Milk Pasteurization and Mortality

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Abstract: Milk was a major disease vector in the early twentieth century, yet the population-level effects of milk safety reforms remain poorly understood. In this paper, we examine the rollout of pasteurization ordinances across U.S. cities and show that pasteurization led to large, sustained improvements in public health. City-level event studies indicate that these ordinances reduced milkborne mortality by 16%, averting approximately 800–1,200 deaths annually. Pasteurization specifically cut typhoid morbidity and mortality by 32-34%, with the largest gains among older children and adults. These declines in milkborne mortality highlight pasteurization as a crucial yet undervalued driver of early 20th-century mortality declines.

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1 Introduction

The rapid decline in infectious disease mortality during the early twentieth century was one of the most important improvements in American living standards (Higgs, 1973; Preston, 1975; Fogel, 1994; Cutler et al., 2006). Economists have linked these changes in the disease environment to higher productivity, faster urbanization, and gains in human capital (Cutler and Miller, 2005; Bleakley, 2010, 2007; Acemoglu and Johnson, 2007; Beach et al., 2016; Beach, 2022; Hoehn-Velasco, 2021). Yet at the turn of the century, American cities remained deeply unhealthy places to live (Haines, 2001). Food- and waterborne illnesses were especially pervasive, reflecting contaminated municipal water systems, inadequate sewage treatment, and long, complex food distribution networks that lacked refrigeration.

Over the following decades, however, mortality from preventable diseases fell sharply as municipal sanitation improved and households benefited from higher incomes, better nutrition, and improved housing (Higgs, 1973; Cutler and Miller, 2005; Anderson et al., 2022; Fogel et al., 2004). One illustration of this mortality transformation is the dramatic decline in typhoid fever. Typhoid mortality fell from 31.3 deaths per 100,000 in 1900 to 1.0 by 1940 (Grove and Hetzel, 1968).[1] This thirty-fold reduction is typically attributed to large-scale water infrastructure, particularly filtration and chlorination (Cutler and Miller, 2005; Beach et al., 2016; Beach, 2022). Yet these well-documented water reforms were only one piece of broader early twentieth-century public health investments.

Like water, milk was widely consumed and distributed in urban areas, making it a prime vehicle for disease transmission. A 1927 public health report documented over 600 milkborne outbreaks, 80 percent of which were attributed to typhoid fever (Armstrong and Parran, 1927). As late as 1938, milk accounted for roughly 25% of all food- and water-borne disease outbreaks (Clark and Harte, 2021). Reformers warned of milk's particular danger to infants, estimating that poor milk quality was responsible for "killing tens of thousands of infants each year" (Ward et al., 2007, pg. 138). As one account put it, "Of all the factors of man's environment, none is more important to his welfare than food. Of all foods, none is more important than milk" (Andrews and Fuchs, 1944, pg. 189). Despite this widespread recognition of milk as a major disease vector, there is less systematic evidence on the aggregate health effects of pasteurization, the most effective milk safety intervention (Straus and Straus, 1913). Prior work has emphasized water filtration and chlorination as the central drivers of early 20th-century health improvements (Troesken, 1999, 2001; Cutler and Miller, 2005; Beach et

[1] See Grove and Hetzel (1968), Table 65.

al., 2016; Anderson et al., 2022; Beach, 2022), but the contribution of clean milk (especially pasteurization) remains comparatively understudied, despite notable contributions from Komisarow (2017); Anderson et al. (2022, 2025).

We address this knowledge gap by examining the public health impact of mandatory milk pasteurization. Over the early 20th century, cities began enacting milk pasteurization laws that mandated the pasteurization of city milk supplies. To evaluate the impacts of these mandates, we use an event-study design spanning 1905 to 1936. This approach leverages variation in ordinance timing across cities and enables us to track the dynamic mortality response to pasteurization, providing one of the first population-level analyses of pasteurization's effects.[2]

Our findings reveal that pasteurization was an important driver of improvements in urban health, with several notable gains in population health attributable to its adoption. First, pasteurization improved milk safety broadly. A composite measure of milkborne mortality, including typhoid, scarlet fever, and non-pulmonary tuberculosis, shows declines in mortality by roughly 16 percent. Counterfactual estimates suggest that this decline corresponds to 800–1,200 averted milkborne deaths each year. We also complement the milkborne mortality analysis with newly digitized data on milkborne outbreaks. We find parallel reductions in documented and traced milkborne outbreaks following the adoption of a pasteurization ordinance.

Second, because much of the literature has focused on reductions in typhoid fever (Beach et al., 2016; Anderson et al., 2022), and because typhoid accounted for nearly 80 percent of documented milkborne outbreaks (Armstrong and Parran, 1927), we also focus on typhoid specifically. We find that pasteurization ordinances reduced typhoid morbidity and mortality by approximately 32-34%. Counterfactual estimates indicate that these mandates prevented 150–300 deaths and 1,500–3,200 cases of typhoid per year. The magnitude of the relative decline in typhoid places pasteurization alongside water filtration among the most effective early twentieth-century public health interventions. While filtration reduced typhoid by about 36 percent (Anderson et al., 2022), pasteurization produced a nearly comparable 32-34 percent reduction. Although the largest proportional effect appears for typhoid, pasteurization's broadest population impact comes from reducing a wide range of milkborne illnesses that burdened cities in this era.

Third, we also find that pasteurization's impact differed by age. The largest declines in typhoid and milkborne mortality occurred among adults, but older children also show some decline in milkborne mortality. By contrast, we find little evidence

[2]Pasteurization has also been demonstrated to be important in two unpublished prior studies, Wang et al. (2014) and Wahlers (2018). Note that Wang et al. (2014) was eventually published as Wang (2016) and focused only on Chicago.

that mandates reduced infant mortality, even though infants were historically viewed as most vulnerable. A possible explanation for infant mortality not responding to pasteurization mandates is that parents fed infants boiled, pasteurized, or certified milk before the ordinances went into effect. These voluntary practices would have insulated infants from unsafe milk. To test this hypothesis, we analyze variation in pasteurization rates across cities. We find suggestive evidence that increases in the supply of pasteurized milk, whether mandated or not, are associated with declines in infant mortality. The importance of the levels of pasteurized milk highlights the interplay between household-level decisions and public regulation in shaping health outcomes.

Our findings make several important contributions to the literature. First, we show that pasteurization was a major, underappreciated component of early twentieth-century public health progress. While the literature has extensively documented the transformative role of water filtration and chlorination (Troesken, 1999, 2001; Cutler and Miller, 2005; Beach et al., 2016; Anderson et al., 2022; Beach, 2022), clean milk programs have received less attention. Existing evidence on milk safety interventions is mixed: Anderson et al. (2025) finds that milk inspections reduced typhoid, while Komisarow (2017) and Anderson et al. (2022) report less apparent effects of clean milk beyond a reduction in diarrheal mortality for one-year-olds in Komisarow (2017). We contribute by focusing on pasteurization, the intervention contemporaries regarded as the most important safeguard against contaminated milk (Straus and Straus, 1913). Our results demonstrate that pasteurization was a high-impact public health program, improving milk safety broadly and preventing hundreds of milkborne deaths annually. In doing so, we elevate pasteurization to close to the importance of clean water in shaping the early twentieth-century disease environment.[3]

We also provide noteworthy new evidence on the impact of public health regulations on typhoid case rates, not just mortality. While deaths from typhoid declined in the 1920s, cities continued to report hundreds of cases annually. For instance, in 1929, New York City reported 586 cases but only 75 deaths. These case rates offer insight into ongoing disease transmission, particularly as populations became healthier and less likely to die from infection (Higgs, 1973; Fogel, 1994). In this context of improving population health, morbidity may be a more sensitive measure of policy effectiveness than mortality alone.

Our results also speak to broader debates over the causes of the mortality transition. Consistent with earlier work, we find that public health played a role in reducing specific causes of death (Troesken, 1999; Haines, 2001; Cutler and Miller, 2005;

[3]Related work shows that substitutes for unsafe water, such as tea (Antman, 2023) or beer (Antman and Flynn, 2023), reduced mortality, underscoring the broad value of safer beverage consumption options.

Bleakley, 2010; Kitchens, 2013; Moehling and Thomasson, 2014; Hoehn-Velasco, 2018; Anderson et al., 2022; Ager et al., 2023). However, we find little evidence that pasteurization mandates affected all-cause mortality, overall child mortality, or infant mortality, suggesting complementary explanations, such as rising incomes, better nutrition, or improved living conditions (McKeown and Record, 1962; Fogel, 1997; Fogel et al., 2004; Anderson et al., 2022).

Finally, our findings remain relevant to contemporary debates. In the United States today, pasteurization continues to face resistance from advocates of raw milk, who argue that pasteurization diminishes the nutritional value and taste of milk (Dickson, 2024; Greenfield, 2025). Public discourse has also increasingly promoted raw milk consumption (Magnoli, 2025; MassLive, 2025; Dickson, 2025) and challenges within the current regulatory framework raise concerns about the capacity to effectively ensure milk safety (Douglas, 2025; Deutsch, Chris, 2025).

While typhoid fever is no longer a major concern in modern milk safety debates, other serious pathogens, such as Campylobacter, Cryptosporidium, E. coli, Listeria, and Salmonella, remain strongly linked to the consumption of raw, unpasteurized dairy products (Centers for Disease Control and Prevention, 2025a,b). Accordingly, official public-health guidelines continue to emphasize the essential role of pasteurization in preventing illness (FDA, 2011). These modern tensions mirror early 20th-century debates over milk oversight and pasteurization (Ayers, 1916; Ward et al., 2007). By revisiting this earlier period, our study provides historical evidence of pasteurization's effectiveness in reducing foodborne disease and underscores pasteurization's importance in the evolution of modern public health.

2 Background

2.1 The Milk Problem

2.1.1 Contaminated Urban Milk Supplies

At the turn of the twentieth century, milk safety posed a serious public health challenge to regulators and consumers (Ward et al., 2007). Milk was transported without refrigeration from unhygienic dairy processing farms and was often riddled with bacteria by the time it arrived at urban milk dealers (North, 1921b; Preston and Haines, 1991). When the milk ultimately reached consumers, it was two to three days old and had passed through many (unwashed) hands in unsealed containers (Straus and Straus, 1913; Ward et al., 2007).

Milk was vulnerable to contamination at every stage of its journey, from the cow to the consumer. On the farm, unsanitary conditions, unhygienic handling practices, and infected cows were common sources of initial contamination. Farmers and milkers were often covered in dirt and animal waste, and handwashing was rare (Ward et al., 2007). Disease transmission was especially likely when a handler was a carrier of an illness, such as typhoid fever, which could be shed into the milk supply without symptoms or detection (Chapin, 1917; Armstrong and Parran, 1927; Ward et al., 2007). Despite the availability of carrier testing, typhoid carriers were a recurring source of contaminated milk supplies (Armstrong and Parran, 1927). Filthy barn environments only amplified these risks, making raw milk a major public health hazard (Ward et al., 2007).

Beyond the farm, contamination could occur during transport and distribution. Milk was frequently handled by intermediaries or consumers who were themselves typhoid carriers (Chapin, 1917; Armstrong and Parran, 1927; Ward et al., 2007). Infected or unclean water sources were another frequent cause of milkborne typhoid outbreaks, as milk containers and equipment were often rinsed or diluted with contaminated water (Ward et al., 2007; Beach et al., 2016). Cost pressures also encouraged adulteration: both dealers and consumers routinely added water to stretch milk supplies, increasing the risk of contamination (Ward et al., 2007).

A further cause of contamination was consumer sampling of milk at purchase (Ward et al., 2007). Milk was often sold in open vats, and consumers would dip their fingers or reuse ladles into the milk before purchase (Ward et al., 2007). The milk that was eventually sold to consumers was often so dirty and discolored by the production and sale process that dealers would add chalk to retain the milk's white color (Ward et al., 2007).

2.1.2 Milkborne Outbreaks and Illness

The contaminated milk supply was directly tied to disease outbreaks. Bacteria flourished in the milk brought from unsanitary farming conditions and through multiple handlers (Armstrong and Parran, 1927). Through these numerous sources of contamination, milk carried various pathogens, including dysentery, scarlet fever, diphtheria, tuberculosis, typhoid fever, septic sore throat (streptococcus), and hoof-and-mouth disease (Ayers, 1916, 1932; North, 1921a).

Raw milk was a well-documented source of disease outbreaks in the early twentieth century, particularly in the summer months when lack of refrigeration accelerated bacterial growth (Armstrong and Parran, 1927). A 1913 bulletin recorded 317 typhoid outbreaks, 125 cases of scarlet fever, and 51 cases of diphtheria linked directly to contaminated milk supplies (Straus and Straus, 1913). A subsequent 1920 report found even more: 375 typhoid outbreaks, 128 outbreaks of scarlet fever, 55 outbreaks of diphtheria, and 22 outbreaks of septic sore throat traced to milk consumption (North, 1921a). Notably, even after pasteurization became widespread, the U.S. Public Health Service reported 170 milkborne typhoid outbreaks, 100 outbreaks of scarlet fever or septic sore throat, 95 outbreaks of gastroenteritis, and 43 outbreaks from other illnesses between 1932 and 1940 (Andrews and Fuchs, 1944). From these outbreaks, typhoid fever was the most frequently reported illness traced to milk, followed by scarlet fever, with diphtheria and septic sore throat playing a smaller but still significant role (Armstrong and Parran, 1927).

To visualize the historical importance of typhoid, we compile nationwide milkborne outbreak data from Armstrong and Parran (1927), a U.S. Public Health Service report that documents milkborne outbreaks in the United States up to 1926. Although the underlying investigations list outbreaks by city and state, the compilation itself is national in scope, encompassing incidents from large cities, small towns, and rural areas. Figure I summarizes the outbreaks reported over 1906–1926 by disease type. Typhoid fever overwhelmingly dominates milkborne outbreaks during this period, accounting for nearly 80 percent of all traced incidents. Other illnesses, including scarlet fever, diphtheria, and septic sore throat, appear far less frequently and jointly comprise the remaining 20 percent. Consistent with Armstrong and Parran (1927), typhoid was the most common, best-documented, and most reliably traced milkborne disease of the early twentieth century, motivating our focus on typhoid mortality in the analysis.[4]

In addition to these specific causes of death, the cleanliness of milk was also considered imperative for child mortality. The milk supply was a known cause of infant and young child mortality and was associated with "killing tens of thousands of infants each year" (Ward et al., 2007, pg. 138). While physicians and public health advocates strongly advised breastfeeding, even in the nineteenth century, breastfeeding was not always possible for mothers, and many children were artificially fed (Straus and Straus, 1913; Woodbury, 1926). In the period before the advent of formula and fully clean water supplies, cow's milk was the next best substitute for breastmilk (Straus and Straus, 1913). Clean milk was considered paramount for young children, and children under five consumed a large portion of their calories from milk (Preston and Haines, 1991). Children frequently died from gastrointestinal illnesses passed

[4] In the appendix (Figure A.3), we also show outbreaks by major cities. Philadelphia and Detroit experienced sharp declines in milkborne outbreaks by the 1910s, while New York continued to experience recurring incidents throughout the period.

through the milk supply, with more than 50 percent of infant deaths occurring from diarrheal infections (Preston and Haines, 1991). In fact, clean milk was so important for children and infants that it was deemed more consequential than improving water supplies in the quest to reduce child mortality (North, 1921a; Preston and Haines, 1991).

2.2 Milk Safety Regulations Before Pasteurization

The scale and severity of milkborne disease prompted cities to experiment with a wide range of regulatory tools well before pasteurization became widespread. In the late nineteenth and early twentieth centuries, municipalities adopted minimum quality standards to deter adulteration of the milk supply (Meckel, 1990; Anderson et al., 2025), mandated tuberculin testing of herds to combat bovine tuberculosis (Olmstead and Rhode, 2004; Czaplicki, 2007; Palmer et al., 2011; Anderson et al., 2022), conducted periodic inspections of farms, depots, and city milk dealers (Perry, 1915; Komisarow, 2017; Anderson et al., 2025), and introduced bacteriological standards that set maximum allowable bacterial counts in raw and pasteurized milk (Fuchs et al., 1939; Anderson et al., 2022). Most cities in the early twentieth century had established some form of milk regulation.

The empirical evidence on the effectiveness of these early regulations reflects this heterogeneity. Anderson et al. (2025) finds that nineteenth-century minimum quality standards and inspections reduced water- and food-borne disease mortality, underscoring that milk regulation could be effective. In contrast, Anderson et al. (2022) shows that early twentieth-century bacteriological standards and tuberculosis testing did not produce measurable declines in mortality. Similarly, Komisarow (2017) demonstrates that twentieth-century milk laws reduced mortality from diarrhea and enteritis among one-year-old children, but found limited effects on other types of mortality. These mixed results are consistent with the practical limitations documented by contemporaries: inspections were costly and infrequent, bacteriological standards were often aspirational rather than binding, and none of these measures fully addressed contamination introduced during transport, distribution, or household handling (Straus and Straus, 1913; Perry, 1915; Chapin, 1917; Parker, 1917; Fuchs et al., 1939). Against this backdrop of partial and uneven regulation, pasteurization emerged as the intervention that public health authorities increasingly viewed as essential for breaking the transmission of typhoid and other milkborne diseases (Straus and Straus, 1913).

2.3 Pasteurization

2.3.1 Early Years: The Voluntary Adoption of Pasteurization

The process of pasteurization was first invented by the French bacteriologist Louis Pasteur in the 1860s for the purpose of purifying contaminated wine (Ayers, 1916, 1932). Although the method was established in the 19th century, milk pasteurization did not become widespread in the United States until the early 20th century (Ayers, 1932). Before milk was commercially pasteurized, some individuals heated milk at home. The practice of heating milk at home began in the late nineteenth century, as early as the 1880s (North, 1921b; Preston and Haines, 1991). Physician Abraham Jacobi was among the first public health advocates to recommend heating milk for infants and young children (Ayers, 1932). He urged the distribution of pasteurized milk to low-income families in the late 19th century (North, 1921a). In part due to his influence, public health campaigns focused on encouraging mothers to heat milk at home, in the period before commercially pasteurized milk was available (Ward et al., 2007). These early efforts placed the responsibility for milk safety on consumers rather than the dairy industry. Examples of these public health messages are shown in Figure A.1.

In the early twentieth century, public health authorities began to increasingly view commercial pasteurization as essential. Advocates emphasized that pasteurization benefited not only infants and children but also reduced outbreaks of milkborne illness in general (Straus and Straus, 1913). Specifically, pasteurization could prevent typhoid, scarlet fever, diphtheria, and other milkborne diseases (Straus and Straus, 1913). The process of commercial pasteurization purifies contaminated milk by heating the liquid to a specified temperature for a certain period of time to kill all pathogenic bacteria that may be present (Ayers, 1932). The liquid is then quickly cooled to prevent further growth of any bacteria (Ayers, 1932).[5] Pasteurization also offers economic advantages: at the time, it was a low-cost intervention, estimated to cost under half a cent per gallon in 1922 (McCullough, 1928). It also extended the milk's shelf life, making it attractive to both regulators and milk distributors (Ayers, 1922, 1932).

Commercially pasteurized milk became available in Cincinnati, New York, Philadel-

[5]In 1916, the gold standard for pasteurization was to heat milk to a temperature of 145 degrees for 30 minutes, followed by a rapid cooling process (Ayers, 1916). While pathogens would perish at 140 degrees, the standard of heating milk to 145 degrees was set to ensure a paramount of safety (Ayers, 1916). This temperature requirement was lowered to 142 degrees in 1932 (Ayers, 1932). When cities began regulating the milk supply, municipalities passed regulations focusing on pasteurization definitions and standards. This was because pasteurization was not foolproof; milk could be heated to too low a temperature for too little time or left too warm after heating. Cities' pasteurization regulations not only specified which milk had to be pasteurized but also included specifications for the required methods (Ayers, 1916, 1922).

phia, and Chicago just before 1900 (Ayers, 1932). Then, it spread to Saint Louis in 1900, and Boston and Chicago in 1908 (Ayers, 1932). From 1905 onward, the process of pasteurization gained traction in U.S. cities (Ayers, 1916). During this period, prior to pasteurization ordinances, city supplies gradually shifted to include more pasteurized milk. In 1905, New York City's milk supply was nearly untouched by pasteurization, but by the mid-1910s, 60 percent was pasteurized. By 1917, 100 percent was pasteurized, after an initial 1912 pasteurization ordinance that was strengthened in 1914 (Boudouin, 1918). Boston was similar to New York in that at the turn of the twentieth century, Boston had almost no pasteurized milk, but in 1915 (before a formal ordinance was passed), over 80 percent of the milk supply was pasteurized (Ayers, 1916; Boudouin, 1918).

Prior to pasteurization mandates, many cities were already implementing some milk control measures, including setting standards for the bacterial content of milk and conducting milk inspections (Anderson et al., 2022, 2025). These milk control measures were not randomly adopted; the majority of large cities adopted milk regulations first. By the 1920s, most large cities had established milk requirements, which varied from pasteurization to grading, tuberculosis testing, inspections, and bacteriological standards for milk (see Appendix Section B for more details on these additional milk regulations). While pasteurization was considered the best and cheapest regulation to protect milk supplies, in 1921, pasteurization was only widely practiced by large cities (Association et al., 1921).

Ensuring the cleanliness of the milk supply was also a more pressing issue in large cities because there were many more suppliers and intermediaries to regulate, and these milk producers were spread over vast distances. For example, in New York City, "*milk came from 44,000 farms in six states and was the product of about 350,000 cows. Some of it had to be transported 400 miles or even more. It was estimated that 127,000 people were engaged daily in handling the milk supply*" (Ayers, 1916, pg. 15).

As a result of the efforts to improve the milk supply in cities, by 1936, most milk-borne outbreaks occurred in small towns with under 10,000 inhabitants (Fuchs et al., 1939). In large cities (>500k), more than 97 percent of the milk supply was pasteurized; in medium cities (100k-499k), 86 percent of the milk supply was pasteurized, and 72 percent was pasteurized in smaller cities (25k-99k) (Fuchs et al., 1939). In cities and towns under 25k, the 1936 pasteurization rates varied from 24.5 percent (1k-2k population) to 58.2 percent (10k-25k population) (Fuchs et al., 1939). The 1936 estimates illustrate the considerable variation in pasteurization rates even toward the end of this study. However, one theme is clear: large cities generally achieved mass pasteurization, while residents in sparsely populated areas were more likely to drink unpasteurized milk supplies.[6]

2.3.2 Opponents to Pasteurization

Despite its public health benefits, pasteurization faced significant opposition from various groups. Critics feared that the process destroyed important enzymes, reduced digestibility, and altered the natural flavor of milk (Ayers, 1916). Some claimed that pasteurization contributed to nutritional deficiencies such as scurvy and rickets (Ward et al., 2007). Some of these concerns had merit, as pasteurization reduces the Vitamin C content in milk; however, public health officials recommended the inclusion of oranges in the diet to prevent scurvy (Ayers, 1932). Public health officials also expressed concern that milk producers might relax their sanitary practices, relying on pasteurization to "clean up" otherwise unsanitary conditions, a concern supported by observational evidence (Ayers, 1916).

Cost was another barrier. Although pasteurization added only a small expense to milk production, milk dealers faced pressure from consumers to lower prices rather than invest in quality improvements (Wessel, 1984). As an alternative, some reformers promoted "certified" or "inspected" milk, which was produced under strict sanitary standards and rigorous inspection protocols. However, certified milk was prohibitively expensive for most families, costing nearly twice as much as other milk varieties (Ayers, 1916; Fuchs et al., 1939). Fuchs et al. (1939)'s 1936 survey of U.S. cities provides some insight into the prices of various types of milk during this period. In large cities in 1936, certified raw milk cost an average of 17.4 cents while high-grade pasteurized milk cost an average of 13.3 cents. The cost of pasteurized milk was slightly lower than that of high-grade raw milk in these large cities, which cost an average of 13.5 cents.

2.3.3 Adoption of Pasteurization: The Path to Pasteurization in Chicago

The first U.S. law that could be considered a "pasteurization law" was passed in Chicago in 1908 (effective in 1909 (Wolf, 2001)).[7] However, this 1908 law was focused primarily on tuberculin testing of cows. Pasteurization was a secondary requirement, and it was only required for herds that tested positive on the most recent tuberculin test (Czaplicki, 2007). The pasteurization requirement was added to allow farmers

[6] Small communities continued to lack access to pasteurized milk even later. In 1944, while most milk in towns over 10,000 was pasteurized, rural areas continued to drink raw milk (Andrews and Fuchs, 1944).

[7] This is the same year that Anderson et al. (2022) also recorded the passage of bacterial standards for milk and tuberculin testing of cows in Chicago (effective 1909).

time to bring their herds up to the tuberculosis standards of the 1908 Chicago milk bill (Czaplicki, 2007). The law also did not specify the standards for pasteurization, leaving the process open-ended. Even though pasteurization was a secondary goal of the legislation, this 1908 law made Chicago the first known city to introduce any pasteurization requirement into legislation (Czaplicki, 2007). However, the state government quickly intervened. Faced with the prospect of slaughtering up to a quarter of Illinois's cattle herd, and the accompanying costs of compensating farmers, the state banned Chicago's tuberculin testing mandate (Czaplicki, 2007).

In response, Chicago adopted a new ordinance in 1912. This regulation introduced a milk grading system: higher-grade milk came from tuberculin-tested herds, while lower-grade milk could be sold if pasteurized and produced under acceptable sanitary conditions (Czaplicki, 2007). Although the 1912 ordinance took effect immediately, its pasteurization provisions phased in over time (USPHS, 1914). Key for our analysis, the pasteurization portion of the law was effective as of 1914.[8]

Despite the ordinance, the transition to universal pasteurization was a gradual process. After the law's pasteurization component became effective in 1914, Chicago's milk supply was still only 85% pasteurized (Boudouin, 1918). It was not until 1916, that the city was spurred by public health crises, such as hoof-and-mouth disease and polio, to pasteurize all milk. 1916 marked the first year that nearly 100% of Chicago's milk supply was pasteurized (USPHS, 1925; Czaplicki, 2007).

Chicago's eight-year journey from partial to near-universal pasteurization involved multiple legislative revisions and complementary public health reforms. This incremental process highlights the difficulty of assigning a single date to Chicago's pasteurization mandate. One could reasonably cite 1908 (first legal reference), 1912 (grading-based regulation), 1914 (enforcement of pasteurization standards), or 1916 (full adoption). In this paper, we use 1914 as the policy date because it was the date when pasteurization became both required and enforceable for a large share of the milk supply. To address ambiguity in policy timing, we complement our difference-in-differences framework with synthetic control methods (Section D), which allow us to empirically test when typhoid mortality began to decline. Importantly, we also verify that our results are robust to omitting these large cities with gradual rollouts of pasteurization in the robustness checks (Section 7).

[8] *"After January 1, 1914, all milk, cream, skim milk, or buttermilk which is not of the grade or kind defined in this section as "Inspected" shall be pasteurized at a temperature no less than 140 degrees F. for not less than 20 minutes or not less than 155 degrees F. for not less than 5 minutes"* (USPHS, 1914, pg 220). The 1912 law not only included requirements regarding the pasteurization of milk, it also regulated the standards for inspected milk, including adding temperature requirements for milk. Thus, the 1912 law in Chicago was not only a pasteurization ordinance; it regulated several aspects of the milk supply, including tuberculin testing, inspections, and temperature standards for milk.

[8] Chicago is not unique in its incremental approach. Cities such as New York and Cincinnati also passed initial pasteurization or grading ordinances in 1912, but did not achieve full adoption until 1914 (USPHS, 1914, 1915; North, 1921a). For consistency, we code the first effective ordinance date.

3 Data

3.1 Milk Pasteurization Ordinances: Dates and the Sample of Cities

Cities gradually began to mandate milk pasteurization through local ordinances, which often applied only to portions of the milk supply and frequently specified the method of pasteurization. Many ordinances exempted certified milk or milk from tuberculin-tested cows, limiting the complete pasteurization of the city's milk supply. As late as 1936, a national survey found that only 65 cities required the pasteurization of all milk by ordinance (Fuchs et al., 1939). However, pasteurization of the entire milk supply had been achieved in 135 cities, indicating that 70 cities reached full pasteurization without a legal mandate. An additional 41 cities had either pasteurized or certified milk widely available, with 36 of these cities having an ordinance in place (Fuchs et al., 1939).

