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Indoor cannabis smoke and children's health

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ABSTRACT

Cannabis use is increasing and cannabis is typically consumed by smoking. This study explored how indoor secondhand cannabis smoke (SCS) was associated with child health. As part of a larger trial, air particle monitors were placed in 298 homes of families with at least one cigarette smoker and one child under 14 years old in San Diego County, California. Assessment included past 7-day indoor cigarette and cannabis use, the youngest child's exposure to cigarette smoke, and 5 smoke-related past-year child health outcomes: emergency department use for coughing/difficulty breathing; physician diagnosis of ear infection, bronchitis/bronchiolitis, asthma, or eczema/atopic dermatitis. An ordinal measure of adverse health outcomes (0, 1, or ≥2) was regressed on reported indoor cannabis smoking—the main measure of exposure (yes/no). Of 221 parents/guardians asked about cannabis use, 192 (86.9%) provided all required data, and 29 (15.1%) reported indoor cannabis smoking; reports were supported by air particle data. Homes without indoor smoking had lower average 7-day particle concentrations (1968 particles/0.01ft^3) than homes with cannabis smoking only (3131 particles/0.01ft^3), cigarette smoking only (3095 particles/0.01ft^3), or both cigarette and cannabis smoking (6006 particles/0.01ft^3). Odds of reporting a greater number of adverse health outcomes were 1.83 (95% CI = 0.89–3.80, $p = 0.10$) times higher for children of families with indoor cannabis smoking vs families without cannabis smoking, after controlling for exposure to cigarette smoke and other covariates. Our results do not indicate a statistically significant association. However, the magnitude of the (non-significant) association between indoor cannabis smoking and adverse health outcomes warrants more studies.

1. Introduction

An estimated 24.0 million persons were current cannabis users in the United States (U.S.) in 2016 and this number has been increasing (Hasin, 2018; Substance Abuse and Mental Health Services Administration, 2017). As of January 2018, the District of Columbia and 8 states in the U.S. had legalized cannabis for both recreational and medicinal use (Hasin, 2018), a phenomenon that coincides with a steady decline in the perception that cannabis use is "risky" to health (Johnston et al., 2016). Smoking is the most common method of cannabis consumption (Schauer et al., 2015). Cannabis smoking most often occurs indoors (Berg et al., 2018, 2015), where it is associated with high concentrations of air particles (Klepeis et al., 2017) that can be inhaled by children and other nonsmokers in the home. In 2015, 5.3 to 8.0 million children in the U.S. lived with a parent who was a current cannabis user, and among parents, both current cannabis use and daily use are increasing (Goodwin et al., 2018). Despite the expansion of legalization and use, there are no laws or restrictions in place to protect children from exposure to cannabis smoke.

Secondhand smoke (SHS) is the smoke exhaled by a smoker or smoke from combusted products such as cigarettes and cannabis. Current literature supports a causal association of exposure to secondhand tobacco smoke (STS) from cigarettes with illnesses in children such as cardiovascular and respiratory damage (Ferrante et al., 2013; Franklin, 2007; Kumar et al., 2015; Li et al., 1999; Patra et al., 2012; Strachan and Cook, 1998). Tobacco and cannabis smoke contain many

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of the same chemicals, such as nitric oxide and aldehydes, which are known to have harmful health effects (Moir et al., 2008). Given that STS and secondhand cannabis smoke (SCS) particles are equally capable of entering the body and have a similar chemical composition (Matt et al., 2004; Wilson et al., 2017), studies of the health effects of SCS are warranted.

Children are more sensitive to respiratory insult from STS exposure because of their underdeveloped respiratory and immune systems (Miller et al., 2002; Ostro et al., 2009). According to the U.S. Surgeon General Reports, the most common health outcomes of child exposure to STS are ear infections, asthma attacks, and poor respiratory health (Centers for Disease Control and Prevention (US), 2010; US Department of Health and Human Services, 2006, 2014). Children may also be exposed to thirdhand smoke (THS), which is SHS that accumulates in dust and on surfaces and includes similar carcinogens and toxicants found in SHS (Matt et al., 2011, 2004). THS exposure can result from contact with contaminated surfaces where STS has settled, from re-emitted particles or gasses, or from new pollutants created when residual smoke components react with other environmental factors (Matt et al., 2011, 2004). Children who have been exposed to THS from tobacco had higher odds of atopic eczema (Kramer et al., 2004). It is unknown whether cannabis exposure (either secondhand or thirdhand) is similarly related to health outcomes since few studies have been conducted on the topic, in part due to the federal illegality of cannabis (Drug Enforcement Administration: Diversion Control Division, 2018).

