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Clouding Up Cognition? Secondhand Cannabis and Tobacco Exposure Related to Cognitive Functioning in Youth

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ABSTRACT

BACKGROUND: Increasing legalization of cannabis, in addition to longstanding rates of tobacco use, raises concerns for possible cognitive decrements from secondhand smoke or environmental exposure, although little research exists. We investigate the relation between cognition and secondhand and environmental cannabis and tobacco exposure in youth.

METHODS: The Adolescent Brain Cognitive Development (ABCD) Study year 2 follow-up (N = 5580; 48% female) cognitive performance and secondhand or environmental cannabis or tobacco exposure data were used. Principal components analysis identified a global cognition factor. Linear mixed-effects models assessed global cognition and individual cognitive task performance by cannabis and/or tobacco environmental exposure. Sociodemographics and other potential confounds were examined. p values were adjusted using the false discovery rate method.

RESULTS: Global cognition was not related to any exposure group after testing corrections and considering confounds. Beyond covariates and family- and site-level factors, secondhand tobacco was related to poorer visual memory (p = .02), and environmental tobacco was associated with poorer visuospatial (p = .02) and language (p = .008) skills. Secondhand cannabis was related to cognition, but not after controlling for potential confounders ($p >$ .05). Environmental cannabis was related to better oral reading (p = .01). Including covariates attenuated effect sizes.

CONCLUSIONS: Secondhand tobacco exposure was associated with poorer visual memory, while environmental tobacco exposure was related to poorer language and visuospatial skills. Secondhand cannabis was not related to cognition after controlling for sociodemographic factors, but environmental cannabis exposure was related to better reading. Because, to our knowledge, this is the first known study of its kind and thus preliminary, secondhand cannabis should continue to be investigated to confirm results.

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Cannabis exposure can be direct or indirect [e.g., secondhand cannabis smoke (SCS) (1)], with either exposure leading to changes in observed cannabis concentrations in biosamples (2–6). Accordingly, measurable concentrations of Δ^9-tetrahydrocannabinol following SCS exposure may influence behavior and cognition (6–8). Given that up to 52% of smoked cannabis is delivered into the environment (1) and SCS has similar carcinogenic properties to tobacco (9), this is a significant public health concern, particularly for children (5). Yet SCS has received limited research attention. Preliminary evidence suggests that maternal cannabis use and SCS are related to increased affective and cognitive difficulties in young children (10), while studies in late childhood are lacking. Investigation into exposure to cannabis-based toxins is needed.

Prevalence of cannabis use among adults with children in the home is rising (4.9% in 2002; 6.8% in 2015) (11). This is particularly true among tobacco smokers and more likely among households with low-income (11) and subsidized (12,13) housing, suggesting increased likelihood of exposure for children from disadvantaged households. A census-weighted study found that 27% of U.S. adults reported SCS in the past week (9% indoor) (14). Younger adults (18–29 years old) identifying as non-White were more likely to report SCS (14), which may in part be tied to structural factors such as placement of cannabis dispensaries within neighborhoods with higher crime, lower income, and/or higher proportions of minorities (15-17). Yet only one known study has investigated the impact of SCS on children. In this small sample (n = 29 SCS exposed), SCS-exposed children had 0.9 to 3.8 times the odds of adverse health events (e.g., asthma) in indoor cannabis smokers' homes relative to nonsmokers (18). Thus, there is need for greater investigation into the impact of SCS in youth.

Similar to cannabis, tobacco is another combustible substance that may impact physical and cognitive functioning through secondhand exposure. Tobacco smoke may influence

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both academic and cognitive performance in youth such as overall intelligence and attention (19). For example, a Finnish study of children 3 to 18 years old, followed for over 30 years, found that objective biosample measurement (serum cotinine) inversely correlated with memory, learning, and working memory, suggesting a potential long-term negative relationship between cognition and secondhand tobacco smoke (STS) (20), although other factors (e.g., unknown baseline cognition, prenatal exposure) obscure interpretation.

