et al., 2002); on this basis, 9% of the study members were classified as maltreated.

Mother's IQ was tested using the SRA (Thurstone & Thurstone, 1973); scores were standardized ($M = 100$, $SD = 15$). Low mother's IQ was defined as a standardized SRA score less than 85.

Child IQ was tested using the Wechsler Intelligence Scale for Children–Revised (WISC-R; Wechsler, 1974); scores were standardized ($M = 100$, $SD = 15$). Low child IQ was defined as a standardized WISC-R score below 85.

Undercontrolled temperament was measured through staff ratings. Ratings were made after observing the child in a 90-min testing session with an unfamiliar examiner. Factor and cluster analyses reduced these ratings to three temperament types, including the undercontrolled type (Caspi & Silva, 1995).

Attention-deficit/hyperactivity disorder (ADHD) was measured using the Diagnostic Interview Schedule for Children (Costello, Edelbrock, Kalas, Kessler, & Klaric, 1982). Data obtained when the study members were ages 5 to 15 were used to make diagnoses according to the criteria of the third edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM–III; American Psychiatric Association, 1980); diagnoses were confirmed through parent or teacher report. Six percent of study members met diagnostic criteria for ADHD.

Adult Outcomes

Substance-use disorders at age 32 were assessed from data obtained in private structured interviews using the Diagnostic Interview Schedule (Robins, Cottler, Bucholz, & Compton, 1995). Diagnoses were made according to DSM–IV criteria (American Psychiatric Association, 1994). For this report, *substance dependence* was defined as cannabis dependence, dependence on other drugs, or alcohol dependence within the past year. Dependence at age 32 signals a substance-use problem serious enough to outlast early adulthood, a developmental period when large numbers of young people can meet criteria for substance disorders on a short-term basis.

Type 2 *herpes infection* at age 32 was assessed using blood samples. Diagnoses were made on the basis of an indirect enzyme-linked immunosorbent assay (HerpeSelect® 2 ELISA IgG, Focus Technologies, Cyprus, CA; Eberhart-Phillips et al., 2001). Herpes infection was diagnosed using a cutoff value of 3.5, and any equivocal result (between 0.9 and 3.5) was resolved using the Western Blot test (Ho, Field, Irving, Packham, & Cunningham, 1993).

Early pregnancy was defined as having at least one pregnancy prior to age 21 and was assessed by females' self-report. The failure to delay pregnancy until age 20 or 21 is associated with economic costs and poor social consequences for both mothers and children in contemporary cohorts (Maynard, 1996).

No educational qualifications was defined as ending secondary education prior to receiving qualifications and not returning to earn qualifications by age 32. In New Zealand, students receive qualifications on the basis of national exams that almost all students take by age 16; the results determine promotion in secondary and technical schools and help people secure better employment in the labor market (Miech, Caspi, Moffitt, Wright, & Silva, 1999).

Number of criminal convictions between ages 17 and 32 was determined for each study member by searching the computerized New Zealand Police database. Convictions for nonviolent and violent crimes were included, but traffic convictions were not.

STATISTICAL ANALYSES

Analyses proceeded in four steps. First, because study members were not randomly assigned to a substance-use condition, potential selection biases were addressed by developing a propensity score for early substance exposure. This score, $e(x)$, represented the conditional probability of early substance exposure ($z = 1$), versus no early substance exposure ($z = 0$), given a combination of key familial, social, and child covariates (x); that is, $e(x)$ represented $P(z = 1|x)$. Propensity scores were calculated for all adolescents using a multivariate logistic regression that included the 13 covariates listed in Table 1. The c statistic for this model was .72 (95% confidence interval: .67–.77), which indicates a fair-to-good ability to discriminate between early-exposed and non-early-exposed adolescents. The propensity scores ranged from .02 to .73.

Second, we used the STATA module PSMATCH2 (Leuven & Sianesi, 2003–2006) to perform 3-to-1 nearest-neighbor propensity-score matching. Specifically, propensity scores were used to match each early-exposed adolescent to 3 non-early-exposed adolescents who had a similar probability of early exposure.

Third, to determine whether early substance exposure influenced adolescents' later lives, we compared the adult outcomes of early-exposed adolescents with the adult outcomes of propensity-matched non-early-exposed adolescents.

Fourth, adolescents were stratified by their conduct-problem history to estimate group-specific treatment effects. More specifically, we tested (a) whether early substance exposure presents a risk for the majority of adolescents, who have no conduct-problem history (*no-conduct-problem group*), and (b) whether adolescents with a prior history of conduct problems (*conduct-problem group*) have a particular vulnerability to early substance exposure.

