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ABSTRACT

Federal prohibition from 1920 to 1933 was one of the most ambitious policy interventions in US history. However, due to the political concessions necessary to bring about repeal, the removal of restrictions on alcohol after 1933 was not uniform. Using new data on city-level variation in alcohol prohibition from 1933 to 1936, we investigate whether the repeal of federal prohibition affected multiple causes of urban (non-infant) mortality. We find that city-level repeal is associated with a 14.7% decrease in homicide rates and a 10.1% decrease in mortality rates associated with other accidents (including accidental poisonings). Thus, the repeal of federal prohibition could have led to an annual reduction of as many as 3,400 urban deaths. Combined with previous results showing large increases in infant mortality, this suggests that nonetheless repeal most likely had negative effects on all-cause mortality and, thereby, public health in the US.

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A data appendix is available at http://www.nber.org/data-appendix/w28181

1. Introduction

2020 marks the centenary of the federal prohibition on the production, sale, and transportation of alcohol in the United States. In the years since, there has been broad interest in understanding why the prohibition movement spread in the years before 1920, how federal prohibition was enforced in the years after 1920, and why it was so quickly repealed in 1933. However, social scientists should be interested in this peculiar episode for more than antiquarian interests. Federal prohibition represents an immense and unprecedented intervention on the economic and social fabric of the United States which could potentially inform policy making in the present, particularly as it relates to the potential legalization of now-illicit substances like cannabis.

And while the federal prohibition period immediately conjures up images of gangsters, jazz, and speakeasies in the popular imagination, there is little-to-no consensus among social scientists on what prohibition did or did not achieve. The reason for this uncertainty is relatively straightforward: there is simply very little research in assessing the outcomes of federal prohibition in the United States. On the one hand, this state of affairs reflects a misunderstanding of the nature of federal prohibition. It was not in fact a uniform policy change with national restrictions on alcohol "turning on" precisely in 1920 and "turning off" precisely in 1933. On the other hand, this state of affairs reflects more prosaic concerns related to data availability on sub-national variation in restrictions on alcohol sales arising from the decentralized nature of American government.

This paper addresses both of these issues head-on and partially fills the gap in our understanding by assessing the short-run effects of federal prohibition's repeal on multiple causes of urban mortality. While the literature has long stressed a possible link among these variables (e.g., Fisher, 1927), it has been silent on the issue of quantification. In considering the effects of the repeal of federal prohibition, we use new data on annual city-level variation in alcohol prohibition and mortality from 1933 to 1936. Thus, we exploit ample geographic and temporal heterogeneity in restrictions on alcohol sales after federal prohibition, allowing

for potential policy externalities in which the prohibition status of US counties may affect neighboring cities.

We find evidence that relaxing restrictions on alcohol sales (that is, transitioning from so-called "dry" to "wet" status) at the city level is associated with a 14.7% decrease in homicide rates and a 10.1% decrease in mortality rates associated with other accidents (importantly including accidental poisonings). In understanding these results, there are various priors which may run counter to the idea that the repeal of prohibition would be associated with reductions in both homicides and other accidents. For instance, one reasonable prior is that repeal would be associated with an increase in other accidents due to drunken misadventure or mishaps. But another equally reasonable prior is that repeal would also be associated with a decrease in accidental poisonings due to renewed access to legal supplies of unadulterated alcohol. Thus, it is fundamentally an empirical question as to which direction such countervailing forces work themselves out. When we combine these estimates with the timing of these transitions by cumulating homicides and other accidents by wet status, a back-of-the-envelope calculation suggests an annual reduction of 3,418 urban deaths (565 fewer homicides plus 2,853 fewer other accidents) that could be attributed to the repeal of federal prohibition.

We argue for the plausible exogeneity of these transitions to wet status in three ways. First, if potential endogeneity is driven by time-invariant alcohol preferences, then fixed-effects estimation in a short-panel context will yield unbiased estimates of becoming wet. Second, in the vast majority of cases, cities are a part of counties, and the vast majority of changes in prohibition status were affected at the county level. Thus, changes in prohibition status at the city level could plausibly be more exogenous than changes in prohibition status at the county level. Third, we have strong evidence that the cities that opted for wet status through local option have similar outcomes as those that went wet through statewide legislation. To the extent that the timing of transition to wet status for the latter set of cities is more exogenous than for the former set of cities, this result suggests that our identification

strategy may be sufficient in dealing with the endogeneity of the timing of changes in prohibition status.

This paper is very closely related to previous work on infant mortality at the county level (Jacks, Pendakur, and Shigeoka, 2017). There, it is found that counties which chose wet status via either local option elections or state-wide legislation saw infant mortality increase by 2.40 additional infant deaths per 1000 live births. Allowing for potential policy externalities from neighboring counties also turns out to be very important in the case of infant mortality: dry counties with wet neighbors saw their baseline infant mortality increase by 2.82 additional infant deaths per 1000 live births. Putting these estimates together with information on the count of live births by the observed prohibition status of counties, 4,493 annual excess infant deaths are attributed to the repeal of federal prohibition. Thus, it is possible to draw immediate comparisons to the estimates derived in this paper wherein repeal lead to an annual reduction of 3,418 urban deaths. This suggests that, on net, repeal most likely had negative effects on all-cause mortality and, thereby, public health in the US.[1]

This paper is also related to previous work on the mortality effects of alcohol control prior to federal prohibition. Law and Marks (2020) study the state-level mortality effects associated with laws on alcohol control prior to 1920. Importantly, they refine their measure of state-level prohibition to explicitly capture the often significant lags in between changes in legislation and when they become effective.[2] Owens (2011, 2014) likewise explores state

[1] Furthermore, from the perspective of assigning the value of a statistical life, any consideration of the respective rates of mortality should put more weight on averting infant – as opposed to adult – deaths.
[2] Another refinement in their measure of exposure is to incorporate the share of a state's population which resided in dry counties prior to federal or state prohibition using data from Sechrist (1983) available on the ICPSR website. In theory, this is a highly sensible approach. In practice, it may be more problematic than first appears. Upon reading contemporary sources and closely inspecting the data for the 1880s and 1890s, we have found substantial errors in the Sechrist data. For example, the entire state of Ohio is coded as dry after it passes a constitutional prohibition in 1883. However, this referenda was deemed invalid by the courts and, thereby, never went into effect. Similar problems were revealed in a handful of other examined states with approximately 75% of the cells reported in Sechrist (1983) being coded incorrectly. Thus, the prohibition status of counties reported there should be taken with abundant caution and verified before use.

level variation to respectively track the effects of both state and federal prohibitions on homicide rates and on the age distribution of homicide victims. In short, she finds that while prohibition did not decisively increase homicide rates it did serve to compress the age distribution of homicide victims, a finding which is consistent with increases in violence observed in contemporary illicit markets.

Finally, this paper speaks to a literature dating from at least Gordon (1953) that tries to locate the sources of the stunning declines in US urban mortality rates from 1900. Famously, Cutler and Miller (2005) revisited this debate, strongly arguing for the primacy of clean water technologies in the form of chlorination and filtration. They find that these interventions were responsible for roughly 50% of the total mortality reduction in major cities with even higher reduction for child and infant mortality, culminating in a stunning social rate of return to these technologies in excess of 2200%. More recently, Anderson, Charles, and Rees (2018) have strongly challenged this received wisdom. On the basis of corrected data on outcomes and new data on other interventions, they are unable to recover the bulk of Cutler and Miller's results, finding a much more limited effect of water filtration in reducing only infant mortality (roughly, -11%) and no role for any of the other interventions considered. Although the present paper does not speak directly to these issues, it considers a further policy innovation – that is, prohibition's repeal – which potentially amplified the general downward trend in urban mortality rates.

The rest of the paper proceeds as follows. Section 2 lays out the historical context related to the emergence of federal prohibition and its eventual repeal. Section 3 introduces the underlying data while Section 4 introduces our empirical model. Section 5 presents our results on urban mortality and considers various samples of the data and specifications of the model. Section 6 concludes by considering the implications of our study in relation to previous work.

2. Context

Coming on the heels of both a long-standing temperance movement and the American entry into World War I on April 6, 1917, the Senate proposed a constitutional amendment to establish a federal prohibition on alcohol on December 18 of that year. Agitation for federal prohibition was motivated by a remarkably wide range of reasons – e.g., patriotism, progressivism, religion, and women's rights – and, thus, appealed to a remarkably wide range of the public (Rorabaugh, 2018). With the approval of 36 states by January 16, 1919, the 18th amendment was thereby ratified with the country becoming dry on January 17, 1920. Over the next three years, fully 46 of the then 48 states eventually ratified the amendment with only Connecticut and Rhode Island as hold outs.

