Explorations in Economic History 89 (2023) 101529

Contents lists available at ScienceDirect

Explorations in Economic History

journal homepage: www.elsevier.com/locate/eeh

Urban mortality and the repeal of federal prohibition ☆

David S. Jacks [a,*], Krishna Pendakur [b], Hitoshi Shigeoka [c]

[a] National University of Singapore, CEPR, and NBER, 1 Arts Link, 117568, Singapore
[b] Simon Fraser University, 8888 University Dr W, Burnaby, BC V5A 1S6, Canada
[c] University of Tokyo and NBER, 7 Chome-3-1 Hongo, Bunkyo City, Tokyo 113-0033, Japan

ARTICLE INFO

JEL classification:
H73
I18
J1
N3

Keywords:
Federal prohibition
Repeal
Urban mortality

ABSTRACT

Federal prohibition was one of the most ambitious policy interventions in US history. However, the removal of restrictions on alcohol after 1933 was not uniform. Using a new balanced panel on annual deaths, we find that city-level repeal is associated with a 11.6% decrease in the rate of death by non-automobile accidents, a category which critically include accidental poisonings. We relate this finding to a large literature which emphasizes – but never precisely quantifies – the mortality effects of adulterated alcohol during federal prohibition. Thus, repeal likely led to a large annual reduction in accidental poisonings. However, combined with previous results showing even larger increases in infant mortality, repeal nonetheless likely had negative contemporaneous effects on public health.

1. Introduction

2020 marked 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 academic and popular interest in understanding why the prohibition movement spread in the years before 1920 (Rorabaugh, 2018; Schrad, 2021), how federal prohibition was enforced in the years after 1920 (McGirr, 2016; Okrent, 2010), and why it was so quickly repealed in 1933 (Kyvig, 2000; Rose, 1996). However, social scientists should be interested in this peculiar episode for more than antiquarian interests. Federal prohibition represents a large and unprecedented intervention on the economic and social fabric of the United States which could potentially inform policy making in the present.

And while federal prohibition 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 achieved. The reason for this ambiguity is straightforward: relative to the scale of the intervention, there is shockingly little research in assessing the outcomes of federal prohibition in the United States.[1] On the one hand, this state of affairs likely reflects a common misunderstanding of the nature of federal prohibition: there was no uniform policy change with restrictions on alcohol "turning on" precisely in 1920 and "turning off" precisely in 1933. On the other hand, this state of affairs definitely 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.

☆ We appreciate feedback from the 2019 Asian and Australasian Society of Labour Economics Conference, the 2021 Australasian Cliometrics Workshop, and the 2021 Canadian Economics Association meetings as well as seminars at National University of Singapore and Wilfred Laurier University. We also gratefully acknowledge research support from Simon Fraser University and the Social Sciences and Humanities Research Council of Canada. Jacks: National University of Singapore, CEPR, and NBER (dsjacks@gmail.com); Pendakur: Simon Fraser University (pendakur@sfu.ca); Shigeoka: University of Tokyo and NBER (hitoshi_shigeoka@sfu.ca).

* Corresponding author.
E-mail address: dsjacks@gmail.com (D.S. Jacks).

[1] Implicitly, we are comparing the handful of papers related to federal prohibition discussed below to the large literature – comprising dozens of papers — on assessing the effects of a broadly contemporaneous policy intervention, namely the New Deal.

https://doi.org/10.1016/j.eeh.2023.101529
Received 29 July 2022; Received in revised form 25 March 2023; Accepted 10 May 2023
Available online 14 May 2023

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 repeal, we use a new balanced panel on annual city-level variation in alcohol prohibition and mortality in the 1930s. Thus, we exploit the ample geographic and temporal heterogeneity in restrictions on alcohol sales which emerged 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 "dry" to "wet" status – at the city level is associated with a 11.6% decrease in the rate of death by non-automobile accidents, a category which critically include 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 such accidents. For instance, one reasonable prior is that repeal would be associated with an increase in non-automobile 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.

In so doing, we first demonstrate that this 11.6% reduction in non-automobile accidents is highly robust to a large number of alternate specifications. Importantly, we also show how our estimates relate to a historical literature which has long emphasized the large morbidity and mortality effects of federal prohibition arising from the consumption of an increasingly toxic supply of alcohol. Once stockpiles of formerly legal alcohol were drawn down, bootleggers first turned to both improvised and highly impure distilled products as well as methanol (wood alcohol) as substitutes for ethanol (Asbury, 1950). They also quickly recognized the potential of scale offered by the redistillation of industrial alcohol (Behr, 1996). Federal authorities responded in kind in the mid-1920s by mandating that industrial alcohol be denatured with very high levels of methanol, a virulent poison which is nearly impossible to remove through redistillation. Existing estimates are wanting in precision and unclear in their methods, but do offer a range of 1600 to 11,700 deaths from poisoned alcohol per year (Blum, 2010; Norris, 1928). In contrast, we offer up a fairly precise and transparent estimate that the repeal of federal prohibition was associated with 3277 fewer urban deaths from non-automobile accidents (read accidental poisonings) per year.

We argue for the exogeneity of these transitions to wet status in four 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. Second, cities are a part of counties in nearly all cases, and the vast majority of changes in prohibition status were affected at the county level. Thus, changes in prohibition status at the city level are plausibly more exogenous than changes in prohibition status at the county level. Third, we also present evidence that the cities that opted for wet status through local option have similar outcomes as those that went wet through statewide legislation. Finally, the patchwork regulatory regime that emerged post-repeal as cities, counties, and states alike reverted back to the status quo prevailing in 1919 suggests that the timing of these transitions was a function of idiosyncratic local factors and likely uncorrelated with other potential policy changes. Thus, we are confident that our identification strategy is sufficient to deal with the potential endogeneity of changes in prohibition status.

This paper is very closely related to previous work on infant mortality at the county level (Jacks et al., 2021). 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.[2]

This paper is also related to previous work on the effects of alcohol control prior to the repeal of 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 between changes in legislation and when they become effective.[3] Owens (2011, 2014) likewise explores state-level variation to track the effects of both state (pre-1920) and federal (post-1920) prohibitions on homicide rates and on the age distribution of homicide victims. In short, she finds that prohibition mainly served to compress the age distribution of homicide victims, a finding which is consistent with the increases in violence observed in contemporary illicit markets. Likewise, Garcia-Jimeno (2016) considers endogenous law enforcement effort from 1911 to 1933 and its effects on the national homicide rate which was increasing in this period.

[2] Our primary data source, the *Mortality Statistics of the United States* is very useful for the purposes of assessing the effect of federal prohibition's repeal, but it is also somewhat haphazard in its presentation of the data. Thus, it separately reports:(a) the annual count of infant deaths for all US counties;(b) the annual count of non-infant, urban deaths by cause for all US cities; and(c) the annual count of non-infant, rural deaths by cause for all US states.This results in separate levels of geographic aggregation across the three categories of death and makes a unified assessment of all-cause mortality rates problematic as (a) through (c) present different challenges to estimation due to issues of aggregation and geospatial concerns, e.g., policy spillovers in the case of county-level data as in Jacks, Pendakur, and Shigeoka (2021).

[3] 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 Sechrist (1983). 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 the small number of other states which we closely examined, *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*.

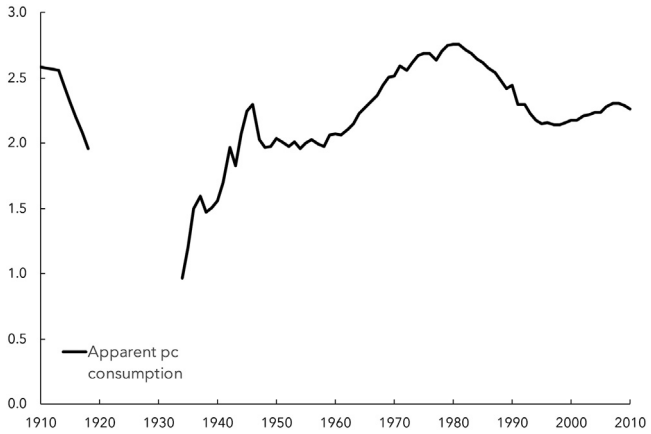

Fig. 1. Apparent per capita alcohol consumption, 1910–2010.
Fig. 1 depicts apparent alcohol consumption on a per capita basis which is derived from alcoholic beverage sales data and is measured in gallons of pure ethanol. Source: LaVallee and Yi (2011).

Finally, this paper speaks to a literature dating from at least Gordon (1953) that seeks to identify the sources of the stunning declines in US urban mortality rates from 1900. 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 reductions for child and infant mortality, translating into a stunning social rate of return to these technologies in excess of 2200%. More recently, Anderson et al. (2022) have strongly challenged these findings. 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, federal 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 and contemporary policy debates.

2. Context

Coming on the heels of both a longstanding temperance movement and the American entry into World War I, the Senate proposed a constitutional amendment to establish a federal prohibition on alcohol in December 1917. Agitation for federal prohibition was motivated by a remarkably wide range of interests — patriotism, progressivism, religion, and women's rights among others — 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. And 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 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).[4]

Instead, individual consumption and possession was subject to varying degrees of restriction at the city, county, and state level. 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 (tax-like) effect suggest that prices were at least five times higher during federal prohibition (Asbury, 1950; 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 Fig. 1. In 1934, the first year of repeal, apparent per capita alcohol consumption was 37% of its pre-prohibition peak in 1910.[5] From there, drinking activity somewhat recovered as the equivalent figure stood at 58% in 1939.

[4] Similarly, distilling was the eighth largest manufacturing industry of the US, according to our calculations.

[5] This figure also likely understates the reduction in consumption due to federal prohibition as one of its unintended consequences was to increase the number of female drinkers, moving drinking away from heavily male-dominated saloons to more mixed evenly mixed clubs, homes,

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 (Asbury, 1950; 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. 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 government revenues was seen to ease growing pressure to raise federal income and inheritance taxes and/or introduce wealth taxes (Kyvig, 2000). Not surprisingly, such a move towards higher levels of taxation was 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 National Prohibition (or Volstead) Act, allowing for resumption of the production and sale of low-alcohol beer and wine (Poelmans et al., 2022). 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 conventions in 38 states.[6] Thus, on December 5, 1933, the 18th Amendment was repealed and federal prohibition came to an end. Of course, many vexing legal issues remained.

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 some continued support for prohibition, a number of important concessions in the 21st Amendment were 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 concession 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 jurisdiction 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.

The transition away from prohibition was nonetheless very rapid. By 1935, 40 states, 2120 counties, and 835 cities became wet — that is, allowed for at least some legal alcohol sales — while 8 states, 991 counties, and 128 cities stayed dry — that is, continued to ban 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.

At the same time, the literature on the patchwork regulatory regime that emerged post-repeal (Childs, 1947; Harrison and Laine, 1936; Pennock and Kerr, 2005) suggests that the timing of city-level transitions was mainly a function of idiosyncratic local factors. That is, they were likely uncorrelated with other potential policy changes. In the main, this situation reflected the dramatic speed with which federal prohibition was repealed and the corresponding legal quagmire that ensued as cities, counties, and states alike reverted back to the *status quo ante* (Clark, 1965; Fosdick and Scott, 1933).

The experience of the two Texarkanas is perhaps instructive in this regard. Prior to federal prohibition, a special election was held in Texas on May 24, 1919. House Joint Resolution 1 proposed to amend the state constitution in order to prohibit "the manufacture, sale, barter or exchange in the state of Texas of spirituous, vinous or malt liquors or medicated bitters capable of producing intoxication, or any intoxicant whatever except for medicinal, mechanical, scientific or sacramental purposes" (Legislative Reference Library of Texas, 1919). This resolution passed with a slim majority (53.3%). Importantly, this statewide prohibition was still in effect when a more decisive majority of Texas voters (61.4%) ratified a referendum in favor of repealing federal prohibition in a special election

and speakeasies (Asbury, 1950; Murdock, 1998; Rose, 1996). At the same time, the first year of repeal corresponds with the first full year of recovery from the Great Depression, so some of this 63% decline might also be attributable to economic conditions and not federal prohibition per se. Cigarettes were another stimulant on which discretionary income could be spent, but for which there was no prohibition: from 1929 to 1933, per-capita cigarette consumption fell by roughly 15% (Warner, 1985). These two factors suggest that federal prohibition's likely effect on individual alcohol consumption was indeed quite large.

[6] 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).

on August 26, 1933.[7] However, it was only on August 24, 1935, that Texas got around to voting in another special election on a resolution to amend the state constitution in order to repeal statewide prohibition. In this instance, only 54.3% of Texans voted in favor of repeal. However, this resolution contained provisions to not only end statewide prohibition, but also authorize the legislature to regulate liquor, prohibit open saloons, and provide for local option elections (Texas Legislative Council, 2022).

In Texarkana, Texas, this meant a reversion to its pre-1919 status as a dry city as its home county of Bowie had voted in favor of local prohibition in 1911. Bowie County was also notable for how evenly divided it was with respect to federal and statewide prohibition: 51% of the county voted in favor of repealing federal prohibition in 1933 while 49% voted in favor of repealing statewide prohibition in 1935 (Endersby, 2012). This created some immediate tensions between the city which favored alcohol sales and the county which favored maintaining its local prohibition (McDermott, 2013). And while low-alcohol beer was allowed to be sold in Texarkana and Bowie County taverns from 1936 and 1950, respectively, unrestricted sales of alcoholic beverages in the whole of Bowie was shot down by a decisive 66% of county voters in 1951. Texarkana would, thus, have to wait until 2006 before mixed beverages could be purchased in restaurants and until 2011 before off-premise consumption of beer and wine was allowed (Texas Alcohol Beverage Commission, various years).

But things were very different just across the state line in its twin city of Texarkana, Arkansas, and its home county of Miller. Like Texas, a decisive majority of Arkansas voters (59.5%) ratified a referendum in favor of repealing federal prohibition in a special election on July 18, 1933 (Johnson, 2005). Unlike Texas, enabling legislation was quickly forthcoming in 1934 which allowed for the sale of low-alcohol beer and wine on the basis of (mandatory) local option elections to be held every two years. This situation radically changed in March 1935 with Act 108 which narrowly passed in both chambers of the legislature. Its quick proposal and passage are explained by pressure from the federal government over New Deal funds which had been misused by the state of Arkansas for ineligible expenditures like schoolteacher salaries (Harper, 2016). Most importantly, this act abrogated the *status quo ante* wherein counties had reverted back to their pre-1920 prohibition status. Instead, all counties of Arkansas were deemed wet by default, and high thresholds for holding now-voluntary, local-option elections were put in place (Johnson, 2005). Consequently, Texarkana, Arkansas and the whole of Miller County went – and stayed – wet overnight.

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 non-infant 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*, resulting in a balanced panel of 3852 city-year observations on the population of non-infant urban death from 1933 to 1936. 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 1000 inhabitants. However, given the large number of covariates to be estimated and multiple causes of death to be examined, it would be problematic to begin our analysis 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 non-infant death into four broader categories based on the classification of deaths given in the

International Statistical Classification of Diseases and Related Health Problems (World Health Organization, 2019):

(i) acute, alcohol-related causes of death ($n = 4$) – auto accidents, homicide, non-automobile accidents, and suicide;
(ii) chronic, alcohol-related causes of death ($n = 3$) – cirrhosis, heart disease, and nephritis;
(iii) potentially alcohol-related causes of death ($n = 6$) – cancer, cerebral hemorrhage, cerebrospinal meningitis, influenza/pneumonia, malaria, and tuberculosis;
(iv) non-alcohol-related causes of death ($n = 12$) – 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 “acute”, “chronic”, “potentially related”, and “non-related” causes before drilling down to more specific causes. Our prior is that repeal should only immediately register in the first category of “acute” as “chronic” causes of death are associated with heavy alcohol consumption over the span of many years. Finally, the third and fourth categories are effectively thought of as a set of placebo tests as the medical literature has established only a weak or no statistical link to alcohol (respectively) for these causes.

To give a sense of the trajectory of mortality in general, Fig. 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

[7] As Endersby (2012) notes, “[this] referendum on the 21st Amendment was actually a choice between two at-large slates of 31 delegates to the state convention for ratification of the proposed repeal amendment.”

