Bismarck's Health Insurance and the Mortality Decline*

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Abstract

We study the impact of social health insurance on mortality. Using the introduction of compulsory health insurance in the German Empire in 1884 as a natural experiment, we estimate difference-in-differences and regional fixed effects models exploiting variation in eligibility for insurance across occupations. Our findings suggest that Bismarck's health insurance generated a significant mortality reduction. Despite the absence of antibiotics and most vaccines, we find the results to be largely driven by a decline of deaths from infectious diseases. Further evidence suggests that statutory access to well-trained doctors was an elementary channel. This finding may be explained by insurance fund physicians transmitting new knowledge on infectious disease prevention.

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"Rarely, if ever, in modern history has a single piece of legislation had such a profound worldwide impact as the German Sickness Insurance Law of 1883 - the cornerstone of German health care policy for almost one century."

– Leichter (1979)

1 Introduction

From the mid of the 19th century, the industrializing world experienced an unprecedented decline in mortality rates. This mortality reduction is central to the demographic transition and had arguably fundamental consequences for long-run growth and productivity.[1] An ongoing debate is concerned with the forces contributing to the mortality decline. Most major advances in medicine such as antibiotics and vaccines did not occur before the 1930s and were not decisive for the considerable reduction of infectious disease mortality during the early phase of the health transition. A large literature discusses the influence of improvements in income and nutrition, directed investments in public sanitation, and scientific advances in health knowledge.[2] Yet, this literature has mostly neglected the emergence of social health insurance at the end of the 19th century.

This paper investigates the impact of Bismarck's health insurance on mortality in Prussia. The Chancellor of the German Empire, Otto von Bismarck introduced the first-ever widely-implemented compulsory health insurance in the world in 1884. Covering large parts of the working population, Bismarck's health insurance (henceforth BHI) was a first move toward democratized access to health care. Subsequently, it acted as a blueprint for Germany's current health system and served as a model for many social insurance systems across the world. Surprisingly, however, econometric studies on the health effects of BHI have been missing so far.

In the first of our two econometric approaches, we exploit the fact that BHI became compulsory for blue-collar workers while other occupations' access to health care remained unchanged. Newly digitized administrative data from Prussia, the largest state in the German Empire, allow us to compute annual occupation-specific mortality rates at the district level for the period 1877 to 1900. We bring these unique panel data to a generalized difference-in-differences (DiD) model that compares the blue-collar mortality trend to the mortality trend of public servants over seven pre-reform and sixteen post-reform years.

DiD estimates indicate that, from its introduction in 1884 to the turn of the century, BHI reduced blue-collar mortality by 6.2–11.4%. Thus, the insurance accounts for 24–45% of the total mortality decline across blue-collar occupations during this period. Common pre-treatment mortality trends across occupations corroborate the validity of the identification strategy. The results are robust to heterogeneous effects of urbanization, the establishment of waterworks, and the roll-out of sewerage. The estimates are

[1] While microeconomic evidence typically confirms the existence of health-induced productivity gains, the relationship between improvements in health and aggregate productivity is subject to ongoing debate. Many studies focus on specific periods of major improvements in health and life expectancy to test this relationship (for some important contributions, see Acemoglu and Johnson, 2007; Ashraf et al., 2008; Bleakley, 2007; Hansen and Lønstrup, 2015; Jayachandran and Lleras-Muney, 2009; Lorentzen et al., 2008; Murtin, 2013; Weil, 2007, 2014).

[2] Seminal contributions include, e.g. Cutler and Miller (2005), Deaton (2013), McKeown (1979), Fogel (2004), and Preston (1975). For recent surveys see Costa (2015) and Cutler et al. (2006).

neither confounded by contemporaneous social reforms nor by improved working conditions or increasing wages for blue-collar workers. The mortality data allow us to capture BHI effects for wives and children of the insured that indicate substantial positive health spillovers within the family, in particular for children. Despite its desirable properties, the DiD approach has some shortcomings. Due to the aggregate nature of the occupation-by-district-level mortality, we cannot exclude issues arising from selection into occupation and spillovers to control-group occupations. Resulting concerns are addressed in a second econometric approach.

The second approach estimates the time-varying impact of the insurance on county-level mortality by exploiting variation in the regional distribution of blue-collar workers fixed at a point in time *before* the introduction of BHI. This fixed effects panel analysis excludes any potential selection into industrial occupations after the introduction of BHI and captures spillovers to other occupations by inspecting overall mortality. The approach also allows us to rigorously control for changes in the age structure of the population, to account for the strength of unions, and to perform specification tests using placebo treatment groups. Fixed effects estimates yield a consistent pattern suggesting that BHI reduced overall mortality in the average county by 0.83 deaths per 1,000 people, thereby explaining 16.5% of the total decline until 1904. This magnitude seems reasonbale given that roughly 15% of the population was eligible for BHI and an additional 25% of the population may have benefited from intra-family spillovers.

We shed light on the channels through which BHI contributed to the mortality decline by inspecting heterogenous effects on various causes of death, the supply of medical professionals, and by comparing the effectiveness of insurance benefits. First, using newly digitized data on causes of death, we show that a large part of the effect is driven by a reduction of mortality due to airborne infectious diseases. Especially the highly infectious tuberculosis, a disease for which a cure was not developed until 1946, declined in response to BHI. Secondly, we show that the BHI effects are larger in counties with a higher share of university-trained doctors and in close proximity to a medical school. Thirdly, using data on insurance benefits, we find that the mortality decline is associated with higher spending on physicians and medical treatment but not with sick pay, the actual main benefit and expense of insurance funds.

These findings lend *prima facie* support to the view that the insurance improved health by providing access to health care rather than through granting sick pay. We interpret the findings to imply that BHI was instrumental in extending access to doctors who acted as transmitters of health knowledge to poor working class families, a group formerly unable or unwilling to afford health care. The view complements earlier conjectures in the historical literature arguing that the insurance provided its members with access to new knowledge regarding hygiene and transmission of infectious diseases (see Condrau, 2000; Koch, 1901; Vögele, 1998). Our interpretation resonates with Mokyr and Stein (1996) who build a model of consumer behavior in which households produce health for its members based on the existing priors about the causes of disease. These priors were radically altered by the germ theory of disease at the end of the 19th century when inputs to the health production function substantially changed. In order for scientific knowledge to affect household priors, a vehicle of transmission is crucial. Indeed, sickness

funds provided such a vehicle by encouraging their licensed physicians to disseminate the new knowledge of infectious disease transmission.

Comparing our results to the literature on historical or recent health interventions is difficult for at least three reasons. First, the health environment, the state of medical knowledge, and the treatment might differ substantially across studies. Secondly, interventions focus on different subgroups of the population with varying health backgrounds. Thirdly, in the absence of clean information on program take-up, many studies report reduced form intention-to-treat effects which are hardly comparable. Keeping these caveats in mind, we may draw parallels between our findings and the literature on modern health insurance. Card et al. (2009) find that extending Medicare eligibility for the 65+ age group led to a mortality reduction by 2–4% within one year. Currie and Gruber (1996) find that expanding Medicaid eligibility to low-income children is associated with a 3.4% reduction in baseline mortality. Our DiD estimates suggest that BHI decreased blue-collar mortality by 6.2–11.4%. The reduced-form BHI effects are therefore considerably larger than extending Medicare or Medicaid eligibility, even though medication was less effective in Bismarck's time.

Because we investigate heterogenous effects by cause of death and for infants, we may also compare these results to historical health interventions. Recent research by Hansen et al. (2017) shows that Danish tuberculosis dispensaries that aimed to prevent the spread of the disease, reduced tuberculosis mortality by 19% between 1907 and 1939. BHI, in comparison, reduced tuberculosis mortality in the average Prussian county by 27%. In the early 20th century, a range of health interventions specifically targeted child mortality. Wüst (2012) finds that a universal home-visiting program in Denmark that advocated breastfeeding and diffused knowledge on nutrition, increased infant survival rates by 0.5–0.8%, which explains 17-29% of the diarrhea-related mortality decline from 1937 to 1949. Moehling and Thomasson (2014) calculate that the U.S. Sheppard-Towner act that provided ongoing access to information and personal interventions, decreased infant mortality rates by 0.7–1.9 deaths per 1,000 births, which explains 9–21% of the decline from 1924 to 1929. Bhalotra et al. (2017) show that a Swedish intervention that provided mothers with nutritional information, non-financial support, and monitoring of infant care, increased infant survival rates by 1.56 percentage points or 24% of the baseline risk in the 1930s. Bütikofer et al. (2019) suggest that Norwegian mother-child health-care centers that granted universal and free access to well-child visits during the first year of life, decreased infant mortality, especially from diarrhea, by 0.8 percentage points, or 18% of the baseline mortality in 1936. While baseline infant mortality in late 19th-century Prussia was substantially higher than in these early 20th-century settings, our estimated magnitudes are roughly similar to those in the literature. Arguably due to intra-family spillovers, BHI reduced infant mortality by 1.6%, which explains 22% of the infant mortality decline until the beginning of the 20th century.

The remainder of the paper is organized as follows. Section 2 discusses the literature on the causes of 19th century's mortality decline, provides background information on Bismarck's health insurance, and discusses consequences for the demand and supply of health services. Section 3 introduces the DiD approach using occupation-specific mortality. Section 4 introduces the FE approach exploiting pre-reform differences in the distribution of

eligible workers. Section 5 provides evidence for potential mechanisms, before Section 6 concludes.

