The Use of Duration Models in the Estimation of Causal Effects: Applications from Health Economics

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Outline of Talk

- Introduction: An Economists Perspective
- Treatment Effects and Policy Evaluation (DiD)
  - Application: discrete time hazard model
- Treatment Effects and Simultaneity (MMPH)
  - Application: bivariate mixed proportional hazard model

Introduction

- Economists are typically interested in identifying causal effects.
- Causality cannot be "proven" on the basis of data or testing.
- Any statement regarding causality ultimately relies on one or more "identifying assumptions" that cannot be tested directly.
  - Look for potential weaknesses with the identification strategy using "robustness" tests to examine sensitivity of findings.
- Good empirical research (and its proper interpretation) requires:
  - a well defined research question (informed by theory and empirical literature)
  - detailed understanding of the data at hand
  - a good knowledge of the available techniques and how they work
  - a strong understanding of the strengths and weaknesses of various approaches

  in order to marry statistical technique with question and data.

Difference in Difference Estimators & Treatment Effects

Policy Evaluation: the Difference in Difference estimator

- In the simplest case, the set-up requires
  - 2 groups: treatment (T=1); control (T=0)
  - 2 time periods (before (t=0); after (t=1)
  - only one group is treated, and treatment occurs in the "after" period
- The DiD estimator in this set-up is:

$$\delta = (\bar{y}_{T=1,t=1} - \bar{y}_{T=1,t=0}) - (\bar{y}_{T=0,t=1} - \bar{y}_{T=0,t=0})$$

  - $(\bar{y}_{T=1,t=1} - \bar{y}_{T=1,t=0})$ eliminates group specific systematic differences
  - $(\bar{y}_{T=0,t=1} - \bar{y}_{T=0,t=0})$ eliminates time effects common to both groups
- Alternatively, we can use a regression framework to obtain the DiD estimator for the impact of treatment, δ:

$$y_i = \alpha + \beta T_i + \gamma t_i + \delta(T_i.t_i) + \varepsilon_i$$

Policy Evaluation: the Difference in Difference estimator

- Alternatively, we can use a regression framework to obtain the DiD estimator for the impact of treatment, δ:

$$y_i = \alpha + \beta T_i + \gamma t_i + \delta(T_i.t_i) + \varepsilon_i$$

$$\begin{aligned} E[y_i|T_i = 0, t_i = 1] &= \alpha + \gamma \\ E[y_i|T_i = 0, t_i = 0] &= \alpha \\ \Delta E[y_i|T_i = 0] &= \gamma \end{aligned}$$

$$\begin{aligned} E[y_i|T_i = 1, t_i = 1] &= \alpha + \beta + \gamma + \delta \\ E[y_i|T_i = 1, t_i = 0] &= \alpha + \beta \\ \Delta E[y_i|T_i = 1] &= \gamma + \delta \end{aligned}$$

- then the DiD estimator is given by:

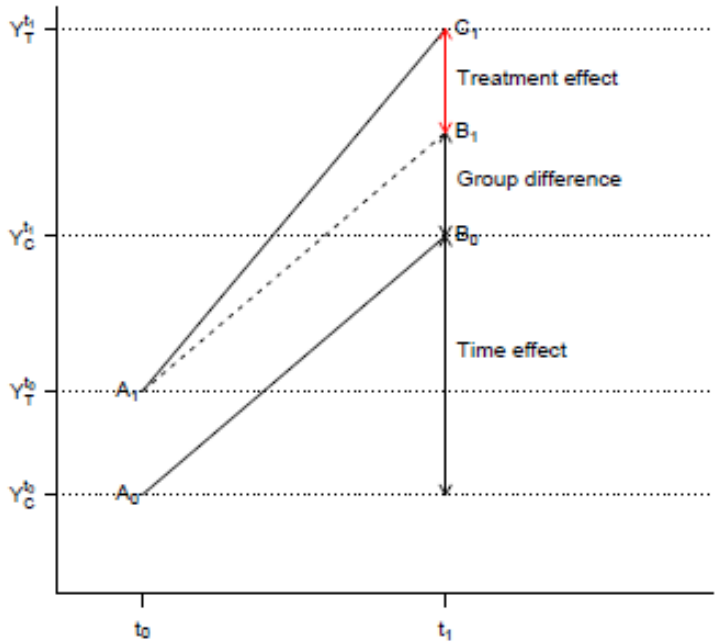
$$\begin{aligned} \Delta E[y_i|T_i = 1] - \Delta E[y_i|T_i = 0] &= \gamma + \delta - \gamma \\ &= \delta \end{aligned}$$

Identifying Assumptions: Common Trend Assumption

$$y_i = \alpha + \beta T_i + \gamma t_i + \delta(T_i.t_i) + \varepsilon_i$$

In addition to assuming that the model is correctly specified, and $E[\varepsilon_i] = 0$, consistency of OLS requires:

1. $cov(\varepsilon_i, T_i) = 0$ (policy exogeneity)
2. $cov(\varepsilon_i, t_i) = 0$ (no compostional change)
3. $cov(\varepsilon_i, T_i.t_i) = 0$ (common trend assumption)



Generalizing Difference-in-Difference Estimation (MRM)

- Observed outcomes can be written as

$$Y_{st} = \gamma_s + \lambda_t + \beta D_{st} + \varepsilon_{st}$$

  - $D_{st} = 1$ if observation belongs to the treatment group and the time period is after treatment has occurred
  - Identifying Assumption: In the absence of treatment, the trend in the outcome for the treated group(s) would have been the same as the trend in the outcome for the control group(s)
- used for treatments that happen at the group-time level
- typically s denotes state since treatment happens at the state level
- examples include compulsory school laws, drink driving laws.....
- requires repeated cross-section sampling from the same aggregate units s (or panel data)
- data can be measured at the group or individual level

Identification and Robustness checks

- The validity of the method depends on having a good control group
- How can you be sure that once systematic differences in group and period have been taken into account, no selection occurs because of anticipation of the unobserved evolution of the potential outcome?
  - "Natural" or "quasi" experiments: sharp changes in the economic environment or changes in state policies that differently affect some categories.
- Robustness checks
  - examine common trends by allowing for state specific pre-existing trends (should not affect estimate of treatment effect)
  - examine causality (Granger sense) by including leads and lags of D_{st} (leads should be jointly zero)
  - use placebo tests (different outcomes, different start date for policy)

Difference in Difference Estimators & Treatment Effects: Application

Application: Does Liberalizing Cannabis Laws Increase Cannabis Use? (JHE, 2014)

This paper is important because:

- Previous research focuses on the impact of decriminalization on participation in cannabis use
  - findings conflicted
- The stock of participants reflect two "flows"
  1. use to non use (quitting) (1/0)
  2. non-use to use (initiators)(0/1)
- Responsiveness (to decriminalization) likely to be greater for the later group
- Therefore conflicted empirical findings likely to be attributable on the age of sample anlayzed
- This paper is conceptually straightforward, focusing on initiation.