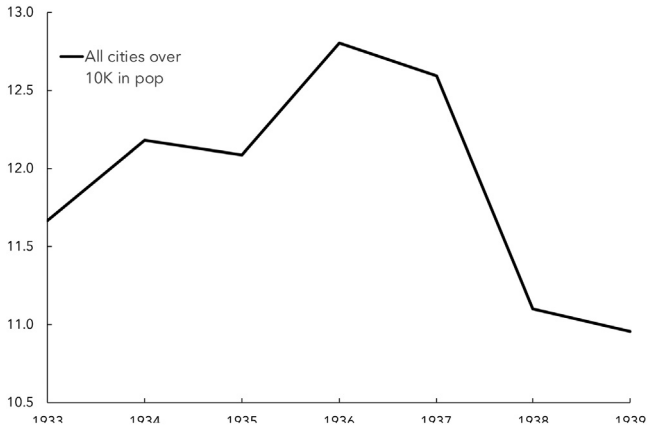

Fig. 2. Urban mortality rate, 1933–1939 (deaths per 1000).
Fig. 2 uses all US cities with a population greater than 10,000 in 1930 (n = 963) and depicts the ratio of total deaths to urban population by year.

was on the rise from the time of federal prohibition's repeal, increasing 9.8% from 11.67 per thousand in 1933 to 12.81 per thousand in 1936. 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 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 et al. (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 between the spread of sulfa 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 and (b) 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 and thereby excludes over 20 million residents of small cities (or roughly one-third of the US urban population). 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. That is, 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.[8] Along these lines, an emergent standard in the literature strongly suggests stopping in 1936 for these exact reasons (Ager et al., 2023; Cutler and Miller, 2005; Feigenbaum and Muller, 2016).

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 nearly zero dry cities in that very long rightward tail after 1933: St. Louis in 1934 is the only dry-city observation with a population greater than 400,000. In other words, the support restriction for difference-in-differences 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 (Garcia-Jimeno, 2016; 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.[9] In light of these concerns, we are yet

[8] 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.

[9] 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.

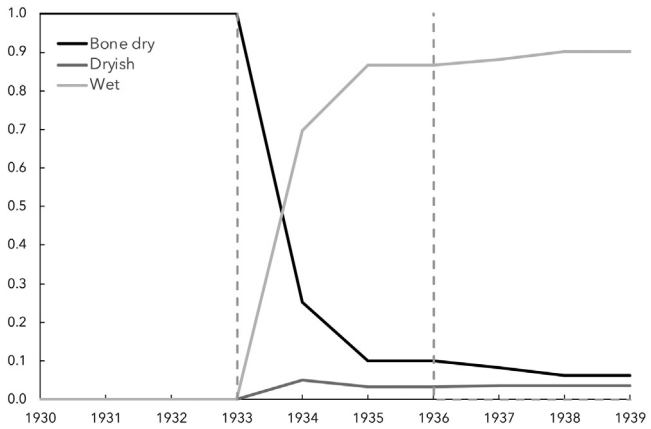

Fig. 3. US cities and towns by prohibition status, 1930–1939.
Fig. 3 uses all US cities with a population greater than 10,000 in 1930 ($n = 963$). Bone dry cities are dry cities more than 30 km from legal sources of alcohol. Dryish cities are dry cities within 30 km from legal sources of alcohol. Wet 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.

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 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 which we explore below.

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 is no source 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 alcohol sales are permitted (dry) to those for which at least some alcohol 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 et al., 2021). 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 away from legal sources of alcohol (that is, bone-dry cities), and cities which are dry but within 30 km of legal sources of alcohol (that is, dryish cities).[10]

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 et al. (2021) reconstructs the prohibition status of all US counties for the key post-repeal period from 1934 to 1939 using an array of sources (Culver and Thomas, 1940; Distilled Spirits Institute, 1935, 1941; Harrison, 1938; Thomas and Culver, 1940). Here, we make manual adjustments to correctly assign dry or wet status to the 963 cities under consideration by consulting editions of the *Annual Report of the Distilled Spirits Institute* (various years) which summarize changes in prohibition status and results of various referenda at a disaggregated level. We then use the distance separating dry cities from wet counties to distinguish between bone-dry and dryish cities. Finally, we note that the distinction between bone-dry and dryish cities likely matters more in principle than in practice as the count of dryish cities is very low throughout (37 cities per year on average).

Fig. 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. Fig. 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

[10] Below, we use different thresholds for bone-dry/dryish as robustness as the choice of 30 km is admittedly somewhat arbitrary. Our results are generally invariant to these changes. What is more, our results are also materially unaffected by the exclusion of the dryish category altogether.

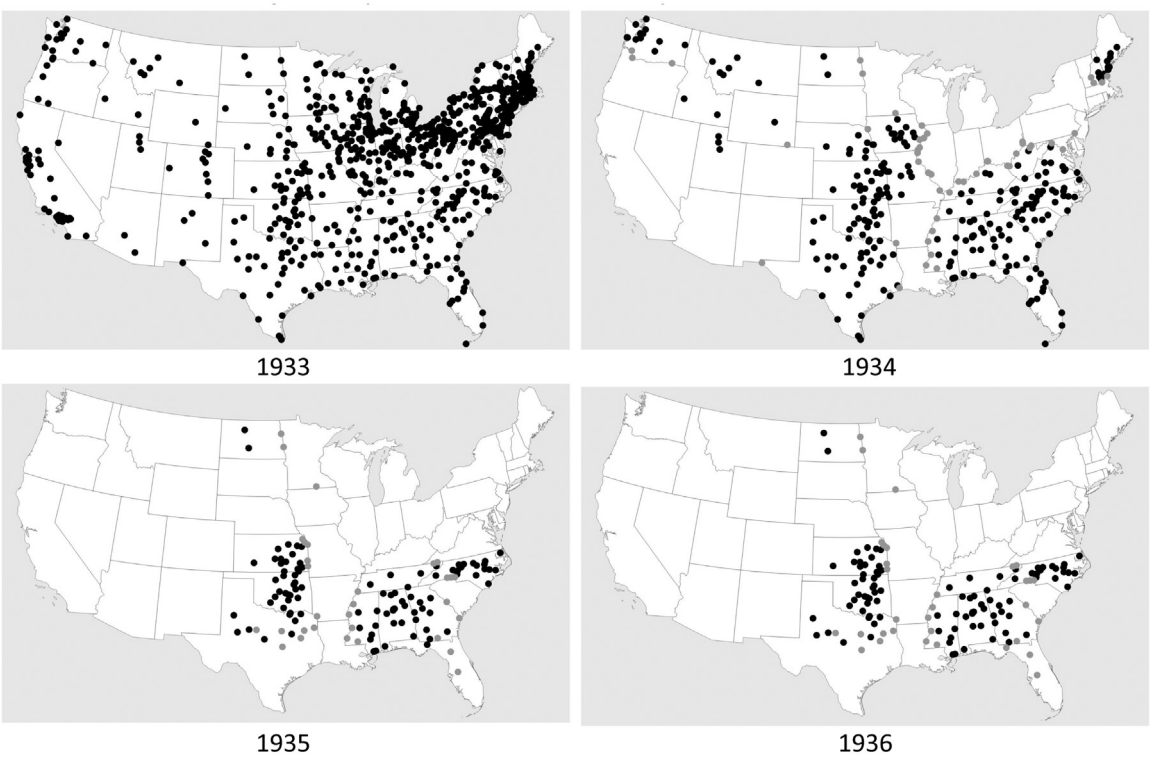

Fig. 4. Spatial distribution of all dry US cities, 1933–1936.
Fig. 4 uses all US cities with a population greater than 10,000 in 1930 (n = 963). The cities in black and gray are bone dry and dryish cities, respectively. Bone dry cities are dry cities more than 30 km from legal sources of alcohol. Dryish cities are dry cities within 30 km from legal sources of alcohol.

the Southeast (Alabama, Georgia, Mississippi, and Tennessee).[11] 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 as seen in Fig. 3.

Consequently, this provides a further rationalization for limiting our sample: the period from 1933 to 1936 represents the minimal dataset for identifying the contemporaneous 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 modeling 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 between when changes in legislation occur and when they become effective and between when changes become effective and when retail outlets for legal alcohol are established.[12]

[11] Of the 41 fully wet states in 1936, only 12 of them, representing 136 cities in our sample, repealed prohibition via state-level legislation. That is, 29 states went fully wet via local option while 699 cities did the same, suggesting that wet status was primarily but not exclusively determined at the local level.

[12] 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 pattern repeats itself at the city level in the immediate post-repeal period.

Table 1
Sample city characteristics by prohibition status.

	(1) All	(2) Dry	(3) Wet	(4) p-value
Acute mortality rate	1.28	1.39	1.21	0.00
	[0.68]	[0.68]	[0.68]	
Chronic 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	0.96
	[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	0.04	0.06	0.00
	[0.02]	[0.02]	[0.02]	
New Deal spending per capita	0.13	0.12	0.13	0.09
	[0.53]	[0.55]	[0.52]	
Hospital beds per 1000	17.47	17.16	17.67	0.70
	[15.60]	[16.55]	[14.95]	
Institutions per 1000	60.26	64.41	57.56	0.03
	[38.33]	[40.82]	[36.38]	
Retail sales per capita	0.64	0.54	0.69	0.00
	[0.18]	[0.16]	[0.17]	
Number of cities	3784	1493	2291	–

Column (1) reports means across all cities and years while columns (2)–(3) report means for dry and wet cities across years, respectively. Standard deviations 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 1000 inhabitants. New Deal spending and retail sales per capita expressed in thousands of USD.

3.3. Data: additional covariates

To identify the effect of repeal on mortality outcomes, we implement a difference-in-differences estimator. Consequently, we include city and year fixed effects in all specifications. Although not required by the difference-in-differences 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 with linear time trends (Acemoglu et al., 2004; Hoynes and Schanzenbach, 2012). 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 county-level, time-varying factors that may be correlated with the treatment.

The number of hospital beds per 1000 inhabitants, the number of medical institutions per 1000 inhabitants, and retail sales per capita are also available for each county-year. We include the levels of these three variables as additional regressors, primarily to address the possibility that new sources of government revenue from alcohol sales may have been directed to medical care at the city level and that repeal might have been most appealing to cities which were the hardest hit by the Great Depression. The inclusion of these county- and time-varying regressors, thus, absorbs variation in economic activity and health services, further reducing the scope of omitted variables bias.[13]

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 four aggregated mortality rates of interest (acute, chronic, 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 specifi-

[13] At the same time, Garcia-Jimenez (2016, p. 530 and Appendix, p. 30) also considers whether fiscal conditions in cities drove their subsequent prohibition status, finding zero correlation between the balance sheet conditions of cities and their pre- and post-Prohibition wet vote shares.

cations. 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.[14]

Finally, we also lean on the facts that cities are a part of counties in nearly all cases 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.

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, s indexes states, 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 1000 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 also mitigate potential omitted variable bias by only considering specifications with city and year fixed effects (iv). To the extent that local preferences which induce changes in prohibition status are fixed over relatively short periods of time, the inclusion of city fixed effects fully accounts for such preference variation. And to the extent that changes in preferences over time is common across cities, the inclusion of year fixed effects fully accounts for such preference variation. Finally, 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.

We estimate our baseline specification using the level of mortality rates and a Poisson pseudo-maximum likelihood estimator. Our reasons for doing so stem from the abundant zeroes in the data. For instance, in our sample of 3784 observations on homicides used in Table 3 below, fully 1443 (or 38%) of them are recorded as zero. In cases like these, Poisson models are preferable to using OLS with a transformed dependent variable like the log of $1 \pm$ the count of deaths (divided by city population) or the inverse hyperbolic sine of mortality rates as the latter can yield incorrect and unstable marginal effects (Manning and Mullahy, 2001; Mullahy and Norton, 2022). In any case, Appendix B shows that our main results presented below are not driven by 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 potential within-city serial correlation of arbitrary form.

5. Results

Our results are presented in five parts: first, we consider our baseline results for aggregated causes of death; second, we consider our baseline results for disaggregated acute causes of death; third, we re-consider our baseline results for both all-acute causes of death and non-automobile accidents, distinguishing between those cities which went wet through state legislation (wet states) and those which went wet through local option (wet cities); fourth, we summarize the results of various robustness exercises on our baseline specification; and finally, we present evidence drawn from a set of event studies using data on a smaller sample of cities but over the longer period from 1928 to 1939.

5.1. Baseline results for aggregated causes of death

Our first step comes in assessing the effects of repeal on the four aggregated mortality rates of interest (acute, chronic, 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 local repeal of prohibition (wet) will occur in the years after the change in status. 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 associated with acute, alcohol-

[14] Appendix A below reports the results from a hazard model on these wet transitions which further motivates our controls and argues for the underlying exogeneity of the timing of the same.

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

	(1) Acute	(2) Chronic	(3) Potentially related	(4) Non-related
Dryish in initial year	−0.023	0.022	−0.008	0.025
	(0.024)	(0.013)	(0.014)	(0.016)
	[0.96]	[1.72]	[0.59]	[1.58]
Dryish in subsequent years	**−0.113**	0.004	−0.037	0.011
	(0.044)	(0.028)	(0.026)	(0.027)
	[2.58]	[0.15]	[1.43]	[0.42]
Wet in initial year	**−0.048**	−0.010	−0.006	−0.020
	(0.017)	(0.010)	(0.011)	(0.011)
	[2.83]	[1.07]	[0.52]	[1.80]
Wet in subsequent years	**−0.072**	−0.019	0.013	0.003
	(0.024)	(0.017)	(0.016)	(0.019)
	[2.99]	[1.18]	[0.76]	[0.15]
N of observations	3784	3784	3784	3784
City & year fixed effects	X	X	X	X
County controls with linear trends	X	X	X	X
State linear trends	X	X	X	X

PPML regression of mortality rates in levels, 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% (or lower) level.

related causes of death (auto accidents, homicide, non-automobile accidents, and suicide). We also see no statistically significant results attached to chronic, alcohol-related causes of death (cirrhosis, heart disease, and nephritis); potentially alcohol-related causes of death (cancer, cerebral hemorrhage, cerebrospinal meningitis, influenza/ pneumonia, malaria, and tuberculosis); or 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). Rationalizing the lack of results on related, but chronic 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 heavy and steady exposure to reveal themselves.

Returning to the results in Column 1 of Table 2, these suggest that the repeal of prohibition was associated 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 7.2% reduction in acute causes of death in wet cities. There is also a significant 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*).

However, these results are only associated with small number of observations (on average, 37 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. What is more, the choice to do so has little bearing on the main conclusions of this paper given the small number of cities involved and, thereby, the small number of deaths potentially affected.[15]

Finally, as our econometric strategy is analogous to difference-in-differences, 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 the parallel-trends assumption, the difference in the rates of change between treated and untreated counties equals the true treatment effect. One way to gage the validity of this assumption is to compare the time trend before any treatments occur (that is, the pre-trend) for cities that are eventually treated with the pre-trend of cities that are never treated. Appendix D considers the pre-trends for acute sources of death, both at the aggregated and disaggregated levels. The main caveat to this exercise is that it can only be conducted for a much more limited sample of 360 cities back to 1928. Bearing this in mind, the results presented there are amenable to the interpretation of parallel pre-trends for aggregated acute sources of death as well as non-automobile 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 disaggregated acute causes of death at our disposal (automobile accidents, homicide, non-automobile accidents, and suicide) by considering the exact same specification as in Table 2. Table 3 reports the baseline results for acute causes of death for the total population of cities. Columns 1 and 2 shows that repeal had no discernible impact on automobile accidents or homicides as the coefficients are generally negative but not statistically significant, apart from that for *dryish in subsequent years* and homicide. Likewise, Column 4 which reports the results for suicide

[15] Appendix B also explores the possibility of differential impacts of prohibition's repeal on the basis of race. We find no evidence of systematic differences across our sample of black and white urban populations. However, we do find evidence of differences across cities with and without significant black populations, suggesting that our headline results may be driven by a relatively small subset of cities in the southern United States.

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

	(1) Automobile accidents	(2) Homicide	(3) Non-auto accidents	(4) Suicide
Dryish in initial year	−0.004	0.016	−0.054	0.024
	(0.043)	(0.053)	(0.044)	(0.063)
	[0.08]	[0.31]	[1.24]	[0.38]
Dryish in subsequent years	−0.115	**−0.234**	−0.099	0.058
	(0.087)	**(0.080)**	(0.059)	(0.075)
	[1.31]	**[2.94]**	[1.69]	[0.77]
Wet in initial year	−0.006	−0.040	**−0.088**	0.022
	(0.027)	(0.047)	**(0.034)**	(0.037)
	[0.22]	[0.85]	**[2.63]**	[0.59]
Wet in subsequent years	−0.059	−0.108	**−0.116**	0.079
	(0.043)	(0.077)	**(0.041)**	(0.061)
	[1.37]	[1.40]	**[2.81]**	[1.29]
N of observations	3784	3784	3784	3784
City & year fixed effects	X	X	X	X
County controls with linear trends	X	X	X	X
State linear trends	X	X	X	X

PPML regression of mortality rates in levels, 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% (or lower) level.

finds positive coefficients but none are statistically significant. Instead, our strongest results emerge in Column 3 for non-automobile accidents.[16]

For non-automobile accidents, the repeal of prohibition was associated with a roughly 11.6% reduction in deaths for *wet in 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.[17] 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 non-automobile accidents. One prior might be 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 non-automobile 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 (see Skorobogatov, 2021 for a contemporary example of these forces at work).