Cannabis smoking is implicated in several adverse health-related outcomes in adults (e.g. symptoms of chronic bronchitis and behavioral change), while evidence of therapeutic effects and the effects of long-term use remain inconsistent (Volkow et al., 2014a, 2014b). The only evidence of SCS-related cardiovascular health effects is from a recent study in rats that showed SCS exposure reduced vascular function more than STS exposure, suggesting cannabis smoke may have worse cardiovascular consequences than tobacco (Wang et al., 2016). In children, the research literature pertaining to the health consequences of cannabis exposure are limited to cases of accidental ingestion (Amirav et al., 2011; Fried and Watkinson, 1990; Macnab et al., 1989; Wang et al., 2011).

This paper explored the relationship between reported indoor SCS and health outcomes in children. Given that there is no risk-free level of exposure to STS (US Department of Health and Human Services, 2006), and the qualitative similarity in chemical composition between SHS from cigarettes and cannabis (Moir et al., 2008), it is plausible that SCS could cause ear infections, asthma, other respiratory dysfunction, and skin conditions. To our knowledge, there is no research on the effects of indoor SCS exposure on health outcomes in children.

2. Methods

2.1. Study design

We examined data from *Project Fresh Air* (PFA), a randomized controlled trial of real-time feedback and coaching aimed to reduce indoor SHS and fine particle levels in low-income homes throughout San Diego County, CA. The cross-sectional analyses presented in this paper used data collected during the period of PFA before any intervention took place.

2.2. Participants

Families were eligible for enrollment if the primary adult participant was at least 18 years old, with no plans of moving for 3 months, and if the household included at least one adult cigarette smoker and at least one child under the age of 14. In each household, one parent/guardian and the youngest child were enrolled. Details of participant recruitment and enrollment are reported elsewhere (Hughes et al., 2018). Two hundred and ninety-eight participating families enrolled. Parents (or guardians) provided written informed consent for inclusion in this study. Study procedures were approved by the San Diego State University Institutional Review Board (IRB).

2.3. Measures

Research assistants (RAs) placed customized Dylos DC1700 air particle monitors in participant homes in the room where the most smoking took place. The monitors counted indoor air particles (0.5–2.5 μm in diameter), which have previously been used to detect indoor smoking (United Sates Environmental Protection Agency, 2016). Air particle monitors continuously collected data throughout the period of study participation. After approximately 7 days of air particle monitoring, RAs administered computer-assisted interviews to assess household behaviors that may affect indoor $PM_{2.5}$ (particulate matter ≤ 2.5 μm) levels (e.g. smoking and air ventilation), demographic information, and health information about the youngest child in the family.

2.3.1. Primary outcome

Parents (or guardians) reported their child's 1) past year use of an emergency department (ED) for coughing or difficulty breathing, and whether in the past year the child had a physician-diagnosed 2) ear infection, 3) bronchitis or bronchiolitis, 4) asthma, or 5) skin conditions such as eczema or atopic dermatitis. From these five survey responses, the cumulative number of health outcomes was computed for each child and categorized into three levels (0, 1, or ≥ 2 outcomes).

2.3.2. Cannabis and tobacco smoke exposure

We measured the enrolled child's potential exposure to cannabis and cigarette smoke with three dichotomous variables derived from responses to three questions. SCS exposure, the primary exposure of interest for this study, was derived from the question, “How often in the past 7 days did anyone smoke medicinal or recreational marijuana in your home?” Responses were coded 1 if one or more days were reported, and 0 otherwise. Exposure to cigarette smoke, which served as our main measure of STS exposure, was derived from the question: “In the past 7 days, was [child's name] exposed to any cigarettes in your home, a car, or any other place?” In a sensitivity analysis, we used the response to the following question as an alternate measure of STS exposure: “How often in the past 7 days did anyone smoke cigarettes in your home?” Responses were coded 1 if one or more days were reported, and 0 otherwise.

2.3.3. Air particle concentrations

Reported indoor cannabis and cigarette smoking behaviors were compared to using 7-day mean air particle concentrations. Air particle monitors counted indoor air particles each second and averaged particle counts every 10 s. Arithmetic mean particle concentrations (counts per 0.01 cubic foot) were computed for each day.

2.3.4. Potential covariates

For the child, the main demographics of interest were age (years), gender (male/female), and race/ethnicity (non-Hispanic White, Hispanic, non-Hispanic Black, or non-Hispanic Other). “Other” included Native American, Asian, Pacific Islander, mixed and unspecified. For the parent or guardian, years of education were categorized as $<$ 12 years, 12 years, and $>$ 12 years and served as a proxy for socioeconomic status.

