Original investigation

Does Starting to Smoke Cigars Trigger Onset of Cannabis Blunt Smoking?

Brian J. Fairman PhD¹, James C. Anthony PhD²

¹Health Behavior Branch, Division of Intramural Population Health, Eunice Kennedy Shriver National Institute of Child Health and Human Development, Bethesda, MD; ²Department of Epidemiology and Biostatistics, Michigan State University, East Lansing, MI

Corresponding Author: Brian J. Fairman, PhD, Division of Intramural Population Health Research, Eunice Kennedy Shriver National Institute of Child Health and Human Development, 6710B Rockledge Dr., Bethesda, MD 20817, USA. Telephone: 301-827-6897; E-mail: brian.fairman@nih.gov

Abstract

Introduction: Among United States teens during the 1990s, increasing cigar use coincided with increasing use of tobacco cigar shells filled with cannabis, called “blunts.” Cigar smokers are more likely to use cannabis, and we hypothesized that starting to smoke cigars might be a probabilistic “trigger” of blunt smoking. We turned to the case-crossover approach to evaluate this hypothesis.

Methods: Within US National Surveys on Drug Use and Health, 2009–2013, we identified a nationally representative sample of newly incident blunt smokers aged 12- to 21-years-old ($n = 4868$) and compared month-of-onsets for smoking of cigars and blunts. Using the subjects-as-their-own-controls case-crossover design, we specified the first month prior to blunt use as a “hazard interval” and the second month prior to blunt use as a “control interval.” We used Mantel–Haenszel (MH) estimators to estimate the matched-pairs odds ratio (OR).

Results: The MH OR estimate was 1.7 (95% CI = 1.3, 2.3), with excess odds of cigar onsets during the hazard interval relative to the control interval. Two alternative control interval specifications yielded congruent estimates (OR = 2.7 and 2.9, respectively).

Conclusions: A short interval right after starting to smoke cigars may be one of increased risk of starting to smoke blunts. We discuss cigar, cigarillo, and “blunt wraps” control approaches that might reduce both tobacco and cannabis-related harms.

Implications: If this evidence is correct, increased market-targeting to promote youthful cigar and cigarillo smoking might be followed by increased rates of blunt smoking in a vulnerable population. As noted by others, enhanced risk of smoking-attributable harms might be a consequence of mixed tobacco-cannabis formulations.

Introduction

Increased United States cigar consumption during the 1990s coincided with resurgence of cannabis use and emergence of using cigar shells as cannabis delivery devices in formulations called “blunts.”¹ Blunts are made by rolling cannabis herb in outer shells of cheap cigars or cigarillos, with constituent tobacco.²

Moreover, while US cigarette sales have been declining, tobacco market share of little cigars and cigarillos shifted upward from 37%

and 25% (in 1990s), respectively, to 47% and 32% (in 2000s).^{3,4} Concurrently, cannabis smoking prevalence increased until the early 2000s and has since remained relatively stable.^{1,5} Golub and colleagues¹ attributed part of the observed rise in cannabis smoking to blunts use.

To our surprise, the epidemiology of blunt smoking largely has been ignored by tobacco and nicotine researchers and much remains to be known, even though blunts may expose cannabis smokers to harmful levels of tobacco from cigar shells themselves or from loose

tobacco.^{6,7} Of note, estimated prevalence of recently active blunt smoking shows modest recent increases to 14% of young people in the United States.⁸ About two-thirds of past-year cannabis users aged 12 and older smoked blunts recently; 8% qualify as daily or near-daily blunt users.⁹ Correlates include being male, younger, black, and urban-dwelling.^{1,8}

If we wished to prevent blunt smoking in the United States, one potentially important modifiable antecedent of smoking blunts is prior cigar smoking experience, independent of cigarette smoking.^{10,11} Blunt-smokers initiate cigars at younger ages, smoke cigars more frequently, and are more likely to be drug dependent.^{9,10,12,13} Smoking of cigars might lead to greater contact with blunt smoking peers, with increased chances to try blunts—akin to tobacco smokers being more likely to encounter cannabis.¹⁴ Familiarity with cigars, their taste, flavorings, and smoke inhalation might promote blunt smoking, especially when users know of possibly enhanced psychoactive effects (eg, from co-use of tobacco and cannabis).^{15–18} Legally marketed “blunt wraps” and other commercial activity shows industry awareness of the blunt phenomena.¹⁹

