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The Developmental Effect of State Alcohol Prohibitions at the Turn of the 20th Century

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Abstract:

We examine the quasi-randomization of alcohol consumption created by state-level alcohol prohibition laws passed in the U.S. in the early part of the 20th century. Using a large dataset of World War II enlistees, we exploit the differential timing of these laws to examine their effects on adult educational attainment, obesity, and height. We find statistically significant effects for education and obesity that do not appear to be the result of pre-existing trends. Our findings add to the growing body of economic studies that examines the long-run impacts of *in utero* and childhood environmental conditions.

JEL: I18, D10, N41, N42

Key words: fetal origins hypothesis, alcohol prohibition, World War II

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"John P. Lennon, treasurer of the American Federation of Labor, says that seventy percent of the drink bill of the United States is contributed by the American laboring man…This means that…liquor money is usually bread money, meat money, shoe money, and money that ought to go for clothing."

American Issue, Maryland Edition, June 12, 1909 as cited in Odegard (1928)

1. Introduction

Work by economists provides considerable evidence consistent with the fetal origins hypothesis—that various chronic health outcomes are prompted by an adverse *in utero* environment (e.g., Deschenes et al. (2009), Banerjee et al. (2010)).[1] While the outcomes and conditions vary across studies, the underlying findings emphasize the risks associated with negative exposures during this critical development period. Most studies in the fetal origins literature exploit the variation afforded by temporary adverse *in utero* shocks (e.g., famines) and focus on early life outcomes (e.g., low birth weight). However, recent research in this area examines the effects in adulthood of positive *in utero* and childhood exposures. For example, Hoynes, Schanzenbach, and Almond (2012) find that the beneficial effects of food stamp access *in utero* and during childhood persist into adulthood, suggesting the potential for positive and sustained environmental changes during gestation and in early childhood to have long lasting impacts.[2] Results from

[1] See also Almond and Currie (2011) and Currie (2011) for more citations from this literature.

[2] Related work by Hoynes, Miller, and Simon (forthcoming) finds a positive impact of increased income, through the Earned Income Tax Credit, on the incidence of low birth

Bleakley (2007) suggest higher adult incomes among cohorts in the American South with more childhood exposure to hookworm eradication efforts.[3]

We contribute to this growing literature by exploiting the quasi-randomization of alcohol consumption created by state-level alcohol prohibition laws passed in the U.S. in the early part of the 20th century. We argue that such laws represented a positive shock to individuals who were *in utero* or who were young children around the times of the laws' adoption. Using a large dataset of World War II enlistees, we examine the long term effects of these state prohibition laws on adult educational attainment, obesity, and height. Although we do not observe alcohol consumption and hence our results provide intent-to-treat estimates, our design avoids the reporting problems associated with using more recent data on alcohol use. We find small but statistically significant effects, which do not appear to be the result of pre-existing trends, for two of the three outcome variables.

2. Background

Reduced consumption of alcohol could lead to improved outcomes for those individuals *in utero* or in early childhood during this period through two channels. First, reduced alcohol consumption by the household members who likely consumed the most alcohol during this period, namely men, may have shifted resources to other members of the household, namely women and children. Second, reduced consumption by pregnant

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weight. Improved prenatal care and less negative maternal health behaviors provide the mechanisms for this result.

[3] See also Baird et al. (2011) and Luca (2014).

women themselves would reduce fetal exposure to alcohol. We provide some historical evidence on the potential relevance of these mechanisms in the context of state-level alcohol prohibitions.

2.1 *Intrahousehold shift in resources to women and young children*

Liquor traffic as the "enemy of the home" was a favorite theme of the Anti-Saloon League and other temperance organizations of the time (Odegard, p. 42).[4] The suggestive titles of pamphlets distributed by such organizations included *Better Babies, Unborn Children*, *Why Babies Die*, and *Boys Worth More Than Taxes*. The obvious intention of such propaganda was to convey the message that the saloon culture, and the alcohol consumption that came with it, resulted in adverse outcomes for children and families that would be reversed under prohibition. Determining whether or not this reversal materialized is difficult given the lack of historical consumption data; state-level data on alcohol consumption are not available for this period. However, national data on consumption and other measures that are likely to be associated with consumption provide some evidence to suggest lower alcohol consumption in the period during which many states adopted alcohol prohibition laws.[5] In addition, an analysis of alcohol

[4] Owens (2011) notes "Bars and saloons were depicted in popular culture as places where men wasted money that could have been spent on their families" (p. 5).

[5] Warburton (1932) suggests declines in "per capita consumption of pure alcohol" during the period from 1910 to 1919. LaVallee and Yi (2011) document small reductions in per capita apparent ethanol consumption during the same period. In contrast, Figure 7 in Dills and Miron (2004) does not indicate a decline in "per capita alcohol consumption" until around 1918; the U.S. annual cirrhosis death rate, also reported in their Figure 7,

consumption during the period surrounding federal Prohibition suggests a sharp reduction in alcohol consumption at the onset of Prohibition, which rebounded to 60-70 percent of its pre-Prohibition level within several years (Miron and Zwibel, 1991).

If men, likely the heaviest consumers of alcohol during this time period, reduced their consumption in response to the state prohibitions, then this may have altered the intrahousehold distribution in ways that shifted resources towards pregnant women and young children.[6] The few studies that examine the long-term effects of changes in economic resources early in life suggest the potential for such shocks to impact height, obesity and educational attainment, the three outcomes on which we focus in our estimation. Banerjee et al. (2010) exploit regional variation in the timing of a 19th

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begins to decline earlier, around 1908. Dills and Miron (2004) argue that state prohibitions contributed little to this decline but ultimately conclude "Thus, we are skeptical that the pre-1920 decline in cirrhosis is mainly due to anti-alcohol policies, but we cannot rule out the possibility" (p. 214). Data reported in Blocker (1994) indicates a downward trend in the number of retail liquor and malt liquor dealers per 1000 population that begins around 1907. Studies using more recent data suggest a positive association between outlet (e.g., retail liquor dealers) density and alcohol consumption (see Campbell et al. (2009) and the citations therein).

[6] While not a direct income transfer, the increase in household resources from reduced alcohol consumption could result in reduced maternal stress, which has been shown to improve birth weight (Aizer et al., 2009; Evans and Garthwaite, 2010). Some evidence from developing countries has shown improved birth outcomes from conditional cash transfer programs (see e.g., Barber and Gertler, 2008)

century blight of French vineyards that resulted in a large negative income shock to households in affected regions. Their results suggest that this resulted in shorter heights in adulthood.

Hoynes et al. (2012) document a significant reduction in the incidence of metabolic syndrome (i.e., obesity, high blood pressure, and diabetes) among individuals with access to food stamps in childhood. This finding is consistent with the Barker hypothesis, in which an adverse pre- and early post-natal environment programs the body, through metabolic adaptations, to survive under scarcity (Barker, 1992; Gluckman and Hanson, 2004). In the event such conditions do not arise (i.e., the nutritional environment improves with age), then these adaptations increase the risk of developing a metabolic disorder as an adult.[7] Hoynes et al. also find increases in educational attainment from childhood access to food stamps among women in their sample, findings consistent with a reduction in the anemia and listlessness that may occur in severely undernourished children. Together, the anecdotal historical record and recent empirical evidence provide a potential channel, an intrahousehold shift of resources, through which state prohibitions may result in higher educational attainment, reduced incidence of obesity, and increased height among those with early life exposure.