2.4. Statistical analysis

Of the 298 families enrolled in PFA, 6 participants were asked about cannabis use but declined to answer and 78 were not asked about cannabis because they were either pilot participants ($n = 36$), or participants ($n = 26$) recruited from a group for whom we did not have IRB

approval to inquire about cannabis use. An additional 16 participants were not asked about cannabis due to interviewer error (administration of the wrong interview). Of those who answered the cannabis use question, 19 did not provide all health information about their youngest child, 2 did not provide information about education, and 1 did not answer the SCS exposure question. Therefore, data from 192 families were available for the present study.

Health outcomes and covariates were summarized using frequencies and means for the total sample and stratified by indoor cannabis smoking. Group differences were tested using Pearson chi-square test, Fisher's Exact Test, and *t*-tests.

Reports of indoor smoking behaviors were confirmed using air particle concentrations. Daily arithmetic mean air particle concentrations were log transformed to better approximate normal distributions. Geometric means (GMs) were computed to summarize particle concentrations during the 7-day period corresponding with the interview for each smoking behavior group: no smoking, cigarette smoking only, cannabis smoking only, or both cigarette and cannabis smoking. Overall group differences were tested using one-way ANOVA. To test the hypothesis that any type of indoor smoking, in relation to no smoking, would increase particle concentrations, between-group differences were computed using Tukey multiple comparison tests.

Ordinal logistic regression was used to assess the relationship of indoor cannabis smoking (yes/no) with the ordinal measure of adverse health outcomes (0, 1, or ≥2 outcomes), adjusting for covariates. Covariates were included in the models if they were related to the number of adverse health outcomes using a threshold of $p < 0.40$ for inclusion. Three ordinal regression models were fit: Model 1 was unadjusted; Model 2 adjusted for two covariates (child's age and parent's education); and to test whether associations were independent of exposure to tobacco smoke, Model 3 added a third covariate (reported child exposure to cigarette smoke). As a sensitivity analysis, we repeated our three-model approach, replacing child exposure to cigarette smoke with reported indoor cigarette smoking. Proportional odds assumptions were tested for each model using the *brant()* function in R (R Foundation for Statistical Computing; Vienna, Austria); no violations were observed (Schlegel and Steenbergen, 2018).

All statistical tests were two-tailed with an alpha of 0.05. Data analysis was performed using R (version 3.5.1 for Mac OS X).

3. Results

3.1. Sample characteristics

Indoor cannabis smoking was reported by 29 (15.1%) parents or guardians (Table 1). Indoor cigarette smoking was most prevalent (48.1%) among those who reported indoor cannabis smoking ($p < 0.01$). Children in cannabis-smoking homes were significantly older (4.72 years) than those who were not (4.12 years; $p < 0.01$). Among enrolled parents or guardians who did not report indoor cannabis smoking, most (65.0%) reported > 12 years of education ($p = 0.03$). While enrolled child racial-ethnic backgrounds among households that reported indoor cannabis smoking were relatively evenly distributed, the majority (55.2%) of enrolled children in households that did not report indoor cannabis smoking were Hispanic ($p = 0.01$). Additional sample characteristics by indoor cannabis smoking status are provided in Table 1.

3.2. Air particle concentration results

GM particle concentrations (counts/0.01ft^3) were used to test our hypothesis that homes with any type of smoking would have higher 7-day average particle levels than non-smoking homes (Table 2). Cannabis-only homes, cigarette-only homes, and dual-smoking homes were all significantly higher in GM particle concentrations than homes that did not report indoor smoking (all p's < 0.05). There was no difference in particle concentrations between cigarette-only and cannabis-only homes ($p = 0.99$) and both groups had higher concentrations than non-smoking homes (p's < 0.05). Overall, results confirmed our assumption of higher particle concentrations in homes that reported indoor cannabis or cigarette smoking, and showed twice as high concentrations in homes with both indoor cannabis and cigarette smoking.

Table 1
Child and household characteristics by indoor cannabis smoking status among study participants in San Diego County, CA ($N = 192$).

Characteristic	No indoor cannabis smoking ($n = 163$) n (%)	Any indoor cannabis smoking ($n = 29$) n (%)	p-value[a]
Past 7-day child exposure to cigarette smoke			0.62
No	51 (31.3)	11 (37.9)	
Yes	112 (68.7)	18 (62.1)	
Indoor cigarette smoking[b]			**< 0.01**
No	127 (78.9)	15 (51.7)	
Yes	34 (21.1)	14 (48.3)	
Age of child[c] (years)	4.12 (3.70)	4.72 (3.84)	**< 0.01**[d]
Gender of child			0.56
Male	86 (52.8)	13 (44.8)	
Female	77 (47.2)	16 (55.2)	
Parent education (years completed)			**0.03**
< 12	30 (18.4)	4 (13.8)	
12	27 (16.6)	11 (37.9)	
> 12	106 (65.0)	14 (48.3)	
Race/ethnicity of child			**0.01**[e]
Hispanic	90 (55.2)	7 (24.1)	
Non-Hispanic Black	20 (12.3)	6 (20.7)	
Non-Hispanic White	25 (15.3)	7 (24.1)	
Non-Hispanic other[f]	28 (17.2)	9 (31.0)	