Passage of the 18th Amendment entailed a near-complete prohibition on the production, sale, and transportation of alcohol. Significantly, federal prohibition did not ban individual consumption and possession of alcohol. It even made allowances for individual production along with exemptions on commercial production and sales for medicinal and religious purposes. To be clear though, these later sources of legal alcohol production could only have been a miniscule fraction of the output of the formerly dominant brewing and distilling industries. In the early 1900s, brewing alone was the fifth largest manufacturing industry of the US on a value-added basis, annually producing nearly 19 gallons of beer for every American (Hernandez, 2016).

Instead, individual consumption and possession was subject to varying degrees of restriction at the city, county, and state levels of government. And while this did not entail the complete unavailability of alcohol – as there were wide differences in enforcement and legislation along these lines – prohibition is best thought as having substantially increased the price of alcohol. Lower bound estimates of this effect suggest that prices were at least five times higher during federal prohibition (Cook, 2007). From the perspective of the present day where impressions of federal prohibition's ineffectual nature abound, surprisingly large effects on quantities were also observed as seen in Figure 1. In 1934, the first year of repeal, apparent per capita alcohol consumption was 37% of its pre-prohibition peak in 1910. From

there, drinking activity somewhat recovered: the relevant figure for apparent per capita alcohol consumption stood at 58% in 1939. Yet the shock of prohibition presumably lingered in the consumption habits of affected Americans throughout their lifetimes as it took until the 1970s for per-capita alcohol consumption to surpass the 1910 peak.

Initial wide-spread support for federal prohibition was eroded throughout the 1920s in the wake of concerns over the new reach of the federal government and doubts related to its efficacy as well as perceptions of rising criminal activity (Okrent, 2010; Garcia-Jimeno, 2016). Punctuating this increasing disillusionment with the national experiment of prohibition was the Great Depression. Faced with a radical decline in commercial activity and concomitant decline in revenue, governments at all levels were exposed to dire fiscal straits. And these straits, in turn, made a return to the pre-prohibition state of the world an appealing prospect on many levels. For one, prior to 1920, roughly 15% of all government revenues came from alcohol taxes (Blocker, 2006) with the federal government collecting fully 35% of its revenue from brewing and distilling in 1914 (Rorabaugh, 2018). Thus, starved of other sources of funding, various levels of government increasingly viewed the sale of alcohol as a potential source of revenue. What is more, the potential repeal of federal prohibition and related rise in alcohol revenues was seen to ease growing pressure to raise federal income and inheritance taxes and/or introduce wealth taxes (Kyvig, 2000). Not surprisingly, this move towards higher levels of taxation was naturally and vehemently opposed by the very wealthy, some of whom were repeal's most ardent and financially generous supporters (Dighe, 2010).

The opening salvo in repealing federal prohibition came on March 22, 1933, when Franklin Roosevelt amended the Volstead Act (or more formally, the National Prohibition Act), allowing for the resumption of low-alcohol beer and wine production and sales (Okrent, 2010). From there, political and popular support for prohibition very quickly eroded. In less than a year, the 21st Amendment to the US Constitution was ratified by special state conventions in 38 states. Of the remaining ten, only South Carolina rejected the amendment altogether whereas North Carolina rejected holding a convention and eight other states failed to propose holding state conventions (Georgia, Kansas, Louisiana, Mississippi,

Nebraska, North Dakota, Oklahoma, and South Dakota). Regardless, on December 5, 1933, the 18th Amendment was duly repealed and federal prohibition came to an end.

Of course, many vexing legal issues remained. For one, most states then reverted to the status quo established by state-level legislation that pre-dated federal prohibition. Thus, many states that found themselves desirous of change in their respective prohibition status had to wait for the arrival and passage of enabling legislation. However, the most contentious issues were related to heterogenous legislation and preferences for alcohol both across and within states that were often in close proximity to one another.

Given the decentralized nature of American government and the existence of continued support for prohibition, there were a number of important concessions in the 21st Amendment needed to shore up support in the various state conventions. One of these relates to potential restrictions on interstate commerce and has been the bone of contention in a number of Supreme Court cases through the years. Namely, imports and transportation of alcohol into states which ratified or retained laws prohibiting alcohol consumption and importation were banned. Thus, there seems to have been at least some acknowledgement of the potential policy externalities arising from repeal and the need to mitigate the same.

Another issue relates to accounting for heterogenous preferences for alcohol. The chief compromise for achieving ratification of the 21st Amendment was in allowing for local option elections to determine liquor laws deemed appropriate for local conditions (Kyvig, 2000). These elections have a long standing in American history and give the electorate the right to vote on liquor control by referendum. That is, local (majority) preferences determine whether a county or municipality prohibits the sale of alcohol. At the same time, many states opted out from local option elections entirely while others allowed for referenda to be held at the state-, county-, city-, or even ward-level. Most importantly then, this compromise ensured that the process of repeal was decidedly not uniform, affording us an important source of variation in prohibition status which we exploit below.

In light of this feature, the transition away from prohibition was nonetheless very rapid: by 1935, 40 states (83%), 2,120 counties (68%), and 835 cities (87%) became wet – that is,

allowed for at least some legal alcohol sales – while 8 states (17%), 991 counties (32%), and 128 cities (13%) stayed dry – that is, banned legal alcohol sales. Naturally, we are concerned about factors which potentially drive both changes in prohibition status and potential risk behaviors at the level of individual cities. Yet the vast historical literature on the prohibition and temperance movements provides us with a healthy list of covariates associated with both anti- and pro-repeal sentiment which we can control for in our empirical model and which we discuss below.

Furthermore, we mitigate potential omitted variable bias by only considering specifications with city-level fixed effects. To the extent that local preferences which induce changes in prohibition status are fixed over relatively short periods of time, the inclusion of county fixed effects fully accounts for such preference variation. And to the extent that change in preferences over time is common across cities, the inclusion of year fixed effects fully accounts for such preference variation. Finally, we include the interaction of all county-level time-invariant characteristics with time trends among the regressors. Since these regressors are thought to influence the decision to become wet, their interaction with time trends should pick up a substantial fraction of any city-level, time-varying factors that are correlated with the treatment.

3. Data

Our data are drawn from three main sources: annual, city-level counts of deaths by cause have been extracted from various issues of the *Mortality Statistics of the United States*; annual, indicators of city-level prohibition status have been constructed from contemporary sources; and other county-level covariates are available from the US Census.

3.1 Data: dependent variables

Annual counts of deaths by 25 consistently-defined and -recorded causes for 963 cities with a population of greater than 10,000 were extracted from various issues of the *Mortality Statistics of the United States*. These were then matched with linearly-interpolated

figures for the population of cities drawn from the 1930 and 1940 US Censuses in order to form mortality rates per 1,000 inhabitants. However, given the large number of covariates to be estimated and multiple causes of death to be examined, it may be problematic to begin by considering these causes one-by-one. In particular, we are concerned about multiple hypothesis testing in which the probability of Type I errors (i.e., false positives) increases, often dramatically, with the number of underlying hypotheses (Shaffer, 1995).

Instead, we first aggregate the 25 causes of death into four broader categories:

(i) acute, alcohol-related causes of death (4) – auto accidents, homicide, other accidents, and suicide;

(ii) chronic, alcohol-related causes of death (3) – cirrhosis, heart disease, and nephritis;

(iii) potentially alcohol-related causes of death (6) – cancer, cerebral hemorrhage, cerebrospinal meningitis, influenza/pneumonia, malaria, and tuberculosis;

(iv) non-alcohol-related causes of death (12) – all other causes, appendicitis, diabetes mellitus, diphtheria, hernia/internal obstruction, other puerperal causes, puerperal septicemia, rheumatism/gout, scarlet fever, syphilis, typhoid/paratyphoid, and whooping cough.

Thus, our baseline regressions feature only "all cause", "acute", "related", "potentially related", and "non-related" causes before drilling down to more specific causes.

To give a sense of the trajectory of mortality in general, Figure 2 uses all US cities with a population greater than 10,000 in 1930 (n = 963) and depicts the ratio of total deaths to total population in any given year. Two features dominate. First, urban mortality was on the rise from the time of federal prohibition's repeal, increasing from 11.67 per thousand in 1933 to 12.81 per thousand in 1936 (i.e., +9.8%). This corresponds with the observation that all-cause mortality for the entire United States (inclusive of non-urban areas) declined during the Great Depression from 1929 to 1933, but began to climb during the years of recovery from 1933 to 1936 (Granados and Roux, 2009). Second, even for this appreciable immediate post-repeal climb, there was a tremendous drop in the all-cause urban mortality rate: first,

tentatively from 1936 to 1937, and then, more decidedly from 1937 to 1939. Thus, by 1939, the all-cause urban mortality rate was fully 14% lower than its peak in 1936.

Locating the sources of this drop is relatively straightforward. Thomasson and Treber (2008) were among the first in definitively associating a related but even stronger drop in maternal deaths to the introduction of sulfa and its interaction with other medical interventions from 1937. Jayachandran, Lleras-Muney, and Smith (2010) followed up on this result and documented equivalently large declines in mortality due to pneumonia and scarlet fever which were clearly related to sulfa's introduction. However, what is much less clear are the patterns governing sulfa's diffusion. As these authors emphasize, there appears to be some rough correlation in between sulfa's diffusion and the size of cities or the presence of major research hospitals. Yet nothing more definitive can be said as we lack any systematic evidence on when and where sulfa was introduced.