2 Literature and institutional background

2.1 Drivers of the mortality decline

Despite its initially high levels of mortality, Germany's mortality decline at the end of the 19th century is considered to be fairly representative of the demographic transition in many European countries (Guinnane, 2011). Figure 1 shows a distinct break in the long-run mortality trend during the mid-1880s. From 1885 to 1913, the crude death rate in Prussia substantially declined by almost 40%, from 27 to about 17 deaths per 1,000 people. Fluctuations due to higher prevalence of epidemics and war notably ceased. A comparison of country-level time series in Online Appendix A confirms that the 'German penalty' (Leonard and Ljungberg, 2010) was evident in the 1870s but diminished until 1913. After 1884, the inception of BHI, the Prussian mortality decline exceeds all other Western and Northern European countries. Between 1875 and 1905, life expectancy increased from 38.4 to 45.5 years at the age of one and from 43.2 to 47.8 years at the age of 20 to 25 (Imhof, 1994). Similar to other European countries, rural life expectancy exceeded urban life expectancy (e.g., Kesztenbaum and Rosenthal, 2011); boys in rural Prussia outlived their urban counterparts by five years (Vögele, 1998).

The precise factors that contributed to the mortality decline remain disputed. McKeown (1979) and Fogel (2004) suggest that improvements in living standards and nutrition were responsible for the mortality decline in 19th-century Europe, ruling out many other factors. In contrast to their views, the literature broadly agrees that the 'urban penalty' in adult and infant mortality was removed through investments in public health measures during the second half of the 19th century (Meeker, 1974; Szreter, 1988; Hennock, 2000). More specifically, improvements in the water supply (Alsan and Goldin, 2019; Brown, 1988; Ferrie and Troesken, 2008; Beach et al., 2016), water purification (Cutler and Miller, 2005), and sewerage systems (Kesztenbaum and Rosenthal, 2017; Alsan and Goldin, 2019) strongly reduced mortality from waterborne diseases. Cutler et al. (2006) and Leonard and Ljungberg (2010) argue that medical treatment hardly contributed to the mortality decline before 1914. As a notable exception, smallpox vaccination had became available in 1796 already and largely reduced infant mortality (Hennock, 1998; Ager et al., 2018). In 1891, the first drugs against diphtheria emerged. A range of antiseptics and anesthetics were widely used. More generally, the end of the 19th century saw a significant progress in chemistry that allowed identifying effective ingredients of medicinal plants and producing new chemical drugs.

In general, scientific medical knowledge considerably deepened towards the turn of the century (Mokyr and Stein, 1996). The prevalent belief that diseases were transmitted through bad smells (*miasmas*) was gradually replaced by scientific findings identifying the role of bacteria as transmitters of diseases. Major breakthroughs in epidemiology, including the well-known discoveries of water as a transmitter for Cholera by John Snow and William Budd, and numerous discoveries in bacteriology by Robert Koch, Louis Pasteur, Ignaz Semmelweis, and others occurred in the second half of the 19th century

(for details see Easterlin, 1999).[3] Advances in bacteriology altered established knowledge across all types of infections related to waterborne and airborne diseases. In fact, Mokyr (2000, p. 15) recognizes germ theory to be "one of the most significant technological breakthroughs in history." However, mere identification of the root cause of infections was insufficient to cure the sick in the absence of remedies. All that physicians could do was to "educate patients on hygiene" (Thomasson, 2013, p. 177).

The role of knowledge diffusion in improving health has recently gained the attention of economists. Deaton (2013) argues that upward shifts of the Preston curve are driven by the *application* of new knowledge. In the early 20th century, hygiene knowledge was disseminated via health care centers, congresses, and public information events around the developed world. Nordic countries were particularly progressive in diffusing knowledge through well-child visits and health care centers. A recent literature shows that such interventions were highly successful in reducing infant mortality and improving long-run health (see Wüst, 2012; Moehling and Thomasson, 2014; Bhalotra et al., 2017; Bütikofer et al., 2019). Hansen et al. (2017) provide evidence that the introduction of tuberculosis dispensaries in early 20th century Denmark significantly decreased TB mortality by disseminating knowledge on disease transmission to patients, their families, and the public.

Public interventions also include the introduction of health insurance across European countries at the turn of the 20th century. According to Vögele (1998), health insurance funds might have contributed to the penetration of new knowledge and health education in the German Empire.[4] Guinnane (2003, p. 45) supports this hypothesis by noting that sickness funds played a major role in strengthening the role of physicians as advocates of hygiene. Kintner (1985) argues that physicians and midwives represented a major source for disseminating information on the health effects of breastfeeding to pregnant women.[5] In addition, Tennstedt (1983, p. 461) suggests that sickness funds fostered prevention of disease transmission by introducing new rules and benefits for workers' families, workers' hygiene and lifestyle, the workplace, and employers' responsibilities.

Econometric evidence for the role of early health insurance programs is scarce. Winegarden and Murray (1998) provide evidence that health insurance coverage across five European countries ia associated with mortality reductions before World War I. They find that a 10% increase in the insured population results in a mortality reduction of 0.9 to 1.6 per 1,000 people. Bowblis (2010) extends this study to eleven countries and shows that a 1 percentage point increase in coverage is associated with a decline of 0.7 infant deaths per 1,000. He speculates that health insurance affected infant mortality by "educating people about the benefits of clean houses, not re-using dirty bath water, washing

[3] The role of hygiene, as an important tool to prevent infectious diseases in hospitals, became generally appreciated in the 1880s, twenty years after the death of Ignaz Semmelweis, the pioneer of modern antisepsis (Murken, 1983). Yet, it was not before Alexander Fleming discovered penicillin in 1928 that antibiotics became widely known as drugs that could fight bacteria.

[4] Vögele (1998, p. 199-208) lists a range of potential channels through which BHI might have improved health, including preventing families to fall into poverty due to sick pay, increasing access to doctors and hospitals, or by allowing the state to systematically educate and control the covered population with respect to their attention to health issues.

[5] Breastfeeding was more widespread in Prussia than in southern Germany. Based on the limited available data, Kintner (1985) and Kintner (1987) find that breastfeeding increased in Baden and Munich whereas it declined in Berlin between 1885 and 1910. Vögele (1998) argues that improvements in the quality and supply of cow milk were marginal in improving infant mortality. Imperial regulations on milk quality occurred after 1901.

hands, and isolation of sick family members from the rest of the household" (Bowblis, 2010, p. 223). Finally, Strittmatter and Sunde (2013) show that the reduction in mortality due to the introduction of public health care systems across Europe translates into positive effects on growth in income per capita and aggregate income.[6] These studies provide an interesting yardstick for our findings that rest on econometric approaches which additionally allow to flexibly control for general mortality trends within the country, to exclude time-invariant factors that are specific to subregions, and to explore the potential channels through which BHI affected mortality.

2.2 Bismarck's health insurance

The Compulsory Health Insurance Bill of 1883 constituted the birth of Germany's social security system. Bismarck's health insurance was the first of the three main branches of the German Social Insurance System, followed by the Accident Insurance Bill (1884) and the Old Age and Disability Insurance Bill (1889, adopted in 1891).[7] Chancellor Otto von Bismarck's decision to introduce the compulsory health insurance was a reluctant reaction to mounting upheavals among the working class. The Industrial Revolution led to increasing social tension between the rising working class and the political and economic elite. The new Socialist Workers' Party of Germany (SAPD) gained support among the lower strata of the population and became a threat to the political stability of conservative dominance in the German parliament. Against this backdrop, Bismarck's attempt to win over votes from the socialist party and the worker unions through the health insurance was coined *mass bribery* by Rosenberg (1967). Furthermore, the reform disburdened public funds by shifting the cost of poor relief to workers and employers. Parliament approved the law on May 31, 1883, against the votes of the Social Democrats who argued that this social reform would not really improve the workers' situation (Tennstedt, 1983). From a political perspective, contemporary supporters of BHI argued that the reform was costly but "bought social peace for Germany" (Leichter, 1979, p. 124).

From December 1st, 1884, BHI was "compulsory for all industrial wage earners (i.e. manual laborers) in factories, ironworks, mines, ship-building yards and similar workplaces" (Bill of June 15, 1883, see Leichter, 1979). Contributions were earnings-related, amounted to an average of 1.5% of the wage[8] and were paid jointly by employers (one-third) and employees (two-thirds). Other occupational groups, including public servants, farmers, domestic servants, day-laborers, or the self-employed were not eligible for BHI. The Bill only specified maximum contributions and minimum benefits so that sickness funds had "considerable discretion to set specific benefits and contribution levels" (Leichter, 1979, p. 123).

In case of sickness, the insured were eligible to receive sick pay amounting to at least 50% of the average local wage for 13 weeks. Further statutory benefits included free medical and dental care for the insured, prescribed medicine, incidental care for up

[6] Their cross-country analysis is based on data from twelve European countries. Due to a lack of data availability, they exclude Germany, the country which introduced the first public health insurance system and boasts the largest share of insured at the turn of the 19th century.

[7] Scheubel (2013) provides an excellent overview of Bismarck's social security system. Fenge and Scheubel (2017) show that the introduction of the old age pension and disability insurance reduced fertility, while Guinnane and Streb (2011) provide evidence for moral hazard effects of the accidence insurance. Lehmann-Hasemeyer and Streb (2018) find that Bismarck's social security system as a whole crowded out private savings.