Bolded p-values indicate statistical significance at $p < 0.05$.
[a] *P*-values are derived from Pearson chi-square tests to test for group differences.
[b] $n = 190$ due to missing responses.
[c] Indicates use of mean and standard deviation.
[d] Indicates *p*-values from two-sample *t*-tests with equal variance.
[e] Indicates p-values from Fisher's Exact Test, used when expected cell sizes were < 5.
[f] "Non-Hispanic Other" includes: Native American, Asian, Pacific Islander, mixed, unspecified.

Table 2
Geometric mean 7-day indoor air particle concentrations by smoking group among study participants in San Diego County, CA (N = 192).

Reported indoor smoking behavior	Mean[a]	SD	p-value[b]
No smoking	1968	1.71	reference
Cannabis only	3131	2.10	**0.01**
Cigarette only	3095	1.85	**< 0.01**
Cannabis and cigarette	6006	2.42	**< 0.01**

There were no significant differences between the means of the "Cannabis only" and "Cigarette only" groups ($p = 0.99$).
The "Cannabis and cigarette" group was significantly different from all other smoking groups (p's < 0.05).
Bolded p-values indicate statistical significance at $p < 0.05$.
[a] Indicates means derived from unadjusted geometric mean particle concentrations (counts/0.01ft^3) from last 7 days before baseline interview.
[b] P-values from Tukey multiple comparisons of geometric means comparing smoking behaviors to "No smoking."

Table 3
Frequency of past-year adverse health outcomes by indoor cannabis smoking behavior among study participants in San Diego County, CA (N = 192).

Outcome		No indoor cannabis smoking	Any indoor cannabis smoking	p-value[a]
		n (%)	n (%)	
Cumulative health outcomes[b]				**0.04**
	0	86 (52.8)	9 (31.0)	
	1	40 (24.5)	13 (44.8)	
	≥2	37 (22.7)	7 (24.1)	
ED visits[c]				0.58[d]
	None	137 (84.1)	26 (86.7)	
	Any	26 (15.9)	3 (10.3)	
Ear infection				0.77[d]
	No	140 (85.9)	24 (82.8)	
	Yes	23 (14.1)	5 (17.2)	
Bronchitis/bronchiolitis				0.67[d]
	No	154 (94.5)	27 (93.1)	
	Yes	9 (5.5)	2 (6.9)	
Asthma				0.99[d]
	No	143 (87.7)	26 (89.7)	
	Yes	20 (12.3)	3 (10.3)	
Skin conditions				0.96
	No	131 (80.4)	24 (82.8)	
	Yes	32 (19.6)	5 (17.2)	

Bolded p-values indicate statistical significance at an alpha < 0.05.
[a] P-values are derived from Pearson chi-square tests to test for group differences.
[b] Cumulative health outcomes include any reports of ED visits, ear infection, bronchitis/bronchiolitis, asthma, or skin conditions in the past year.
[c] Visits to an emergency department (ED) for coughing or difficulty of breathing.
[d] Indicates p-values derived from Fisher's Exact Test, used when expected cell sizes were < 5.

3.3. Health outcomes

Among the children who lived in households with indoor cannabis smoking, 31.0% had no adverse health outcomes, 44.8% reported 1 outcome, and 24.1% reported 2 or more outcomes Detailed information about specific health outcomes are in Table 3.

In Model 1 (unadjusted), the odds of reporting a greater number of adverse health outcomes were 1.75 (95% CI = 0.86–3.54; $p = 0.12$) times higher for children of families *with* indoor cannabis smoking compared to families *without* indoor cannabis smoking (Table 4). Adjustment for demographic confounders (Model 2) did not substantially change this association (adjusted OR = 1.84; 95% CI = 0.89–3.81; $p = 0.10$). After additional adjustment for exposure to cigarette smoke in the past 7 days (Model 3), the odds of reporting a greater number of adverse health outcomes remained essentially the same (adjusted OR = 1.83; 95% CI = 0.89–3.80; p = 0.10). These results were unchanged in our sensitivity analysis, in which we adjusted for indoor cigarette smoking instead of reported cigarette smoke exposure (Supplemental Table 1). Interactions between cannabis smoke exposure and any of the three covariates were not statistically significant.