Secondhand smoke, or smoke that is exhaled or sidestream from smoked products, remains problematic even after smoke dissipates (18,21–23). Toxins arise from residual smoke products that accumulate on surfaces, which can result in additional environmental exposure through ingestion, inhalation, or absorption through skin [e.g., thirdhand tobacco smoke (23) or thirdhand cannabis smoke], leading to long-lasting opportunities for negative sequelae. Evidence suggests that nicotine in particular has a high affinity for absorption into surfaces, and tobacco chemicals are oxygenated and nitrogenated into secondary pollutants that are not present from tobacco smoke alone (23). This has been found in residential homes and commercial facilities years after smoking occurred (23), giving reason to suspect that broad environmental smoke exposure represents a pressing concern (22) and a likely increased health risk (24). This is well established within the tobacco literature (22,23). Yet, it is also likely that cannabis may leave behind toxins and pollutants. Even well-ventilated examination rooms designed to fully exchange airflow within 2 minutes were found to have detectable levels of cannabinoids on room surfaces after use in a cannabis vaporization administration study (21). While direct exposure to adults smoking cannabis or nicotine is concerning, overall environmental toxins are also important even when smoke is not overtly apparent.

Prevalence of secondhand smoke is not equal across all populations. Previous studies have found that socioeconomic status (SES) (household income) correlates negatively with tobacco exposure in the home, such that children in low SES households are at highest risk for exposure (25–27). Although not as researched, SCS is more prevalent in multiunit housing (13), suggesting exposure to those who are already economically marginalized. A report from the National Health and Nutrition Examination Survey from 2011 to 2018 found that the prevalence of secondhand smoke exposure was higher among non-Hispanic Blacks than non-Hispanic Whites (28); however, given that race and ethnicity are social constructs that are proximal to social inequities, more studies are needed to determine the social factors underlying this health disparity of exposure to tobacco. Therefore, to assess the relationship between secondhand toxins and cognitive performance, sociodemographics must also be accounted for.

This study investigated the influence of toxins from both secondhand and environmental cannabis and/or tobacco exposure on cognitive performance in preadolescent children. We leveraged parental report of youth smoke exposure from the large, diverse Adolescent Brain Cognitive Development (ABCD) Study. To assess the direct influence of secondhand smoke, we examined secondhand exposure relative to no exposure separately by drug (cannabis or tobacco) in relation to global and specific domains of cognitive functioning. We expected that youth with SCS or STS exposure would demonstrate deficits across cognitive domains. Further, to examine the influence of environmental exposure (caregiver report of someone smoking in the home/vehicle when the child is not present), we compared environmental exposure to either substance with no exposure. We hypothesized that environmental exposure would similarly be related to poorer cognitive performance relative to those unexposed in the home.

METHODS AND MATERIALS

Full recruitment and protocol information for the ABCD Study have been described elsewhere (29). Data used in this study were derived from annual data release 3.0 (National Institute of Mental Health Data Archive; https://nda.nih.gov/abcd; https://doi.org/10.15154/1519007), collected from September 2018 to February 2020 (half of year 2 follow-up data; 10–13 years old). Participants with complete data for the planned models were included in these analyses (N = 5580). All aspects of the ABCD protocol were approved by the centralized Institutional Review Board.

ABCD Protocol

Participants and a parent/guardian presented at local research sites for their annual visit. Written assent/consent was provided, and participants underwent clinical interviews, cognitive testing, and magnetic resonance imaging scan. All participants who were initially recruited (29,30) were invited to return for this visit.

Measures

Secondhand and Environmental Substance Use Exposure. Parents/guardians reported whether their child had spent a significant amount of time at their household and, if so, how many adults and youth in the home use each type of drug. If any individuals in the home were reported to use a substance (e.g., cannabis), they were asked, "Did anyone smoke marijuana (medical OR non-medical) inside the house when your child was home or in a vehicle your child was in?" Similar questions were asked regarding cigarettes, e-cigarettes, and other tobacco products. Reporting any household member smoking a cannabis or tobacco product in the home or vehicle with the child present was coded as indicating secondhand exposure (SCS, n = 88; STS, n = 144; SCS + STS, n = 21). Reporting that a household member smoked cannabis or tobacco when the child was not present was coded as environmental exposure (given likely thirdhand exposure; environmental cannabis [Envi-C], n = 737; environmental tobacco [Envi-T], n = 746; environmental exposure to both, n = 222).

Cognitive Tasks. Seven cognitive tasks were assessed as outcome variables. Task details can be reviewed elsewhere (31). For NIH Toolbox tasks (picture vocabulary, which measures language; flanker, which measures attention; pattern comparison, which measures processing speed; picture memory, which measures visual memory; and oral reading, which measures reading ability and academic achievement), uncorrected summary scores were used. For the Little Man Task (measures visuospatial attention/abilities), total correct responses were calculated. For the Rey Auditory Verbal

Learning Task, which measures verbal learning and memory, an overall sum score of total correct responses was used.