Against this background, we hypothesized that cigar smoking onset might be a precipitant of blunt smoking among young people, as one variant of widely-discussed gateway processes.²⁰ Alternative variants such as a “reverse gateway” have been suggested, but the idea that tobacco-smoking can serve as a “gateway” toward cannabis-smoking is not a dead idea.^{21,22}

To localize this cigar-blunts investigation, we focused on 12- to 21-year-olds in the United States and harnessed the epidemiologic case-crossover design and its subject-as-own-control approach in order to constrain alternative causal influences while estimating the degree to which cigar-smoking-onset might foster an increased risk of blunt-smoking-onset. Here, our cigar focus stems from our concerns about sample size, including our forecast that available samples were inadequate for evaluation of cigars and cigarettes as interdependent causal influences on blunt smoking. Of these two tobacco possibilities (cigars vs. cigarettes), we chose to investigate cigar-onsets.

The case-crossover study approach addresses the following type of causal question, “Was this event-outcome triggered by something unusual that happened just before?”²³ Exposure to a hypothesized trigger-event is studied within “hazard intervals,” specified as intervals before event-outcomes in a non-deterministic causal sequence, such that the “hazard intervals” are those during which there is hypothesized elevated probability of the event-outcome.²³ Unusualness of triggering-events is gauged relative to “control intervals,” most often occurring before hazard intervals and typically specified with control interval duration equal to hazard interval duration. Evidence that favors triggering-event hypotheses is seen when odds of triggering-events during hazard intervals exceeds odds of triggering-events during control intervals.²⁴

General population tests of triggering-event hypotheses can be formulated for discrete-time survival analysis data with triggering-events treated as time-varying covariates and with right-censoring by event-outcomes. However, causal inference from general population tests face unmeasured or difficult-to-measure individual-level antecedents of event-outcomes.²³ For cigar-to-blunt triggering hypotheses, long-standing propensities or unwillingness to inhale fumes from smoked compounds are examples of these rarely measured antecedents. Case-crossover (CC) approaches were designed to constrain such otherwise confounding individual-level antecedents via self-matching in study design and analysis.²³ That is, focused on

specified epidemiological study populations, CC approaches target specific affected cases who have experienced event-outcomes under study, followed by estimation of the odds of triggering-events being seen during each case’s hazard interval relative to odds of triggering-events being seen during the same case’s control interval. As such, each case’s individual-level confounding variables (eg, willingness to inhale fumes) are constrained to have comparable values even when they are unmeasured. Accordingly, estimates of the exposure odds ratio (OR) tend toward 1.0 when the null hypothesis is correct (“no triggering”), and otherwise depart from 1.0, as illustrated in a growing series of drug epidemiology investigations.^{24–32}

Methods

Study Design, Population, Sample, and Data Collection

The population under study was sampled, recruited, and assessed for US National Surveys on Drug Use and Health (NSDUH), 2009–2013, with a strictly cross-sectional design. Each year’s NSDUH multi-stage probability sampling design collects data on a nationally representative sample of non-institutionalized US civilians aged 12 years or older. De-identified public release data files provided information on ~55 000 participants per year. Additional details of general NSDUH design and methods have been published elsewhere (www.icpsr.umich.edu/icpsrweb/ICPSR/series/64). Analysis of NSDUH de-identified data were deemed “not human subjects research” by cognizant institutional review boards.

Among individuals surveyed from 2009–2013 ($n = 282\,470$), we identified 4868 newly incident blunt smokers who met inclusion criteria: (1) were 12- to 21-years-of-age (49% of total); (2) were newly incident blunt smokers (16% of 12–21 year olds who had ever smoked blunts were newly incident—ie, onset within 12 months before assessment, with focus on this short interval to constrain memory errors); (3) had non-missing data on timing of blunt and cigar onsets. Among all 12- to 21-year-olds who had ever smoked blunts ($n = 28\,769$), fewer than four percent had missing data required for analysis (~3.7%).

Trained field staff followed standardized survey protocols in or near participant dwellings. Audio computer-assisted self-interviews (ACASI) collected data on drug use and other health topics, offering a highly confidential means to provide private information on such topics.