Identifying the exact sources of this estimated reduction in non-automobile accidents is partially clouded by a lack of fine-grained data on mortality in this period. One useful resource in this regard is the Longitudinal, Intergenerational Family Electronic Micro-database (LIFE-M) on causes of death for Ohio in the early 20th century (Bailey et al., 2023). This source reports the cause of death for roughly 185,000 individuals from 1908 to 1953 of which over 10% fall under the heading of "accidents, injuries, and poisoning". Admittedly, Ohio may not be the ideal testing ground in that the entire state went wet in 1934, but by comparing pre-repeal (1931–1933) and post-repeal (1934–1936) sample means, we may make some progress in understanding the mechanisms at work here. First, whereas overall mortality for the US increased between the two periods (Granados and Roux, 2009), overall mortality for Ohio actually fell by 16.7% from 1931–1933 to 1934–1936. With respect to more specific causes, there was a 30% reduction in "other acute poisonings". Unfortunately, the records lack any further granularity for us to investigate.

We can, however, supplement this analysis with an abundant literature which speaks to the potential scale of acute poisonings during federal prohibition. Contemporary accounts emphasize the sometimes severe morbidity and mortality consequences of adulterated alcohol supplies in this period. Drawing from his experience as the chief medical examiner of New York City in the late 1920s,

[16] In Appendix D, we also separately consider: (i) the three 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 Table 2. For chronic causes, there are zero statistically significant coefficients across the four parameters of interest and the three dependent variables considered (that is, zero coefficients for 12 parameters of interest). For potentially alcohol-related causes, there is one statistically significant coefficient for the 24 parameters of interest. And for non-alcohol-related causes, there are three statistically significant coefficients for the 48 parameters of interest. In sum, four statistically significant – but potentially spurious – coefficients for 84 (or 4.76%) parameters of interest is roughly to be expected when using a 5% level of significance.

[17] A natural question related to the results might be how they compare to the estimated (but unreported) coefficients associated with the controls. Across all specifications, the only control which ever matters in a quantitative/statistical sense is retail sales per capita. For acute and non-auto-accident death rates as reported in Tables 2 and 3, there is are significant coefficients for retail sales per capita at +0.42 and +0.49, respectively. Thus, a one-standard-deviation increase in retail sales per capita (=0.18 from Table 1) is associated with an increase of 0.076 and 0.088. These respectively compare with the estimated effects on *wet in subsequent years* of −0.072 and −0.116 reported in Tables 2 and 3.

Norris (1928) begins with an anecdote intended to underline just how common and devastating mass poisoning events were: in just three days of October 1928, 25 individuals in the city died from wood alcohol poisoning. He goes on to claim that such "poison liquor" was responsible for (an oddly precise) 11,700 deaths in the US in 1927, citing the fact that the Police Bureau of Philadelphia had determined that across thousands of samples of prohibition-era liquor fully 95% contained discernible poisons.

One of the more notorious episodes in the history of federal prohibition relates to federal regulations stipulating the use of known poisons like benzine, formaldehyde, gasoline, and methanol (wood alcohol) in order to denature industrial alcohol, a common source of illegal ethanol when redistilled. These regulations originated in the early 1900s when denatured alcohol was deemed exempt from federal excise taxes on potable spirits. But by the mid-1920s, they were deemed insufficient, and federal standards on denaturing industrial alcohol were tightened, now requiring the use of very high levels of methanol, a virulent poison which is nearly impossible to remove through redistillation. A large increase in the number of deaths from poisoned alcohol were forthcoming (Asbury, 1950). Behr (1996) reports that after New Year's Eve celebrations in 1926, 41 people died in one New York City hospital alone while a further 163 people died in Chicago and 307 died in Philadelphia. By some estimates, this federally mandated adulteration of industrial alcohol alone led to at least 1600 deaths per year (Blum, 2010).

Finally, how do our results compare to other estimates in the literature? Fishback et al. (2007) and Fishback et al. (2010) find no significant effects of relief spending on disaggregated causes of death like homicides and (presumably) non-automobile accidents, making comparisons in this regard a little difficult. The former does, however, document that a one standard-deviation increase in New Deal spending was associated with significant declines in infant mortality (of around 5%) and some forms of non-infant mortality, including bronchitis, influenza, and pneumonia as well as suicide (of around 13%). In more recent work, Galofré Vilà (2020) finds that the expansion of conditional cash transfers under the Aid to Dependent Children program of the 1935 Social Security Act reduced infant and non-infant mortality by roughly 10 to 20% while Galofré Vilà, McKee, and Stuckler (2022) find that the simultaneous introduction of the Old Age Assistance program reduced mortality for those over the age of 65 by roughly 30 to 40%. One might argue then that the magnitude of our results are not only reasonable — but also potentially modest — in comparison.

5.3. Results for all-acute causes & non-automobile accidents, wet cities vs wet states

One of the largest threats 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 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 the former category of "wet" into two bins, "wet cities" and "wet states", while no changes are made to dryish. Otherwise, it fully replicates the specifications of Tables 2 and 3. For all-acute causes in Column 1, some interesting results emerge for cities which went wet through state legislation. In particular, the coefficient for *wet states in subsequent years* at −0.131 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 −0.061. Thus, these results are economically meaningful and individually statistically significant, but they are not statistically distinguishable from one another. That is, 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 the 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 potential endogeneity of the timing of changes in prohibition status.

In Column 2, we extend the analysis of acute causes of death by considering non-automobile accidents. The results largely conform to those in Table 3: the original coefficient for *wet in subsequent years* of −0.116 from Table 3 is matched by respective values of −0.100 and −0.223 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 F through H carry out various robustness exercises, over and beyond alternative definitions of our dependent variables as in Appendix B. They consider the use of different fixed effects, different thresholds for defining the set of dryish cities, and different samples of cities and years.[18] 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. The two panels of Table 5 consider mortality rates for all-acute causes of death and

[18] Furthermore, Appendix D of Jacks, Pendakur, and Shigeoka (2021) considers the possibility that individuals may have migrated to counties in response to the respective maintenance or repeal of prohibition at the local level. Analysis of county-level measures of net migration in 1940 finds

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

	(1) All-acute causes	(2) Non-auto accidents
Dryish in initial year	−0.024	−0.057
	(0.024)	(0.044)
	[0.99]	[1.30]
Dryish in subsequent years	**−0.113**	−0.099
	(0.044)	(0.059)
	[2.56]	[1.68]
Wet cities in initial year	**−0.039**	**−0.073**
	(0.018)	**(0.036)**
	[2.12]	**[1.99]**
Wet cities in subsequent years	**−0.061**	**−0.100**
	(0.026)	**(0.044)**
	[2.36]	**[2.25]**
Wet states in initial year	**−0.093**	**−0.177**
	(0.033)	**(0.055)**
	[2.83]	**[3.22]**
Wet states in subsequent years	**−0.131**	**−0.223**
	(0.056)	**(0.087)**
	[2.36]	**[2.57]**
N of observations	3784	3784
City & year fixed effects	X	X
County controls with linear trends	X	X
State linear trends	X	X

PPML regression of mortality rates in levels, 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% (or lower) level.

non-automobile accidents for the baseline specification in Tables 2 and 3 (Columns 1 and 7 of Table 5, respectively) for a common set of robustness exercises:

(i) the inclusion of state-year fixed effects (Columns 2 and 8);
(ii) the use of 10 km and 50 km as the threshold defining dryish (Columns 3–4 and 9–10); and (iii) the inclusion of larger cities and all cities/all years through 1939 (Columns 5–6 and 11–12).

For all-acute causes of death and *wet in subsequent years* (top panel), there is a very high degree of correspondence between our benchmark result in Column 1 and those in Columns 2 through 6 as the latter all register as negative and statistically significant but not statistically different from one another. For non-automobile accidents and *wet in subsequent years* (bottom panel), there is also a high degree of correspondence between our benchmark result in Column 7 and those in Columns 9 through 12 as the latter all register as negative and statistically significant but not statistically different from one another. The remaining coefficient in Column 8 relates to the substitution of state-by-year fixed effects for state linear trends which yields a negative, similar-in-magnitude, but a 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 cities which opt for wet status and cities within states which opt for wet status. By including state-year fixed effects, we thereby eliminate all variation coming from wet states.[19]

5.5. Event studies

Another approach to determining repeal's effects on contemporaneous urban mortality, of course, come from event studies. As it stands, the results reported in the paper represent the best compromise we can come up with as we are confronted with insurmountable limitations in the data and must choose between:

(a) a shorter panel from 1933 to 1936 which is complete across both 25 causes ofdeath and 963 cities which form the full population of US urban areas; and
(b) a longer panel from 1928 to 1939 which is restricted to only the 360 largest UScities with a population greater than 25,000.

no relationship between changes in prohibition status and county-level changes in population. Thus, there is little evidence to the effect that changes in prohibition status drove intercounty – and presumably, intercity – migration patterns in this period.

[19] Appendices I through K report the results of further robustness exercises related to the use of a new DiD estimator, corrections to our reported p-values, and the stability of our results in light of the addition of successive controls. All of these exercises serve to strongly vindicate the baseline results reported here.

Table 5
Robustness on acute causes of death.

	(1) Baseline all-acute causes	(2) State-year fixed effects	(3) Dryish <10 km	(4) Dryish <50 km	(5) Cities >25K	(6) All cities, all years
Dryish in initial year	−0.023	−0.021	0.000	−0.031	0.011	0.006
	(0.024)	(0.029)	(0.026)	(0.022)	(0.029)	(0.033)
	[0.96]	[0.71]	[0.00]	[1.37]	[0.39]	[0.18]
Dryish in subsequent years	**−0.113**	**−0.143**	**−0.129**	**−0.101**	−0.087	−0.041
	(0.044)	**(0.050)**	**(0.045)**	**(0.037)**	(0.055)	(0.030)
	[2.58]	**[2.86]**	**[2.89]**	**[2.70]**	[1.56]	[1.34]
Wet in initial year	**−0.048**	−0.052	**−0.042**	**−0.050**	−0.039	**−0.064**
	(0.017)	(0.062)	**(0.017)**	**(0.017)**	(0.021)	**(0.016)**
	[2.83]	[0.85]	**[2.47]**	**[2.95]**	[1.85]	**[3.93]**
Wet in subsequent years	**−0.072**	**−0.152**	**−0.063**	**−0.075**	**−0.066**	**−0.110**
	(0.024)	**(0.068)**	**(0.024)**	**(0.024)**	**(0.030)**	**(0.024)**
	[2.99]	**[2.22]**	**[2.64]**	**[3.09]**	**[2.23]**	**[4.66]**
	(7) Baseline non-auto accidents	(8) State-year fixed effects	(9) Dryish <10 km	(10) Dryish <50 km	(11) Cities >25K	(12) All cities, all years
Dryish in initial year	−0.054	−0.024	−0.030	**−0.089**	−0.061	0.022
	(0.044)	(0.045)	(0.048)	**(0.040)**	(0.055)	(0.063)
	[1.24]	[0.55]	[0.63]	**[2.23]**	[1.12]	[0.35]
Dryish in subsequent years	−0.099	−0.123	**−0.124**	−0.110	−0.093	−0.011
	(0.059)	(0.064)	**(0.058)**	(0.060)	(0.081)	(0.050)
	[1.69]	[1.92]	**[2.16]**	[1.84]	[1.15]	[0.23]
Wet in initial year	**−0.088**	−0.084	**−0.081**	**−0.099**	−0.078	**−0.069**
	(0.034)	(0.109)	**(0.033)**	**(0.034)**	(0.044)	**(0.026)**
	[2.63]	[0.77]	**[2.43]**	**[2.91]**	[1.79]	**[2.71]**
Wet in subsequent years	**−0.116**	−0.199	**−0.107**	**−0.128**	**−0.105**	**−0.093**
	(0.041)	(0.109)	**(0.041)**	**(0.042)**	**(0.053)**	**(0.030)**
	[2.81]	[1.83]	**[2.61]**	**[3.07]**	**[1.99]**	**[3.16]**
N of observations	3784	3784	3784	3784	1464	6741
City & year fixed effects	X	X	X	X	X	X
County controls with linear trends	X	X	X	X	X	X
State linear trends	X		X	X	X	X

PPML regression of mortality rates in levels, 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% (or lower) level.

For (b), the sample restriction on larger cities excludes over 20 million residents of small cities (that is, roughly one-third of the US urban population) which is far from ideal for purposes of representativeness. We also re-emphasize that any results including data from the years after 1936 should be taken with a large grain of salt in light of the fact that the diffusion of sulfa drugs across cities was almost certainly correlated with cities' transition to wet status as discussed in Section 3.1 and Appendix E.

Here, we consider whether (b) above can corroborate our baseline results. To this end, we present results for event studies on "wet" for the sub-sample of larger cities from 1928 to 1939. Our specification for these event studies is one in which we replace *dryish in initial year* and *dryish in subsequent years* as well as *wet in initial year* and *wet in subsequent years* with dryish and wet indicators respectively which are interacted with a set of indicator variables for each year, ranging from ten years prior to a change in prohibition status to five years after the same change (with $t = 0$ representing the year in which the transition occurred). We also estimate these event studies via PPML and include the same set of controls as before to maintain consistency.

In the main, we find the results reassuring as they confirm the story told in the paper regarding repeal's effect on acute causes of death and non-automobile accidents. At the same time, they somewhat "flip the script" when it comes to deaths due to automobile accidents. Panels A through E of Fig. 5 depict the event studies for the effects of transitions to wet status on all-acute and disaggregated causes of death in the sample of large cities from 1928 to 1939 ($n = 360$). In all of them, the point estimates are plotted as a solid line while the associated 95% confidence intervals are plotted as dotted lines. The point estimates are also normalized to be equal to zero in the year before the transition to wet while the coefficient at $t = 0$ corresponds to "Wet in initial year" in the tables of the main text and the coefficients for $t \geq 0$ corresponds to "Wet in subsequent years" of the same.

Panel A shows no discernible pre-trends for all-acute causes of death in $t \leq 0$. For $t = 0$, the coefficient value is −0.104 which compares to the figure of −0.048 reported for "wet in initial year" in Table 2 of the text. For $t \geq 0$, the average value of the coefficients −0.102 compares to the figure of −0.072 for "wet in subsequent years" in the same. What is more, the event study suggests that this was likely a transitory effect as the coefficient is insignificant by $t = 4$. Along the same lines, Panel D shows no discernible pre-trends

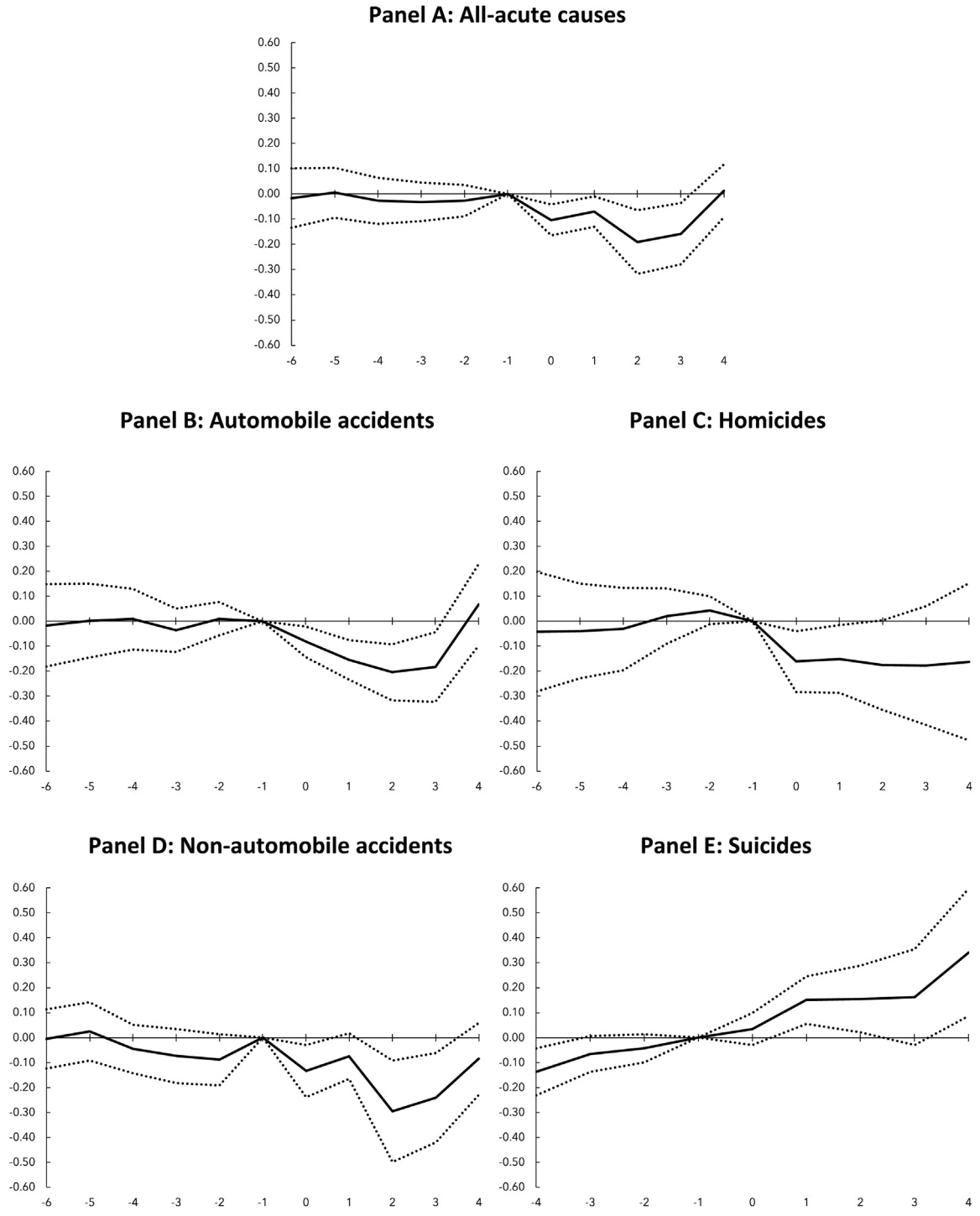

Fig. 5. Event studies for wet on urban mortality, 1928–1939.
Fig. 5 depicts event study analysis for acute sources of death in a sample of large cities from 1928 to 1939 (n = 360). Point estimates are plotted as a solid line while the associated 95% confidence intervals are plotted as dotted lines.

for non-automobile accidents in $t \leq 0$. For $t = 0$, the coefficient value is −0.134 which compares to the figure of −0.088 reported for "wet in initial year" in Table 3 of the text. For $t \geq 0$, the average value of the coefficients −0.174 compares to the figure of −0.116 for "wet in subsequent years" in the same. Once again, the event study hints at transitory effects as the coefficient is insignificant by $t = 4$.