Caregiver-Reported Prenatal Substance Use Exposure. Parents/guardians reported whether the biological mother had used drugs at any point during pregnancy. Prenatal substance use was scored as 1 if the biological mother used cannabis, nicotine, or alcohol while pregnant with the participant (prenatal exposure, n = 1843).

Parental History of Substance Problems. Parents/guardians reported whether either biological parent had a history of alcohol/substance problems. Positive parental history was scored between 0.5 and 2, with a history of either alcohol problems or substance problems each scoring 0.5 per parent (e.g., mother with alcohol problems and father with substance problems = 1; mother with alcohol and substance problems and father with substance problems = 1.5; family history $>$ 0, n = 1037).

Self-reported Substance Use. Youth participants were queried first as to whether they heard of each drug class and, if so, whether they used each drug (32). For this study, analyses focused on the most commonly used substances by adolescents (i.e., alcohol, nicotine, cannabis). Thus, participants were coded as either reporting any substance use (i.e., more than a sip or puff; n = 30; SCS, n = 1; STS, n = 0) or not (n = 5550). Analyses were run both including and excluding those who self-reported substance use; results remained unchanged regardless, and so all presented results include those who reported substance use.

Sociodemographics. Parents/guardians reported family income ($<$\$50,000, \$50,000–$<$\$100,000, or $\geq$\$100,000), highest parental education of either parent/guardian, their child's race/ethnicity (Asian, Black, Hispanic, White, or Other), age, and their child's sex as assigned at birth; each were included as covariates. Sociodemographics were entered in the second models for all analyses, given prior secondhand literature (19).

Statistical Analysis

R version 4.1.0 was used in RStudio (https://www.R-project.org/).

Principal Component Analysis. Previous work within the ABCD Study cohort demonstrated three overall components of cognitive abilities, suggesting that broader domains of cognitive functioning can be detected (33). However, although three components were identified, the ABCD protocol changed such that two of the neurocognitive tasks were removed, precluding use of these components. Prior research suggests that cannabis and tobacco use are often associated with deficits in specific, widespread cognitive domains (34–37), with occasional findings of global decline (38,39). Because this has not been tested in SCS exposure, we tested both composite-based and domain-based cognition. Therefore, principal component analysis in this sample with all available task data was run using FactoMineR (40) and missMDA (41) packages. Examination of eigenvalues and the proportion of variance across the domains that each eigenvector explained determined utility of components in further analyses.

Regression Models. Linear mixed-effects models were run with the package 'lme4' (42). Separate linear mixed-effects models predicted cognitive performance across each cognitive domain by three exposure group contrasts, such that one contrast was included per model: 1) SCS versus environmental (not secondhand) cannabis exposure versus no exposure; 2) STS versus environmental (not secondhand) tobacco exposure versus no exposure; and 3) any secondhand exposure versus any environmental (not secondhand) exposure versus no exposure. In all models, no exposure served as a reference group (thus preventing direct comparison of SCS/STS to environmental groups). Given the dearth of prior studies assessing SCS and cognition, all contrasts and models were tested hierarchically, first to determine if a relationship was present, then to confirm that relationships persisted above and beyond potential confounding variables. Site and family were included as random intercepts with the group contrast variable in the first analyses (labeled model 1). If the first model contrasts were significant, models were then run with the group variable, potential confounding fixed effects (sociodemographics), and nested random effects (labeled model 2). Sensitivity analyses were run with additional covariates as model 3: prenatal exposure, self-reported substance use, and parental history of substance use problems. Finally, cannabis models included a dichotomous variable indicating STS exposure; in nicotine models, SCS was included. Benjamini-Hochberg (BH) false discovery rate corrections (43) were applied to all p values within the first model to mitigate the issue of heightened false discovery for both cannabis and tobacco exposure.

As noted above, cannabis and tobacco use in adolescence are associated with domain-specific cognitive deficits (34–37). Thus, secondary analyses were conducted with each task considered as a dependent variable. The same exposure group contrasts were used, with the exception that the any exposure group was not included to investigate substance-specific correlates. Separate linear mixed-effects models predicted cognitive performance across each domain by exposure group contrast. Hierarchical models were run as described above. Seven outcomes per contrast were examined, with BH corrections within the first models (i.e., seven models with two p values each for two contrasts; 28 p values). The next model was run if the group contrast was significant after correcting for multiple comparisons. Finally, sensitivity analyses were run (model 3) for significant contrasts at model 2. Outcome variables were standardized to facilitate interpretation.