A few considerations motivate our choice of sample period. First, the choice of a start date in 1933 is predicated by the fact that: (a) this is the last year in which federal prohibition is fully in effect; (b) there is no city-level data on disaggregated causes of death in 1930; and (c) the number of cities drops sharply in 1931 and 1932 when the original source only records mortality for those cities with a population greater than 25,000. The latter gap in the data is particularly unfortunate as it reduces the available set of cities from a gross count of 963 to 360. Second, the choice of an end date in 1936 is predicated by the very large drop in urban mortality following the introduction of sulfa in 1937. The fear here is that by including 1937, 1938, and 1939 we may unwittingly introduce omitted variable bias for the fact that we have no means of controlling for the uneven diffusion of sulfa drugs across cities. Thus, to the extent that prohibition status may be correlated with city-level and potentially time-varying characteristics also governing sulfa's introduction, we are on safer ground by using the

period from 1933 to 1936 in our baseline estimation and reserving the period from 1933 to 1939 for robustness exercises.[3]

In a similar vein, we initially restrict our attention to cities where the population is less than 400,000. This sample restriction is driven by two observations. First, the distribution of city size in the United States at this time was highly concentrated around 40,000 but with a very long rightward tail with only 17 cities registering a population count greater than 400,000. What is more, there are very few dry cities in that very long rightward tail after 1933. Indeed, St. Louis in 1934 with a population of over 800,000 is the only dry-city observation with a population greater than 400,000. In other words, the support restriction for difference-in-difference is violated for the largest cities as they were all wet after 1934. Thus, we initially restrict the sample in order to establish a more valid comparison across dry and wet cities.

Second, in this exercise of identifying the effects of repeal using changes in *de jure* prohibition status, we have to contend with a historical literature which suggests that *de facto* changes in prohibition status may have been far more muted, particularly in large cities (Okrent, 2010; Rorabaugh, 2018). That is, we should be concerned whether cities like Chicago, New York City, and San Francisco were ever really dry at all during the period of federal prohibition.[4] In light of these concerns, we are yet again on safer ground by using the restricted sample of cities where the population is less than 400,000 (n = 946) in our baseline estimation and reserving the full sample of all cities (n = 963) for robustness exercises.

Finally, another attractive feature of the mortality data is that they are further broken down for cities in which the non-white population numbered at least 10,000 and/or

[3] The choice of an end date in either of 1936 or 1939 is also predicated by the fact that the vast majority of changes in prohibition status had occurred by then. We also wish to avoid any confounding effects of the mobilization effort for World War II and so only consider the 1930s.
[4] Indeed, this observation might also explain the wide-spread opinion that federal prohibition was ineffective in changing alcohol consumption, even in the face of high quality data like LaVallee and Yi (2011) which suggests otherwise. Namely, popular impressions of prohibition – both at the time and today – would have been overwhelmingly influenced by accounts, media, and news based in or drawn from America's largest cities.

represented at least 10% of the total population. This then allows us to determine if there was any differential impact of repeal on non-white and white mortality rates.

3.2 Data: treatment variables

Ideally, we would like individual-level information on alcohol consumption or at least equivalent aggregate information for cities. Of course, this type of data is not available before, especially during, or even after prohibition. Another possibility would be to rely on other legal restrictions on alcohol. Yet liquor laws in the United States appear in stunningly diverse forms: among other things, individual cities, counties, and states continue to limit the maximum alcohol content of specific types of beverages sold within their borders, specify whether alcohol can be sold for off- or on-premise consumption for specific types of establishments, and/or place restrictions on the day and time of alcohol sales. At this time, there exists no data that captures all of these features across cities/counties and years.

Instead, we rely on the sharpest distinction in prohibition status possible: dry versus wet. That is, we seek to compare outcomes for those cities for which no sales of alcohol are permitted (dry) to those for which at least some sales are permitted (wet). Also, previous work finds that explicitly recognizing the possibility of policy externalities across administrative borders matters for estimation and interpretation (Jacks, Pendakur, and Shigeoka, 2017). Thus, after the repeal of federal prohibition, it is not only an individual city's choice of prohibition status which may matter but also the prohibition status of its neighbors. In this manner, we distinguish among cities which allow for the sale of alcohol within their borders (that is, wet cities), cities which are dry and more than 30 km from legal sources of alcohol (that is, bone-dry cities), and cities which are dry but within 30 km from legal sources of alcohol (that is, dryish cities).[5]

[5] Below, we used different thresholds for bone-dry/dryish as robustness as the choice of 30 km is admittedly somewhat arbitrary.

Thereby, we assign all dry cities into either the bone dry or dryish categories. To achieve this goal, we build on previous data collection efforts. Jacks, Pendakur, and Shigeoka (2017) reconstructs the prohibition status of all continental US counties for the key post-repeal period from 1934 to 1939. Here, we make manual adjustments to correctly assign dry or wet status to the 946 cities under consideration and then use the distance separating dry cities from wet counties to distinguish between bone-dry and dryish cities (with 30 km, again, as the relevant threshold). Finally, we note that this distinction of bone-dry versus dryish cities likely matters more in principle than in practice as the count of dryish cities is very low throughout (i.e., 48 cities in 1934, 31 cities in 1935, and 32 cities in 1936).

Figure 3 depicts the proportion of all US cities by prohibition status for the longer period from 1930 to 1939. There, we treat all cities as bone dry from 1930 to 1933. By 1939, this proportion had dropped from 100% to 6.2%. Likewise, we observe the proportion of wet cities rising from 0% in 1933 to 90.1% in 1939 and the proportion of dryish cities rising from 0% to only 3.6% in 1939. Figure 4 depicts the spatial distribution of bone dry and dryish cities by year from 1933 to 1936. It makes clear that by 1936 the remaining hold-out states for prohibition were along the central axis of the US (Kansas, North Dakota, and Oklahoma) along with large parts of the Southeast (Alabama, Georgia, Mississippi, and Tennessee). This constellation of dry cities remained relatively stable into 1939: although Alabama and North Dakota jettisoned state-wide prohibitions in 1937 and there were some changes in city-level prohibition status in Georgia, Tennessee, and Texas in later years, the vast majority of changes in prohibition status had occurred by 1935.

Consequently, this provides a further rationalization for limiting our sample: the period from 1933 to 1936 represents the minimal dataset for identifying the effects of repeal. That is, including the years prior to 1933 and after 1936 adds very little by way of variation in our independent variable of interest, namely individual cities' prohibition status. Including those years would also increase the restrictiveness of the parallel trends assumption by forcing it to cover more years. What is more, our short panel approach also circumvents the concerns outlined in Section 3.1 related to the dramatic effects of sulfa's introduction and the

uncertainty over the forces driving its diffusion across cities. It is also beneficial in that we believe that underlying attitudes on and preferences for alcohol availability are unlikely to have changed very much over such a short period of time.

Finally, in all of our specifications, we not only distinguish among bone dry, dryish, and wet cities but also distinguish between initial and subsequent effects. This modelling choice reflects our prior that any effects of repeal will primarily occur after the recorded change in status. This is due to the fact that we only observe prohibition status at an annual frequency and, thus, there is uncertainty about when in a particular year the change in status occurred. Moreover, even in the case where a precise date of status change is known, there are likely to be lagged effects due to discrepancies in the timing in between when changes in legislation occur and when they become effective and in between when changes become effective and when retail outlets for legal alcohol are established.[6]

3.3 Data: additional covariates

To identify the effect of repeal on mortality outcomes, we implement a difference-in-difference estimator. Consequently, we include city and year fixed effects in all specifications. Although not required by the difference-in-difference framework, we also condition on covariates suggested by the historical literature. This literature points to strong preferences for dry status before and during the period of repeal among Baptists/Methodists and the native-born (Kyvig, 2000; Okrent, 2010; Rorabaugh, 2018). To this list, we also have information at the county level on cumulative New Deal spending per capita, the proportion of black people, and the unemployed-to-population ratio. As all these variables are observed in the cross-section and we already include city fixed-effects, we interact these regressors

[6] Of these three elements, we believe that the second one is likely the most important. Law and Marks (2020) report that for the 18 state-level prohibitions in the early 20th century (that is, prior to federal prohibition), two were effective in the same year they were enacted. Ten were effective in the calendar year after they were enacted. And fully six were effective two or more calendar years after they were enacted. We suspect this type of pattern repeats itself at the county level in the immediate post-repeal period.

with linear time trends (Acemoglu, Autor, and Lyle, 2004; Hoynes and Schanzenbach, 2010). Here, the idea is to control for potential differences in trends across cities which may be correlated with their prohibition status. The number of hospital beds per 1,000 inhabitants, the number of medical institutions per 1,000 inhabitants, and retail sales per capita are also available for each county-year. We include the levels of these variables as additional regressors. The inclusion of these county- and time-varying regressors absorbs variation in economic activity and health services, further reducing the scope of omitted variables bias.