2.2 *Reduced in utero exposure to alcohol*

Consistent with fostering a healthy *in utero* environment, medical professionals have discouraged alcohol consumption in pregnant mothers for decades. The U.S. Centers for Disease Control (CDC) urges pregnant women not to drink any amount of

[7] Metabolic disorders include obesity, hypertension, type II diabetes, and cardiovascular disease.

alcohol at any time during pregnancy. The primary basis for this recommendation stems from studies that document associations between fetal alcohol exposure and memory limitations, a lack of coordination, learning disabilities, impaired reasoning and judgment skills, language delays, hyperactivity disorder, as well as a host of physical issues.[8] The diagnostic criteria for fetal alcohol syndrome (FAS), the most severe consequence of fetal alcohol exposure, include growth problems, specifically, prenatal height or weight or postnatal height or weight measured at any one point in time putting the individual at or below the 10th percentile for the person's age, sex, and race.[9] Since 2003, fetal alcohol spectrum disorder (FASD) has been used as an umbrella term describing the full range of adverse effects that can occur in an individual whose mother consumed alcohol during pregnancy. FASD may include "physical, mental, behavioral, and/or learning disabilities with possible lifelong implications" (Bertrand et al., 2004; p. 4).

A childhood height deficit is among the criteria for diagnosing fetal alcohol syndrome. Additionally, economic research proposes the use of height as a marker of early life health (Case and Paxson, 2008; Case and Paxson, 2010) and documents associations between adult height and a range of non-health outcomes (e.g., earnings, cognitive ability, employment), including educational attainment. While childhood weight deficits are also among the FAS diagnotic criteria, Klug et al. (2003) suggest that height deficits from FAS persist into adulthood while those in weight begin to dissipate in childhood. Thus, to the extent that we estimate significant effects of prohibition on adult

[8] http://www.cdc.gov/ncbddd/fasd/alcohol-use.html

[9] See http://www.cdc.gov/ncbddd/fasd/documents/fas_guidelines_accessible.pdf for the full criteria.

obesity, our findings underscore the importance of the first causal mechanism, an intrahousehold shift of resources.

There is now general consensus among public health professionals that fetal alcohol exposure is a causal factor in these various adverse outcomes. However, this knowledge is a modern finding, which postdates the period of analysis for our study.[10] Thus, in order to establish a potential casual role for reduced *in utero* exposure to alcohol as a result of state prohibitions, we must establish that women at the end of the 19th century and turn of the 20th century consumed alcohol and therefore may have been less likely to do so as a result of state alcohol prohibitions. Historical sources characterize the degree of alcohol abuse by women of the period. For example, Murdock (1998) indicates that about 15% of patients admitted for treatment at inebriate homes and hospitals were women.

Characterizing women's temperate drinking during this period is more difficult. Murdock (1998) explains the challenge as follows: "The dearth of primary sources on women's moderate drinking has led to the widespread conclusion that nineteenth-century women, or at least middle-class women, did not drink." (p. 51). However, available alternative sources do indicate moderate alcohol consumption by many women of the period. In contrast to consumption by men during this period, which often occurred in public saloons, sources such as cookbooks and etiquette books suggest that consumption by women was more likely to occur in the home (Murdock, 1998). While consuming alcohol at public saloons by women was uncommon (but not unheard of), many saloons, especially those in urban areas, sold alcohol to women for consumption off-site.

[10] Jones and Smith (1973) provide the first description of FAS.

Murdock (1998) describes the common practice of "rushing the growler", filling buckets of beer at the saloons for consumption at home. Murdock also notes that brewers' advertisement campaigns during the period promoted beer's "sterility and nutritional value, a reasonable claim in light of the poor quality of urban milk and water" (p. 54). Alcohol for medicinal purposes was "highly popular and easy to acquire" (Murdock, 1998, p. 52). Physicians treated pain associated with menstruation, pregnancy, childbirth, among other conditions with alcohol. These historical references provide evidence of both alcohol abuse and moderate alcohol consumption by women in the late 1800s and early 1900s and thus support a potential underlying mechanism by which state prohibition laws may have reduced the incidence of fetal alcohol exposure and its attendant adverse effects.

3. Research design and data

Our research design exploits the differential timing of state-level alcohol prohibition laws adopted in the early 1900s to examine average within-cohort effects of alcohol restrictions on health and non-health endpoints among a sample of individuals who were *in utero* or were young children during this period. Compared to the federal Prohibition that was in place between 1920 and 1933, state-level alcohol prohibition laws provide us with more variation in presumed access to alcohol. However, as Dills and Miron (2004) note, prohibition laws varied across states with some states adopting various exemptions (e.g., for home manufacture, importation for personal consumption) and others adopting more restrictive rules (e.g., bone dry prohibition). Dills and Miron (2004) and Owens (2011) provide more detailed discussions of the heterogeneity in state laws. We follow

the convention adopted by Dills and Miron and refer to state laws restricting access to alcohol as state "prohibition" laws; in contrast, Owens refers to these same laws as state "temperance" laws. Figure 1 illustrates adoption years for most states that passed state-level alcohol prohibition laws after 1900 (Dills and Miron, 2004).[11] The distribution of states that adopted state-level alcohol prohibitions was not random. Relative to non-adopting states, adopting states were less industrial, less populated, and more likely to be located in the south or west (Dills and Miron, 2004; Lewis, 2008). As discussed in more detail below, this is not problematic for our research design as we restrict attention to adopting states and exploit differences in the timing of the state-level prohibitions.

[11] Figure 1 includes only those states represented in our analysis. Kansas, Maine, and North Dakota adopted alcohol prohibition before 1900 and are excluded from our analysis. Alabama passed statewide prohibition twice during our study period, first in 1908 (repealed in 1911) and again in 1914. Our research design precludes us from including Alabama in our analysis. New Hampshire first adopted statewide prohibition in 1855 but repealed it in 1903. For most states, the adoption year is the year the law was passed according to Dills and Miron (2004). We confirmed these adoption years using information from the Anti-Saloon League (ASL), specifically maps from the ASL Year Books for the period 1908-1918 provided by the Westerfield Public Library in Westerfield, Ohio. For West Virginia, the two data sources conflict. West Virginia passed statewide prohibition in 1912 but the law did not take effect until sometime in 1914. West Virginia's adoption date as listed in Figure 1 reflects this updated information.

Our identification strategy faces two primary challenges.[12] First, the validity of our design is compromised if the timing of state prohibition laws reflects pre-existing trends in state-level characteristics that may be related to our outcome variables. Our identification strategy addresses this in two ways. First, as in Bailey (2006), we include in our specifications state linear time trends to capture gradually changing unobserved state of birth characteristics. Second, we follow Acemoglu et al. (2004) and Hoynes and Schanzenbach (2009) and include in our specifications interactions between pre-adoption state characteristics and a linear time trend.