[8] Contributions were confined to a maximum range of 3-6% of the wage.

to 13 weeks, and treatment in hospitals for up to 26 weeks. Funds were allowed but not obligated to extend free health care to dependent family members.[9] Moreover, the insurance provided maternity benefits encompassing free medical attention and a cash benefit (*Wochenhilfe*) for three weeks after giving birth. In case of death, the insurance paid a death grant to the family of the insured.

The health insurance system was administered in a decentralized manner by local sickness funds (*Krankenkassen*). Where possible, Bismarck built upon pre-existing funds, i.e., in the building trade, in mining (*Knappschaften*), by local guilds, and various industrial sickness funds.[10] This saved both time and state resources and was sensible from a political perspective because it respected the guilds' and unions' traditional position as insurance providers for their members. In addition, two new types of funds, the local funds (*Ortskrankenkassen*) and the parish funds (*Gemeindekrankenkassen*) insured all eligible workers not covered by other funds. Local and parish funds attracted the lion's share of the newly insured workers and served 59% of insured individuals by 1905. Each fund was self-governed by an elected board consisting of the insurance contributors, i.e., workers and employers. Because contributions were statutory, funds were able to collect them in accordance with public law (Wagner-Braun, 2002, p. 77).

Figure 2 depicts the development of health insurance coverage over time.[11] Prior to 1885, health insurance was only mandatory in the mining industry and a few local funds. The data suggest minuscule increases in the insured population until 1876, the latest available pre-BHI data point, after which the coverage surged from 3% to 8% in 1885 and further increased to 11.6% within five years. By the turn of the century, the share of insured in the population had quintupled (from 3% to 15%). After issuing the Bill in 1883, municipalities and other institutions had more than a year of preparatory time to set up the insurance funds. Yet, the very early period of BHI did not pass without frictions.[12]

Workers were initially unenthusiastic about the insurance and concerned about employer's resentments regarding their share of contributions. As a result, in 1885, 19% of workers remained registered with the voluntary funds, which did not require contributions by employers, and employers preferred to hire workers with such insurances (Tennstedt, 1983, pp. 318–322). With time, suspicions dropped and the share of workers in voluntary funds steadily declined to less than 10% in 1893. Huerkamp (1990, p. 74) argues that "insured workers increasingly relied on physicians in all cases of illness and thus accepted them as professional experts" and Labisch (1985, p. 608) infers that workers started to accept "the offer of value-free 'health' as a scientifically-based mode of life". Similarly, employers gradually became sympathetic towards the compulsory health insurance. In particular, the large scale and heavy industries embraced the reform because the compulsory nature of BHI levelled the playing field such that competitors could no longer gain a

[9] Ewald (1914, p. 522) conjectures that approximately 10% of the insurance funds provided family benefits.

[10] Only few workers from minor industries were covered by these particular insurance funds; most of them were members of the *Knappschaften* which provided insurance against accident, illness, and old age from 1854 (see also Guinnane and Streb, 2011).

[11] Table B.1 in the Online Appendix shows the exact numbers of insured and total population of Prussia over the years.

[12] Based on the occupation census of 1882, officials in Dresden were expecting 45,000 workers from 8,665 firms to be liable for compulsory insurance. By mid 1885, only 30,000 workers were registered and 3,000 employers had yet to report their workers. Similar compliance rates were reported from Leipzig (Tennstedt, 1983, p. 319).

cost advantage at the expense of the social security of workers (Tauchnitz, 1999, p. 58). Employers also expressed satisfaction that the standardized organization and compulsory nature of BHI had a "socially integrative and disciplining effect" on workers (Tauchnitz, 1999, p. 66). Finally, employers gained access to the sickness funds' administration through which they were better able to enforce their notion of order against unions and workers (Tauchnitz, 1999, p. 66). This provided a major incentive for employers to register workers with mandatory funds rather than voluntary funds. Compared to the eligible population share of 14.9% (see Table B.2 in the Online Appendix), the actual coverage of 12.6% in 1895 suggests a broad take-up ten years after BHI was introduced.

2.3 The changing demand and supply of health services

Historical accounts convey that BHI increased the demand for health goods and services. The insured consulted physicians far earlier and more frequently than the uninsured and Huerkamp (1985, p. 202) argues that a large share of the newly insured would not have been able to afford consulting a physician in the absence of BHI.[13] Specifically, Huerkamp (1985, pp. 207–208) suggest that uninsured patients would pay four to five times the consultation rates reimbursed by the funds for their members. Considering an average compensation of 2.55 Marks for insurance physicians, the price for uninsured patients would amount to approximately 60–70% of a week's wage for the average blue-collar worker. Because insured patients did not bear the immediate costs of doctor visits, they did not face a sudden trade-off between consuming health care and basic subsistence goods. Consequently, they increasingly made use of consultation hours. The stereotypical doctor's complaint that patients came for consultation only when it was too late turned into complaints that patients came in for petty indispositions (Huerkamp, 1985, p. 201).[14] Moreover, BHI became a key driver of the increased utilization of hospital capacity in the 1890s, where the majority of inpatients were treated for non-communicable skin, eye and surgical diseases, i.e. treatment of injuries and wounds (Spree, 1996). In 1901, half of the patients in Düsseldorf hospitals were compulsory insured (Vögele et al., 1996).

Seeing a doctor was a necessary prerequisite for being eligible for insurance benefits. In particular, insured individuals were obligated to consult an insurance physician after three days of absence from work. Unless the doctor attested the inability to work, the worker would not receive sick pay. In principle, sick pay constitutes an improvement to the previous situation in which workers received no income while being sick at home. However, contemporaries typically criticized that workers were hardly able to subsist on half of the local daily wage and historical accounts suggest that they preferred returning to work as soon as possible (Ellerkamp, 1991). Sickness inspectors (*Krankenkontrolleure/Krankenbesucher*), initially hired to detect malingering via unannounced home visits, eventually became mandated to monitor the curfew and medication intake of pa-

[13] The chairman of the Imperial Insurance Agency Tonio Bödiker claimed that less than half of the worker's families would have consulted a doctor before the introduction of the compulsory insurance (Huerkamp, 1985, pp. 207–208).

[14] This notion is supported by contemporary sources suggesting that only half of the consultations justified a period of sick leave (Huerkamp, 1985, p. 202).

tients.[15] [16] Moreover, physicians and inspectors specifically aimed at triggering changes in the hygienic behavior of all members of the household through particularly addressing women, who were typically in charge of care and food (Frevert, 1981).[17]

The historical literature agrees that there was no shortage of physicians in the German Empire. In fact, the steep increase in the demand for health services by the insured was accompanied by an increase in supply of university trained doctors (employed by the sickness funds). From 1870 to 1890, the number of medical students in Prussian universities more than tripled (from 2,600 to 8,724), whereas the number of students in other disciplines roughly doubled. Between 1876 and 1909, the number of approbated doctors increased from 13,728 to 30,558 whereas the patient-to-doctor-ratio declined by 36% from 3,112 to 1,984 in the German Empire (Huerkamp, 1985, p. 151). This contrasts with the situation in Britain, where the patient-to-doctor-ratio in the same period declined only by 26% (Weindling, 1991, p. 212). It is therefore unlikely that BHI led to a crowding out of uninsured patients. Weindling (1991, p. 212) rather asserts an 'overproduction of medical graduates during the 1880s'. There was 'stiff competition' (Huerkamp, 1990, p. 73) between doctors as insurance treatment was concentrated 'in the hands of relatively few company doctors' (Huerkamp, 1990, p. 76) so that physicians were increasingly forced to move from urban areas into the countryside.

German scholars were leading in many medical fields and attracted international students to German universities (Richmond, 1954; Tomes, 1997). Germany's reliance on licenses to practice medicine (*Approbationen*) facilitated the standardized medical training of doctors. The legal requirements for approbations granted by imperial authorities comprised at least nine semesters of university training, including two semesters of clinical internship, and a successful state examination. The exam consisted of six written and clinical tests of knowledge in anatomy, physiology, pathology, surgery and ophthalmology, medicine, obstetrics and gynecology, and an oral interview in hygiene.[18] Weindling (1991, p. 214) argues that the reliance on scientific qualification was characteristic of the professionalization in German medicine that stands in "marked contrast to the attitude towards science adopted by the Anglo-American medical profession". Different from Britain or the US, where "scientific superiority was not necessary for professional success", social status derived from university training and academic titles helped doctors to attract patients in Germany (Weindling, 1991, p. 204).

These developments were highly supportive for successful treatment and surgery. Due to their living conditions, the incidence of infectious diseases was high among blue-collar workers and they may have benefited more from such progress than other groups in society. However, germ theory had yet to become public knowledge. Only after the turn of the century, insurance funds also initiated public educational talks by insurance doctors

[15] In 1896, the municipality fund of Leipzig conducted 79,332 visits by voluntary inspectors and 149,899 visits by professional inspectors (Tennstedt, 1983, p. 451).

[16] The sickness attestation by the doctors included information on mandatory bed rest and curfew hours. Patients lost their claim to sick pay in case of noncompliance.

