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Is marijuana really a gateway drug? A nationally representative test of the marijuana gateway hypothesis using a propensity score matching design

Cody Jorgensen, Ph.D[1] · Jessica Wells, Ph.D[1]

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Abstract

Marijuana use has been proposed to serve as a "gateway" that increases the likelihood that users will engage in subsequent use of harder and more harmful substances, known as the marijuana gateway hypothesis (MGH). The current study refines and extends the literature on the MGH by testing the hypothesis using rigorous quasi-experimental, propensity score-matching methodology in a nationally representative sample. Using three waves of data from the National Longitudinal Study of Adolescent to Adult Health (1994–2002), eighteen propensity score-matching tests of the marijuana gateway hypothesis were conducted. Six of the eighteen tests were statistically significant; however, only three were substantively meaningful. These three tests found weak effects of frequent marijuana use on illicit drug use but they were also sensitive to hidden bias. Results from this study indicate that marijuana use is not a reliable gateway cause of illicit drug use. As such, prohibition policies are unlikely to reduce illicit drug use.

Keywords Marijuana · Gateway · Substance use · Propensity score matching

Introduction

Originally proposed in the mid-1970s, the marijuana gateway hypothesis (MGH) suggests that use of various classes of drugs follows a predictable pattern. Individuals who use licit substances, such as tobacco and alcohol, are at a greater risk for use of marijuana. Subsequently, marijuana use presents an increased propensity of drug use escalation to harder substances (Kandel 1975). In an effort to reduce hard drug use, public policies surrounding the criminalization of marijuana have often cited the MGH as a justification for its prohibition. In the 1980s, President Reagan quipped that

✉ Cody Jorgensen, Ph.D
codyjorgensen@boisestate.edu

[1] Boise State University, University Drive, Mail Stop 1955, Boise, Idaho 83725-1955, USA

marijuana may be the most dangerous drug because it was a gateway to harder drugs and the argument is still commonly presented. This marijuana gateway hypothesis currently remains a consistent justification for harsh policies controlling the plant.

Despite the proliferation of policies based on the MGH, research suggests that MGH arguments may not be as accurate as widely assumed. Rather, research suggests that while there may be an association between marijuana and other drug use, causal arguments are not strongly supported. If marijuana use causes hard drug use, there must be an association that includes correlation and temporal ordering that is not explained by other external variables. Competing models, such as the common liability model, suggest that while there is an association between marijuana use and hard drug use, this can largely be explained by other risk factors, or "third" variables (Vanyukov et al. 2003).

Research generally finds support for the correlational and temporal aspects of the MGH. This aspect is commonly referred to as sequencing. However, sequencing is not causal. For example, people who ride motorcycles rode bicycles prior. Additionally, before we ride bicycles, we typically ride a tricycle. There is a sequence: tricycle to bicycle to motorcycle. Yet, no one would intuitively think that riding a tricycle causes riding a bicycle and that riding a bicycle causes riding a motorcycle. There are a host of other variables related to motorcycle riding that have not been taken into account. As such, the MGH is plagued by issues associated with spuriousness calling into question any claims of causality. While a body of research has attempted to control for extraneous variables that could explain the association between marijuana use and subsequent hard drug use, these attempts have been rather methodologically limited. For example, in a study that found little evidence in support of the gateway hypothesis, Nkansah-Amankra and Minelli (2016) examined this process using generalized estimation models. Although they attempted to directly control for a number of potential confounds, the variety and complexity of potential extraneous confounds limits the ability to fully understand the extent to which the MGH may occur.

Improvements in the salience of findings regarding the MGH are needed in order to better understand the complex relationship between marijuana and hard drug use and to discover if that relationship is causal. The current study advances this body of research by applying propensity score matching to a longitudinal, nationally representative sample. The founder of the MGH has been critical of MGH research making appeals to spuriousness: "In practice, control for all relevant confounding factors is an ideal that cannot be achieved, because the critic can always invoke the lack of control for important non-measured factors" (Kandel 2003, p. 470). The propensity score matching (PSM) methodology of the current study provides a viable alternative to traditional approaches (e.g., regression analysis using unbalanced data) in testing the MGH when true experimental methods are not an option. PSM is a selection on observables method based on the counterfactual approach to hypothesis testing that allows researchers to estimate causal effects (Apel and Sweeten 2010; Heckman and Hotz 1989).