Application: Does Liberalizing Cannabis Laws Increase Cannabis Use? (JHE, 2014)

This is the first study to empirically study whether decriminalizing cannabis use leads to an increase in uptake.

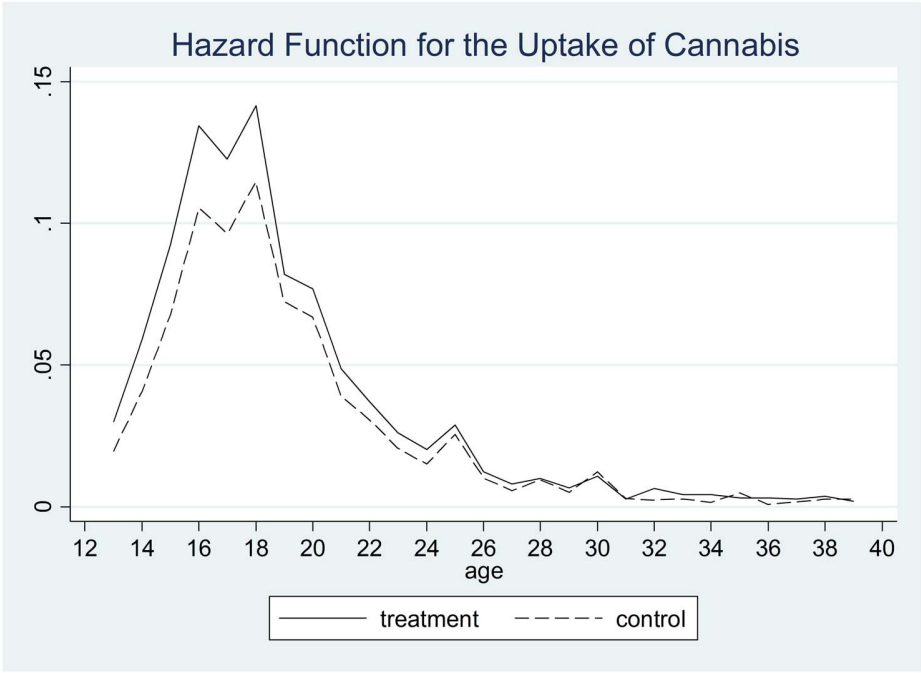
- We identify the causal effect of decriminalization by exploiting a natural experiment:
  - exogenous variation in the timing of the introduction of decriminalization in half of Australia's states and territories.
- Our empirical approach marries the difference-in-difference framework with a discrete time hazard model for the transition into cannabis use.
- We extend the framework to permit
  - heterogeneous treatment effects
  - dynamic treatment effects.

Data: National Drug Strategy Household Surveys

- We pool cross-sectional data from 1998, 2001, 2004 , 2007 and 2010.
- The sample consists 20-40 year olds at the time of survey, N=39,087.
- Retrospective information on age at first use allows us to construct histories assuming individuals at risk of uptake from age of 12.
- This leverages data collected over the period 1998-2010 to cover the period 1970-2010
  - includes the introduction of decriminalization in **all** of the decriminalizing states.
    - ★ 1987: South Australia
    - ★ 1992: Australian Capital Territory (applies to minors & adults)
    - ★ 1996: Northern Territory
    - ★ 2004: Western Australia

Table: Summary Statistics

	Full Sample	Treatment Sample	Control Sample
ever use cannabis	0.57	0.63	0.55
start age	17.51	17.30	17.60
male	0.41	0.43	0.40
age at survey	30.88	31.03	30.81
low education	0.31	0.32	0.31
Australian born	0.79	0.79	0.80
Aboriginal	0.02	0.03	0.02
Lives in a capital city	0.68	0.78	0.65
survey_yr01	0.24	0.23	0.25
survey_yr04	0.24	0.23	0.25
survey_yr07	0.18	0.18	0.18
survey_yr10	0.20	0.19	0.21
VIC	0.21	0.00	0.29
QLD	0.21	0.00	0.30
WA	0.10	0.35	0.00
SA	0.08	0.27	0.00
TAS	0.05	0.00	0.06
ACT	0.05	0.19	0.00
NT	0.05	0.19	0.00
N	39087	11088	27999


Hazard Function for the Uptake of Cannabis
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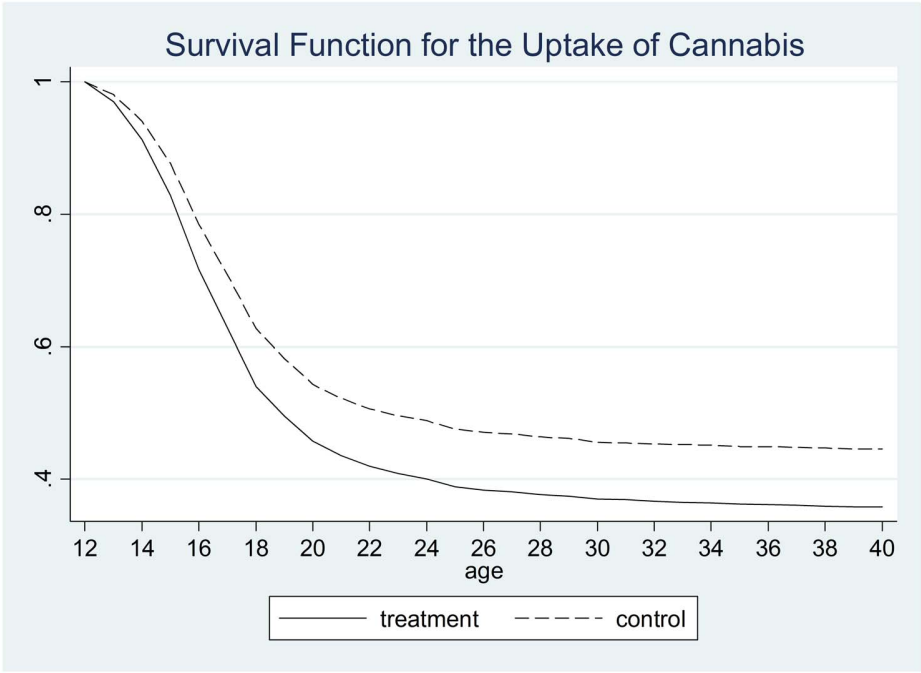
.05
0
12 14 16 18 20 22 24 26 28 30 32 34 36 38 40
age
treatment
control