[17] According to Tennstedt (1983, p. 458) funds specifically considered to deploy female inspectors to give advice to *their uneducated sisters* on *how to ventilate and clean the apartment, curtains and other dust catchers.*

[18] "For the clinical subjects, the student was assigned the care of six patients (two each in medicine, surgery, and gynecology) for a period of eight days, during which he was quizzed about his diagnoses, recommended treatments and prescriptions, and knowledge of surgical procedures. [...] In no other country were such uniformly high standards for practice found before the twentieth century." see Bonner (1995, p. 254)

(see Ewald, 1914). Talks were well attended by workers only if they were organized by health insurance funds (Vögele, 2001, p. 375 ff). The self-administration gave the events a working-class appeal so that they were not condemned as bourgeois-elitist attempts to patronize workers (Tennstedt, 1983, p. 462). Insurance doctors thus provided a necessary gateway to gain access to new medication and knowledge about the importance of hygiene in avoiding infections.[19]

3 Difference-in-differences: eligibility by occupation

In this section, we exploit the fact that BHI became mandatory for blue-collar workers in 1884, but not for other occupations. This constitutes a natural setting for a reduced form DiD model, in which we compare the mortality trends of the treated group to a control group. Two characteristics qualify public servants as our preferred control group. First, similar to blue-collar workers, public servants are likely to live in urban areas and thus experience the same structural changes to their living environment. Second, public servants did not become eligible for compulsory health insurance before 1914. Prussian civil servants were, however, eligible for continuation of salary payment during illness and a pension in case of disability or old age.[20] Yet, public servants did not receive benefits such as free doctor visits and medication. Most importantly for our identifying assumption, their benefits were not subject to change in the period 1873 to 1914.

3.1 Data and econometric specification

The DiD specification draws on unique administrative data from Prussia, the largest state in the German Empire. The Royal Prussian Statistical Office published annual death statistics (see KSBB (1861–1934)) by occupational group for all 36 Prussian districts.[21] We extract fourteen occupational groups that are consistently reported between 1877 and 1900 and can be identified as either blue collar or public servant following the classification of the statistical office.[22] Within each occupational group, we can distinguish between deaths of adult men, adult women as well as male and female children below the age

[19] Labisch (1985, pp. 609–610) describes the situation as follows: "[...] it was that health insurance and communal health services became the entry-gates through which medico-hygienic ideas percolated among the workers and their political representatives. [...] housing inspectors, advised by socialist doctors, gave working-class women practical instruction in how to air and clean the home, how dust and dirty curtains helped to transmit tuberculosis, how foodstuffs (summer diarrhoea for the babies!) should be kept, and so on. Here, too, there was fertile ground for utopian plans: 'Every Saturday, one workers' housing quarter should be flooded with health inspectors, the women observed as they wash and clean their steps, rooms and houses, and instructed about the main risks they are exposing themselves to in the process, and reports on all this should then be made to the centre, the *Krankenkasse* [sickness fund]; where there is resistance, there must be further inspections and calls, so that in the course of a year the whole of Berlin will have been visited and educated in hygienic culture. Thus, it was the workers' doctors and the *Krankenkassen* [sickness funds], committed on the one hand to the scientific concept of social hygiene and on the other to the political concept of social democracy, who formed the personal and institutional link between the scientific idea of 'health' and the world of the working class. And it was with eager enthusiasm that the workers adapted themselves to this new form of life."

[20] These benefits were confirmed after German unification by the Imperial Law on the Legal Relationship with Public Servants of 1873.

[21] In the Prussian administrative hierarchy the district (*Regierungsbezirk*) ranks above the county (*Landkreis*).

[22] Blue-collar occupations encompass thirteen groups listed as sector B in the statistics: mining and turf, minerals, metals, machinery, chemicals, fossil fuels, textiles, paper and leather, wood, food, apparel and cleaning, construction, and printing. Public servant occupations, listed as sector E in the statistics, include: public administration, military, church and education, health, and arts and entertainment.

of fourteen. Deaths of non-employed females and children are allocated to occupational groups according to the occupation of their husband or father, respectively.[23]

Due to the rapid industrialization of Germany in this period, the occupational groups experienced different growth rates leading to differences in the growth of the population at risk (see Table B.2 in the Online Appendix for details).[24] Therefore, we supplement the death statistics with data from occupation censuses to compute occupation-specific mortality rates. The occupation censuses were exclusively conducted in the years 1882, 1895, and 1907 under the supervision of the Imperial Statistical Office (KSA (1884–1942)). Because the occupation censuses follow the same classification, we were able to consistently match the death data to their respective population for the fourteen occupational groups. Gaps between census years were filled by linear interpolation and extrapolation to obtain annual estimates of the respective size of each occupational group.[25] The resulting dataset consists of mortality rates for 12,096 occupation-by-district-by-year observations coming from 14 occupational groups, 36 districts, and 24 years.

In Figure 3, we plot the mean crude death rate of blue-collar groups (black solid line) and the mean crude death rate of public servants (black dashed line) against years. The grey solid line depicts the counterfactual mortality trend of the treatment group, i.e., the hypothetical mortality trend followed in the absence of BHI, assuming that the mortality trend of public servants resembles an untreated mortality trend of blue-collar groups. Throughout the entire period, blue-collar mortality is higher than the mortality of public servants. Prior to BHI, both groups follow approximately the same mortality trend. Only after the introduction of BHI, blue-collar mortality falls more steeply than the mortality of public servants. This can be seen most clearly in the considerable departure of the actual from the counterfactual mortality trend. This descriptive pattern suggests a negative treatment effect of BHI on blue-collar mortality.[26]

We bring these statistics to an econometric framework by estimating a DiD model expressed by the following Equation 1:

$$Death_{oit} = \alpha_{oi} + \theta_{it} + \sum_{t=1877}^{1900} \beta_t (BlueCollar_{di} \cdot T_t) + X'_{it} BlueCollar_{di} \gamma_{+} \varepsilon_{oit} \qquad (1)$$

[23] In 1882, 4.2% (0.4%) of all women and 1.4% (0.2%) of married women were blue-collar workers (public servants). Consequently, female mortality data predominantly reflects non-employed wives of blue-collar (public servant) husbands. Similarly, child mortality reflects the mortality of children of blue-collar (public servant) fathers because Prussian legislation prohibited employment of children below the age of thirteen from 1855.

[24] Note that this period is not a period of substantial warfare. The Franco-Prussian war of 1871 did not affect Prussian territory and civilians. The war created 30,000 veterans suffering from long-term disabilities and/or diseases. Even so, there is no evidence for the year 1884 constituting a crucial turning point in veteran's mortality.

[25] Different from the death statistics, the occupation censuses report non-working family members in a single category. Thus, when calculating occupation-specific mortality rates for children and women, we can only use the total occupational population (the sum of workers, dependent females, and children, but excluding servants). We tested the robustness of our results to replacing this denominator with the occupation-specific male working population. The results are qualitatively similar. We also tested interpolation by spline or based on urban population growth, which does not require linear extrapolation of years prior to 1882. The results are qualitatively similar.

[26] In Figure C.1 in the Online Appendix, we plot the crude death rates for each of the eleven provinces of Prussia to show that the decline occurs across all regions. In this figure, the province of Brandenburg contains the imperial city of Berlin and the province Rheinland contains Sigmaringen due to the external administration of the health sector. In Figure C.2 in the Online Appendix, we plot crude death rates after collapsing the deaths of all blue-collar occupations o and districts i into one mortality rate thereby adjusting for differential size of the occupational groups.

$Death_{oit}$ is the mortality rate of occupational group o in district i in year $t \in (1877-1900)$. α_{oi} are occupation-by-district fixed effects accounting for any time-constant mortality differences between occupational groups in each district. θ_{it} are district-by-period fixed effects that flexibly allow mortality trends to differ across districts. These fixed effects pick up a range of shocks to the district-level health environment affecting all occupational groups, such as overall improvements in nutrition due to variation in harvests and food prices, or differences in temperature especially affecting infant mortality. $BlueCollar_{di}$ is a dichotomous variable that nests occupational groups and is unity for the 13 blue-collar groups and zero for public servants. We interact this treatment indicator with all year dummies T_t. Under the assumption that the mortality of blue-collar groups and public servants follow the same time trend in absence of the treatment, the β_t coefficients measure unbiased intention-to-treat effects (ITT) of BHI. By letting β vary over time, we generalize the standard DiD model to perform placebo tests in the pre-treatment period and allow for potentially time varying ITT effects. ε_{iot} is a mean zero error term. Standard errors are clustered at the occupation-by-district level to allow for serial occupation-specific autocorrelation within districts.

We introduce a vector of time-varying control variables X'_{it} to address concerns regarding changes specific to the urban environment that may coincide with the introduction of the health insurance. This vector includes measures for public health interventions frequently used to explain declining mortality in 19th-century Europe and the U.S., i.e., waterworks and sewage, as well as the urbanization rate. Using administrative data from Galloway (2007), we calculate urbanization rates the are linearly interpolated between the quinquennial census years. We combine this data with information from Grahn (1898-1902) and Salomon (1906-1907) who report city level dates of the establishment of public water supply and sewerage systems, respectively. Assuming that the entire city population benefited from the introduction of sanitation infrastructure, we calculate the district-level share of the population with access to waterworks and sewerage.[27] These control variables can be constructed for the district level but cannot be broken down by occupational group. To avoid multicollinearity with the θ_{it} fixed effects, specification 1 explicitly allows these covariates to have differential effects for the treatment and control group by introducing the interaction term $X'_{it}BlueCollar_{id}$. Allowing urbanization rates to differentially affect blue-collar groups and public servants accounts for the fact that working-class city quarters may have been differentially affected by changes in population density due to city growth at the intensive margin. Occupation-specific interactions of waterworks and sewerage will exclude that the results are confounded by heterogenous access to sanitation infrastructure.