4. Discussion

4.1. Summary of findings

To our knowledge, this is the first study of the relationship between reported indoor SCS and health outcomes in children. We observed that children living in homes with indoor cannabis smoke had 83% higher odds of adverse health outcomes compared to children in homes with no indoor cannabis smoking. Our exposure of interest, indoor SCS, did not reach thresholds for statistical significance (p = 0.10), possibly due in part to insufficient statistical power. Notably, however, the magnitude and direction of this association persisted even after further adjustment for two different measures of STS exposure. This suggests that exposure to SCS, independent of exposure to STS, may be related to some of the same adverse health outcomes with which STS has been shown to be associated.

We confirmed reports of indoor smoking with objective measures from air particle monitors, giving us higher confidence in our measure of SCS exposure. The analysis also showed that homes with one indoor smoking behavior had higher particle concentrations than homes without indoor smoking. Homes with both smoking behaviors had two times the daily mean particle concentrations of homes in any other group (all p's < 0.01), suggesting that children may be exposed to more smoke in dual-use homes. This is particularly important given that cannabis users primarily smoke indoors (Berg et al., 2018, 2015) and are more likely to smoke tobacco as well (Goodwin et al., 2018; Moore et al., 2005; Wilson et al., 2017).

4.2. Relation to previous work

Recent studies have examined the acute effects of SCS in rats and human adults. Flow-mediated dilation (FMD) is used to examine vasodilator function and has been shown to be associated with cardiovascular events in humans (Thijssen et al., 2011). One study examined the effects of STS and SCS exposure on femoral artery FMD in rats (Wang et al., 2016). Investigators concluded that both SCS and STS impaired FMD, suggesting that SCS might have the same effect as STS in humans. The authors also found that neither tetrahydrocannabinol (THC), the main psychoactive component in cannabis, nor nicotine were required in their respective SHS for rats to exhibit FMD impairment, suggesting that the cardiotoxic effects of SHS might be

Table 4
Association of number of past-year adverse health outcomes with indoor cannabis smoke exposure, in three ordinal regression models (N = 192).

Variable		Model 1			Model 2			Model 3		
		OR	(95% CI)	p-value	aOR	(95% CI)	p-value	aOR	(95% CI)	p-value
Indoor cannabis smoking				0.12			0.10			0.10
	None	1.00	(reference)		1.00	(reference)		1.00	(reference)	
	Any	1.75	(0.86–3.54)		1.84	(0.89–3.81)		1.83	(0.89–3.80)	

The three-level health outcome variable was used as the dependent variable in all 3 models.
Specifications of independent variables in the 3 models were as follows:
Model 1: Indoor cannabis smoking.
Model 2: Model 1, and age of child, and education level of parent (or guardian).
Model 3: Model 2, and past 7-day child exposure to cigarette smoke.
OR = Odds ratio.
aOR = Adjusted odds ratio.
CI = Confidence interval.

attributable to the smoke rather than the main active ingredients in cigarettes and cannabis. In humans, Herrmann et al. showed that exposing non-smokers to SCS resulted in significantly higher blood/urine THC levels and decreased cognitive ability, effects that depended on the ventilation levels in the smoking chamber (Herrmann et al., 2015). In a clinical sample of children presenting to a Colorado hospital with bronchiolitis, the urinary biomarker for SCS exposure, 11-nor-9-carboxy-THC (COOH-THC), was detected among 16% of those tested (Wilson et al., 2017). The prevalence of exposure was even higher among children with urinary-cotinine-confirmed exposure to cigarette SHS, with 56% testing positive for COOH-THC (Wilson et al., 2017). These findings suggest that children are exposed to both STS and SCS in the real world and their exposure to SHS could have played a role in the children's hospitalization.

To our knowledge, previous research has not explored the health effects of chronic exposure to SCS. However, our current findings and those from Wilson et al. (2017) suggest the possibility of important health effects that warrant further exploration. Given our results and recent rise in cannabis smoking (Hasin, 2018; Substance Abuse and Mental Health Services Administration, 2017), it is crucial for studies investigating the effects of STS exposure on child health to measure and test for effects of SCS. Failure to consider SCS may result in an underestimate of the overall effects of smoke exposure on children. Future epidemiologic studies that include SCS biomarkers and/or measures of exposure duration, the environment in which exposure occurs, and THC potency may better characterize exposure and provide more detail about the nature of associations (Wei et al., 2015). Future longitudinal studies should follow initially healthy children of parents who regularly use cannabis and parents who do not use cannabis to prospectively assess the relationship between SCS and child health outcomes.

5. Strengths and limitations

There are three main strengths to this study. First, examination of the health effects of SCS exposure on children represents a novel and important area of research. Second, the study is timely due to the recent increases in recreational cannabis legalization at the state level in the U.S. Third, subjective measures of smoking behaviors were confirmed using objective measures of air particle concentrations.