Measures

Blunts were described as follows: “Sometimes people take some tobacco out of a cigar and replace it with marijuana. This is sometimes called a ‘blunt.’” Calendar months/years of blunt onset were assessed when self-reported age-of-onset for blunts was equal to or 1 year less than the age at interview: “Did you first smoke part or all of a cigar with marijuana in it in (Current year/Current year–1/Current year–2)”; “In what month did you first smoke part or all of a cigar with marijuana in it?” Using these data, we defined a variable that captured the month/year of blunt onset, storing it as a discrete number of months since January 1, 1960. Equivalent variables were defined for month/year of onset for cannabis (all forms), tobacco cigarettes, cigars, snuff, and chew, all assessed in similar modes. NSDUH didn’t elicit onset month/year when onsets occurred > 24 months prior to survey interview.

The month/year variable for cigar onset was then subtracted from the month/year variable for blunt onset, yielding numbers of months prior to blunt onset when cigar onset occurred. For example,

a value of “0” on this variable indicates that the individual initiated cigars and blunts in the same month, a “1” means that cigar onset occurred in the first calendar month prior to the month of blunt onset, and so on. From this constructed variable, a pair of dichotomous variables was derived to indicate whether cigar onset had occurred in the individual’s case-crossover hazard interval or control interval. Similar pairs of indicator variables were defined for tobacco cigarettes, snuff, and chew.

Covariates help describe the sample: age at interview, sex, race-ethnicity, and residential subtype. Via self-matching, these covariate values were held constant for hazard and control intervals in our primary study analysis.

Analysis

For reasons explained elsewhere,^{33,34} any CC approach to NSDUH month-wise data can be made complex if hazard intervals are specified to include the month of occurrence of event-outcomes, “*t*” (eg, due to uncertainty about temporal sequencing of triggering-events and event-outcomes within that same calendar month). To avoid these complexities, we specified our CC “hazard interval” as the first calendar month prior to the blunt onset month (ie, *t*-1). We specified the “control interval” (I) to be the calendar month before the hazard interval month (ie, *t*-2).

Mantel-Haenszel (MH) estimators for matched-pair odds ratios (OR) are based on the logic used in studies of environmental differences between case-discordant monozygotic twin pairs, which derive from a more general instance of matched-pair case-control studies.^{25,33} Accordingly, each CC case is assigned to a 2 × 2 table, as depicted in Table 1, based on whether exposure to hypothesized triggering-events occurred in the hazard interval and/or in the control interval (yes vs. no). For MH OR estimation, informative cases are those exposed in the hazard interval but not in the control interval (cell “B”), as well as those exposed in the control interval but not in the hazard interval (cell “C”; ie, discordant or incongruous cases). Concordant cases (ie, cells A and D) are those not exposed in either the hazard or control interval (A), or exposed in both the hazard and the control intervals (D), and are non-informative with respect to point estimation of MH OR.

With the 2 × 2 table formulated on this basis, the MH OR in simplified form³⁴ is $OR = (B/C)$ with standard deviation (SD) simplified as $\ln(OR) = (1/B + 1/C)^{1/2}$. We report 95% confidence intervals (CI) based on exact methods for OR estimates, and *p*-values based on McNemar’s chi-square test statistic. All analyses are from Stata Version 11.³⁵

Table 1. Illustration of Analysis Approach for Case-Crossover Data Using a Simplified Mantel-Haenszel Matched-Pair Odds Ratio Estimator

		Exposure to triggering-event during the hazard interval	
		No	Yes
Exposure to the triggering-event during the control interval	No	A	B
	Yes	C	D

Odds ratio (OR) is calculated as $OR = B/C$. Standard deviation can be calculated as $\ln(OR) = (1/B + 1/C)^{1/2}$. Cells A and D do not contribute to OR estimation. When the hypothesized triggering-event is “absorbing” (occurs once and only once), cell D is a structural zero.