3.2 Main results

Similar to an event study, Figure 4 plots β-coefficients, estimated from a generalized DiD model corresponding to Equation 1, over time. Estimates marked by a circle derive from a specification without control variables. The pattern confirms the impression obtained from the descriptive Figure 3, namely that BHI reduced mortality and that this negative effect

[27] The waterworks data is extrapolated to cover the post 1898 period. Figures C.3 and C.4 in the Online Appendix show waterworks and sewerage systems established in Prussian cities per year as well as their cumulative distribution functions. Table D.3 in the Online Appendix provides summary statistics on the waterworks, sewerage and urbanization variables.

gradually increases over time. Estimates marked by diamonds add urbanization rates and access to sanitation infrastructure (sewerage and waterworks) as control variables. Finally, estimates marked by rectangles derive from a specification, which additionally controls for potentially diverging pre-treatment trends between treatment and control group.[28] The estimates are equally stable across these three specifications.

Most of the annual pre-treatment coefficients are small and statistically not different from zero. This means that treatment and control group indeed followed a very similar mortality trend prior to Bismarck's reform, which corroborates the parallel trends assumption. Yet, we would like to cautiously qualify this conclusion by pointing out that the year 1883 constitutes an inexplicable outlier to the general pattern.

After the introduction of BHI, blue-collar mortality slightly increases for two years. Considering the initial frictions in the roll-out, we do not expect that major improvements in health occur immediately after the reform but that any effects should unfold over time. Such a delayed health response to the insurance is also well in line with the literature on historical health interventions (see Hansen et al., 2017, p. 26). This, however, does not explain the marginally significant short-lived deterioration in health in 1886. Later, we show that this short-lived positive effect on mortality is not a general phenomenon but one driven by the specific subgroup of male children (see Section 3.3) and one that is not robust across specifications (see Section 4.2). Therefore, we avoid over-interpreting this result.

Following two years of rising mortality after the introduction of BHI, a sustained mortality decline occurs. The emerging negative effects are statistically significant, economically meaningful, and increase in magnitude over time. BHI reduced blue-collar mortality by 1.158 to 2.138 deaths per 1,000, i.e., by 6.2–11.4% (given a baseline mortality of 18.755 in 1884) at the end of the 19th century. In other words, BHI accounts for 24–45% of the total mortality decline across blue-collar groups ($18.755 - 13.980 = 4.775$) in this period. Note that these DiD estimates depict reduced form intention-to-treat effects. Provided that full insurance take-up of blue-collar workers was delayed, the increasing magnitude of effects may exclusively be driven by increasing compliance. If we rescale the ITT estimates by the corresponding share of insured among blue-collar workers (computed from Tables B.1 and B.2), roughly a third of the increase in the coefficients can be attributed to the expansion of coverage. The remaining two thirds can be attributed to increases in the intensive margin of the insurance effect.

We include several robustness checks for the DiD specification in Online Appendix C. We estimate Equation 1 after collapsing the mortality of the blue-collar occupations into one sector-mortality rate, thereby adjusting for differential size of the occupational groups[29] (see Figure C.2) and subsequently into blocks of four years, thereby reducing the influence of outlier years (see Table C.2). We test robustness to the inclusion of several district-level covariates (i.e., average age, SPD vote share, wheat yields, and Protestant share, all interacted with occupational group indicators; see Figure C.5). We address

[28] We adjust mortality for occupation-specific linear trends. In line with Wolfers (2006), these trends were estimated based on data of the pre-treatment period 1877–1884 only and then extrapolated for the post-treatment years.

[29] While our main DiD specification assigns the same weight to each occupational group, collapsing mortality across groups essentially weighs each occupational group according to its size.

concerns related to the linear interpolation of the denominator between occupational censuses by using a spline interpolation (see Figure C.6). Finally, we estimate log-linear DiD models using the natural logarithm of deaths as a dependent variable and controlling for the log size of the occupational group (see Figure C.7). This addresses the concern that achieving an accentuated drop in mortality may be easier if the pre-existing level is high. All these analyses univocally confirm our previous findings.

3.3 Effect heterogeneity: men, women, and children

The aggregate BHI effects might hide interesting effect heterogeneity between men, women, and children. Different from blue-collar men, most blue-collar women and virtually all blue-collar children are non-employed (thus, in general not eligible for BHI) and could thus benefit from BHI only indirectly via their insured husband, or father, respectively. We disaggregate the occupation-specific death rates to obtain separate death rates for men, women, and children and use these as outcome variables in the DiD. Figure 5 shows that BHI's negative mortality effects are present for adult males and children of both gender, whereas we do not find any significant reduction in adult female mortality.

The mortality decline for children indicates that uninsured individuals could also benefit from changes brought about by the health insurance. At least three channels may explain our findings but remain undistinguishable with the given data. First, some sickness funds voluntarily extended treatment and medication to all family members. Second, sick pay may result in stabilized household income, thereby facilitating continuous calorie intake and nutritional prospects of household members. Third, treatment and medication may affect health of uninsured family members because medication and knowledge on infectious disease transmission received during treatment by physicians can be shared with household members.[30] Such intra-family spillovers have recently been confirmed by Chen et al. (2018) who find that, even today, children hugely benefit from access to health information in the family, i.e., when one of their parents is a health professional. Moreover, Fletcher and Marksteiner (2017) show that individuals benefit from adopting behavioral changes of their spouses during health interventions related to alcohol and cigarette consumption.

Intra-family spillover effects may be larger for children than for adult females because children react more strongly to changes in the health environment than adults. This argument is prevalent in the literature on early human capital development, nutritional status, and the fetal origin hypothesis (Subramanian and Deaton, 1996; Deaton, 2007; Case and Paxson, 2008; Douglas and Currie, 2011).[31] Another reason may be associated with the role of women as caregivers in the industrial household. Mokyr (2000) argues that the new knowledge on hygiene increased the workload of homemakers. This increasing

[30] Until the end of the 19th century, hygiene education was not part of schools' curricula, teachers were not particularly trained to educate students in hygiene issues, and doctors were not systematically integrated in the school system to examine pupils and inform parents (e.g., Leuhuscher, 1914; Krei, 1995; Umehar, 2013). Therefore, positive knowledge- and health-spillovers from children to their parents are rather unlikely.

[31] For the case of air pollution, it is argued that children are more vulnerable because their lungs and their immune system is still immature. They spend more time outdoors and are therefore exposed to higher levels of air pollution and in particular to a higher volume of air exchange relative to body mass (see, e.g., Currie and Neidell, 2005; Currie et al., 2009; Coneus and Spiess, 2012; Beatty and Shimshack, 2014; Rodrigues-Villamizar et al., 2015). Currie et al. (2013) bring forward analogous arguments for the case of water pollution.

burden may have offset benefits for adult females.[32] Yet, note that in Section 4.2, we will show that the lack of an adult female effect is not consistent across specifications.

3.4 Threats to identification

Bismarck's old age and disability insurance

Bismarck's old age and disability insurance, the third pillar of his welfare system, was introduced in 1891, i.e., seven years after BHI. Different from the health insurance, workers in agriculture, white collar occupations, and domestic services were eligible for this type of insurance in addition to blue-collar workers. While the actual number of contributors was unknown to the administration, recent estimates amount to 23% of the total population (see Kaschke and Sniegs, 2001, p. 14). The retirement age of 70 was, however, rarely attained (1.1–1.6% of insured in 1891–1904) and the pension averaged at only 18% of the wage. Disability claims amounted to twice as many cases than old age claims by 1900, increased steadily in numbers, but resulted in slightly lower pensions.[33] Using 1891 as the baseline year, we find that the introduction of the third pillar does not constitute a considerable trend break (see Figure C.8 in the Online Appendix). Blue-collar mortality declines well before 1891 and proceeds to do so thereafter. Indeed, there is no particular pattern in the data suggesting that the year 1891 changed relative blue-collar mortality in a meaningful way.

Working conditions and factory regulation

If the introduction of BHI coincided with improvements in industrial working conditions, leading to a stronger mortality decline for blue-collar workers than for public servants, our estimates may be biased. Such a scenario is rather unlikely, as the period under analysis is a period of ongoing, rapid industrialization. The typical industrial job was physically demanding, workers were remunerated via piece rate schemes, working hours were extensive, breaks were irregular, and food intake during working hours insufficient (see, e.g., Pietsch, 1985; Paul, 1987; Berg et al., 1989). What is more, the relationship between workers and their employers was characterized by an authoritarian style, where employers disciplined employees using harsh measures (see, e.g., Frevert, 1981).

The Trade, Commerce and Industry Regulation Act (*Gewerbeordnung*) of 1878 adopted the Prussian industrial code of 1855 into an imperial law, barring all children below the age of thirteen from any work in factories, mines, foundries and stamping mills. According to Hennock (2007, p.83), this marked "the end of the development of factory legislation in Germany for the next thirteen years." Bismarck strongly opposed any further attempts

[32] Mokyr (2000, p. 21) argues that the "primary cause of increased housework, however, was that large segments of the population over the past century have acquired more knowledge about the connection between what they consume and their health. Homemakers spent more time cleaning, nursing, laundering, cooking, and looking after their children because they had become convinced that the health of the members of their household was under their control and part of their responsibility. They had been persuaded that wholesome food, clean clothes and bedsheets, breastfeeding, and a hygienic environment were critical variables in the determination of good health and longevity."