Literature review

Previous research indicates that marijuana use is associated with long-lasting detrimental effects on a variety of life domains. Among others, marijuana use has been found to be associated with decreased educational attainment, unemployment, and reduced IQ

and working memory (Castellanos-Ryan et al. 2017; Fergusson and Boden 2008; Horwood et al. 2010; Lynskey and Hall 2000; Solowij and Battisti 2006; Townsend et al. 2007; van Ours and Williams 2009; Zhang et al. 2016). Research also indicates that marijuana use is associated with hard drug use. As proposed by Kandel (1975), the marijuana gateway hypothesis may be useful in explaining this pattern of progression of substance use. The MGH suggests that substance use follows a predictable pattern from licit drug use, such as nicotine and alcohol, to marijuana, to other hard drug use. According to the MGH, the use of marijuana causes an increased risk for the use of other, more dangerous drugs. Research examining the MGH largely couches this argument through adherence to either biological and psychological alterations or differential association with delinquent peers. The first of these explanations argues that marijuana causes future hard drug use by altering the individual user such that the user seeks out more potent intoxicating effects. The second argues that use of marijuana increases associations with peers that also engage in drug use and thus the user learns about and is influenced by the drug use of others. Despite this theoretical framework, there is fairly robust evidence that the MGH is flawed, at best (see generally, Mosher and Akins 2014). For the MGH to be supported, three elements of the association between marijuana use and harder drug use must be established: correlation, sequencing, and non-spuriousness. While the theoretical framework and research testing the MGH is largely in support of these first two elements, discord in the field continues concerning whether the progression from marijuana use to hard drug is actually causal due to the potential contribution of one or more extraneous variable on both marijuana and other drug use.

Correlation and sequencing

The association and sequencing of marijuana use and hard drug use is fairly robust. Research has found that individuals that initiate marijuana use before the age of 21 were 157 times more likely to engage in subsequent hard drug use. Furthermore, those that initiate marijuana use after 21 were 24 times more likely to engage in subsequent hard drug use (Morral et al. 2002). This finding has been replicated in longitudinal, adolescent research that also suggests that marijuana use significantly related to later hard drug use. Studies limiting examination of correlation and sequencing of drug use trajectories to adolescents have found support for these two elements of the MGH (Choo et al. 2008). Evaluations that extend further into the life course have found similar support for correlation and sequencing (Degenhardt et al. 2009; Deza 2015; Fergusson et al. 2015; Fergusson et al. 2006; Fergusson and Horwood 2000; Lessem et al. 2006). Extending from age 15 to 35, evaluation of the longitudinal Christchurch Health and Human Development Study (CHDS) found a significant association between earlier marijuana use and later hard drug use (Fergusson et al. 2015). While this supports correlation and sequencing of marijuana and hard drug use, this research also suggests that if the MGH holds, this process is dynamic across the life-course with earlier use initiation being more deleterious than later onset marijuana use.

While the vast majority of marijuana users never escalate to later hard drug use, the majority of those who engage in hard drug use have a history of marijuana use (Fergusson et al. 2015). Analysis of drug use trajectories has found that, while marijuana use is a significant predictor of later hard drug use for a small group of

individuals, the risk of hard drug use following marijuana use is minimal for a much larger proportion of individuals (Melberg et al. 2010). Some studies have found moderate estimates wherein approximately 29% of marijuana users continue on to harder drug use (Golub and Johnson 2002). Despite these variations in the effect of marijuana use on hard drug use, there is ample evidence that among hard drug users, marijuana use does vastly precede hard drug use. A study of the National Household Survey of Drug Abuse (NHSDA) found that only 1.6% of hard drug users initiated hard drug use prior to use of marijuana (Morral et al. 2002). A study of the CHDS similarly found that only 1% of hard drug users aged 15–21 did not previously engage in marijuana use (Fergusson and Horwood 2000).

While the normative sequential pattern suggested by the MGH may have some merit with low-level users, more severe patterns of use have been found to deviate from the expected sequence—that is, those with more severe drug use patterns may initiate illicit substance use prior to licit substance use. Although few studies have examined this reverse patterning, the sequence has been explained by variation in neighborhood physical environment and exposure to drugs as well as neglectful parents (Tarter et al. 2006). This sequencing may also be explained by measurement variation in which more chronic hard drug users are less likely to follow the "typical" pattern from licit drug use, to marijuana, to hard drug use (Golub and Johnson 2002).

Non-spuriousness

Common liability explanations for the association between marijuana and hard drug use suggest that marijuana use does not cause hard drug use. Rather, there is some individual or collective "third" variable that explains use of both marijuana and hard drugs. If this "third" variable is capable of explaining increased liability for substance use, the MGH cannot stand on causal grounds (Vanyukov et al. 2003, 2012). While support for correlation and sequencing is strong, and supportive of MGH, research regarding "third" variable explanations casts doubt on the validity of MGH. Again, the MGH is a causal argument stating that marijuana use causes hard drug use.

Variation in marijuana use patterns may be explained by a host of individual characteristics that vary between early and chronic marijuana users and those with no use, late-onset use, and occasional marijuana use (Hall and Lynskey 2005). In a longitudinal study of male adolescents and young adults, those who initiated marijuana use before the age of 22 affiliated with more delinquent peers, were less involved in school, and were generally more deviant than other participants (Tarter et al. 2006). More recently, chronic marijuana use trajectories were found to be associated with increased sensation seeking, emotional dysregulation, and unconventional behavior (Brook et al. 2016). These factors are particularly relevant to claims of spuriousness in the MGH as there appears to be a dose-dependent response. That is, individuals with chronic use are more likely to engage in subsequent hard drug use as compared to those with lower-level usage patterns (Fergusson et al. 2015; Fergusson and Horwood 2000; Morral et al. 2002; Ward et al. 2010). An obvious explanation for this observed pattern is self-control (Gottfredson and Hirschi 1990). Those lower in self-control are more likely to frequently use marijuana and to use hard drugs than those with higher levels of self-control thereby rendering the relationship between frequent marijuana use and hard drug use possibly spurious.