Things are a little more ambiguous in the consideration of automobile accidents, homicide, and suicide. Panels B and C suggest negative, significant, but transitory effects of wet status on automobile accidents and homicide for $t \geq 0$. The results in Panel B, of course, run counter to those in Table 3 above and somewhat counter to expectations since it is hard to understand why greater access to legal sources of alcohol should lead to a decline in automobile accidents. Rationalizing the results in Panel C is more straightforward as the point estimates from the event study point to a roughly 15% drop in homicide in the first two to three years after repeal. Garcia-Jimeno (2016) considers endogenous law enforcement effort in the period from 1911 to 1933 and attributes the same with a 15% increase in the homicide rate, a finding which is remarkably consistent with these estimates. Panel E might suggest an increase in urban mortality coming from a greater number of suicides after repeal, but the presence of a clear pre-trend would strongly argue against this interpretation.

On balance, we find the event studies useful, but find ourselves still leaning towards our baseline results, simply for their significantly larger sample sizes, consequently greater representativeness, and substantially lower chance of being driven by omitted variable bias. They do, however, underline the fact that some of the results presented here may be more suggestive than definitive, given constraints on the data.

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, non-automobile accidents. We find little evidence that policy externalities greatly mattered in this context, but this is likely due to the relatively small number of potentially treated (dryish) cities in our sample. 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 wet status. Thus, our preferred estimates suggest that city-level repeal was associated with a roughly 11.6% decrease in mortality rates for non-automobile accidents (including accidental poisonings).

One way of contextualizing these results would be in terms of a nationwide count of the reduction in non-automobile accidents due to the repeal of federal prohibition. We can provide a 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 32,974 deaths attributable to non-automobile accidents on an annual basis for the period from 1933 to 1936 (relative to an urban population of 60,264,042 in the sample). Applying our benchmark estimate of −11.6% yields an annual reduction of 3825 deaths 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 non-automobile accidents by wet status yields an annual reduction of 3277 urban deaths in the short run.

Is it possible to contextualize the latter number? In previous work by Jacks et al. (2021) 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 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, 4493 annual excess infant deaths can be attributed to the repeal of federal prohibition.

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, this immediate 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. These results suggest that, on net, repeal likely had negative contemporaneous effects on all-cause mortality and, thereby, public health in the US. However, there were other associated components of repeal which remain unexplored and which should be added to any reckoning of prohibition's legacy. Chief among these is the potential effect on later-life mortality due to chronic disease, particularly for those exposed as young adults to dramatically easier access to alcohol, which would only serve to heighten repeal's potentially negative mortality effects.

Finally, we can briefly speculate about the lessons of federal prohibition. Given its scope, our integrated assessment of its effects on public health might inform policy making in the present, particularly as it relates to the currently evolving legalization of cannabis and the potential de jure decriminalization – but de facto legalization — of even harder drugs. First, the historical experience suggests that legalization of formerly illicit substances is likely to have significant unanticipated effects on public health. The pro-repeal literature of the 1920s and 1930s was consistent in its messaging that not only alcohol was potentially good for one's health but also prohibition's repeal would lead to fewer deaths by external causes like homicide and poisoning. However, to our knowledge, none of the literature, whether for or against repeal, made the link to infant mortality. And while it is important to recognize the different addictive, psychoactive, and potentially toxic properties of alcohol and other drugs, our work suggests that, at a minimum, any future episode of legalization is unlikely to result in the uniformly positive change in public health touted by its proponents.

Second, a key insight of our larger project is that mortality in this period was driven by not only any individual county's choice of prohibition status but also its neighbours' choice of prohibition status. That is, a city, county, or state's choice to go wet and allow for the sale of alcohol in its borders strongly affected mortality in neighbouring jurisdictions that chose to remain dry. It is telling that the legal debate on the relative merits and demerits of state-level legalization of cannabis and other drugs has failed to adequately address the possibility of such cross-jurisdictional externalities. And while we live in a world of different costs and modes of transaction, and vastly more information on the health effects of intoxicants like alcohol and other drugs, concerns over the possibility of cross-border policy spillovers as seen in the immediate period of repeal seem as relevant as ever, suggesting that if legalization is to proceed, it should be enacted with a careful eye to such prospective spill-over effects.

Data availability

All data and code used in this paper are available at www.davidjacks.org

APPENDIX A: HAZARD MODEL OF WET TRANSITIONS

Table A1 below reports the results of a hazard model ran at the city level ($n = 946$) for the period from 1933 to 1936. We use the number of years before a city becomes wet as the outcome and regress this time to transition on predetermined county characteristics at 1933 for time-varying county characteristics and 1930 for variables coming from that year's Census. There are a few plausible stories here: chiefly that cities with a higher black share of the population were slower to become wet while cities with a higher foreign-born share of the population were quicker to become wet. However, these do not appear to be quantitatively large effects: for instance, a one standard-deviation increase (0.18) in the black share of a city's population is predicted to lengthen the transition to wet status by roughly one day ($0.18 * -0.014 * 365$).

Table A1
Hazard analysis on years before becoming wet.

% black	**0.014**
	(0.003)
	[4.03]
% foreign-born	**−0.028**
	(0.006)
	[4.88]
% Baptist/Methodist	0.003
	(0.004)
	[0.61]
Unemployed-population ratio	−0.010
	(0.029)
	[0.35]
New Deal spending per capita	0.000
	(0.001)
	[0.67]
Hospital beds per 1000	−1.004
	(2.644)
	[0.38]
Institutions per 1000	−2.043
	(1.104)
	[1.85]
Retail sales per capita	0.094
	(0.261)
	[0.36]
Number of cities	946

Figures in bold are significant at the 5% (or lower) level.

APPENDIX B: INVERSE HYPERBOLIC SINES, LOGS, AND QUARTICS

Table 2 reports our baseline results for aggregated causes of death, using PPML with mortality rates in levels. In Tables B1 through B3, we consider the same specifications but instead substitute the inverse hyperbolic sine of mortality rates, scaled log rates (ln((deaths ± 1)/population) and the quartic root of mortality rates as the dependent variables, respectively. All of these transformations preserve the zeros in the data and allow us to instead estimate everything in the context of OLS.

Starting with Columns 1 through 4 in the three tables, we see statistically significant results are only consistently associated with acute, alcohol-related causes of death. Just as in Table 2, we also see no statistically significant results attached to either chronic, alcohol-related causes of death or potentially alcohol-related causes of death. For non-alcohol-related causes of death, we see a small but still significant effect for *wet in initial year* in Tables B1 through B3 unlike Table 2.

Table B1
Repeal's effect on aggregated causes of death, inverse hyperbolic sine.

	(1) Acute	(2) Chronic	(3) Potentially related	(4) Non-related
Dryish in initial year	−0.020	0.022	−0.014	0.026
	(0.020)	(0.013)	(0.014)	(0.016)
	[0.96]	[1.64]	[1.02]	[1.69]
Dryish in subsequent years	**−0.082**	−0.002	−0.037	0.018
	(0.039)	(0.027)	(0.027)	(0.025)
	[2.09]	[0.07]	[1.38]	[0.73]
Wet in initial year	**−0.040**	−0.008	−0.007	**−0.023**
	(0.013)	(0.010)	(0.010)	**(0.010)**
	[2.98]	[0.89]	[0.68]	**[2.26]**
Wet in subsequent years	**−0.049**	−0.016	0.012	0.001
	(0.019)	(0.016)	(0.016)	(0.018)
	[2.60]	[0.96]	[0.72]	[0.08]
N of observations	3784	3784	3784	3784
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 mortality rates after transformation, 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% (or lower) level.

Table B2
Repeal's effect on aggregated causes of death, in logs.

	(1) Acute	(2) Chronic	(3) Potentially related	(4) 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	3784	3784	3784	3784
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 mortality rates after transformation, 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% (or lower) level.

Tables B4 through B6 replicate the same type of exercise but do so in a consideration of the disaggregated acute causes of death (that is, automobile accidents, homicide, non-automobile accidents, and suicide). The same broad pattern emerges as in Table 3, namely that there are consistent, significant negative effects of repeal for non-automobile accidents but not for automobile accidents or suicide. The only really notable difference comes for *wet in subsequent years* and homicide wherein the coefficients are significant in the specifications with scaled log and quartic root rates. However, as mentioned in Section 4 of the text, in our sample of 3784 observations on homicides used in Table 3, fully 1443 (or 38%) of them are recorded as zero. In cases like these, Poisson models are preferable to using OLS with a transformed dependent variable like the log of 1 ± the count of deaths or the inverse hyperbolic sine as the latter can yield incorrect and unstable marginal effects (Manning and Mullahy, 2001; Mullahy and Norton, 2022).

Table B3
Repeal's effect on aggregated causes of death, in quartic roots.

	(1) Acute	(2) Chronic	(3) Potentially related	(4) Non-related
Dryish in initial year	−0.006	0.008	−0.005	0.009
	(0.007)	(0.005)	(0.005)	(0.006)
	[0.93]	[1.68]	[0.92]	[1.65]
Dryish in subsequent years	**−0.027**	0.000	−0.013	0.006
	(0.013)	(0.010)	(0.010)	(0.009)
	[2.06]	[0.00]	[1.32]	[0.60]
Wet in initial year	**−0.014**	−0.003	−0.002	**−0.008**
	(0.005)	(0.003)	(0.004)	**(0.004)**
	[3.01]	[0.89]	[0.52]	**[2.21]**
Wet in subsequent years	**−0.019**	−0.006	0.005	0.001
	(0.007)	(0.006)	(0.006)	(0.006)
	[2.83]	[0.99]	[0.90]	[0.09]
N of observations	3784	3784	3784	3784
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 mortality rates after transformation, 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% (or lower) level.

Table B4
Repeal's effect on acute causes of death, inverse hyperbolic sine.

	(1) Automobile accidents	(2) Homicide	(3) Non-auto accidents	(4) Suicide
Dryish in initial year	0.000	−0.002	−0.030	0.004
	(0.016)	(0.011)	(0.025)	(0.011)
	[0.02]	[0.21]	[1.22]	[0.40]
Dryish in subsequent years	−0.040	**−0.072**	−0.049	0.009
	(0.035)	**(0.023)**	(0.036)	(0.012)
	[1.12]	**[3.14]**	[1.36]	[0.73]
Wet in initial year	−0.006	**−0.017**	**−0.046**	0.004
	(0.009)	**(0.006)**	**(0.018)**	(0.007)
	[0.59]	**[2.74]**	**[2.54]**	[0.65]
Wet in subsequent years	−0.023	−0.017	**−0.052**	0.015
	(0.015)	(0.010)	**(0.022)**	(0.011)
	[1.51]	[1.73]	**[2.37]**	[1.38]
N of observations	3784	3784	3784	3784
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 mortality rates after transformation, 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% (or lower) level.

Table B5
Repeal's effect on acute causes of death, in logs.

	(1) Automobile accidents	(2) Homicide	(3) Non-auto accidents	(4) 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	3784	3784	3784	3784
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 mortality rates after transformation, 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% (or lower) level.

Table B6
Repeal's effect on acute causes of death, in quartic roots.

	(1) Automobile accidents	(2) Homicide	(3) Non-auto accidents	(4) Suicide
Dryish in initial year	0.006	−0.016	−0.013	0.010
	(0.010)	(0.015)	(0.010)	(0.015)
	[0.63]	[1.04]	[1.34]	[0.69]
Dryish in subsequent years	−0.008	**−0.054**	−0.018	0.014
	(0.020)	**(0.026)**	(0.015)	(0.021)
	[0.40]	**[2.06]**	[1.23]	[0.68]
Wet in initial year	0.000	−0.019	**−0.021**	0.005
	(0.007)	(0.011)	**(0.008)**	(0.009)
	[0.03]	[1.78]	**[2.71]**	[0.53]
Wet in subsequent years	−0.010	**−0.039**	**−0.025**	0.018
	(0.011)	**(0.018)**	**(0.010)**	(0.013)
	[0.93]	**[2.13]**	**[2.64]**	[1.35]
N of observations	3784	3784	3784	3784
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 mortality rates after transformation, 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% (or lower) level.

APPENDIX C: DIFFERENTIAL IMPACTS OF PROHIBITION'S REPEAL

As mentioned in the text, an 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 represented at least 10% of the total population. By and large, non-white in this context can be interpreted as black in that the latter represented approximately 95% of the non-white population at the time (Gibson and Jung, 2002). However, there was significant variation in this ratio across the US, particularly in border states like California and Texas.

Here, we limit our attention to cities with overwhelmingly black populations due to inconsistencies in how non-white populations are classified in the sample period. In particular, the Mexican population of the United States went from being tabulated as non-white to white in between the 1930 and 1940 Censuses:

Until 1930, Mexicans, the dominant Hispanic national origin group, had been classified as white. A "Mexican" race category was added in the 1930 census, following a rise in immigration that dated to the Mexican Revolution in 1910. But Mexican Americans

Table C1
Aggregated causes of death, black population, restricted sample.

	(1) Acute	(2) Chronic	(3) Potentially related	(4) Non-related
Dryish in initial year	−0.008	0.009	−0.011	−0.014
	(0.060)	(0.037)	(0.027)	(0.026)
	[0.14]	[0.25]	[0.41]	[0.53]
Dryish in subsequent years	**−0.191**	−0.003	−0.039	−0.021
	(0.072)	(0.053)	(0.044)	(0.049)
	[2.63]	[0.06]	[0.88]	[0.42]
Wet in initial year	−0.086	0.017	0.005	0.038
	(0.051)	(0.044)	(0.034)	(0.036)
	[1.71]	[0.38]	[0.15]	[1.06]
Wet in subsequent years	**−0.225**	−0.015	0.066	0.054
	(0.077)	(0.080)	(0.052)	(0.068)
	[2.93]	[0.19]	[1.27]	[0.80]
N of observations	540	540	540	540
City & year fixed effects	X	X	X	X
County controls with linear trends	X	X	X	X
State linear trends	X	X	X	X

PPML regression of mortality rates in levels, 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% (or lower) level.

(helped by the Mexican government) lobbied successfully to eliminate it in the 1940 census and revert to being classified as white, which gave them more legal rights and privileges. (Pew Research Center, 2015, p. 26)

Consequently, this introduces "an element of incomparability into the race-specific rates [reported in the *Vital Statistics*] for the years 1932 to 1934″ (Linder and Grove, 1947, p. 103). Our work-around then comes in only considering cities both for which non-white deaths are reported as a separate category and which are located in states in which black people represented >90% of the non-white population. Thus, we collected data on causes of death for non-white people in 243 cities and matched these with black population counts for 135 cities in 1930 taken from US Bureau of the Census (1935). Critically, we note that of the 135 cities in this restricted sample, 121 are in the southern United States.