In order to identify the appropriate pre-adoption state characteristics for inclusion in our model, we use data from the 1900 Public Use Microsample and the 1901 Statistical Abstract of the United States. For each state listed in Figure 1, we create a "time to adoption" variable that indicates the number of years that elapsed between 1904 and the adoption year. We regress our time to adoption variable individually on various state-level characteristics.[13] Table 1 reports the results of these regressions. The results

[12] Aside from these two primary challenges, our identification strategy would be in question if the adoption of state alcohol prohibitions was temporally clustered with the adoption of other relevant reforms (e.g., women's suffrage). Using the information on state-level suffrage laws in Miller (2008), we regress state prohibition adoption year on the year of women's suffrage for our sample of states. The coefficient on women's suffrage is -0.056 and insignificant, suggesting no discernible relationship. Miller reports similar results using the full sample of states.

[13] This is consistent with Bailey (2006) but differs somewhat from the technique used by Hoynes and Schanzenbach (2009), which would involve regressing our "time to

identify three state characteristics that significantly delayed implementation of statewide alcohol prohibition; states with a smaller percentage of the population living on a farm, black, and native born were slower to adopt state prohibitions. Even in these three models, the predictive power of the observables is low (i.e., the range of R^2 values is 0.19 to .21), which suggests that much of the variation in adoption years is likely idiosyncratic. Our models include interactions between four pre-adoption state characteristics (% population living on a farm, native born, and black; South) and time trends to control for observable differences in state trends that may be spuriously correlated with adoption. We include an interaction with South because the variable was marginally significant in a multivariate time to adoption regression.

Second, an obvious difficulty in using historical state prohibition laws is finding individual-level outcome data to exploit the variation, given how far in the past these changes occurred. Our analysis relies on the Electronic Army Serial Merged File (EASMF), a dataset of World War II enlistment records that have recently been digitized and made available through the National Archives and Records Administration (NARA).[14] The full dataset includes information for the majority of individuals who

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adoption" variable on all of the state-level characteristics simultaneously. In such a model, which has an R^2 of 0.43, only the variable measuring race is individually significant (South is marginally significant) so we opted for the univariate regressions.

[14] The original sources for the digitized data were punch cards, which contained basic information about enlistees, recorded at the time they entered service. The punch cards were destroyed after being microfilmed. See Hull (2006) for a more detailed discussion of the dataset's history.

enlisted in the United States Army during World War II, comprising information for over nine million individuals.[15] The EASMF sample is representative of men who served but not necessarily of the U.S. population of draft-age men due to various service criteria (Bleakley et al., 2014; Acemoglu et al., 2004; Goldin and Olivetti, 2013).

The data contain limited control variables. However, importantly for our study, the data include the individual's birth year, state of birth, race, enlistment year, and educational attainment, as well as the individual's height and weight for those who enlisted prior to 1943 (Hull, 2006).[16] We use the height and weight information to calculate body mass index (BMI) and classify those men with BMIs greater than or equal to 30 as obese.

Like Bleakley et al. (2014), we implement a set of sample restrictions to obtain samples that are more likely to be representative by cohort. We construct two primary estimation samples, one for the education outcome and another for the obesity and height outcomes.[17] The first set of restrictions applies to both samples. First, we drop duplicate observations from the raw data set as well as observations with invalid values for enlistment year and missing values for birth state. We also drop members of the Enlisted

[15] Thirteen percent of the original records were unreadable (Hull, 2006).

[16] Beginning in 1943, the "height" and "weight" fields were used for other purposes (i.e., to indicate Military Occupational Specialty).

[17] We are unable to implement the exact sample restrictions used in Bleakley et al. (2014) due to our focus on height and weight and the limited availability of these measures in the EASMF data. Our restrictions on age of enlistment, race, gender, height, and weight mimic theirs.

Reserve Corps due to the potential for miscoding errors among these observations.[18] About 8.3 million observations survive this process.[19] Second, we restrict our sample to white men. This excludes members of the Women's Army Auxiliary Corps (WAAC) and non-white men. Members of the WAAC are a self-selected sample of women and are therefore unlikely to be representative of the general female population during the study period.[20] Black men were much less likely to have served (Acemoglu, Autor, and Lyle, 2004; Goldin and Olivetti, 2013). Third, we restrict the sample to those men born between 1904 and 1923.[21] Forth, we include only those men who enlisted between the ages of 20 and 45, with the latter restriction consistent with formal enlistment

[18] Members of the Enlisted Reserve Corps may also differ systematically from regular Army enlistees and generally from other members of their birth cohorts.

[19] The raw data include 9,200,232 observations. 162,266 of these are duplicate observations; of these 41,896 have invalid values for enlistment year; of these 495,588 missing values for birth state; of these 207,637 represent members of the Enlisted Reserve Corps.

[20] WAAC members represent less than 2% of the raw EASMF dataset. 80% of the observations in raw dataset represent white individuals.

[21] 1903 is the year in which New Hampshire repealed its first statewide alcohol prohibition, adopted in 1855. The 1923 restriction is consistent with Bleakley et al. (2014).

requirements.[22] Fifth, we limit attention to individuals who were born in the states listed in Figure 1, which accounts for almost half of this sample. Sixth, we restrict the sample to draftees (i.e., "selectees") and therefore exclude men who voluntarily enlisted. Relative to voluntary enlistees, draftees are more likely to be representative of their respective cohorts.

Finally, the samples include only those men born more recently than ten years before the adoption of prohibition in their birth states, which excludes from our analysis men who were first exposed after age 10.[23] Of those observations that remain before this imposing this restriction, less than three percent were first exposed after age 10. These men are among the oldest in the sample; the mean age among these men is 36 compared to 25 for other men in the sample. As such they are likely to be systematically different from other members of their cohorts.

The obesity/height sample reflects additional restrictions. Due to the data limitation noted earlier, these samples include only those men who enlisted between 1938 and 1942. Consistent with drafting criteria, the obesity/height sample includes men with heights between 60 and 78 inches who weighed at least 105 pounds. Table 2 presents summary statistics for the two samples. Because we observe educational attainment for individuals regardless of their enlistment year, the education sample includes almost a

[22] The age ranges of the samples we ultimately use in estimating our models are somewhat narrower due to other exclusion restrictions (e.g., based on year of birth) and data limitations (e.g., on our height variable).

[23] We report the robustness of our results to relaxing this assumption in the appendix. See the related discussion in footnote 27.

million more observations than the obesity/ height sample.[24] Relative to the education sample, the obesity/height sample is slightly younger with lower educational attainment and less exposure to state alcohol prohibition. The prevalence of obesity in our data is 2.25%, which is low relative to contemporary comparisons but in line with other estimates of obesity rates in the early 1900s. Helmchen and Henderson (2004) estimate the prevalence of obesity at around 3.7% among a sample of non-Hispanic white men between the ages of 40-49 years old in 1890-1894. To provide some basic evidence of representativeness, we compare the educational attainment for our samples to the U.S. population using Census data. According to the 1940 Census, 38.9% of white males between the ages of 25 and 29 completed 4 years of high school or more. Among white male draftees between the ages of 25 and 29 in our education (obesity/height) sample, 39.54% (39.93%) completed at least four years of high school.