Table 1 reports summary statistics for our sample of cities over the years from 1933 to 1936. Specifically, we report the sample means of the five aggregated mortality rates of interest (all cause, acute, related, potentially related, and non-related) along with the sample means of city populations and our county-level control variables. Nearly all of the mortality rates and control variables display significant differences in mean across dry and wet cities, strongly arguing for the inclusion of city-level fixed effects in our specifications. What is more, if potential endogeneity is driven by time-invariant attitudes on and preferences for alcohol availability, then fixed-effects estimation in a short panel-data context will yield unbiased estimates of becoming wet.

Finally, we also lean on the facts that cities – in the vast majority of cases – are a part of counties[7] and that the vast majority of changes in prohibition status were affected at the county level. Thus, changes in prohibition status at the city level could plausibly be more exogenous than changes in prohibition status at the county level. That is, (at least some of) a city's inhabitants could have preferences for remaining dry but find themselves residing in a county with preferences for becoming wet. Thus, such cities in wet counties may be thought of as rough analogs to their dryish counterparts.

[7] The cities of Virginia are a notable exception to this observation. Under its state constitution, all municipalities were incorporated as independent cities following the Civil War and are thereby not part of any county.

4. Empirical Model

Our baseline specification for estimating the effects of prohibition's repeal on urban mortality is the following:

$$Y_{c,t} = \beta_1 dryish_{c,t=0} + \beta_2 dryish_{c,t>0} + \beta_3 wet_{c,t=0} + \beta_4 wet_{c,t>0} + \gamma \cdot controls_{c,t} + v_c + w_t + \Theta_s \cdot trend + \varepsilon_{c,t}$$

where *c* indexes cities and *t* indexes years. That is, we seek to explain variation in city-level mortality rates as a function of:

(i) cities' prohibition status (either dryish or wet with bone dry acting as our control group), allowing for differential effects across years (in the initial year of status change where $t = 0$ versus in all subsequent years where $t > 0$);

(ii) county-level, time-invariant controls interacted with linear time trends (% Baptist/ Methodist, % black, % foreign-born, New Deal spending per capita, and the unemployed-to-population ratio);

(iii) county-level, time-varying controls (hospital beds and medical institutions per 1,000 inhabitants and retail sales per capita);

(iv) city and year fixed effects;

(v) state-specific linear trends.

Previously, we have discussed the rationale for including (i) in Section 3.2 and the rationale for including (ii) and (iii) in Section 3.3. We include city and year fixed effects (iv) to control for all remaining city-level, time-invariant unobservables and all remaining common, time-varying unobservables, respectively. To account for the possibility that urban mortality rates evolved at different rates in states that allowed for prohibition's repeal as compared to states that did not, we include state-specific linear trends (v) as well.

Following much of literature (e.g. Anderson, Charles, and Rees, 2018; Anderson *et al*., 2019), we use the log of city-level mortality rates as our preferred transformation of the

dependent variable. In order to preserve the oftentimes abundant zeros in the data,[8] we add +1 to the count of deaths before first normalizing by city population and then taking the log. Jacks, Pendakur, and Shigeoka (2017) shows that logged mortality rates are suitable dependent variables if the denominator is large enough and all regressions are appropriately weighted. Regardless, Appendix A shows that the results presented below are not dependent on any particular transformation of the dependent variable.

Finally, all regressions are weighted by city population, and all standard errors are clustered on cities to account for within-city serial correlation of arbitrary form.

5. Results

Our results are presented in four parts: first, we consider our baseline results for aggregated causes of death; second, we consider our baseline results for acute causes of death; then, we re-consider our baseline results for both aggregated and acute causes of death, distinguishing between those cities which went wet through state legislation (wet states) and those which went wet through local option (wet cities); and finally, we summarize the results of various robustness exercises.

5.1 Baseline results for aggregated causes of death

Our first step comes in assessing the effects of repeal on the five aggregated mortality rates of interest (all cause, acute, related, potentially related, and non-related). Our baseline specification includes city and year fixed effects, county-level controls interacted with linear trends (if time invariant), other county-level time-varying regressors, and state linear trends. We use cities with populations less than 400,000 and the period from 1933 to 1936. Again, our expectation is that most of the effects of a city being exposed to legal sources of alcohol either through the actions of a neighboring county or state (dryish) or a local repeal of

[8] For instance, in our sample of 3,784 observations on homicides used in Table 3 below, fully 1,443 (or 38%) of them are recorded as zeroes.

prohibition (wet) will occur in the years after the change in status. This is related to uncertainty over the timing of status changes within years, timing discrepancies in between when changes in legislation occur and their effective dates, and timing discrepancies in between effective dates and the establishment of retail outlets for legal alcohol. Accordingly, we focus our attention on the results for *dryish in subsequent years* and *wet in subsequent years*.

Table 2 reports our baseline results for aggregated causes of death for the total population of cities (that is, the non-white and white populations of cities combined). In Columns 1 through 4, we see statistically significant results are only consistently associated with acute, alcohol-related causes of death (auto accidents, homicide, other accidents, and suicide). We also see no statistically significant results attached to either chronic, alcohol-related causes of death (cirrhosis, heart disease, and nephritis) or potentially alcohol-related causes of death (cancer, cerebral hemorrhage, cerebrospinal meningitis, influenza/ pneumonia, malaria, and tuberculosis). Rationalizing the lack of results on related causes of death is straightforward: in a short panel such as ours, it is unreasonable to expect any significant results on such conditions as the effects of alcohol consumption generally take years of steady exposure to reveal themselves.

For non-alcohol-related causes of death (all other causes, appendicitis, diabetes mellitus, diphtheria, hernia/internal obstruction, other puerperal causes, puerperal septicemia, rheumatism/gout, scarlet fever, syphilis, typhoid/paratyphoid, and whooping cough), we see a small effect only for *wet in initial year* which we chalk up to most likely being due to inherent randomness. Underneath this column heading (and all others like it), we have estimated in excess of 1,000 parameters, some of which are bound to register as statistically significant.

Returning to the results in Column 1 of Table 2, these suggest that the repeal of prohibition was associated only with a significant reduction in deaths by acute causes in cities which transitioned from dry to wet status (*wet in subsequent years*). Taken at face value, repeal was then responsible for a roughly 6.6% reduction in acute causes of death in wet

cities. There is also a marginally insignificant reduction in deaths by acute causes in cities which remained dry but which had easy access to legal sources of alcohol (*dryish in subsequent years*). This last non-result may be explained by the relatively small handful of observations (on average, 41 observations across all years) which simultaneously satisfy all the underlying criteria for inclusion in this category (i.e., this must be a dry city within 30 km to legal sources of alcohol in the years after a neighboring county switches to wet status but before 1937). Therefore, we do not emphasize this or other results for *dryish in subsequent years* due to inconsistencies in magnitude and significance throughout.[9]

Finally, as our econometric strategy is analogous to difference-in-difference, the key assumption in estimation is that treated counties would have followed the same time trend as untreated counties had they themselves not been treated. Under this common-trends assumption, the difference in the rates of change between treated and untreated counties equals the true treatment effect. One way to gauge the validity of this assumption is to compare the time trend before any treatments occur (that is, the pre-trend) for counties that are eventually treated with the pre-trend of counties that are never treated. Appendix C considers the pre-trends for acute sources of death, both at the aggregated and dis-aggregated levels. The main caveat to this exercise is that it can only be conducted for the much more limited sample of 360 cities available prior to 1933. However, the results presented there are highly amenable to the interpretation of parallel pre-trends for acute sources of death (as well as homicide and other accidents).

5.2 Baseline results for acute causes of death

Our second step comes in drilling down further into the data to consider the four dis-aggregated acute causes of death at our disposal (automobile accidents, homicide, other

[9] Appendix B also explores the possibility of differential impacts of prohibition's repeal on the basis of race. For better or worse, we find no evidence of systematic differences across black and white urban populations.

accidents, and suicide).[10] Again, our baseline specification includes city and year fixed effects, county-level controls interacted with linear trends (if time invariant), other county-level time-varying regressors, and state linear trends and is limited in its consideration to cities with populations less than 400,000 and to the period from 1933 to 1936. Again, our expectation is that most of the effects of a city being exposed to legal sources of alcohol either through the actions of a neighboring county or state (dryish) or a local repeal of prohibition (wet) will occur in the years after a change in prohibition status. And again, we focus our attention on the results for *dryish in subsequent years* and *wet in subsequent years*.

Table 3 reports the baseline results for acute causes of death for the total population of cities). Column 1 shows that repeal had no discernible impact on automobile accidents as the coefficients are not only statistically insignificant but also fairly small in magnitude. Likewise, Column 4 which reports the results for suicide finds slightly larger magnitudes in coefficient size but none are statistically significant. Instead, our strongest results emerge in Column 2 for homicide and Column 3 for other accidents.