Table C1 presents the results from considering the respective aggregated mortality rates for black people, using the restricted sample. A qualitatively similar set of results emerges as in the main text. For chronic, potentially related, and non-related causes of black mortality, the repeal of prohibition does not seem to have left any trace. And for acute causes of death alone, we find significant effects. However, the repeal of prohibition was associated with a reduction in deaths by acute causes for *wet in subsequent years* which was roughly 3x larger than in Table 2 (and roughly 2x for *dryish in subsequent years*). At first glance then, these results seem to indicate potentially large differential impacts of prohibition's repeal on the basis of race (albeit the respective coefficients in Table C1 are not significantly different from their counterparts in Table 2).

Table C2 explores this possibility in more depth. Here, we consider white mortality rates for aggregated causes of death in the same restricted sample of 135 cities with significant black populations. The point estimates for acute causes are very close in value to those attached to black mortality in the same cities. Consequently, the respective coefficients in Table C2 are not significantly different from their counterparts in Table C1. We therefore cannot then rule out the possibility that the differential outcomes reported in Table C1 are driven by sample selection issues. In particular, 89.6% of the cities in the restricted sample are in the southern United States, so any differential outcomes on the basis of race more likely reflect underlying heterogeneity in outcomes in that particular region.

Table C3 confirms this suspicion. It returns to a consideration of total (non-white plus white) mortality by aggregated causes of death. However, it differs from Table 2 by only considering the full sample of cities less the restricted sample of cities with significant black populations used in Tables C1 and C2 (that is, $n = 946 - 135 = 811$). In this instance, for acute, chronic, potentially related, and non-related causes of total mortality alike, we see no significant effects associated with prohibition's repeal (apart from the coefficient on chronic causes of death for *dryish in initial year*). The point estimate for *wet in subsequent years* is, however, significantly different from both those reported in Table C1 and C2. Thus, the headline results reported in the main text may be driven by effects in a relatively small subset of cities.

Tables C4, C5, and C6 report equivalent results for disaggregated acute causes of death by respectively considering black mortality in the restricted sample, white mortality in the restricted sample, and total mortality in the full-less-restricted sample. The results suggest that: (1) there are no significant differences in the point estimates for the black and white populations in the restricted sample (Tables C4 and C5); and (2) there may be some reason to believe that the results for acute causes of death reported in the main text may also be driven by effects in a relatively small subset of cities (Table C6).

Table C2
Aggregated causes of death, white population, restricted sample.

	(1) Acute	(2) Chronic	(3) Potentially related	(4) Non-related
Dryish in initial year	**−0.077**	−0.026	−0.016	0.008
	(0.036)	(0.023)	(0.025)	(0.024)
	[2.13]	[1.15]	[0.65]	[0.33]
Dryish in subsequent years	**−0.136**	−0.043	−0.033	0.054
	(0.058)	(0.045)	(0.036)	(0.039)
	[2.35]	[0.95]	[0.93]	[1.38]
Wet in initial year	**−0.105**	−0.014	−0.009	−0.010
	(0.042)	(0.020)	(0.023)	(0.023)
	[2.51]	[0.70]	[0.37]	[0.46]
Wet in subsequent years	**−0.156**	−0.028	0.023	0.013
	(0.053)	(0.031)	(0.030)	(0.043)
	[2.94]	[0.89]	[0.78]	[0.30]
N of observations	540	540	540	540
City & year fixed effects	X	X	X	X
County controls with linear trends	X	X	X	X
State linear trends	X	X	X	X

PPML regression of mortality rates in levels, 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% (or lower) level.

Table C3
Aggregated causes of death, total population, full-less-restricted sample.

	(1) Acute	(2) Chronic	(3) Potentially related	(4) Non-related
Dryish in initial year	−0.008	**0.056**	−0.012	0.043
	(0.032)	**(0.021)**	(0.021)	(0.025)
	[0.25]	**[2.70]**	[0.56]	[1.71]
Dryish in subsequent years	−0.072	0.056	−0.038	−0.032
	(0.054)	(0.034)	(0.035)	(0.034)
	[1.33]	[1.65]	[1.11]	[0.96]
Wet in initial year	−0.020	0.015	0.005	−0.014
	(0.018)	(0.011)	(0.012)	(0.013)
	[1.10]	[1.45]	[0.42]	[1.09]
Wet in subsequent years	−0.028	0.008	0.013	0.007
	(0.029)	(0.020)	(0.021)	(0.022)
	[0.99]	[0.39]	[0.61]	[0.33]
N of observations	3244	3244	3244	3244
City & year fixed effects	X	X	X	X
County controls with linear trends	X	X	X	X
State linear trends	X	X	X	X

PPML regression of mortality rates in levels, 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% (or lower) level.

Table C4
Acute causes of death, black population, restricted sample.

	(1) Automobile accidents	(2) Homicide	(3) Non-auto accidents	(4) Suicide
Dryish in initial year	−0.149	0.030	−0.006	0.369
	(0.150)	(0.069)	(0.100)	(0.230)
	[1.00]	[0.43]	[0.06]	[1.60]
Dryish in subsequent years	**−0.440**	−0.141	−0.144	0.494
	(0.204)	(0.093)	(0.131)	(0.616)
	[2.15]	[1.52]	[1.10]	[0.80]
Wet in initial year	−0.018	−0.032	−0.169	−0.344
	(0.140)	(0.084)	(0.103)	(0.274)
	[0.13]	[0.37]	[1.64]	[1.26]
Wet in subsequent years	−0.120	−0.133	**−0.416**	0.100
	(0.210)	(0.138)	**(0.156)**	(0.446)
	[0.57]	[0.96]	**[2.67]**	[0.22]
N of observations	540	540	540	540
City & year fixed effects	X	X	X	X
County controls with linear trends	X	X	X	X
State linear trends	X	X	X	X

PPML regression of mortality rates in levels, 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% (or lower) level.

Table C5
Acute causes of death, white population, restricted sample.

	(1) Automobile accidents	(2) Homicide	(3) Non-auto accidents	(4) Suicide
Dryish in initial year	−0.050	0.062	**−0.152**	−0.021
	(0.068)	(0.166)	**(0.075)**	(0.096)
	[0.74]	[0.37]	**[2.02]**	[0.22]
Dryish in subsequent years	−0.073	**−0.521**	**−0.158**	−0.040
	(0.118)	**(0.181)**	**(0.068)**	(0.091)
	[0.62]	**[2.88]**	**[2.35]**	[0.44]
Wet in initial year	−0.065	0.108	**−0.185**	−0.075
	(0.052)	(0.118)	**(0.094)**	(0.087)
	[1.24]	[0.92]	**[1.97]**	[0.87]
Wet in subsequent years	−0.131	0.107	**−0.284**	−0.018
	(0.088)	(0.172)	**(0.109)**	(0.158)
	[1.49]	[0.62]	**[2.60]**	[0.12]
N of observations	540	540	540	540
City & year fixed effects	X	X	X	X
County controls with linear trends	X	X	X	X
State linear trends	X	X	X	X

PPML regression of mortality rates in levels, 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% (or lower) level.

Table C6
Acute causes of death, total population, full-less-restricted sample.

	(1) Automobile accidents	(2) Homicide	(3) Non-auto accidents	(4) Suicide
Dryish in initial year	0.023	0.104	−0.011	0.020
	(0.070)	(0.152)	(0.060)	(0.106)
	[0.33]	[0.69]	[0.18]	[0.19]
Dryish in subsequent years	0.073	0.230	0.009	0.098
	(0.157)	(0.363)	(0.122)	(0.243)
	[0.47]	[0.63]	[0.07]	[0.40]
Wet in initial year	0.029	0.088	−0.038	0.025
	(0.051)	(0.115)	(0.034)	(0.061)
	[0.57]	[0.77]	[1.11]	[0.41]
Wet in subsequent years	0.000	0.066	−0.055	0.065
	(0.076)	(0.163)	(0.053)	(0.094)
	[0.01]	[0.41]	[1.04]	[0.68]
N of observations	3244	3244	3244	3244
City & year fixed effects	X	X	X	X
County controls with linear trends	X	X	X	X
State linear trends	X	X	X	X

PPML regression of mortality rates in levels, 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% (or lower) level.

APPENDIX D: PRE-TRENDS FOR ACUTE MORTALITY RATES

As our econometric strategy is analogous to difference-in-differences, 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 gage 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.

Figs. D1 through D5 tracks raw mortality rates (expressed as deaths per 1000 population) for one aggregated and four disaggregated causes of death in the period from 1928 to 1932 (acute, automobile accidents, homicide, non-automobile accidents, and suicide). However, because of changes in data collection from year to year, the sample is limited to only the 360 largest US cities as opposed to the 946 cities used in the text. This sample restriction serves to exclude over 20 million residents of small cities (roughly 1/3 of the US urban population).

With this caveat in mind, we employ two mutually exclusive categories, always dry and ever wet. Always dry cities are those which remain dry throughout the post-repeal period. Ever wet cities are those which allow for legal alcohol sales at some point in the post-repeal period. Thus, the composition of cities is held constant.

Starting with Fig. D1, we observe a general, but moderate decline in mortality rates from acute causes of death for the two city types, and the general ordering is strongly preserved when considering the years between 1928 and 1932: acute mortality rates are

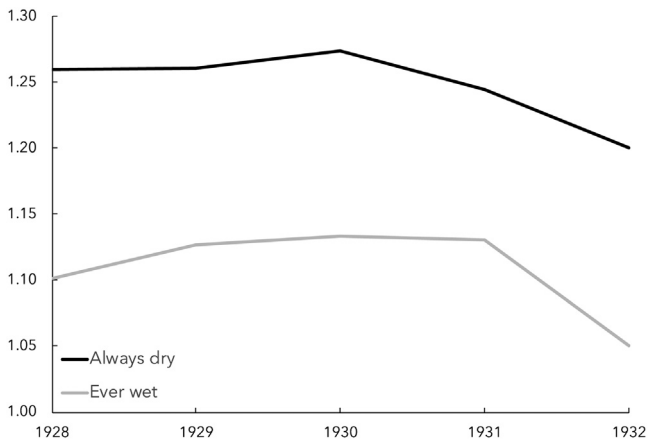

Fig. D1. Pre-trends in acute sources of urban mortality, 1928–1932.

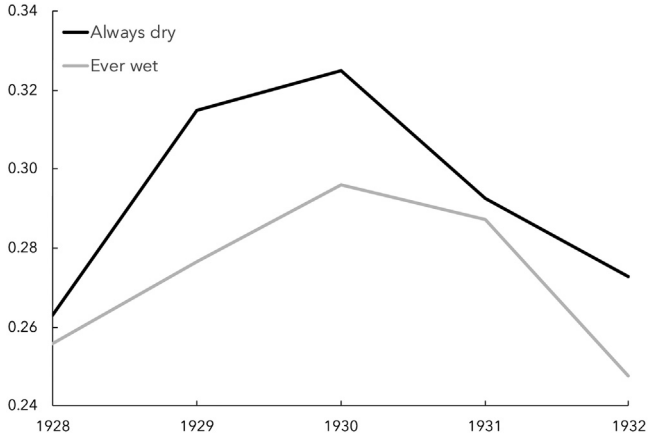

Fig. D2. Pre-trends in deaths due to automobile accidents, 1928–1932.

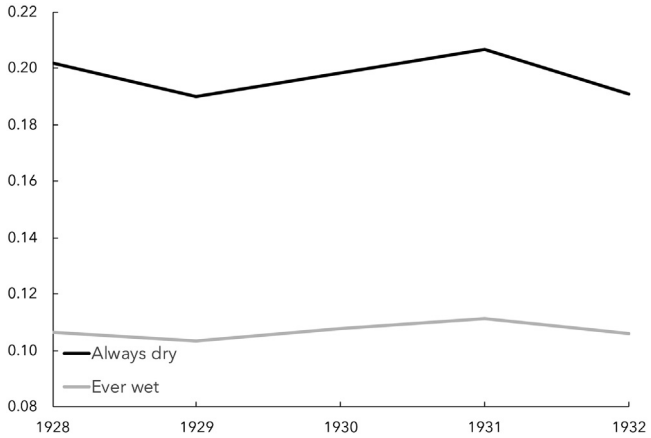

Fig. D3. Pre-trends in deaths due to homicide, 1928–1932.

highest for always-dry cities and lowest for ever-wet cities. In these years, they also exhibit highly similar pre-trends. In other words, Fig. D1 suggests that the common-trend assumption holds for this restricted sample of cities, thereby validating the results reported in the text.

Similarly, we find reassuring evidence related to the common-trend assumption for homicide and non-automobile accidents in Figs. D3 and D4, respectively. In both instances, always-dry cities both begin and end with higher mortality rates than ever-wet cities. Moreover, the difference between the two is roughly constant. This is reassuring, given that non-automobile accidents were the single component of acute sources of death for which consistently significant effects were found in the text. Turning to the other two components of acute sources of death for which consistently insignificant effects were found in the text, the implications of Figs. D2 and D5 for automobile accidents and suicide, respectively, are less clear. For automobile accidents, always-dry cities have consistently higher mortality rates than ever-wet cities, but the difference varies considerably over time. Likewise, for suicide, ever-wet cities have consistently higher mortality rates than always-dry cities, but the difference narrows considerably over time.

Fig. D1 depicts a sample of large cities from 1928 to 1932 ($n = 360$). Always dry cities remain dry for the entire post-repeal period. Ever wet cities allow for legal alcohol sales at some point in the post-repeal period. Both series are expressed as deaths per 1000.

Fig. D2 depicts a sample of large cities from 1928 to 1932 ($n = 360$). Always dry cities remain dry for the entire post-repeal period. Ever wet cities allow for legal alcohol sales at some point in the post-repeal period. Both series are expressed as deaths per 1000.

Fig. D3 depicts a sample of large cities from 1928 to 1932 ($n = 360$). Always dry cities remain dry for the entire post-repeal period. Ever wet cities allow for legal alcohol sales at some point in the post-repeal period. Both series are expressed as deaths per 1000.

Fig. D4 depicts a sample of large cities from 1928 to 1932 ($n = 360$). Always dry cities remain dry for the entire post-repeal period. Ever wet cities allow for legal alcohol sales at some point in the post-repeal period. Both series expressed as deaths per 1000.

Fig. D5 depicts a sample of large cities from 1928 to 1932 ($n = 360$). Always dry cities remain dry for the entire post-repeal period. Ever wet cities allow for legal alcohol sales at some point in the post-repeal period. Both series expressed as deaths per 1000.

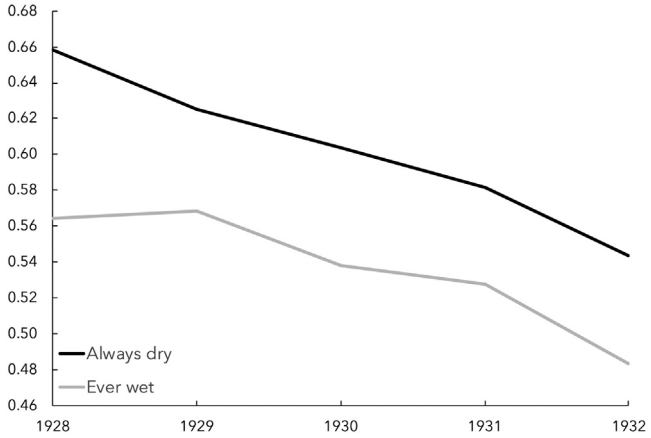

Fig. D4. Pre-trends in deaths due to non-automobile accidents, 1928–1932.

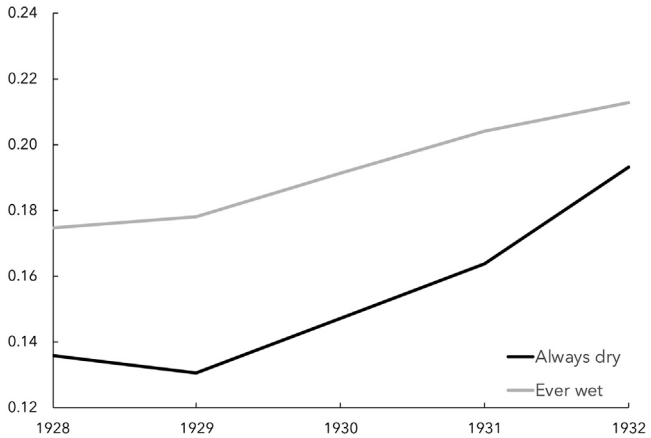

Fig. D5. Pre-trends in deaths due to suicide, 1928–1932.

APPENDIX E: RESULTS FOR ALL OTHER DISAGGREGATED CAUSES OF DEATH

In the text, we consider four disaggregated acute causes of death, motivated by the results in Tables 2 which detects consistently signed and significant effects for their summed value.