Genetically informed models also highlight the potential for third-variable explanations. An analysis of sibling pairs, discordant for marijuana use, found that a significant proportion of the association between adolescent marijuana use and later hard drug use was attributable to shared environmental effects between sibling (i.e., parenting, family, and neighborhood effects) (Lessem et al. 2006). Further research has shown that the majority of shared environmental effects on marijuana use initiation and abuse are attributable to neighborhood effects (Gillespie et al. 2009). In another early study examining twins discordant for marijuana use, Lynskey et al. (2003) found that genetic and shared environmental influences were negligible. As such, they found support for the MGH. More recently, however, Cleveland and Wiebe (2008) improved on a host of methodological weaknesses of Lynskey et al. (2003). Using data from the National Longitudinal Study of Adolescent to Adult Health (Add Health), the authors found that the trajectory of drug use could not be explained by the MGH, but rather, was largely heritable (Cleveland and Wiebe 2008).

Collectively, this research suggests that there may be important variation in individuals' characteristics between those marijuana users and non-users as well as those who subsequently progress to hard drug use. Despite a large body of literature arguing that third-variable explanations account for the marijuana-hard drug use association, some research controlling for such factors continues to find an association between early marijuana use and later hard drug use. In an analysis of the National Drug Use and Health data using a "subject-as-own-control" method, O'Brien, Comment, Liang, and Anthony (2012) found that marijuana use was a significant predictor of cocaine use. Similarly, analysis of the National Youth Survey controlling for variables from strain, social bonding, and differential association theories found a significant effect of marijuana use on later hard drug use (Rebellon and Gundy 2006).

Given the mixed evidence regarding "third" variable explanations, further research that allows for causal inference is needed to examine whether elements of common liability may account for the association between marijuana use and later hard drug use. The current study presents the first study to use propensity-score matching, allowing for causal inference. Analyzed in this way, we examine the MGH as it relates to the use of marijuana causing the use of harder drugs using a nationally representative dataset. While this approach has been utilized in other research testing similar processes, see (Timberlake et al. 2009), the strength of this approach has not been used frequently to inform the MGH. Utilizing longitudinal data from a nationally representative sample, the current study tests the MGH by systematically controlling for a host of potential common liability factors. It should be noted that PSM is a selection on observables method and is subject to omitted variable bias if relevant antecedents are absent in the vector of covariates. However, PSM differs from traditional regression based methods by following a counterfactual approach that approximates the randomized control trial, the standard for establishing causality. As such, the PSM approach allows for causal inference estimation.

Methods

The data examined for this analysis come from the publicly available National Longitudinal Study of Adolescent to Adult Health (Add Health), a nationally representative

multi-wave cohort study (Udry 2003). The first three waves of data were included in this analysis. The available data from wave IV were not used because participants have aged out of the age-crime curve by this time and it is unlikely that including wave IV would produce any unique findings that were not discovered using the first three waves[1]. The first wave of data was collected between 1994 and 1995, when respondents were between 7th and 12th grades. The second wave of data was collected 2 years later. Data from wave III were collected between 2001 and 2002. The Add Health covers a wide range of topics interesting to social scientists including demographics, crime/delinquency, substance use, family life, personality traits, neighborhood context, etc. Sample sizes for the three waves were 6360, 4749, and 4865, respectively. Sample size attrition observed in this study is a common limitation of large scale longitudinal studies. At wave I, 49% of the sample was male and 66% of the sample was white. Demographic representivity remains rather consistent across waves[2].

Measures

The independent/treatment variable in this study was marijuana use. The frequency of use was recoded for this study at three different usage levels. The first usage level measured whether or not respondents had used marijuana within the last 30 days (0 = no, 1 = yes). Any lifetime use of marijuana was not chosen as an independent variable. This was done to limit recall bias, ensure that covariates were antecedent to the treatment, and to save space in the manuscript. We reasonably assumed that individuals who used hard drugs did not stop using marijuana. The second usage level captured recreational or moderate marijuana use measured as use of marijuana more than once but less than 5 times within the last 30 days were coded (0 = no, 1 = yes). The third usage level measured heavy/frequent marijuana use measured as use of marijuana five or more times within the last 30 days (0 = no, 1 = yes) which captures the top quartile of marijuana use. These three usage levels represent the treatment variable from a quasi-experimental context. The categorical measurement strategy was necessary to differentiate frequent marijuana users from infrequent marijuana users. The literature discussed in the prior section shows that the likelihood of graduating to harder drug use is higher for frequent marijuana users. This measurement approach allows for a kind of "tipping point" test of the MGH. After all, it could be the case that the MGH holds only for frequent marijuana users but not for infrequent marijuana users.