[33] The average benefits per insured were approximately 2.74 Marks in pensions and 0.25 Marks in health care in the old age and disability insurance, whereas they were 6.63 Marks (compensation) and 7.55 Marks (health care) in the health insurance during the period 1891–1903. Since we do not find compensation payments in the health insurance to be significantly associated with mortality (see Section 5.3), we conclude that the old age and disability insurance could have exacerbated the BHI effect by as much as 3%, i.e., the increase in health care spending due to the disability insurance after 1891.

aimed at improving working conditions since he considered new factory regulations to be detrimental to economic development.[34] Hennock (2007) argues that Bismarck's health insurance might even have delayed any major safety and health regulations in factories. Indeed, the 1880s saw only few improvements in workplace regulation. Federal regulatory reforms were minor and restricted to very specific industries.[35] It seems highly unlikely that such improvements can generate the aggregate BHI effects. After Bismarck resigned from office in 1890, regulations were passed to reduce maximum working hours for women. Similar legal restrictions in working hours for men were introduced only in 1919 (e.g. Hennock, 2007, pp.125-128). In 1891, an amendment of the Trade, Commerce and Industry Regulation Act (*Gewerbeordnung*) formally tightened regulations regarding safety at work. In line with earlier considerations regarding the introduction of the old age insurance, the absence of a particular trend break in mortality in 1891 mollifies concerns of confounding safety regulations (see Figure C.8 in the Online Appendix).

The absence of formal improvements in workplace regulation does not exclude the possibility that employers voluntarily improved working conditions due to changing incentives.[36] For example, the benefits of a healthy workforce may increase in the task-specific human capital of incumbent workers. We argue that the ratio of skilled to unskilled workers with little task specific human capital was likely relatively low at the end of the 19th century. Employers thus had little incentives to incur the marginal costs of improving working conditions to gain the marginal benefits of a more healthy but less disposable workforce. Due to the lack of employment protection, employers were legally unrestricted to substitute workers at any time.[37]

We gain additional empirical insights regarding improved working conditions as a potential confounder by exploiting the heterogeneity of our occupation-specific mortality data. Presume that workers in some industries were more successful in improving working conditions and/or employers in some industries improved working conditions voluntarily. As a result of such uncoordinated, local activity, we would expect working conditions to improve in different industries and regions at different points in time. To investigate whether this is the case, we estimate Equation 1 for each of the thirteen blue-collar occupations individually. The results of this exercise are displayed in Figure 6. Three findings are noteworthy: First, the negative mortality effect is not driven by one single (large) in-

[34] An anecdotal account is characteristic of Bismarck's position: As the Pomerian factory inspector R. Hertel admonished Bismarck that there was a risk of explosion at his own paper factory in Varzin, he grumpily countered: "Where is danger ever completely ruled out?" (Lerman, 2004, p.182).

[35] In particular, these improvements consist of a regulation of the use of white phosphorus in the manufacture of matches (1884), a regulation for the manufacture of lead paints and lead acetate (1886) and for the manufacture of hand-rolled cigars (1888).

[36] Similarly, improvements in working conditions may have been a byproduct of technological progress, for example if steam power is replaced by electricity. However, the widespread use of electricity developed much later than BHI. Merely 2.7% of steam engines produced electricity in 1891. Furthermore, by 1900 approximately 80% of electrical energy was used for lighting (see Herzig et al., 1986).

[37] The historical narrative is mostly supportive of this view. Even by 1912, a metal workers' union reported the typical factory air condition to be extremely hot, dusty and toxic due to insufficient ventilation. Yet, employers refused to provide workers with free protective masks and goggles (see, e.g., Deutscher Metallarbeiter-Verband, 1912, p.545). Even if inspectors criticized employers for providing insufficient safety for their workers, employers had no incentives to comply because they could hardly be prosecuted (Bocks, 1978). Next to such complaints, the metal workers' union reports that the number of non-fatal accidents per worker in the workplace increased considerably from 1886 until 1909. Below, we provide evidence from causes of death data confirming that the mortality decline is not driven by a reduction of workplace accidents.

dustry but occurs systematically across many industries.[38] Secondly, the mortality decline occurs at the same point in time across industries, namely shortly after the introduction of BHI.[39] Thirdly, we do not find a substantial post-1884 mortality decline in the mining industry, the only sector that introduced *compulsory* health insurance prior to BHI in 1854 and therefore did not experience a fundamental change in health benefits during the period of observation.[40] We consider these findings to be convincing evidence against a substantial role of working conditions in confounding the blue-collar mortality decline.

Wages and income

If blue-collar workers' wages grew more rapidly than public servants' wages, the BHI effects might be confounded by income increases and related improvements in nutritional status. To be precise, a relative increase of blue-collar wages would prevent the clean identification of the insurance effect only if the reason for the income rise is unrelated to BHI.[41] Unfortunately, there are no administrative data available that systematically report annual wages by occupation *and* district. However, based on an individual-level dataset of household accounts by Fischer (2011), we identify approximately 2,500 blue-collar workers reporting income and consumption during a year in the period 1870–1910. We find no sign of a trend break in mean disposable income or food consumption around 1884 that could explain the sudden decline in mortality rates (see Figure B.1 in the Online Appendix).

Time series wage data for 19th-century Germany, reported by Hoffmann (1965), suggest the largest wage growth between 1884 and 1900 occurred in the construction (43%) and the wood (39%) industries, whereas metals (23%) and textiles (23%) experienced considerably lower wage growth. If our findings were indeed confounded by an income effect, we would expect the mortality decline to be more salient in industries with the largest increases in wages. Again drawing on the heterogeneity across individual industries displayed in Figure 6, we find the mortality decline in low wage-growth industries such as metals (5.2 deaths per 1,000) and textiles (3.4) to be similar or even significantly larger than in high wage-growth industries such as wood (3.6) and construction (1.6) by 1900. Based on these admittedly limited comparisons, we find no indication of income as a confounder of the BHI effect.

Spillovers and selection

As indicated above, we find spillover effects of BHI within the family of the insured. Since families do not live in isolation, there may also be spillovers to untreated individuals outside the family. We selected our control group to be as similar as possible to our

[38] Results do not show a clear pattern and coefficients are imprecisely estimated for occupations that constitute less than 1% of the industrial labor force, i.e., chemicals (0.9%), fossil fuels (0.6%), and printing (0.8%). Changes in mortality in such small populations are a very noisy measure that does not allow a meaningful evaluation of the BHI effect.

[39] When estimating the model in a sample of industries with least concerning pre-treatment coefficients, i.e., metals, textiles, and construction, we find that post-treatment coefficients are larger compared to the full sample of industries.

[40] The fact that miners' mortality stopped increasing after 1884 could be interpreted to show that BHI ameliorated miners' pre-existing mandatory health insurance by providing sufficient benefits to check deteriorating health.

[41] If employers shift their part of insurance contribution to employees by decreasing wages, this would constitute a channel of the BHI effect rather than a confounding factor.

treatment group, e.g., both should live in an urban environment. This comes with the downside that members of the control group could have indirectly benefitted from BHI because they lived in the same improved disease environment. Such spillovers from blue-collar workers to public servants due to reduced risk of disease transmission imply that our estimates constitute a lower bound of the BHI effects on mortality.

Our ITT design identifies reduced form effects of BHI eligibility on mortality. We cannot completely rule out that compliance of blue-collar workers is incomplete or that public servants voluntarily bought additional health insurance.[42] Consequently, there might be uninsured individuals in the treatment and insured individuals in the control group. On the positive side, the ITT design rules out any issues arising from the endogenous selection into insurance take-up.

The ITT design, however, does not rule out that selection into occupations might bias the results. People whose health characteristics are systematically different from incumbent industrial workers may have selected into blue-collar occupations after the insurance was introduced. If an increasing amount of young and healthy people from rural areas migrated to cities to pick up an industrial occupation, the average age structure of blue-collar workers would change. This would result in lower aggregate mortality in the treatment group and produce an upward bias. On the other hand, if necessity drove poor and unhealthy rural agricultural workers into blue-collar occupations, the estimates would be biased downwards. A similar downward bias occurs if new workers from rural areas particularly suffered under the dismal living conditions of urban working-class quarters and fell sick, leading to higher aggregate mortality in the treatment group. Thus, the direction of the bias arising from selection into the industrial sector is a priori unclear. The root cause of the problem is that occupation-specific mortality data do not allow us to fix assignment to treatment to a date before the introduction of BHI.

4 Fixed effects: pre-reform differences at the regional level

In this section, we use a county fixed effects model that compares the mortality trends of counties with varying shares of blue-collar workers in 1882. By holding this treatment intensity variable fixed at a point in time before the introduction of BHI, we exclude selection into treatment eligibility. Furthermore, we capture potential across-ocupation spillovers by estimating the effect of BHI on the full county population.

4.1 Data and econometric specification

The fixed effects approach draws on regionally aggregated mortality rates for the period from 1875 to 1904. Using administrative census data from Galloway (2007), we generate mortality rates from annual death statistics and interpolated quinquennial population censuses for 441 Prussian counties reflecting the borders of 1875.[43] For some specifications, the data will be aggregated to 36 districts. Next to total mortality, the data include

[42] Moreover, the death statistics do not distinguish between dependent workers and the self-employed who would not be captured by the mandatory nature of BHI.

[43] A county is the administrative unit below the district. In its borders of 1867, Prussia consists of 441 counties with an average area of less than 800 square kilometers. If counties were split during administrative reforms after 1867, we aggregate the data to consistently match the initial borders.

gender-specific mortality and infant mortality by legitimacy status.[44] Summary statistics are provided in Tables D.1 and D.3 in the Online Appendix.