For homicide, the repeal of prohibition was associated with roughly a 14.7% reduction in deaths in cities which transitioned from dry to wet status (*wet in subsequent years*) and roughly a 19.1% reduction in deaths in cities which remained dry but which had ready access to legal sources of alcohol (*dryish in subsequent years*). We do not emphasize the differences in magnitude on *dryish in subsequent years* and *wet in subsequent years* as the coefficients are not different from one another in terms of statistical significance. For other accidents, the repeal of prohibition was associated with roughly a 10.1% reduction in deaths for *wet in*

[10] In Appendix D, we also consider: (i) the three dis-aggregated chronic, alcohol-related causes of death; (ii) the six potentially alcohol-related causes of death; (iii) and the 12 non-alcohol-related causes of death. The results there strongly support those reported in Tables 2. For related causes, there is one statistically significant coefficient across the four parameters of interest and the three dependent variables considered (that is, one out of 12 coefficients of interest). For potentially alcohol-related causes, there are zero statistically significant coefficients out of the 24 coefficients of interest. And for non-alcohol-related causes, there are five statistically significant coefficients out of the 48 coefficients of interest. In sum, six statistically significant – but potentially spurious – coefficients out of 84 coefficients of interest (or 7.14%) is roughly to be expected when using a 5% level of significance.

subsequent years while there is no statistically significant result for *dryish in subsequent years*. This leads us to further down-weight the results on *dryish in subsequent years*, both here and throughout.

In understanding the results on *wet in subsequent years* in particular, there are various priors which may run counter to the idea that the repeal of prohibition would be associated with reductions in the mortality rates for both homicide and other accidents. For instance, one reasonable prior is that repeal would be associated with an increase in homicide due to a commiserate increase in alcohol-related violence. But another equally reasonable prior is that repeal would also be associated with a decrease in homicide due to the legalization of alcohol sales and its reduction in gang enforcement/warfare and related extra-judicial killings (Owens, 2011, 2014).

With respect to deaths by other accidents, one prior is that repeal would be associated with an increase in such deaths due to drunken misadventure or mishaps, but there are also very good reasons for the opposite expectation. Under this heading of other accidents are items as varied as attacks by venomous animals and death by lightning (US Bureau of the Census, 1931). However, for our purposes, there a few key sub-headings which are particularly bearing, namely accidental poisoning, denatured alcohol poisoning, methyl alcohol poisoning, and wood alcohol poisoning among others. That is, another prior is that repeal would be associated with a decrease in such poisonings due to renewed access to legal supplies of unadulterated alcohol. Contemporary accounts emphasize the sometimes severe morbidity and mortality consequences of adulterated alcohol supplies during federal prohibition (Norris, 1928). Likewise, some have claimed that the federally mandated adulteration of industrial alcohol alone led to an estimated 10,000 deaths during federal prohibition (Blum, 2010).

5.3 Results for aggregated and acute causes of death, wet cities versus wet states

The number one threat to these results, of course, relates to the exogeneity of wet status. That is, what is the role of unobservables in driving the estimated effects of wet status?

Previously, we had leaned on the facts that cities — in the vast majority of cases — are a part of counties and that the vast majority of changes in prohibition status were affected at the county level. Thus, changes in prohibition status at the city level could plausibly be more exogenous than changes in prohibition status at the county level. And even though we have included city fixed effects and a battery of county-level controls in all specifications, the possibility remains that other time-varying unobservables are driving both a city's mortality rates and its prohibition status.

To this end, we make a distinction in between those cities which went wet through state legislation (wet states) and those which went wet through local option (wet cities). The reason for doing so is that the former changes in prohibition status are arguably even more exogenous than the latter from the perspective of individual cities. That is, a city's inhabitants could have strong preferences for remaining dry but find themselves residing in a state with strong preferences for becoming wet. Thus, such cities in wet states may be thought of as rough analogs to their dryish counterparts.

Table 4 presents results which separate cities with wet status into two bins, wet cities and wet states, while no changes are made to dryish. Otherwise, it fully replicates the specifications of Table 2. The dependent variables are the four aggregated mortality rates (acute, related, potentially related, and non-related) for the total population of cities, and the controls are city and year fixed effects, county-level controls interacted with linear trends (if time invariant), other county-level time-varying regressors, and state linear trends. At first pass, the same pattern emerges in Table 4 as in Table 2: non-results for related, potentially related, and non-related mortality, but with clear negative results for acute causes of death.

For acute causes in particular, some interesting results emerge for cities which went wet through state legislation. In particular, the coefficient for *wet states in subsequent year*s at -0.136 is large in magnitude and statistically significant. Likewise, for cities which went wet through local option, the coefficient for *wet cities in subsequent years* is a statistically significant -0.053. Thus, these results are economically meaningful and individually statistically significant, but not statistically distinguishable from one another. Thus, cities that

opted for wet status through local option have similar outcomes as those that went wet through statewide legislation. To the extent that the timing of transition to wet status for the latter set of cities is more exogenous than for the former set of cities, this result suggests that our identification strategy may be sufficient to deal with the endogeneity of the timing of changes in prohibition status.

In Table 5, we extend the analysis of acute causes of death by considering the four disaggregated causes contained therein (automobile accidents, homicide, other accidents, and suicide). The results largely conform to those in Table 3 as automobile accidents and suicide are not meaningfully affected while homicide and other accidents evidence clear declines related to repeal. For homicide, the original coefficient for *wet in subsequent years* of -0.147 from Table 3 is matched by respective values of -0.149 and -0.108 for *wet cities in subsequent years* and *wet states in subsequent years*. Furthermore, neither of these values can be statistically distinguished from that in Table 3 or from one another. Equivalent results emerge for other accidents: the original coefficient for *wet in subsequent years* of -0.101 from Table 3 is matched by respective values of -0.076 and -0.245 for *wet cities in subsequent years* and *wet states in subsequent years*. Again, neither of these values can be statistically distinguished from that in Table 3 or from one another. In sum, this exercise partially validates our assumption of exogeneity in the timing of changes in prohibition status.

5.4 Summary of robustness exercises

Appendices E through G carry out various robustness exercises, over and beyond alternative definitions of our dependent variables (which produce qualitatively the same results). In particular, they respectively consider the use of different fixed effects, different thresholds for defining the set of dryish cities, and different samples of cities and years. In the interests of space, we have collated the main results and summarize our findings, leaving the full set of robustness results in the appendices. Tables 6, 7, and 8 respectively consider mortality rates for (aggregated) acute causes of death, homicide, and other accidents for the

baseline specification in Tables 2 and 4 (Column 1 of each table) for a common set of robustness exercises:

(i) the inclusion of state-year fixed effects (Column 2 of each table);

(ii) the use of 10 km and 50 km as the threshold defining dryish status (Columns 3 and 4 of each table); and

(iii) the inclusion of large cities and all years through 1939 (Column 5 of each table).

Table 6 is our first step by considering mortality rates for acute causes of death. For *wet in subsequent years*, there is a very high degree of correspondence in between our benchmark result in Column 1 and those in Columns 3 through 5 as the latter all register as negative and statistically different from zero but not statistically different from one another. In Column 2, the substitution of state-by-year fixed effects for state linear trends yields a negative, but marginally statistically insignificant coefficient. However, it is an open question about how much interpretive weight to place on the non-significance of these results as the category of wet includes both counties which opt for wet status and counties within states which opt for wet status. By including state-year fixed effects, we thereby eliminate all variation coming from wet states. For *dryish in subsequent years*, the results from three of the four robustness specifications (Columns 2 through 4) are now not only negative as before but also now statistically significant. However, the results in Column 5 are much smaller in magnitude and statistically insignificant. This non-result related to expanding the sample to include all cities and all years does not seem to be exclusively driven either by the inclusion of cities with a population of 400,000 or by the inclusion of years after 1936 (see Appendix G). Rather, both are associated with a lack of statistical significance when entered separately.

Table 7 is our next step by considering mortality rates for homicide. For *wet in subsequent years*, two of the coefficients from the robustness exercises are negative and statistically different from zero (Columns 3 and 5). Neither of these values can be statistically distinguished from that in Column 1 or from one another. Of the other two coefficients, that in Column 4 is practically indistinguishable from either Column 1 or 3, its closest comparators: the coefficient is of almost the exact same magnitude and is only marginally

statistically insignificant. Again, the substitution of state-by-year fixed effects for state linear trends yields a negative, but marginally statistically insignificant coefficient. For *dryish in subsequent years*, all of the coefficients are of variable magnitude, and only that for Columns 2 and 4 are statistically significant.