Table 1 shows the sample of cities that passed pasteurization ordinances during the study period. To find pasteurization ordinances, we searched public health reports and newspaper articles. We include a detailed description of the source for each city in the online appendix. Table 1 shows the years that cities passed pasteurization ordinances in the first column and the respective pasteurization rates over time in subsequent columns (Ayers, 1916, 1922, 1926, 1932; Boudouin, 1918; Frank, 1933). Table 1 reveals that, in most cases, milk pasteurization began before the ordinances went into effect.

We employ more than one control group for pasteurization ordinance cities. For our main event-study analyses, our control group consists of larger cities (population 50,000 or more in 1930) that had no pasteurization ordinance in place as of 1936 **or** had pasteurization rates of 80 percent or less in 1930 or 1931 (Ayers, 1932; Frank, 1933).[9] Table A.1 shows these primary control cities. Though we show that our results remain robust when restricting the sample to control cities that we verified did not have pasteurization ordinances in place by 1936 (see Figure V). In robustness checks, we also expand the control group to include smaller cities (<50k population in 1930), and add state-by-year fixed effects as well. The results are similar whether or not we include smaller cities in the analysis (Figure A.16).

[9]We verify no pasteurization in Lincoln, NE, Atlanta GA, Memphis TN, Washington DC, Kansas City MO, Louisville KY, New Orleans LA, and Saint Paul MN. We also include the cities listed as having no ordinance but full pasteurization in Fuchs et al. (1939).

3.2 Mortality Data

We construct an unbalanced panel of city-level mortality and morbidity (or case counts) from *Bureau of the Census, United States Vital Statistics Division (1890-1938)*. The published mortality counts are available by cause and have been used in prior work (Feigenbaum et al., 2019; Hoehn-Velasco and Wrigley-Field, 2021; Ager et al., 2024). With the mortality data, we focus on the period from 1905 to 1936. We stop the analysis in 1936 to have six post-period years. 1937 also marks the year sulfa drugs were introduced (Thomasson and Treber, 2008; Jayachandran et al., 2010), which could affect several of the illnesses in our main analysis. We begin the analysis in 1905 because our first pasteurization ordinance did not go into effect until 1912. However, the results are similar if we extend the sample to 1900-1940 (see Panel C of Figure A.17). When constructing mortality rates, we use published population numbers interpolated between census years, as in prior work Feigenbaum et al. (2019); Hoehn-Velasco and Wrigley-Field (2021). We prefer the published population counts because the IPUMS city variable undercounts populations for certain cities.[10]

For our primary analysis, we create a composite measure of milkborne mortality. Our measure of milkborne mortality is the combination of typhoid, scarlet fever, and non-pulmonary tuberculosis. Among milkborne illnesses, typhoid was the most common, best documented, and most reliably traced disease, motivating an extra focus on typhoid mortality throughout the analysis (Straus and Straus, 1913; North, 1921a; Andrews and Fuchs, 1944). But scarlet fever and non-pulmonary tuberculosis also were linked to milk supplies, mostly through infected handlers (Ayers, 1916, 1932; North, 1921a; Currier and Widness, 2018).

In addition to these main causes of milkborne mortality, diarrhea and diphtheria are also potentially milkborne. General diarrhea could be linked to milk and milkborne outbreaks (Currier and Widness, 2018), and we consider diarrhea and diarrhea under age two individually in the findings. We consider diarrhea separately because it encompasses a range of non-specific causes of death. We also consider diphtheria separately because diphtheria's medical treatment changed substantially over the period. Diphtheria toxoid immunization was rolled out in the 1920s through the 1930s (Rosen, 1993; Harden, 1985).

Pasteurization may have influenced other illnesses that were not reported individually in the mortality statistics, such as septic sore throat and general gastroenteritis

[10] When constructing the population controls, we incorporate the "Minor Civil Division" (MCD) variable as well as the "Standard City." We attempt to match the populations to those in the mortality statistics and use the MCD when there are large discrepancies. The MCD combination overcounts the population at points, as compared to the approach with just the "Standard City", which undercounts.

caused by foodborne or waterborne illnesses. To capture these non-specific causes of mortality, and test the theory outlined by Cutler and Miller (2006) that a reduction in infectious (specifically waterborne) illnesses might have positive spillovers and reduce overall mortality, we also consider overall mortality, child mortality, and infectious disease mortality, in addition to the by-cause measures of mortality.[11]

We also incorporate newly digitized annual case counts of key notifiable illnesses reported in U.S. cities, using data from Public Health Reports (USPHS, 1912-1929). Separate volumes are available for small cities (population 10,000–100,000, available 1912–1929) and large cities (over 100,000, available 1912–1930). While the list of reportable illnesses varies somewhat over time, typhoid, another disease of focus based on its prevalence in milkborne outbreaks (see Figure I), is consistently included throughout the morbidity series.

3.3 Census Controls

We add census controls for city characteristics. These controls are derived from the IPUMS Restricted Complete Count Census Data (Minnesota Population, Center and Ancestry.com (2017); Ruggles et al. (2020)). Years between the census decades are linearly interpolated. For the main set of controls, we include the share white, which is expected to be related to mortality conditions in cities, as non-white mortality was significantly higher than white mortality, especially urban infectious disease mortality (Feigenbaum et al., 2019). We control for the share over 65, the share female, and the share foreign. The share foreign, in particular, is expected to be tied to mortality conditions, as it affects aspects of living conditions like crowding (Ager et al., 2024). We also control for health care resources with the number of physicians per 10,000s.

3.4 Summary Statistics

Table 2 presents the summary statistics across cities that adopted a pasteurization ordinance. Cities that adopt pasteurization ordinances tend to have slightly lower overall mortality levels. However, for specific mortality measures, such as scarlet fever and diphtheria mortality, cities that adopt pasteurization requirements had higher mortality than control cities. Our main outcome, milkborne mortality, is not significantly different between treatment and control cities.

[11] Infectious disease, in particular, is expected to decline after pasteurization if several by-cause measures of mortality decline after pasteurization began in the city limits. Infectious disease mortality includes 19 causes of mortality and is described in detail in Feigenbaum et al. (2019); Ager et al. (2024).

4 Empirical Strategy

For our primary analysis, we rely on an event-study specification. We choose a Poisson pseudo-maximum likelihood framework to consider the impact of pasteurization on mortality. In additional results, and for other mortality measures, we also consider the linear specification with the log of mortality.[12]

Formally, our preferred specification appears as:

$$\text{Mortality}_{jst} = exp(\alpha + \sum_{m=-6}^{7} \beta_m \text{ Pasteurization}_{jm} + \mathbf{X}'_{jst}\gamma + a_j + \eta_t)\epsilon_{jst} \tag{1}$$

where Mortality_{jst} is the mortality rate in city j, state s, and year $t = 1905, ..., 1936$. We model the rate using the death count as the outcome and the population as the exposure. This yields results equivalent to weighting by population and modeling the rate as the outcome.

The effect of the pasteurization ordinance is captured by $\text{Pasteurization}_{jm}$. $\text{Pasteurization}_{jm}$ represents the passage of a pasteurization ordinance in city j during period m (see dates in Tables 1), where m captures six years before and seven years after the ordinance. The main treatment effect of pasteurization is captured by the post-treatment dummy variables, $m = 0, 1, ..., 7$, which are relative to the pre-legislation year, $m = -1$. Endpoints are binned at $m = -6$ and $m = 7$, though we do not display endpoints in the main graphs. When considering typhoid morbidity, given that we only have data from years $t = 1912, ..., 1930$, we examine four years prior to pasteurization, with the endpoint binned at $m = -4$.[13]

We compare the effect of pasteurization against cities that did not pass pasteurization ordinances during the sample time frame. For our main event-study analyses, our control group consists of larger cities (population 50,000 or more in 1930) that had no pasteurization ordinance in place as of 1936 **or** had pasteurization rates of 80 percent or less in 1930 or 1931 (Ayers, 1932; Frank, 1933).[14] These control cities are

[12]We primarily rely on a Poisson model, which is more appropriate for the skewed distribution of lower-count mortality measures like typhoid. Poisson models have been chosen in similar settings such as Myers and Ladd (2020); Myers (2021a,b); Hollingsworth et al. (2022); Farin et al. (2024), where the outcomes of interest are rates that have a non-trivial number of zeros. We prefer the Poisson model over a log-level OLS specification due to the fact that the log is undefined at zero. Similar to the log-level model, Poisson coefficients also have the benefit of being an estimated semi-elasticity, or percentage change. Much of the existing literature Jayachandran et al. (2010); Alsan and Goldin (2019a); Anderson et al. (2020) models mortality changes as proportional changes rather than linear changes because areas have very different levels of mortality on average. Poisson models also avoid common pitfalls associated with log transformations, such as the log plus a constant and the inverse hyperbolic sine (Cohn et al., 2022). The semi-elasticity models any mortality decline as a proportional change, allowing for different average levels across cities. By contrast, the linear model estimates the decline in the mortality rate as similar across cities.

[13]When we present grouped post-period estimates, the point estimate corresponds to the grouping of the post-period event study coefficients from $m = 1$ onward in Equation 1. The DiD equation is presented in Section C.

[14]We verify no pasteurization in Lincoln, NE, Atlanta GA, Memphis TN, Washington DC, Kansas City MO, Louisville KY, New Orleans LA, and Saint Paul MN. We also include the cities listed as having no ordinance but full pasteurization in Fuchs et al.

shown in Table A.1. $\mathbf{X}_{jst}$ contains city-level controls. Because of contamination from multiple treatments, we only include demographic controls in the main specification (Hull, 2018; Goldsmith-Pinkham et al., 2024; De Chaisemartin and d'Haultfoeuille, 2023; Hoehn-Velasco et al., 2024). Controls include the share of the population that is white, the share of the population over 65, the share female, the share foreign, and the physicians per 10,000 persons. In additional results, we add policy controls for other public health policies from Anderson et al. (2022), as well as water purification and disinfection from USPHS (1926). Finally, a_j addresses the time-invariant city fixed effects, and η_t captures the year fixed effects. ϵ_{jst} is the regression error, which is clustered at the city level throughout the results.

4.1 Potential Threats to Validity

Several threats to validity are apparent in this primary specification. First, we present our main results using the canonical two-way fixed effects estimator (TWFE) and Poisson Model. However, the TWFE estimator is known to make improper comparisons between units (Borusyak et al., 2018; Sun and Abraham, 2020; Callaway and Sant'Anna, 2020; Goodman-Bacon, 2021). Thus, in robustness tests, we also implement a log-linear OLS model with the Interaction-Weighted (IW) estimator from Sun and Abraham (2020). This estimator, proposed by Sun and Abraham (2020), addresses concerns that dynamic TWFE event study estimates may be contaminated by effects from other time periods (Goodman-Bacon et al., 2019; Callaway and Sant'Anna, 2020; De Chaisemartin and d'Haultfoeuille, 2023). The results using this IW estimator are consistent with our main results using the TWFE estimator (see Figure A.18).

Second, cities also invested in water infrastructure alongside milk ordinances. Table A.2 presents the dates of milk regulation (for available cities) as well as water infrastructure investment from several sources (*Filtration Plant Census, 1924*, August, 1925; USPHS, 1926; Anderson et al., 2022). We separate the effects of clean water from clean milk by presenting compelling subsample analyses in the robustness checks.

Third, in addition to city-level ordinances, several states enacted their own milk regulations. For instance, California mandated either pasteurization or tuberculin testing in 1914 (USPHS, 1911-1922), and New York implemented a milk grading ordinance in 1913 (effective 1914), which included pasteurization as a core requirement (USPHS, 1911-1922). Other states, including Indiana, New Jersey, and Colorado, followed with similar laws during the 1920s (USPHS, 1923-1928). These state-level policies are a potential confounding factor for our city-level analysis, particularly if they also influ-

(1939). We also show that our results remain robust when restricting the sample to control cities that we verified did not have pasteurization ordinances in place by 1936 (see Figure A.17).

enced cities in the control group. To address this, we include state-by-year fixed effects in robustness checks (see Figure A.16). These controls absorb the impact of state policies that are uniform within each state and over time.

Fourth, our empirical strategy assumes that the timing of pasteurization mandates is exogenous, meaning that adoption is not driven by prior trends in mortality rates. This assumption would be violated if cities with higher mortality levels were systematically more likely to adopt ordinances earlier. We test this in Appendix Table A.3 with a Cox proportional Hazard model. For the most part, past and current levels of mortality and morbidity do not significantly predict the adoption of the pasteurization ordinance with controls in the specification.

Finally, our empirical strategy assumes that pasteurization ordinances acted as an exogenous shock to raise the pasteurization rate in cities. And through this increase in city-level milk pasteurization, city-level health improves. Table A.4 confirms this: across both weighted and unweighted specifications, adopting a pasteurization ordinance increases the city's pasteurization rate by roughly 15–18 percentage points. These first-stage estimates indicate that the ordinances produced large and meaningful shifts in the share of pasteurized milk, consistent with our interpretation of pasteurization as a substantive change in local milk safety.

5 Results

5.1 Pasteurization Improves Milk Safety

We begin by assessing whether pasteurization improved milk safety in a broad sense, or milkborne mortality. Milk in the early twentieth century carried multiple disease risks, including typhoid, scarlet fever, and non-pulmonary tuberculosis (Winslow, 1908, 1909; Rosenau, 1912; North, 1921b; Armstrong and Parran, 1927). If pasteurization effectively reduced contamination events in the milk supply, we should observe declines across the combination of these illnesses.

Figure II.A presents a composite measure of milkborne mortality that includes typhoid, scarlet fever, and non-pulmonary tuberculosis. The event-study estimates show a sustained post-adoption decline in milkborne mortality of approximately 16%, with flat, statistically insignificant pre-periods, indicating no evidence of differential trends prior to pasteurization.

On the right side of Figure II, we calculate the estimated decline in the number of deaths using counterfactual predictions from the Poisson model. The counterfactual series, plotted in light pink lines, is generated by removing the estimated contribution of pasteurization in treated cities. The observed levels of milkborne mortality (light gray) closely track the predicted series (maroon), while the green dashed line highlights the gap between the predicted and counterfactual counts of milkborne deaths. This counterfactual series indicates that pasteurization prevented 800–1,200 milkborne deaths annually across adopting cities. This reflects the substantial prevalence of milkborne illness in the early twentieth century and highlights the scale of mortality declines directly attributable to unsafe milk.

Importantly, these results also isolate the direct effect of milk safety. Neither scarlet fever nor non-pulmonary tuberculosis is waterborne. Scarlet fever spreads through respiratory droplets and, crucially for this context, through milk contaminated by infected milk handlers (Rosenau, 1912; Straus and Straus, 1913; Armstrong and Parran, 1927; Guthrie, 1931). Likewise, non-pulmonary tuberculosis is transmitted through infected milk (Winslow, 1908, 1909; Rosenau, 1912; North, 1921b; Armstrong and Parran, 1927). Thus, declines in milkborne mortality following pasteurization provide compelling evidence that these declines are attributable to improvements in milk safety, rather than to coincident water infrastructure investments. Supporting this interpretation, when we restrict the composite measure to only scarlet fever and non-pulmonary tuberculosis, mortality from these two illnesses alone still falls by roughly 25% (Figure A.4).

5.2 Pasteurization Reduces Typhoid Fever Cases and Deaths

Typhoid fever warrants particular focus because it provides the clearest and most historically definitive measure of milk safety. Typhoid accounted for nearly 80 percent of all documented milkborne outbreaks in the early twentieth century (Armstrong and Parran, 1927). Typhoid was also sensitive to improvements in sanitation, both water and milk, making it a precise indicator of changes in the disease environment (Beach et al., 2016; Anderson et al., 2022). Focusing on typhoid allows us to compare our findings across water investments of the early twentieth century. A final advantage of focusing on typhoid is that we observe both mortality and morbidity. Case counts provide a less severe but highly informative measure of milkborne illness, offering a novel complement to the mortality data typically used in this literature. For these reasons, typhoid serves as a natural benchmark against which to evaluate the public health impact of pasteurization.

Figures II.B and II.C show that pasteurization ordinances led to large, immediate, and persistent declines in typhoid morbidity and mortality. Across cities, typhoid case rates fall by roughly 32 percent, and typhoid mortality declines by 34 percent following the ordinance. The post-period effects remain statistically significant throughout, while pre-trends are flat and insignificant, supporting a causal interpretation.

To quantify the magnitude of these declines, we compare observed typhoid levels to a counterfactual series generated by removing the estimated effect of pasteurization. The resulting gap indicates that pasteurization prevented approximately 150–300 typhoid deaths and 1,500–3,200 typhoid cases per year in adopting cities. The largest absolute reductions occurred during the 1910s and 1920s, when baseline typhoid incidence remains high.

These results underscore that pasteurization rivaled some of the most influential public-health interventions of the era. In percentage terms, pasteurization is comparable to, though slightly smaller than, the impact of major water infrastructure investments that occurred during the same era (Beach et al., 2016; Cutler and Miller, 2005; Anderson et al., 2022). For example, Anderson et al. (2022) finds a 36% reduction in typhoid mortality after the introduction of water filtration plants. While our headline estimate is slightly smaller at 34%, the magnitude of the relative decline emphasizes that pasteurization played a major role in reducing typhoid mortality in cities. Taken together, the evidence places pasteurization alongside foundational water sanitation measures as a crucial public health intervention.

5.3 Evidence from Milkborne Outbreaks

We corroborate the mortality patterns using newly digitized data on milkborne outbreaks. Armstrong and Parran (1927) documented annual counts of traced milkborne outbreaks across the U.S. from 1906 to 1926, providing a direct measure of disease transmission through contaminated milk. Using a difference-in-differences design, we test whether the frequency of these outbreaks declined after pasteurization ordinances. To estimate the effect on outbreaks, we use a grouped post-period version of Equation (1) (see also Equation (C)). Table 3 shows consistent, though modest, declines in outbreak frequency following adoption. The largest reductions appear when aggregating all milkborne outbreaks. These results reinforce the interpretation that pasteurization directly reduced contamination events in the milk supply, complementing the mortality and case rate evidence in Figure II.

5.4 Summary

Taken together, these findings show that pasteurization delivered broad and substantial public health gains. Across cities, pasteurization ordinances produced large, immediate, and persistent reductions in typhoid morbidity and mortality by 32-34%

and reduced broader milkborne mortality by 16%. In absolute terms, the largest population benefit comes from the composite reduction in milkborne mortality: pasteurization averted roughly 800–1,200 deaths per year, compared to 150–300 typhoid deaths. These declines, reinforced by documented reductions in traced milkborne outbreaks, provide consistent evidence that pasteurization meaningfully improved the safety of the milk supply. Overall, the evidence indicates that pasteurization ordinances were a central, underappreciated, driver of early twentieth-century improvements in urban health.

6 Additional Results: Pasteurization Does Not Reduce Other Causes of Death

6.1 All-Cause and By-Cause Mortality

Appendix Figures A.5, A.6, A.7, A.8 and A.9, present estimates for causes of death beyond typhoid. Across these other measures of mortality, we find little evidence that pasteurization had a consistent effect on mortality. Only a few post-pasteurization ordinance declines are visible, primarily for scarlet fever and non-pulmonary tuberculosis. Both of these causes of death are incorporated into milkborne mortality in Figure II.

One notable finding is that pasteurization does not cause a sharp break in diarrheal mortality. Pasteurization could influence diarrheal mortality either through misclassification of typhoid deaths or through real reductions in gastrointestinal illness. In either scenario, we would expect diarrheal mortality to exhibit a post-pasteurization decline similar to that observed with typhoid. However, the event-study estimates show no significant post-period drop in diarrheal mortality, including among children under age two (Figure A.10). The absence of a detectable decline in diarrheal deaths suggests that pasteurization's effects were confined solely to specific milkborne deaths rather than to broader gastrointestinal conditions.

6.2 Overall Mortality Among Infants and Young Children

Historical accounts suggest pasteurization should have substantially reduced infant and child mortality, given the central role of cow's milk in early nutrition (Preston and Haines, 1991). Yet, as shown in Figure A.10, mortality among children does not decline sharply after pasteurization ordinances.[15] Aside from a modest post-period drop in some specifications, the event-study estimates show limited changes, and even di-

[15]We report both Poisson and OLS estimates for child mortality.

arrheal mortality under age two shows no meaningful reduction after pasteurization. These results align with Anderson et al. (2025), which found that milk inspections reduced gastrointestinal causes of mortality but did not reduce infant mortality. A potential explanation is that infants were already receiving comparatively safer milk before the pasteurization ordinances. Families often boiled milk or purchased pasteurized milk, and many cities operated infant milk depots that distributed pasteurized milk to low-income households. In New York City, for example, milk depots established between 1908 and 1910 "proved beyond question their great value in the reduction of infant mortality" (North, 1921a, p. 241). Such programs may have protected infants before pasteurization became mandatory, a hypothesis we test further in Section 8.

6.3 Did pasteurization reduce cause-specific mortality among infants and young children?

Figure III confirms that pasteurization's largest and clearest reduction in mortality occurs at older ages. Milkborne mortality in Panel A shows the clearest declines for adolescents and adults of 13–18%. Infants show some decline, but it is statistically insignificant. Typhoid in Panel B shows similar age-specific effects. Older children and adults experience significant reductions of 25–30%, while infant estimates are smaller and less precise. Together, these patterns indicate that pasteurization substantially improved milk safety but had a limited detectable impact on infants. Thus, unlike water infrastructure investment, which was most important for infants (Anderson et al., 2022), our results show that clean milk was comparatively more important for older children and adults.[16]

7 Robustness

We perform a battery of robustness tests to confirm our main findings. Because we would like to identify the causal effect of the pasteurization ordinance, it is important to demonstrate that the estimated reduction in typhoid is not driven by bundled public-health reforms, differential pre-trends, or compositional changes in the set of treated or comparison cities. Throughout this section, we continue to rely primarily on the event-study framework because it allows us to assess whether pasteurization caused sharp changes in mortality and whether there are preexisting trends prior to the ordinances. In nearly every specification, the post-treatment decline in milkborne

[16] Figures A.12 and A.11 also show the scarlet fever and other tuberculosis deaths by age. Figure A.12 shows the log-linear OLS results. Figure A.13 breaks out the larger age groups – individual ages 1,2,3,4,5-9, and 20-29. Figure A.13 shows some declines in milkborne mortality for ages 1-2. The most apparent declines are for those 20 and over for both typhoid and milkborne mortality. We choose to combine ages into larger buckets for the main results to have larger counts of each illness in each age group.

mortality falls between 10-20 percent, and typhoid mortality falls within a range of approximately 20–40 percent. The consistency of the decline strengthens our interpretation that the adoption of pasteurization ordinances played an independent role in reducing mortality in U.S. cities.

7.1 Pasteurization Reduces Typhoid Fever in Subsample Analyses with "Clean" Treatments

A central concern is that pasteurization ordinances were enacted during a period of rapid expansion of public health measures, particularly water filtration and chlorination. If these investments were clustered around the same time as pasteurization, or systematically occurred in cities with higher disease burdens, our baseline event-study estimates could inadvertently capture the effect of cleaner water rather than the effect of clean milk.

To directly address this concern, Figure IV A-E presents a series of subsample analyses that intentionally restrict the treated and comparison groups to cities with "clean" treatment timing. Figure IV Panel A excludes cities that installed chlorination systems within seven years of pasteurization; Panel B removes cities that adopted water filtration; Panel C omits cities that experienced either type of water infrastructure investment within this window; and Panel D removes cities with filtration plants using an alternative filtration dataset (*Filtration Plant Census, 1924*, August, 1925). In all cases, the event-study pattern closely mirrors the main estimates. The similarity of these results across Panels A-D indicates that the decline in typhoid is not due to clean water investment, and rather, that the effect of pasteurization persists across "clean" samples.

Figure IV Panel E shifts the focus to milk-specific regulatory reforms: bacteriological standards and tuberculin testing of cow herds. These regulations were sometimes adopted close in time to pasteurization, and the effects of the two interventions could potentially be confounded. Using data from Anderson et al. (2022), we remove cities that adopted bacteriological standards or tuberculin testing at the same time as pasteurization, thereby isolating the impact of pasteurization from other milk-safety measures. The results again show a clear decline in typhoid following pasteurization. The consistency of the decline in typhoid suggests that pasteurization is not merely a proxy for general milk regulation.

As a final step, Figure V Panels A-B take a more direct approach and add controls for public health interventions. In Panel A, we control for the water and milk-quality controls compiled by Anderson et al. (2022), including indicators for filtration, chlorination, tuberculin testing, bacteriological standards, and sewage treatment or diversion. Because these controls exist for only 25 cities, the resulting estimates are necessarily less precise. Even so, the event-study pattern remains effectively unchanged: the decline in typhoid following pasteurization mirrors the baseline trajectory. Taken together with the subsample evidence, this specification reinforces that the estimated effect of pasteurization is not simply picking up the influence of contemporaneous public-health investments, but instead reflects an independent decline attributable to pasteurization.

Because Panel A has fewer than 25 cities, in Figure V Panel B, we broaden the sample coverage to include controls for water filtration and disinfection data from USPHS (1926). Using these dates for water infrastructure investments yields a larger sample than in Panel A. Still, the estimated decline in Panel B aligns closely with the baseline, a reduction in typhoid fever by more than 30 percent and a decline in milkborne mortality by 15%. The consistency of the specifications, along with additional controls across the two distinct data sources, reinforces the conclusion that pasteurization's health improvements do not result from related public health improvements.

7.2 Unweighted Estimates and the Influence of Large Cities

Another concern is that population-weighted estimates might reflect mortality declines concentrated in a few very large cities rather than an average effect across U.S. cities. To evaluate this possibility, Panel C of Figure V reports unweighted event-study estimates. These estimates give each city equal importance, thereby revealing the "average city's" response to pasteurization. The resulting trajectory is similar to the weighted specification, showing a clear and persistent post-adoption decline. This result indicates that the estimated effect is not driven by Chicago, New York, or a handful of other large cities, but instead reflects a widespread reduction in typhoid across urban areas.

Relatedly, in Figures A.14 and A.15, we individually drop large cities in the sample. This leave-one-out analysis ensures that none of the largest cities drives the entire treatment effect. Even when each of these large cities is removed, a continued decline in typhoid mortality persists following the introduction of pasteurization.

7.3 Adjusting for Population Size and City Characteristics

Because pasteurization was more common in larger and faster-growing cities, we also test whether population dynamics could bias the estimates. Figure V Panel D incorporates population-quartile-by-year fixed effects, which flexibly control for differential mortality trends among cities of different sizes. If large cities were already on a downward trajectory for typhoid, these fixed effects would eliminate declines in specific city-size groups. Yet with these population-quartile-by-year fixed effects, the resulting estimates remain nearly identical to the main results.

7.4 Alternative Control Groups

Another concern is that our results might be driven by the particular set of comparison cities used in the baseline specification. First, in Figure V Panel E, we restrict the sample to cities that did not pass pasteurization ordinances. Here, the results are similar to the baseline.

Then, Figure A.16 broadens the comparison group: first to all cities with pasteurization rates below 80 percent in 1930–1931 (Panels A–B), then to the full universe of cities regardless of pasteurization status (Panels C–D). Across all these expanded control sets, the event-study profiles remain nearly identical to the baseline. Moreover, adding state-by-year fixed effects in Figure A.16, which absorb any policy changes or shocks common to cities within the same state in a given year, produces similarly large and precise reductions in typhoid mortality and morbidity. These results indicate that neither selective comparison groups nor differential state-level policy adoption can account for the estimated declines, strengthening the causal interpretation of pasteurization.

Figure A.16 Panel E also constructs a more observably similar comparison group using nearest-neighbor matching and propensity-score reweighting (Figure A.16 Panel E). First, we match cities based on 1910 observable characteristics (our main controls as well as the log of typhoid mortality in 1910. We run a logistic regression of the adoption of pasteurization ordinances in 1910 based on these characteristics. Then, we construct the propensity scores and use these scores to choose the city most observably similar to the treatment cities in 1910. This nearest neighbor matching selects one match for each treated city. After performing the nearest neighbor matching, we run our event-study analysis with propensity score weights applied. With these propensity score weights, after the pasteurization ordinance goes into effect, typhoid mortality and morbidity still decline.