Survival Function for the Uptake of Cannabis
1
.8
.6
.4
12 14 16 18 20 22 24 26 28 30 32 34 36 38 40
age
treatment
control

Empirical Approach: Discrete Time Hazards

- We observe an individual's spell for interval (year) j, $j = 1, ...J$.
- The spell is either completed ($\delta_i = 1$) or censored ($\delta_i = 0$).
- Define the discrete-time hazard for non-censored observations:

$$\begin{aligned}\lambda_i(j \mid x_{ij}, policy_{ij}) &= P(T_i = j \mid T_i \geq j, x_{ij}, decrim_{ij}) \\ &= \lambda_i(j)\end{aligned}$$

- The corresponding discrete-time survivor function:

$$S_i(j) = \Pr[T_i \geq j)] = \prod_{s=1}^{j}[1 - \lambda_i(j)]$$

- Accounting for censored spells, the density for failure at $T_i = j$ by individual i be written

$$f_i(j)^{\delta_i} S_i(j)^{(1-\delta_i)}$$
$$[S_i(j-1) - S_i(j)]^{\delta_i} S_i(j)^{(1-\delta_i)}$$

Discrete Time Hazards

- Taking logs and summing over individuals we obtain the log likelihood function

$$\begin{aligned}\ln L &= \sum_{i=1}^{N} \delta_i \ln f_i(j) + \sum_{i=1}^{N} (1-\delta_i) \ln S_i(j) \\ &= \sum_{i=1}^{N} \delta_i \ln \left[\frac{\lambda_i(j)}{1-\lambda_i(j)}\right] + \sum_{i=1}^{N}\sum_{k=1}^{j} \ln[1-\lambda_i(k)]\end{aligned}$$

- Define a new binary variable y_{ik} indicating whether the spell of individual i ends in interval k. Then:
- In this case the log-likelihood becomes:

$$\begin{aligned}\ln L &= \sum_{i=1}^{N}\sum_{k=1}^{j} y_{ik} \ln \left[\frac{\lambda_i(j)}{1-\lambda_i(j)}\right] + \sum_{i=1}^{N}\sum_{j=1}^{k} \ln[1-\lambda i(k)] \\ &= \sum_{i=1}^{N}\sum_{k=1}^{j} \{y_{ik} \ln \lambda_i(k) + (1-y_{ik}) \ln [1-\lambda_i(k)]\}\end{aligned}$$

Empirical Approach: Discrete Time Hazard

- We assume a cloglog functional form for the hazard rate
  - this is a discrete time approximation to the proportional hazard model

$$\lambda_{ij} = 1 - \exp(-exp(\theta(j) + \beta' x_{ij} + \gamma' decrim_{ij}))$$

  - x_{ij} contains observed covariates
  - $\theta(j)$ is the duration dependence modeled flexibly using age dummies
  - $decrim_{ij}$ is an indicator equal to 1 if person i faces a decriminalized cannabis regime at age j
  - The parameter of interest is the coefficient on *decrim*, γ.

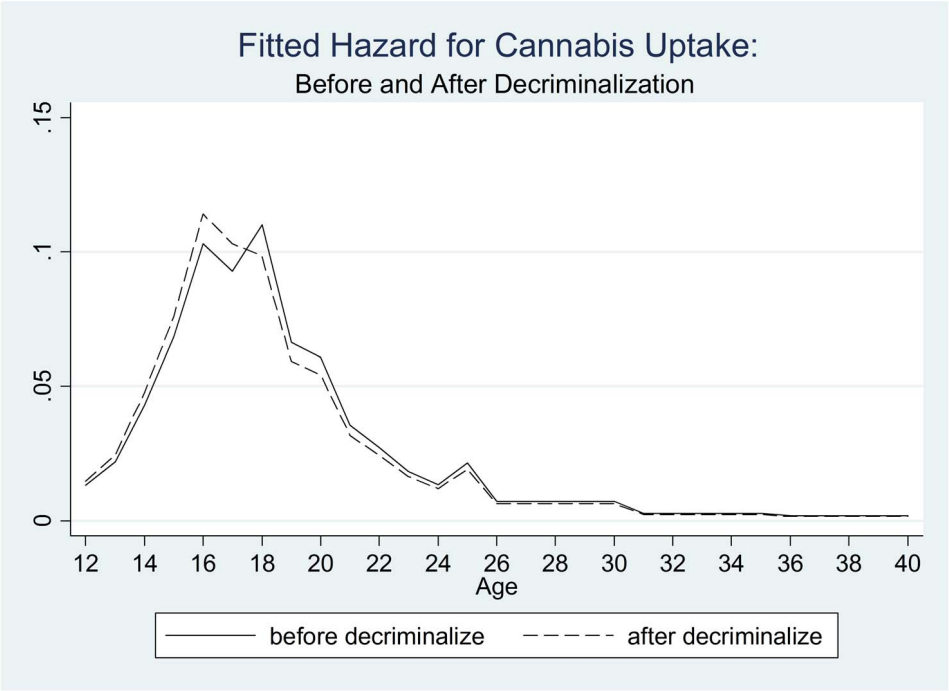
Identification

- The coefficient on *decrim* is the analagous to the d-i-d estimator in a linear model,
- in the hazard setting it is actually a ratio of ratios:
  - the percentage increase in uptake when a treatment state changes from criminalization to decriminalization, relative to the change that occurs over the same calendar time period in a control state
- Identification of the causal effect of decriminalization relies on policy exogeneity
  - In the duration framework, the timing of the introduction of the policy that is important
  - The substantial uncertainty about whether and when legislation related to liberalizing cannabis laws is introduced suggests plausibly exogeneity
  - Plausible exogeneity of the policy strengthened by including state and year indicators

Baseline Results: Hazards Model for Cannabis Uptake

	(1)	(2)	(3)	(4)
decriminalized	0.115**	-0.0905	0.00928	-0.116***
	(0.0572)	(0.0661)	(0.0395)	(0.0303)
decrim*age $<$ 18				0.225***
				(0.0690)
control states				
VIC		-0.0770***	-0.599***	-0.596***
		(0.00455)	(0.0251)	(0.0248)
QLD		0.0314**	-0.356***	-0.356***
		(0.0148)	(0.00757)	(0.00760)
TAS		-0.0675**	-0.841***	-0.842***
		(0.0315)	(0.0130)	(0.0129)
treatment states				
WA		0.306***	-0.0734***	-0.0867***
		(0.00404)	(0.00862)	(0.00682)
SA		0.219***	0.0816***	0.0526***
		(0.0595)	(0.0116)	(0.0107)
ACT		0.250***	-0.0492	-0.0726**
		(0.0181)	(0.0382)	(0.0363)
NT		0.410***	0.413***	0.391***
		(0.0167)	(0.0298)	(0.0255)
state time trends	NO	NO	YES	YES
duration dependence	YES	YES	YES	YES
individual level controls	YES	YES	YES	YES
calendar year fixed effects	YES	YES	YES	YES

Note: The table reports coefficient estimates and standard errors in parentheses; standard errors are clustered at the state level; ***, **, * indicates significance at a 1% 5% or 10% level.