RESULTS

Does Propensity-Score Matching Create Balance Between Early- and Non-Early-Exposed Adolescents?

As Table 1 shows, early-exposed adolescents were well matched to non-early-exposed adolescents following propensity-score matching. Prior to propensity-score matching, the standardized bias (SB) between groups ranged from −8% to 85% across the background covariates; the average SB was 17%. After propensity-score matching, the SB between groups ranged from −6% to 8% across the background covariates, and the average SB was 0%. We obtained a similar reduction in SB within the conduct-problem subgroups after propensity-score matching:

TABLE 1

Risk Factors in Adolescents With Versus Without Early Substance Exposure, Before and After Propensity-Score Matching

	Before propensity-score matching			After propensity-score matching		
	Mean score or prevalence			Mean score or prevalence		
Risk factor	No early exposure (n = 813)	Early exposure (n = 114)	*SB* (%)	No early exposure (n = 342)	Early exposure (n = 114)	*SB* (%)
Family history of alcohol or drug disorder	0.14	0.21	43*	0.20	0.21	5
Conduct problems, age 7	1.65	1.90	16	1.97	1.90	−5
Conduct problems, age 9	1.47	1.75	18*	1.83	1.75	−6
Conduct problems, age 11	1.36	1.72	23*	1.79	1.72	−6
Conduct problems, age 13	1.31	2.49	85*	2.45	2.49	3
Parent criminal conviction (%)	25.0	31.8	16	29.3	31.8	6
Low socioeconomic status (%)	20.0	20.0	0	17.8	20.0	7
Maltreatment (%)	8.4	10.0	6	10.0	10.0	1
Low mother's IQ (%)	14.1	18.1	11	15.7	18.0	8
Low child IQ (%)	13.1	15.2	6	16.3	15.2	−4
Undercontrolled temperament (%)	10.7	8.2	−8	9.1	8.2	−4
ADHD (%)	6.1	7.2	5	7.9	7.2	−3
Male (%)	50.6	51.8	3	53.3	51.8	−4
Average	—	—	17	—	—	0

Note. The sample included 927 adolescents, categorized as early-exposed (i.e., exposed to alcohol or drugs on multiple occasions before age 15) and non-early-exposed (i.e., not exposed to alcohol or drugs on multiple occasions before age 15). Standardized bias (*SB*) between the treatment (exposed) group and the control (unexposed) group (subscripts T and C, respectively) was computed as follows: $SB = M_T - M_C/\sqrt{[\sigma_T^2 + \sigma_C^2)/2]}$. Negative values of *SB* indicate greater risk in the non-early-exposed group than in the early-exposed group. ADHD = attention-deficit/hyperactivity disorder.

*$p < .10$.

TABLE 2

Effects of Early Substance Exposure on Adolescents' Adult Outcomes, Before and After Propensity-Score Matching

	Before propensity-score matching			After propensity-score matching		
	Mean risk			Mean risk		
Adult outcome	No early exposure (n = 813)	Early exposure (n = 114)	Effect size	No early exposure (n = 342)	Early exposure (n = 114)	Effect size
Substance dependence at age 32 (%)	11.1	28.8	3.25** (2.04–5.18)	14.7	28.8	2.25** (1.35–3.73)
Herpes infection at age 32 (%)	17.0	28.0	1.90** (1.18–3.07)	16.1	28.0	2.02** (1.18–3.44)
Early pregnancy (prior to age 21)[a] (%)	18.1	44.7	3.65** (1.93–6.86)	22.5	44.7	2.78** (1.38–5.57)
No educational qualifications by age 32 (%)	14.7	32.1	2.74** (1.76–4.27)	19.3	32.1	1.97** (1.22–3.19)
Number of criminal convictions between ages 17 and 32[b]	1.31	7.12	5.35** (2.85–10.05)	1.74	7.12	3.95** (2.23–6.97)

Note. Study members were categorized as early-exposed (i.e., exposed to alcohol or drugs on multiple occasions before age 15) versus non-early-exposed (i.e., not exposed to alcohol or drugs on multiple occasions before age 15). The reported effect sizes are odds ratios for all outcomes except number of criminal convictions, for which incidence-rate ratios are reported. The numbers in parentheses are 95% confidence intervals. All findings remained statistically significant after adjustment of standard errors via bootstrapping in PSMATCH2. [a]Early pregnancy was estimated for females only and was defined as having at least one pregnancy prior to age 21; sex-specific propensity scores were used for this female-only analysis. [b]Negative binomial regressions were applied to model incidence-rate ratios for the number of criminal convictions.