7.5 Alternative Sample Windows and Sample Restrictions

To assess whether our findings depend on the choice of sample window or the construction of population denominators, Figure A.17 Panel A extends the analysis to 1900–1940 and replaces published population figures with IPUMS-based counts. Despite these changes in panel length and population measurement, the estimated post-treatment decline closely matches the baseline. Panel B further subsets to a balanced panel, ensuring that cities neither enter nor exit the sample early. Still, the balanced sample leaves the treatment effect essentially unchanged. We also subset to the largest urban centers in Panel C. Panel C restricts the sample to cities with populations above 100,000, producing estimates nearly identical to those from the full sample. Similarly, limiting the analysis to a shorter window (1905–1930) and to cities adopting pasteurization before 1926 (Panel D) yields results that mirror the baseline.

7.6 TWFE-Alternative and Alternative Functional Form

Next, in Appendix Figure A.18, we present the estimates from a log-linear OLS specification that considers the log of the milkborne mortality rate, the log of the typhoid mortality rate, and the log of the typhoid case rate. The results also display the estimates from the Interaction-Weighted estimator alongside the estimates from TWFE specifications (Sun and Abraham, 2020). This IW estimator addresses concerns that dynamic TWFE event study estimates may be contaminated by effects from other time periods (Goodman-Bacon et al., 2019; Callaway and Sant'Anna, 2020; De Chaisemartin and d'Haultfoeuille, 2023). The plotted points in Figure A.18 display a decline similar to the estimates from the Poisson specification. Following the pasteurization ordinance, both typhoid mortality and the case rate decline by over 30%, milkborne mortality declines by more than 15%.

7.7 Synthetic-Control DiD Event Studies

We next evaluate whether the estimated effect of pasteurization persists when we replace the traditional event-study design with a synthetic-control-based event study in Figure A.19 Panel A. This estimator, following Arkhangelsky et al. (2021) and Ciccia et al. (2024), constructs counterfactual outcomes using weighted combinations of untreated cities and provides an alternative form of identification. We include only the years 1909-1929 because we need a balanced panel of cities. Many cities are missing typhoid data before 1908 and many cities also entered the sample in 1930 and dropped out in 1931. We use the log of typhoid mortality as the outcome, which also results in the loss of some observations due to zero values. However, before the late 1920s, most cities had higher mortality rates. Cities are compared to the full group of control cities with pasteurization rates of 80 percent or less, and where we verify no pasteurization ordinance. We include all cities due to requirements on the number of controls.[17] The

[17] The analysis was performed with 100 bootstrap replications. We only show the main event window from m=-10 to 10. We cannot consider morbidity, because it is difficult to construct a balanced panel with this data, since it starts later than the mortality data.

synthetic-control event study yields results that are strikingly similar to the primary analysis. The synthetic estimates reveal a clear break at the time of the pasteurization ordinance, followed by a persistent decline in mortality. Though, the break in mortality is clearer for typhoid than milkborne mortality.

7.8 Formal Pre-Trends Tests

To further assess the validity of the event-study design, we conduct formal tests for pre-treatment parallel trends following the framework of Roth (2022) and Caceres-Bravom (2024). These tests assess whether the estimates in the years preceding pasteurization differ systematically from zero. Figure A.19 Panel B shows that the estimated pre-trends for both typhoid mortality and case rates run in the opposite direction of the post-treatment effects. This pattern reinforces the argument that the observed decline in typhoid outcomes is not driven by pre-existing trends.

7.9 Permutation-Based Placebo Tests

Next, to determine whether the estimated decline could have arisen by chance, we implement a permutation (or "randomization inference") placebo test. Specifically, we assign each city a random draw of the year of the pasteurization ordinance from the original distribution of adoption years, preserving the staggered rollout structure. Following the approach of Chetty et al. (2009), Buchmueller et al. (2011), Ohrn (2018), and Baron et al. (2020), we re-estimate our baseline event-study model (Equation 1) using a grouped post-treatment indicator. This simulation is repeated 1,000 times, with each iteration drawing a new random assignment. The resulting cumulative distribution function (CDF) of simulated treatment effects is shown in Figure A.20. Comparing the empirical estimate to this null distribution yields a non-parametric p-value, which indicates that the observed treatment effect is statistically significant at the 1% level.

7.10 Difference-in-Differences: Interaction of Pasteurization and Water Infrastructure

In Appendix Section C, we present a complementary difference-in-differences analysis that parallels the main event-study design. Because the event studies are better suited to evaluating pre-trends and dynamic treatment effects, we place the full DiD results in the appendix. However, one result from the DiD framework warrants direct discussion: the interaction between pasteurization and investments in water infrastructure.

Understanding this interaction is important for several reasons closely tied to the identification concerns raised earlier in the paper. First, as noted in Section 4, pasteurization did not emerge in isolation. Cities often pursued milk-safety reforms during periods of investment in water infrastructure. If pasteurization closely tracked these investments, or if its effectiveness depended on improvements to water systems, our baseline estimates could mistakenly attribute water-related reforms to milk safety interventions. Evaluating the interaction term between water infrastructure investment and pasteurization, therefore, allows us to test whether pasteurization works because it coincides with cleaner water or whether it exerts an independent effect. Second, prior work by Alsan and Goldin (2019b) emphasizes that public health investments often act as complementary investments. If clean water and clean milk reinforce each other, by either reducing exposure or by lowering the overall typhoid burden, then we would expect interaction terms between pasteurization and water investments to show larger declines in typhoid mortality. Detecting such complementarities is particularly relevant in this setting, where multiple public health reforms were adopted within a short time frame.

Table 4 presents the DiD results with these interaction effects, incorporating indicators for filtration, chlorination, and sewage treatment from Anderson et al. (2022) and USPHS (1926). Across most specifications, the interaction terms provide little evidence of complementarities between clean milk and clean water, particularly for typhoid. For milkborne mortality, Panel B shows modest interaction between pasteurization and water disinfection: the interaction coefficient is negative and statistically significant in several columns, suggesting that disinfection may slightly amplify the milk-safety gains from pasteurization. Though, the interaction term does not show the same sign and significance in Panel C-D, using the the filtration and chlorination measures from Anderson et al. (2022).

For the most part, Table 4 indicates that pasteurized milk and water improvements functioned as independent channels of disease reduction, rather than mutually reinforcing interventions. For typhoid especially, the pattern is clear, pasteurization did not become more or less effective in cities with water filtration or disinfection, nor is there evidence that water treatment required pasteurized milk to yield its full benefits. Importantly, the main effects remain large and robust: pasteurization reduces typhoid deaths and cases by roughly 30–40%, and water filtration produces similar reductions. The persistence of these main effects even after adding interaction terms reinforces the conclusion that each intervention independently improved public health.

Taken together, the interaction results reinforce that pasteurization was not simply a proxy for contemporaneous water infrastructure investments, nor were its benefits dependent on parallel improvements in water infrastructure. Instead, the evidence points to two distinct public-health channels: cleaner milk and cleaner water each reduced typhoid fever independently. Although Table C.1 shows that the relative magnitudes of pasteurization and water-treatment effects vary somewhat by specification, the overall pattern is consistent, both interventions yield sizable and broadly comparable reductions in typhoid mortality. The absence of meaningful interaction effects, combined with the stability and similarity between the coefficients, underscores that pasteurization provided an independent contribution to early twentieth-century mortality declines.

8 Voluntary Pasteurization is Associated with Reductions in Child Mortality

Pasteurization is expected to yield its greatest benefits for infants and young children, who relied heavily on cow's milk in the early twentieth century (Preston and Haines, 1991). Yet, there is little overall mortality decline from pasteurization ordinances for infants or children (Figure A.10). Aside from modest post-period dips, the event studies reveal no sharp or systematic decline, and even diarrheal mortality under age two remains largely unaffected by pasteurization.

A key reason pasteurization ordinances may have muted effects on infants and young children is that many families in large U.S. cities already had access to safer milk before the ordinances were imposed. In the decades leading up to adoption, a substantial share of the urban milk supply was voluntarily pasteurized, and families with infants often purchased higher-quality or boiled milk at home before local regulations. Milk depots and philanthropic organizations also distributed pasteurized milk to low-income families well before cities enacted mandatory ordinances (Rosenau, 1912). In this context, ordinances may have simply formalized practices already widespread among households most sensitive to milk safety.

We assess whether health improvements occurred during periods of rising voluntary pasteurization, by considering whether infant mortality responds to changes in the share of locally pasteurized milk. In order to test whether the supply of pasteurized milk affects mortality, we digitize pasteurization rates from Ayers (1916, 1922, 1926, 1932), Boudouin (1918), and Frank (1933). These pasteurization rates are collected from surveys sent to health officials in each city (Ayers, 1922, 1926, 1932).[18]

While these results cannot be interpreted causally, they still provide suggestive

[18] We keep only cities that reported their pasteurization rates at the beginning of the sample, 1921, and at the end of the sample, 1931, the first and last comprehensive years in which pasteurization rates were reported. Examples of city-specific pasteurization rates are shown in Tables 1 and A.1.

evidence about whether increases in the supply of safer milk are associated with reductions in infant mortality. These results complement the main results, and show the effects of marginal changes in city-level milk pasteurization, speaking to the effect of increasing pasteurized milk within cities over time. Similarly, but distinct, the main results estimate the effect of an exogenous shock to pasteurization of the city's milk supply, or moving from partial to near-universal pasteurization due to the city-level mandates. As shown in Table A.4, pasteurization ordinances sharply increase the availability of pasteurized milk in cities.

Formally, we consider whether increasing the city-level pasteurization rate affects urban mortality as:

$$\text{Mortality}_{jt} = exp(\alpha + \beta \ \text{ Pasteurization Rate}_{jt} + \mathbf{X}'_{jt}\gamma + a_j + \eta_t)\epsilon_{jt} \tag{2}$$

where Mortality_{jt} is the mortality rate in city j and year $t = 1905, 1910, 1911, 1912, 1913,$ $1914, 1915, 1916, 1921, 1924, 1930, 1931$. As in the main empirical strategy, we model the mortality rate with the death count as the outcome and the population as the exposure. In this specification, we focus on mortality because we are primarily interested in child mortality. The effect of pasteurization is captured by the $\text{Pasteurization Rate}_{jt}$ in city j during year t. As in the main empirical strategy, $\mathbf{X}_{jt}$ contains city-level controls, a_j addresses the time-invariant city fixed effects, and η_t captures the year fixed effects. ϵ_{jt} is the regression error, which is clustered at the city level. We do not include city-level trends because specifications with certain controls and state-by-year fixed effects have too few observations.

While there are limitations to this analysis, Equation (2) provides more than plain cross-sectional correlational evidence. Equation (2) examines whether changes in the pasteurization rate over time are associated with changes in mortality in that city. However, omitted factors may cause cities to adopt voluntary milk pasteurization and also lead to reductions in child mortality. While in many cases these differences will be captured by city-fixed effects, omitted factors may still be time-varying.

8.1 Voluntary Pasteurization is Associated with Reductions in Infant and Child Mortality

Table 5 presents the relationship between the availability of pasteurized milk and mortality. Across Columns (5)–(8), higher pasteurization rates are consistently associated with reductions in infant mortality and mortality under age two. These results point to a potential explanation for why pasteurization mandates had limited effects on infants: parents were likely early adopters of pasteurized milk. Historical accounts indicate that pasteurized milk carried a price premium (Wessel, 1984), meaning that informed, higher-risk families may have selectively purchased safer milk in advance of regulation. In this setting, mandatory ordinances potentially had little additional impact on child mortality because the most vulnerable had already shifted behavior voluntarily (Chapin, 1921; Tomes, 1990).

By contrast, the availability of pasteurized milk only shows weak and inconsistent associations with typhoid and waterborne mortality. This contrast also helps reconcile the results in Table 5 with the main findings: infant mortality responds to any additional pasteurized milk, whereas typhoid responds primarily when the milk supply becomes universally pasteurized through ordinances. This lack of a clear relationship between pasteurization rates and typhoid & waterborne diseases suggests that broad-based public health interventions, such as mandatory pasteurization and clean water infrastructure, were necessary to fully eliminate foodborne illness in cities. This explanation is supported by the fact that the pasteurization ordinance is more important for typhoid fever and waterborne mortality than the pasteurization rate in Panel B of Table 5. Because typhoid and waterborne illnesses affected all demographic groups, not just infants and young children, selective private action was likely insufficient to completely eliminate the spread of typhoid.

We further test the robustness of the voluntary pasteurization rates in Panels C-E of Table 5, incorporating state-by-year fixed effects, controls for water purification and disinfection, and additional public health controls from Anderson et al. (2022). Further, we present the results from an OLS specification using the log of the mortality rate. Throughout these modifications, infant mortality consistently declines with higher milk pasteurization rates. Infant mortality declines appear to be more closely related to the city-level milk pasteurization rate than to either the pasteurization ordinance or investments in clean water infrastructure.

Taken together, the results suggest that voluntary access to pasteurized milk was more closely associated with reductions in infant mortality than either pasteurization mandates or investments in clean water. Infant mortality rates fell most significantly where pasteurized milk became widely available, regardless of the presence of ordinances. By contrast, typhoid and waterborne mortality responded more clearly to universal interventions. While parents could individually protect their children by purchasing pasteurized milk, controlling diseases like typhoid, which spread broadly across populations, depended on citywide improvements in milk safety and water quality. These public health mandates likely fill the gap that private behavior alone could not overcome.

9 Conclusion

This paper provides new evidence that improvements to the milk supply through mandatory pasteurization played a major and underappreciated role in the early twentieth-century decline in infectious disease. Contaminated milk was a pervasive source of illness in American cities, responsible for hundreds of documented outbreaks and a wide range of milkborne illnesses (Straus and Straus, 1913; Ayers, 1932; Armstrong and Parran, 1927). Consistent with this historical record, our results show that pasteurization mandates substantially improved overall milk safety. A composite measure of milkborne mortality, including typhoid, scarlet fever, and non-pulmonary tuberculosis, fell by roughly 16 percent following pasteurization ordinances. In absolute terms, this corresponds to 800–1,200 fewer milkborne deaths each year.

We also examine typhoid fever specifically because it accounted for the majority of milkborne outbreaks. We find that pasteurization ordinances reduced typhoid mortality and case rates by approximately 32-34 percent, preventing an estimated 150–300 deaths and 1,500–3,200 cases annually. These reductions place pasteurization alongside water filtration in terms of the relative reductions in typhoid fever. Whereas filtration lowered typhoid mortality by about 36% (Anderson et al., 2022), pasteurization achieved a nearly comparable decline of 32-34%.

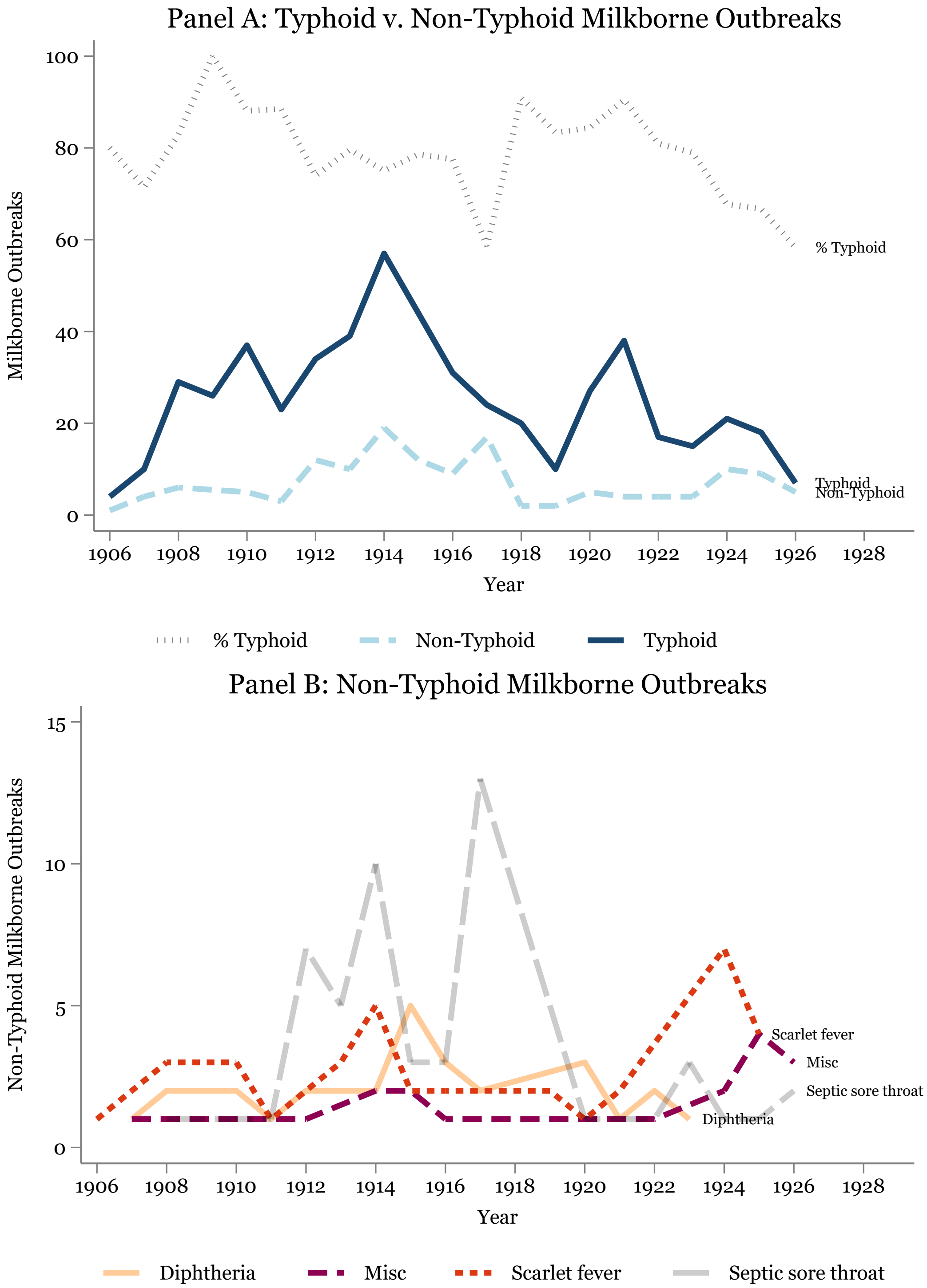
From a public cost-benefit perspective, pasteurization mandates also differed importantly from other major public health investments of the era. Large-scale water infrastructure projects required substantial upfront capital expenditures and ongoing public maintenance costs borne directly by municipal governments (Cutler and Miller, 2005). In contrast, pasteurization ordinances relied primarily on regulatory standards and enforcement, shifting much of the implementation cost to private producers and consumers while requiring comparatively modest public outlays. By setting and monitoring minimum safety standards for a widely consumed good, cities were able to generate large population health gains without undertaking major infrastructure investments. Pasteurization thus functioned as a scalable regulatory complement to water sanitation, expanding the toolkit available to cities seeking to reduce infectious disease.

Overall, our results demonstrate that pasteurization ordinances provided essential, population-wide protection against foodborne disease, particularly typhoid fever. The influential role of clean milk is not merely historical; recent political controversies have revived these tensions. Today, advocates of raw milk argue that drinking raw milk improves the immune system, symptoms of asthma, and lactose intolerance (FDA, 2011), and public discourse has increasingly promoted raw milk consumption

(Magnoli, 2025; MassLive, 2025; Dickson, 2025). Simultaneously, challenges within the current regulatory framework have raised concerns about the capacity to effectively safeguard the milk supply (Douglas, 2025; Deutsch, Chris, 2025). Still, public health experts and the U.S. Food & Drug Administration continue to assert the dangers of raw milk and supply evidence against these misconceptions (FDA, 2011). These debates mirror the tensions that shaped pasteurization campaigns a century ago (Ayers, 1916; Ward et al., 2007; Dickson, 2024; Greenfield, 2025). By revisiting the historical implementation of pasteurization, our findings highlight the profound public health benefits of community-wide standards for food safety.

10 Figures

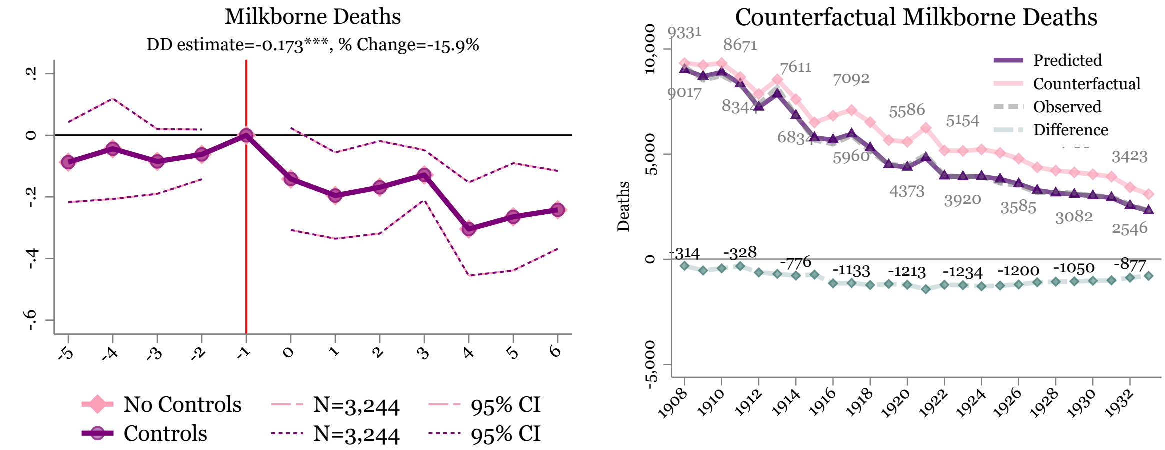
Figure I: Milkborne Outbreaks



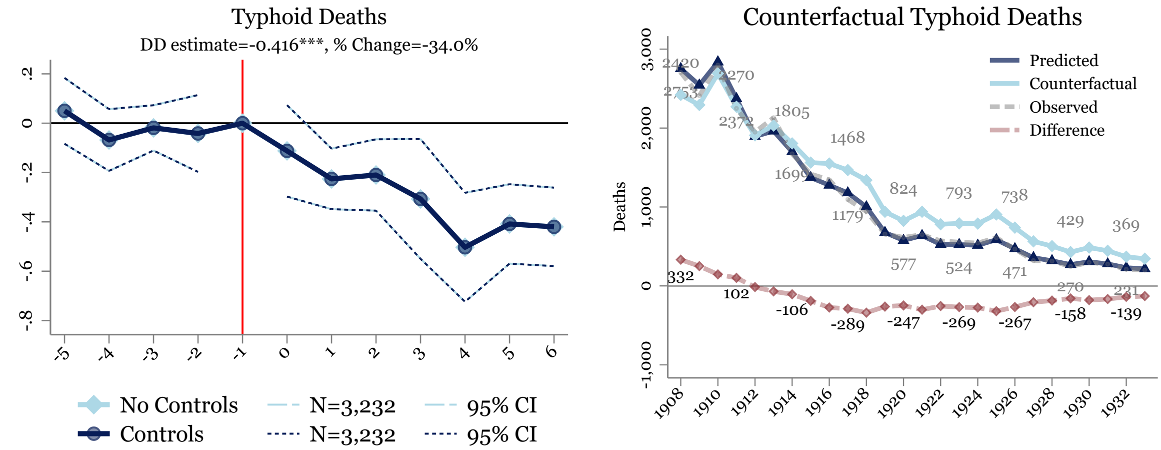
Notes and Sources: Data on outbreaks from Armstrong and Parran (1927), a U.S. Public Health Service report that documents all published milkborne outbreaks in the United States up to 1926. The compilation itself is national in scope and the survey includes outbreaks from cities, towns, and rural areas. Non-typhoid outbreaks include septic sore throat, scarlet fever, diphtheria, and miscellaneous/diarrhea. Typhoid includes paratyphoid. When outbreaks in Armstrong and Parran (1927) are listed as occurring in multiple places, they are counted more than once.

Figure II: Main Results–Pasteurization, Milkborne Deaths and Typhoid Fever

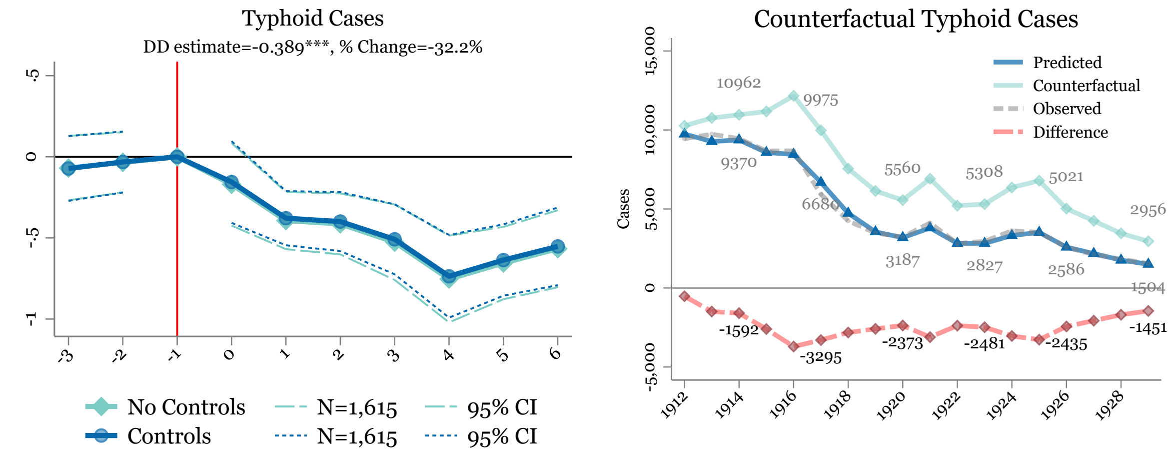
Panel A: Milkborne Deaths



Panel B: Typhoid Deaths

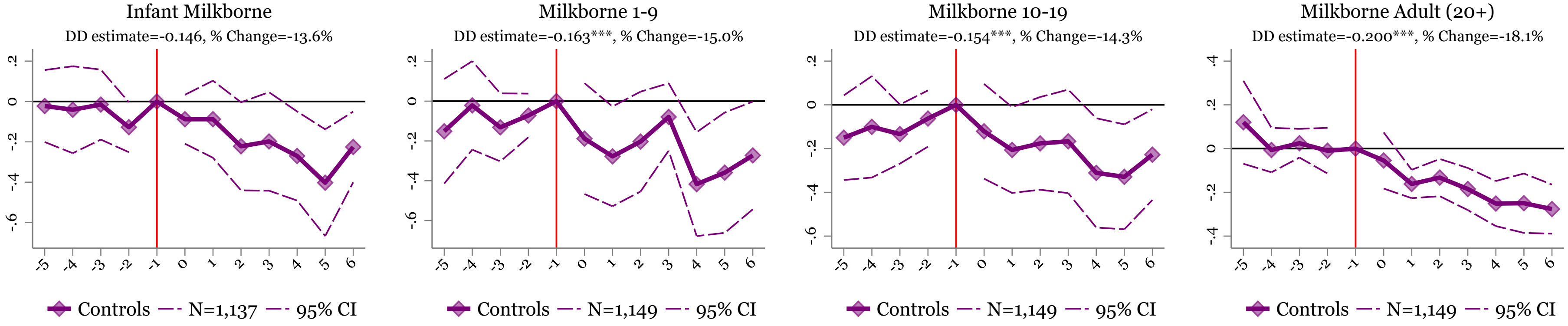


Panel C: Typhoid Cases

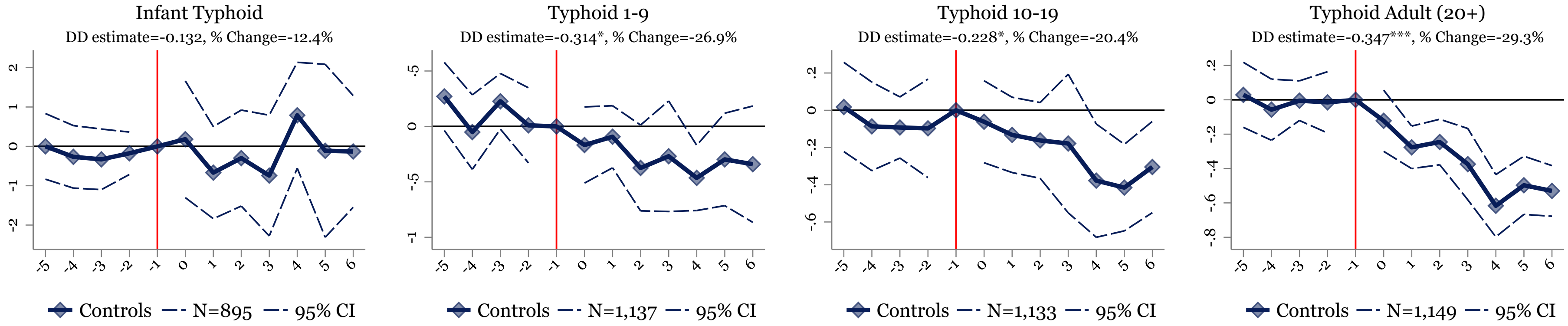


NOTES: For **left graphs**, estimated coefficients from a Poisson model, using the death count (or case count) as the outcome and the exposure set as the population. Baseline fixed effects include year fixed effects and city fixed effects. Plotted coefficients are dummy variables for each year before and after the passage of the pasteurization ordinance. The period just before the ordinance is the excluded period (-1)–indicated by the vertical line. In Panels A-B, the left endpoint is binned at $m = -6$, and the right endpoint is binned at $m = 7$, though we do not display endpoints in the main graphs. In Panel C, because we only have years $t = 1912, \ldots, 1930$ available (versus 1905-1936 for mortality), we consider four years before pasteurization, with the endpoint binned at $m = -4$. Dashed and dotted lines reflect 95% confidence intervals. Robust standard errors clustered at the city level. Controls include the share of the population that is white, the share of the population over 65, the share female, the share foreign, and the physicians per 10,000 persons. Grouped post period from Equation (C). For **right panel**, graphs represent counts after estimating Poisson regression models for deaths and cases. From each model, we generate predicted values based on the full specification with controls. To construct counterfactual predictions, we keep the treated units, and we remove the contribution of pasteurization by subtracting the relevant coefficients from the predicted coefficients. Then both the predicted and counterfactual terms are exponentiated to recover the counterfactual expected counts. The difference between the predicted and counterfactual series captures the estimated treatment effect (red line). We sum these counts for all treated cities across all years.