Fitted Hazard for Cannabis Uptake:
Before and After Decriminalization
0
.05
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.15
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36
38
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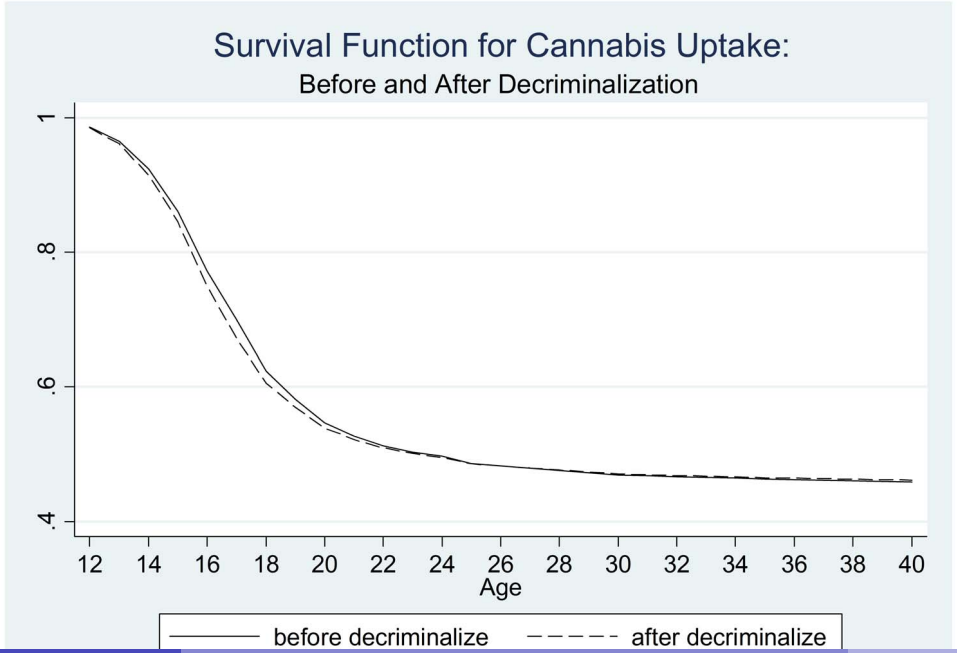
Age
before decriminalize
after decriminalize


Survival Function for Cannabis Uptake:
Before and After Decriminalization
.4
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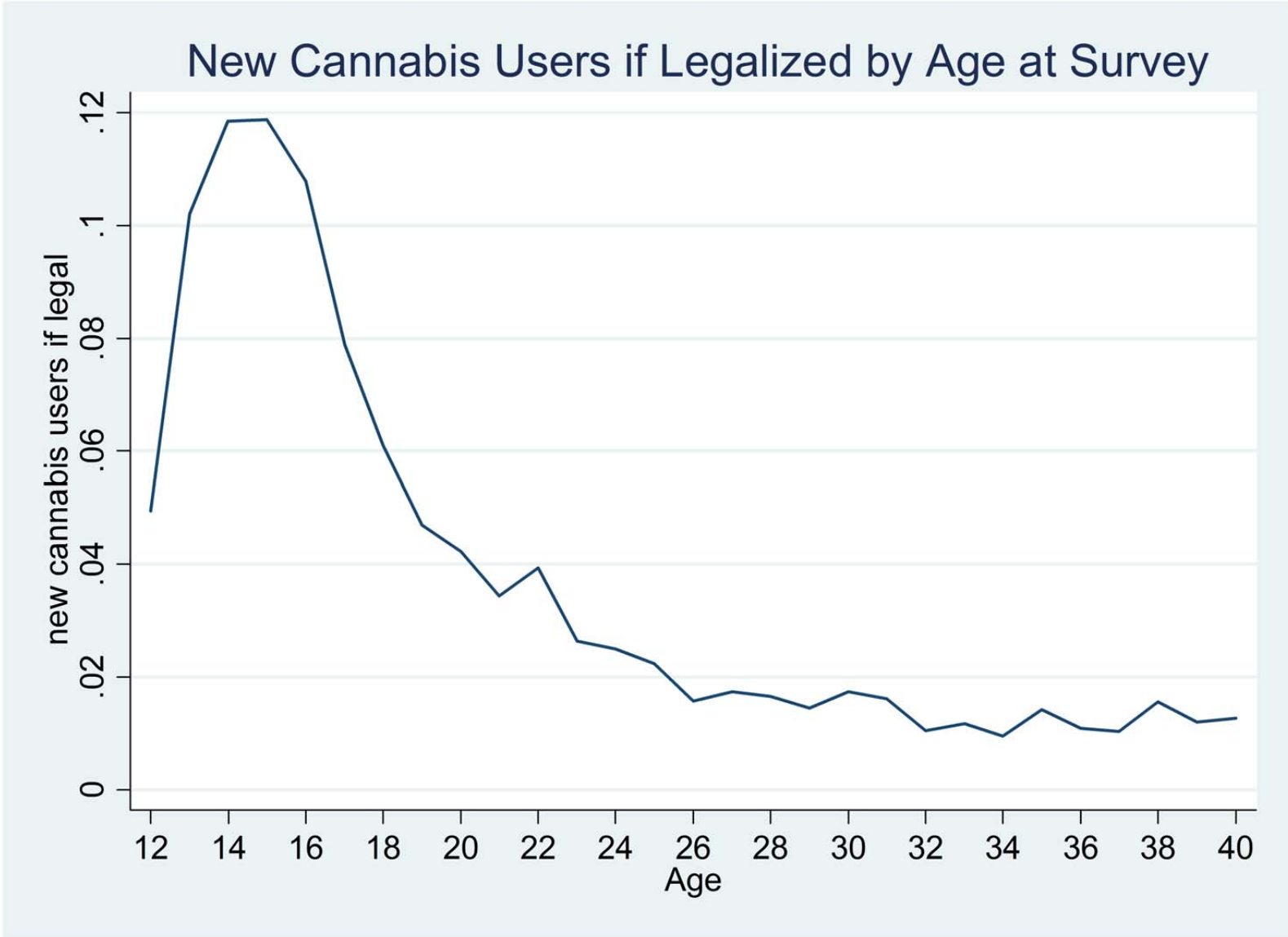
Age
before decriminalize
after decriminalize