In the interests of space, the text thereby neglects a separate consideration:

(i) of the three disaggregated chronic, alcohol-related causes of death;
(ii) of the six potentially alcohol-related causes of death; and
(iii) of the 12 non-alcohol-related causes of death.

Here, we consider each of the 21 other disaggregated causes of death in turn. These results strongly support our decision to focus on acute causes of death.

For chronic, alcohol-related causes, there are zero statistically significant coefficients across the four parameters of interest and the three dependent variables considered (that is, zero coefficients for 12 parameters of interest). These results are displayed in Table E1. For potentially alcohol-related causes, there is exactly one statistically significant coefficient across the four parameters of interest and the six dependent variables considered (that is, one coefficient for 24 parameters of interest). These results are displayed in Table E2 below.

And for non-alcohol-related causes, there are three statistically significant coefficients across the four parameters of interest and the 12 dependent variables considered (that is, three coefficients for 48 parameters of interest. These results are displayed in Tables E3 and E4 below. In sum, four statistically significant – but potentially spurious – coefficients for 84 (or 4.76%) parameters of interest is roughly to be expected when using a 5% level of significance.

In Tables E5 through E8 below, we have repeat this exercise, but now with the inclusion of all data through 1939. This results in a larger number of apparently statistically significant results for slower-moving chronic, only partially related, and even non-related causes. Rather than four statistically significant – but potentially spurious – coefficients for 84 (or 4.76% of all) parameters of interest,

Table E1
Repeal's effect on chronic causes of death.

	(1) Cirrhosis	(2) Heart disease	(3) Nephritis
Dryish in initial year	0.016	0.034	−0.006
	(0.091)	(0.018)	(0.033)
	[0.17]	[1.90]	[0.20]
Dryish in subsequent years	−0.164	0.003	0.016
	(0.117)	(0.033)	(0.040)
	[1.41]	[0.09]	[0.39]
Wet in initial year	0.047	−0.019	0.007
	(0.055)	(0.011)	(0.020)
	[0.85]	[1.68]	[0.35]
Wet in subsequent years	0.009	−0.036	0.021
	(0.089)	(0.019)	(0.032)
	[0.11]	[1.84]	[0.65]
N of observations	3784	3784	3784
City & year fixed effects	X	X	X
County controls with linear trends	X	X	X
State linear trends	X	X	X

PPML regression of mortality rates in levels, weighted by city population for the years from 1933 to 1936. 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% (or lower) level.

Table E2
Repeal's effect on potentially related causes of death.

	(1) Cancer	(2) Cerebral hemorrhage	(3) Cerebrospinal meningitis	(4) Influenza/ pneumonia	(5) Malaria	(6) Tuberculosis
Dryish in initial year	−0.032	0.016	**0.339**	−0.008	−0.128	−0.019
	(0.021)	(0.027)	**(0.166)**	(0.038)	(0.172)	(0.039)
	[1.50]	[0.60]	**[2.05]**	[0.21]	[0.75]	[0.49]
Dryish in subsequent years	0.003	−0.057	−0.129	−0.062	0.001	−0.022
	(0.040)	(0.053)	(0.287)	(0.048)	(0.207)	(0.051)
	[0.08]	[1.08]	[0.45]	[1.30]	[0.01]	[0.43]
Wet in initial year	0.001	−0.030	−0.026	−0.034	0.136	0.020
	(0.015)	(0.018)	(0.155)	(0.022)	(0.154)	(0.022)
	[0.05]	[1.64]	[0.17]	[1.56]	[0.88]	[0.92]
Wet in subsequent years	−0.000	−0.031	0.046	0.025	0.235	0.008
	(0.025)	(0.028)	(0.225)	(0.033)	(0.281)	(0.037)
	[0.00]	[1.11]	[0.20]	[0.76]	[0.84]	[0.21]
N of observations	3784	3784	3784	3784	3784	3784
City & year fixed effects	X	X	X	X	X	X
County controls with linear trends	X	X	X	X	X	X
State linear trends	X	X	X	X	X	X

PPML regression of mortality rates in levels, weighted by city population for the years from 1933 to 1936. 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% (or lower) level.

these new results feature 20 statistically significant – but potentially spurious – coefficients for 84 (or 23.81% of all) parameters of interest.

Also, since nearly all of the statistically significant results from the longer panel are negatively signed, this suggests that wet prohibition status was likely correlated with the diffusion of sulfa across cities. Thus, we are again more comfortable with the results from the shorter panel as these likely represent the most conservative estimates related to repeal.

Table E3
Repeal's effect on non-related causes of death, part 1.

	(1) All other causes	(2) Appendicitis	(3) Diabetes mellitus	(4) Diphtheria	(5) Hernia/ Internal obstruction	(6) Other puerperal causes
Dryish in initial year	0.020	0.075	−0.018	**0.358**	−0.004	−0.010
	(0.020)	(0.057)	(0.063)	**(0.121)**	(0.062)	(0.075)
	[1.00]	[1.31]	[0.29]	**[2.97]**	[0.07]	[0.13]
Dryish in subsequent years	−0.006	0.060	0.079	0.299	−0.018	−0.136
	(0.031)	(0.082)	(0.066)	(0.184)	(0.077)	(0.123)
	[0.18]	[0.73]	[1.19]	[1.62]	[0.24]	[1.11]
Wet in initial year	−0.006	−0.046	−0.042	0.101	0.035	−0.055
	(0.013)	(0.030)	(0.031)	(0.106)	(0.036)	(0.052)
	[0.43]	[1.55]	[1.34]	[0.96]	[0.98]	[1.05]
Wet in subsequent years	0.012	−0.079	0.023	0.183	0.017	−0.000
	(0.022)	(0.046)	(0.047)	(0.161)	(0.062)	(0.084)
	[0.53]	[1.71]	[0.48]	[1.14]	[0.27]	[0.00]
N of observations	3784	3784	3784	3784	3784	3784
City & year fixed effects	X	X	X	X	X	X
County controls with linear trends	X	X	X	X	X	X
State linear trends	X	X	X	X	X	X

PPML regression of mortality rates in levels, weighted by city population for the years from 1933 to 1936. 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% (or lower) level.

Table E4
Repeal's effect on non-related causes of death, part 2.

	(7) Puerperal septicemia	(8) Rheumatism/ gout	(9) Scarlet fever	(10) Syphilis	(11) Typhoid/ paratyphoid	(12) Whooping cough
Dryish in initial year	−0.037	−0.131	−0.020	0.009	0.122	−0.011
	(0.099)	(0.127)	(0.238)	(0.077)	(0.112)	(0.158)
	[0.37]	[1.04]	[0.09]	[0.12]	[1.08]	[0.07]
Dryish in subsequent years	**0.267**	0.164	0.149	−0.087	−0.106	0.114
	(0.115)	(0.243)	(0.380)	(0.111)	(0.147)	(0.221)
	[2.33]	[0.68]	[0.39]	[0.78]	[0.72]	[0.51]
Wet in initial year	−0.051	−0.132	0.069	−0.034	0.007	**−0.320**
	(0.061)	(0.084)	(0.145)	(0.049)	(0.101)	**(0.119)**
	[0.83]	[1.57]	[0.48]	[0.69]	[0.07]	**[2.70]**
Wet in subsequent years	−0.002	−0.118	0.132	−0.033	−0.009	−0.414
	(0.103)	(0.129)	(0.256)	(0.077)	(0.170)	(0.226)
	[0.02]	[0.91]	[0.52]	[0.42]	[0.05]	[1.83]
N of observations	3784	3784	3784	3784	3784	3784
City & year fixed effects	X	X	X	X	X	X
County controls with linear trends	X	X	X	X	X	X
State linear trends	X	X	X	X	X	X

PPML regression of mortality rates in levels, weighted by city population for the years from 1933 to 1936. 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% (or lower) level.

Table E5
Repeal's effect on chronic causes of death.

	(1) Cirrhosis	(2) Heart disease	(3) Nephritis
Dryish in initial year	**0.171**	**0.034**	0.024
	(0.084)	**(0.014)**	(0.026)
	[2.04]	**[2.35]**	[0.89]
Dryish in subsequent years	0.007	0.012	0.019
	(0.071)	(0.019)	(0.032)
	[0.10]	[0.66]	[0.61]
Wet in initial year	0.002	−0.016	−0.018
	(0.040)	(0.009)	(0.013)
	[0.06]	[1.75]	[1.36]
Wet in subsequent years	−0.104	**−0.041**	**−0.048**
	(0.055)	**(0.016)**	**(0.020)**
	[1.90]	**[2.57]**	**[2.44]**
N of observations	6741	6741	6741
City & year fixed effects	X	X	X
County controls with linear trends	X	X	X
State linear trends	X	X	X

PPML regression of mortality rates in levels, weighted by city population for the years from 1933 to 1939. 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% (or lower) level.

Table E6
Repeal's effect on potentially related causes of death.

	(1) Cancer	(2) Cerebral hemorrhage	(3) Cerebrospinal meningitis	(4) Influenza/ pneumonia	(5) Malaria	(6) Tuberculosis
Dryish in initial year	0.008	0.018	**0.353**	0.010	−0.179	0.024
	(0.020)	(0.021)	**(0.157)**	(0.033)	(0.152)	(0.030)
	[0.38]	[0.82]	**[2.24]**	[0.31]	[1.17]	[0.78]
Dryish in subsequent years	0.069	−0.009	0.122	−0.021	**0.310**	0.029
	(0.044)	(0.025)	(0.272)	(0.036)	**(0.155)**	(0.031)
	[1.57]	[0.37]	[0.45]	[0.57]	**[2.00]**	[0.95]
Wet in initial year	−0.006	−0.033	0.285	**−0.059**	0.002	0.012
	(0.013)	(0.015)	(0.149)	**(0.018)**	(0.088)	(0.015)
	[0.46]	[2.30]	[1.92]	**[3.27]**	[0.03]	[0.84]
Wet in subsequent years	−0.026	−0.041	**−0.361**	**−0.103**	−0.146	−0.015
	(0.018)	(0.021)	**(0.170)**	**(0.031)**	(0.129)	(0.023)
	[1.42]	[1.91]	**[2.12]**	**[3.32]**	[1.13]	[0.67]
N of observations	6741	6741	6741	6741	6741	6741
City & year fixed effects	X	X	X	X	X	X
County controls with linear trends	X	X	X	X	X	X
State linear trends	X	X	X	X	X	X

PPML regression of mortality rates in levels, weighted by city population for the years from 1933 to 1939. 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% (or lower) level.

Table E7
Repeal's effect on non-related causes of death, part 1.

	(1) All other causes	(2) Appendicitis	(3) Diabetes mellitus	(4) Diphtheria	(5) Hernia/ Internal obstruction	(6) Other puerperal causes
Dryish in initial year	0.014	0.061	−0.032	**0.291**	0.064	−0.011
	(0.020)	(0.044)	(0.040)	**(0.091)**	(0.049)	(0.064)
	[0.70]	[1.39]	[0.79]	**[3.20]**	[1.31]	[0.18]
Dryish in subsequent years	0.030	0.020	−0.012	0.189	0.039	−0.120
	(0.034)	(0.068)	(0.041)	(0.151)	(0.067)	(0.092)
	[0.88]	[0.30]	[0.29]	[1.26]	[0.58]	[1.30]
Wet in initial year	−0.018	**−0.075**	**−0.056**	0.056	**−0.056**	**−0.117**
	(0.014)	**(0.028)**	**(0.023)**	(0.084)	**(0.028)**	**(0.038)**
	[1.26]	**[2.71]**	**[2.41]**	[0.67]	**[1.99]**	**[3.12]**
Wet in subsequent years	−0.008	**−0.121**	−0.039	0.045	**−0.081**	**−0.106**
	(0.023)	**(0.042)**	(0.032)	(0.116)	**(0.039)**	**(0.052)**
	[0.34]	**[2.86]**	[1.24]	[0.38]	**[2.07]**	**[2.02]**
N of observations	6741	6741	6741	6741	6741	6741
City & year fixed effects	X	X	X	X	X	X
County controls with linear trends	X	X	X	X	X	X
State linear trends	X	X	X	X	X	X

PPML regression of mortality rates in levels, weighted by city population for the years from 1933 to 1939. 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% (or lower) level.

Table E8
Repeal's effect on non-related causes of death, part 2.

	(7) Puerperal septicemia	(8) Rheumatism/ gout	(9) Scarlet fever	(10) Syphilis	(11) Typhoid/ paratyphoid	(12) Whooping cough
Dryish in initial year	0.052	−0.071	0.003	0.001	0.128	0.284
	(0.096)	(0.108)	(0.191)	(0.089)	(0.101)	(0.146)
	[0.54]	[0.65]	[0.02]	[0.02]	[1.27]	[1.95]
Dryish in subsequent years	**0.215**	0.076	**0.507**	−0.005	0.082	0.240
	(0.090)	(0.146)	**(0.244)**	(0.071)	(0.124)	(0.132)
	[2.39]	[0.52]	**[2.08]**	[0.07]	[0.66]	[1.811]
Wet in initial year	−0.087	−0.083	0.013	−0.004	0.060	**−0.066**
	(0.047)	(0.062)	(0.104)	(0.041)	(0.072)	**(0.087)**
	[1.84]	[1.34]	[0.13]	[0.09]	[0.84]	**[0.76]**
Wet in subsequent years	−0.075	−0.033	0.099	−0.031	0.078	0.190
	(0.069)	(0.085)	(0.163)	(0.057)	(0.101)	(0.099)
	[1.08]	[0.39]	[0.61]	[0.55]	[0.77]	[1.91]
N of observations	6741	6741	6741	6741	6741	6741
City & year fixed effects	X	X	X	X	X	X
County controls with linear trends	X	X	X	X	X	X
State linear trends	X	X	X	X	X	X

PPML regression of mortality rates in levels, weighted by city population for the years from 1933 to 1939. 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% (or lower) level.

APPENDIX F: ROBUSTNESS, VARIATIONS ON CITY, REGIONAL, AND STATE EFFECTS

In the text, Table 5 collates the results from various robustness exercises for acute causes of death and non-automobile accidents. Here, we explore the full set of robustness results related to variations on the state fixed effects used as controls. In particular, we consider the substitution of state-by-year fixed effects, Census region-by-year fixed effects, and city linear trends for state linear trends, in turn.

Table F1 introduces state-by-year fixed effects into our baseline specification for the four aggregated mortality rates (acute, chronic, potentially related, and non-related) for the total population of cities. For acute causes, *dryish in subsequent years* and *wet in subsequent years* both register as negative, of a similar magnitude as the coefficients reported in the main text, and statistically significant.

Table F2 introduces state-by-year fixed effects into our baseline specification for the four acute causes of death (automobile accidents, homicide, non-automobile accidents, and suicide) for the total population of cities. In this case, the results for non-automobile accidents are consistent in sign and size relative to the baseline specification, but are marginally statistically insignificant. Here, we

Table F1
Repeal's effect on aggregated causes of death, state-by-year fixed effects.

	(1) Acute	(2) Chronic	(3) Potentially related	(4) Non-related
Dryish in initial year	−0.021	0.030	−0.009	0.027
	(0.029)	(0.017)	(0.021)	(0.018)
	[0.71]	[1.80]	[0.45]	[1.49]
Dryish in subsequent years	**−0.143**	0.008	−0.048	0.013
	(0.050)	(0.029)	(0.030)	(0.026)
	[2.86]	[0.28]	[1.57]	[0.49]
Wet in initial year	−0.052	0.011	−0.070	0.020
	(0.062)	(0.052)	(0.040)	(0.045)
	[0.85]	[0.21]	[1.76]	[0.44]
Wet in subsequent years	**−0.152**	0.015	−0.051	−0.004
	(0.068)	(0.051)	(0.048)	(0.050)
	[2.22]	[0.30]	[1.07]	[0.09]
N of observations	3784	3784	3784	3784
City & year fixed effects	X	X	X	X
County controls with linear trends	X	X	X	X
State-year fixed effects	X	X	X	X

PPML regression of mortality rates in levels, 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% (or lower) level.

Table F2
Repeal's effect on acute causes of death, state-by-year fixed effects.

	(1) Automobile accidents	(2) Homicide	(3) Non-auto accidents	(4) Suicide
Dryish in initial year	−0.057	0.059	−0.024	−0.010
	(0.050)	(0.060)	(0.045)	(0.076)
	[1.14]	[0.99]	[0.55]	[0.14]
Dryish in subsequent years	−0.144	**−0.271**	−0.123	−0.018
	(0.094)	**(0.094)**	(0.064)	(0.088)
	[1.53]	**[2.88]**	[1.92]	[0.21]
Wet in initial year	0.073	−0.142	−0.084	−0.095
	(0.113)	(0.180)	(0.109)	(0.199)
	[0.64]	[0.79]	[0.77]	[0.48]
Wet in subsequent years	−0.044	−0.259	−0.199	−0.117
	(0.130)	(0.178)	(0.109)	(0.146)
	[0.34]	[1.46]	[1.83]	[0.80]
N of observations	3784	3784	3784	3784
City & year fixed effects	X	X	X	X
County controls with linear trends	X	X	X	X
State-year fixed effects	X	X	X	X

PPML regression of mortality rates in levels, 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% (or lower) level.