Figure III: Additional Results–Pasteurization and Age-Specific Mortality
Panel A: Milkborne Mortality



Panel B: Typhoid Mortality



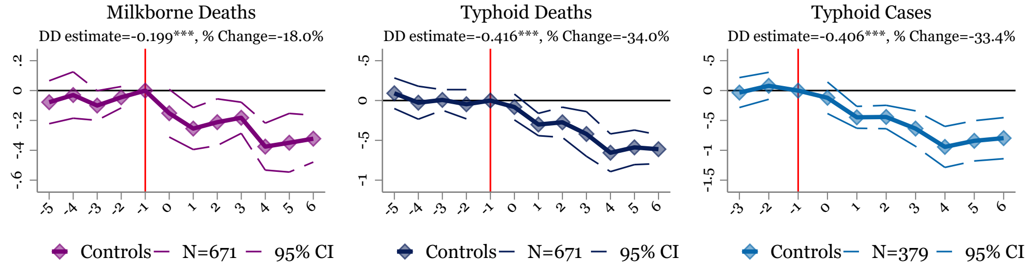
NOTES: Reflects Figure II except considering the age-specific mortality rates. Here the exposure is the population in each age group. See Figure A.13 for finer ages. We combined ages here into larger buckets to have more cases of each illness in each age group.

Figure IV: Robustness Check–Clean Subsamples

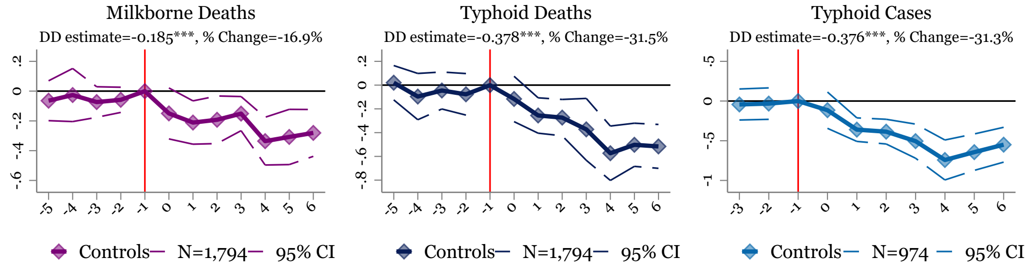
NOTES: Reflects Figure II except with modifications. Panel A removes cities that invested in water disinfection (i.e., chlorination, from USPHS (1926)) within seven years of pasteurization (before or after). Panel B drops cities that invested in water purification technology (i.e., filtration, from USPHS (1926)). Panel C omits cities with both recent disinfection efforts (within seven years of pasteurization) and cities that adopted purification in the sample period (USPHS, 1926). In Panel D, we perform a similar check, where we remove cities that reported filtration in the *Filtration Plant Census, 1924* (August, 1925). Panel E removes cities that passed bacteriological standards or tuberculin testing in the same year as pasteurization, based on Anderson et al. (2022).

Figure V: Additional Robustness Checks

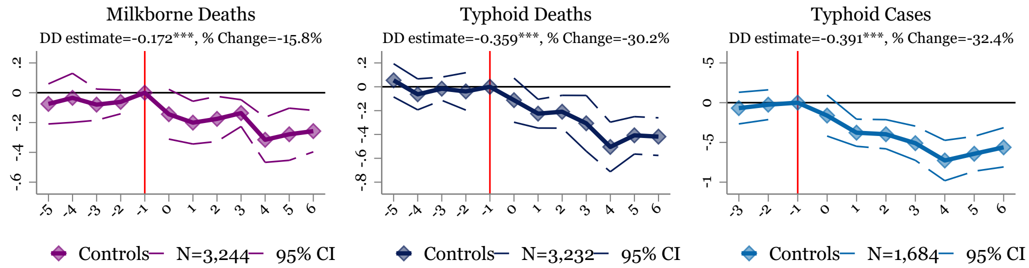
Panel A: Anderson et al Public Health Controls



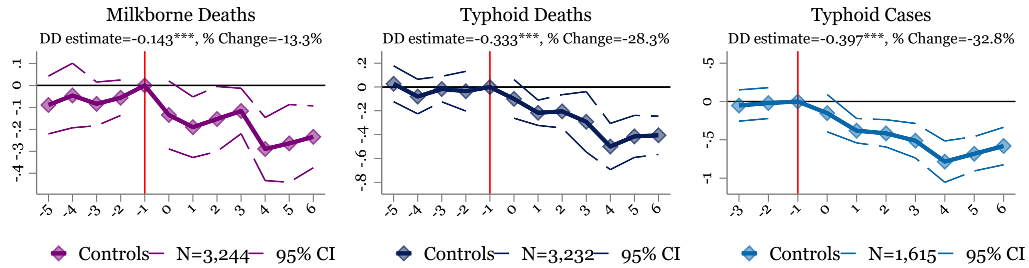
Panel B: Purification and Disinfection Controls



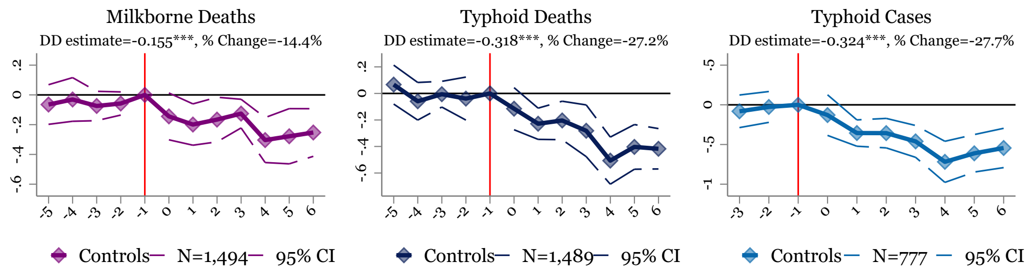
Panel C: No Population Weights



Panel D: Population Size x Year FE



Panel E: Control Group No Pasteurization Ordinance



NOTES: Reflects Figure II except with modifications. Panel A adds controls from Anderson et al. (2022), subsetting to a smaller group of cities. Panel B controls for an expanded set of water purification and disinfection interventions from USPHS (1926). Panel C omits population weights. Panel D adds population-quartile-by-year fixed effects. Panel E include cities without verified pasteurization ordinances at the end of the sample based on both our research and Fuchs et al. (1939).

11 Tables

Table 1: Pasteurization Date and Pasteurization Rates - Treatment Cities

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)	(13)	(14)
	City	Year	Rate 1905	Rate 1910	Rate 1911	Rate 1912	Rate 1913	Rate 1914	Rate 1915	Rate 1916	Rate 1921	Rate 1924	Rate 1930	Rate 1931
1	New York	1912	0				60			100	98	98	98	98
2	Chicago	1914					80	85	85	100	98	99	100	100
3	Philadelphia	1914									98		99	100
4	Detroit	1915	0	1	1	3	5	10	100	100	98	99		100
5	Cleveland	1916									98	98	99	99
6	Saint Louis	1915								80	92	98		100
7	Baltimore	1917							56	65	98	98	98	99
8	San Francisco	1916									85	97	98	96
9	Milwaukee	1916	20	50	60	70	85	85	90	92	98	100		100
10	Buffalo	1918	25	25	25	25	25	25	35	90	100		100	100
11	Minneapolis	1920								60	94	96	96	96
12	Cincinnati	1912									98	98	100	100
13	Indianapolis	1916	10	20	30	40	50	55	70	90				98
14	Rochester	1922	0	10	10	15	15	20	25	25	65	95	97	98
15	Jersey City	1915	0	0	0	0	0	0	95	95			89	98
16	Toledo	1915										100		100
17	Syracuse	1924							25	38	66	92	97	99
18	Dayton	1919				50	55	60	65	75	95	95	98	100
19	Grand Rapids	1917									90	90	93	96
20	Hartford	1924									70		86	89
21	Scranton	1920	10	10	10	10	10	50	75	85		90	99	95
22	Norfolk	1931									50			100
23	Trenton	1922									60			100
24	New Bedford	1925									40	98	99	98
25	Reading	1920						25	35	50	96	95		99
26	Wilkes-Barre	1922												85
27	Altoona	1914									97	97	99	99
28	Racine	1923									85		100	100
29	Charleston	1919									100			100
30	Madison	1928										75	95	96
31	Hamilton	1919									100			100
32	Poughkeepsie	1921									95	85		
33	Ogden	1924									50	85		80
34	Norwood	1915										97		
35	Elyria	1917									85		100	100
36	Framingham	1926									50	55		85
37	Vallejo	1922										90		
38	Waycross	1930										0	100	100

Notes and Sources: See online appendix for details on pasteurization dates. Pasteurization rates from Ayers (1916, 1922, 1926, 1932); Boudouin (1918); Frank (1933).

Table 2: Summary Statistics by Adoption of a Pasteurization Ordinance

	Pasteurization Ordinance		No Ordinance		Difference Treatment-Control
	Mean	*Std. Dev.*	*Mean*	*Std. Dev.*	*Est.*
Mortality					
Overall Deaths Per 10,000	140.90	33.35	149.84	44.41	-8.94***
Infectious Deaths Per 10,000	41.42	20.32	44.78	25.63	-3.36***
Under 5 Deaths Per 1,000 Under 5	30.53	10.94	30.31	11.78	0.22
Infant Deaths Per 1,000 Under 1	100.96	38.40	97.39	38.27	3.58*
By-Cause					
TB Deaths Per 10,000	10.95	6.30	13.11	10.28	-2.16***
Non-Pulmon. Tuberculosis Rate	1.63	0.91	1.67	1.08	-0.04
Typhoid Deaths Per 10,000	1.07	1.44	1.42	1.75	-0.34***
Typhoid Cases Per 10,000	3.47	4.63	5.66	7.07	-2.20***
Diphtheria Deaths Per 10,000	1.36	1.29	1.08	1.05	0.28***
Scarlet Fever Deaths Per 10,000	0.53	0.74	0.30	0.49	0.24***
Milkborne Deaths Per 10,000	3.24	2.32	3.38	2.54	-0.15
Milkborne Deaths (No Typhoid) Per 10,000	1.61	1.75	1.71	1.88	-0.11
Characteristics					
Share White	0.93	0.11	0.85	0.15	0.08***
Share Over 65	0.04	0.01	0.04	0.02	0.00
Share Foreign	0.18	0.10	0.13	0.11	0.05***
Physicians per 10,000	0.00	0.00	0.00	0.00	-0.00***
Share Females	0.50	0.02	0.51	0.02	-0.01***
N	1,216		2,496		3,712

SOURCES: City-level demographic characteristics are calculated from the IPUMs Restricted Complete Count U.S. Census data. Mortality statistics are from *US Bureau of the Census, United States Vital Statistics Division (1890-1938)*, with the population numbers interpolated between census years. Case counts of key illnesses reported in large cities and small cities from USPHS (1912-1929). Separate volumes are published for small cities (population 10,000–100,000, available 1912–1929) and large cities (over 100,000, available 1912–1930). Pasteurization ordinances from a variety of sources; see online appendix for full description. Mortality rates are per 10,000 persons. Age-specific rates are per the relevant populations.

Table 3: Pasteurization and Number of Outbreaks, Poisson Model

	Milkborne Outbreaks		Typhoid Outbreaks		Non-Typhoid Outbreaks	
	(1)	(2)	(3)	(4)	(5)	(6)
1(Pasteurization Ordinance)	-1.2597**	-1.0323**	-1.1422*	-0.9055	-1.9050*	-1.8945
	(0.5550)	(0.5255)	(0.6096)	(0.5802)	(1.0085)	(1.4702)
N	641	641	536	536	119	119
City and Year FE	X	X	X	X	X	X
Controls		X		X		X

NOTES: Reflects Table C.1 except considering the number of outbreaks in a city based on Armstrong and Parran (1927). Only includes years 1906-1926. See Figure I and Figure A.3 for descriptions of the outbreaks. In Columns (1) and (2), outbreaks include both typhoid and other illnesses. Grouped post period from Equation (C).

Table 4: Interaction of Pasteurization and Water Treatment

	Milkborne Deaths			Typhoid Deaths			Typhoid Cases		
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
Panel A: Purification Interaction									
1(Pasteurization)=1	-0.2446***	-0.2182***	-0.1726***	-0.3821***	-0.3909***	-0.2618***	-0.4357***	-0.3646***	-0.2703***
	(0.0386)	(0.0427)	(0.0565)	(0.1304)	(0.1072)	(0.0674)	(0.1030)	(0.0872)	(0.0695)
1(Pasteurization)=1 × 1(Water Purification)=1	0.1245**	0.0892*	0.1116	-0.0685	0.0326	0.0832	-0.0063	-0.0227	0.0231
	(0.0627)	(0.0464)	(0.0687)	(0.1831)	(0.1582)	(0.1256)	(0.1505)	(0.1484)	(0.1219)
1(Water Purification)=1	-0.1247**	-0.1328**	-0.1631***	-0.2499*	-0.3350***	-0.3155***	-0.1703	-0.1265	-0.1319**
	(0.0587)	(0.0583)	(0.0445)	(0.1317)	(0.1220)	(0.0892)	(0.1089)	(0.1073)	(0.0664)
1(Water Disinfection)	0.0512	0.0440	0.0306	0.0293	0.0310	0.0244	-0.0770	-0.1206	0.0074
	(0.0359)	(0.0374)	(0.0353)	(0.0654)	(0.0659)	(0.0493)	(0.0883)	(0.0906)	(0.1178)
N	1,794	1,794	1,794	1,794	1,794	1,794	974	974	974
Pseudo R-squared	0.949	0.950	0.954	0.863	0.868	0.884	0.897	0.898	0.915
Panel B: Disinfection Interaction									
1(Pasteurization)=1	-0.0842**	-0.0786	-0.0568	-0.3971***	-0.3023***	-0.1070	-0.1200	-0.0987	0.1737
	(0.0412)	(0.0545)	(0.0412)	(0.1464)	(0.0957)	(0.0841)	(0.1284)	(0.0953)	(0.1724)
1(Pasteurization)=1 × 1(Water Disinfection)=1	-0.1175**	-0.1157**	-0.0843**	-0.0150	-0.0797	-0.1297	-0.3355**	-0.2972**	-0.4603**
	(0.0476)	(0.0566)	(0.0401)	(0.1590)	(0.1208)	(0.0965)	(0.1468)	(0.1265)	(0.1801)
1(Water Purification)	-0.0576	-0.0870	-0.1299**	-0.2747**	-0.3230***	-0.3008***	-0.1708	-0.1359	-0.1341**
	(0.0659)	(0.0570)	(0.0535)	(0.1292)	(0.1071)	(0.0925)	(0.1081)	(0.1003)	(0.0655)
1(Water Disinfection)=1	0.0478	0.0422	0.0296	0.0354	0.0311	0.0222	-0.0665	-0.1080	0.0317
	(0.0374)	(0.0384)	(0.0375)	(0.0700)	(0.0698)	(0.0489)	(0.0896)	(0.0935)	(0.1184)
N	1,794	1,794	1,794	1,794	1,794	1,794	974	974	974
Pseudo R-squared	0.949	0.950	0.954	0.863	0.868	0.884	0.897	0.898	0.915
Panel C: Anderson et al Public Health Controls									
1(Pasteurization)=1	-0.2604**	-0.2906***	-0.2094*	-0.7637***	-0.6095***	-0.4538***	-0.2000**	-0.1596	0.0241
	(0.1212)	(0.0985)	(0.1265)	(0.1470)	(0.1274)	(0.1564)	(0.0785)	(0.1018)	(0.0770)
1(Pasteurization)=1 × 1(Water Chlorine)=1	0.0451	0.1009	0.0851	0.2487**	0.2098	0.2526*	-0.2740**	-0.2727**	-0.2418**
	(0.1146)	(0.0917)	(0.1191)	(0.1237)	(0.1438)	(0.1495)	(0.1163)	(0.1175)	(0.1118)
1(Water Chlorine)=1	0.0584	0.0631	-0.0082	-0.0134	-0.0260	-0.1027	0.1980	0.1418	0.0919
	(0.0482)	(0.0525)	(0.0458)	(0.0961)	(0.0627)	(0.0672)	(0.1378)	(0.1257)	(0.1500)
1(TB Testing of Cows)	-0.0222	0.0007	-0.0176	0.1096	0.0357	-0.0712	0.0479	-0.0117	-0.0164
	(0.0658)	(0.0570)	(0.0325)	(0.1551)	(0.1315)	(0.0793)	(0.0804)	(0.0898)	(0.0956)
1(Bacteriological Standard for Milk)	0.1007**	0.0904**	0.0994**	0.1107	0.1186	0.1153	0.1261	0.1289	-0.0294
	(0.0432)	(0.0398)	(0.0409)	(0.1105)	(0.1050)	(0.0835)	(0.1079)	(0.0972)	(0.0985)
1(Sewage Treatment/Diversion)	0.0699	0.0495	-0.0940**	0.0383	0.0896	0.0757	0.0093	0.0574	0.2222
	(0.0799)	(0.0744)	(0.0449)	(0.1484)	(0.1190)	(0.0707)	(0.1547)	(0.1527)	(0.2422)
1(Water Filtration)	-0.0093	-0.0382	-0.1595***	-0.2625*	-0.2524**	-0.3843***	-0.0227	0.0454	0.0039
	(0.0680)	(0.0497)	(0.0602)	(0.1457)	(0.1220)	(0.0599)	(0.1968)	(0.1845)	(0.1418)
N	671	671	671	671	671	671	379	379	379
Pseudo R-squared	0.951	0.952	0.959	0.882	0.888	0.909	0.910	0.912	0.929
Panel D: Anderson et al Public Health Controls									
1(Pasteurization)=1	-0.2623***	-0.2298***	-0.1823***	-0.4845***	-0.4191***	-0.2239***	-0.4069***	-0.3247***	-0.1240
	(0.0559)	(0.0531)	(0.0478)	(0.1757)	(0.1271)	(0.0819)	(0.1221)	(0.0996)	(0.0960)
1(Pasteurization)=1 × 1(Water Filtration)=1	0.1303	0.1028*	0.1605***	-0.1198	0.0092	-0.0059	-0.1259	-0.2045	-0.1743
	(0.0839)	(0.0584)	(0.0595)	(0.2035)	(0.1754)	(0.1202)	(0.1346)	(0.1484)	(0.1314)
1(Water Filtration)=1	-0.0815*	-0.0898*	-0.2085***	-0.2248*	-0.2580***	-0.3836***	0.0484	0.1536	0.0869
	(0.0466)	(0.0478)	(0.0492)	(0.1241)	(0.0954)	(0.0556)	(0.1839)	(0.2014)	(0.1421)
1(TB Testing of Cows)	0.0029	0.0144	-0.0131	0.0804	0.0323	-0.0787	0.0419	-0.0112	-0.0015
	(0.0602)	(0.0569)	(0.0343)	(0.1312)	(0.1266)	(0.0822)	(0.0851)	(0.0799)	(0.0869)
1(Bacteriological Standard for Milk)	0.0782*	0.0813**	0.0989***	0.1357	0.1230	0.1258	0.1239	0.1069	-0.0705
	(0.0421)	(0.0381)	(0.0356)	(0.1138)	(0.1068)	(0.0850)	(0.1000)	(0.0976)	(0.1012)
1(Sewage Treatment/Diversion)	0.0629	0.0499	-0.0985**	0.0466	0.0890	0.0679	0.0192	0.0648	0.2794
	(0.0792)	(0.0750)	(0.0485)	(0.1517)	(0.1202)	(0.0708)	(0.1497)	(0.1487)	(0.2512)
1(Water Chlorine)	0.0841	0.0935**	0.0193	-0.0057	-0.0075	-0.0839	0.1628	0.0953	0.0596
	(0.0527)	(0.0465)	(0.0454)	(0.1108)	(0.0771)	(0.0745)	(0.1330)	(0.1219)	(0.1456)
N	671	671	671	671	671	671	379	379	379
Pseudo R-squared	0.952	0.953	0.959	0.882	0.887	0.909	0.910	0.912	0.929
City and Year FE	X	X	X	X	X	X	X	X	X
Controls		X	X		X	X		X	X
Ciy Linear Trends			X			X			X

NOTES: Reflects grouped post period described in Section C except adding interaction terms for water cleanliness and pasteurization.

Table 5: Voluntary Pasteurization–City-level Pasteurization Rates and Mortality

	Milkborne Deaths		Typhoid Deaths		Infant Deaths		Under 2 Deaths	
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Panel A: Baseline								
Pasteurization Rate	-0.0006	-0.0009	-0.0011	-0.0031**	-0.0016***	-0.0016***	-0.0017***	-0.0012**
	(0.0008)	(0.0007)	(0.0015)	(0.0013)	(0.0005)	(0.0005)	(0.0006)	(0.0006)
N	612	612	592	592	575	575	284	284
Pseudo R-squared	0.958	0.959	0.876	0.879	0.991	0.992	0.991	0.992
Panel B: Pasteurization Controls								
Pasteurization Rate	0.0006	0.0002	0.0005	-0.0017	-0.0012***	-0.0013**	-0.0013***	-0.0012*
	(0.0008)	(0.0008)	(0.0013)	(0.0011)	(0.0004)	(0.0005)	(0.0005)	(0.0006)
1(Pasteurization Ordinance)	-0.1951***	-0.2077***	-0.3085***	-0.3520***	-0.0772**	-0.0518	-0.0707*	-0.0456
	(0.0629)	(0.0622)	(0.0854)	(0.0972)	(0.0370)	(0.0361)	(0.0410)	(0.0358)
N	535	535	521	521	503	503	263	263
Pseudo R-squared	0.960	0.960	0.878	0.881	0.992	0.992	0.992	0.993
Panel C: State x Year FE								
Pasteurization Rate	-0.0004	-0.0009	-0.0053***	-0.0055***	-0.0037***	-0.0039***	-0.0041***	-0.0040***
	(0.0008)	(0.0007)	(0.0016)	(0.0016)	(0.0007)	(0.0008)	(0.0009)	(0.0006)
N	504	504	486	486	476	476	183	183
Pseudo R-squared	0.966	0.967	0.888	0.888	0.993	0.993	0.995	0.995
Panel D: Purification and Disinfection Controls								
Pasteurization Rate	-0.0003	0.0002	0.0007	-0.0013	-0.0016***	-0.0014*	-0.0019***	-0.0012
	(0.0009)	(0.0010)	(0.0017)	(0.0018)	(0.0006)	(0.0007)	(0.0006)	(0.0008)
1(Water Disinfection)	0.0243	-0.0261	-0.2209*	-0.2317*	0.0174	-0.0200	0.0410	-0.0069
	(0.0708)	(0.0721)	(0.1176)	(0.1195)	(0.0435)	(0.0387)	(0.0475)	(0.0459)
1(Water Purification)	-0.1490	-0.2277***	-0.5697***	-0.5598***	-0.0725	0.0016	-0.0321	0.0313
	(0.1054)	(0.0798)	(0.1231)	(0.1014)	(0.0772)	(0.0397)	(0.0858)	(0.0459)
N	314	314	314	314	305	305	276	276
Pseudo R-squared	0.954	0.956	0.871	0.875	0.991	0.991	0.991	0.992
Panel E: Anderson et al Public Health Controls								
Pasteurization Rate	-0.0009	-0.0011	-0.0013	-0.0037*	-0.0018**	-0.0020**	-0.0017**	-0.0017**
	(0.0012)	(0.0014)	(0.0015)	(0.0022)	(0.0007)	(0.0008)	(0.0008)	(0.0007)
1(Water Filtration)	-0.1043	-0.1893**	-0.4729***	-0.4958***	-0.0710	-0.0209	-0.0262	0.0152
	(0.0726)	(0.0924)	(0.1163)	(0.0873)	(0.0618)	(0.0392)	(0.0727)	(0.0431)
1(Water Chlorine)	-0.0786	-0.0571	-0.2276*	-0.2610**	0.0462	0.0082	0.0473	0.0086
	(0.0857)	(0.0930)	(0.1263)	(0.1046)	(0.0437)	(0.0325)	(0.0479)	(0.0374)
1(Bacteriological Standard for Milk)	0.1849*	0.1618	-0.0130	0.1873	-0.0675	0.0036	-0.0561	0.0157
	(0.1044)	(0.0988)	(0.1581)	(0.1250)	(0.0843)	(0.0571)	(0.0955)	(0.0613)
1(TB Testing of Cows)	0.1120	0.0832	0.2304	0.0993	0.0824*	0.0446	0.0651	0.0356
	(0.0745)	(0.0941)	(0.1438)	(0.1340)	(0.0440)	(0.0353)	(0.0481)	(0.0358)
1(Sewage Treatment/Diversion)	0.0971	-0.0271	0.3342**	0.3139**	0.0385	0.0629	0.0443	0.0516
	(0.1161)	(0.1046)	(0.1497)	(0.1409)	(0.0353)	(0.0441)	(0.0413)	(0.0409)
N	138	138	138	138	137	137	137	137
Pseudo R-squared	0.953	0.956	0.874	0.880	0.991	0.992	0.992	0.993
Panel F: Log-level Specification								
Pasteurization Rate	-0.0009	-0.0005	-0.0001	-0.0015	-0.0016***	-0.0014***	-0.0016**	-0.0011*
	(0.0008)	(0.0008)	(0.0020)	(0.0020)	(0.0006)	(0.0005)	(0.0007)	(0.0005)
N	609	609	496	496	575	575	284	284
Adjusted R-squared	0.841	0.846	0.846	0.856	0.889	0.908	0.921	0.941
City and Year FE	X	X	X	X	X	X	X	X
Controls		X		X		X		X

NOTES: Estimated coefficients from a city-level Poisson model. In Columns (1)-(2), the outcome is milkborne deaths, and the exposure is the city-level population. In Columns (3)-(4), the outcome is typhoid deaths, and the exposure is the city-level population. In Columns (5)-(6), the outcome is infant deaths, and the exposure is the population under one. In Columns (7)-(8), the outcome is under-two deaths, with the exposure the population under age two. Includes pasteurization rates from Ayers (1916, 1922, 1926, 1932); Boudouin (1918); Frank (1933). City and year fixed effects included. Robust standard errors clustered at the city level. ***, **, * represent statistical significance at 1, 5, and 10 percent levels. The sample includes only cities that reported their pasteurization rates in 1921, the first comprehensive year pasteurization rates were reported, and 1931, the last comprehensive year. We do not include city-level trends because specifications with certain controls and state-by-year fixed effects have too few observations. Panel A reflects the baseline Poisson model, Panel B controls for the pasteurization ordinances, omitting cities with known pasteurization ordinances but for which we are missing the date (from Fuchs et al. (1939)). Panel C adds state-by-year fixed effects. Panel D adds water purification and disinfection controls from USPHS (1926). Panel E adds controls from Anderson et al. (2022), subsetting to a smaller group of cities. Panel F presents the estimates from a log-linear OLS specification.