Table 8 is our final step by considering mortality rates for other accidents. For *wet in subsequent years*, three of the coefficients from the robustness exercises are negative and statistically different from zero (Columns 3, 4, and 5). None of these values can be statistically distinguished from that in Column 1 or from one another. The remaining coefficient in Column 2 again relates to the substitution of state-by-year fixed effects for state linear trends which yields a negative, similar-in-magnitude, but statistically insignificant coefficient. For *dryish in subsequent years*, there is exactly one negative and statistically significant coefficient in Column 3 (but again, even the baseline failed to register much of an effect in this case). The remaining coefficients are mostly of the same magnitude while all suffer from a low underlying observation count (and thereby, low precision). Most tellingly, the coefficient in Column 5 is not only statistically insignificant but also anomalously positive.

In sum, we are on safer ground by acknowledging the sensitivity of our results for dryish cities, exercising caution in their interpretation, and instead emphasizing our results for wet cities.

6. Discussion and Conclusion

In considering the effects of the repeal of federal prohibition, we use new data on city-level variation in alcohol prohibition from 1933 to 1936. We find evidence that relaxing restrictions on alcohol sales lead to decreases in deaths by acute causes and, in particular, homicide and other accidents. We find little evidence that policy externalities greatly mattered in this context, likely due to the relatively small number of potentially treated (dryish) cities. Instead, our strongest set of results — both in the estimated magnitude of the effect and in the number of specifications for which it holds — relates to cities transitioning from bone dry to fully wet status. Thus, our benchmark estimates that city-level repeal is

associated with roughly a 14.7% decrease in homicide rates and roughly a 10.1% decrease in mortality rates associated with other accidents (including accidental poisonings).

One way of contextualizing these results would be in terms of a nationwide count of the reduction in homicide and other accidents due to the repeal of federal prohibition. We can provide a rough back-of-the-envelope calculation by extrapolating the estimates from our sample of cities to the national population in the following manner. In all cities with a population greater than 10,000, there were an average of 6,144 deaths attributable to homicide and 32,974 deaths attributable to other accidents on an annual basis for the period from 1933 to 1936. Applying our benchmark estimates of -14.7% and -10.1% yields an annual reduction of 4,233 deaths (903 fewer homicides plus 3,330 fewer other accidents) in the strong counterfactual in which every US city transitioned from dry to wet status from December 1933. In reality, this transition was delayed and incomplete as seen previously. Taking into account the actual timing of these transitions by cumulating homicides and other accidents by wet status yields an annual reduction of 3,418 urban deaths (565 fewer homicides plus 2,853 fewer other accidents).

And how should we contextualize the latter number? In previous work by Jacks, Pendakur, and Shigeoka (2017) on infant mortality at the county level, counties which chose wet status via local option elections or state-wide legislation saw infant mortality increase by 2.40 additional infant deaths per 1000 live births. Allowing for potential policy externalities from neighboring counties also turns out to be very important in the case of infant mortality: dryish status raised baseline infant mortality by 2.82 additional infant deaths per 1000 live births. Putting these estimates together with information on the count of live births by the observed prohibition status of counties, the annual number of excess infant deaths which could potentially be attributed to the repeal of federal prohibition is 4,493.

Thus, for whatever benefits the repeal of federal prohibition conferred in terms of consumer welfare, diminished expenditure on law enforcement, and/or freedom of choice, it also came at the cost of increasing baseline infant mortality in both dryish and wet counties. In the context of this paper then, this increase in baseline infant mortality was not fully offset

by equivalent declines in non-infant urban mortality. Furthermore, from the perspective of assigning the value of a statistical life, any consideration of balancing the respective rates of mortality should put more weight on averting infant – as opposed to adult – deaths. Naturally, there were other associated components of repeal which remain unexplored in this paper and which should be added to any reckoning of prohibition's legacy (such as the potential effects of repeal on non-infant rural mortality). However, our cumulative results suggest that, on net, repeal had potentially negative effects on public health and mortality in the US.

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Figure 1: Apparent per capita alcohol consumption, 1910-2010

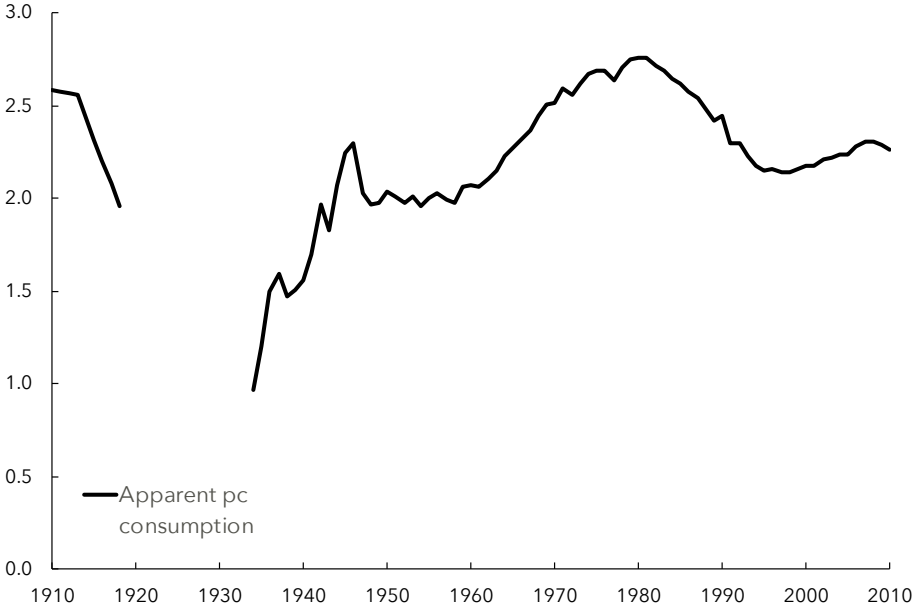

Figure 1 depicts apparent alcohol consumption on a per capita basis which is derived from alcoholic beverage sales data and is measured in gallons of pure ethanol. Source: LaVallee and Yi (2011).

Figure 2: Urban mortality rate, 1933-1939 (deaths per 1,000)

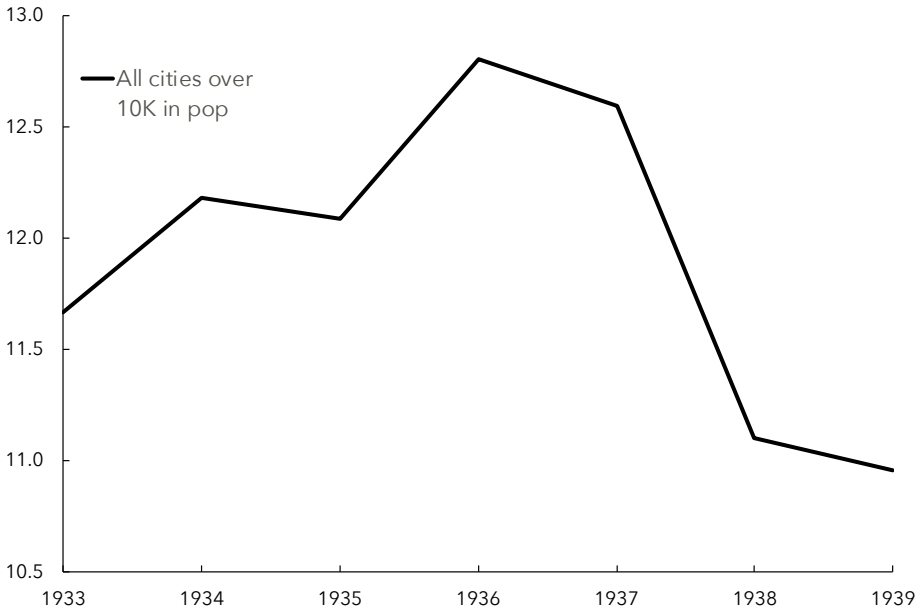

Figure 2 uses all US cities with a population great than 10,000 in 1930 (n = 963) and depicts the ratio of total deaths to urban population by year.

Figure 3: US cities and towns by prohibition status, 1930-1939

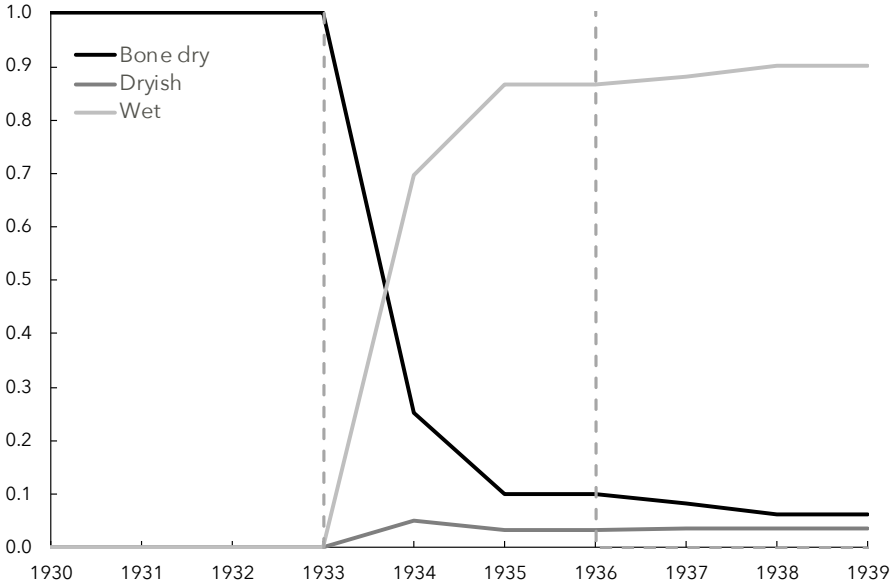

Figure 3 uses all US cities with a population great than 10,000 in 1930 (n = 963). Bone dry cities are dry cities more than 30 km from legal sources of alcohol. Dryish cities are dry cities within 30 km from legal sources of alcohol. Wet cities are those which allow for alcohol sales within their borders. The two vertical dashed lines correspond to the beginning (1933) and end (1936) of our sample period.