Table F3
Repeal's effect on aggregated causes of death, Census region-by-year fixed effects.

	(1) Acute	(2) Chronic	(3) Potentially related	(4) Non-related
Dryish in initial year	−0.036	0.016	−0.012	0.018
	(0.022)	(0.013)	(0.014)	(0.015)
	[1.62]	[1.23]	[0.89]	[1.18]
Dryish in subsequent years	**−0.117**	0.001	−0.032	0.006
	(0.041)	(0.026)	(0.023)	(0.024)
	[2.84]	[0.04]	[1.35]	[0.24]
Wet in initial year	**−0.047**	−0.010	−0.015	−0.012
	(0.020)	(0.010)	(0.012)	(0.010)
	[2.37]	[1.03]	[1.23]	[1.23]
Wet in subsequent years	**−0.060**	0.028	−0.018	0.003
	(0.023)	(0.016)	(0.015)	(0.016)
	[2.57]	[1.74]	[1.16]	[0.17]
N of observations	3784	3784	3784	3784
City & year fixed effects	X	X	X	X
County controls with linear trends	X	X	X	X
Region-year fixed effects	X	X	X	X

PPML regression of mortality rates in levels, 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% (or lower) level.

Table F4
Repeal's effect on acute causes of death, Census region-by-year fixed effects.

	(1) Automobile accidents	(2) Homicide	(3) Non-auto accidents	(4) Suicide
Dryish in initial year	−0.017	0.009	−0.075	0.022
	(0.043)	(0.050)	(0.040)	(0.063)
	[0.39]	[0.19]	[1.86]	[0.36]
Dryish in subsequent years	−0.112	**−0.142**	**−0.157**	0.068
	(0.085)	**(0.069)**	**(0.056)**	(0.092)
	[1.30]	**[2.07]**	**[2.79]**	[0.74]
Wet in initial year	−0.028	−0.008	**−0.072**	−0.035
	(0.028)	(0.047)	**(0.032)**	(0.033)
	[1.02]	[0.17]	**[2.21]**	[1.07]
Wet in subsequent years	−0.060	−0.095	−0.063	−0.051
	(0.039)	(0.068)	(0.034)	(0.049)
	[1.53]	[1.39]	[1.82]	[1.03]
N of observations	3784	3784	3784	3784
City & year fixed effects	X	X	X	X
County controls with linear trends	X	X	X	X
Region-year fixed effects	X	X	X	X

PPML regression of mortality rates in levels, 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% (or lower) level.

question how much interpretive weight to place on the non-significance of the results for *wet in subsequent years* in particular, 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.

Tables F3 and F4 replicate these exercises in robustness by substituting the state-by-year fixed effects in Tables F1 and F2 with Census region-by-year fixed effects. This mainly serves to also replicate the results in the preceding tables, albeit with small changes in coefficient values.

Finally, Tables F5 and F6 report the results from the introduction of city linear trends (instead of state linear trends) into our baseline specification for the four aggregated mortality rates and the four acute causes of death, respectively. We exercise caution in interpreting these results and only present them for the sake of completeness as, in general, this particular specification gives rise to frequent problems in estimation. The high number of parameters to be estimated (nearly 2000), the high degree of collinearity among the variables, and the large number of clusters leads to potentially singular VCV matrices and instability in the value of point

Table F5
Repeal's effect on aggregated causes of death, city linear trends.

	(1) Acute	(2) Chronic	(3) Potentially related	(4) Non-related
Dryish in initial year	−0.008	0.020	−0.015	0.013
	(0.025)	(0.012)	(0.016)	(0.022)
	[0.30]	[1.70]	[0.93]	[0.62]
Dryish in subsequent years	−0.048	0.007	−0.048	−0.040
	(0.059)	(0.043)	(0.030)	(0.049)
	[0.81]	[0.17]	[1.58]	[0.81]
Wet in initial year	**−0.041**	−0.013	−0.002	−0.025
	(0.018)	(0.010)	(0.011)	(0.012)
	[2.29]	[1.31]	[0.15]	[1.56]
Wet in subsequent years	**−0.059**	−0.023	0.021	0.006
	(0.026)	(0.017)	(0.017)	(0.020)
	[2.28]	[1.33]	[1.22]	[0.30]
N of observations	3784	3784	3784	3784
City & year fixed effects	X	X	X	X
County controls with linear trends	X	X	X	X
City linear trends	X	X	X	X

PPML regression of mortality rates in levels, 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% (or lower) level.

Table F6
Repeal's effect on acute causes of death, city linear trends.

	(1) Automobile accidents	(2) Homicide	(3) Non-auto accidents	(4) Suicide
Dryish in initial year	0.021	0.056	−0.054	0.074
	(0.052)	(0.056)	(0.049)	(0.073)
	[0.40]	[1.00]	[1.10]	[1.02]
Dryish in subsequent years	−0.003	−0.017	−0.110	0.273
	(0.118)	(0.121)	(0.112)	(0.178)
	[0.03]	[0.14]	[0.98]	[1.54]
Wet in initial year	0.003	0.073	**−0.081**	0.043
	(0.028)	(0.052)	**(0.036)**	(0.039)
	[0.12]	[1.39]	**[2.27]**	[1.10]
Wet in subsequent years	−0.042	0.079	**−0.101**	0.113
	(0.046)	(0.086)	**(0.045)**	(0.066)
	[0.92]	[0.92]	**[2.25]**	[1.72]
N of observations	3784	3784	3784	3784
City & year fixed effects	X	X	X	X
County controls with linear trends	X	X	X	X
City linear trends	X	X	X	X

PPML regression of mortality rates in levels, 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% (or lower) level.

estimates depending on which program and/or routine is used. Thus, we choose to deemphasize these results but are reassured by the close correspondence between the magnitude of the effects for *wet in subsequent years* reported here and in the main text.

APPENDIX G: ROBUSTNESS, VARIATIONS ON THRESHOLD FOR DRYISH

In the text, Table 5 collates the results from various robustness exercises for acute causes of death and non-automobile accidents. Here, we explore the full set of robustness results related to variations in how the threshold for dryish is defined. In particular, we compare the results from our baseline specification where dryish cities are dry cities within 30 km from legal alcohol to those in which this distance is 10, 50, 70, or 90 km.

Table G1 introduces these other thresholds for dryish into our baseline specification for acute causes of death in the total population of cities. Column 1 of Table G1 corresponds with Column 2 of Table 2 (our benchmark specification). Naturally, there is a very high degree of correspondence among all of the results for *wet in subsequent years* in Table G1 as, regardless of what threshold is used, the number of wet cities is large and the number of dryish cities is small. For *dryish in subsequent years*, there is no distinguishable

Table G1
Repeal's effect on acute causes of death, other thresholds for dryish.

	(1) Acute (Dryish <30 km)	(2) Acute (Dryish <10 km)	(3) Acute (Dryish <50 km)	(4) Acute (Dryish <70 km)	(5) Acute (Dryish <90 km)
Dryish in initial year	−0.023	0.000	−0.031	−0.023	−0.019
	(0.024)	(0.026)	(0.022)	(0.022)	(0.021)
	[0.96]	[0.00]	[1.37]	[1.06]	[0.89]
Dryish in subsequent years	**−0.113**	**−0.129**	**−0.101**	−0.069	−0.044
	(0.044)	**(0.045)**	**(0.037)**	(0.039)	(0.038)
	[2.58]	**[2.89]**	**[2.70]**	[1.79]	[1.15]
Wet in initial year	**−0.048**	**−0.042**	**−0.050**	**−0.048**	**−0.048**
	(0.017)	**(0.017)**	**(0.017)**	**(0.017)**	**(0.018)**
	[2.83]	**[2.47]**	**[2.95]**	**[2.80]**	**[2.73]**
Wet in subsequent years	**−0.072**	**−0.063**	**−0.075**	**−0.072**	**−0.071**
	(0.024)	**(0.024)**	**(0.024)**	**(0.024)**	**(0.025)**
	[2.99]	**[2.64]**	**[3.09]**	**[2.96]**	**[2.87]**
N of observations	3784	3784	3784	3784	3784
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	X

PPML regression of mortality rates in levels, 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% (or lower) level.

Table G2
Repeal's effect on non-automobile accidents, other thresholds for dryish.

	(1) Non-auto accidents (Dryish <30 km)	(2) Non-auto accidents (Dryish <10 km)	(3) Non-auto accidents (Dryish <50 km)	(4) Non-auto accidents (Dryish <70 km)	(5) Non-auto accidents (Dryish <90 km)
Dryish in initial year	−0.054	−0.030	**−0.089**	**−0.083**	**−0.071**
	(0.044)	(0.048)	**(0.040)**	**(0.036)**	**(0.034)**
	[1.24]	[0.63]	**[2.23]**	**[2.31]**	**[2.05]**
Dryish in subsequent years	−0.099	**−0.124**	−0.110	−0.108	−0.090
	(0.059)	**(0.058)**	(0.060)	(0.056)	(0.052)
	[1.69]	**[2.16]**	[1.84]	[1.92]	[1.74]
Wet in initial year	**−0.088**	**−0.081**	**−0.099**	**−0.100**	**−0.100**
	(0.034)	**(0.033)**	**(0.034)**	**(0.035)**	**(0.035)**
	[2.63]	**[2.43]**	**[2.91]**	**[2.90]**	**[2.81]**
Wet in subsequent years	**−0.116**	**−0.107**	**−0.128**	**−0.131**	**−0.131**
	(0.041)	**(0.041)**	**(0.042)**	**(0.042)**	**(0.044)**
	[2.81]	**[2.61]**	**[3.07]**	**[3.09]**	**[3.00]**
N of observations	3784	3784	3784	3784	3784
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	X

PPML regression of mortality rates in levels, 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% (or lower) level.

difference across Columns 1, 2, and 3. The effect continues in relative magnitude but not statistical significance once the threshold reaches 70 km. The effect is roughly halved and again statistically insignificant once the threshold reaches 90 km.

Table G2 introduces these other threshold for dryish into our baseline specification for non-automobile accidents in the total population of cities. Column 1 of Table G3 corresponds with Column 3 of Table 3 (our benchmark specification). With respect to *wet in subsequent years* in all columns of Table G3, the magnitude of the effect remains quite consistent across specifications and maintains statistical significance. For *dryish in subsequent years*, the magnitude of the effect remains roughly the same in Columns 2 through 5, skirts the margins of statistical significance in Columns 3 through 5, only becoming statistically significant with a threshold of 10 km in Column 2. Thus, there is some evidence speaking to the robustness of *wet in subsequent years* for non-automobile accidents as it relates to other thresholds for dryish, but again, our benchmark estimate on dryish seems somewhat fragile. In sum, these and other results lead us to down weight *dryish in subsequent years* as a headline result for the paper.

APPENDIX H: ROBUSTNESS, VARIATIONS ON SAMPLE

In the text, Table 5 collates the results from various robustness exercises for acute causes of death and non-automobile accidents. Here, we explore the full set of robustness results related to variations on the sample used for estimation.

Table H1 introduces these other samples into our baseline specification for acute causes of death in the total population of cities. Column 1 of Table H1 corresponds with Column 2 of Table 2 (our benchmark specification). Column 2 of Table H1 expands the number of cities to include those with more than 400,000 inhabitants ($n = 17$) but retains the sample period from 1933 to 1936. Column 3 of Table H1 retains the original sample of cities but expands the sample period to 1939. Column 4 of Table H1 relaxes both of these constraints. For *wet in subsequent years*, there is no substantive difference across Columns 1 through 4. For *dryish in subsequent years*, the effect appreciably declines in magnitude in Columns 3 and 4 nor is it statistically significant.

Table H1
Repeal's effect on acute causes of death, extensions of sample.

	(1) Acute (<1937, <400 K)	(2) Acute (<1937, all cities)	(3) Acute (all years, <400 K)	(4) Acute (all years, all cities)
Dryish in initial year	−0.023	0.009	−0.018	0.006
	(0.024)	(0.037)	(0.024)	(0.033)
	[0.96]	[0.26]	[0.75]	[0.18]
Dryish in subsequent years	**−0.113**	**−0.093**	−0.045	−0.041
	(0.044)	**(0.045)**	(0.028)	(0.030)
	[2.58]	**[2.04]**	[1.58]	[1.34]
Wet in initial year	**−0.048**	**−0.062**	**−0.044**	**−0.064**
	(0.017)	**(0.017)**	**(0.016)**	**(0.016)**
	[2.83]	**[3.64]**	**[2.80]**	**[3.93]**
Wet in subsequent years	**−0.072**	**−0.070**	**−0.077**	**−0.110**
	(0.024)	**(0.028)**	**(0.020)**	**(0.024)**
	[2.99]	**[2.51]**	**[3.94]**	**[4.66]**
N of observations	3784	3852	6622	6741
City & year fixed effects	X	X	X	X
County controls with linear trends	X	X	X	X
State linear trends	X	X	X	X

PPML regression of mortality rates in levels, 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% (or lower) level.

Table H2
Repeal's effect on non-automobile accidents, extensions of sample.

	(1) Non-auto accidents (<1937, <400 K)	(2) Non-auto accidents (<1937, all cities)	(3) Non-auto accidents (all years, <400 K)	(4) Non-auto accidents (all years, all cities)
Dryish in initial year	−0.054	0.022	−0.046	0.022
	(0.044)	(0.077)	(0.039)	(0.063)
	[1.24]	[0.28]	[1.19]	[0.35]
Dryish in subsequent years	−0.099	−0.050	−0.034	−0.011
	(0.059)	(0.069)	(0.046)	(0.050)
	[1.69]	[0.72]	[0.73]	[0.23]
Wet in initial year	**−0.088**	**−0.097**	**−0.060**	**−0.069**
	(0.034)	**(0.033)**	**(0.026)**	**(0.026)**
	[2.63]	**[2.91]**	**[2.26]**	**[2.71]**
Wet in subsequent years	**−0.116**	**−0.098**	**−0.085**	**−0.093**
	(0.041)	**(0.049)**	**(0.027)**	**(0.030)**
	[2.81]	**[1.99]**	**[3.20]**	**[3.16]**
N of observations	3784	3852	6622	6741
City & year fixed effects	X	X	X	X
County controls with linear trends	X	X	X	X
State linear trends	X	X	X	X

PPML regression of mortality rates in levels, 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% (or lower) level.

Table H2 introduces these other samples into our baseline specification for non-automobile accidents in the total population of cities. Column 1 of Table H2 corresponds with Column 3 of Table 3 (our benchmark specification) while Columns 2, 3, and 4 respectively expand the number of cities, expand the sample period to 1939, and relax both of these constraints. With respect to *wet in subsequent years* in Table H2, there is no statistically distinguishable difference across Columns 1, 2, 3, and 4. For *dryish in subsequent years*, the effects dramatically decline in value in Columns 2 through 4. They also tend towards gross statistical insignificance. Again, these and other results lead us to down weight *dryish in subsequent years* as a headline result for the paper.

APPENDIX I: ROBUSTNESS, ALTERNATIVE ESTIMATORS

Here, we implement the Sun and Abraham (2022) estimator to ensure our estimates are not jeopardized by ignoring recent critiques of TWFE. We report the results below in Table I1. In the first column, we simply replicate our baseline estimates for all-acute causes of death using PPML. To aid comparability with what comes next, the second column reports the same specification but using scaled OLS, that is, the log of ((acute causes + 1) / city population). As noted in Appendix B, these two sets of results are virtually indistinguishable, principally for the fact that there are no zero-valued observations for the all-acute variable. The third and fourth columns also use the scaled log of all-acute causes but do so in the context of the Sun and Abraham estimator and not OLS. We put more interpretive weight on column (4) as we believe the Sun and Abraham estimator technically does not allow for multiple treatments.

The results are highly consistent with the baseline and one another.

Table I1
Robustness on TWFE for all-acute causes of death.

	(1) PPML all-acute	(2) OLS logs	(3) Sun & Abraham	(4) Sun & Abraham
Dryish in initial year	−0.023	−0.023	−0.014	
	(0.024)	(0.026)	(0.028)	
	[0.96]	[0.90]	[0.49]	
Dryish in subsequent years	**−0.113**	−0.089	−0.106	
	(0.044)	(0.046)	(0.070)	
	[2.58]	[1.92]	[1.51]	
Wet in initial year	**−0.048**	**−0.049**	**−0.051**	**−0.053**
	(0.017)	**(0.018)**	**(0.020)**	**(0.019)**
	[2.83]	**[2.76]**	**[2.62]**	**[2.74]**
Wet in subsequent years	**−0.072**	**−0.066**	**−0.080**	**−0.083**
	(0.024)	**(0.025)**	**(0.034)**	**(0.034)**
	[2.99]	**[2.62]**	**[2.34]**	**[2.46]**
N of observations	3784	3784	3784	3784
City & year fixed effects	X	X	X	X
County controls with linear trends	X	X	X	X
State linear trends	X	X	X	X

PPML regression of mortality rates in levels, 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% (or lower) level.