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Online Appendix

A Appendix

Figure A.1: Example Public Health Notices
Panel A: How to Buy Safe Milk

Panel B: How to Pasteurize Milk

SOURCE: Ward et al. (2007).

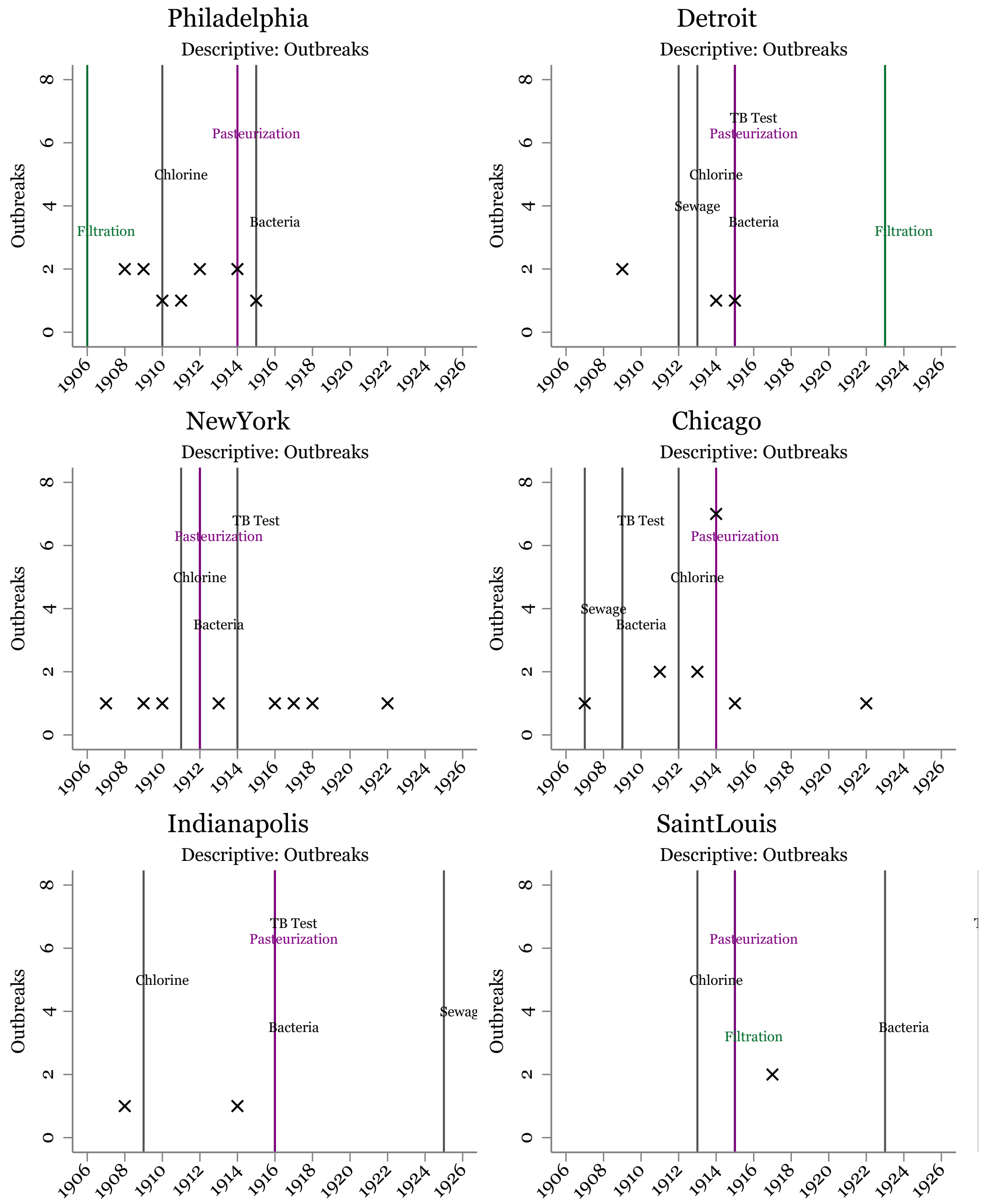
Figure A.2: The Problem of Unclean Milk

"I DRINK TO THE GENERAL DEATH OF THE WHOLE TABLE."

This cartoon was awarded first prize by the American Medical Association.

SOURCE: Straus and Straus (1913, pg. 2).

Figure A.3: Milkborne Outbreaks by City



NOTES AND SOURCES: Armstrong and Parran (1927). Only includes select cities.

Table A.1: Rates of Pasteurization - Control Cities

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)	(13)
	City	Rate 1905	Rate 1910	Rate 1911	Rate 1912	Rate 1913	Rate 1914	Rate 1915	Rate 1916	Rate 1921	Rate 1924	Rate 1930	Rate 1931
1	Washington	0	5	20	35	50	65	80	85	91	95	97	97
2	New Orleans										20	64	
3	Kansas City									50	50	50	50
4	Seattle						67	70	75	85	86		38
5	Louisville									85		98	96
6	Portland								50	55	67		75
7	Houston									50	67		
8	Saint Paul								60	60		81	80
9	Atlanta								0			58	
10	Dallas									70			30
11	Birmingham										65	48	50
12	Memphis									50	50		74
13	San Antonio												69
14	Omaha									30			70
15	Hempstead												
16	Oklahoma City									75			
17	Nashville									40		66	60
18	San Diego											79	76
19	Long Beach									80	80	79	77
20	Tulsa										50	75	
21	Paterson									80		76	
22	Jacksonville									70	50	44	40
23	Kansas City									65	25	60	65
24	Spokane	7	60	60	60	65	70	80	85		80	80	
25	Wichita										80		66
26	Miami										75	65	70
27	Tacoma												50
28	Knoxville										33	61	56
29	Peoria												
30	El Paso									33	75		59
31	Duluth									46	87	53	58
32	Tampa									8		70	70
33	Lowell									34		77	
34	Waterbury										40	59	
35	Lawrence										85		
36	Savannah										1	40	33
37	Charlotte												26
38	Little Rock									50			22
39	Saint Joseph									46	50		47
40	Saginaw									41	45	75	
41	Pawtucket												
42	Shreveport											50	
43	Pasadena												52
44	Lincoln									70	76	75	80
45	Huntington											67	
46	Winston-Salem									50	50		47
47	East Saint Louis												
48	Troy											38	40
49	Mobile										5		15
50	New Britain									20	70	68	68
51	East Orange												
52	Atlantic City												
53	Montgomery									50	14	21	23
54	Topeka									40	5	33	50
55	Glendale												
56	Wheeling									75	72	76	76
57	Davenport										55	75	80
58	Charleston										75	74	35
59	Augusta									20		19	25
60	Lancaster									50	80	65	70
61	Medford										80		
62	Hoboken												
63	Beaumont										2		54
64	San Jose									70	67		67
65	Springfield											42	49
66	Decatur										45		
67	Irvington												
68	Hamtramck												
69	New Rochelle												
70	Macon										25		30
71	Greensboro											39	
72	Galveston									15		68	80
73	Waco											56	50
74	Durham									15			58
75	Columbia									15			20
76	Dearborn												
77	Asheville										75		70
78	Pueblo									55		61	60

Sources: Pasteurization rates from Ayers (1916, 1922, 1926, 1932); Boudouin (1918); Frank (1933).

Table A.2: Milk Control and Water Infrastructure Investment Dates

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)
		Milk Pasteurization	Milk Bac. Standards	Milk TB Testing	Water Filtation	Water Chlorination	Sewage Investment	Water Purification	Water Disinfection	Water Sand Filtation
1	New York	1912	1912	1914		1911	1937		1910	
2	Cincinnati	1912	1914	1907	1907	1918		1907	1911	1907
3	Philadelphia	1914	1915	1930	1906	1910		1912	1913	1902
4	Chicago	1914	1909	1909		1912	1907		1912	
5	Altoona	1914								
6	Jersey City	1915	1915	1915		1908	1924	1902	1902	
7	Saint Louis	1915	1923	1928	1915	1913		1904	1908	1914
8	Detroit	1915	1915	1915	1923	1913	1912	1923	1913	1923
9	Toledo	1915						1910	1910	1910
10	Norwood	1915								
11	Indianapolis	1916	1916	1916	1904	1909	1925	1904	1910	1903
12	Milwaukee	1916	1908	1908	1939	1910	1925		1910	
13	Cleveland	1916	1906	1906	1918	1911	1922	1919	1911	1917
14	San Francisco	1916	1909	1909		1922			1923	1910
15	Baltimore	1917	1913	1917	1915	1911	1911	1915	1911	1914
16	Grand Rapids	1917						1913	1913	1913
17	Elyria	1917								1903
18	Buffalo	1918	1918		1926	1914	1938		1914	
19	Dayton	1919							1914	
20	Charleston	1919								1902
21	Hamilton	1919								
22	Minneapolis	1920	1907	1895	1913	1910	1938	1913	1910	1911
23	Scranton	1920						1910	1908	1910
24	Reading	1920						1903	1921	1903
25	Poughkeepsie	1921								1874
26	Rochester	1922	1907	1922		1925	1917			
27	Trenton	1922						1916	1911	1914
28	Wilkes-Barre	1922						1918		1895
29	Vallejo	1922								
30	Racine	1923								
31	Hartford	1924						1918	1913	
32	Syracuse	1924							1919	
33	Ogden	1924								
34	New Bedford	1925								
35	Framingham	1926								
36	Madison	1928								
37	Waycross	1930								
38	Norfolk	1931								

Notes and Sources: Pasteurization dates based on our collection of dates, see the online appendix for details. Anderson et al. (2022) the source for bacteriological standards, tuberculin testing of cow herds, and filtration/chlorination. Water purification/disinfection from USPHS (1926). Rapid and slow sand dates from *Filtration Plant Census, 1924* (August, 1925).

Table A.3: Adoption of a Pasteurization Ordinance

	Adoption of Pasteurization		
	(1)	(2)	(3)
L.Log of Milkborne Mortality	-0.2570		
	(0.5653)		
L.Log of Typhoid Mortality Rate		-0.1870	
		(0.3297)	
L.Log of Typhoid Case Rate			-0.2014
			(0.3288)
Log of Population	1.2995***	1.4547***	1.4164***
	(0.2694)	(0.2913)	(0.3197)
Share White	9.3229*	7.1966	18.0826***
	(4.7902)	(4.7955)	(4.9827)
Share Over 65	-6.7686	16.0410	-9.5593
	(24.1000)	(26.2735)	(28.1092)
Share Foreign	-8.9371***	-9.7343***	-12.4012***
	(3.4592)	(3.6504)	(3.7016)
Physicians per 10,000	-1.70e+03***	-2.44e+03***	-1.81e+03***
	(611.2801)	(600.4868)	(699.5000)
Share Females	-10.7204	-22.4904*	-17.3746
	(9.1164)	(13.0240)	(14.5412)
N	2,518	2,292	1,240
Pseudo R-squared	0.155	0.184	0.195
City and Year FE	X	X	X

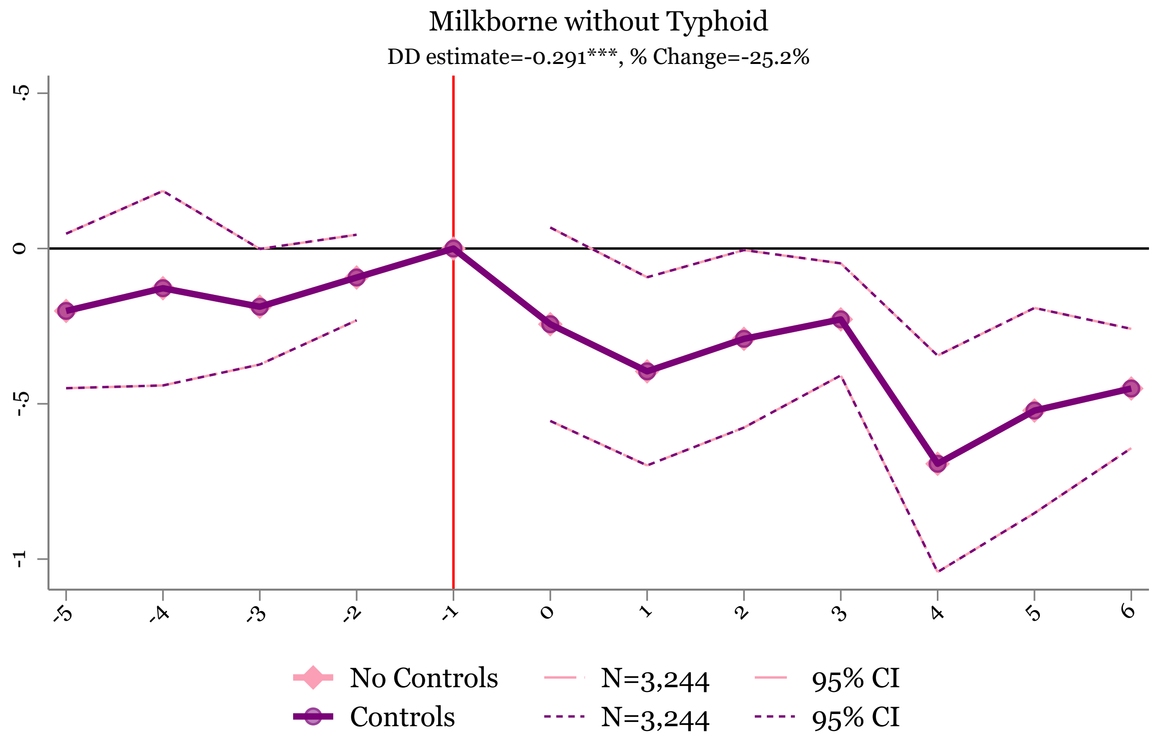
NOTES: Estimates from an OLS regression that uses an indicator in the year that the pasteurization ordinance was adopted. The sample stops in the year of adoption. The OLS regression considers whether the log of the previous year's typhoid mortality and case rate predicts the timing of a pasteurization law. L. denotes the lag. Robust standard errors clustered at the city level. ***, **, * represent statistical significance at 1, 5, and 10 percent levels.

Table A.4: First Stage Effects of Pasteurization Ordinance on Pasteurization Rates

	Weighted		Unweighted	
	Outcome: Pasteurization Rate			
	(1)	(2)	(3)	(4)
1(Pasteurization Ordinance)	17.7805**	17.1982**	16.2765***	15.7045***
	(8.8139)	(7.3232)	(5.3177)	(4.9236)
N	899	899	1,128	1,126
Adjusted R-squared	0.769	0.789	0.746	0.749
City and Year FE	X	X	X	X
Controls		X		X

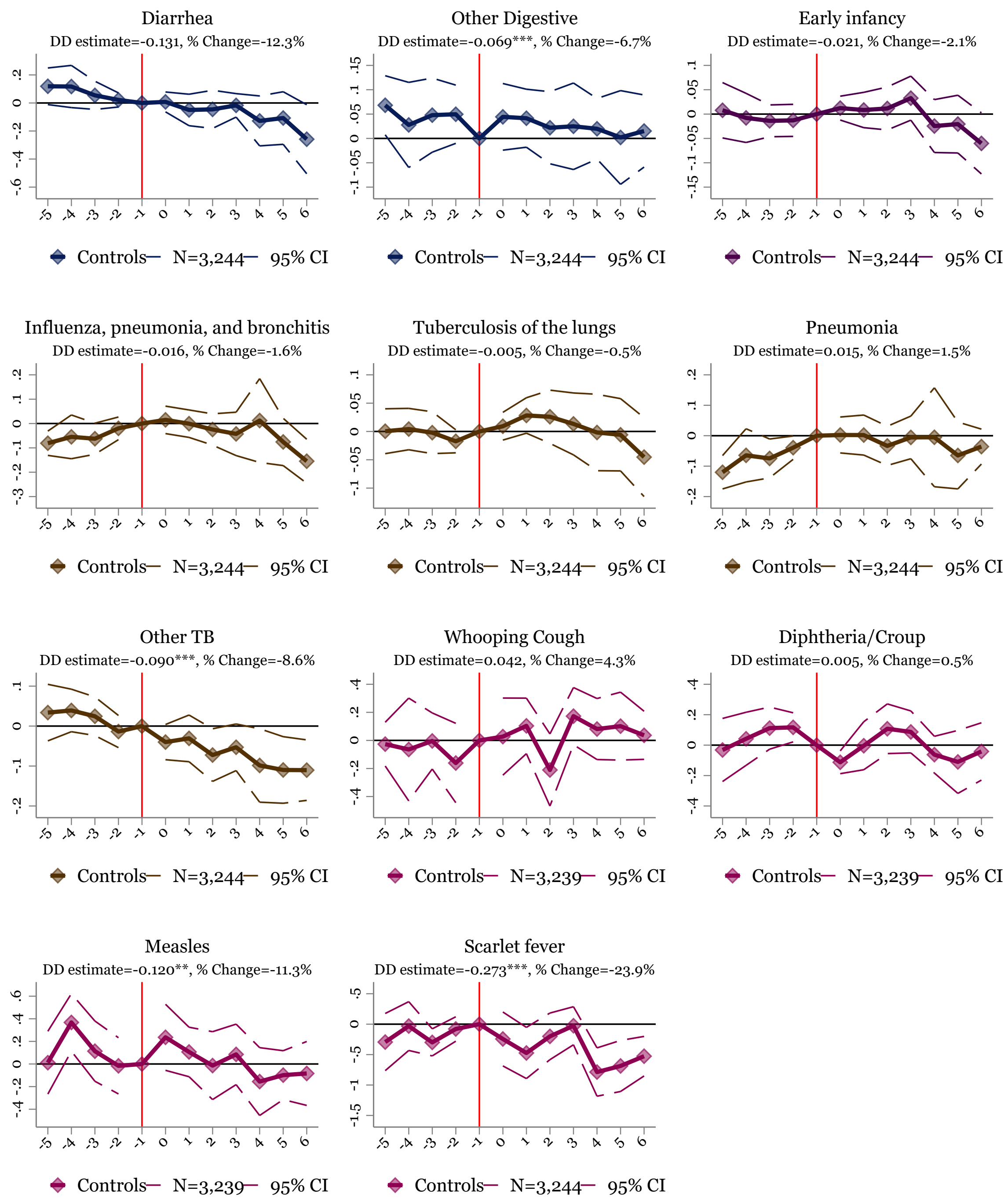
NOTES: Pasteurization rates for select years from Ayers (1916, 1922, 1926, 1932), Boudouin (1918), and Frank (1933). Estimates from an OLS regression that uses an indicator in the year that the pasteurization ordinance was adopted. The OLS regression considers whether mandatory pasteurization impacts the pasteurization rate in a city. Robust standard errors clustered at the city level. ***, **, * represent statistical significance at 1, 5, and 10 percent levels.

Figure A.4: Pasteurization and Milkborne Mortality–Excluding Typhoid



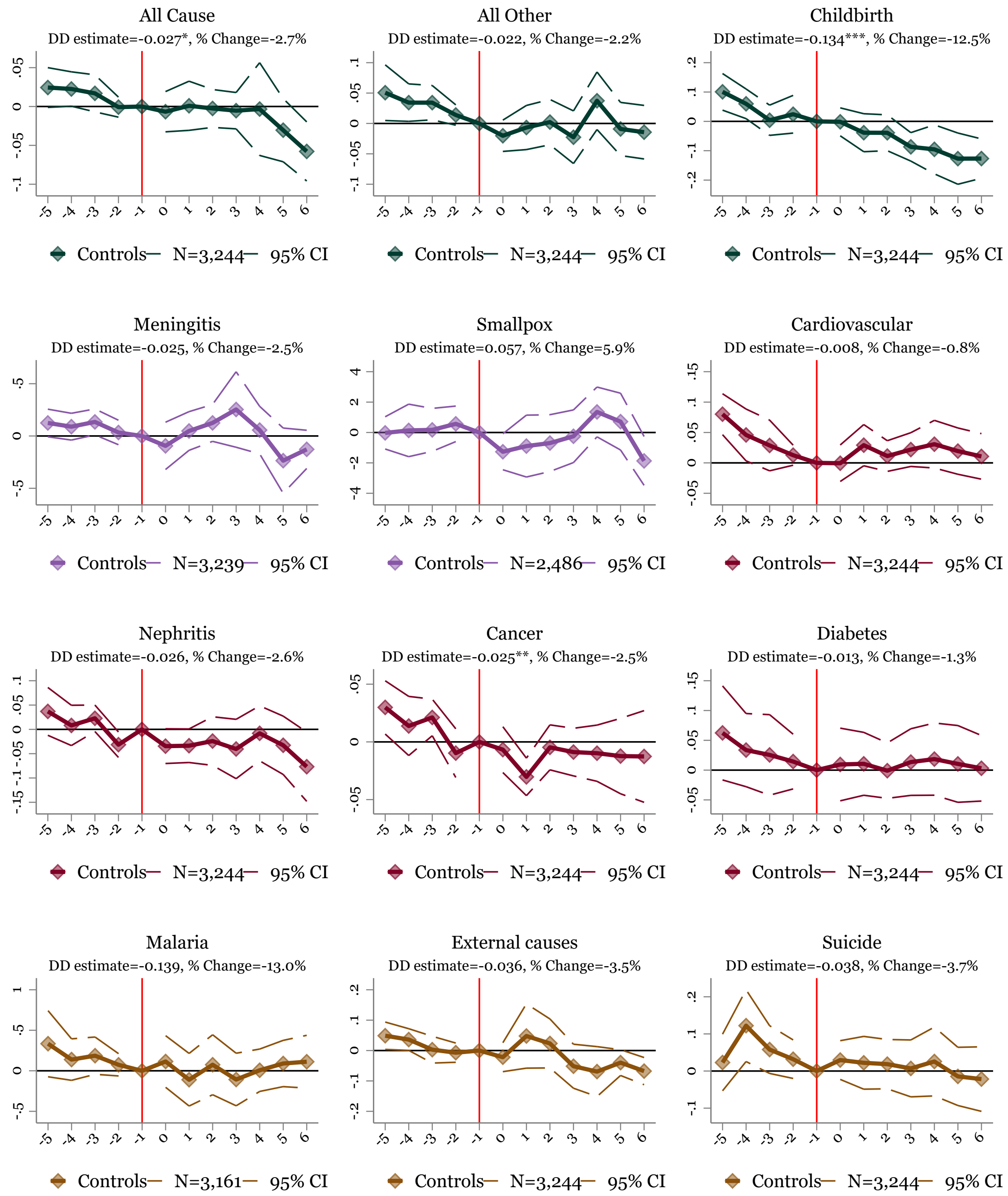
NOTES: Reflects the Poisson specification from Figure II except modifying the outcome to include deaths from non-pulmonary tuberculosis and scarlet fever.

Figure A.5: Pasteurization–Other Causes of Death I, Poisson Specification



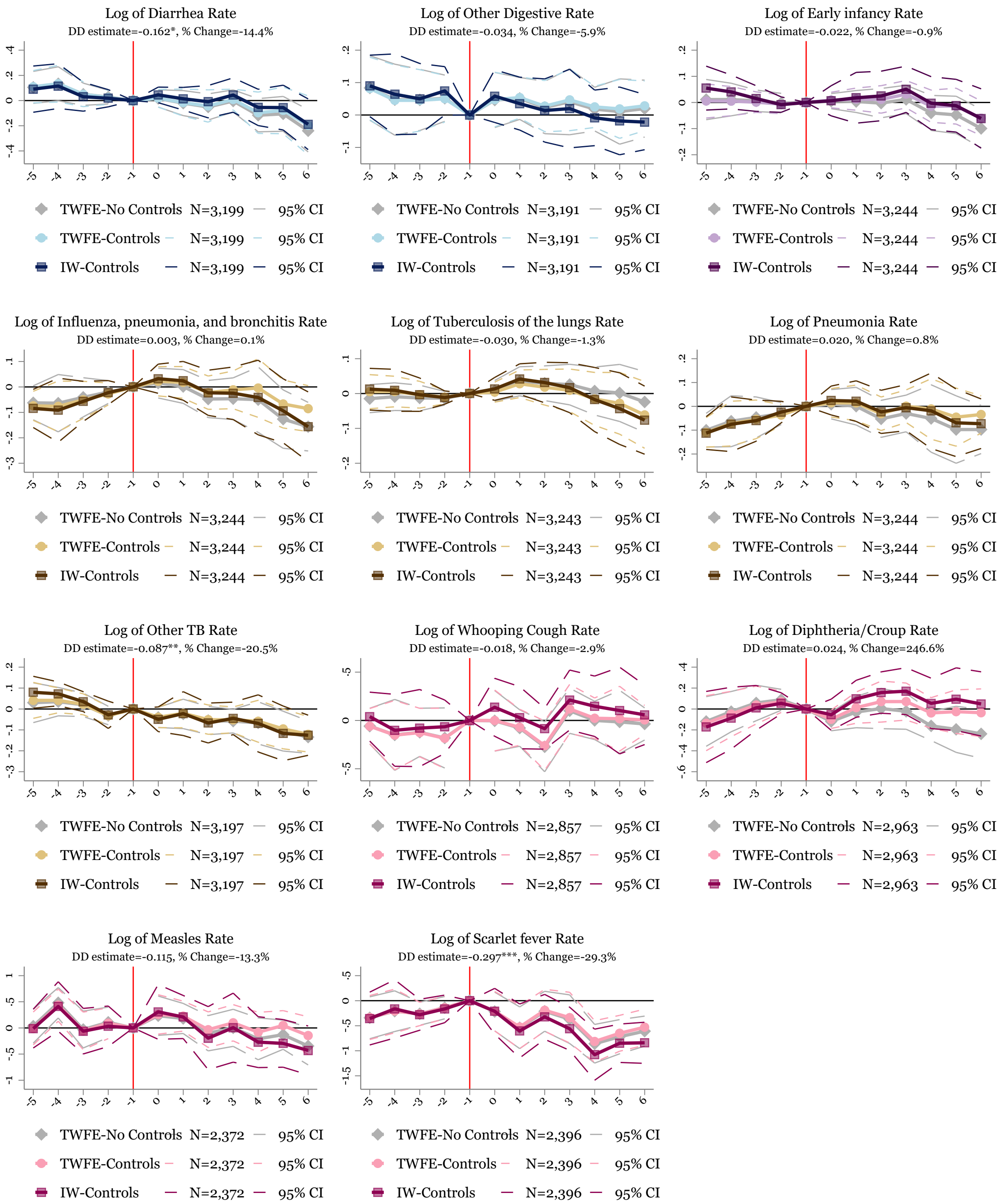
NOTES: Reflects Figure II except modifying the outcome in each graph.