New Cannabis Users if Legalized by Age at Survey
new cannabis users if legal
0
.02
.04
.06
.08
.1
.12
12
14
16
18
20
22
24
26
28
30
32
34
36
38
40
Age

Placebo Test: Hazard Models for Cigarette Uptake

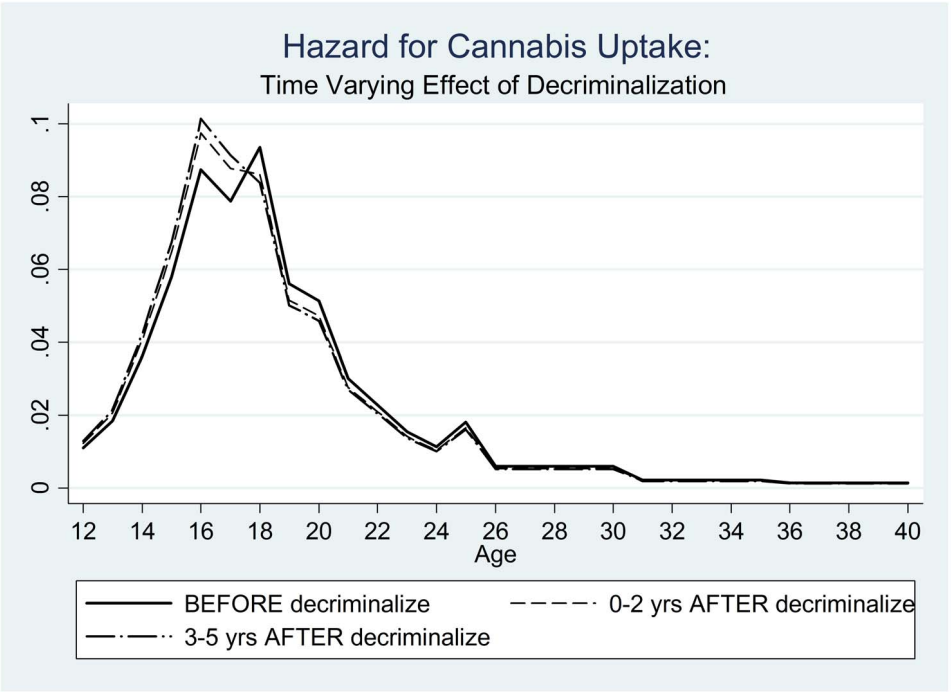
	(1)	(2)	(3)	(4)
decriminalized	-0.0260	-0.119***	0.00672	-0.0331
	(0.0371)	(0.0382)	(0.0458)	(0.118)
decrim*age $<$ 18				0.0535
				(0.113)
control states				
VIC		0.123***	0.187***	0.187***
		(0.00314)	(0.0165)	(0.0165)
QLD		0.0814***	0.125***	0.125***
		(0.00706)	(0.0160)	(0.0160)
TAS		0.0481***	0.0124	0.0121
		(0.0147)	(0.0181)	(0.0180)
treatment states				
WA		0.137***	0.282***	0.280***
		(0.00278)	(0.0146)	(0.0162)
SA		0.152***	0.389***	0.385***
		(0.0279)	(0.0202)	(0.0262)
ACT		0.225***	0.352***	0.348***
		(0.0150)	(0.0260)	(0.0317)
NT		0.200***	0.487***	0.483***
		(0.00906)	(0.0267)	(0.0325)
state time trends	NO	NO	YES	YES
duration dependence	YES	YES	YES	YES
individual level controls	YES	YES	YES	YES
calendar year fixed effects	YES	YES	YES	YES

Note: The table reports coefficient estimates and standard errors in parentheses; standard errors are clustered at the state level; ***, **, * indicates significance at a 1% 5% or 10% level.

Sensitivity Analysis: Hazard Models for Cannabis Uptake

specification	decrim	decrim*age < 18
baseline	-0.116***	0.225***
	(0.0303)	(0.0690)
baseline plus		
age at survey	-0.116***	0.225***
	(0.0302)	(0.0690)
honesty	-0.124***	0.228***
	(0.0277)	(0.0685)
cohort effects	-0.0941***	0.187***
	(0.0347)	(0.0632)
state * <18 interacitons	-0.172***	0.337***
	(0.0202)	(0.0684)
policies for minors	-0.112***	0.232***
	(0.0306)	(0.0734)
males only	-0.0872**	0.205**
	(0.0339)	(0.0835)
females only	-0.131***	0.248***
	(0.0405)	(0.0717)

Note: Each row adds the specified additional controls to the baseline.
The the table reports coefficient estimates and standard errors in parentheses;
standard errors are clustered at the state level;
***, **, * indicates significance at a 1% 5% or 10% level.


Hazard for Cannabis Uptake:
Time Varying Effect of Decriminalization
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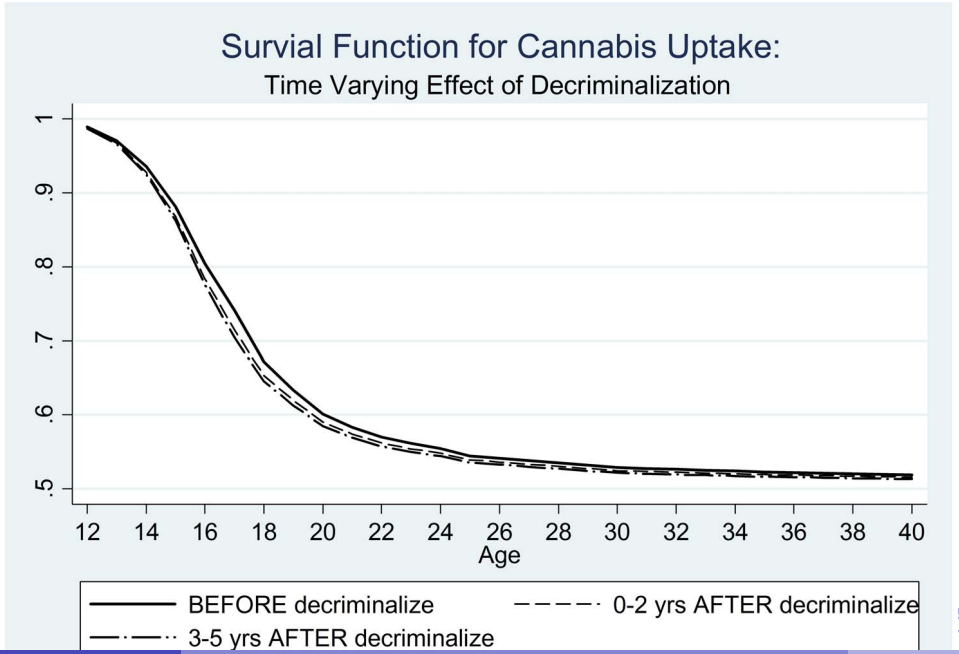
Age
BEFORE decriminalize
0-2 yrs AFTER decriminalize
3-5 yrs AFTER decriminalize