Our primary limitation was our measure of potential cannabis exposure to children. Although we obtained parent/guardian reports of child exposure to indoor cigarette smoking, we did not have a parallel question soliciting reports of child exposure to indoor cannabis smoking. Therefore, the best proxy for indoor cannabis smoke exposure was the report of frequency of indoor cannabis smoking.

The analyses were limited by the small proportion of homes in which marijuana smoking was reported. The high ratio of unexposed to exposed limited statistical power. Recreational cannabis use was illegal in California during the PFA study period, and therefore reported measures of indoor cannabis smoking may have been especially prone to reporting bias or social-desirability bias. Underreporting of indoor cannabis smoking would likely reduce the magnitude of association observed in our study. Our results may lend to conclusions of a null association. However, given the consistency of the magnitude of association after successive control for confounding variables, it is plausible that a true effect exists, justifying additional research.

Our sample was primarily Hispanic and low income, limiting representativeness. An additional limitation was the low sensitivity of the past-year health outcomes measures. We did not collect data about the extent to which the adverse health outcomes were exacerbated by any type of SHS. We also did not ask if the outcomes manifested prior to the past year of report. Therefore, we did not capture acute or long-term health effects of any SHS exposure. Future studies should ask questions about the duration and severity of past-year diagnoses in children. Furthermore, the cross-sectional design did not lend to drawing conclusions about a possible causal relation of SCS to health consequences.

6. Conclusions

This study provides some of the first findings concerning potential health implications for children exposed to secondhand cannabis smoke. We found a relatively strong though not statistically significant association (adjusted OR = 1.83; 95% CI = 0.89–3.80; $p = 0.10$) between indoor cannabis smoking and adverse health outcomes in children. Assuming the null hypothesis is true, that there is no association between indoor cannabis smoking and adverse health outcomes in children, the probability is 10% that we would observe an odds ratio of this magnitude in our study. Especially given the potential health risks at stake, the increasing rates of cannabis use, and the less than ideal characteristics of our study (e.g., use of reported measures and limited statistical power), the observed association is sufficient to warrant further research to more decisively determine possible health impacts of indoor cannabis smoking. Ideally, future studies should use higher fidelity measures (e.g., biomarkers of cannabis and cigarette exposure, medical record data), and a stronger design (e.g., larger sample size, longitudinal cohort). With the rise of cannabis use in the U.S., we need to understand its health consequences. Such information is needed to help healthcare providers, lawmakers, and researchers encourage parents and other caregivers to protect children from potentially harmful exposure to SCS.

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Conflicts of interest

The authors declare that no competing interests exist.

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References

Amirav, I., Luder, A., Viner, Y., Finkel, M., 2011. Decriminalization of cannabis - potential risks for children? Acta Paediatr. 100, 618–619. https://doi.org/10.1111/j.1651-2227.2010.02081.x.

Berg, C.J., Buller, D.B., Schauer, G.L., Windle, M., Stratton, E., Kegler, M.C., 2015. Rules regarding marijuana and its use in personal residences: findings from marijuana users and nonusers recruited through social media. J. Environ. Public Health 2015, 1–7. https://doi.org/10.1155/2015/476017.

Berg, C.J., Haardörfer, R., Wagener, T.L., Kegler, M.C., Windle, M., 2018. Correlates of allowing alternative tobacco product or marijuana use in the homes of young adults. Pediatrics 141, S10–S20. https://doi.org/10.1542/peds.2017-1026E.

Centers for Disease Control and Prevention (US), 2010. How tobacco smoke causes disease: the biology and behavioral basis for smoking-attributable disease. http://www.ncbi.nlm.nih.gov/pubmed/21452462, Accessed date: 7 February 2017.

Drug Enforcement Administration: Diversion Control Division, 2018. list of scheduling actions, controlled substances and regulated chemicals. https://www.gpo.gov/fdsys/, Accessed date: 4 October 2018.

Ferrante, G., Simoni, M., Cibella, F., Ferrara, F., Liotta, G., Malizia, V., Corsello, G., Viegi, G., Grutta, S. La, 2013. Third-hand smoke exposure and health hazards in children. Monaldi Arch. Chest Dis. 79, 38–43. https://doi.org/10.4081/monaldi.2013.108.

Franklin, P.J., 2007. Indoor air quality and respiratory health of children. Paediatr. Respir. Rev. 8, 281–286. https://doi.org/10.1016/j.prrv.2007.08.007.

Fried, P.A., Watkinson, B., 1990. 36- and 48-month neurobehavioral follow-up of children prenatally exposed to marijuana, cigarettes, and alcohol. J. Dev. Behav. Pediatr. 11, 49–58.