Figure A.6: Pasteurization–Other Causes of Death II, Poisson Specification



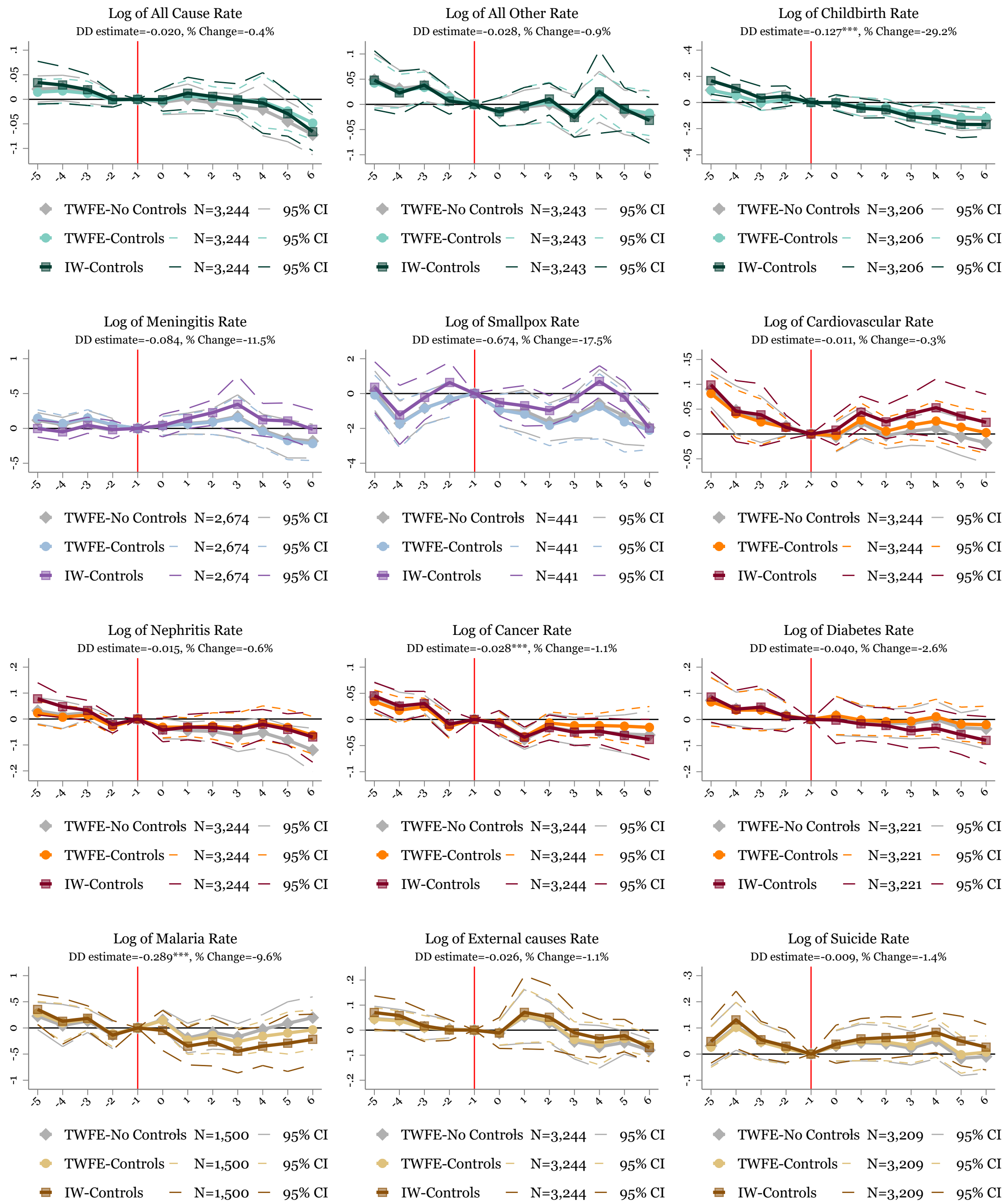
NOTES: Reflects Figure II except modifying the outcome in each graph.

Figure A.7: Pasteurization–Other Causes of Death I, OLS



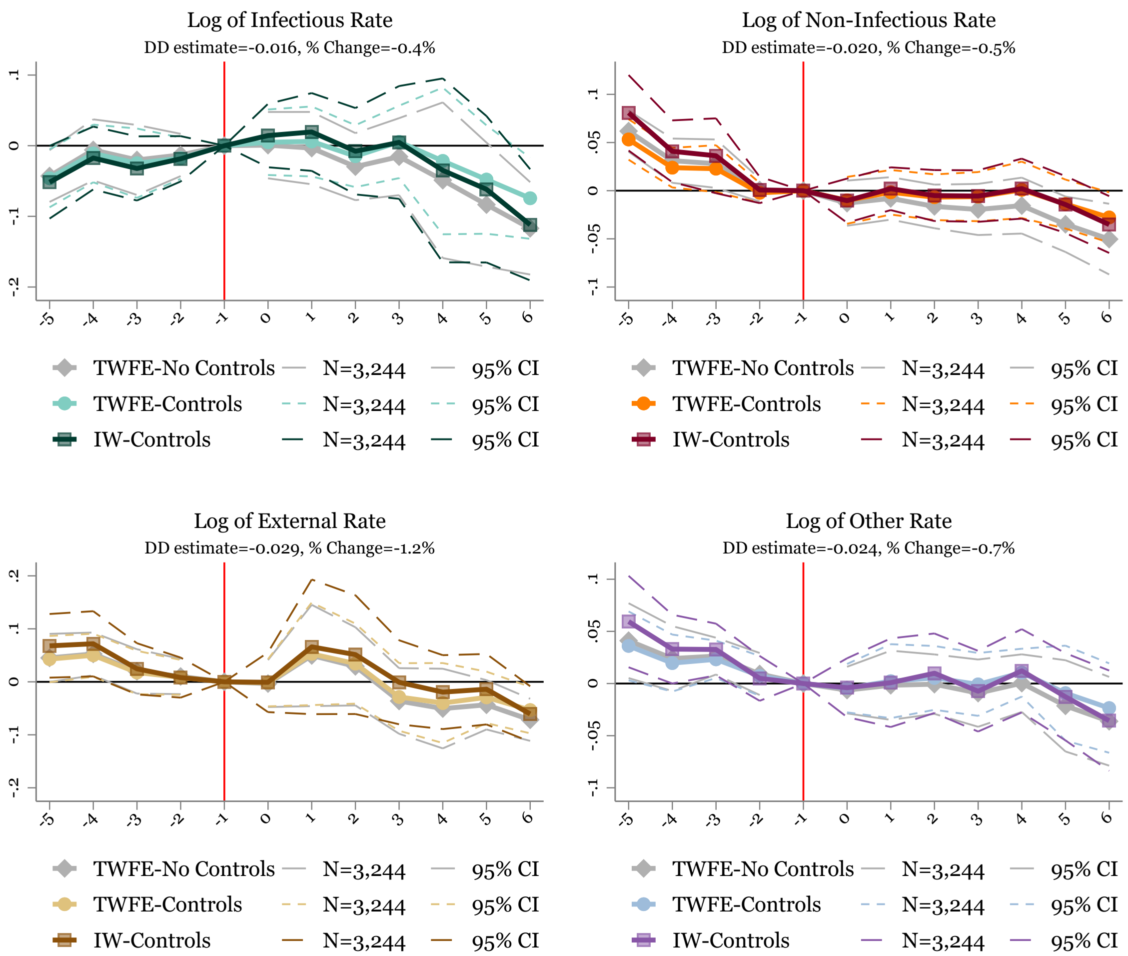
NOTES: Estimated coefficients from an OLS regression. Baseline fixed effects include year fixed effects and city fixed effects. Plotted coefficients are dummy variables for each year before and after the passage of the pasteurization ordinance. The period just before the ordinance is the excluded period (-1)–indicated by the vertical line. Controls include the share of the population that is white, the share of the population over 65, the share female, the share foreign, and the physicians per 10,000 persons. We use the log of mortality for all OLS specifications. For the majority of mortality measures, the mortality rate is the deaths per 10,000 persons. Regressions weighted by the denominator of the rate in each specification.

Figure A.8: Pasteurization–Other Causes of Death II, OLS



NOTES: Estimated coefficients from an OLS regression. Baseline fixed effects include year fixed effects and city fixed effects. Plotted coefficients are dummy variables for each year before and after the passage of the pasteurization ordinance. The period just before the ordinance is the excluded period (-1)–indicated by the vertical line. Controls include the share of the population that is white, the share of the population over 65, the share female, the share foreign, and the physicians per 10,000 persons. We use the log of mortality for all OLS specifications. For the majority of mortality measures, the mortality rate is the deaths per 10,000 persons. Regressions weighted by the denominator of the rate in each specification.

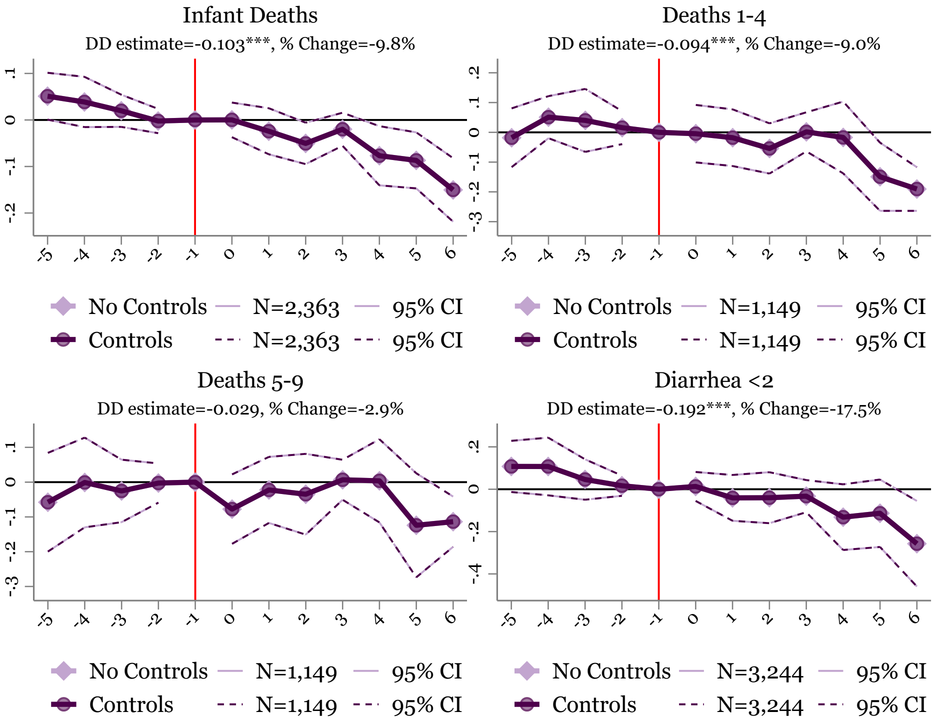
Figure A.9: Pasteurization–Additional Grouped Causes of Death III, OLS



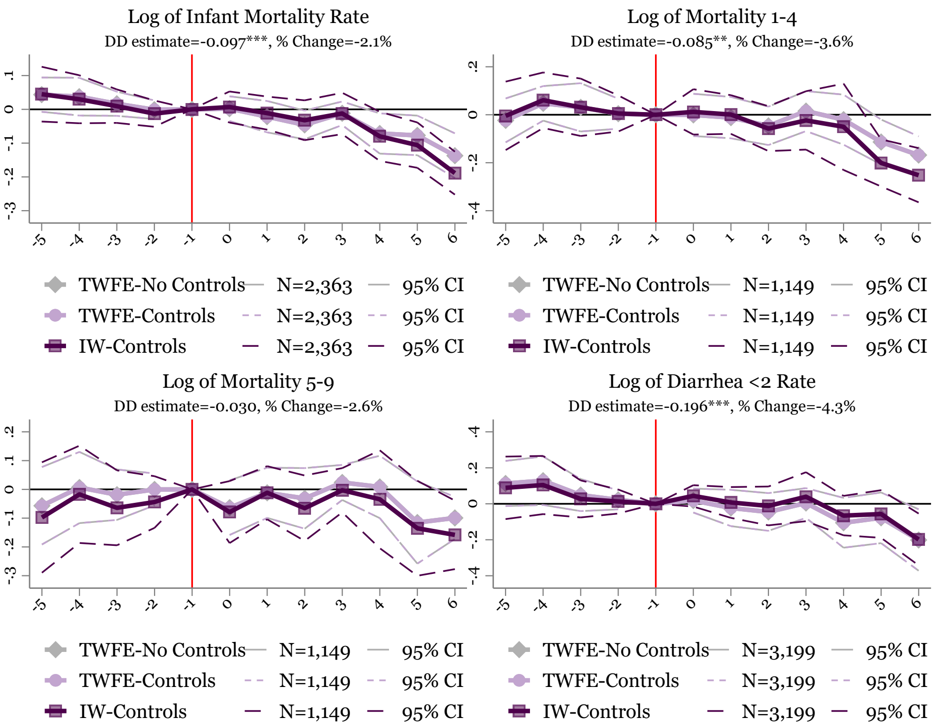
NOTES: Estimated coefficients from an OLS regression. Baseline fixed effects include year fixed effects and city fixed effects. Plotted coefficients are dummy variables for each year before and after the passage of the pasteurization ordinance. The period just before the ordinance is the excluded period (-1)–indicated by the vertical line. Controls include the share of the population that is white, the share of the population over 65, the share female, the share foreign, and the physicians per 10,000 persons. We use the log of mortality for all OLS specifications. For the majority of mortality measures, the mortality rate is the deaths per 10,000 persons. Regressions weighted by the denominator of the rate in each specification.

Figure A.10: Pasteurization–Infant Mortality and Child Mortality

Panel A: Poisson Specification



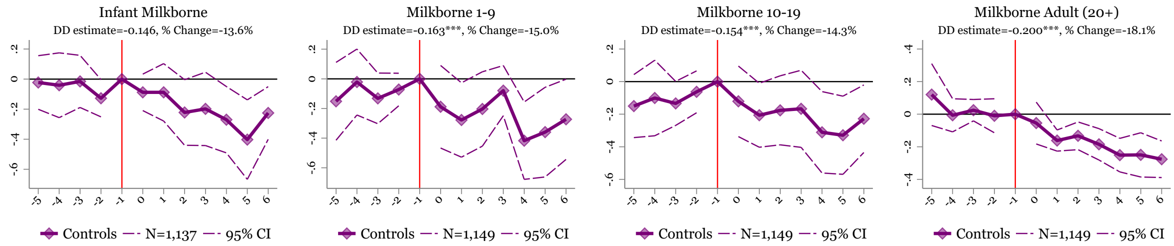
Panel B: OLS Specification



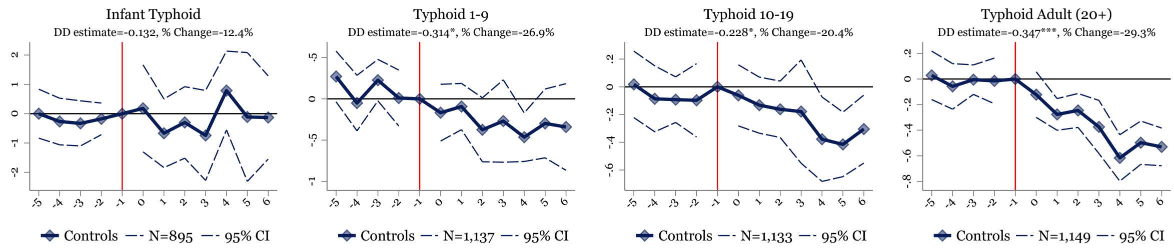
NOTES: In Panel A: Reflects Figure II except modifying the outcome and exposure in each graph. In Panel B: Estimated coefficients from an OLS regression. Baseline fixed effects include year fixed effects and city fixed effects. Plotted coefficients are dummy variables for each year before and after the passage of the pasteurization ordinance. The period just before the ordinance is the excluded period (-1)–indicated by the vertical line. Controls include the share of the population that is white, the share of the population over 65, the share female, the share foreign, and the physicians per 10,000 persons. We use the log of mortality for all OLS specifications. For the majority of mortality measures, the mortality rate is the deaths per 10,000 persons. Regressions weighted by the denominator of the rate in each specification.

Figure A.11: Additional Results–Pasteurization and Age-Specific Mortality

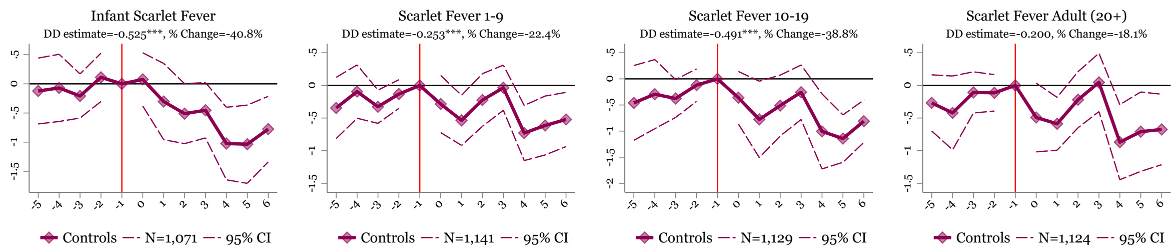
Panel A: Milkborne-Mortality



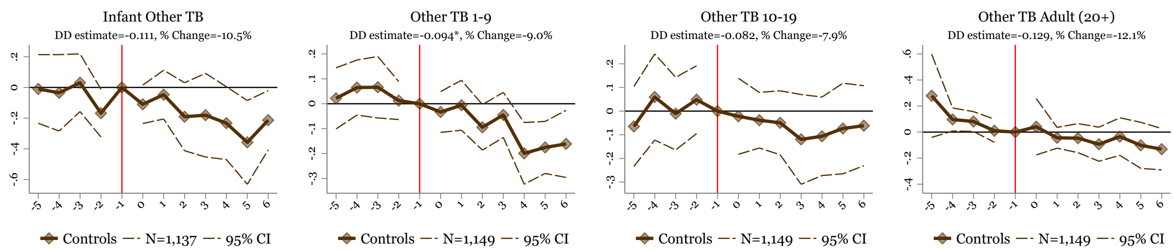
Panel B: Typhoid Mortality



Panel C: Scarlet Fever Mortality



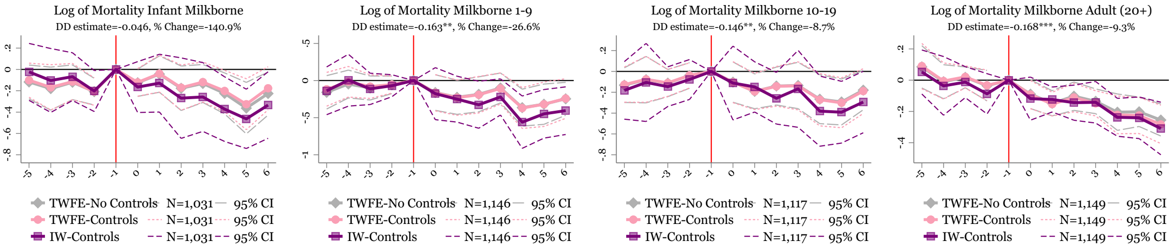
Panel D: Other Tuberculosis



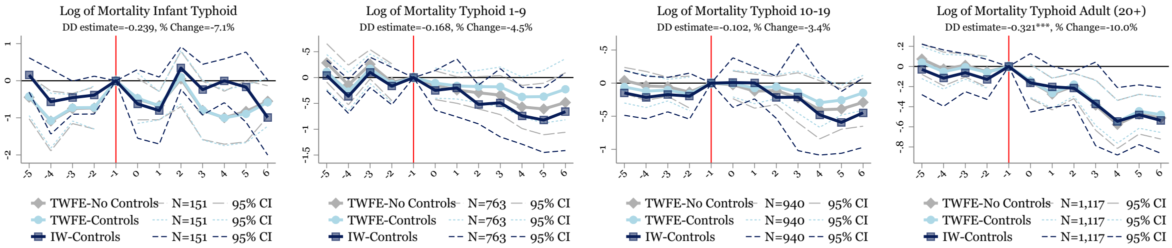
NOTES: Reflects Figure II except considering the age-specific mortality rates.

Figure A.12: Additional Results–Pasteurization and Age-Specific Mortality

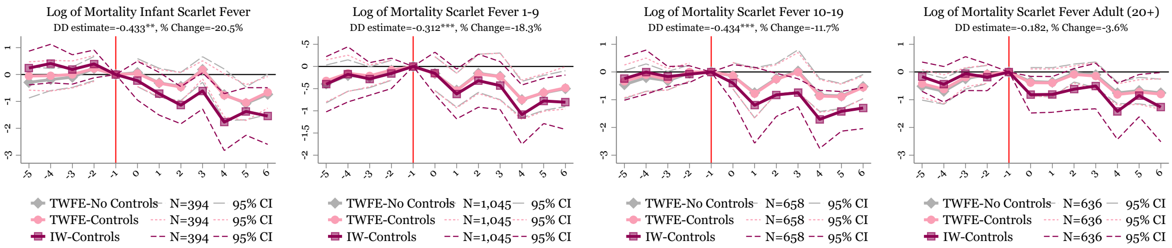
Panel A: Milkborne-Mortality



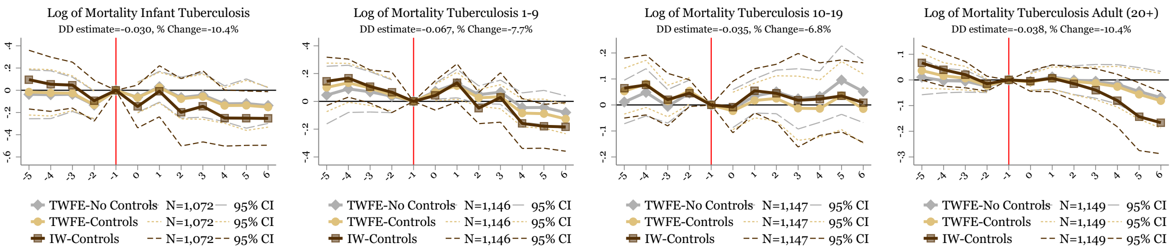
Panel B: Typhoid Mortality



Panel C: Scarlet Fever Mortality

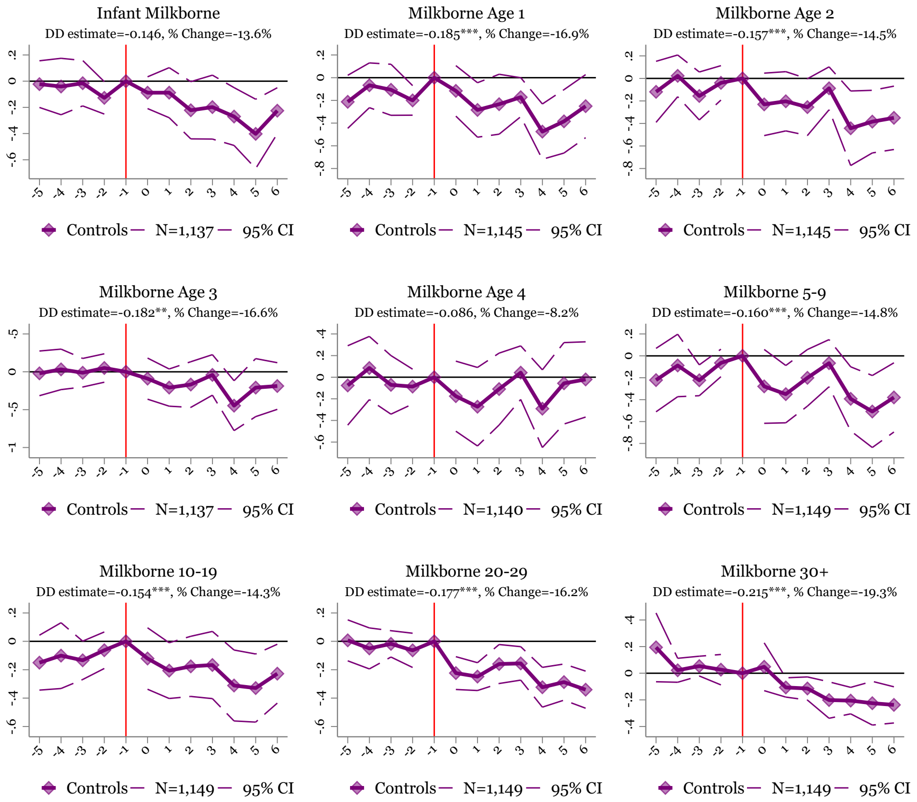


Panel D: Other Tuberculosis

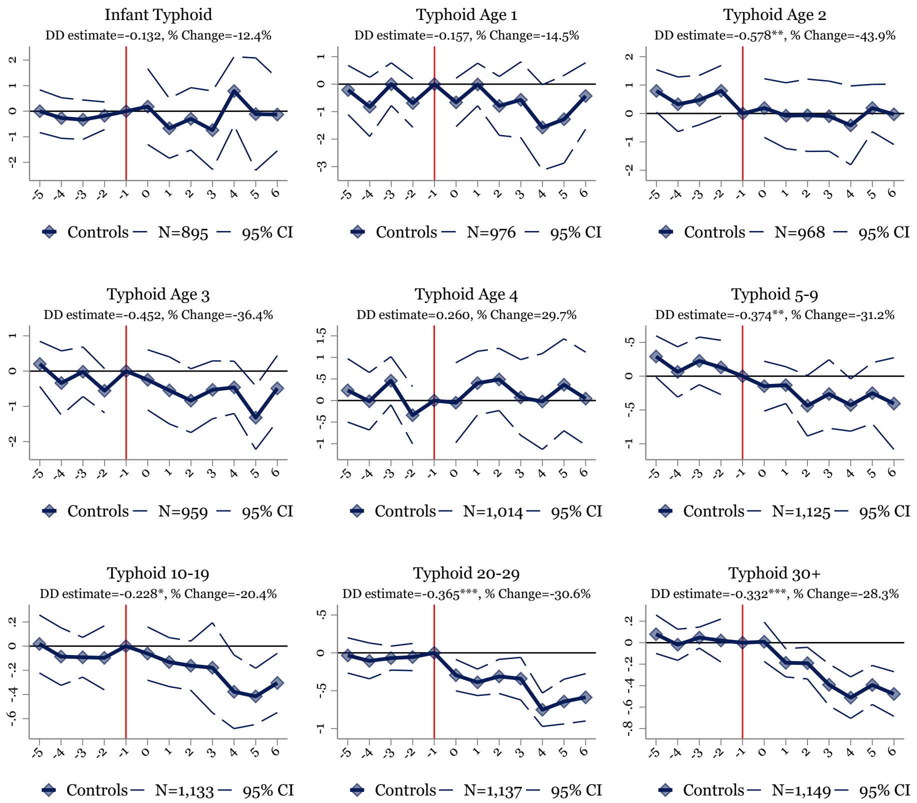


NOTES: Reflects Figure II except considering the age-specific mortality rates and using OLS and the log of the mortality rate as in Figure A.7.