Our analysis plan pre-specified post-estimation exploratory data analyses (EDA) to evaluate MH OR estimates based on two alternative control intervals and to eliminate potentially confounding influences of cigarette smoking onsets. As has been done previously,³¹ one alternative control interval (labeled as II in our table and text) involves use of the entire 11-month period from month *t*-2 through *t*-12 with a simple arithmetic correction to calibrate the 1-month duration of the hazard interval (*t*-1) versus the 11-month duration of the control (II) interval (ie, calibrated by dividing by 11 and rounding to the lowest integer result). The second alternative control interval (labeled as III in table and text) is a specification of *t*-12 as the control interval for the original hazard interval (*t*-1). That is, in this post-estimation EDA, the MH OR estimate based on the III control interval is gauged in relation to the odds of the triggering-event in the same calendar month as the blunt-onset hazard interval, worked backward 1 year.

Our plan to address potential confounding by tobacco cigarette smoking involved two post-estimation EDA: (1) to exclude from the analysis sample all individuals who had smoked a tobacco cigarette, even once, and (2) exclusion from the analysis sample those individuals for whom cigarette-smoking-onset occurred in the same month as cigar-smoking-onset.

Anonymous reviewers encouraged us to complete several new analyses we had not planned: (1) estimation of MH OR with a substitution of other nicotine exposures as the triggering-event (ie, cigarette-onset, snuff-onset, chew-onset), and (2) estimates for the distribution of covariates in the study population (although these covariates are held constant via the self-matching CC design). We have provided these estimates in our Results section. We also plotted the frequency of cigar onsets per month among those who initiated cigars in the 12 months prior to blunt onset (*n* = 650) in order to provide a visual sense of when cigar onsets occurred relative to blunt onsets in the analysis sample.

Results

As depicted in Table 2, weighted to the population, newly incident blunt smokers aged 12–21 years were evenly split between males and females (Table 2). A slightly larger proportion were 12–17 years old (58.1%) versus 18–21 years. Most were non-Hispanic white (55.0%), followed by Hispanic (21.4%), and non-Hispanic black (16.9%). Most lived in a large metropolitan area (56.8%), while less than one-third lived in a small metro area (30.5%). For about 3 in 10 (29.8%), blunt initiation co-occurred in the same month as cannabis initiation in any form, but we cannot say which came first. A majority reported never having smoked cigars (54.2%), while slightly more than one-third (35.6%) had never smoked tobacco cigarettes.

Figure 1 depicts month-wise sample frequencies when cigar onsets were observed prior to blunt onset (*n* = 650 newly incident blunt smokers). For instance, 128 started smoking cigars in the first calendar month prior to blunt onset (*t*-1), 74 in the second month (*t*-2), etc. After month 4, this per-month number of cigar onsets remained relatively stable.

MH OR estimates are depicted in Table 3. Among the newly incident blunt smokers, 128 had cigar onset in hazard interval (*t*-1) and 74 had cigar onset in our study’s primary control interval (I), while 95% of them had no cigar onset in either of these intervals. The resulting OR estimate indicates excess odds of starting to smoke cigars in the hazard versus control (I) interval (ie, $OR = 128/74 = 1.7$, 95% CI [1.3, 2.3]; *p* < .001).

Table 2. Characteristics of Newly Incident Blunt Smokers Assessed at Age 12–21 Years ($n = 4868$)

	Sample size (n)	Weighted population estimate (%)	95% confidence interval	
Sex				
Female	2427	49.4	47.4	51.3
Male	2441	50.6	48.7	52.6
Age				
12–17 years	3245	58.1	56.1	60.2
18–21 years	1623	41.9	39.8	43.9
Race/Ethnicity				
Non-Hispanic white	2722	55.0	52.9	57.1
Non-Hispanic black	791	16.9	15.3	18.5
Hispanic	909	21.4	19.7	23.2
Other than the above	446	6.7	5.8	7.7
Residential area sub-type				
Large metro	2285	56.8	54.4	59.1
Small metro	1695	30.5	28.4	32.7
Nonmetro	888	12.7	11.4	14.0
Cannabis onset, irrespective of form ^a				
Not within the same month as blunts	3493	70.2	68.3	71.9
Within the same month	1375	29.8	28.1	31.7
Cigar smoking				
Never used	2585	54.2	52.4	56.0
Ever used	2283	45.8	44.0	47.6
Tobacco cigarette smoking				
Never used	1733	35.6	33.5	37.8
Ever used	3125	64.4	62.2	66.5

Data from United States National Surveys on Drug Use and Health, 2009–2013.