Data Missingness. A total of 5580 participants out of 6571 (85%) of participants in ABCD Annual Release 3.0 had complete core variables (i.e., neurocognitive, secondhand/environmental exposure, and sociodemographic data), and individual neurocognitive tasks were missing for 1% to 4% of

participants. Because most cases were complete, we expected minimal bias and loss in statistical power conducting analyses with completed cases only. Moreover, because we could not confirm whether data were missing completely at random, alternative missing data methods (e.g., multiple imputation) may provide minimal (if any) improvements to power, accuracy, and precision of estimated effects in this data scenario (44). Thus, we elected to use listwise removal of missing data as a viable missing data strategy.

RESULTS

Sociodemographics and Other Confounds

Tables 1 and 2 contain sociodemographics by cannabis and tobacco grouping, respectively. Participants were 10.58 to 13.58 years old. Sociodemographics for this subsample of ABCD and the full baseline cohort are presented in Table S1. Only 30 (<1%) youth self-reported alcohol, tobacco, or cannabis use (more than a sip or puff).

Principal Component Analysis

All seven neurocognitive outcomes were included in the principal component analysis. Eigenvalues are provided in the scree plot in Figure S1. The first component consisted of an eigenvector of 8.77, constituting 63% of the shared variance among all seven neurocognitive predictors, indicating a global cognition component. Variables were moderately to strongly correlated (r = 0.52–0.69). The next component had an eigenvector of 1.91 with 14% shared variance and consisted primarily of flanker and pattern comparison performance. Given that these tasks are subsumed within the first component and the amount of variance shared within this first component relative to the next component, only the first component was chosen for further analysis.

Model 1: Smoke Exposure and Global Cognition Without Sociodemographic Controls

The first models predicted cognitive performance from smoke exposure, controlling for random effects of site and family, without controlling for sociodemographic covariates (Tables 1 and 2). In the cannabis model, after BH corrections, there were no significant relationships with global cognition. For the tobacco model, both STS and Envi-T were associated with lower global cognitive performance (BH-corrected p values $< .001$). Similarly, in the model jointly considering cannabis and tobacco, any secondhand (b = −0.26, p = .002, BH p = .005, partial R^2 = 0.002, 95% CI, −0.42 to −0.09) or environmental (b = −0.07, p = .03, BH p = .03, partial R^2 = 0.001, 95% CI, −0.14 to −0.01) exposures were associated with lower global cognition performance.

Model 2: Environmental Exposure and Cognition With Core Sociodemographic Covariates

Significant contrasts were run again controlling for random effects of site and family and fixed effects of sociodemographic factors. There were no significant relationships between exposure groupings and global cognitive performance (p values $> .05$; see the Supplement for model details).

Table 1. Sociodemographics and Other Confounds by Cannabis Exposure Group

Variable	SCS, n = 88	Envi-C, n = 737	No Exposure, n = 4755	p Value[a]
Age, Years, Mean (SD)	11.81 (0.63)	11.94 (0.64)	11.97 (0.64)	p = .054
Female, %	53%	47%	48%	p = .57
Parental Education, %				p = .15
<High school	1%	2%	3%	
High school	7%	6%	7%	
Some college	31%	26%	25%	
Bachelor's	28%	31%	28%	
Postgraduate	33%	35%	37%	
Income, %				p = .08
<$50,000	35%	24%	27%	
≥$50,000 and <$100,000	24%	33%	29%	
≥$100,000	41%	43%	44%	
Race/Ethnicity, %				$p < .001$[b]
Asian	0%	1%	2%	
Black	14%	9%	11%	
Hispanic	15%	16%	19%	
Other	15%	13%	9%	
White	57%	61%	58%	
Prenatal Exposure, %	66%	52%	29%	$p < .001$[b]
Parental History Substance Problems, %	39%	26%	18%	$p < .001$[b]
Self-reported Substance Use, %	1%	1%	1%	p = .06

No exposure indicates no exposure (secondhand or environmental). Sum totals of categories that are less than 100% are due to participants reporting "Don't Know" or "Refuse to Answer"; sum totals of categories that are greater than 100% are due to rounding.

ANOVAs, analyses of variance; Envi-C, environmental cannabis exposure; SCS, secondhand cannabis smoke.