Survial Function for Cannabis Uptake:
Time Varying Effect of Decriminalization
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Age
BEFORE decriminalize
0-2 yrs AFTER decriminalize
3-5 yrs AFTER decriminalize

Contribution of this study

- We demonstrate the importance of allowing for heterogenous treatment effects
  - imposing a homogenous treatment effect lead to the conclusion that decriminalization has no significant effect on uptake
  - this average effect masked an increase in uptake amongst youth and a decrease in uptake amongst adults.
- We reveal the importance of accounting for dynamics in evaluating the effect of a policy change
  - In the short run, decriminalization increases uptake by youth and reduces uptake by adults
  - This largely represents an earlier starting age amongst those who would have otherwise started alter, thus produces only a very small increase in overall use.
  - In the long run, there is no effect of decriminalization on uptake of cannabis for adults or youth.

Bivariate Duration Models & Treatment Effects

Bivariate Duration Models & Treatment effects

- What is the impact of using cannabis on mental health and well being?
- The negative association between cannabis use and mental may be due to
  - Cannabis use adversely affecting mental health and well being
  - Cannabis use occurring in response to poor mental health
  - Common counfounders.
- Suicidal ideation is a measure of (extreme) mental distress.
- The two durations of interest are
  - the duration until the onset of suicidal ideation
  - the duration until initiation into cannabis use
- The treatment effect of interest is the impact of initiation into cannabis use on the onset of suicidal ideation
- The uptake of cannabis is endogenous since
  - unobserved characteristics associated with the onset of suicidal ideation may also associated with the uptake of cannabis
  - the onset of suicidal ideation may lead to the uptake of cannabis

Bivariate Duration Models & Treatment effects

- The hazard rate for suicidal ideation at t conditional on observed x, previous uptake of cannabis, and and unobserved characteristics ν is denoted by $\theta_s(t|x, t_c, \nu)$
- The hazard for or suicidal ideation is assumed to have the MPH specification

$$\theta_s(t|x, t_c, \nu) = \lambda_s(t) \exp(x'\beta_s + \delta_c \cdot \mathrm{I}(t_c < t) + \nu)$$

where

  - x is assumed to be time invariant
  - x is assumed to be independent of ν.
  - $\lambda_s(t)$ is the baseline hazard (duration dependence)
  - $\mathrm{I}(t_c < t)$ is an indicator equal to one of the uptake of cannabis occurred prior to the current period

- The conditional density function for completed durations until the onset of suicidal ideation $t|x, t_c, \nu$ is

$$f_s(t|x, t_c, \nu) = \theta_s(t|x, t_c, \nu) \exp\left(-\int_0^t \theta_s(s|x, t_c, \nu)\, dz\right)$$

Bivariate Duration Models & Treatment effects

- The hazard rate for cannabis uptake at time t_c is given by

$$\theta_c(t_c|x, t_s, u) = \lambda_u(t) \exp(x'\beta_c + \delta_s \cdot \mathrm{I}(t_s < t) + u)$$

  where
  - x is time invariant
  - u is independent of x
  - $\mathrm{I}(t_s < t)$ is an indicator variable, equal to one if the onset of suicidal ideation occurred in a period prior to the current period
  - $\lambda_c(t)$ represents the duration dependence.
- The conditional density function of completed durations until cannabis at $t|x, t_s, u$ can be written as

$$f_c(t|x, t_s, u) = \theta_c(t|x, t_s, u) \exp\left(-\int_0^t \theta_c(z|x, t_s, u)\, dz\right)$$

Bivariate Duration Models & Treatment effects

- Now consider the joint distribution of t_c and t_s conditional on x, u and ν
  - If it is assumed that t_c is exogenous to t_s, then u and ν are independent
    - ★ we would have a standard duration model for $t_s|x, t_c$ in which $\mathrm{I}(t_c < t)$ can be treated as a time-varying regressor that is orthogonal to the unobserved heterogeneity term ν.
  - However, if ν and u are not independent, inference on $t_s|x, t_c$ has to be based on $t_s, t_c|x$.
- Let $G(\nu, u)$ be the joint distribution function of the unobserved characteristics (ν, u).
- The joint density function of t_s, t_c conditional on x is given by

$$f_{s,c}(t_s, t_c|x) = \int_{\nu} \int_{u} f_s(t_s|x, \nu, t_c) f_c(t_c|x, u, t_s) \, dG(\nu, u)$$

Bivariate Duration Models & Treatment effects

- The joint distribution of the unobserved heterogeneity terms ν and u is
  - bivariate
  - discrete
  - has two unrestricted mass-point locations for each term
- Let ν_1, ν_2, u_1 and u_2 denote the points of support of ν and u
- The associated probabilities are denoted as follows:

$$\begin{aligned} \Pr(\nu = \nu_1, u = u_1) &= p_1 \quad \Pr(\nu = \nu_2, u = u_1) = p_3 \\ \Pr(\nu = \nu_1, u = u_2) &= p_2 \quad \Pr(\nu = \nu_2, u = u_2) = p_4 \end{aligned}$$

  with $0 \leq p_i \leq 1$ for $i = 1, \ldots, 4$, and $p_4 = 1 - p_1 - p_2 - p_3$.
- ν and u are independent iff $\operatorname{cov}(\nu, u) = 0$

Bivariate Duration Models & Treatment effects: Identification

- Aberring and van den Berg (2003) show that the (non-parametric) identification of the model

$$\theta_1(t_1|x,\nu_1) = \psi_1(t) \cdot \theta_{0,1}(x) \cdot \nu_1$$
$$\theta_2(t_1|x,\nu_2) = \psi_2(t) \cdot \theta_{0,2}(x) \cdot e^{\delta I(t1<t2)} \cdot \nu_2$$

  requires the standard regularity conditions for each of the hazards plus:
  - independence of x from ν_1, ν_2
  - assumptions on the first moments of ν_1, ν_2
    $E(\nu_1) < \infty$ and $(E(\nu_1\nu_2) < \infty$
  - no anticipation
- The treatment effect is identified without relying on
  - exclusion restrictions
  - parametric functional form assumptions about the distribution of ν_1, ν_2
- This is because the timing of events conveys useful information on the treatment effect.