Figure 4: Spatial distribution of all dry US cities, 1933-1936

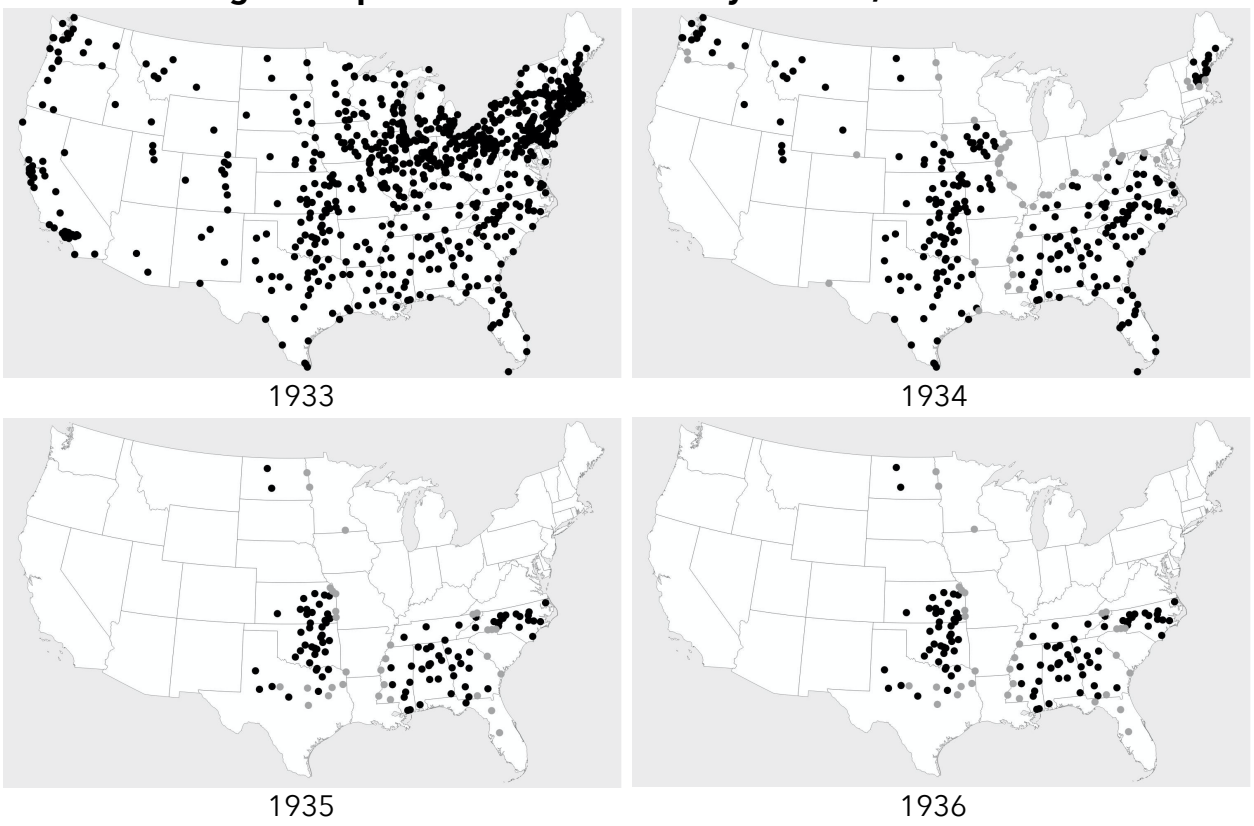

Figure 4 uses all US cities with a population great than 10,000 in 1930 (n = 963). The cities in black and gray are bone dry and dryish cities, respectively. Bone dry cities are dry cities more than 30 km from legal sources of alcohol. Dryish cities are dry cities within 30 km from legal sources of alcohol.

Table 1: Sample city characteristics by prohibition status

	(1) All	(2) Dry	(3) Wet	(4) p-value
All cause mortality rate	12.72	13.31	12.34	0.00
	[5.01]	[4.64]	[5.20]	
Acute mortality rate	1.28	1.39	1.21	0.00
	[0.68]	[0.68]	[0.68]	
Related mortality rate	3.84	3.68	3.94	0.00
	[1.44]	[1.31]	[1.51]	
Potentially related mortality rate	3.83	3.97	3.74	0.00
	[1.61]	[1.47]	[1.69]	
Non-related mortality rate	2.91	3.29	2.66	0.00
	[1.74]	[1.73]	[1.70]	
City population (1000s)	37.79	37.74	37.82	0.97
	[50.58]	[51.89]	[49.72]	
% black	0.10	0.16	0.06	0.00
	[0.18]	[0.24]	[0.12]	
% foreign-born	0.17	0.11	0.21	0.00
	[0.13]	[0.11]	[0.13]	
% Baptist/Methodist	0.16	0.21	0.13	0.00
	[0.15]	[0.18]	[0.11]	
Unemployed-population ratio	0.05	0.05	0.06	0.00
	[0.02]	[0.02]	[0.02]	
New Deal spending per capita	127.11	122.45	130.15	0.00
	[53.35]	[56.10]	[51.87]	
Hospital beds per 1,000	17.47	17.16	17.67	0.34
	[15.60]	[16.55]	[14.95]	
Institutions per 1,000	60.26	64.41	57.56	0.00
	[38.33]	[40.82]	[36.38]	
Retail sales per capita	635.02	544.18	694.22	0.00
	[181.47]	[163.28]	[167.76]	
Number of cities	3,784	1,493	2,291	--

Column (1) reports means across all cities and years while columns (2)–(3) report means for dry and wet cities across years, respectively. Standard errors in brackets. Column (4) reports p-values for the null hypothesis that the means are the same across dry and wet. Reported mortality rates expressed as deaths per 1,000 inhabitants.

Table 2: Repeal's effect on aggregated causes of death

	(1)	(2)	(3)	(4)
	Acute	Related	Potentially related	Non-related
Dryish in initial year	-0.023	0.023	-0.015	0.027
	(0.026)	(0.014)	(0.014)	(0.016)
	[0.90]	[1.66]	[1.06]	[1.67]
Dryish in subsequent years	-0.089	-0.003	-0.039	0.019
	(0.046)	(0.028)	(0.028)	(0.026)
	[1.92]	[0.11]	[1.42]	[0.73]
Wet in initial year	**-0.049**	-0.008	-0.007	**-0.025**
	(0.018)	(0.010)	(0.011)	**(0.011)**
	[2.76]	[0.85]	[0.68]	**[2.26]**
Wet in subsequent years	**-0.066**	-0.016	0.012	-0.000
	(0.025)	(0.017)	(0.017)	(0.020)
	[2.62]	[0.92]	[0.72]	[0.02]
N of observations	3,784	3,784	3,784	3,784
City & year fixed effects	X	X	X	X
County controls with linear trends	X	X	X	X
State linear trends	X	X	X	X

Regression of logged mortality rates, weighted by city population. Standard errors are in parentheses and clustered at the city level; t-statistics reported below standard errors in brackets in brackets. Figures in bold are significant at the 5% level.

Table 3: Repeal's effect on acute causes of death

	(1)	(2)	(3)	(4)
	Automobile accidents	Homicide	Other accidents	Suicide
Dryish in initial year	0.018	-0.014	-0.057	0.045
	(0.041)	(0.058)	(0.042)	(0.060)
	[0.44]	[0.24]	[1.36]	[0.75]
Dryish in subsequent years	-0.051	**-0.191**	-0.076	0.078
	(0.087)	**(0.078)**	(0.065)	(0.070)
	[0.59]	**[2.44]**	[1.16]	[1.11]
Wet in initial year	-0.005	-0.065	**-0.082**	0.027
	(0.027)	(0.042)	**(0.033)**	(0.037)
	[0.17]	[1.55]	**[2.54]**	[0.74]
Wet in subsequent years	-0.050	**-0.147**	**-0.101**	0.075
	(0.044)	**(0.073)**	**(0.041)**	(0.060)
	[1.13]	**[2.03]**	**[2.46]**	[1.24]
N of observations	3,784	3,784	3,784	3,784
City & year fixed effects	X	X	X	X
County controls with linear trends	X	X	X	X
State linear trends	X	X	X	X

Regression of logged mortality rates, weighted by city population. Standard errors are in parentheses and clustered at the city level; t-statistics reported below standard errors in brackets. Figures in bold are significant at the 5% level.