^aNSDUH assessed month and year of onset of cannabis use (any form) and separately assessed month and year of first blunt smoking.

Figure 1. Frequency of newly incident blunt smokers ($n = 650$) by the number of months from cigar onset to blunt onset, with a restriction to newly incident blunt smokers who also were newly incident cigar smokers. Data from National Surveys on Drug Use and Health, 2009–2013.

Our post-estimation exploration of alternative specifications for the control interval disclosed somewhat larger case-crossover OR estimates as compared with this primary study estimate (Table 3). Expanding the control interval (II) to capture all 11 months prior to the hazard interval yielded 522 cigar-onsets for an average number of 47 cigar-onsets per month (ie, $522/11 = 47.5$, rounded down). When compared to cigar smoking onsets in the hazard month, the result was an $OR = 128/47 = 2.7$ and 95% CI = 1.9, 3.8. The second alternative control interval (III), based on cigar-onsets exactly one calendar year prior to the month of blunt onset, produced a similar sized estimate ($OR = 2.9$; 95% CI [2.1, 4.1]; $n = 44$ for this specified “control interval”).

For our additional pre-specified exploratory analyses, we re-estimated whether the primary study OR estimate of 1.7 changed appreciably when tobacco cigarette smoking was controlled in two ways. First, with exclusion of newly incident blunt-smokers who had any history of tobacco cigarette smoking ($n = 3135$; 64.4%), the resulting OR estimate was 2.9 (95% CI [1.5, 5.7]). Second, after exclusion of newly incident blunt-smokers with tobacco cigarette onset in the same month as cigar-smoking onset ($n = 170$; 3.6%), the resulting OR estimate was 1.9 (95% CI [1.4, 2.6]). (Estimates not shown in a table.)

In the final set of unplanned analyses suggested by others, we explored whether onset of snuff use might qualify as a triggering-event; here, the result was consistent with the null hypothesis ($OR = 1.2$; 95% CI [0.6, 2.3]). The corresponding estimate for chewing tobacco onset also was null ($p > .05$). However, readers interested in the originally specified “gateway process” might appreciate that the month after onset of tobacco cigarette smoking also appears to be a month of excess risk for starting blunt smoking ($OR = 1.4$; 95% CI [1.04, 1.8]). Here, the triggering-event was specified to be onset of tobacco cigarette smoking in the hazard interval relative to our initially specified control interval (I). As we had forecast, the study sample was not large enough to produce a statistically useful estimate of combined cigar-cigarette onsets within the same calendar month.

Discussion

We started this study expecting that cigar-onset might be a “triggering” event with respect to risk of starting to smoke the cannabis-tobacco formulation known as a blunt. The resulting non-null OR estimate of 1.7, derived with strengths of a subject-as-own-control design, suggests that the month following cigar smoking onset is a period of heightened risk for starting to smoke blunts. Exclusions of all tobacco

Table 3. Estimated Odds Ratios for Initiating Cigar Smoking in the Month Prior to Blunt Smoking Onset (Hazard Interval) Compared to Cigar Initiation in Different Control Intervals (*n* = 4868)

Triggering event during:	<i>n</i> (%)	Triggering Event in Hazard Interval ^a		Odds ratio (OR)	95% Confidence interval	<i>P</i>
		No	Yes			
Control (I) interval ^b	No	4666 (96%)	128 (3%)	1.7	[1.3, 2.3]	<.001
	Yes	74 (2%)	0			
Control (II) interval ^c	No	4218 (87%)	128 (3%)	2.7	[1.9, 3.9]	<.001
	Yes	522/11 = 47 (11%)	0			
Control (III) interval ^d	No	4624 (96%)	128 (3%)	2.9	[2.1, 4.2]	<.001
	Yes	44 (1%)	0			

Data from US National Surveys on Drug Use and Health, 2009–2013.

^aDefined as the first calendar month prior to month of blunt onset.

^bDefined as the second calendar month prior to month of blunt onset.

^cDefined as the second through 12th calendar months prior to month of blunt onset. Division by 11 and rounded to the nearest integer was necessary to correct for the longer control interval.