The dependent/outcome variable, illicit/hard drug use, was assessed by asking participants about their frequency of cocaine, methamphetamine, and heroin use. Prevalence of illicit/hard drug use was captured dichotomously in what is conceptualized as the recreational hard drug use variable (0 = no illicit/hard drug use, 1 = illicit/hard drug use once or twice within the past 30 days). The second usage level measured heavy/frequent illicit drug use measured as use of any hard drug three times or more within the last 30 days which captures the top quartile of the illicit drug use distribution. Given the distribution of drug use, using the top quartile as a cutoff point was the most reasonable choice. Doing so discriminated between frequent and infrequent users

[1] Additionally, included wave IV would add a substantial amount of length to the manuscript.
[2] Readers may consult the documentation provided by the Add Health study at their website (see the Appendix 1).

Table 1 Percentage of sample by type and level of drug use within the last 30 days

Drug use	W1	W2	W3
Any marijuana use	13%	15.1%	21.8%
1–4 marijuana uses	7.5%	8.8%	11%
5+ marijuana uses	6%	6.9%	12%
1–2 illicit drug uses	3.8%	3.8%	6.4%
3+ illicit drug uses	2.2%	1.7%	3.2%

considering our conceptualization while also providing enough observations in each group for comparison purposes. The categorical measurement strategy for the dependent variable follows the logic of the independent variable. These measures represent the outcome variable from a quasi-experimental context. Table 1 below shows the percentages of the sample that fall into these measures at each wave.

Categorizing the independent and dependent variables was necessary to meet the goals of this analysis and to examine whether or not a "tipping point" into the gateway effect exists. For example, it may be the case that infrequent marijuana use has no gateway effect but frequent use does. Categorizing the variables of interest makes testing this effect fairly straight forward. Additionally, the continuous distributions of both marijuana use and illicit drug use are extremely skewed.

The vector of covariates included 33 controls including various measures of demographics, crime and delinquency, tobacco and alcohol use, family life, peer associations, school and neighborhood characteristics, psychological and personality characteristics, and religiosity. Both the independent/treatment variable and dependent/outcome variable measure drug use and both have similar covariates, meaning that both the independent variables and dependent variables are drug using behaviors and both are explained by similar theoretical constructs. The vector of covariates selected in the current study represents a comprehensive (although maybe not exhaustive) list of salient covariates. See Appendix 1 for a complete list of controls used in this analysis. The covariates selected are commonly used in substance use research and include numerous theoretically related factors predicting drug use as specified by prominent criminological theories[3].

Missing data

In the logit models used to create propensity scores, missing data were handled via listwise deletion. This was done for a few reasons. First, listwise deletion is a standard practice in social science when working with large datasets. Second, imputation procedures are not typically ideal for PSM when using longitudinal datasets due to attrition (Kupzyk and Beal 2017). Third, few observations were missing and after dropping missing observation there were enough data to achieve sufficient common support. At wave I, all but three covariates contained less than 10% missing

[3] See Appendix 1 for the list of the covariates used in this study. Information on how survey items were measured, response options, and descriptive statistics for each wave are readily available at the Add Health website. Refer to Appendix 1 for appropriate Internet links.

observations. Most covariates had only a few percent missing. Cold relationship with the father, father's education, and condition of the neighborhood had 30%, 34%, and 29% missing cases, respectively. These covariates were included in the model since they are theoretically relevant to drug using behavior and therefore non-ignorable. Relationship with the father is an important aspect of the social bond and father's education and condition of neighborhood are indicators of the respondent's socio-economic status[4]. Listwise deletion still left over 4200 observations in the logit model at wave I. Wave II had similar issues with these covariates yet still left more than 3400 observations. Participant attrition is a common reason for reduced observations in subsequent waves of data.

Analytical strategy

The biggest limitation of the existing research testing the MGH is the lack of experimental methods. Due to practical and ethical constraints surrounding testing of the MGH using randomized control trials, a preferable alternative is to employ a quasi-experimental technique that approximates the randomized controlled trial. A quasi-experimental approach using observational data such as the Add Health data is therefore necessary to test the MGH to understand if marijuana use truly has a causal effect on hard drug use later on in life. Propensity score matching is such an alternative (Apel and Sweeten 2010; Guo and Fraser 2009). PSM improves on regression-based techniques because the test is non-parametric thereby reducing bias in the calculated effect size as compared with fitting a linear model, and, assuming adequate care has been taken to include all relevant variables, allows researchers to be more confident that findings could be causal. To the best of the authors' knowledge, no published tests of the effect marijuana use has on harder drug use that implement a PSM design using a nationally representative dataset exist. Nguyen et al. (2019) used a propensity weighting approach, analyzing Add Health data to test the effect marijuana use has on daily cigarette smoking. This study is similar but different from the current study in several important ways. First, Nguyen et al. (2019) tested a reverse MGH hypothesis, predicting that marijuana use increases the risk of cigarette smoking, which is generally regarded as less deviant given its legal status. Second, the current study assesses wave-to-wave effects across three waves. Finally, the current study refines marijuana usage by categorizing users by their level of use (past month non-users, recreational, and heavy users) to test potential variation in results that may be dependent on the frequency of use.