**$p < .05$.

Within the no-conduct-problem subgroup, the average SB for early- versus non-early-exposed adolescents was reduced from 2% to 1%, and within the conduct-problem subgroup, the average SB was reduced from 11% to 0%. For all analyses, the SB was reduced to below 20% for each of the 13 covariates following propensity-score matching, an indication of a high degree of similarity in the distributions of the background covariates.

Does Early Substance Exposure Predict Poor Adult Outcomes for Adolescents After Propensity-Score Matching?

As Table 2 shows, early-exposed adolescents were at an increased risk for the adult outcomes of substance dependence, herpes infection, early pregnancy, failure to obtain educational qualifications, and criminal convictions. The adjusted effects after propensity-score matching remained statistically significant for all of these outcomes. Early-exposed adolescents were approximately 2 to 3 times more likely than non-early-exposed adolescents to be substance dependent, to have herpes infection, to have had an early pregnancy, and to have failed to obtain educational qualifications; early-exposed adolescents also had significantly more criminal convictions than non-early-exposed adolescents.

Does Early Substance Exposure Influence the Majority of Ordinary Adolescents With No Prior History of Conduct Problems?

As expected, early substance exposure was not a random event; adolescents with a conduct-problem history were 2 times (odds ratio = 2.1, confidence interval = 1.4–3.1) more likely to be exposed to illicit substances prior to age 15, compared with adolescents without a conduct-problem history. More specifically, 17.0% of adolescents with a conduct problem history, versus 9.1% of adolescents with no conduct-problem history, experienced early substance exposure. Fifty-six of the 114 early-exposed adolescents were assigned to the no-conduct-problem subgroup; this means that approximately 50% of adolescents exposed to substances prior to age 15 had no prior history of conduct problems.

Table 3 presents the group-specific effects of early substance exposure on adolescents' adult outcomes. Adjusted effects after propensity-score matching are presented separately for adolescents with versus without a conduct-problem history. The results in the table illustrate two main findings. First, after propensity-score matching, ordinary adolescents without a conduct-problem history were at an increased risk for adult substance dependence, herpes infection, early pregnancy, and criminal convictions if they had experienced early substance exposure, although early substance exposure did not increase risk for not finishing school in this subgroup. Second, early substance exposure further elevated the risk for adult substance dependence, early pregnancy, failure to obtain educational qualifications, and criminal convictions—but not herpes infection—among adolescents who were already at risk because of their conduct-problem history.

Sensitivity Analyses

Are the effects of early substance exposure different for alcohol versus illicit drugs? The substance most frequently used by

TABLE 3

Propensity-Adjusted Effects of Early Substance Exposure on Adolescents' Adult Outcomes, for Adolescents With Versus Without a Conduct-Problem History

	No conduct-problem history			Some conduct-problem history		
	Mean risk			Mean risk		
Adult outcome	No early exposure (n = 168)	Early exposure (n = 56)	Effect size	No early exposure (n = 165)	Early exposure (n = 55)	Effect size
Substance dependence at age 32 (%)	7.6	23.2	3.64** (1.57–8.45)	15.3	32.0	2.60** (1.24–5.46)
Herpes infection at age 32 (%)	15.4	32.7	2.66** (1.26–5.60)	20.5	23.4	1.18 (0.54–2.60)
Early pregnancy (prior to age 21)[a] (%)	12.3	34.4	3.75** (1.45–9.69)	35.9	69.2	4.02** (1.04–15.46)
No educational qualifications by age 32 (%)	10.9	14.2	1.37 (0.56–3.34)	28.7	49.0	2.38** (1.24–4.57)
Number of criminal convictions between ages 17 and 32	0.65	1.59	2.86** (1.2–6.6)	2.52	13.12	4.96** (2.5–10.0)

Note. Study members were categorized as early-exposed (i.e., exposed to alcohol or drugs on multiple occasions before age 15) and non-early-exposed (i.e., not exposed to alcohol or drugs on multiple occasions before age 15). The reported effect sizes are odds ratios for all outcomes except number of criminal convictions, for which incidence-rate ratios are reported. The numbers in parentheses are 95% confidence intervals. All findings remained statistically significant after adjustment of standard errors via bootstrapping in PSMATCH2.
[a]The within-group treatment effects for pregnancy prior to age 21 are exploratory because each subgroup contained fewer than 50 early-exposed females.
**$p < .05$.

adolescents in our cohort was alcohol, and the vast majority of early-exposed adolescents were exposed to alcohol. However, 65 of these adolescents were exposed to alcohol only and did not use drugs. We tested whether adolescents exposed only to alcohol differed from adolescents with no early substance exposure. After propensity-score matching, early alcohol exposure alone predicted a cumulative index of poor adult outcomes, including substance dependence, herpes infection, failure to obtain educational qualifications, and criminal convictions (Cohen's $d = 0.51$, $p < .01$).