4. Econometric specification and results

4.1 *Event study*

Before proceeding to our primary specifications, we report the results of an event study to provide some intuition and a graphical depiction of our data. To do so, we create a variable "years-from-dry", which indicates the number of years between an individual's birth and the year in which his state adopted a statewide alcohol prohibition provision. That is, for a particular individual, years-from-dry is equal to the individual's birth year

[24] We follow Bleakley et al. (2014) and assign an educational attainment equal to 4.5 for individuals whose educational attainment is listed in the data as exactly 8 years. The data do not include values of educational attainment less than 8 years.

minus the year in which his birth state adopted prohibition. This variable will be negative (positive) for individuals born before (after) their birth state adopted prohibition and zero for individuals born in the year of adoption. Values of years-from-dry around one denote individuals who were *in utero* during their birth state's adoption.[25] We create a set of fixed effects, one for each value of years-from-dry, and use these to flexibly explore the effects of state alcohol prohibition. In contrast to a sharp research design, this flexible design allows us to identify potentially different effects of statewide prohibition adoption on individuals of different ages (e.g., *in utero*, in early childhood). However, an important weakness of this design is its failure to account for pre-existing trends in state-level characteristics that may be related to our outcome variables. For our application, the event study design is not amenable to the identification strategy we describe above and therefore it should be viewed with this limitation in mind.

For our event study analyses, we estimate equations of the general form:

$$Y_{ibs} = \alpha + d_y'\beta_y + \eta_s + \varpi_c + \varepsilon_{ibs} \qquad (1)$$

where Y_{ibs} denotes educational attainment, binary obesity status, or ln(height) of individual *i* born in year *b* in state *s* and d_y denotes the set of years from dry fixed effects. State of birth and cohort-by-age at enlistment fixed effects are denoted η_s and ϖ_c, respectively. β_y denotes the coefficient vector of interest. Standard errors are clustered on state and year of birth.

[25] Because we know only year of birth (not month or date) and the year in which the state adopted statewide prohibition (not the month or date of adoption), we cannot identify the "years from dry" values that correspond to individuals who were *in utero* with certainty.

Figures 2 through 4 display the estimated coefficients and 95 percent confidence intervals on the years-from-dry fixed effects from our event study analysis. The reported coefficients are interpreted relative to the excluded category of -10 (i.e., denoting men who were born ten years before their birth state adopted prohibition). The three figures illustrate a similar pattern in that significant effects of exposure generally occur around a years-from-dry value of zero, which indicates those men born in the year of adoption. However, the estimated effects of exposure are more pronounced for educational attainment and obesity than for height. Figure 2 suggests significant positive effects of exposure on educational attainment for years-from-dry values between -4 and 8 (i.e., men born between four years before and eight years after their birth states adopted prohibition). Figure 3 indicates negative and significant effects of exposure on obesity for those born between about four years before and four years after their birth states adopted prohibition.

For extreme values of years-from-dry, the magnitudes of the estimated coefficients become smaller and our estimates become noisier. We offer two explanations for this result. First, individuals with high values of years-from-dry are more likely to have been *in utero* or in early childhood during World War I which may help to explain the shape of the figures. Brown (2011) provides evidence of lower income, health, and education of the parents of the 1919 birth cohort, relative to surrounding cohorts and argues that U.S. involvement in World War I in 1918 explains this result. Individuals who were children during World War I were affected in other ways (e.g., death or injuries of fathers, changing role of mothers in household, more caregiving responsibilities for younger siblings) that could contribute to our results.

Second, due to the composition of the data, high values of years-from-dry represent significantly fewer states and fewer birth years than moderate values, diminishing our ability to obtain precise estimates. While our event study results are suggestive of significant effects of exposure to state prohibitions at early ages, they do not allow us to rule out the possibility that the observed effects are due merely to underlying trends in our three outcome variables. Our main econometric specifications address this issue using the identification strategy we introduced above.

4.2 *Primary specifications*

For our main analysis, we estimate models of the following general form:

$$Y_{ibs} = \alpha + \gamma Pro_{bs} + \eta_s + \lambda_a + \varpi_c + \eta_s \cdot b + \theta S1900 \cdot b + \varepsilon_{ibs} \quad (2)$$

where Y_{ibs}, η_s, λ_a, and ϖ_c are defined as in equation (1). Pro_{bs} is the measure of exposure to state alcohol prohibition (i.e., the treatment) in early life. The coefficient of interest is γ. Linear state of birth trends, $\eta_s \cdot b$, control for unobservable state-specific trends.[26] The specifications also include interactions between pre-adoption characteristics of the state of birth and linear trends in year of birth ($S1900 \cdot b$). Standard errors are clustered on state and year of birth.

Our various measures of exposure are in the spirit of Hoynes et al. (2012) with some modifications to reflect our reliance on state and year of birth variation for identification. Our exposure measures use information on birth year and the year in

[26] Our main results are qualitatively similar if we enhance the set of fixed effects to include state-by-enlistment year fixed effects or state-by-age at enlistment fixed effects. Results are also robust to excluding the state-specific trends.

which each state implemented prohibition as we do not observe the specific date of birth or the exact date on which prohibition took effect. Our main exposure measures, *Exp8* and *Exp10*, indicate the number of years of exposure to state alcohol prohibition before ages eight and ten, respectively. Summary statistics for our exposure measures are given in Table 2.

Table 3 contains the results of estimating equation (2) for the three outcome variables, education, obese, and ln(height).[27] The first columns of the table indicate significant education and obesity effects of early exposure to state alcohol prohibition. We do not detect significant treatment effects for height although the estimated impacts are positive. For the education models, the estimated coefficient on *Exp8* suggests that an additional year of exposure to state alcohol prohibition before age eight increases educational attainment by about 0.04 years. [28] Because our estimates are intent-to-treat,

[27] Appendix Table A1 reports results from the same models estimated with the sample of all enlistees. The estimated effects with the inclusion of voluntary enlistees, in addition to draftees, are qualitatively similar to those reported in Table 3. Appendix Table A2 reports results from the models estimated without the years-from-dry restriction using the sample of draftees. In general the estimated coefficients are smaller and less precisely estimated than those reported in Table 3. We also estimated the models using samples that exclude draftees who were born in 1918 and therefore may have been exposed *in utero* to Spanish influenza (see Almond, 2006). Our results (unreported but available from the authors) are also robust to this change.