Bivariate Duration Models & Treatment effects: Identification

Standard regularity conditions for the univariate MPH model

1. $\theta_0(x)$ is positive for all values of x
2. $\psi(t)$ is positive and continuous on $[0, \infty)$, except $lim_{t \downarrow 0} \psi(t)$ may be infinite. For all t $\int_0^t \psi(\tau) d\tau < \infty$, $lim_{t \to \infty 0} \int_0^t \psi(\tau) d\tau = \infty$
3. $G(\nu)$ in the inflow satisfies $Pr[0 < \nu < \infty] = 1$
4. the individual value of ν is time invariant
5. in the inflow, ν is independent of x
6. variation in observed explanatory variables (x takes on at least 2 values)
7. normalizations; for some chosen t_0 and x_0, $\int_0^{t_0} \psi(\tau) d\tau = 1$ and $\theta_0(x_0) = 1$
8. tail of unobserved heterogeneity distribution: $E(\nu) < \infty$

Bivariate Duration Models & Treatment Effects: Application

Application of bivariate MPH model: Cannabis use and suicidal ideation (JHE, 2013)

This paper provides new insights into the complicated relationship between cannabis use and mental health and wellbeing.

- The potential for reverse causality in addition to omitted common confounders need to be addressed.
- The episodic and cyclical nature of suicidal behaviors make identifying the causal effect of cannabis use especially challenging.
- The innovation of our approach is that we consider the relationship between the onset of suicidal ideation and the uptake of regular cannabis use.
  - affords some confidence that we are able to empirically discern the direction of causal pathways linking substance use and suicidal behavior, and quantify the strength of these effects.
- Allowing for differential effects of cannabis use by
  - age of onset
  - intensity of use

provide new insights that resolve conflicts in the empirical literature.

Application of bivariate MPH model: Cannabis use and suicidal ideation (JHE, 2013)

- We provide new evidence on the causal impact of cannabis use on suicidal behaviour of youth.
- The analysis uses the Christchurch Health and Development Study, a 30 year longitudinal study of a cohort of children born in 1977 in Christchurch, NZ
- The contribution of this paper lies in its focus on transitions into the onset of suicidal ideation and the onset of regular cannabis use
  - use a bivariate hazard framework with correlated discretely distributed unobserved heterogeneity
  - this framework permits us to address both sources of endogeniety – reverse causality & unobserved confounders

Prevalence suicidal ideation and monthly cannabis use (percentages)

	Females		Males	
Suicidal ideation	No cannabis	Cannabis	No cannabis	Cannabis
No	220 (73.1)	76 (42.7)	169 (78.6)	146 (59.8)
Yes	81 (26.9)	102 (57.3)	46 (21.4)	98 (40.2)
Total	301 (100.0)	178 (100.0)	215 (100.0)	244 (100.0)

Suicidal ideation and monthly cannabis use – timing of events (percentages)

	Monthly cannabis use	
	Females	Males
Cannabis use first	21 (4.4)	43 (9.4)
Cannabis use same age	18 (3.8)	12 (2.6)
Cannabis use later	63 (13.2)	43 (9.4)
Suicidal ideation, no cannabis	81 (16.9)	46 (10.0)
Cannabis, no suicidal ideation	76 (15.9)	146 (31.8)
No cannabis, no suicidal ideation	220 (45.9)	169 (36.8)
Total	479 (100.0)	459 (100.0)

Empirical Set-up

- We use a bivariate mixed proportional hazard framework:
  - Allow for a direct effect of cannabis use on suicidal ideation AND a direct effect of suicidal ideation on cannabis use
  - Account for correlation in transitions by modeling unobserved heterogeneity as drawn from a joint discrete distribution
- Cannabis use that occurred prior to t can impact on the onset of suicidal ideation at t & only suicidal ideation that occurred prior to t can impact on cannabis at uptake at t
- Identification of treatment effect comes from the timing of events (Abbring & Van den Berg, 2003)

Bivariate Hazard Model

- We specify the joint density function for the duration of time until first suicidal thought and cannabis uptake as

$$h_{c,s}(t_c, t_s \mid x) = \int_u \int_v f_c(t \mid x, t_s, u) f_s(t \mid x, t_c, v) dG(u, v)$$

- $G(u, v)$ is assumed to be a discrete distribution with 4 points of support (u_1, v_1), (u_2, v_1), (u_1, v_2),
- the associated probabilities are modeled using a multinomial logit