APPENDIX J: ROBUSTNESS, Q-VALUES

In order to more formally account for the possibility of false discovery, we can implement proposed corrections in the literature on p-values by authors like Anderson (2008). Thankfully, there is now packaged code in Stata for multiple hypothesis test corrections along these lines. We report the results below in Tables J1 and J2. Materially, these do not alter are take-home message: the "sharpened" q-values are indeed larger than our original p-values for all-acute causes and non-automobile accidents, but they still indicate statistical significance at the 5% level. However, there is a curious drawback to most – if not all – p-value corrections we encountered: namely, they cannot account for correlations among the p-values themselves. As this is not the main focus of our paper, we simply report them here for the sake of completeness.

Table J1
Repeal's effect on aggregated causes of death.

	(1) Acute	(2) Chronic	(3) Potentially related	(4) Non-related
Dryish in initial year	−0.023	0.022	−0.008	0.025
	(0.34)	(0.09)	(0.56)	(0.11)
	[0.51]	[0.29]	[0.71]	[0.33]
Dryish in subsequent years	**−0.113**	0.004	−0.037	0.011
	(0.01)	(0.88)	(0.15)	(0.68)
	[0.05]	[0.92]	[0.38]	[0.77]
Wet in initial year	**−0.048**	−0.010	−0.006	−0.020
	(0.01)	(0.29)	(0.60)	(0.07)
	[0.04]	[0.47]	[0.71]	[0.29]
Wet in subsequent years	**−0.072**	−0.019	0.013	0.003
	(0.00)	(0.24)	(0.45)	(0.88)
	[0.04]	[0.44]	[0.58]	[0.92]
N of observations	3784	3784	3784	3784
City & year fixed effects	X	X	X	X
County controls with linear trends	X	X	X	X
State linear trends	X	X	X	X

PPML regression of mortality rates in levels, weighted by city population. Original p-values in parentheses; Anderson's "sharpened" q-value reported below in brackets. Figures in bold are significant at the 5% (or lower) level.

Table J2
Repeal's effect on acute causes of death.

	(1) Automobile accidents	(2) Homicide	(3) Non-auto accidents	(4) Suicide
Dryish in initial year	−0.004	0.016	−0.054	0.024
	(0.93)	(0.75)	(0.21)	(0.70)
	[0.69]	[0.62]	[0.45]	[0.62]
Dryish in subsequent years	−0.115	**−0.234**	−0.099	0.058
	(0.19)	**(0.00)**	(0.09)	(0.44)
	[0.45]	**[0.04]**	[0.42]	[0.62]
Wet in initial year	−0.006	−0.040	**−0.088**	0.022
	(0.82)	(0.39)	**(0.01)**	(0.55)
	[0.62]	[0.62]	**[0.04]**	[0.62]
Wet in subsequent years	−0.059	−0.108	**−0.116**	0.079
	(0.17)	(0.16)	**(0.00)**	(0.20)
	[0.45]	[0.45]	**[0.04]**	[0.45]
N of observations	3784	3784	3784	3784
City & year fixed effects	X	X	X	X
County controls with linear trends	X	X	X	X
State linear trends	X	X	X	X

PPML regression of mortality rates in levels, weighted by city population. Original p-values in parentheses; Anderson's "sharpened" q-value reported below in brackets. Figures in bold are significant at the 5% (or lower) level.

APPENDIX K: ROBUSTNESS, SUCCESSIVE ADDITION OF CONTROLS

Here, we consider the possibility that our identifying assumption that selection on unobservable is not driving our results is somehow violated. We do so by considering the successive addition of our controls, starting from a bare-bones TWFE model. Our reported results are highly stable across the successive inclusion of controls and fixed effects as seen in Table K1 below. In short, the exclusion/inclusion of controls beyond TWFE do not seem to have much bearing on our results.

Table K1
Repeal's effect on acute causes of death, aggregated, successive controls.

	(1) Baseline	(2) TWFE	(3) (2) + Retail	(4) (3) + Institutions	(5) (4) + Hospital beds
Dryish in initial year	−0.023	−0.040	−0.040	−0.040	−0.040
	(0.024)	(0.023)	(0.022)	(0.022)	(0.022)
	[0.34]	[1.78]	[1.80]	[1.81]	[1.81]
Dryish in subsequent years	**−0.113**	**−0.115**	**−0.116**	**−0.116**	**−0.116**
	(0.044)	**(0.034)**	**(0.040)**	**(0.041)**	**(0.041)**
	[2.58]	**[3.36]**	**[2.87]**	**[2.85]**	**[2.85]**
Wet in initial year	**−0.048**	**−0.045**	**−0.047**	**−0.047**	**−0.047**
	(0.017)	**(0.015)**	**(0.016)**	**(0.016)**	**(0.016)**
	[2.83]	**[2.89]**	**[3.04]**	**[3.03]**	**[3.03]**
Wet in subsequent years	**−0.072**	**−0.066**	**−0.073**	**−0.072**	**−0.072**
	(0.024)	**(0.020)**	**(0.020)**	**(0.020)**	**(0.020)**
	[2.99]	**[3.34]**	**[3.65]**	**[3.63]**	**[3.63]**
N of observations	3784	3784	3784	3784	3784
City & year fixed effects	X	X	X	X	X
County controls with linear trends	X				
State linear trends	X				

	(6) (5) + Black (trend)	(7) (6) + Foreign (trend)	(8) (7) + Baptist (trend)	(9) (8) + Unemp. (trend)	(10) (9) + ND exp. (trend)
Dryish in initial year	−0.041	−0.037	−0.037	−0.037	−0.037
	(0.022)	(0.023)	(0.023)	(0.023)	(0.023)
	[1.83]	[1.63]	[1.64]	[1.64]	[1.63]
Dryish in subsequent years	**−0.120**	**−0.113**	**−0.114**	**−0.114**	**−0.114**
	(0.041)	**(0.040)**	**(0.040)**	**(0.039)**	**(0.039)**
	[2.95]	**[2.83]**	**[2.89]**	**[2.91]**	**[2.90]**
Wet in initial year	**−0.046**	**−0.044**	**−0.044**	**−0.044**	**−0.044**
	(0.016)	**(0.016)**	**(0.016)**	**(0.016)**	**(0.016)**
	[2.89]	**[2.75]**	**[2.80]**	**[2.80]**	**[2.78]**
Wet in subsequent years	**−0.068**	**−0.059**	**−0.060**	**−0.060**	**−0.060**
	(0.021)	**(0.021)**	**(0.021)**	**(0.021)**	**(0.021)**
	[3.20]	**[2.80]**	**[2.88]**	**[2.88]**	**[2.85]**
N of observations	3784	3784	3784	3784	3784
City & year fixed effects	X	X	X	X	X
County controls with linear trends					X

PPML regression of mortality rates in levels, weighted by city population. Standard errors are in parentheses and clustered at the city level; t-statistics reported below standard errors in brackets.

References

Acemoglu, D., Autor, D.H., Lyle, D., 2004. Women, war, and wages: the effect of female labor supply on the wage structure at midcentury. J. Polit. Econ. 112 (3), 497–551.

Ager, P., Feigenbaum, J.J., Hansen, C.W., Tan, H.R., 2023. How the other half died: immigration and mortality in US cities. Rev. Econ. Stud. forthcoming.

Anderson, D.M., Charles, K.K., Rees, D.I., 2022. Re-examining the contribution of public health efforts to the decline in urban mortality. Am. Econ. J.: Appl. Econ. 14 (2), 126–157.

Asbury, H., 1950. The Great Illusion: An Informal History of Prohibition. Doubleday and Company, Garden City.

Behr, E., 1996. Prohibition: 13 Years that Changed America. Arcade Publishing, New York.

Blocker, J.S., 2006. "Did prohibition really work? Alcohol Prohibition as a public health innovation. Am. J. Public Health 96 (2), 233–243.

Blum, D., 2010. The Poisoner's Handbook: Murder and the Birth of Forensic Medicine in Jazz Age New York. Penguin Press, New York.

Childs, R.W., 1947. Making Repeal Work. Penn Alcoholic Beverage Study, Philadelphia.

Clark, N.H., 1965. The Dry Years. University of Washington Press, Seattle.

Cook, P., 2007. Paying the Tab: The Costs and Benefits of Alcohol Control. Princeton University Press, Princeton.

Culver, D.C., Thomas, J.E., 1940. State Liquor Control Administration. Bureau of Public Administration, University of California-Berkeley.

Cutler, D., Miller, G., 2005. The role of public health improvements in health advances: the twentieth-century United States. Demography 42 (1), 1–22.

Dighe, R.S., 2010. Pierre S. du Pont and the making of an anti-prohibition activist. Soc. Hist. Alcohol Drugs 24 (2), 97–118.

Distilled Spirits Institute (various years), *Annual Report of the Distilled Spirits Institute*. Washington, DC: Distilled Spirits Institute.

Distilled Spirits Institute, 1935. Summary of State Liquor Control Laws and Regulations Relating to Distilled Spirits. Distilled Spirits Institute, Washington, DC.

Distilled Spirits Institute, 1941. Summary of State Liquor Control Laws and Regulations Relating to Distilled Spirits. Distilled Spirits Institute, Washington, DC.

Endersby, J.W., 2012. Prohibition and repeal: voting on statewide liquor referenda in Texas. Soc. Sci. J. 49, 503–512.

Feigenbaum, J.J., Muller, C., 2016. Lead exposure and violent crime in the early twentieth century. Explor. Econ. Hist. 62, 51–86.

Fishback, P.V., Haines, M.R., Kantor, S., 2007. Births, deaths, and new deal relief during the great depression. Rev. Econ. Stat. 89 (1), 1–14.

Fishback, P.V., Johnson, R.S., Kantor, S., 2010. Striking at the roots of crime. J. Law Econ. 53 (4), 715–740.
Fisher, I., 1927. The economics of prohibition. Am. Econ. Rev. Papers Proc. 17 (1), 5–10.
Fosdick, R.B., Scott, A.L., 1933. Toward Liquor Control. Harper & Brothers, New York.
Galofré Vilà, G., 2020. Quantifying the impact of aid to dependent children. Explor. Econ. Hist. 77 (3), 101332.
Galofré Vilà, G., McKee, M., Stuckler, D., 2022. Quantifying the mortality impact of the 1935 Old-age assistance. Eur Rev Econ Hist 26 (1), 62–77.
Garcia-Jimeno, C., 2016. The political economy of moral conflict: an empirical study of learning and law enforcement under prohibition. Econometrica 84 (2), 511–570.
Gibson, C., Jung, K., 2002. Historical census statistics on population totals by race, 1790 to 1990, and by Hispanic Origin, 1970 to 1990, for the United States, Regions, Divisions, and States. Population Division Working Paper 56.
Gordon, T., 1953. Mortality in the United States, 1900-1950. Public Health Rep. 68 (4), 441–444.
Granados, J.A.T., Roux, A.V.D., 2009. Life and death during the great depression. Proc. Natl Acad. Sci. 106 (41), 17290–17295.
Harper, J.W., 2016. A spirited revolution: local option elections and the impending death of prohibition in Arkansas. Univ. Arkansas Little Rock Law Rev. 38 (3), 527–557.
Harrison, L.V., 1938. The Local Option Fallacy. Distilled Spirits Institute, Washington, DC.
Harrison, L.V., Laine, E., 1936. After Repeal, A Study of Liquor Control Administration. Harper, New York.
Hernandez, C.E. (2016), "Adaptation and survival in the brewing industry during prohibition." https://www.aeaweb.org/conference/2017/preliminary/paper/SFDQizbG
Hoynes, H.W., Schanzenbach, D.W., 2012. Work incentives and the food stamp program. J. Public Econ. 96 (1), 151–162.
Jacks, D.S., Pendakur, K., Shigeoka, H., 2021. Infant mortality and the repeal of federal prohibition. Econ. J. 131 (639), 2955–2983.
Jayachandran, S., Lleras-Muney, A., Smith, K.V., 2010. Modern medicine and the twentieth century decline in mortality: evidence on the impact of sulfa drugs. Am. Econ. J.: Appl. Econ. 2 (2), 118–146.
Johnson, B.F., 2005. John Barleycorn Must Die: The War Against Drink in Arkansas. University of Arkansas Press, Fayetteville.
Kyvig, D.E., 2000. Repealing National Prohibition. Kent State University Press, Ashland.
LaVallee, R.A., Yi, H., 2011. Apparent per capita alcohol consumption: national, state, and regional trends, 1977-2009. NIAAA Surveillance Report 92.
Law, M.T., Marks, M.S., 2020. Did early twentieth century alcohol prohibition affect mortality? Econ. Inq. 58 (2), 680–697.
Legislative Reference Library of Texas (1919), *Acts 1919, 36th Regular Session, House Joint Resolution 1*. https://lrl.texas.gov/LASDOCS/36R/HJR1/HJR1_36R.pdf
Linder, F.E., Grove, R.D., 1947. Vital Statistics of the United States 1900-1940. Government Printing Office, Washington, DC.
Manning, W.G., Mullahy, J., 2001. Estimating log models: to transform or not to transform? J. Health Econ. 20, 461–494.
McDermott, R., 2013. Alcohol flows through city's history. Texarkana Gazette Dec. 1.
McGirr, L., 2016. The War on Alcohol. W.W. Norton, New York.
Mullahy, J., Norton, E.C., 2022. Why Transform Y? In: NBER Working Paper, p. 30735.
Murdock, C.G., 1998. Domesticating Drink. Johns Hopkins University Press, Baltimore.
Norris, C., 1928. Our essay in extermination. North Am Rev 226 (6), 645–652.
Okrent, D., 2010. Last Call: The Rise and Fall of Prohibition. Scribner, New York.
Owens, E.G., 2011. Are underground markets really more violent? Evidence from early 20th Century America. Am. Law Econ. Rev. 13 (1), 1–44.
Owens, E.G., 2014. The American temperance movement and market-based violence. Am. Law Econ. Rev. 16 (2), 433–472.
Pennock, P.E., Kerr, K.A., 2005. In the Shadow of Prohibition: domestic American Alcohol Policy since 1933. Bus. Hist. 47 (3), 383–400.
Pew Research Center (2015), "Multiracial in America: proud, diverse and growing in numbers." Washington, DC.
Poelmans, E., Taylor, J.E., Raisanen, S., Holt, A.C., 2022. Employment gains attributable to beer legalization in spring 1933. Explor. Econ. Hist. 84, 101427.
Rorabaugh, W.J., 2018. Prohibition: A Concise History. Oxford University Press, Oxford.
Rose, K.D., 1996. American Women and the Repeal of Federal Prohibition. New York University Press, New York.
Schrad, M.L., 2021. Smashing the Liquor Machine. Oxford University Press, Oxford.
Sechrist, R.P., (1983), "Prohibition movement in the United States, 1801-1920." Interuniversity Consortium for Political and Social Research.
Shaffer, J.P., 1995. Multiple hypothesis testing. Annu. Rev. Psychol. 46, 561–584.
Skorobogatov, A.S., 2021. The effect of alcohol sales restrictions on alcohol poisoning mortality. Health Econ. 30 (6), 1417–1442.
Texas Alcohol Beverage Commission (various years), *TABC Annual Report*. Austin: TABC.
Texas Legislative Council (2022), "Amendments to the Texas Constitution Since 1876." https://tlc.texas.gov/docs/amendments/Constamend1876.pdf
Thomas, J.E., Culver, D.C., 1940. Protection of dry areas. Law Contemp. Probl. 7 (4), 696–708.
Thomasson, M.A., Treber, J., 2008. From Home to hospital: the evolution of childbirth in the United States, 1928–1940. Explor Econ Hist 45 (1), 76–99.
US Bureau of the Census, 1931. Manual of the International List of Causes of Death Based On the Fourth Decennial Revision by the International Commission, Paris, October 16 to 19, 1929. Government Printing Office, Washington, DC.
US Bureau of the Census, 1935. Negro Population, 1930: A Listing of the 695 Cities and Urban Places Having 1,000 Or More Negro Inhabitants. GPO, Washington, DC.
Warner, K.E., 1985. Cigarette Advertising and media coverage of smoking and health. N. Engl. J. Med. 312, 384–388.
World Health Organization (2019), *International statistical classification of diseases and related health problems*, 10th revision. https://icd.who.int/browse10/2019/en