Goodwin, R.D., Cheslack-Postava, K., Santoscoy, S., Bakoyiannis, N., Hasin, D.S., Collins, B.N., Lepore, S.J., Wall, M.M., 2018. Trends in cannabis and cigarette use among parents with children at home: 2002 to 2015. Pediatrics 141. https://doi.org/10.

1542/peds.2017-3506.
Hasin, D.S., 2018. US epidemiology of cannabis use and associated problems. Neuropsychopharmacology 43, 195–212. https://doi.org/10.1038/npp.2017.198.
Herrmann, E.S., Cone, E.J., Mitchell, J.M., Bigelow, G.E., LoDico, C., Flegel, R., Vandrey, R., 2015. Non-smoker exposure to secondhand cannabis smoke II: effect of room ventilation on the physiological, subjective, and behavioral/cognitive effects. Drug Alcohol Depend. 151, 194–202. https://doi.org/10.1016/j.drugalcdep.2015.03.019.
Hughes, S.C., Bellettiere, J., Nguyen, B., Liles, S., Klepeis, N.E., Quintana, P.J.E., Berardi, V., Obayashi, S., Bradley, S., Hofstetter, C.R., Hovell, M.F., 2018. Randomized trial to reduce air particle levels in homes of smokers and children. Am. J. Prev. Med. 54, 359–367. https://doi.org/10.1016/J.AMEPRE.2017.10.017.
Johnston, L.D., O'Malley, P.M., Bachman, J.G., Schulenberg, J.E., Miech, R.A., 2016. Monitoring the future of national survey results on drug use, 1975-2015: volume 2, college students and adults ages 19-55. http://www.monitoringthefuture.org/pubs/monographs/mtf-vol2_2016.pdf, Accessed date: 21 October 2017.
Klepeis, N.E., Bellettiere, J., Hughes, S.C., Nguyen, B., Berardi, V., Liles, S., Obayashi, S., Hofstetter, C.R., Blumberg, E., Hovell, M.F., 2017. Fine particles in homes of predominantly low-income families with children and smokers: key physical and behavioral determinants to inform indoor-air-quality interventions. PLoS One 12, e0177718. https://doi.org/10.1371/journal.pone.0177718.
Kramer, U., Lemmen, C.H., Behrendt, H., Link, E., Schafer, T., Gostomzyk, J., Scherer, G., Ring, J., 2004. The effect of environmental tobacco smoke on eczema and allergic sensitization in children. Br. J. Dermatol. 150, 111–118. https://doi.org/10.1111/j.1365-2133.2004.05710.x.
Kumar, S.R., Davies, S., Weitzman, M., Sherman, S., 2015. A review of air quality, biological indicators and health effects of second-hand waterpipe smoke exposure. Tob. Control. 24, i54–i59. https://doi.org/10.1136/tobaccocontrol-2014-052038.
Li, J.S.M., Peat, J.K., Xuan, W., Berry, G., 1999. Meta-analysis on the association between environmental tobacco smoke (ETS) exposure and the prevalence of lower respiratory tract infection in early childhood. Pediatr. Pulmonol. 27, 5–13. https://doi.org/10.1002/(SICI)1099-0496(199901)27:1<5::AID-PPUL3>3.0.CO;2-5.
Macnab, A., Anderson, E., Susak, L., 1989. Ingestion of cannabis: a cause of coma in children. Pediatr. Emerg. Care 5, 238–239.
Matt, G.E., Quintana, P.J.E., Hovell, M.F., Bernert, J.T., Song, S., Novianti, N., Juarez, T., Floro, J., Gehrman, C., Garcia, M., Larson, S., 2004. Households contaminated by environmental tobacco smoke: sources of infant exposures. Tob. Control. 13, 29–37. https://doi.org/10.1136/tc.2003.003889.
Matt, G.E., Quintana, P.J.E., Destaillats, H., Gundel, L.A., Sleiman, M., Singer, B.C., Jacob, P., Benowitz, N., Winickoff, J.P., Rehan, V., Talbot, P., Schick, S., Samet, J., Wang, Y., Hang, B., Martins-Green, M., Pankow, J.F., Hovell, M.F., 2011. Thirdhand tobacco smoke: emerging evidence and arguments for a multidisciplinary research agenda. Environ. Health Perspect. 119, 1218–1226. https://doi.org/10.1289/ehp.1103500.
Miller, M.D., Marty, M.A., Arcus, A., Brown, J., Morry, D., Sandy, M., 2002. Differences between children and adults: implications for risk assessment at California EPA. Int. J. Toxicol. 21, 403–418. https://doi.org/10.1080/10915810290096630.
Moir, D., Rickert, W.S., Levasseur, G., Larose, Y., Maertens, R., White, P., Desjardins, S., 2008. A comparison of mainstream and sidestream marijuana and tobacco cigarette smoke produced under two machine smoking conditions. Chem. Res. Toxicol. 21, 494–502. https://doi.org/10.1021/tx700275p.