The current study therefore employs PSM analysis to test the MGH (i.e., marijuana causes hard drug use) using a nationally representative and longitudinal dataset. Of the 18 analyses conducted, the first six tests examine the gateway effect of three usage levels of marijuana use at wave I on two usage levels of hard drug use at wave II. The

[4] However, it seems that the statistically significant relationships between marijuana use and heavy drug use found in this analysis are differentially affected when considering these missing data. The correlation between recreational marijuana use at wave 1 and heavy illicit drug use at wave 3 reduced from .06 to .03 when missing values on cold relationship with the father were taken into account. This correlation reduced to .045 when missing values on father's education was taken into account. The correlation between heavy marijuana use at wave 2 and light illicit drug use at wave 3 reduced from .14 to .08 when missing values on condition of the neighborhood were taken into account. The significant results found in Tables 3 and 4 should be interpreted with caution.

next six tests assess the effect of marijuana use at wave I on hard drug use at wave III, and the following six tests identify the effect of marijuana use at wave II on hard drug use at wave III. The analyses were conducted using the *psmatch2* and *pstest* commands in STATA 14. Logit models were employed to calculate propensity scores.

The specifications for the PSM models were selected to maximize balance and precision while maintaining a sufficient sample size of matched pairs. One-to-one nearest neighbor matching without replacement and a .01 caliper was the default specification throughout this analysis. A caliper of .01 is quite conservative and produces closely matched pairs. The advised caliper as described by Rubin (2001) was .0525 for this study. In ten cases, this specification produced a sufficient number of matched pairs. However, this specification had to be relaxed in eight of the tests to maximize the number of matched pairs. In these cases, a .05 caliper was specified to widen the bandwidth of potential matches to be selected from and the nearest neighbor specification was increased by one unit with replacement until the maximum number of matched pairs was achieved (e.g., 1:1, 1:2, 1:3). The relaxed specifications produced less precise matches, yet were still within acceptable ranges commonly used in social science research and was necessary to obtain the most matched pairs possible (Guo and Fraser 2009). Sufficient common support and overlap between the treatment group and control group was achieved throughout this analysis. Histograms and maximum and minimum propensity score values showed that the distributions of propensity scores for the treatment and comparison groups substantially overlapped allowing for a sufficient number of matched pairs to conduct meaningful tests of the MGH[5]. Adding the propensity scores as a covariate did not yield more matches in the models that had fewer than 100 match pairs. The propensity scores created for matching were calculated for the appropriate wave of data used for each test. When marijuana use at wave I was being tested, propensity scores were calculated using wave I data. When marijuana use at wave II was being tested, propensity scores were calculated using wave II data. Lastly, all covariates were antecedent, or reasonably assumed to be antecedent, to the independent variable given that only marijuana use within the last 30 days was captured. The PSM models estimated an average treatment effect of the treated (ATT)[6] which is the default effect calculated in the *psmatch2* command in STATA and we assumed a non-directional two-tailed test with a .05 alpha level. It could be the case that marijuana use was perceived as unpleasant by the user who therefore quit using. Covariate balance was tested via the *pstest* command which uses inferential techniques to test the difference in the mean value of a covariate prior to matching from the mean value of that same covariate after the matching algorithm has been applied. Finally, sensitivity analyses using Rosenbaum's R-bounds to check for hidden bias were completed. Hidden bias is the bias affecting the treatment effect from unobserved covariates. Testing for hidden bias assesses the robustness of the treatment effects. Although this analysis included 33 covariates to create propensity scores, it could be the case that unmeasured covariates influencing the treatment, outcome, or both could be missing.

[5] Histograms are not recreated here to save space but are available upon request.

[6] **ATT** = E[{E[Yi\P(Xi),Di = 1] − E[Yi\P(Xi),Di = 0]}\Di = 1]

Table 2 PSM results marijuana use at W1 on hard drug use at W2

IV	DV	# matches	Off support	Effect size	*t*	Balance	
						UM[a]	M[b]
Mar. last 30	Light illicit	129	81	.04	1.09	26	0
Recreational	Light illicit	112	0	.05	1.28	23	0
Heavy	Light illicit	82	16	.08	1.32	26	0
Mar. last 30	Heavy illicit	129	81	.01	.34	25	0
Recreational	Heavy illicit	112	0	.03	1.29	23	0
Heavy	Heavy illicit	82	16	.04	.91	26	0

[a] Number of covariates that statistically differ between the treatment group and control group prior to matching

[b] Number of covariates that statistically differ between the treatment group and control group after matching

Results

The results assessing the effects of various usage levels of marijuana use at wave I on harder drug use at wave II are presented in Table 2. At wave I, the subjects of the survey were between the 7th and 12th grades. Wave II was collected 2 years later. No support for the MGH was found in these analyses as none of the tests were statistically or substantively meaningful. The treatment and control groups were balanced in these tests. The unmatched (UM) and matched (M) values for these tests suggest that prior to the matching algorithm being applied, the treatment group and control group significantly differed on 23–26 control variables. After matching, however, the treatment group and control group did not significantly differ on any control variable leaving marijuana use as the only difference between the treatment group and control group given the covariates included in the analysis.