^dDefined as the calendar month 1 year prior to month of blunt onset.

cigarette smokers or those who started smoking tobacco cigarettes in the same month as cigars did not affect the cigar-blunt OR.

Even so, our post-estimation exploration also suggested that a modified hypothesis may be in order. Namely, onsets of “either” cigar smoking “or” tobacco cigarette smoking, “or both,” might qualify as triggering-events for blunt smoking onset. Given that no excess risk was observed in analyses with snuff onset or chew onset substituted as the triggering-event, we now can speculate that some part of the observed associations might be general with respect to inhalation (“smoking”) of tobacco fumes, possibly due to route of administration commonalities.¹⁵ Even so, our somewhat larger cigar-blunts association may indicate a possible specificity with cigars, particularly when the OR estimate did not differ appreciably when cigarette smoking was controlled. These issues deserve examination in more recently gathered data, including data from the longitudinal PATH study now underway (<https://pathstudyinfo.nih.gov/UI/StudyOverviewMobile.aspx>, last accessed 3 January 2017).

The observed associations also may qualify as special variations of tobacco-cannabis gateway processes specified more generally, long ago.^{14,21} We can speculate about several potential mechanisms. As one example, smoking cigars could increase the likelihood of being offered a blunt.^{36–40} Further, passing around a cigar, cigarillo, or cigarette after taking a turn at smoking a blunt, a practice called “chasing”, is also known to occur.⁴⁰ Analogous processes might enhance likelihood of blunt-onsets among those with recent cigar-onsets.

Cigar-onsets also might affect risk perceptions about blunts directly, or indirectly via a generalized effect to the extent that engaging in one risky behavior might prompt down-regulation of perceptions about harm from other unhealthy behaviors.^{41,42} An alternative is that cigars/cigarillos might be perceived as more “natural” and thereby less harmful, similar to justifications for using cannabis.^{40,43,44} If so, new cigar smokers may see little added risk for engaging in blunt smoking. In addition, knowledge about other aspects of using blunts to consume cannabis might be relevant, based on qualitative studies of users who report achieving a larger subjectively-felt “high” from cannabis-tobacco co-use, as compared to smoking cannabis-only joints.^{39,45} Other possibilities involve rapid learning that a flavored cigar wrapper might be used to mask an unwanted cannabis odor; alternately, a blunt in cigar form might help conceal cannabis smoking in public.⁴⁰ Knowledge on such topics (eg, from peers)

might precede or follow cigar-onsets, but might gain increased salience after cigar-onsets.

Leaving these uncertain speculations aside, we think the OR estimates from this case-crossover study qualify as a noteworthy addition to the research literature on the epidemiology of tobacco cigar smoking and cannabis smoking in general, as well as blunt smoking in particular. Strengthened by the subject-as-own-control facet of case-crossover research, this approach constrains many (but not all) alternative individual antecedents that otherwise might account for an association linking prior cigar smoking with later blunt smoking. While these findings suggest possibilities of a “transient” risk of blunt smoking due to cigar onset, CC designs were not intended to speak to the “cumulative” risk of event-outcomes over lifetimes of individuals. Clearly, any claim that cigar onset is the only determinant or the strongest determinant of blunt smoking would be misplaced. As our estimates show, a majority of newly incident blunt smokers in this US study population did not report any prior cigar use; a great many did not have cigar-onsets in the entire year before blunt onset. Therefore, cigar smoking itself does not guarantee blunt use, nor can all cases of blunt smoking be explained by cigar smoking.

As for other limitations faced in this research, we note self-report data. Retrospective self-reports are subject to recall and reporting errors, constrained here by limiting the time required for recollection to 24 months at most. We judge reliability of the resulting month-by-month reports about drug-onsets to be adequate for this study’s purposes, but no definitive evidence exists on this matter. We also note that a potentially generalized under-reporting of illegal or sensitive behaviors, including blunt/cannabis use, with possibly reduced validity of the study estimates. Second, fine-grained data on the time-sequencing of cigar and blunt onsets (ie, on the scale of days) might be preferred; some CC studies gained advantages via hour-by-hour and day-by-day temporal sequencing data. Third, a strength of CC designs is self-matching control over many sources of uncontrolled confounding, but we cannot completely rule out confounding by time-varying antecedents occurring during the hazard/control intervals, which also might function as alternative explanations for onset of blunt smoking. Here, we ruled out confounding by tobacco cigarette smoking via exclusions, but a more comprehensive assessment plan, prospectively gathered data, and more fine-grained time-sequencing data definitely would help provide more definitive evidence for or against our “triggering” hypothesis about cigar-onsets and subsequent blunts-onsets.