Figure A.13: Main Results–Pasteurization and Additional Ages
Panel A: Milkborne-Mortality

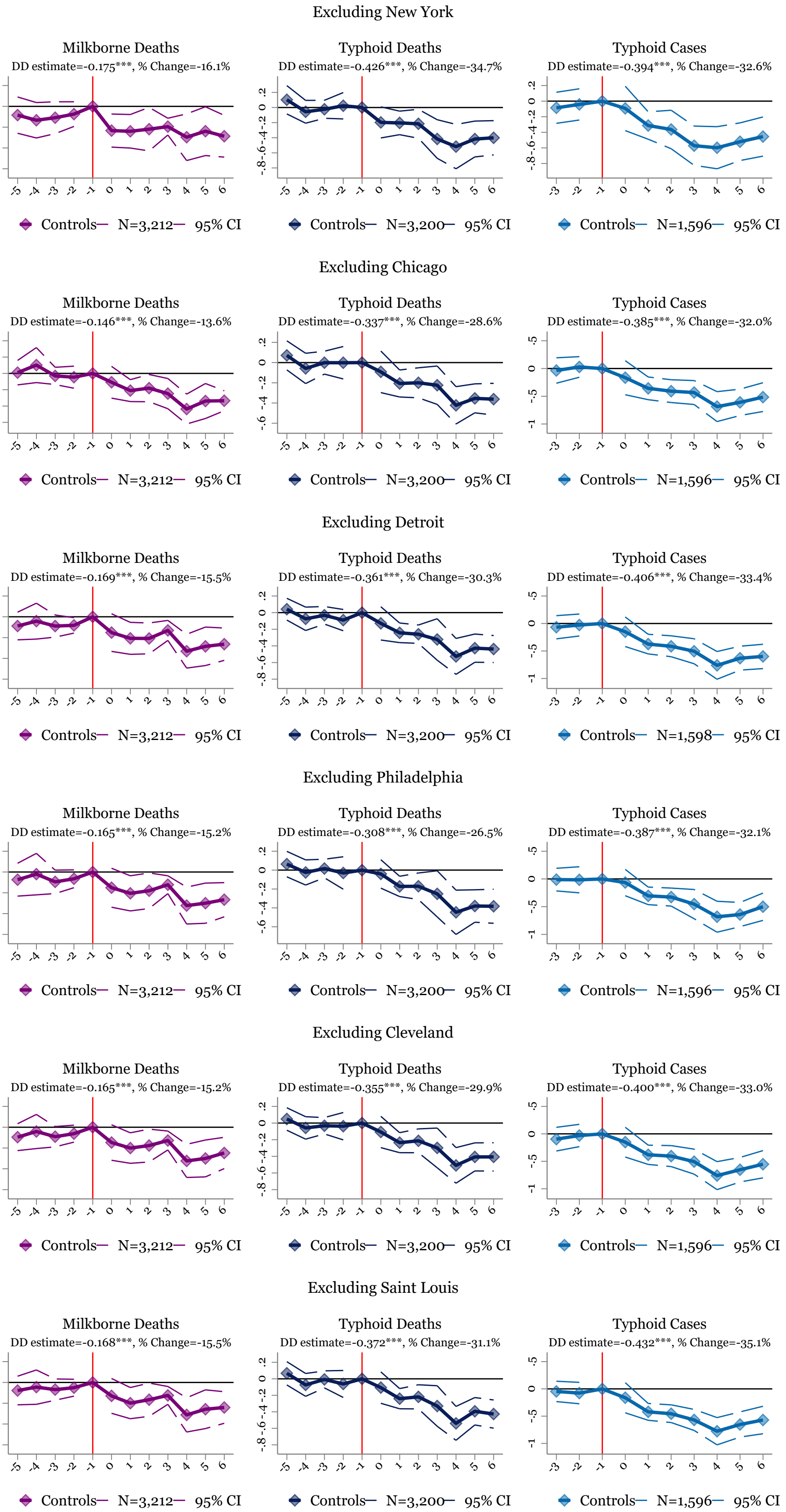


Panel B: Typhoid Mortality



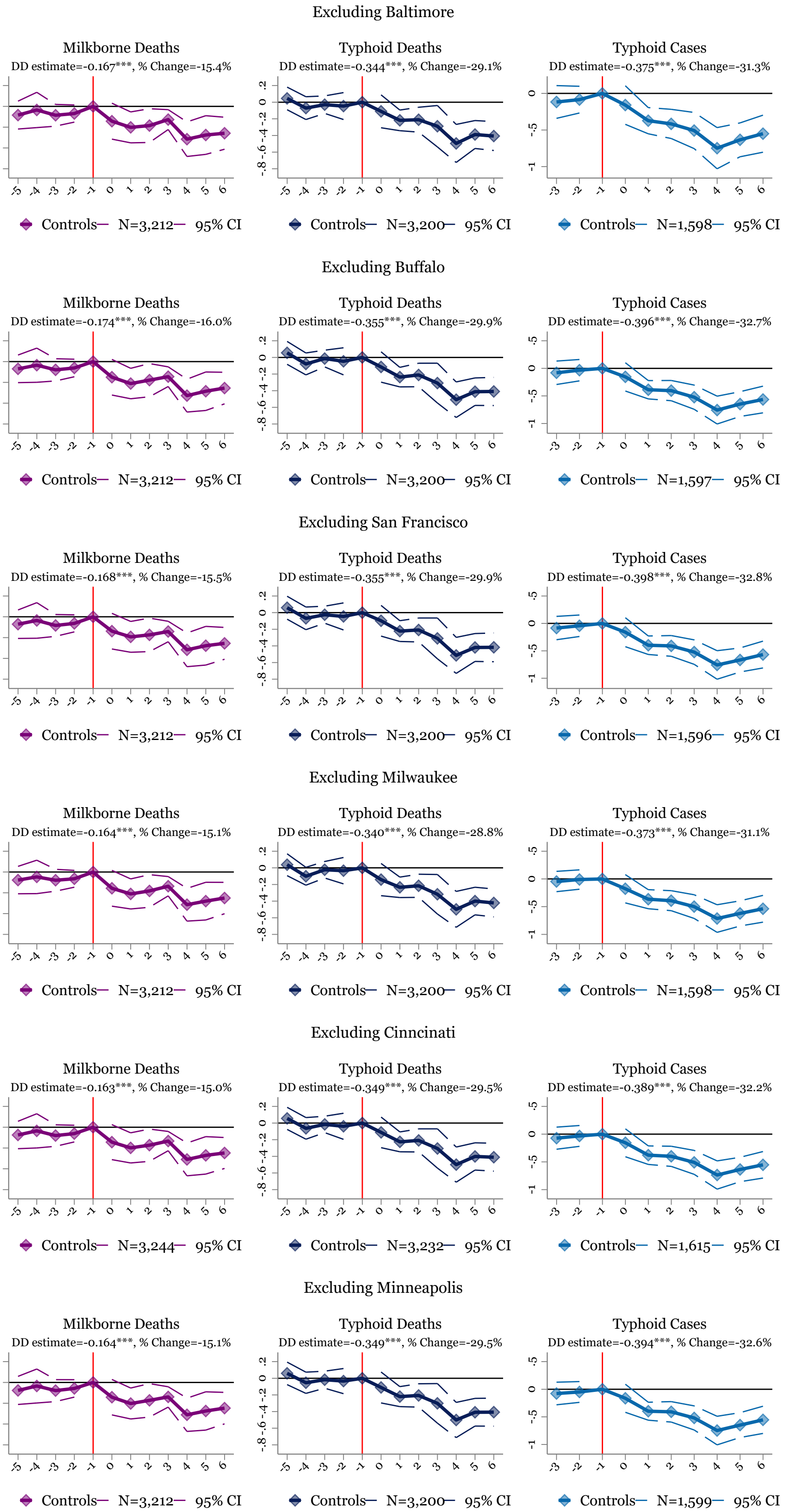
NOTES: Reflects Figure II except considering the age-specific mortality rates for additional ages.

Figure A.14: Dropping Large Cities (I)



NOTES: Reflects Figure II except dropping each large city noted in the title from the analysis.

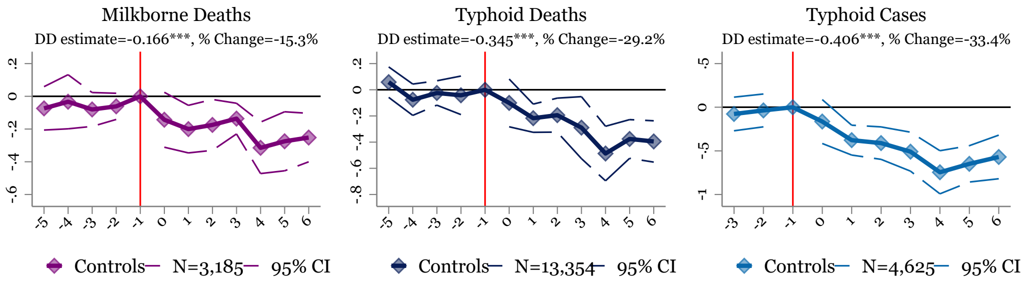
Figure A.15: Dropping Large Cities (II)



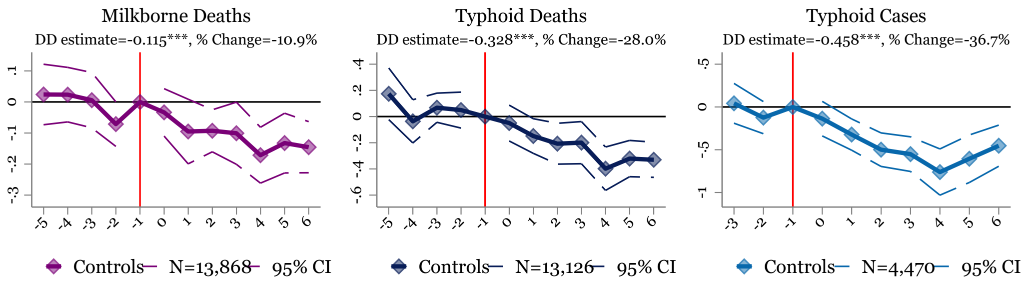
NOTES: Reflects Figure II except dropping each large city noted in the title from the analysis.

Figure A.16: Additional Robustness Checks (I)

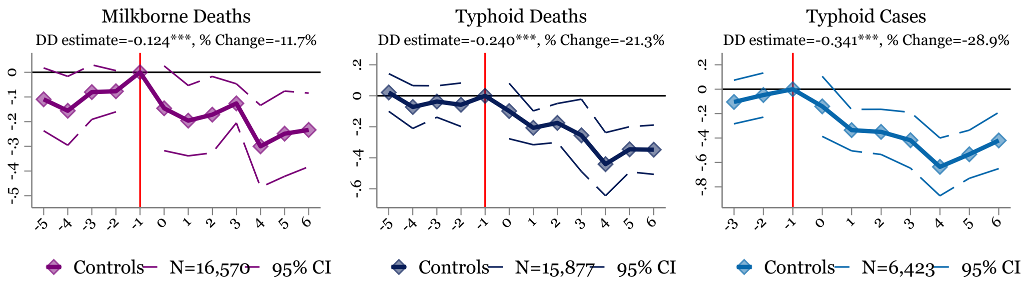
Panel A: Expanded Sample of Cities



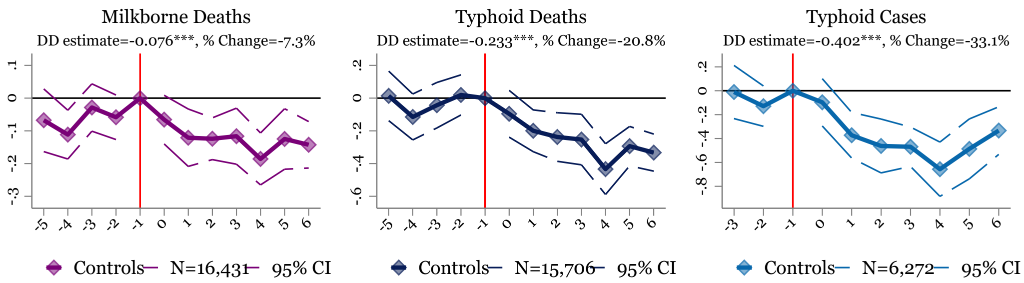
Panel B: Expanded Sample of Cities with State x Year FE



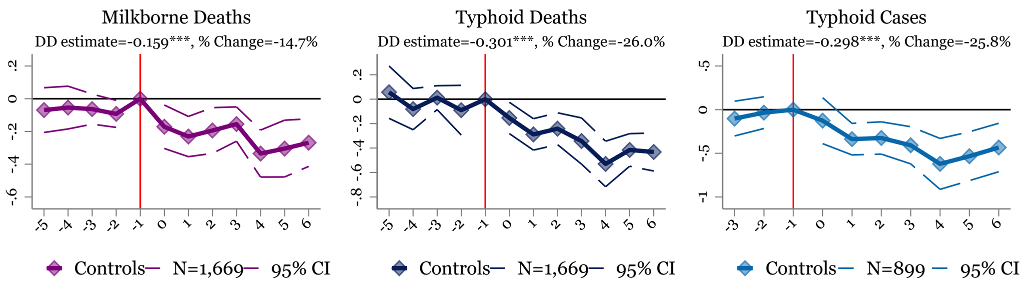
Panel C: Synthetic Controls Sample



Panel D: Synthetic Controls Sample, with State x Year FE



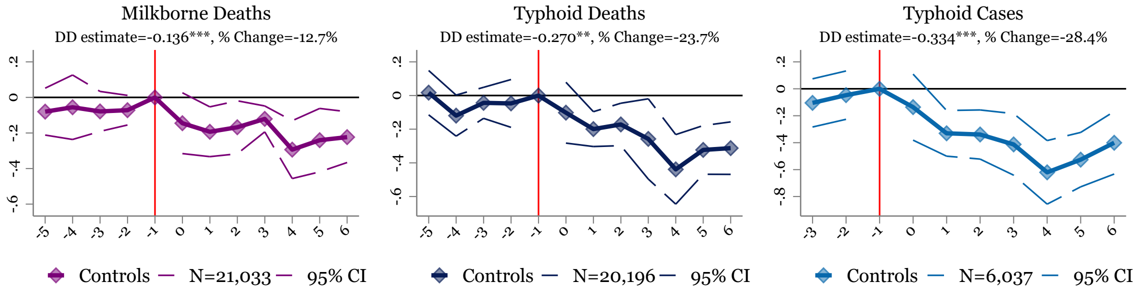
Panel E: Propensity Score Weights and Nearest Neighbor Matching



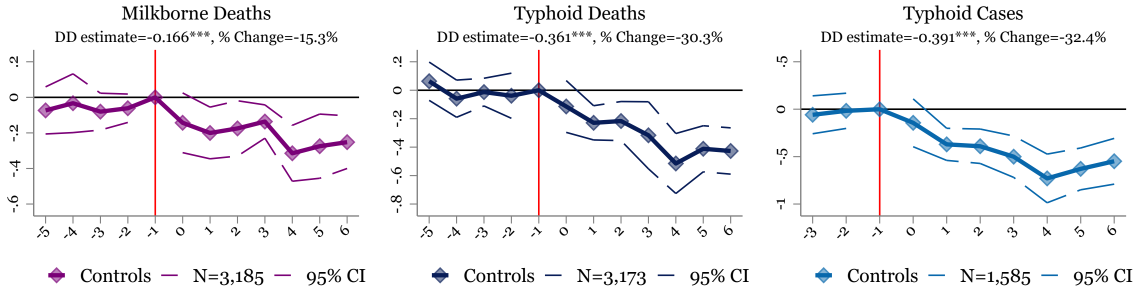
NOTES: Panel A broadens the control group to include all cities with pasteurization rates of 80 percent or below (adding in cities with less than 50k individuals). Panel B uses the same sample as in Panel A but adds state-by-year fixed effects. Panel C shows the synthetic control sample, cities without missing pasteurization dates based on Fuchs et al. (1939). Panel D shows the same sample as in Panel C, but with the addition of state-by-year FE. Panel E uses propensity score reweighting and nearest neighbor matching to select control cities.

Figure A.17: Additional Robustness Checks (II)

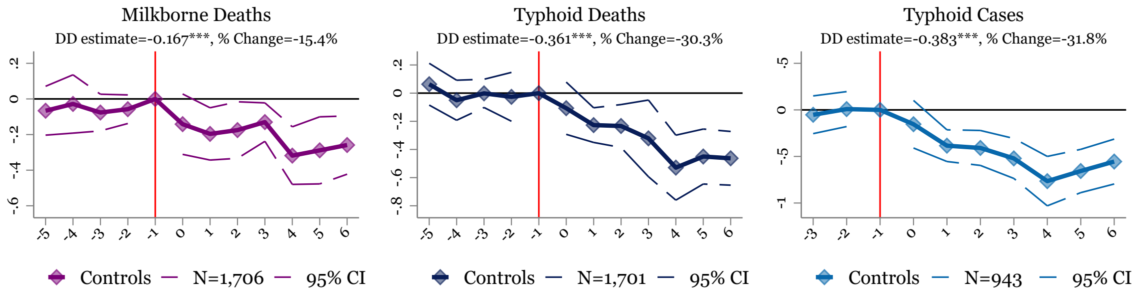
Panel A: 1900-1940 and IPUMs Population



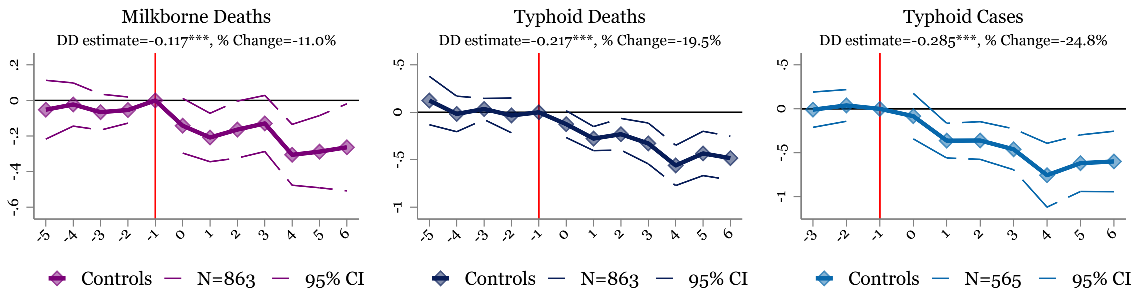
Panel B: Balanced Event Time (Deaths)



Panel C: Cities 100,000+

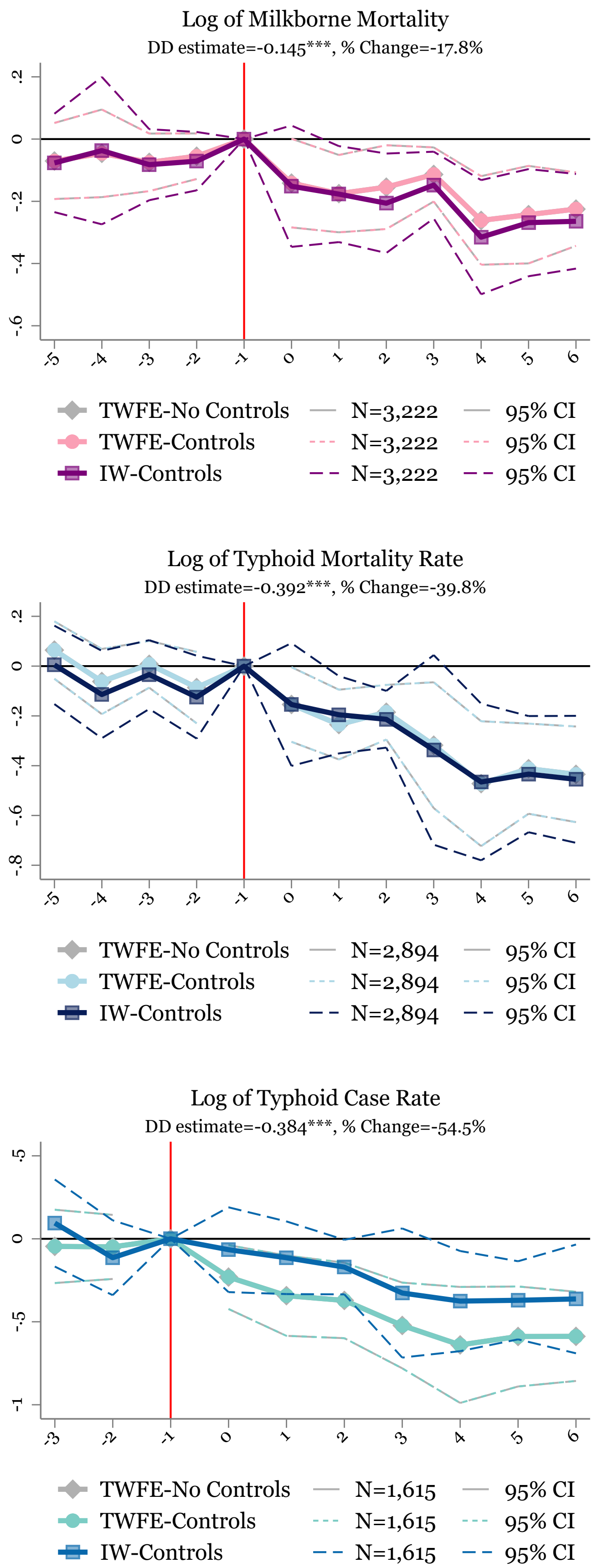


Panel D: 1905-1930, Year Pasteurization up to 1926



NOTES: Panel A extends the analysis to 1900 and 1940 and includes the IPUMS population as the exposure rather than the published census counts. Panel B shows the main sample with balanced event time for typhoid mortality (no cities entering or exiting early). Panel C subsets to only cities with 100,000 or more persons. Panel D subsets to only 1905-1930 and only cities that passed pasteurization up to 1926.

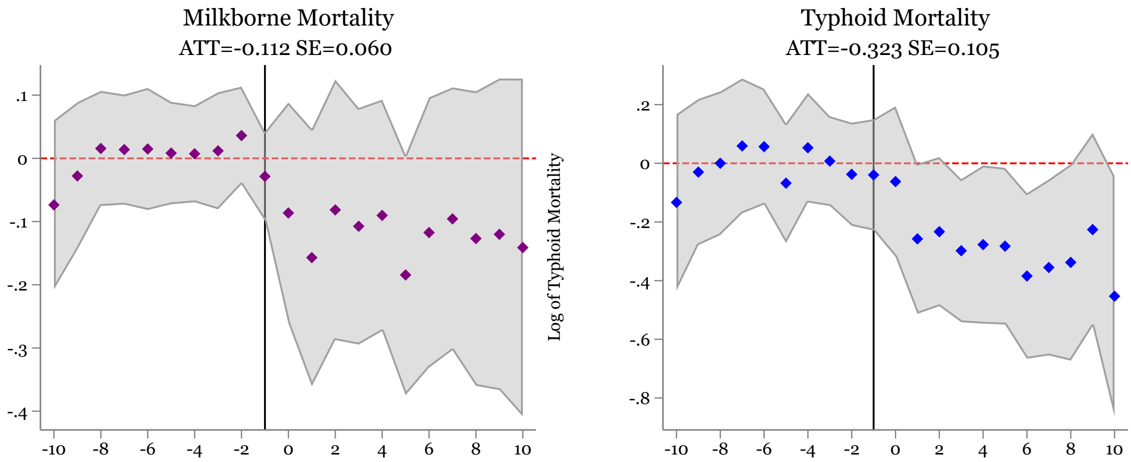
Figure A.18: Robustness Checks–OLS and TWFE-alternative



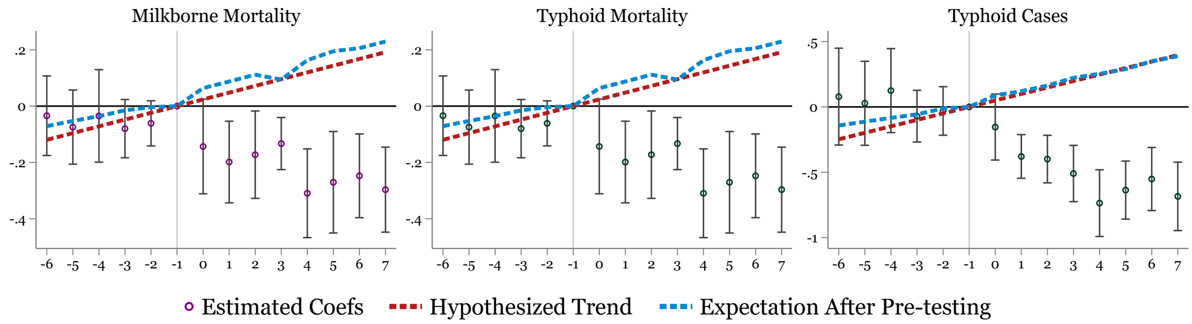
NOTES: Reflects Figure II except presents the estimates from a log-linear OLS specification, where the outcome is the log of the typhoid mortality rate and case rate, and the regressions are weighted by the city-level population.

Figure A.19: Pre-trends test and Synthetic Difference-in-Difference Estimator

Panel A: Synthetic Difference-in-Difference Estimator



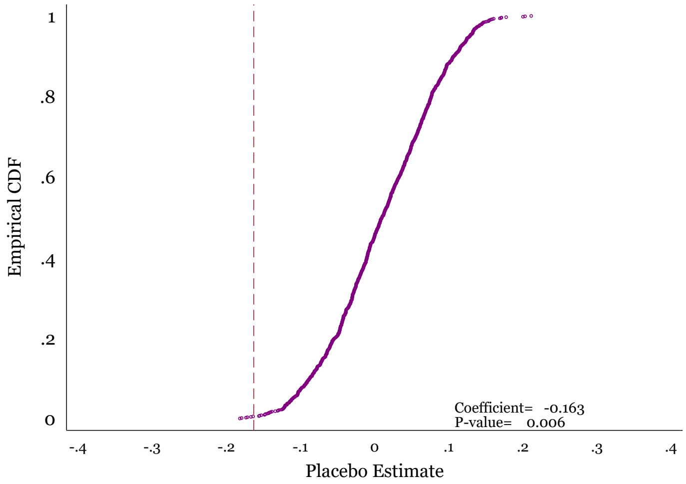
Panel B: Pasteurization Ordinance with Overlaid Pretrends



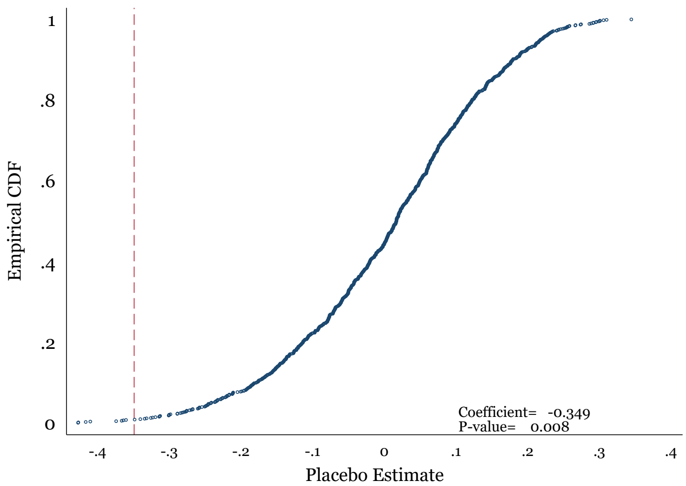
NOTES: Panel A reflects Figure II except considering the OLS specification with the log of the typhoid fever mortality rate, and considering the synthetic difference-in-differences from Arkhangelsky et al. (2021); Ciccia et al. (2024). We include only the years 1909-1929. We need a balanced panel for the regression, and more cities are missing typhoid before 1908. Cities also entered in 1930. We use the log of typhoid mortality as the outcome, which also results in the loss of some observations due to zero values. Cities are compared to the full group of cities (including less than 50k cities) due to requirements on the number of controls. The analysis was performed with 100 bootstrap replications. We only show the main event window, m=-10 to m=10. Panel B reflects Figure II with controls, except considering the test for pretrends from Roth (2022); Caceres-Bravom (2024).

Figure A.20: Permutations on the Year of the Pasteurization Ordinance

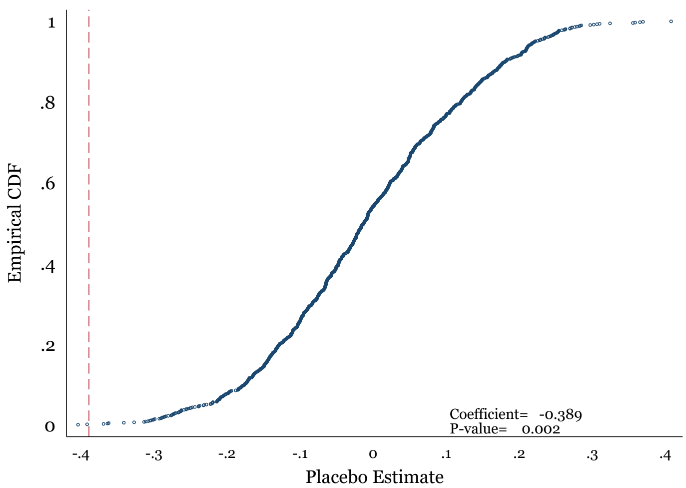
Panel A: Milkborne Mortality



Panel B: Typhoid Mortality



Panel C: Typhoid Case Rate



NOTES: Represents city-level specification from Figure II, except randomizing the treated cities across the pasteurization years. We randomly assign cities and consider a grouped post-period estimation of Equation 1. Each plotted point represents a separate regression. The randomization is performed 1,000 times. The plotted CDF represents the distribution of estimates from these placebo simulations, with the estimated coefficient for our 'actual' difference-in-differences estimate indicated by the vertical line. The non-parametric p-value is calculated as the number of placebo observations that are greater than the estimated effect, divided by the sample size of all permutation estimates.

B Non-Pasteurization Milk Quality Control Regulations

During the early twentieth century, cities employed several strategies in their quest to ensure the cleanliness of the milk supply. As early as 1900, regulations on milk sales were in place in 44 states, with 25 states employing at least one public health official dedicated to the enforcement of these laws (Selitzer, 1976). The measures employed by cities during this time included pasteurization, minimum quality standards, tuberculin testing of cow herds, milk inspections, the setting of bacteriological standards, and the grading of milk.

B.1 Minimum Quality Standards

Minimum quality standards (MQSs) were aimed at curbing the deliberate adulteration of milk with foreign substances such as water, salt, sugar, and boric acid in order to ensure that babies received adequate nutrition from milk and that unsafe substances were not being introduced to the milk supply (Meckel, 1990). Many cities adopted these laws in the late 1800's, and they usually set a minimum threshold for solids that could be enforced by use of a lactometer (Meckel, 1990). Anderson et al. (2025) finds evidence that these standards reduced mortality from waterborne and food-borne diseases by 12 percent after five years and 19 percent after ten. However, MQSs were not a foolproof method for detecting adulteration, nor did it do anything to address unintentional contamination by pathogens.

B.2 Tuberculin Testing

In the year 1900, tuberculosis stood as a primary cause of death in the United States (Crimmins and Condran, 1983). Both the human and bovine forms of tuberculosis could infect humans and could be transmitted through dairy products. While it was accepted that milk could become contaminated through handling by an infected individual, around this time, there was a growing awareness that bovine tuberculosis could pass from cows to humans via meat or milk from infected animals (Currier and Widness, 2018).