[28] Note that the predicted relationship between health improvements in early life and educational attainment is ambiguous; if brawn is of relatively greater value than brain in

an assessment of the magnitude of this effect requires information on the exposed population. Only those individuals born to drinking households would be potentially affected by the treatment. The paucity of information on the demographic profile of drinkers during this historical time period makes it difficult to obtain a precise estimate of this figure. We, can, however, use the available statistics to provide a rough range of the exposed population. The earliest available estimates characterize alcohol consumption in the 1940s, two to three decades after the time period of analysis for our study. According to Efron and Keller (1963), 75% of men and 56% of women were drinkers in 1946. Efron and Keller also provide estimates of the average number of alcoholics in a given year between 1940 and 1945 by gender—2,970,000 million men and 530,000 women or 4.5 percent of the male population and 0.81 percent of the female population.[29] These figures allow us to develop rough bounds on the treatment-on-the-treated estimates. Applying the figures for drinkers to our education results suggests treatment-on-the-treated estimates between 0.53 and 0.71 additional years of education per year of exposure under age 8. These estimates are of course larger, 0.89 and 4.94 additional

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the labor market, then improvements in child health could actually increase the opportunity cost of schooling. See Yamaguchi (2008), Bleakley (2010), Pitt et al. (2012), Bleakley et al. (2014).

[29] This calculation assumes a total male population of 66,061,592 and female population of 65,607,683 (Grove and Hetzel, 1968). Efron and Keller arrive at estimates of the total number of alcoholics by multiplying by a factor of five estimates of the number of "alcoholics with complications" based on the Jellinek formula. The Jellinek formula uses information on the number of deaths from cirrhosis of the liver.

years of education respectively, when we use instead the percentages of alcoholics in the population. Excluding the implausibly large effect of almost five additional years based on the estimated proportion of female alcoholics, these estimated effects imply percentage increases in educational attainment between 5.8% and 9.8% per year of exposure up to age 8.

For obesity, the negative and significant estimated coefficients on *Exp8* and *Exp10* suggest a reduction in the probability of obesity with additional years of exposure to state prohibition in childhood. Based on estimates of the proportion of drinkers, the range of treatment-on-the-treated estimates is from a 0.11 to a 0.14 percentage point reduction in the probability of obesity with each additional year of exposure up to age 8. Given the sample mean value of obese, 2.25 percent, the estimated coefficient on *Exp8* corresponds to a treatment-on-the-treated effect of about 5%.

Although we fail to estimate statistically significant effects of exposure to state prohibitions on height, it remains instructive to gauge the magnitude of effects implied by the estimated coefficients. Again applying estimates of the proportion of drinkers in the population, the estimated coefficient on *Exp8* implies an increase in height of between 0.01 and 0.013 inches for each year of exposure up to age eight. With eight years of exposure, this would translate into about an additional 0.09 inches. While this effect appears small given the sample mean height of 68.61 inches, we can also compare the estimated effect to the increase in height experienced by men during this time period. According to Fogel et al. (1983), mean height among U.S. males grew at a rate of 1.2 inches per generation (i.e., 30 years) between cohorts born in 1906 and 1921. Viewed in this light, the estimated effect of exposure on height is larger but remains fairly modest.

4.3 *Mechanisms*

We explore the potential mechanisms that may underlie our results through three additional exercises. First, we examine the relative importance of being first exposed to state prohibition *in utero* and in early childhood exposures by defining alternative exposure variables. As in Hoynes et al. (2012), exposure in this context is "from above", which implies that someone exposed *in utero* was also exposed as a child. The variable, *Child_exp*, takes the value of one for individuals born between five years before and one year before their birth state adopted prohibition (i.e., men who were first exposed between the ages of about five and one). *Full_exp* takes the value of one for individuals born in the year of adoption or after adoption (i.e., men who were exposed *in utero* and as children).[30] With both of these exposure variables included in equation (1), the excluded category is men who were first exposed to state alcohol prohibition between the ages of about five and ten.

Table 4 reports the results of estimating our primary specifications with these two alternative exposure measures for education, obesity, and ln(height). The fourth column of the table reports p-values for tests of equivalence between the estimated coefficients on *Child_exp* and *Full_exp*. The final column reports p-values for tests of joint significance. In general, the coefficients are less precisely estimated in these models. For education, the estimated coefficients suggest higher educational attainment for men first exposed as young children or *in utero* compared to men first exposed as older children. Although the latter result is statistically insignificant, the two coefficients are

[30] Because we observe only the year of birth and the year a state adopted prohibition, this variable provides a measure of approximate *in utero* exposure.

jointly significant. We find a similar result for height but the two estimated coefficients in the obesity model are not jointly significant at conventional levels. For all three outcome variables, we fail to detect a significant difference between the two estimated coefficients. As a result, the results reported in Table 4 do not allow us to distinguish between *in utero* and early childhood initial exposure as the primary driver of our earlier results but rather suggest potentially important exposure effects during both developmental periods.

The fact that both periods of exposure appear contribute to the observed effects does, however, provide some insight into the relative importance of the two mechanisms.[31] Nilsson (2014) finds that a policy that sharply increased alcohol

[31] A third plausible mechanism for our findings, but one which we unfortunately are unable to explore empirically, is a reduction in violence associated with lower alcohol consumption. While early time series evidence found a positive association between the temperance movement and crime (Dills and Miron, 1999), results from more recent panel data analysis indicate a positive association between dry laws and the homicide rate in most states (Owens, 2010). Other recent studies also find a reduction in crime with restrictions on drinking. Bleakley and Owens (2010) find that the passage of county-level dry ordinances reduced the incidence of lynchings. One of the mechanisms proposed to explain this result is a changed pattern of social behavior resulting in young men spending "less time in saloons, and more time engaged with family members..." (p. 3). Results from Luca et al. (2014) suggest lower rates of violence against women among Indian states with higher minimum legal drinking ages. See also Cook and Durrance (2013).

availability, and alcohol consumption, during a short period in Sweden in the 1960s resulted in lower wages and educational attainment for those individuals exposed to the policy *in utero*. Similar effects were not detected among those cohorts exposed to the policy as young children. If Nilsson's results are driven, as he suggests, by increased maternal alcohol consumption, then they indicate that this channel has important long-term effects for those exposed *in utero* but not as young children. Given this, the similar effects that we detect for both periods of exposure provide some evidence that an intrahousehold shift in resources, not a reduction in maternal alcohol consumption, is the primary mechanism underlying our results.

Second, we follow Owens (2011) in constructing a proxy for the demand for illegal alcohol in a state during prohibition and allow the effect of early exposure to vary with this measure. Owens (2011) proposes the ratio of wet (i.e., against) to dry (i.e., for) votes for the state prohibition law as a proxy for the demand for illegal alcohol. We construct a similar measure, denoted, *Vote_ratio*, using information reported in her Table 1 (p. 6). Unfortunately, vote counts are unavailable for eight states (Georgia, Mississippi, Alabama, Arkansas, Iowa, Indiana, and New Hampshire) represented in our early analyses. As a result, draftees born in these states are excluded from the estimating samples for this robustness check. If the reduction in alcohol consumption due to state prohibition is lower in states with a higher demand for illegal alcohol, then the effect of early exposure should be attenuated in these states. Alternatively, because the wet-dry vote ratio indicates the strength of resistance within the state to passing state prohibition, it may also provide a measure of alcohol consumption within the state prior to the state prohibition. If pre-prohibition alcohol consumption was high and the state prohibition

was effective in reducing consumption, then we would expect larger reductions in consumption following prohibition. This would suggest a larger effect of early exposure in states with high values of *Vote_ratio*.