[a]p values by χ^2 and ANOVAs between groups.

[b]Indicates stastically significant results wherein $p < .05$.

Table 2. Sociodemographics and Other Confounds by Tobacco Exposure Group

Variable	STS, n = 144	Envi-T, n = 746	No Exposure, n = 4690	p Value[a]
Age, Years, Mean (SD)	11.87 (0.59)	11.95 (0.65)	11.96 (0.64)	p = .23
Female, %	46%	46%	48%	p = .86
Parental Education, %				$p < .001$[b]
<High school	10%	4%	3%	
High school	19%	11%	6%	
Some college	56%	41%	21%	
Bachelor's	13%	25%	29%	
Postgraduate	2%	3%	41%	
Income, %				$p < .001$[b]
<$50,000	67%	40%	23%	
≥$50,000 and <$100,000	27%	33%	29%	
≥$100,000	6%	27%	47%	
Race/Ethnicity, %				$p < .001$[b]
Asian	0%	1%	2%	
Black	28%	13%	10%	
Hispanic	13%	21%	18%	
Other	10%	15%	9%	
White	49%	51%	60%	
Prenatal Exposure, %	74%	57%	28%	$p < .001$[b]
Parental History Substance Problems, %	44%	34%	16%	$p < .001$[b]
Self-reported Substance Use, %	0%	1%	1%	p = .40

No exposure indicates no exposure (secondhand or environmental). Sum totals of categories that are less than 100% are due to participants reporting "Don't Know" or "Refuse to Answer"; sum totals of categories that are greater than 100% are due to rounding.

ANOVAs, analyses of variance; Envi-T, environmental tobacco exposure; STS, secondhand tobacco smoke.

[a]p values by χ^2 and ANOVAs between groups.

[b]Indicates stastically significant results wherein $p < .05$.

Secondary Task-Based Analysis

Given that substance use may be associated with domain-specific deficits that would not be detected on a global cognition measure, task-based analyses were considered (Tables 3–5).

Cannabis Models. SCS was marginally associated with poorer Little Man visual task performance (BH p = .08). Envi-C was associated with better oral reading (BH p = .004) and marginally associated with better picture vocabulary (BH p = .09). There were no significant relationships between any level of cannabis exposure and the other tasks.

After controlling for sociodemographics, the relationship between Envi-C and oral reading remained (p = .004) (Table S5 for full models).

When including other covariates (prenatal substance use, parental substance problems, STS), oral reading and Envi-C exposure were still significantly related (b = 0.09, p = .01, partial R^2 = 0.001, 95% CI, 0.02 to 0.18) (Table S6).

Tobacco Models. Both STS and Envi-T were associated with poorer picture vocabulary (STS: BH $p < .001$; Envi-T: $p < .001$, BH $p \leq .001$), picture memory (STS: BH p = .07; Envi-T: BH p = .07), oral reading (STS: BH p = .002; Envi-T: BH $p < .001$), visuospatial (STS: BH p = .001; Envi-T: BH $p < .001$), and Rey Auditory Verbal Learning Task performance (STS: BH p = .002; Envi-T: BH $p < .001$). STS was associated with poorer pattern comparison processing speed (BH p = .09).

After controlling for sociodemographics, poorer picture vocabulary (p = .03) and poorer visuospatial skills (p = .03) remained significantly associated with Envi-T, while poorer picture memory was associated with STS (p = .01). Full models with covariates are presented in Table S7.

Finally, with all covariates, Envi-T remained associated with poorer picture vocabulary (p = .008) and visuospatial skills (p = .02) and STS remained associated with poorer picture memory (p = .02) (Table S8 for full model details).

DISCUSSION

In this novel and preliminary investigation, caregiver-reported secondhand cannabis and tobacco exposure were associated with modest differences in global cognitive performance only when not accounting for sociodemographic or other confounds. In assessing domain-specific performance by smoke exposure groupings, STS remained significantly related to poorer picture memory, Envi-T exposure was related to poorer picture vocabulary and visuospatial performance, and Envi-C exposure was related to better oral reading after considering all potential covariates. SCS was approaching significance with poorer visual task performance prior to inclusion of covariates. Partial R^2 is largely invariant or even increases with the inclusion of covariates of most models, and all effect sizes were small. While a growing body of literature has considered how personal cannabis use may impact cognition in adolescents (45,46), involuntary exposure through secondhand and environmental exposure is underrepresented in the field. Similarly, despite knowledge of health consequences