Table 3 presents the results of the effects of marijuana use at wave I on hard drug use at wave III, when the subjects of the survey were between 18 and 25 years old and at the peak to desistance portion of the age-crime curve. Light Illicit drug use at wave III was significantly predicted by wave I prevalence of marijuana use (ATT = .08, $t = 2.41$, $p < .05$), recreational marijuana use (ATT = .08, $t = 1.99$, $p < .05$), and heavy marijuana use (ATT = .16, $t = 2.37$, $p < .05$)[7]. Sensitivity analyses show that each of these significant effects were sensitive to hidden bias caused by an unobserved covariate. Gamma became no longer significant at the upper bound (alpha = .05) at 1.3, 1.2, and 1.3, respectively, for the findings detailed above. Of these statistically significant associations, only the association between heavy marijuana use and light illicit drug use was substantively meaningful with a treatment effect of .16 (Cooper et al. 2016). This suggests a weak gateway effect. It should also be noted that this finding comes from the least conservative model in the series of tests in Table 3 and was rather sensitive to hidden bias. This test found a maximum of 97 matched pairs using a .05 caliper and 1:2 nearest neighbor matching algorithm which resulted in the treatment group and control group being unbalanced on five control variables. The mean percentage difference between the treatment group and control group for time spent

[7] These findings may be biased due to missing data on cold relationship with the father and father's education.

Table 3 PSM results marijuana use at W1 on hard drug use at W3

IV	DV	# matches	Off support	Effect size	*t*	Balance	
						UM[a]	M[b]
Mar. last 30	Light illicit	138	85	.08	2.41*	25	0
Recreational	Light illicit	100	23	.08	1.99*	22	0
Heavy	Light illicit	97	3	.16	2.37*	25	5
Mar. last 30	Heavy illicit	138	85	.03	1.18	26	0
Recreational	Heavy illicit	100	23	.05	1.93	22	0
Heavy	Heavy illicit	97	3	.09	1.56	26	4

[a] Number of covariates that statistically differ between the treatment group and control group prior to matching

[b] Number of covariates that statistically differ between the treatment group and control group after matching

with friends was 11%. Cold relationship with mother, alcohol use, saying prayers, and importance of religion differed by 17%, 23%, 22%, and 25%, respectively. All of which are theoretically related to drug use, therefore it was decided to leave these variables in the model. Because the mean percentage differences of these five covariates are relatively large, the link between marijuana use and illicit drug use could be spurious. Relaxing the specification of the model further also did not yield better balance. Additionally, seven years separates the IV and DV, which is a significant amount of time.

Table 4 presents the results of the effects of marijuana use at wave II on hard drug use at wave III. Heavy marijuana use at wave II was associated with an increase in wave III light and heavy illicit drug use (ATT = .17, $t = 2.28$, $p < .05$; ATT = .11, $t = 2.09$, $p < .05$)[8]. Sensitivity analyses show that the upper bounds became insignificant at the .05 alpha level when gamma = 1.5 for both tests. Similar to the statistically significant associations found between Wave I marijuana use and Wave III illicit drug use, the statistically significant effects here were substantively weak. Again, these findings stem from the least conservative models of these tests and are somewhat sensitive to hidden bias. Further, the relaxed specification (.05 caliper; 1:2 nn matching) only found a maximum of 70 matched pairs; however, balance was achieved.

The results of this analysis also support a "tipping point" effect. Categorizing the independent and dependent variables was useful in the current study. For comparative purposes, PSM models were also estimated with illicit drug use measured continuously and the results varied from the categorical approach. For example, frequent marijuana use (measured categorically) at wave II increased frequent illicit drug use (measured categorically) at wave III. However, frequent marijuana at wave II use was not statistically associated with illicit drug use in wave III when illicit drug use was measured continuously (ATT = .44). When illicit drug use was measured continuously at wave III, infrequent marijuana use and frequent marijuana use at Wave I had identical statistically significant treatment effects (ATT = 1.3, $p < .05$). These results are different from the results shown in Table 3 measuring illicit drug use categorically

[8] The correlation between heavy marijuana use and illicit drug use changes based on missing data according to neighborhood condition. This finding should be interpreted with caution.

Table 4 PSM results marijuana use at W2 on hard drug use at W3

IV	DV	# matches	Off support	Effect size	*t*	Balance	
						UM[a]	M[b]
Mar. last 30	Light illicit	107	87	−.02	−.43	27	0
Recreational	Light illicit	101	13	−.06	−1.18	22	0
Heavy	Light illicit	70	10	.17	2.28*	27	0
Mar. last 30	Heavy illicit	107	87	−.02	−.59	27	0
Recreational	Heavy illicit	101	13	−.06	−1.55	22	0
Heavy	Heavy illicit	70	10	.11	2.09*	25	0

[a] Number of covariates that statistically differ between the treatment group and control group prior to matching

[b] Number of covariates that statistically differ between the treatment group and control group after matching

that found infrequent marijuana use did not have a substantively meaningful effect on illicit drug use, but frequent marijuana use did[9].