Moore, B.A., Augustson, E.M., Moser, R.P., Budney, A.J., 2005. Respiratory effects of marijuana and tobacco use in a U.S. sample. J. Gen. Intern. Med. 20, 33–37. https://doi.org/10.1111/j.1525-1497.2004.40081.x.
Ostro, B., Roth, L., Malig, B., Marty, M., 2009. The effects of fine particle components on respiratory hospital admissions in children. Environ. Health Perspect. 117, 475. https://doi.org/10.1289/EHP.11848.
Patra, S., Sharma, S., Behera, D., 2012. Passive smoking, indoor air pollution and childhood tuberculosis: a case control study. Indian J. Tuberc. 1515, 151–155.
Schauer, G.L., King, B.A., Bunnell, R.E., Promoff, G., Mcafee, T.A., 2015. Toking, vaping, and eating for health or fun. Am. J. Prev. Med. 50, 1–8. https://doi.org/10.1016/j.amepre.2015.05.027.
Schlegel, B., Steenbergen, M., 2018. brant: test for parallel tegression assumption. In: R Package Version 0.2–0.
Strachan, D.P., Cook, D.G., 1998. Parental smoking and childhood asthma: longitudinal and case-control studies. Thorax 53, 204–212. https://doi.org/10.1136/thx.53.3.204.
Substance Abuse and Mental Health Services Administration, 2017. Key substance use and mental health indicators in the United States: results from the 2016 National Survey on drug use and health. https://www.samhsa.gov/data/, Accessed date: 1 July 2018.
Thijssen, D.H.J., Black, M.A., Pyke, K.E., Padilla, J., Atkinson, G., Harris, R.A., Parker, B., Widlansky, M.E., Tschakovsky, M.E., Green, D.J., 2011. Assessment of flow-mediated dilation in humans: a methodological and physiological guideline. Am. J. Physiol. Circ. Physiol. 300, H2–H12. https://doi.org/10.1152/ajpheart.00471.2010.
United Sates Environmental Protection Agency, 2016. Air quality designations for particle pollution. https://www.epa.gov/particle-pollution-designations, Accessed date: 2 July 2017.
US Department of Health and Human Services, 2006. The health consequences of involuntary exposure to tobacco smoke: a report of the Surgeon General. http://www.ncbi.nlm.nih.gov/pubmed/20669524, Accessed date: 2 July 2017.
US Department of Health and Human Services, 2014. The health consequences of smoking: 50 years of progress. A report of the Surgeon General. https://www.surgeongeneral.gov/library/reports/50-years-of-progress/full-report.pdf, Accessed date: 2 July 2017.
Volkow, N.D., Baler, R.D., Compton, W.M., Weiss, S.R., 2014a. Adverse health effects of marijuana use. N. Engl. J. Med. 370, 2219–2227. https://doi.org/10.1056/NEJMra1402309.
Volkow, N.D., Wang, G.-J., Telang, F., Fowler, J.S., Alexoff, D., Logan, J., Jayne, M., Wong, C., Tomasi, D., 2014b. Decreased dopamine brain reactivity in marijuana abusers is associated with negative emotionality and addiction severity. Proc. Natl. Acad. Sci. U. S. A. 111, E3149–E3156. https://doi.org/10.1073/pnas.1411228111.
Wang, G.S., Narang, S.K., Wells, K., Chuang, R., 2011. A case series of marijuana exposures in pediatric patients less than 5 years of age. Child Abuse Negl. 35, 563–565. https://doi.org/10.1016/j.chiabu.2011.03.012.
Wang, X., Derakhshandeh, R., Liu, J., Narayan, S., Nabavizadeh, P., Le, S., Danforth, O.M., Pinnamaneni, K., Rodriguez, H.J., Luu, E., Sievers, R.E., Schick, S.F., Glantz, S.A., Springer, M.L., 2016. One minute of marijuana secondhand smoke exposure substantially impairs vascular endothelial function. J. Am. Heart Assoc. 5, e003858. https://doi.org/10.1161/JAHA.116.003858.
Wei, B., Wang, L., Blount, B.C., 2015. Analysis of cannabinoids and their metabolites in human urine. Anal. Chem. 87, 10183–10187. https://doi.org/10.1021/acs.analchem.5b02603.
Wilson, K.M., Torok, M.R., Wei, B., Wang, L., Robinson, M., Sosnoff, C.S., Blount, B.C., 2017. Detecting biomarkers of secondhand marijuana smoke in young children. Pediatr. Res. 81, 589–592. https://doi.org/10.1038/pr.2016.261.