Our model can be described by the following Equation 2:

$$Death_{it} = \alpha_i + \theta_t + \sum_{t=1875-1879}^{1900-1904} \beta_t (BlueCollar_{i,1882} * T_t) + X'_{it}\gamma + \varepsilon_{it} \tag{2}$$

where $Death_{it}$ is the death rate of county (district) i averaged over five-year periods $t \in (1875-1879, 1880-1884, 1885-1889, 1890-1894, 1895-1899, 1900-1904)$. α_i are county (district) fixed effects capturing unobserved time-invariant heterogeneity between counties (districts), and θ_t are period fixed effects that flexibly account for general time trends. $BlueCollar_{i,1882}$ is the share of employed blue-collar workers in county (district) i in year 1882 and as such a measure of treatment intensity (Source: Galloway, 2007).[45] For a map of the cross-sectional distribution of blue-collar workers in 1882, see Figure D.1 in the Online Appendix. We interact this variable with period indicators T_t where the omitted reference period is 1880–1884. Thus, the β_t capture any period specific associations between the share of blue-collar workers in 1882 and the outcome variable. By holding the share of blue-collar workers constant at the 1882 level, we avoid any issues due to potentially systematic selection into the industrial sector after the introduction of BHI. In some specifications, the treatment variable will be replaced by the actual share of insured, or by other occupational groups to perform placebo treatment tests. X'_{it} is a vector of time-varying covariates, including the urbanization rate and the share of population with access to sewerage and waterworks discussed in Section 3.1, here generated for the county level. ε_{it} is a mean-zero error component. Standard errors are clustered at the county level to account for serial autocorrelation within counties.

In order for the β_t to identify reduced form ITT effects of BHI, we rely on the assumption that there are no time-varying unobserved determinants of mortality that are correlated with the share of blue-collar workers in 1882. In other words, conditional on X'_{it}, counties with a high or low share of blue-collar workers in 1882 follow the same mortality trend in absence of the treatment. Again, we can inspect whether β_t is zero in the pre-treatment years to corroborate the common trend assumption.

4.2 Main results

In column 1 of Table 1, we document that the established pattern of results can also be found using a basic version of the county fixed effects model described in Equation 2. Similar to the DiD specification, the reduced form effect of Bismarck's health insurance increases over time. The small and insignificant pre-treatment coefficient corroborates the validity of this approach. In column 2, we add the urbanization rate, the share of population with access to waterworks, and the share of population with access to sewerage to control for changes in urbanization patterns and the roll-out of public health

[44] We follow the standard approach to calculate crude death rates as the total number of deaths per year per 1,000 people. Infant mortality is defined as the number of deaths of children below the age of one per year per 1,000 live births. This applies for legitimate and illegitimate deaths and live-births, respectively.

[45] Since this treatment variable aims at capturing the share of insured in the population, it does not include wives and children of blue-collar workers.

infrastructure. The reduced form effects become slightly smaller but stay statistically significant and economically meaningful.[46] In the average county (with a blue-collar share of 7.4% in 1882), BHI reduced aggregate mortality by 0.83 deaths per 1,000, i.e., 3.1% as compared to the mortality level in the baseline period 1880–1884 (26.4). Thus, the BHI effect accounts for 16.5% of the average mortality decline of 5.02 deaths per 1,000 between 1880–84 and 1900–04. This magnitude seems reasonable given that roughly 15% of the total population could benefit directly from BHI at the turn of the century because they were insured (see Table B.1). If we consider that dependent wives and children could indirectly benefit from BHI via intra-family spillovers, roughly 40% of the population may have benefited from BHI directly or indirectly (see Panel B of Table B.2).

In columns 3 to 7 of Table 1, we break down the outcome variable to analyze the effects of BHI on males, females, and infants separately. We find statistically significant negative effects for all three groups. In the average county (with a blue-collar share of 7.4% in 1882), BHI reduced infant mortality by roughly 3.8 deaths per 1,000 births (column 5), or 1.6% of the mortality level in the baseline period 1880–1884. This explains 22% of the infant mortality decline of 17.0 deaths per 1,000 births. Once we distinguish between legitimate infants (column 6) and illegitimate infants (column 7), we find highly significant negative effects for the former but not for the latter.[47] The lack of BHI-induced reductions for illegitimate infants might be explained by the lack of intra-family diffusion of insurance benefits for this group of infants: If a single mother is not a blue-collar worker, the absence of a father leaves the family without access to insurance benefits. Finally, note that in those cases where the pre-treatment interaction is significant, the sign is always negative, implying increasing mortality in counties with a high blue-collar workers' share. Consequently, if we accounted for these deviating trends in the pre-treatment period, the treatment effects would become even larger.[48]

Column 8 reports the results of a placebo test using the 1882 *self-employed* industrial workers share as a placebo treatment variable. Because the 1882 occupation census distinguishes by employment status between employed and self-employed, we can check if self-employed workers, who were not eligible for BHI, create a similar pattern of results as the employed blue-collar workers. As a group, these individuals are arguably highly comparable to the blue-collar workers. We find only small and insignificant coefficients rejecting that this group creates similar mortality effects. In column 9, we execute a similar placebo test by using the 1882 public servants share as a placebo treatment variable. Again we find no significant relationship with changes in mortality. Consequently, we rule out that our treatment indicator picks up mortality trends common to other occupational groups.[49]

[46] Note that a substantial share of overall mortality is infant mortality. Results are qualitatively similar if we deduct deaths before the age of one year. Findings are virtually unaffected when adding baseline control variables measured in 1880–84 interacted with time dummies to allow for differential effects over time (see Table D.7 in the Online Appendix) or when adding baseline dependent variables measured in 1880-84 interacted with time dummies to account for mean reversion (see Table D.8 in the Online Appendix).

[47] From 1880 to 1884, the average death rate of infants across all counties was 229.1; it was 228.2 for legitimate and 368.5 for illegitimate infants. Deaths of illegitimate infants accounted for 11.5% of all deaths of children below age one.

[48] In further unreported regressions, we observe significant negative effects of BHI on the mortality of individuals younger than 15, and of individuals aged between 15 and 44.

[49] Note that running a horse race regression between the share of blue-collar workers and the placebo groups yields similar results (available upon request).

4.3 Robustness checks

Selective migration

Selective rural to urban migration does not pose a threat to our county fixed effects model as long as migration happens *within* county borders. Yet, selective *between* county migration might confound the estimates. In Table 2, we move the level of analysis from the county level to the district level to alleviate this concern. In particular, selective rural to urban migration *between* counties is now innocuous for our estimates as long as it stays *within* district borders. Column 1 shows that a baseline regression using the standard control variables yields point coefficients that are large and fully in line with the county-level findings, although they are less precisely estimated due to a lower number of observations.[50] [51] Moving from the county to the district level allows us to address a range of further concerns with additional data that are only available at this higher level of aggregation.

Changes in the age structure

Changes in the age composition may confound the BHI effect if they are correlated with its share of blue-collar worker in 1882. In this case, the estimates are biased if changes in the age composition do not arise due to the mortality effect of BHI. Rosenbaum and Rubin (1984) suggest to include age composition variables on the right-hand side of the regression instead of using age-adjusted outcome variables.[52] We follow their suggestion and run regressions controlling for changes in the age composition, using newly digitized district-level data from population censuses reported in KSBB (1861–1934) to generate variables that depict the age structure of the population during the period 1875 to 1904 in two ways. We capture compositional changes by including either the mean age of the population or more fine-grained cohort population shares for ten-year age intervals (1–9, 10–19, 20–29, 30–39, 40–49, 50–59, 60–69, 70 plus) in columns 2 and 3. Our findings are virtually unaffected when controlling for the age structure.

Strength of unions

The earlier concern of confounding improvements in working conditions and wages is addressed again in column 4 – this time by controlling for a proxy for the strength of workers' movements and unions. In particular, we add the district-level vote share of the workers' party SPD (and its predecessors) as a control variable.[53] This variable is expected to be highly correlated with spatial and intertemporal heterogeneity in the

[50] For a full replication of Table 1 at the district-level see Table D.9 in the Online Appendix.

[51] Using census data that reports residents by place of birth, we calculate that the population that reported to live in their county of birth declined by 6.7% between 1885 and 1900. If we assume that all of them migrated across district borders, selected from a rural (mortality: 26.62 deaths per 1,000) into an urban municipality (mortality: 27.31 deaths per 1,000), and fully maintained their better health status, the urban-rural mortality differential would decline by 0.12 deaths per 1,000. Thus, even under such extreme assumptions, selective migration would only explain 20% of our mortality effect of 0.83 deaths per 1,000.

[52] Rosenbaum and Rubin (1984) show that adjusting mortality rates for age does not resolve issues related to compositional changes unless all explanatory variables are adjusted in the same way. In the absence of age-adjusted controls, it is preferable that the age composition enters as a control variable.

[53] Using data from Galloway (2007), we calculate the district-level vote share of the workers party SPD (and its predecessors) in the general elections of 1874, 1878, 1884, 1890, 1893 and 1898. These elections were chosen because they precede the first year of each five year interval to which we aggregate.

power of unions.[54] Again, our findings remain virtually unaffected by the inclusion of this variable.

Share of insured individuals

The district level aggregation also allows us to use data on the actual share of individuals registered with health insurance funds (Source: KSA (1884–1942)). Columns 5–8 of Table 2 repeat columns 1–4 but substitute the share of blue-collar workers with the share of insured as the treatment intensity variable.[55] Similar to previous specifications, we hold the share of health insured constant at the earliest possible year in 1885, right after the introduction of BHI, and interact it with time dummies.[56] Comparing columns 1–4 and 5–8, we observe that results from both treatment variables are qualitatively similar, which corroborates our interpretation that the blue-collar worker mortality decline is in fact related to health insurance. As expected, the point coefficients for the health insured (columns 5–8) are consistently larger than the point coefficients for blue-collar workers (columns 1–4) because the share of insured is a more direct measure for the treatment.