Table 3. Environmental Exposure and Domain-Specific Cognitive Performance With Random Effects and Without Confounds

	b	95% CI	Partial R^2	p	BH p	b	95% CI	Partial R^2	p	BH p
Cognitive Outcome	SCS vs. No Exposure					Environmental Cannabis vs. No Exposure				
Global Cognition	0.09	−0.27 to 0.15	<0.001	.55	.55	0.09	0.01 to 0.17	0.001	.03	.06
Picture Vocabulary	0.02	−0.19 to 0.23	<0.001	.22	.89	0.08	0.001 to 0.16	<0.001	.046	.09
Flanker	−0.05	−0.26 to 0.16	<0.001	.65	.76	0.04	−0.04 to 0.12	<0.001	.29	.51
Pattern Comparison	0.001	−0.21 to 0.21	<0.001	.99	.99	0.005	−0.07 to 0.08	<0.001	.90	.94
Picture Memory	−0.06	−0.28 to 0.15	<0.001	.56	.70	0.03	−0.05 to 0.11	<0.001	.46	.67
Oral Reading	−0.08	−0.14 to 0.29	<0.001	.48	.67	0.13	0.05 to 0.21	0.002	.001	.004[a]
Little Man	−0.23	−0.44 to −0.02	0.001	.03	.08	0.04	−0.04 to 0.12	<0.001	.34	.156
RAVLT	−0.06	−0.27 to 0.15	<0.001	.57	.70	0.02	−0.06 to 0.10	<0.001	.56	.22
	STS vs. No Exposure					Environmental Tobacco vs. No Exposure				
Global Cognition	−0.46	−0.63 to −0.28	0.006	<.001	<.001[a]	−0.22	−0.30 to −0.14	0.006	<.001	<.001[a]
Picture Vocabulary	−0.36	−0.54 to −0.19	0.004	<.001	<.001[a]	−0.23	−0.31 to −0.15	0.007	<.001	<.001[a]
Flanker	−0.07	−0.24 to 0.10	<0.001	.42	.65	−0.01	−0.09 to 0.06	<0.001	.72	.80
Pattern Comparison	−0.18	−0.35 to −0.002	0.001	.047	.09	−0.06	−0.14 to 0.02	<0.001	.17	.32
Picture Memory	−0.45	−0.63 to −0.28	0.006	<.001	<.001[a]	−0.09	−0.17 to −0.01	0.001	.03	.07
Oral Reading	0.32	−0.49 to −0.14	0.003	<.001	.002[a]	−0.17	−0.25 to −0.09	0.004	<.001	<.001[a]
Little Man	−0.32	−0.49 to −0.15	0.003	<.001	<.001[a]	−0.17	−0.25 to −0.09	0.004	<.001	<.001[a]
RAVLT	−0.30	−0.54 to −0.19	0.003	<.001	.001[a]	−0.17	−0.31 to −0.15	0.004	<.001	<.001[a]

Global cognition was run separately from and before the specific-domain models and BH corrections were thus separate. Partial R^2 was extracted for fixed effect. Each model included a cognitive outcome predicted by smoke exposure group and random effects (site and family).

BH, Benjamini-Hochberg; RAVLT, Rey Auditory Verbal Learning Task; SCS, secondhand cannabis smoke; STS, secondhand tobacco smoke.

[a]Significant ($p < .05$) results.

linked to STS in utero or in early childhood (47), there is limited research considering the cognitive impact of STS in older youth (19).

No SCS and cognition relationships survived correction for multiple comparisons, although visuospatial performance was marginally related to SCS within the first models establishing potential relations. Notably, our study consisted of only 88 preadolescents whose caregivers reported that the child had been exposed to SCS (1.6% of the sample); because other research suggests that nearly 7% of adults with children in the home use cannabis (11), it may be that there was not sufficiently accurate reporting to detect relationships and/or too small of a sample. Results should be interpreted with caution and require replication. Future research should also devise and then employ methods for objective measurement of Envi-C exposure, because scant methods currently exist (48). Despite null relationships, this study adds to the growing literature related to SCS, such as that secondhand exposure is more common in individuals in lower income areas (12,13) and has potential associations with increased adverse health