We are hopeful that the previously mentioned PATH study should be helpful due to its epidemiologically credible study population sample, its prospective design, and its recording of individual trajectories of tobacco and other drug involvement, including consideration of e-cigarettes (<https://pathstudyinfo.nih.gov/UI/StudyOverviewMobile.aspx>, last accessed 3 January 2017). Nevertheless, as was true in the current study, PATH sample size might not be large enough for estimation of any modified hypothesis about specificity of cigar-blunt associations. That is, we focused on cigar-blunt associations because the anticipated sample was not large enough for three alternative hypotheses about (1) cigars specifically, (2) tobacco cigarettes specifically, and (3) either cigars or tobacco cigarettes (ie, a generic “smoking” hypothesis). We anticipate that the PATH sample size also will face constraints in this regard.

These limitations and constraints on sample size notwithstanding, the findings of the present study are of interest for several reasons. Public health harms attributed to low-cost cigars and cigarillos have been recognized with flavorings and their sale as singletons, the latter of which affects cost thresholds for access and attractiveness to adolescent, novice, and minority users.^{17,46} If this study’s findings are reproducible and are found to withstand challenges to validity, then it should be of interest that a previously unanticipated consequence of starting to smoke cigars might be an increased incidence of cannabis use in the form of blunt smoking. In turn, blunt smoking might reinforce tobacco exposure via the tobacco content of blunts themselves⁶ as well as non-blunt cigar use via the “chasing” behavior described previously in this discussion section. This increased tobacco exposure might contribute to tobacco-attributable illnesses, interfere with users’ ability to quit either tobacco or cannabis, and impair treatment outcomes for tobacco and cannabis co-users.⁴⁷

Our findings could have implications for public health policies regarding the sale and marketing of cigars, cigarillos, and other tobacco products used for making blunts, which now appear to have been targeted specifically to black communities and to residential areas with greater density of young adults.⁴⁶ Adult cigarillo users have noted that certain brands are easier to split apart with a perforated line on the cigar shell for this purpose.¹⁹ Some tobacco companies have started to market “blunt wraps,” consisting of the outer shell of a cigar. These products are ready-made for construction of the tobacco-cannabis blunt formulation. Therefore, policies regarding these products may deserve greater scrutiny, especially in areas where adolescents have easy access to both cigar/cigarillos and cannabis, including jurisdictions with recently liberalized cannabis policies. As a policy example, the City of Boston has restricted the sale of cigarillos in the form of singletons, as well as blunt wraps and flavored cigar products.⁴⁸ We note that local ordinances of this type, systematically applied, might create opportunities for future drug policy experiments or quasi-experiments pertinent to the cigar-blunt linkage.

Conclusions

This study’s estimates for 12- to -21-year-olds in the United States prompt the conclusion that the month following onset of cigar smoking might be one of increased risk for onset of blunt smoking. The several OR estimates suggest that cigar-onset functions as a specific triggering-event, but there are residual possibilities that involve “tobacco smoking” in general or “inhalation” (eg, of e-cigarette “vapors”), which could not be studied in detail here.

Ours might be the first epidemiological study to progress beyond descriptive identification of blunt smoking correlates in the direction

of a specific modifiable causal influence (ie, cigar-onsets) with both measured and unmeasured alternative explanations held constant. A study replication should become possible before 2020 because the current NSDUH plan is for accrual of new samples through 2018 when a completely new and independent sample of newly incident blunt smokers will become available for study.

Even though blunts research currently has greatest policy and public health relevance within the United States, this formulation could become more common in other countries. Mixing tobacco and cannabis (“mulling”) is a typical practice elsewhere, and few jurisdictions now enforce policy restrictions on youthful access to “blunt wraps,” even where restrictions on cigars and cigarillos are in place.^{49–52}

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Declaration of Interests

None declared.

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