To put these findings in context, PSM models were also estimated testing the independent gateway effect of both tobacco and alcohol on hard drug use. The findings from these tests nearly mimicked the findings from the marijuana gateway tests. The results are shown in Tables 5 and 6 below. These findings suggest that both tobacco and alcohol independently have roughly equivalent weak gateway effects on hard drug use as compared with marijuana. These findings are supportive of prior research in the public health literature that tends to find that tobacco and alcohol use are predictive of marijuana use and hard drug use (Nkansah-Amankra and Minelli 2016). If the MGH is being invoked to justify its prohibition, then the results presented in Tables 5 and 6 suggest the same argument could be made to prohibit the use of tobacco and alcohol, an argument unlikely to win many supporters.

Discussion

Considering the findings from all 18 tests together, it can be concluded that the hypothesis that marijuana is a gateway drug is unsupported. Summarily, if marijuana "really" is a gateway drug we would expect to see stronger and more consistent causal gateway effects. These tests found no support for the MGH in 12 of the 18 tests, scant support in 3 with treatment effects less than .10, and weak support in 3 tests with treatment effects between .11 and .17. The three significant and substantive findings were weak, derived from the poorest models, did not achieve complete balance in one

[9] Using illicit drugs was a rare event in the sample. The large majority of respondents did not use illicit drugs. As such, there were excessive zeros in the dependent variables. Subsequent analysis on the matched samples examined whether the excessive zeros would bias the results of the propensity score models. The findings from the subsequent analysis were generally similar to those of the propensity score models. However, some differences were observed. Heavy marijuana use was not significantly related to heavy illicit drug use at any wave. Additionally, heavy marijuana use at W1 was weakly associated with light illicit drug use at W2, and having used marijuana at all in the last 30 days at W1 was weakly associated with light illicit drug use at W3. We interpret these results as generally congruent to the results of the propensity score models.

Table 5 Gateway effects of tobacco and alcohol use at W1 on hard drug use at W2

IV	DV	# matches	Off support	Effect size	*t*	Balance	
						UM[a]	M[b]
Light smoking	Light illicit	317	88	.06	2.89*	27	0
Heavy smoking	Light illicit	144	3	.11	2.74*	25	0
Light drinking	Light illicit	496	232	.03	2.38*	25	4
Heavy drinking	Light illicit	283	1	.03	1.06	26	0
Light smoking	Heavy illicit	317	88	.03	2.71*	27	0
Heavy smoking	Heavy illicit	144	3	.08	2.50*	26	0
Light drinking	Heavy illicit	496	232	.01	1.08	25	4
Heavy drinking	Heavy illicit	283	1	.02	1.41	26	0

[a] Number of covariates that statistically differ between the treatment group and control group prior to matching

[b] Number of covariates that statistically differ between the treatment group and control group after matching

of the tests, were sensitive to hidden bias, may be affected by missing observations, and were limited only to the gateway effect of heavy marijuana use. After controlling for a host of common liability factors, the causal association between marijuana use and hard drug use was largely unfounded in the current study.

Marijuana legalization is controversial in the American public discourse. There is evidence that Americans are developing more accepting attitudes toward legal cannabis because it is viewed as less dangerous than other drugs on the drug schedule. Results from a 2019 Gallup poll showed that 66% of Americans favored legalizing marijuana. As of this writing, Colorado, Washington, Oregon, Alaska, Arizona, New York, Michigan, New Jersey, California, Nevada, Maine, Massachusetts, Vermont, and Illinois have legalized the recreational use of marijuana (in defiance of federal law), and 34 states plus Washington, DC, have legalized the use of medicinal marijuana. In

Table 6 Gateway effects of tobacco and alcohol use at W2 on hard drug use at W3

IV	DV	# matches	Off support	Effect size	*t*	Balance	
						UM[a]	M[b]
Light smoking	Light illicit	323	147	.05	2.02*	31	0
Heavy smoking	Light illicit	62	18	.13	1.87*	24	0
Light drinking	Light illicit	451	140	.02	1.4	25	0
Heavy drinking	Light illicit	265	0	.02	.67	26	0
Light smoking	Heavy illicit	323	147	.04	2.12*	31	0
Heavy smoking	Heavy illicit	174	0	.03	1.01	26	0
Light drinking	Heavy illicit	451	140	.02	1.12	25	0
Heavy drinking	Heavy illicit	265	0	.01	.33	25	0

[a] Number of covariates that statistically differ between the treatment group and control group prior to matching

[b] Number of covariates that statistically differ between the treatment group and control group after matching

short, there appears to be shifting attitudes in America about the acceptability of marijuana in recent years.

As attitudes toward marijuana among the American public change, policymakers should take note and enact legislation that suits the will of the public. As noted earlier, political discourse advocating marijuana prohibition commonly hinges on the assumption that marijuana causes hard drug use. The MGH is by far the most common justification for prohibiting the use of cannabis (Mosher and Akins 2014). However, the current study provides further evidence that common liability arguments are more in line with substance use patterns observed in the USA. What is more, legalizing marijuana would remove the subcultural aspects of common liability arguments which would further reduce the correlation between marijuana use and hard drug use. As such, attempts to restrict access to marijuana may not have the expected impact on reducing hard drug use. Additionally, the enforcement of laws that are viewed by most members of the public as illegitimate or unnecessary is likely to hurt the perceived legitimacy of the criminal justice system which, in turn, reduces the likelihood of voluntary compliance and cooperation from the public (Tyler 2006).