Table 4. Environmental Exposure and Domain-Specific Cognitive Performance With Random Effects and Sociodemographic Covariates

	b	95% CI	Partial R^2	p	b	95% CI	Partial R^2	p
Cognitive Outcome	SCS vs. No Exposure				Environmental Cannabis vs. No Exposure			
Global Cognition	0.006	−0.18 to 0.20	<0.001	.95	0.07	−0.001 to 0.14	<0.001	.05
Oral Reading	–	–	–	–	0.11	0.04 to 0.2	0.002	.004[a]
	STS vs. No Exposure				Environmental Tobacco vs. No Exposure			
Global Cognition	−0.05	−0.21 to 0.11	<0.001	.57	−0.07	−0.14 to 0.007	<0.001	.08
Picture Vocabulary	−0.03	−0.13 to 0.19	<0.001	.70	−0.08	−0.15 to −0.01	0.001	.03[a]
Picture Memory	−0.21	−0.20 to 0.14	<0.001	.01[a]	−0.001	−0.09 to 0.07	<0.001	.99
Oral Reading	0.02	−0.15 to 0.19	<0.001	.80	−0.05	−0.13 to 0.03	<0.001	.21
Little Man	−0.09	−0.26 to 0.08	<0.001	.31	−0.09	−0.17 to −0.01	0.001	.03[a]
RAVLT	−0.05	−0.22 to 0.12	<0.001	.58	−0.08	−0.16 to 0.001	0.001	.05

Global cognition was run separately from and before the specific-domain models. No SCS results survived corrections in the first model and so no further SCS models were run. Partial R^2 was extracted for fixed effect. Each model included a cognitive outcome predicted by smoke exposure group and random effects (site and family) and fixed effects of sociodemographics (parental education, household income, race/ethnicity, sex, and age).

RAVLT, Rey Auditory Verbal Learning Task; SCS, secondhand cannabis smoke; STS, secondhand tobacco smoke.

[a]Significant ($p < .05$) results.

Table 5. Environmental Exposure and Domain-Specific Cognitive Performance With Random Effects, Sociodemographic Covariates, and Other Relevant Confounds

	b	95% CI	R^2	p	b	95% CI	R^2	p
Cognitive Outcome	STS				Environmental Tobacco			
Picture Vocabulary	−0.002	−0.19 to 0.15	<0.001	.83	−0.1	−0.18 to −0.03	0.002	.02[a]
Picture Memory	−0.18	−0.38 to −0.03	0.001	.02[a]	0.02	−0.08 to 0.09	<0.001	.66
Little Man	−0.11	−0.27 to 0.08	<0.001	.19	−0.10	−0.17 to −0.01	0.001	.02[a]

Partial R^2 was extracted for fixed effects. Each model included a cognitive outcome predicted by smoke exposure group and random effects (site and family) and fixed effects of sociodemographics (parental education, household income, race/ethnicity, sex, age) and other controlled variables (prenatal exposure, parental history of alcohol/substance problems, self-reported substance use, or SCS).

SCS, secondhand cannabis smoke; STS, secondhand tobacco smoke.

[a]Significant ($p < .05$) results.

outcomes in children (18). Given the rise in legalization of cannabis with a myriad of different approaches to governing laws, it is important to understand the breadth of the impact cannabis may have. If null findings persist with future study, this will be beneficial to know. Regardless, it is important to understand the potential impact, if any, of SCS, particularly among those already disproportionately affected by system inequities, including economic disadvantage.

STS is also concerning for cognition in youth, because poorer visual memory performance was exhibited by youth exposed to STS and was robust to consideration of potential confounders. Preclinical research suggests potential mechanisms of action, because STS in young mice is associated with altered BDNF (brain-derived neurotrophic factor); decreased synaptogenesis in the hippocampus, prefrontal cortex, striatum, and cerebellum; and impaired myelination (49–51); these mice also demonstrated memory deficits (49) and anxious behaviors (50). STS-exposed adolescent rats demonstrated regionally specific increased orbitofrontal cortex excitation with reported corresponding behavioral inhibition deficits (52). Other clinical research suggests that STS confers risk for cognitive deficits (20,53). As with SCS, the percentage reporting secondhand exposure to tobacco may be underreported because only 2.5% of caregivers reported direct STS, while another study found that 20% of adults with children in the home reported smoking cigarettes (11). The ABCD Study has recently broadened its collection of urinary cotinine (a primary metabolite of nicotine), which provides more objective measurement of nicotine exposure. Future analyses should include this data because it may provide greater opportunities to tease apart self-report and biological measurement of tobacco and associated outcomes.