Table 5 reports coefficient estimates from specifications that include exposure measured by *Exp8* as well as an interaction between *Exp8* and *Vote_ratio*.[32] The signs of the estimated coefficients on the interaction terms for all three outcome variables suggest a larger effect of exposure in states with a higher wet-dry ratio. Thus, the empirical results are more consistent with the wet-dry ratio proxying for the level of pre-prohibition consumption of alcohol than for the demand for illegal alcohol post-prohibition. To facilitate comparisons with our main results, the fourth column of the table reports the estimated effect of early exposure evaluated at the sample mean of *Vote_ratio* for the two estimating samples. The estimated effects of early exposure at the mean of *Vote_ratio* are significant for education and obesity but not for height, consistent with our main results.

The final exercise explores the potential effects of heterogeneity in state prohibition laws on the estimated effects of exposure to state prohibitions. As mentioned earlier, some state prohibition laws were more stringent than others. A priori the effect of exposure to a more stringent prohibition law relative to a less stringent law is ambiguous. On the one hand, a more stringent law could encourage a more active underground market and potentially more potent alcohol as people resorted to home production. On the other hand, a more stringent law could be more effective in curbing consumption. To explore this empirically, we create a dummy variable, *Prohib*, which takes the value of

[32] Results are similar for *Exp10*.

one for states that adopted outright (i.e., bone dry) prohibition, and zero for states with prohibition laws that allowed importation or home production for personal use (i.e., temperance) (Owens, 2011). We then interact this variable with our measure of exposure, *Exp8*. Table 6 reports the results. The final column reports the estimated coefficient of an additional year of exposure before age eight under outright prohibition (i.e., the sum of the coefficients on *Exp8* and *Exp8*prohib*). The results for education suggest a significantly larger effect of exposure in states with outright prohibition, relative to temperance states while the results for obesity suggest the opposite. The inconsistent results across these two outcome variables may be explained by some systematic unobserved difference in the set of states that adopted outright prohibition rather than temperance.

5. Conclusion

Recent research in the fetal origins literature suggests the potential for positive changes in the *in utero* and/or early childhood environment to have long lasting effects that persist into adulthood. We document such effects associated with pre- and early post-natal exposure to statewide alcohol prohibitions at the turn of the 20th century. Specifically, we find that those adult men in our sample exposed to prohibition *in utero* and as young children enjoy an increase in educational attainment and a decrease in the likelihood of obesity. We also find small, positive effects on adult height but these effects are never statistically significant. These findings are consistent with the hypothesis that prohibition impacted *in utero* and early childhood environmental conditions in positive ways. While our data prevent us from definitively identifying the precise channel through which these

effects arise, our findings are more consistent with an intrahousehold shift in resources than with reduced maternal consumption of alcohol.

It's important to note that while our analysis documents positive benefits of alcohol prohibition during this historical time period, it does not speak to the attendant costs. In addition, because of important differences between the alcohol culture in the early 1900s and the modern-day alcohol and drug cultures, we caution against extrapolating our results to current debates on alcohol and drug policies.

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Table 1. 1900 state-level predictors of statewide alcohol prohibition timing

Variable	Sample mean (standard deviation)	Estimated coefficient (robust standard error)	R^2
% of population living on farm	45.89 (17.22)	-0.091* (0.038)	0.20
% of population black	13.12 (18.57)	-0.088* (0.038)	0.21
% of population unemployed	8.64 (2.28)	-0.061 (0.34)	0.0015
% of population native born	88.89 (9.05)	-0.17* (0.066)	0.19
% of population age 5 to 18 enrolled in school	73.45 (11.84)	0.029 (0.052)	0.0095
Population density	25.68 (25.22)	-0.0069 (0.026)	0.0024
South	0.34 (0.48)	-2.22 (1.60)	0.092

Table notes: * denotes significance at 5% level. The first three variables were created using PUMS data and second three variables were created using the 1901 Statistical Abstract of the United States.

Table 2. Variable descriptions and summary statistics

Variable name	Description	Mean (standard deviation) or percent of sample for binary variables	
		Education sample	Obesity/height sample
Age	Age at time of enlistment	25.56 (4.57)	25.28 (4.30)
Height	Height in inches at time of enlistment	--	68.51 (2.55)
Weight	Weight in pounds at time of enlistment	--	149.57 (23.89)
Obese	= 1 if body mass index at time of enlistment is greater than or equal to 30, = 0 otherwise	--	2.25
Education	Educational attainment between eight (grammar school) and seventeen (post-graduate) at time of enlistment.	9.06 (3.72)	8.94 (3.78)
Exp8	Number of years of exposure up to age 8	6.73 (2.36)	6.66 (2.40)
Exp10	Number of years of exposure up to age 10	8.69 (2.50)	8.61 (2.55)
Child_exp	= 1 for men who were first exposed between the ages of about five and one	20.45	22.21
Full_exp	= 1 for men who were first exposed *in utero*	69.66	67.46
Range of enlistment years represented in sample		1939-1946	1940-1942
Range of ages represented in sample		20-41	20-38
Range of birth years represented in sample		1904-1923	1904-1922
Number of observations		1,704,191	1,389,781

Table 3. Estimated effects of early exposure to state alcohol prohibition

Exposure variable	Dependent variable					
	education		obese		ln(height)	
	Estimated coefficient (standard error)					
Exp8	0.042** (0.011)	--	-0.00080** (0.00027)	--	0.00011 (0.00011)	--
Exp10	--	0.054** (0.012)	--	-0.0012** (0.00030)	--	0.00018 (0.00012)
R^2	0.092	0.092	0.0057	0.0057	0.024	0.024

Table notes: Models include fixed effects for birth state and cohort-by-age at enlistment; state linear time trends; pre-adoption state characteristics and trend interactions. Standard errors are corrected for clustering on birth state by year. ** denotes significance at 1% level. Number of observations is 1,704,191 for the education sample and 985,118 for the obesity/height sample.

Table 4. Estimated effects of first exposure to state alcohol prohibition in early childhood and *in utero*

Dependent variable	Estimated coefficient (standard error)		p-value for equivalency of estimated coefficients	p-value for joint significance of estimated coefficients
	Child_exp	Full_exp		
education	0.089* (0.036)	0.078 (0.053)	0.67	0.011
obese	-0.0017 (0.0011)	-0.0014 (0.0015)	0.65	0.15
ln(height)	0.00070* (0.00030)	0.00028 (0.00051)	0.15	0.0018

Table notes: Models include fixed effects for birth state and cohort-by-age at enlistment; pre-adoption state characteristics and trend interactions. Standard errors are corrected for clustering on birth state by year. *denotes significance at 5% level. Number of observations is 1,704,191 for the education sample and 985,118 for the obesity/height sample.