Table 4: Repeal's effect on acute causes of death, wet cities versus wet states

	(1)	(2)	(3)	(4)
	Acute	Related	Potentially related	Non-related
Dryish in initial year	-0.024	0.023	-0.015	0.029
	(0.026)	(0.014)	(0.014)	(0.016)
	[0.93]	[1.64]	[1.05]	[1.80]
Dryish in subsequent years	-0.088	-0.003	-0.039	0.018
	(0.046)	(0.028)	(0.027)	(0.025)
	[1.90]	[0.11]	[1.42]	[0.72]
Wet cities in initial year	**-0.042**	-0.007	-0.009	**-0.033**
	(0.019)	(0.010)	(0.011)	**(0.012)**
	[2.16]	[0.70]	[0.78]	**[2.86]**
Wet cities in subsequent years	**-0.053**	-0.017	0.011	-0.013
	(0.027)	(0.019)	(0.018)	(0.020)
	[1.97]	[0.92]	[0.59]	[0.65]
Wet states in initial year	**-0.090**	-0.015	0.002	0.021
	(0.036)	(0.017)	(0.019)	(0.025)
	[2.47]	[0.88]	[0.12]	[0.84]
Wet states in subsequent years	**-0.136**	-0.011	0.023	0.073
	(0.060)	(0.027)	(0.032)	(0.042)
	[2.26]	[0.39]	[0.70]	[1.73]
N of observations	3,784	3,784	3,784	3,784
City & year fixed effects	X	X	X	X
County controls with linear trends	X	X	X	X
State linear trends	X	X	X	X

Regression of logged mortality rates, weighted by city population. Standard errors are in parentheses and clustered at the city level; t-statistics reported below standard errors in brackets. Figures in bold are significant at the 5% level.

Table 5: Repeal's effect on acute causes of death, wet cities versus wet states

	(1)	(2)	(3)	(4)
	Automobile accidents	Homicide	Other accidents	Suicide
Dryish in initial year	0.017	-0.011	-0.060	0.045
	(0.041)	(0.057)	(0.043)	(0.060)
	[0.40]	[0.20]	[1.40]	[0.76]
Dryish in subsequent years	-0.051	**-0.190**	-0.075	0.079
	(0.087)	**(0.078)**	(0.065)	(0.070)
	[0.59]	**[2.43]**	[1.14]	[1.12]
Wet cities in initial year	0.004	**-0.083**	-0.065	0.024
	(0.029)	**(0.038)**	(0.035)	(0.039)
	[0.14]	**[2.18]**	[1.86]	[0.61]
Wet cities in subsequent years	-0.040	**-0.149**	-0.076	0.077
	(0.048)	**(0.075)**	(0.044)	(0.063)
	[0.84]	**[1.97]**	[1.74]	[1.22]
Wet states in initial year	-0.054	0.039	**-0.179**	0.046
	(0.045)	(0.102)	**(0.060)**	(0.069)
	[1.19]	[0.38]	**[2.97]**	[0.68]
Wet states in subsequent years	-0.110	-0.108	**-0.245**	0.069
	(0.087)	(0.163)	**(0.092)**	(0.123)
	[1.27]	[0.66]	**[2.66]**	[0.56]
N of observations	3,784	3,784	3,784	3,784
City & year fixed effects	X	X	X	X
County controls with linear trends	X	X	X	X
State linear trends	X	X	X	X

Regression of logged mortality rates, weighted by city population. Standard errors are in parentheses and clustered at the city level; t-statistics reported below standard errors in brackets. Figures in bold are significant at the 5% level.

Table 6: Robustness on acute causes of death (aggregated)

	(1)	(2)	(3)	(4)	(5)
	Baseline, acute	State-year fixed effects	Dryish <10 km	Dryish <50 km	All cities, all years
Dryish in initial year	-0.023	-0.027	0.005	-0.029	0.006
	(0.026)	(0.033)	(0.027)	(0.024)	(0.038)
	[0.90]	[0.80]	[0.20]	[1.20]	[0.15]
Dryish in subsequent years	-0.089	**-0.116**	**-0.100**	**-0.084**	-0.021
	(0.046)	**(0.053)**	**(0.047)**	**(0.037)**	(0.034)
	[1.92]	**[2.19]**	**[2.11]**	**[2.23]**	0.61
Wet in initial year	**-0.049**	-0.041	**-0.043**	**-0.051**	**-0.072**
	(0.018)	(0.060)	**(0.018)**	**(0.018)**	**(0.004)**
	[2.76]	[0.69]	**[2.40]**	**[2.85]**	**[4.55]**
Wet in subsequent years	**-0.066**	-0.123	**-0.058**	**-0.068**	**-0.126**
	(0.025)	(0.069)	**(0.025)**	**(0.025)**	**(0.022)**
	[2.62]	[1.78]	**[2.30]**	**[2.70]**	**[5.69]**
N of observations	3,784	3,784	3,784	3,784	6,741
City & year fixed effects	X	X	X	X	X
County controls with linear trends	X	X	X	X	X
State linear trends	X		X	X	X
State-by-year fixed effects		X			

Regression of logged mortality rates, weighted by city population. Standard errors in parentheses and clustered at the city level; t-statistics reported below standard errors in brackets. Figures in bold are significant at the 5% level.

Table 7: Robustness on homicide

	(1)	(2)	(3)	(4)	(5)
	Baseline, homicide	State-year fixed effects	Dryish <10 km	Dryish <50 km	All cities, all years
Dryish in initial year	-0.014	0.006	0.014	0.020	-0.045
	(0.058)	(0.061)	(0.063)	(0.051)	(0.046)
	[0.24]	[0.10]	[0.23]	[0.40]	[0.98]
Dryish in subsequent years	**-0.191**	**-0.205**	-0.105	**-0.141**	-0.054
	(0.078)	**(0.091)**	(0.092)	**(0.068)**	(0.046)
	[2.44]	**[2.26]**	[1.13]	**[2.08]**	[1.16]
Wet in initial year	-0.065	-0.076	-0.059	-0.057	**-0.096**
	(0.042)	(0.152)	(0.041)	(0.042)	**(0.030)**
	[1.55]	[0.50]	[1.42]	[1.34]	**[3.22]**
Wet in subsequent years	**-0.147**	-0.237	**-0.137**	-0.138	**-0.145**
	(0.073)	(0.153)	**(0.072)**	(0.073)	**(0.038)**
	[2.03]	[1.55]	**[1.96]**	[1.88]	**[3.76]**
N of observations	3,784	3,784	3,784	3,784	3,784
City & year fixed effects	X	X	X	X	X
County controls with linear trends	X	X	X	X	X
State linear trends	X		X	X	X
State-by-year fixed effects		X			

Regression of logged mortality rates, weighted by city population. Standard errors in parentheses and clustered at the city level; t-statistics reported below standard errors in brackets. Figures in bold are significant at the 5% level.

Table 8: Robustness on other accidents

	(1)	(2)	(3)	(4)	(5)
	Baseline, accidents	State-year fixed effects	Dryish <10 km	Dryish <50 km	All cities, all years
Dryish in initial year	-0.057	-0.029	-0.032	**-0.086**	0.024
	(0.042)	(0.046)	(0.046)	**(0.038)**	(0.073)
	[1.36]	[0.62]	[0.69]	**[2.24]**	[0.33]
Dryish in subsequent years	-0.076	-0.097	**-0.122**	-0.096	0.014
	(0.065)	(0.069)	**(0.059)**	(0.060)	(0.053)
	[1.16]	[1.41]	**[2.05]**	[1.59]	[0.27]
Wet in initial year	**-0.082**	-0.066	**-0.075**	**-0.091**	**-0.075**
	(0.033)	(0.102)	**(0.032)**	**(0.033)**	**(0.024)**
	[2.54]	[0.65]	**[2.33]**	**[2.75]**	**[3.13]**
Wet in subsequent years	**-0.101**	-0.161	**-0.092**	**-0.112**	**-0.110**
	(0.041)	(0.107)	**(0.041)**	**(0.042)**	**(0.026)**
	[2.46]	[1.50]	**[2.27]**	**[2.68]**	**[4.24]**
N of observations	3,784	3,784	3,784	3,784	6,741
City & year fixed effects	X	X	X	X	X
County controls with linear trends	X	X	X	X	X
State linear trends	X		X	X	X
State-by-year fixed effects		X			

Regression of logged mortality rates, weighted by city population. Standard errors in parentheses and clustered at the city level; t-statistics reported below standard errors in brackets. Figures in bold are significant at the 5% level.