Figure: Transition rates to first suicidal ideation

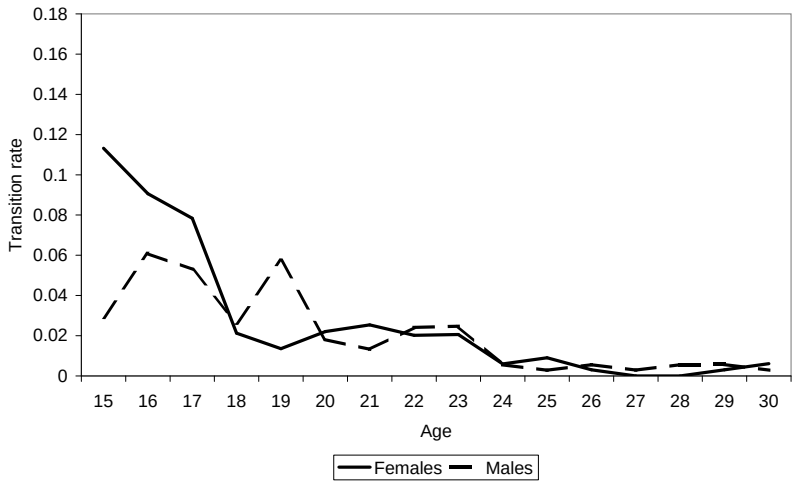


Figure: Transition rates to monthly cannabis use

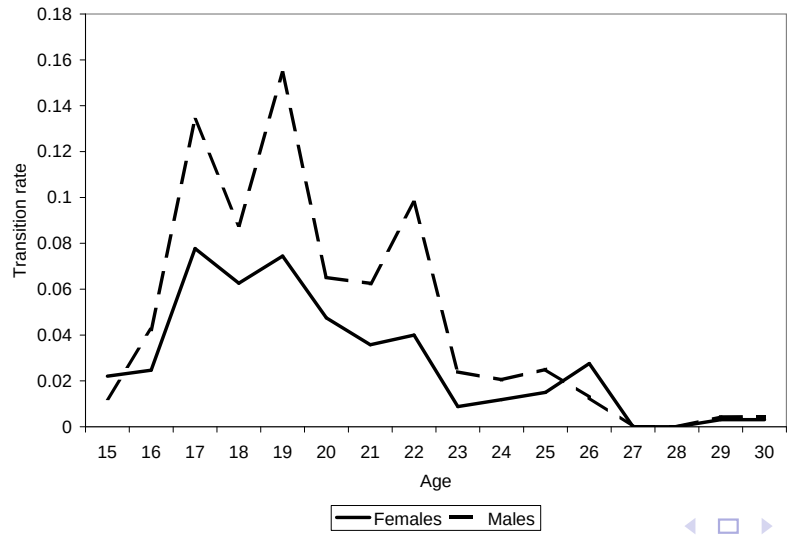


Figure: Cumulative starting probability for the onset of suicidal ideation

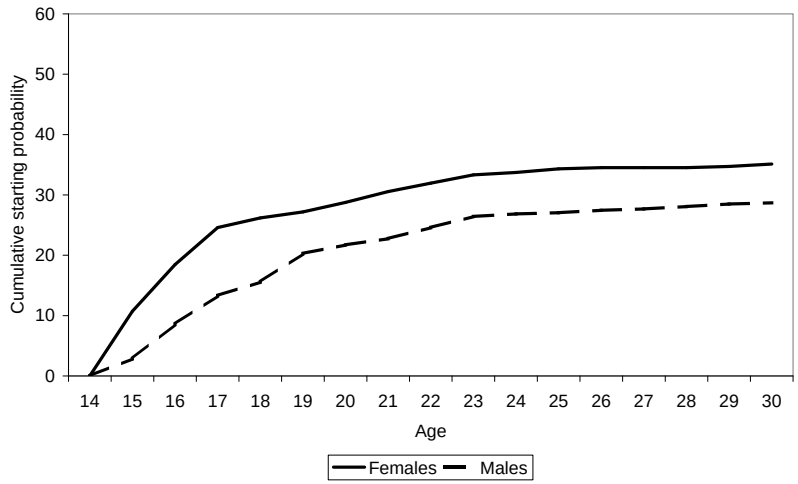
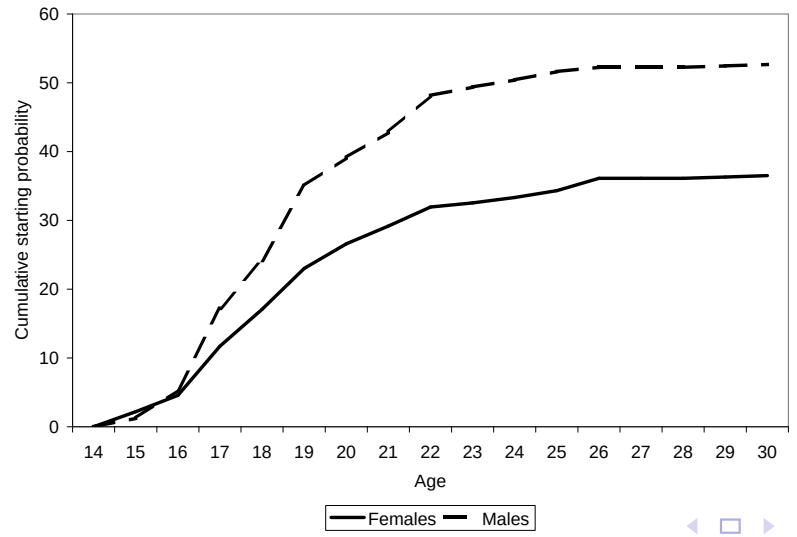


Figure: Cumulative starting probability for the uptake of monthly cannabis use



Results: Distribution of correlated unobserved heterogeneity (monthly cannabis use)

		Females (%)			Males(%)		
Cannabis use		Ever	Never	Total	Ever	Never	Total
Suicidal	Ever	25	20	45	21	10	**31**
Ideation	Never	15	40	55	33	36	69
	Total	40	60	100	54	46	100

Results – Males:
Coefficient estimates (t-statistics)

Cannabis use	Effect Cannabis Use on Suicidal Ideation	Effect Suicidal Ideation on Cannabis use	-Loglikelihood
a. Bivariate MPH Model			
1. At least monthly	0.70 (2.5)**	-0.03 (0.2)	1372.8
2. At least weekly	1.05 (3.8)**	-0.20 (0.9)	1287.7
3. Several times per week	1.33 (5.1)**	-0.01 (0.0)	1187.9
4. Daily	2.81 (7.4)**	-0.08 (0.2)	1048.2
b. Independent MPH Models			
1. At least monthly	0.92 (3.5)**	-0.00 (0.0)	1379.8**
2. At least weekly	1.27 (4.7)**	-0.16 (0.8)	1296.4**
3. Several times per week	1.59 (6.3)**	0.03 (0.1)	1196.3**
4. Daily	3.02 (9.6)**	0.52 (1.8)*	1053.5**

** indicates statistical significance at 5%

Sensitivity Analysis

	Effect		-likelihood
a. Baseline	0.70	(2.5)**	1372.8
d. At least monthly but less than weekly	0.03	(0.1)	
At least weekly but less than several times per week	-0.36	(0.5)	
Several times per week but less than daily	0.80	(1.2)	
Daily	2.83	(2.1)**	1393.8
e. At least weekly but less than several times per week	-0.36	(0.5)	
Several times per week but less than daily	0.76	(2.0)**	
Daily	2.76	(3.2)**	1394.6
f.Several times per week but less than daily	0.79	(2.2)**	
Daily	3.16	(9.4)**	1394.8

Simulations: Proportion of those susceptible who have transitioned into suicidal ideation

		Cannabis use at age 17		Cannabis use at age 20	
	No	More than weekly		More than weekly	
Age	cannabis	Less than daily	Daily	Less than daily	Daily
17	46	46	46	46	46
18	50	**77**	**98**	50	50
19	70	**94**	**100**	70	70
20	75	**98**	**100**	75	75
21	79	**99**	**100**	**91**	**99**
25	90	**100**	**100**	**100**	**100**
30	96	**100**	**100**	**100**	**100**

Conclusions

- Unobserved factors that make individuals more susceptible to suicidal ideation also make them more susceptible to cannabis use
- After accounting for this we find:
  - using cannabis at least several times per week leads to the onset of suicidal ideation in susceptible males
  - suicidal ideation does not lead to the onset of cannabis use in either males or females
- Our results also indicate that
  - the earlier that intense use first occurs, the faster susceptible individuals start having suicidal thoughts, and
  - the higher the frequency of cannabis use, the faster susceptible individuals start having suicidal thoughts.

Summary

- This talk attempts to give a brief overview of some of the methods used in economics in pursuit of identification of causal effects.
- Good empirical work involves thinking about hard about what questions can be reasonably answered with the data at hand
  - what are the strengths of the approach
  - what are the weaknesses of the approach
  - how reasonable are the assumptions required for the identification strategy
- Without identifying assumption, one cannot move beyond correlational analysis for many important questions
  - RCTs are not practical, well-suited, or feasible for many issues
- Governments make policies in the absence of good evidence
- A strong evidence base that makes appropriate use of data and identification strategies is the best defense against poor policy