Further, the gateway hypothesis holds that licit substance use should precede marijuana use. The current study found greater support for this stage of substance use escalation. Alcohol is legal, widely used, easily accessible, and often celebrated. Generally, alcohol is a much more harmful substance to individuals and to society compared with marijuana (Nutt et al. 2010). A 2010 report prepared by the Independent Scientific Committee on Drugs and published in the *Lancet* found that, overall, alcohol was by far the most harmful drug to individual users and society. The study showed that alcohol was 3.6 times more harmful than marijuana. Additionally, the study found that tobacco was more harmful to the individual user and more harmful to society compared with marijuana, although only slightly. What is more, research suggests that the public and even police officers agree that marijuana is not more harmful or dangerous than alcohol (Jorgensen 2018; Stylianou 2003).

This study fills a methodological gap in testing the marijuana use explanations for escalation in substance use. In particular, propensity score matching offers a meaningful option when testing gateway-type hypotheses when true experimental conditions are not appropriate or viable. To the best of the authors' knowledge, this analysis is the first to use PSM to test the effect of marijuana use on hard drug use using nationally representative data. Future research should attempt to replicate the findings from this study using methods that allow for causal inference.

Although this study makes a contribution to our understanding of the MGH, several limitations need to be addressed. Several tests detailed above yielded fewer paired matches than desired (although the number of matched pairs was acceptable), most likely because relatively few respondents in the sample used hard drugs or used marijuana frequently. Additionally, the tests with the fewest matches were also the least precise models that did not achieve balance even after further relaxing specifications which resulted in even fewer matched pairs. Caution should be taken when interpreting the results from these tests as the specifications allowed more room for error and spurious findings. Another important limitation is the absence of biological, genetic, and physiological control variables. As noted in the literature review, biological forces do play a significant role in drug using behavior and addiction, and it could be the case that the findings from this study would be different had biological factors

been included in the analysis. In short, the models may suffer from omitted variable bias as not all possible "third" variables have been accounted for. However, these potential third variables being absent likely inflated the relationship between marijuana use and hard drug use which suggests that the significant findings from this study would not be as strong had these third variables been included. This further weakens the MGH argument. Future research should incorporate biologically informed variables into the vector of covariates in PSM models.

Conclusion

In sum, the findings from the current study using PSM to test the MGH provide further support of previous research questioning the causal claims of the MGH. While there is strong support for correlation and sequencing in marijuana and hard drug use, correlation and sequencing alone cannot provide sufficient evidence for causality. Factors other than marijuana use such as genetic predisposition, peer associations, or access to the illicit drug market could be the primary causes of hard drug use instead of marijuana use itself. As such, any public policy that prohibits the use of marijuana in an attempt to curb hard drug use is unlikely to succeed. Policy makers should also consider the negative consequences of making a criminal out of an otherwise law-abiding pot smoker. Former President Carter once said in a 1977 address to Congress, "the penalty for drug possession should not be more harmful to an individual than the use of the drug itself." It could be argued that this is the case for marijuana.

Appendix 1. Vector of covariates

Gender
Age
Age squared
Ethnicity
Grade in school
Ever been suspended from school
Mother's education
Father's education
Neighborhood condition
Urbanity
Perception of living in a safe neighborhood
How often respondents hang out with friends
Relationship with the mother
Relationship with the father
Frequency of feeling depressed
Drug use in the home
Frequency of tobacco use
Frequency of alcohol use
Ever been high at school
Frequency of vandalism

Importance of religion
Frequency of prayer
Suicidal feelings
Perception of intelligence
Reliance on gut feelings
Frequency of being upset by difficult problems
Perception of having good qualities
Frequency of shoplifting
Frequency of running away from home
Frequency of burglary
Frequency of robbery
Frequency of selling drugs
Frequency of taking part in a group fight
Frequency of theft

Readers are advised to consult the Add Health website to view the survey item wording, response options, and descriptive statistics for each covariate at each wave of data collection. This information can be accessed via the links below:

https://addhealth.cpc.unc.edu/documentation/codebooks/

https://addhealth.cpc.unc.edu/wp-content/uploads/docs/documentations/ACE_Instructions_2015-05-12_scd.pdf

https://addhealth.cpc.unc.edu/documentation/codebook-explorer/#/

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Cody Jorgensen is an Assistant Professor in the Criminal Justice department at Boise State University. He earned a PhD in Criminology from the University of Texas at Dallas in 2014. His main research interests include drug policy, gun violence, and biosocial criminology.

Jessica Wells is an Assistant Professor of Criminal Justice at Boise State University. She received her PhD in Criminal Justice and Criminology from Sam Houston State University in 2017. Her research is in the area of psychological and biosocial perspectives on criminal offending. Her publications focus on the consequences of stress and trauma exposure in samples of community members, incarcerated individuals and criminal justice professionals.