Table 5. Estimated effects of early exposure with the effect of exposure varying with a measure of the demand for alcohol

Dependent variable	Estimated coefficient (standard error)		Estimated effect of early exposure at mean of vote_ratio (standard error)	Sample mean (standard deviation) of Vote_ratio
	Exp8	Exp8* vote_ratio		
education	-0.089** (0.031)	0.0016** (0.00042)	0.035** (0.013)	76.85 (18.84)
obese	-0.00056 (0.00076)	-0.0000039 (0.0000099)	-0.00086** (0.00031)	77.14 (18.51)
ln(height)	-0.0011** (0.00030)	0.000015** (0.0000041)	0.000097 (0.00013)	

Table notes: Models include fixed effects for birth state and cohort-by-age at enlistment; pre-adoption state characteristics and trend interactions. Standard errors are corrected for clustering on birth state by year. ** denotes significance at 1% level. Number of observations is 1,221,807 for the education sample and 717,548 for the obesity/height sample.

Table 6. Estimated effects of exposure with the effect of exposure varying with a dummy variable for outright prohibition

Dependent variable	Estimated coefficient (standard error)		Estimated effect of early exposure under outright prohibition (standard error)
	Exp8	Exp8* prohib	
education	0.028* (0.012)	0.0066** (0.015)	0.094** (0.014)
obese	-0.0010** (0.00027)	0.00011* (0.00044)	0.00009 (0.00046)
ln(height)	0.000014 (0.00012)	-0.00017 (0.00012)	-0.000026 (0.00013)

Table notes: Models include fixed effects for birth state and cohort-by-age at enlistment; pre-adoption state characteristics and trend interactions. Standard errors are corrected for clustering on birth state by year. * denotes significance at 5% level. ** denotes significance at 1% level. Number of observations is 1,704,191 for the education sample and 985,118 for the obesity/height sample.