The remaining early-exposed adolescents ($n = 49$) were exposed to multiple substances. This reflects the reality of adolescent substance use: It is extremely rare for a young adolescent to specialize in the use of cannabis, inhalants, or any other illicit drug. Rather, adolescents who use illicit drugs typically use alcohol as well. After propensity-score matching, early poly-substance exposure predicted a cumulative index of poor adult outcomes (Cohen's $d = 1.15$, $p < .01$). Thus, although early alcohol exposure alone was a significant predictor of adult risk, early poly-substance exposure was associated with more pronounced risk.

DISCUSSION

This application of propensity-score matching within a 30-year prospective study helps to advance what is known about the effects of early substance exposure in two ways. First, the prior consensus in child psychology and psychiatry has been that adolescents who go on to develop substance dependence are not normal adolescents who are experimenting with substances, but rather are highly likely to be adolescents with a prior history of conduct problems (Armstrong & Costello, 2002). If this is the case, the documented association between early substance exposure and adult outcomes would not be due to exposure per se, but instead would be the result of *who* is exposed (Wells, Horwood, & Fergusson, 2004). Prior research has not resulted in a consensus regarding the causal status of substance exposure (Agrawal, Neale, Prescott, & Kendler, 2004; Kandel, 2003; Lynskey et al., 2003; Prescott & Kendler, 1999). However, results from this study are consistent with a causal effect of early substance exposure among adolescents with no prior history of conduct problems. That is, early-exposed adolescents with no conduct-problem history, although they did not have an increased risk of failing to complete school, were more likely than their matched non-early-exposed counterparts to develop substance dependence, test positive for herpes, have an early pregnancy, and be convicted of criminal offenses.

Second, findings from this prospective study support a causal link between early substance exposure and a wide range of adult outcomes. Propensity-score-adjusted effects indicate that early substance exposure more than doubles the odds of adult substance dependence, herpes infection, early pregnancy, and criminal convictions. With a few notable exceptions (Brook, Brook, Zhang, Cohen, & Whiteman, 2002; Dooley, Prause, Ham-Rowbottom, & Emptage, 2005; Fergusson & Horwood, 1997; Wells et al., 2004), most prior tests of the association between early substance exposure and adult outcomes have been based on cross-sectional surveys of adults, which are

limited to retrospective recall of both childhood behaviors and age of onset for substance use.

This study has limitations. First, although propensity-score matching is designed to mimic the desirable features of randomized control trials within observational studies, the lack of random assignment to a treatment condition (which in this case would have involved administering illicit drugs to children) restricts an ideal test of causal association. Second, children with conduct problems are a heterogeneous group; future studies are required to estimate the impact of early substance exposure separately for distinct subtypes of children with conduct problems. Third, this study was based on a single New Zealand cohort and requires replication across ethnicities and cultures. Fourth, the culture of drug and alcohol use among adolescents has shifted over time. The cohort we studied was exposed to substances for the first time in 1984 through 1987, which meant that exposure was restricted mainly to alcohol and cannabis. Ideally, a study would isolate the effects of specific types of drugs on adolescents' future lives. However, the reality is that, aside from adolescents who are exposed to alcohol only, adolescents exposed to substances prior to age 15 do not specialize in their substance use. Only 1.2% of the cohort restricted their substance use to cannabis prior to age 15, and less than 1% used inhalants or other drugs exclusively. Therefore, we could not examine the specific effects of cannabis, inhalants, or other drugs separately, as the necessary groups of adolescents did not exist. Research needs to examine the effects of other substances that are now used more frequently by adolescents (e.g., cocaine, ecstasy). Future studies should also be designed to include a more rigorous assessment of early substance exposure based on multiple informants and finer-grained measurement of the precise timing and dosage of exposure.

With these limitations in mind, the implications of these results for public policy and prevention can be noted. With respect to public policy, out results are consistent with the U.S. Surgeon General's call to action to prevent early substance exposure. Results support the policy position that early substance exposure poses independent risks for adolescents' future lives; these risks extend beyond the development of later substance dependence, to include risks for herpes infection, early pregnancy, and crime. With respect to prevention, it is important to note that in our study, 50% of the adolescents exposed to substances before age 15 did not have a prior history of conduct problems, yet they experienced many of the same ill consequences as their peers who were already at risk. This means that prevention efforts should not focus solely on at-risk or conduct-problem adolescents. Moreover, this research provides a rationale for evaluating the cost-benefit ratios of substance-use prevention programs separately for adolescents with versus without conduct problems. In short, universal interventions are required to ensure that all children—not only those entering early adolescence on at at-risk trajectory—receive an adequate dose of prevention.

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