After controlling for sociodemographic and other factors, only two correlates of Envi-T exposure were revealed, such that Envi-T was associated with poorer picture vocabulary and visuospatial skills. Although independent analyses of Envi-T with cognition revealed lower cognitive performance across various cognitive tasks, these findings were attenuated when controlling for potential confounding sociodemographic factors, including SES (household income, parental education); small effect sizes also remained largely consistent regardless of exact model specifications. While lower SES has been correlated with lower cognitive performance, SES is also linked to increased risk of social adversity due to systemic inequities that directly influence the quality of their environments. Indeed, creators of the NIH Toolbox caution interpretation of results due to inherent limitations in the norming population (54), and other ABCD analyses have found robust links between SES and cognition (55). Further, the sociodemographic makeup by exposure group is not dissimilar to other national studies (28). Thus, it is unclear to what extent differences in cognitive performance may be attributable to systemic inequities in the environments of children or to the cultural biases in the cognitive tasks and, in these analyses, attenuate the associations for environmental smoke exposure. Indeed, this is consistent with a systematic review that found that, while there are some intellectual and attention deficits in youth exposed to STS, adding socioeconomic and other factors complicates the picture (19). A more thorough analysis of how individual sociodemographics and larger systemic issues moderates and interacts with substance exposure in youth is needed.

When assessing Envi-C exposure, youth with exposure exhibited modestly better oral reading. Several explanations are considered. First, while sociodemographic differences in the Envi-C group are modest (i.e., significant differences by race/ethnicity; marginal differences by household income), inclusion of sociodemographics attenuated relationships, suggesting that systemic issues may be present. The exact determination of these differences is difficult, particularly because race/ethnicity is a social construct and a proxy for numerous other factors that should be appropriately considered and contextualized (56), and the influence of SES on cognition in ABCD participants (55) may prevent distinguishing between factors common across groups (e.g., lower SES and smoke exposure). Further, oral reading itself has been suggested as better representing educational opportunities than broader functioning (57) and is more experience based (58). It may also be that parents who use cannabis but not around their children are more aware of potential harms and take appropriate preventive actions; may be using cannabis for medicinal reasons, allowing for potential improved functioning, which could lead to positive outcomes for the entire family; and/or may be using a minimal volume of cannabis (e.g., one joint a week) relative to what one might experience with tobacco (e.g., one pack a day), reducing the potential impact. More research is needed to evaluate whether this finding remains stable over time and across different sociodemographic conditions.

Unique strengths of this study include its large, diverse sample; robust cognitive battery; and consideration of important novel secondhand and environmental smoke factors. The study is limited by its self-report nature of exposure only in the

home/household vehicles. Presentation biases may have prevented some parents/guardians from fully disclosing details regarding substance use details. It is also possible that environmental smoke may be present in some children's homes from more traditional sources of thirdhand smoke such as living in older buildings where previous occupants had smoked. Robust detection methods of second- and thirdhand smoke should be utilized in the future to carefully assess levels of exposure and other health and cognitive outcomes. Here, we used a dichotomous variable to control for prenatal exposure to any of the three most common drugs of abuse; however, other literature suggests that cannabis use after maternal knowledge of the pregnancy may be an important factor in later cognitive outcomes (59). Future research should continue to investigate more nuanced data regarding prenatal substance use. Effect sizes were modest, which, while not inconsistent with a study of this size (60), raises the question of clinical significance. As a cross-sectional analysis, no causality can be established; however, future data releases from the ABCD Study will allow for ongoing monitoring of these participants and implementation of causal inference models. Finally, interpretation of development is complex (61) and, as noted above, identifying the unique effects of secondhand or environmental smoke exposure is difficult due to the myriad of factors that contribute to an individual's environment, including sociodemographic factors (19). Thus, caution is warranted in not overinterpreting findings.

Taken together, preliminary findings indicate that STS is associated with poorer visual memory and Envi-T is associated with poorer picture vocabulary and visuospatial skills in preadolescent youth, while Envi-C is associated with better reading ability. Even so, given the vast changes in cannabis legalization policy, potential for adverse health events associated with SCS, and the lack of perceived harm from SCS, continued assessment of potential public health concerns that SCS may present are needed. Further, continued efforts to reduce STS exposure in youth are needed.

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The ABCD data repository grows and changes over time. The ABCD data used in this report came from ABCD Release 3.0 (DOI: 10.15154/1519007).

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