Figure 1. Adoption years for state-level alcohol prohibition laws

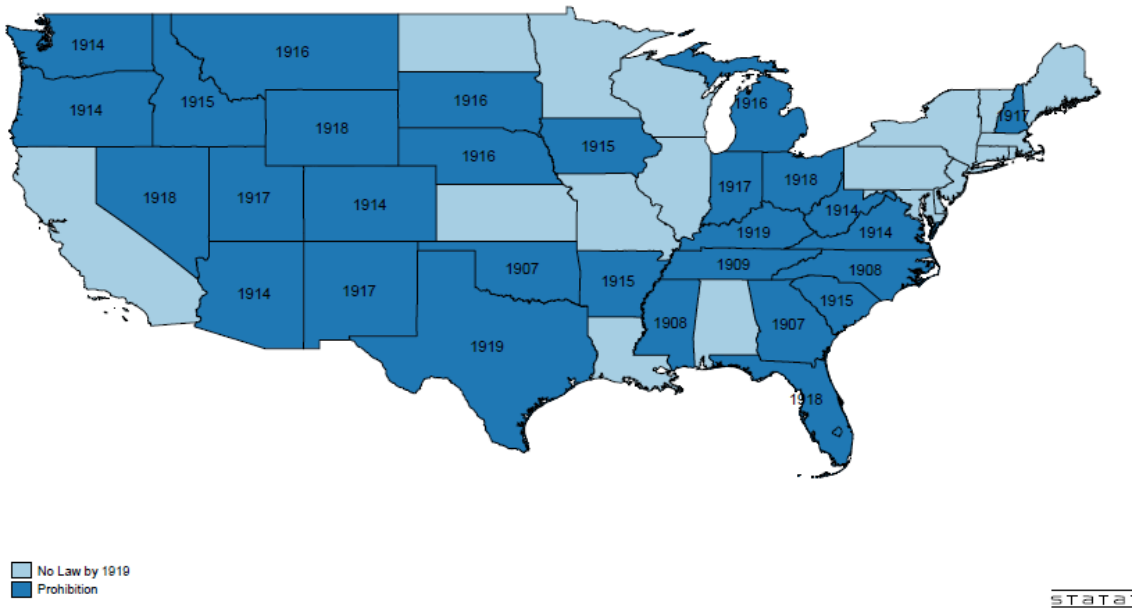


Figure 2. Results of event study for education

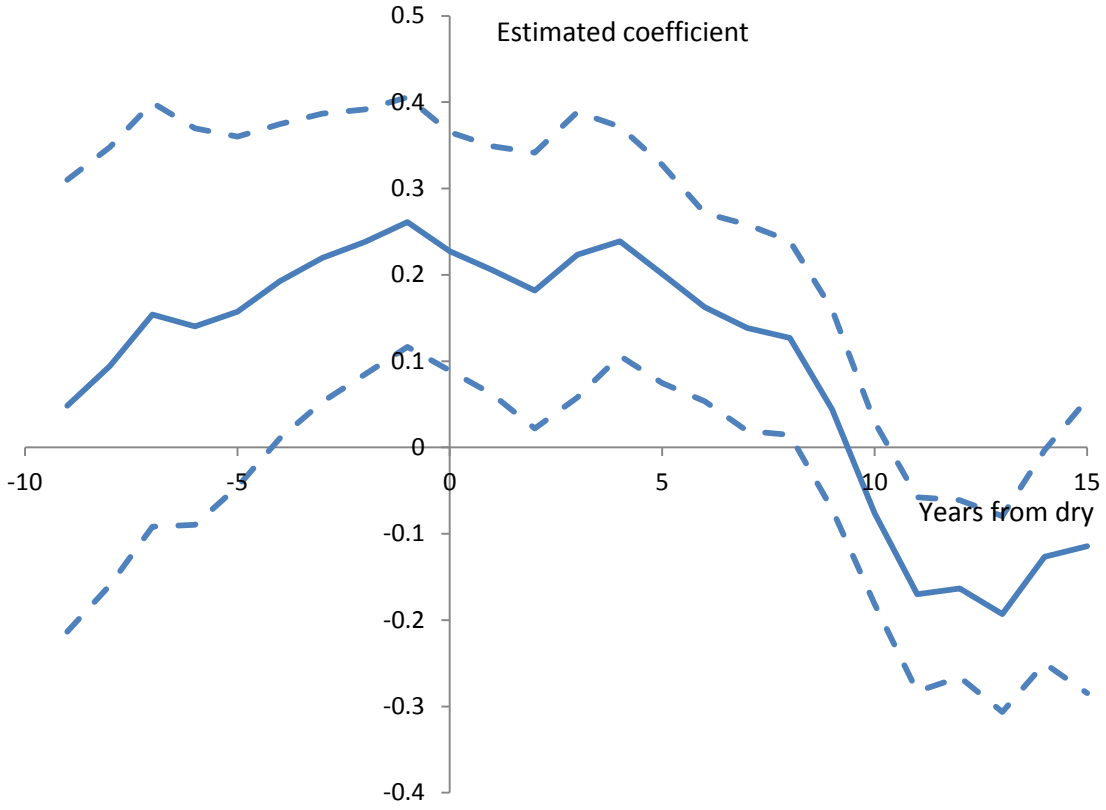


Figure 3. Results of event study for obese

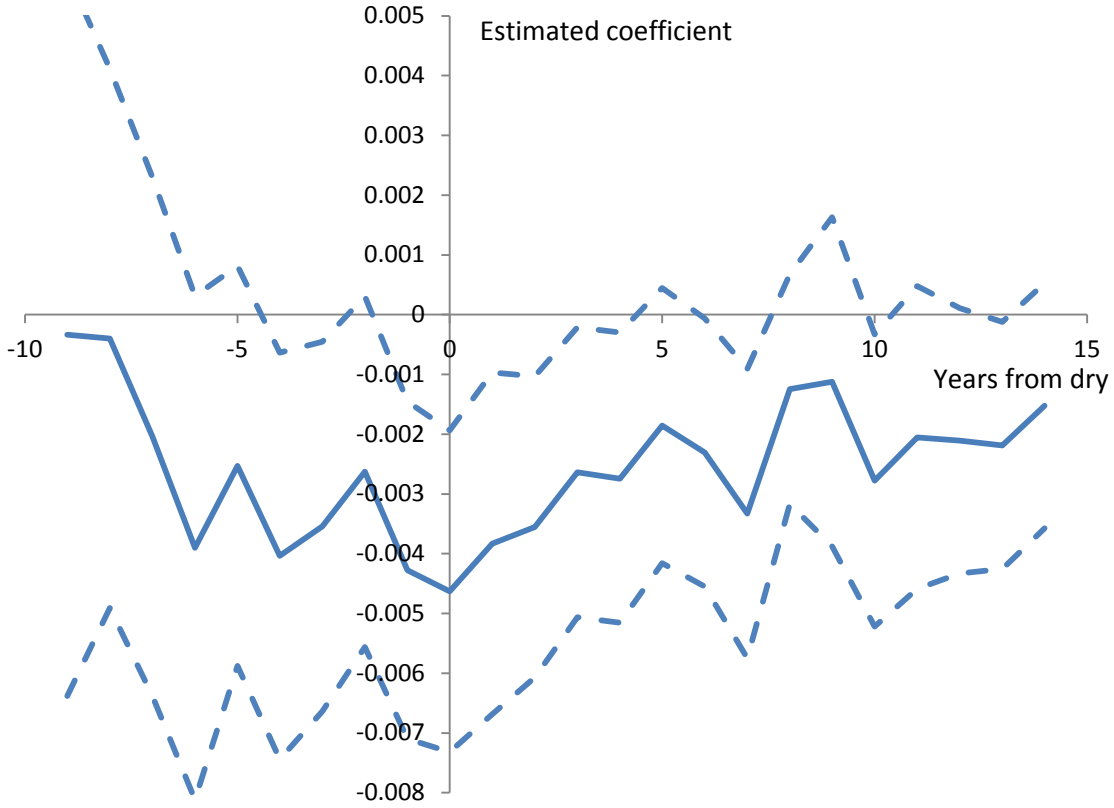
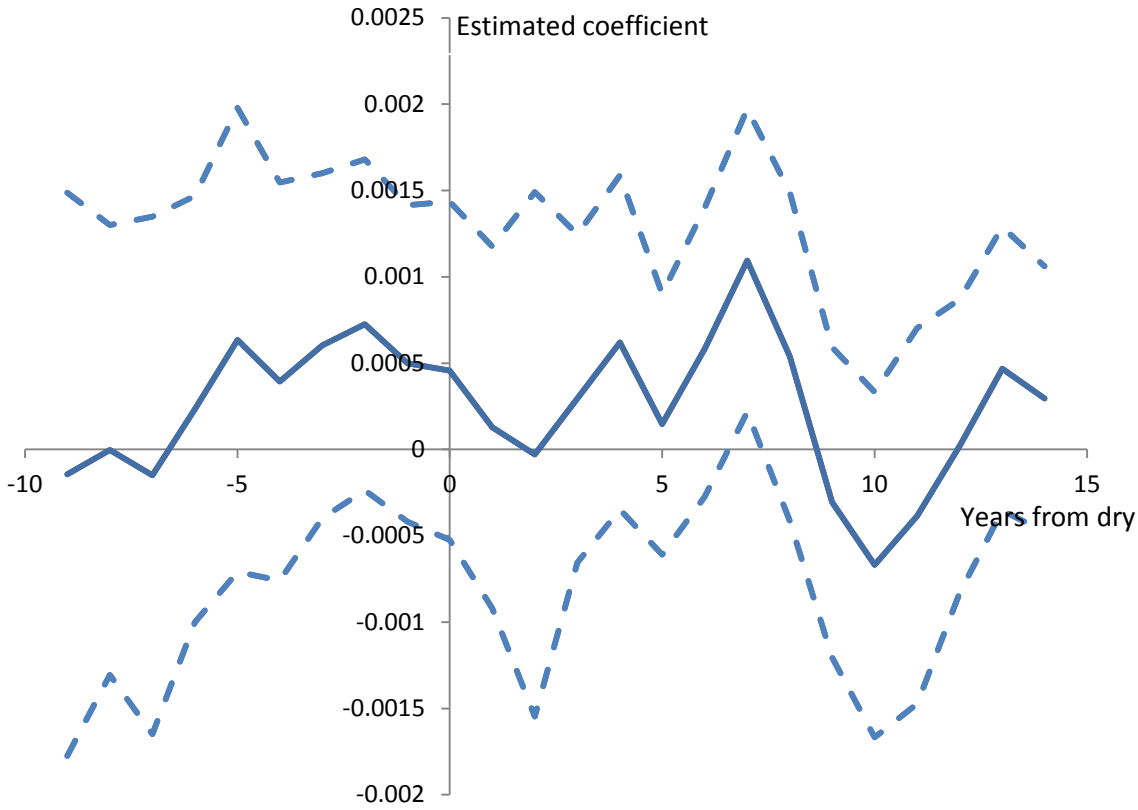


Figure 4. Results of event study for ln(height)



Appendix. Additional robustness tests

Table A1. Estimated effects of early exposure to state alcohol prohibition among all enlistees

Exposure variable	Dependent variable					
	education		obese		ln(height)	
	Estimated coefficient (standard error)					
Exp8	0.024** (0.0094)	--	-0.00074** (0.00023)	--	0.000081 (0.000093)	--
Exp10	--	0.032** (0.010)	--	-0.0011** (0.00025)	--	0.00012 (0.00010)
R^2	0.088	0.088	0.0065	0.0065	0.026	0.026

Table notes: Models include fixed effects for birth state and cohort-by-age at enlistment; pre-adoption state characteristics and trend interactions. Standard errors are corrected for clustering on birth state by year. ** denotes significance at 1% level. Number of observations is 2,255,750 for the education sample and 1,389,781 for the obesity/height sample.

Table A2. Estimated effects of early exposure to state alcohol prohibition—no restriction on years from dry

Exposure variable	Dependent variable					
	education		obese		ln(height)	
	Estimated coefficient (standard error)					
Exp8	0.014 (0.0078)	--	-0.00043† (0.00024)	--	-0.000013 (0.000086)	--
Exp10	--	0.025** (0.0092)	--	-0.00060* (0.00026)	--	0.000029 (0.000091)
R^2	0.091	0.091	0.0062	0.0062	0.025	0.025

Table notes: Models include fixed effects for birth state and cohort-by-age at enlistment; pre-adoption state characteristics and trend interactions. Standard errors are corrected for clustering on birth state by year. ** denotes significance at 1%; * denotes significance at 5% level; † denotes significance at 10% level. Number of observations is 1,756,737 for the education sample and 1,022,815 for the obesity/height sample.