As a result of this new understanding that diseased cows could spread bovine tuberculosis to humans, along with the discovery of a new method for tuberculosis testing of cows, municipalities began implementing the tuberculin testing of cow herds (Palmer et al., 2011). Tuberculin testing of herds involved injecting cows with a substance called "tuberculin" and observing them for up to 24 hours for signs of hyperthermia (Palmer et al., 2011).[19] Those displaying an elevated temperature were found to test positive for bovine tuberculosis and were removed from the herd. In cases of widespread infection, entire herds would be destroyed (Palmer et al., 2011). Mandatory tuberculin testing of herds at the city and state level grew to be widespread. In one survey of cities for the year 1935, 84 percent of cities reported practicing tuberculin testing of herds (Fuchs et al., 1939). At the state level, tuberculin testing was also more common than pasteurization. As of 1935, no states required full pasteurization of the milk supply, but 23 states required tuberculin testing of herds (Fuchs et al., 1939).[20]

However, tuberculin testing of herds was relatively costly for municipalities to implement

[19] This method was discovered by Robert Koch, who was searching for a cure for tuberculosis, and adapted by others for the purpose of identifying diseased cows.

[20] The popularity of mandatory tuberculin testing arose at least partially from concerns that places without this requirement would become "dumping grounds" for tubercular cows from states with stricter requirements (Czaplicki, 2007; Palmer et al., 2011).

as compared to pasteurization. Cities and states varied in their capacity to carry out this public health measure. Many early tuberculin-testing ordinances on the books were mere "paper measures" (Czaplicki, 2007) because the cities and small towns did not have the capacity to enforce the laws. On the other hand, larger cities had more state capacity to enforce such laws. For example, when Chicago passed a mandate requiring tuberculin testing of dairy herds, the city also subsequently increased the number of sanitary inspectors from 12 to 67 (Czaplicki, 2007).

Even when cities had the capacity to carry out testing, tuberculin testing fell short of ensuring a safe milk supply from a public health standpoint. Critically, milk from tuberculin-tested herds was still susceptible to contamination with non-tuberculosis illnesses (Straus and Straus, 1913). Further, tuberculosis itself was still known to contaminate milk from herds where there was diligent removal of cattle with tuberculosis. Straus and Straus (1913) argues that the tubercle bacilli was still found in 6.7 percent of milk from tested herds. While the tuberculin testing of herds was a significant public health achievement in that it would eventually go on to succeed in largely eradicating tuberculosis in cow herds (Olmstead and Rhode, 2004; Palmer et al., 2011), it fell short of producing the holistically safe milk that cities sought.

B.3 Milk Inspections

During this period, cities also conducted milk inspections that have been tied to mortality declines (Anderson et al., 2025; Komisarow, 2017). While the scope and frequency of milk inspections varied across place, Chicago provides an illustrative example. In 1915, Chicago conducted milk inspections on dairy farms, creameries, and pasteurization plants located in the country, as well as on milk platforms, milk depots & stores, and pasteurization plants located within the city (Perry, 1915). In many large cities, a feature of milk inspections included conducting regular bacteriological examinations and chemical analyses on milk and cream samples at depots to ensure that the dairy products complied with any existing standards (Perry, 1915).

However, similar to other pasteurization alternatives, milk inspections proved costly in terms of time and labor, and many localities struggled to conduct them with adequate frequency and thoroughness to ensure the cleanliness of the milk supply. For example, in 1915, Chicago employed only fifteen country milk inspectors, each of which was tasked with inspecting an average of 933 farms each year, as well as any creameries and pasteurization plants within their dairy district, resulting in each dairy farm being inspected only twice per year on average (Perry, 1915). More broadly, although 91 percent of cities with local milk control measures had at least a part-time inspector in 1935, only 12 percent had an inspector in charge of full-time milk control work (Fuchs et al., 1939). Most states had at least one part-time inspector as of 1935 (only seven did not), and roughly 29 states had full-time inspectors (Fuchs et al., 1939). In addition to their infrequency, milk inspections struggled to address issues arising during milk's transportation, such as milk exceeding a safe temperature during rail transport, and during final distribution to consumers (Perry, 1915).

B.4 Bacteriological Standards & Grading

Cities and states also adopted bacteriological standards for raw and pasteurized milk (Fuchs et al., 1939). The bacteriological standards that were set by cities encoded into law a maximum allowable bacteriological count per cubic centimeter. This bacteriological standard was used to establish either as an overall minimum standard or to assign milk a grade based upon its quality. Over half of cities surveyed reporting grading milk in 1935, and this practice was especially common among the largest cities with a population of over 500,000 (Fuchs et al., 1939).

While previous work in Anderson et al. (2022) has suggested that bacteriological standards were essentially pasteurized milk ordinances, bacterial ordinances were distinct. This is made clear by the fact that separate bacterial standards were adopted for pasteurized and raw milk, and that even when bacteriological standards were adopted, the pasteurization rate of milk remained low in many cities (Fuchs et al., 1939). In Boston, both tuberculin testing of cows and bacteriological standards for milk went into effect in 1905. However, Boston's rate of pasteurization five years later, in 1910, was still below 50 percent. In fact, Boston's pasteurization rate did not approach 90 percent until 1921 (see Table 1). Baltimore presents another similar case to Boston, where milk pasteurization rates remained around 50 percent in 1916 even though bacteriological standards for milk were passed in 1913 (Anderson et al., 2022). Thus, in many cases, our dates of pasteurization are distinct from Anderson et al. (2022)'s dates for bacteriological standards.

Cities varied as to whether bacteriological standards were adopted before, concurrently with, or after pasteurization ordinances. Table A.2 demonstrates that, among cities that adopted both bacteriological standards and pasteurization during the period of study, bacteriological standards were most commonly adopted before pasteurization ordinances. However, several cities adopted them in the same year as pasteurization ordinances, while several other cities, such as Cincinnati, Philadelphia, and Saint Louis, adopted bacteriological standards after pasteurization. Finally, it is also apparent that some cities adopted only one or neither of these milk control measures.

Bacteriological standards did not provide a new means of directly improving the quality of milk; rather, they were set aspirationally as an incentive for milk producers to improve milk quality in a measurable way. A study of bacterial counts in eight cities found that log average counts were higher than the bacterial standard in seven out of the eight cities (Dahlberg et al., 1953).[21] Milk exceeding municipal bacteriological standards was also found to be common in Kansas (Kansas State Board of Health, 1922).

As another example, Chicago's 1912 ordinance mandated a winter bacteriological standard of 100,000 per cubic centimeter for raw milk (150,000 in the summer) and 50,000 for pasteurized milk (100,000 in the summer) (Perry, 1915). However, the health department reported an average bacterial count of 1,000,000 per c.c. in raw milk (6.7-10x the standard) and 100,000 per c.c. in market pasteurized milk (1-2x the standard) for the year 1914 (Perry, 1915). Additional shortcomings to this method of milk control were the significant resources required to conduct regular bacterial counts, the inability to produce bacterial counts quickly enough to prevent the sale of milk with high counts, inconsistent counts between different laboratories, and the understanding that the measure could not provide information on the type of bacteria present (Parker, 1917).

[21] These cities were Birmingham, Boston, Houston, Louisville, Minneapolis, Rochester, Sacramento, and Washington.

C Difference-in-differences Specification

For completeness, we also show difference-in-differences results in Table C.1. An advantage of showing these difference-in-differences point estimates is that we can display competing public health interventions. This allows us to compare coefficients and see which efforts had the largest impact on city-level mortality.

Formally, our difference-in-differences specification appears similar to the event study for city j, in state s and year t as:

$$\text{Mortality}_{jst} = \exp(\alpha + \beta \, \text{Pasteurization Ordinance}_{jst} + \mathbf{X}'_{jst}\gamma + a_j + \eta_t)\, \epsilon_{jst}, \tag{2}$$

where Mortality_{jst} is the mortality rate in city j, state s, and year $t = 1905, ..., 1936$. We model the rate using the death count as the outcome and the population as the exposure. The indicator $\text{Pasteurization Ordinance}_{jst}$ equals one the year after city j adopts a pasteurization ordinance, and is zero otherwise. The coefficient β represents the average post-ordinance proportional change in mortality associated with pasteurization.

As in Equation (1), $\mathbf{X}_{jst}$ denotes time-varying city covariates, a_j are city fixed effects, and η_t are year fixed effects, with ϵ_{jst} the error term, clustered at the city level. In the difference-in-differences we also add city-specific trends only where indicated, $\theta_j t$.

C.1 Results

Table C.1 Panel A repeats our baseline estimation of a Poisson model with a grouped post-period. Column (1) shows the results without controls, Column (2) adds controls, and Column (3) adds linear trends. The results suggest mortality declines across all specifications in Panel A. Then, in Panel B, we expand the sample and add the state-by-year fixed effects, where the mortality declines are similar to Panel A.

Next, in Panel C, we add controls for purification and disinfection of the water supply (USPHS, 1926). Pasteurization and water purification produce typhoid mortality declines, with the effects similar in magnitude but varying slightly across specifications. Only pasteurization consistently impacts the typhoid case rate and milkborne mortality. In Panel D, we also present similar results but expand the sample and add state-by-year fixed effects to remove state-level policies. Here the results show a decline for all measures of mortality and typhoid case rates.

Then, we add controls for the Anderson et al. (2022) public health interventions in Panel E. Here, the sample shrinks again, but pasteurization still reduces milkborne mortality, typhoid morbidity, and mortality. Whether pasteurization or filtration has a bigger impact on typhoid mortality depends on the specification of focus. As with Panels C and D, water filtration (analogous to purification) produces a competing decline in mortality with pasteurization. However, throughout Table C.1, water infrastructure investments do not affect the case rate, an outcome that was not examined in past studies.

Table C.1: Pasteurization Ordinance and Typhoid Fever, Poisson Model

	Milkborne Deaths			Typhoid Deaths			Typhoid Cases		
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
Panel A: All Pasteurization									
1(Pasteurization Ordinance)	-0.1733***	-0.1629***	-0.1305***	-0.4162***	-0.3494***	-0.2076***	-0.4400***	-0.3892***	-0.2557***
	(0.0338)	(0.0332)	(0.0420)	(0.1153)	(0.0993)	(0.0501)	(0.0783)	(0.0751)	(0.0766)
N	3,244	3,244	3,244	3,232	3,232	3,232	1,615	1,615	1,615
Pseudo R-squared	0.948	0.948	0.952	0.855	0.859	0.875	0.896	0.897	0.914
Panel B: Expanded Sample, State x Year FE									
1(Pasteurization Ordinance)	-0.1078***	-0.1151***	-0.1265***	-0.3783***	-0.3280***	-0.2424***	-0.4515***	-0.4576***	-0.2893***
	(0.0297)	(0.0349)	(0.0449)	(0.0625)	(0.0591)	(0.0597)	(0.0668)	(0.0644)	(0.0635)
N	13,868	13,868	13,868	13,126	13,126	13,126	4,470	4,470	4,470
Pseudo R-squared	0.932	0.932	0.935	0.840	0.841	0.849	0.898	0.898	0.916
Panel C: Purification and Disinfection Controls									
1(Pasteurization)	-0.1954***	-0.1852***	-0.1349***	-0.4116***	-0.3778***	-0.2300***	-0.4390***	-0.3760***	-0.2603***
	(0.0365)	(0.0362)	(0.0413)	(0.1205)	(0.0968)	(0.0511)	(0.0814)	(0.0763)	(0.0779)
1(Water Purification)	-0.0576	-0.0865	-0.1296**	-0.2747**	-0.3229***	-0.3004***	-0.1727	-0.1349	-0.1262*
	(0.0663)	(0.0571)	(0.0533)	(0.1292)	(0.1072)	(0.0925)	(0.1077)	(0.1003)	(0.0665)
1(Water Disinfection)	0.0387	0.0337	0.0259	0.0348	0.0283	0.0203	-0.0770	-0.1200	0.0097
	(0.0363)	(0.0378)	(0.0371)	(0.0687)	(0.0687)	(0.0484)	(0.0887)	(0.0917)	(0.1167)
N	1,799	1,799	1,799	1,799	1,799	1,799	974	974	974
Pseudo R-squared	0.949	0.950	0.954	0.863	0.868	0.884	0.897	0.898	0.915
Panel D: Expanded Sample, State x Year FE, Purification and Disinfection Controls									
1(Pasteurization Ordinance)	-0.1145**	-0.1302**	-0.1276**	-0.1865**	-0.2348***	-0.2524***	-0.2753***	-0.3015***	-0.1681**
	(0.0448)	(0.0552)	(0.0562)	(0.0787)	(0.0658)	(0.0747)	(0.0710)	(0.0707)	(0.0663)
1(Water Purification)	-0.1075*	-0.1190**	-0.1468**	-0.2752**	-0.2549*	-0.2415*	0.0303	-0.0669	-0.2117
	(0.0555)	(0.0556)	(0.0627)	(0.1308)	(0.1322)	(0.1430)	(0.0918)	(0.1085)	(0.2155)
1(Water Disinfection)	0.0004	-0.0073	-0.0272	-0.1400**	-0.1356**	-0.1271**	-0.0124	0.0334	0.0948
	(0.0478)	(0.0487)	(0.0378)	(0.0577)	(0.0585)	(0.0544)	(0.1256)	(0.1294)	(0.1548)
N	1,457	1,457	1,457	1,455	1,455	1,455	759	759	759
Pseudo R-squared	0.965	0.965	0.967	0.905	0.906	0.909	0.941	0.942	0.947
Panel E: Anderson et al Public Health Controls									
1(Pasteurization)	-0.2187***	-0.1993***	-0.1350***	-0.5310***	-0.4160***	-0.2260***	-0.4534***	-0.4059***	-0.1903**
	(0.0537)	(0.0491)	(0.0434)	(0.1929)	(0.1211)	(0.0761)	(0.1042)	(0.0812)	(0.0802)
1(Water Filtration)	-0.0099	-0.0391	-0.1597***	-0.2647*	-0.2552**	-0.3843***	-0.0287	0.0407	-0.0066
	(0.0684)	(0.0507)	(0.0606)	(0.1480)	(0.1232)	(0.0608)	(0.1975)	(0.1867)	(0.1429)
1(Water Chlorine)	0.0650	0.0778*	0.0036	0.0085	-0.0088	-0.0833	0.1715	0.1084	0.0592
	(0.0473)	(0.0451)	(0.0464)	(0.0968)	(0.0617)	(0.0675)	(0.1307)	(0.1193)	(0.1441)
1(Bacteriological Standard for Milk)	0.1026**	0.0945**	0.1051**	0.1170	0.1238	0.1258	0.1078	0.1116	-0.0538
	(0.0450)	(0.0416)	(0.0441)	(0.1080)	(0.1029)	(0.0848)	(0.1071)	(0.0948)	(0.0946)
1(TB Testing of Cows)	-0.0234	-0.0021	-0.0216	0.1050	0.0310	-0.0787	0.0492	-0.0134	-0.0197
	(0.0665)	(0.0572)	(0.0355)	(0.1554)	(0.1313)	(0.0822)	(0.0836)	(0.0922)	(0.0975)
1(Sewage Treatment/Diversion)	0.0702	0.0501	-0.0976**	0.0401	0.0891	0.0678	0.0170	0.0629	0.2460
	(0.0799)	(0.0744)	(0.0442)	(0.1489)	(0.1182)	(0.0705)	(0.1563)	(0.1515)	(0.2426)
N	671	671	671	671	671	671	379	379	379
Pseudo R-squared	0.951	0.952	0.959	0.882	0.887	0.909	0.909	0.911	0.928
City and Year FE	X	X	X	X	X	X	X	X	X
Controls		X	X		X	X		X	X
Ciy Linear Trends			X			X			X

NOTES: Estimated coefficients from a city-level Poisson model. City and year fixed effects are included. The outcome is the (linear) deaths and case counts, and the exposure is the city-level population. Robust standard errors clustered at the city level. ***, **, * represent statistical significance at 1, 5, and 10 percent levels. Controls include the share of the population that is white, the share of the population over 65, the share female, the share foreign, and the physicians per 10,000 persons.
SOURCES: See Figure II. Water purification from Anderson et al. (2022) and USPHS (1926).

D Suggestive Trends: Comparing Each City's Typhoid Mortality Rate Against a Synthetic Control Group

D.1 Methods

We complement our difference-in-differences analysis using synthetic control methods (SCM) and descriptive mortality trends. This approach illustrates the timing and magnitude of declines in typhoid mortality following the pasteurization ordinances. For the SCM, we focus on the log of typhoid mortality as our primary outcome because water filtration has been shown to affect typhoid, and the SCM helps visually distinguish whether the mortality decline was due to pasteurization or waterborne effects.

Because the log transformation is undefined at zero, and the SCM analysis requires a balanced panel, we restrict the SCM analysis to cities with non-zero typhoid mortality from 1909 to 1926 (mostly large cities).[22] For each individual SCM analysis, control cities only include cities without a recorded pasteurization date based on Table 1 or Fuchs et al. (1939).

D.2 Analysis of Pasteurization and the Log of Typhoid Mortality

Figures D.1 and D.2 present results for each city. Figure D.1 focuses on larger cities with ordinance dates from Anderson et al. (2022), while Figure D.2 includes larger cities with less apparent effects from pasteurization (Panels A–E) as well as smaller cities (Panels F–N).

Focusing on Figure D.1, for each city, the top panel shows the raw data, and the bottom panel presents the synthetic control estimates. In these plots, the treated city appears as a dark blue solid line, the synthetic control as a light blue dashed line, and the ordinance year is marked by a solid purple vertical line. Pasteurization rates, when available, are shown in purple text.

We begin with Chicago (Figure D.1, Panel A), which experienced a sharp decline in typhoid mortality beginning in 1914, the year of its pasteurization ordinance. Although chlorination began two years earlier, the magnitude and timing of the reduction in typhoid suggest a strong and immediate effect of the pasteurization ordinance. San Francisco (Panel B) exhibits a similar pattern: a pronounced drop in typhoid mortality following its 1916 ordinance, with no other major public health interventions implemented concurrently.

Baltimore (Panel C) also shows a drop in typhoid after its pasteurization ordinance, though water filtration was introduced shortly beforehand, and the mortality reduction appears to occur gradually, rather than immediately. In Panels D and E, both Milwaukee and Minneapolis enacted pasteurization ordinances largely in isolation from other public health investments; both show clear post-ordinance declines in typhoid mortality, although their match with their synthetic controls is weaker than in the top panels.

Panel F presents the more complex case of New York City. The city implemented an initial ordinance in 1912 and strengthened it in 1914. Typhoid mortality fell beginning in 1912, but accelerated in 1914. The overlapping interventions in New York, including chlorination and stricter bacterial standards, make it difficult to attribute the observed decline solely to

[22] Modeling the log of mortality rates allows us to estimate proportional changes rather than absolute differences in mortality.

pasteurization.

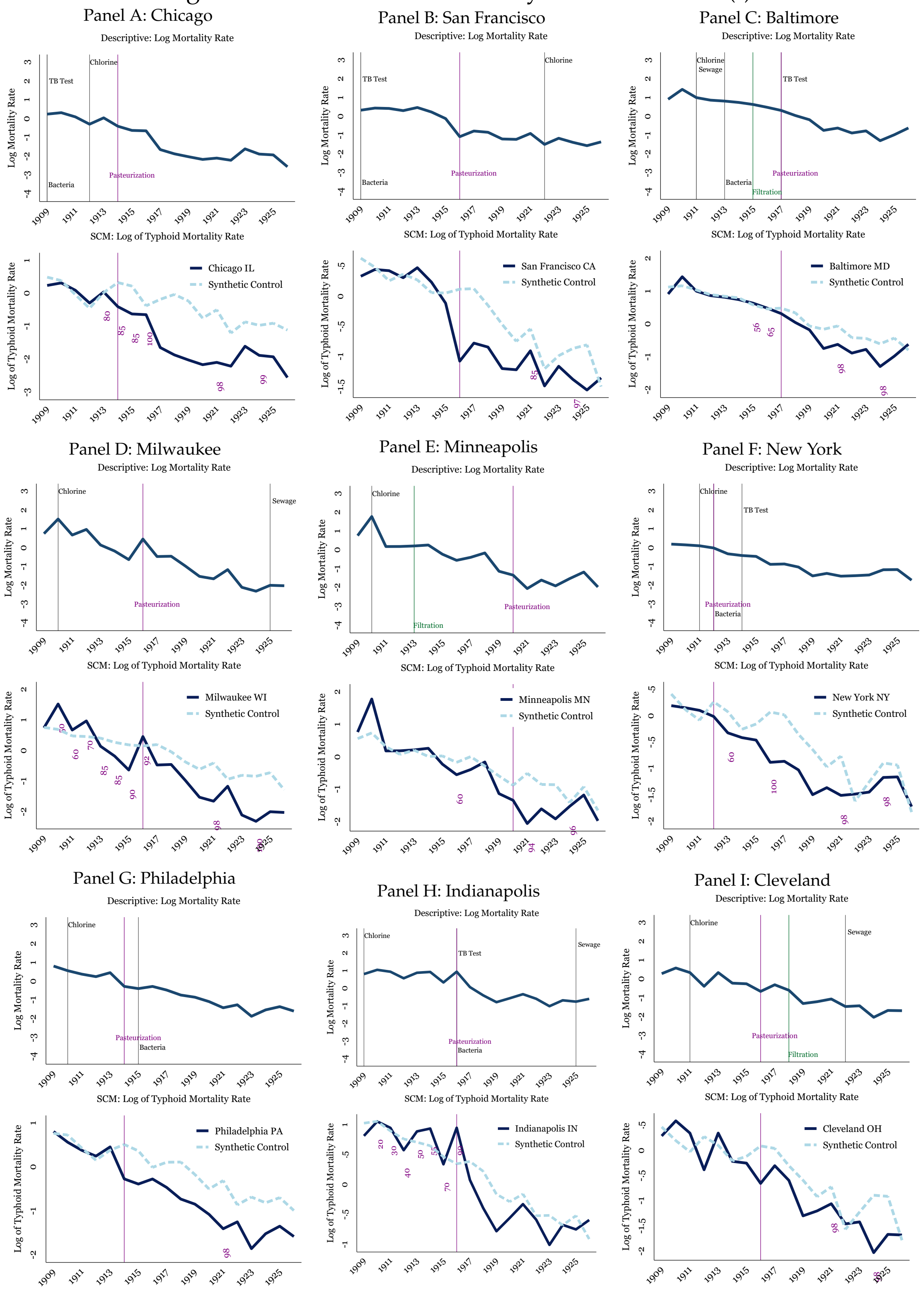
Panel G shows Philadelphia, where chlorination predated pasteurization. Nevertheless, a distinct break in typhoid mortality occurs immediately after pasteurization. Finally, Panels H and I present Indianapolis and Cleveland. Both cities show some decline after pasteurization, but the effects are less pronounced than the remainder of the cities in Figure D.1.

Then, Panels A–E of Figure D.2 include cities where the impact of pasteurization appears limited or ambiguous. In Detroit and Jersey City, both of which rapidly transitioned to near-universal pasteurization, there is no immediate mortality break; Detroit shows some decline only after several years.

Panels F–N of Figure D.2 show smaller cities. About half display evidence of a post-ordinance decline. Grand Rapids shows the most pronounced effect, while Toledo and Dayton experience modest reductions. In contrast, Scranton, Altoona, and Trenton show little or no change following pasteurization.

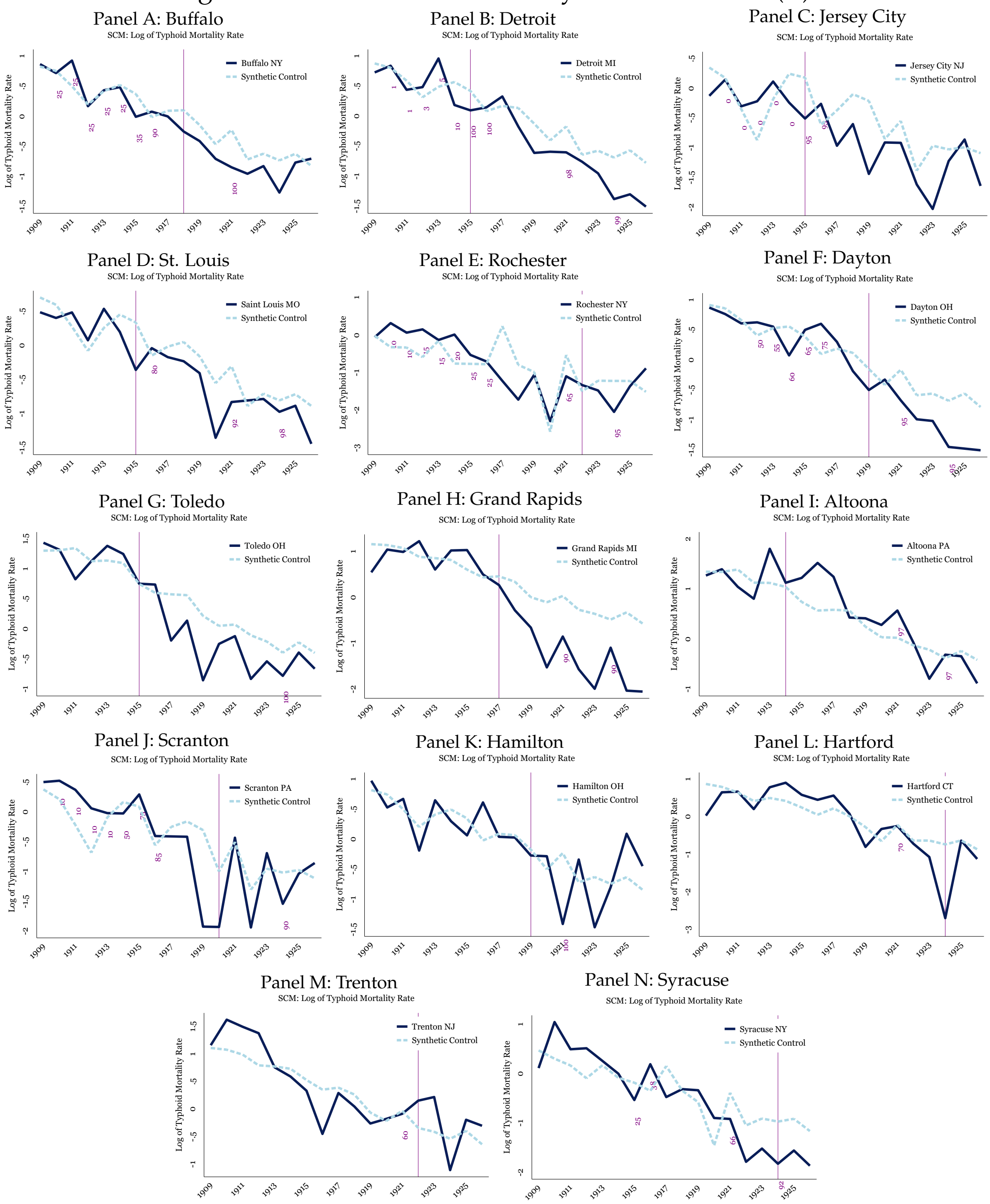
Taken together, these results reveal heterogeneity in the effects of pasteurization ordinances. Cities such as Chicago and San Francisco experienced clear and immediate declines in typhoid mortality, while others saw more modest, delayed, or negligible effects. These patterns highlight the importance of local context, the nuances of policy implementation, and potential interactions with concurrent public health interventions, which we examine in greater depth in Section 7.10.

Figure D.1: Pasteurization and Synthetic Controls (I)



NOTES: Synthetic control methods from synth_runner package (Galiani and Quistorff, 2017). Separately compares the outcome, the log of typhoid fever mortality, for each treated city against a synthetic control city over the years 1909-1926. The synth_runner package requires a balanced panel, so only cities with a balanced panel of non-zero typhoid mortality are considered. The synthetic control group is constructed by matching the log of typhoid mortality over the pre-pasteurization years for each city, 1909, through the pre-pasteurization year (varies by city), and identifying a weighted comparison group based on untreated cities (Abadie et al., 2010). The control group is comprised of cities where we are not missing pasteurization dates based on Fuchs et al. (1939) or Table 1. The year of pasteurization is shown by the solid purple vertical line. Each city's pasteurization rate is in purple text. The top graph shows the public health investment dates from Anderson et al. (2022).

Figure D.2: Pasteurization and Synthetic Controls (II)



NOTES: See notes in Figure D.1. While Panels A-E also have public health investment dates from Anderson et al. (2022), for brevity, we do not show the dates because there is only a limited decline in typhoid fever for these cities. Panels F onward are smaller cities not included in Anderson et al. (2022).