5 Understanding the channels

5.1 Evidence from the causes of death

In this section, we use panel data on causes of death to better understand the channels via which BHI reduced mortality rates. In particular, we draw on newly-digitized district-level data from KSBB (1861–1934) that consistently report the full universe of thirty different fatalities annually from 1875 to 1902 to compute cause-specific death rates.[57] Since the causes of death data are not recorded by occupation, we employ the regional fixed effects model as described by Equation 2.

Column 1 of Table 3 shows that we do not find any negative BHI effects on deaths due to accidents. This implies that BHI's overall mortality effect is not driven by a lower number of work-related accidents arising from workers' improved fitness and health. Furthermore, this speaks against the concern that our results are confounded by the accident insurance introduced in 1885.[58]

[54] From 1878 to 1890, the Anti-Socialist Law prohibited assemblies of social democratic groups, in particular of the Socialist Worker Party and related organizations such as unions. Consequently, the strength of unions was curbed until the ban was lifted and organized strikes were suspended. Due to the lack of information on union activity, we resort to using information on the election of social democratic politicians who were still allowed to run as individuals without party affiliation. The vote share of social democratic politicians is probably the single available and best proxy for the strength of worker movements and unions in our period of observation.

[55] Although the source KSA (1884–1942) provides annual district-level records on the number of health insurance contributors for the entire German Empire, we confine our analysis to Prussia since our detailed mortality data are only available for the Prussian territory.

[56] The choice of the first year is admittedly arbitrary to some degree. Since results are qualitatively similar when using other years, we decided to use 1885 to exclude the most pressing concerns of reverse causation and selection.

[57] The data distinguish between deaths from waterborne infectious diseases such as typhus, typhoid fever, or diarrhea, and deaths from airborne infectious diseases such as smallpox, scarlet fever, measles, diphtheria, pertussis, scrofula, tuberculosis, tracheitis, or pneumonia. Besides, the data report the number of deaths due to accidents, maternal deaths and deaths from non-infectious diseases such as cancer, edema, stroke, heart disease, brain disease, and kidney disease. Due to changes in the original reporting of the causes of death after 1902, average mortality is only constructed from three years of data in the last period (1900–1902).

[58] In fact, most post-reform coefficients are positive and significant. Such results are in line with Hennock (2007) who argues that BHI delayed any major safety and health regulations in factories and with Guinnane and Streb (2015) who show that the accident insurance compensated workers for their losses due to accidents but did not result

While BHI had no effects on waterborne disease mortality (column 2), we find considerable negative effects on airborne disease mortality (column 3). We argue that the diffusion of knowledge about disease transmission and avoidance of infection is one of the most important channels through which BHI affected mortality. Preventing airborne disease transmission requires hygiene knowledge, whereas preventing waterborne disease transmission requires hygiene knowledge and hygiene technology. If individuals learn about the risk of contaminated water, they might boil drinking water, yet remain at higher risk of contact with contaminated water until proper infrastructure is installed. Consequently, investment in infrastructure is a necessary condition for the reduction of waterborne diseases. In contrast, knowledge transmission channels are sufficient for reducing the risk of infection with airborne diseases.

Among airborne infectious diseases, tuberculosis was responsible for 12% of overall mortality and 30% of deaths among males in the age group 20-70 in Prussia in 1884. A major breakthrough in fighting TB was achieved in 1882, when Robert Koch identified the bacterium causing tuberculosis.[59] Yet, as the cure was not developed until 1946, the focus was set on preventing infections.[60] Robert Koch (1901, p. 575) himself argued that only preventive action could reduce tuberculosis mortality, including the diffusion of knowledge about its contagiousness to increasingly larger circles.[61] Since TB was widespread among the working age population, the sickness insurance funds were particularly interested in reducing its incidence and the hygienic situation of workers' housing became the center of attention. A characteristic excerpt from Tennstedt (1983, p.458) mentions that *research by Preysing and Schütz found tuberculosis germs underneath 21.2% of 66 toddler's fingernails, which they picked up when crawling on the floors of worker dwellings contaminated by sputum.* Such deficits in the hygienic situation were detected by sickness inspectors (*Krankenkontrolleure/Krankenbesucher*) who became instrumental in educating workers on hygiene. As blue-collar workers' access to hygiene information was fundamentally improved with the introduction of BHI, they gained a head start in TB prevention over other occupational groups that did not have free access to doctors and health inspectors.

Our results suggest that these measures were successful. As can be seen from columns 4 and 5 in Table 3, BHI caused a decline in the number of deaths by lung diseases and especially tuberculosis (and the related scrofula).[62] Compared to the baseline period, BHI decreased TB mortality in the average district with a blue-collar worker share of 8.3% by 27% $((10.502 * 0.083)/3.24)$ in 1900–04. In column 6 of Table 3, we see that BHI also reduced deaths due to non-infectious diseases although coefficients are smaller in magnitude than for infectious airborne diseases. This result suggests that BHI did not exclusively affect communicable disease mortality and leaves room for explanations related to improved access to medication such as antiseptics and the provision of sick pay. However, to the degree that cardiovascular diseases can ultimately be triggered by infectious diseases

in a decline in work-related accidents. Note, however, that, similar to the health insurance, the accident insurance provided sick pay and disability pensions to casualties of work accidents that likely improved living standards.

[59] Tuberculosis was initially assumed to be hereditary since usually the entire family suffered from its symptoms.

[60] Once a vaccine was found in 1921, it had a major impact on infection rates and longevity (see Bütikofer and Salvanes, 2015).

[61] For recovery, physicians sent infected patients to TB sanatoria. The very first sanatorium, the "Genesungshaus Königsberg" near Goslar, was opened by Hannover's province insurance on May 1st, 1895.

[62] As technology to detect the actual cause of death was limited, deaths classified as unspecified lung diseases may in fact have been tuberculosis and vice versa.

such as simple influenza, we cannot exclude a certain overlap. We find some evidence for a reduction in maternal deaths (column 7), which may be related to several channels including doctor visits, medical treatment, and sick pay.

Concerns regarding the quality of the causes of death data from this period have been raised in the literature (Kintner, 1999; Lee et al., 2007). Indeed, identification of the ultimate cause of death is a complex task. Since there were no mandatory inquests, causes of death were certified by laymen rather than doctors in most regions of Prussia. Therefore, it is possible that improved knowledge about diseases also allowed registrars to better identify the accurate cause of death during our period of observation. If changes in the ability to correctly specify the cause are systematically related to a districts' share of insured, our results may be biased and the direction of the bias is ambiguous. We address this issue by inspecting the category 'unknown' cause of death that we expect to decline by more in regions with more blue-collar workers if ability increased by more in these regions. Results in column 8 reveal that there is hardly any evidence for a systematic change of 'unknown' cause of death. Only after the turn of the century, the positive treatment coefficient becomes marginally significant suggesting that, if anything, districts with a high blue-collar workers share became worse in understanding the cause of death. On a cautionary note, such a regression cannot exclude systematic shifts in misspecified categories of known causes of death. Spree (1981) argues that classification-quality concerns are most pressing for infant and child mortality.[63] Combined with the fact that roughly 90% (78,494 out of 88,056) of individuals reported to have died from tuberculosis in 1885 were older than 15 years, we are cautiously optimistic that the findings that are based on the causes of death data are trustworthy.

5.2 Evidence from the supply of health services and health knowledge

We argue that BHI improved health because it provided workers with access to health knowledge and medication via scientifically trained doctors. We further test this hypothesis using the fixed effects setting of Equation 2 and adding a triple interaction of various health service variables measured in 1882, the 1882 blue-collar worker share and time-period dummies. The coefficients on these triple interactions indicate whether the negative effects of BHI on mortality were stronger in regions with better supply of health services.

Column 1 of Table 4 shows that the mortality decreasing effect of BHI is indeed stronger in regions with a higher share of medical professionals. Since the county level data from KSBB (1861–1934) does not distinguish between doctors, midwives, nurses, and other health workers, we also draw on district level data on the distribution of approbated physicians from KSB HB (1903). Column 2 confirms the previous pattern by showing that the negative effect of BHI on mortality is stronger in districts with a higher share of approbated physicians. While we do not observe the level of scientific training of doctors, we can plausibly assume that the diffusion of recent graduates with new scientific knowledge on germ theory declines with geographic distance to a medical school. In column 3, we find BHI effects to be stronger in counties with a lower Euclidean distance

[63] Causes of death data for individuals aged 15 and older, however, seem reliable already in 1877. Spree (1981) draws this conclusion from the fact that mortality of specific causes looks very similar when comparing Prussia and Berlin (where reporting was regulated more heavily).

to the next university with a medical school in the German Empire. Column 4 presents consistent evidence from the district level by showing that the mortality reducing BHI effects are driven by districts with a medical school.

Table D.10 in the Online Appendix, shows that regions with a larger share of blue-collar workers do not have a systematically higher density of approbated physicians (column 1).[64] This suggests that the negative mortality effects are not confounded by changes on the supply side; rather, the BHI effects are just stronger in regions with better health service supply. Column 2 of Table D.10 shows that BHI increased the demand for hospital treatment, approximated by hospital inpatients per capita. Since insurance benefits covered treatment in hospitals, this result does not come as a surprise. This finding supports the historical narrative of Spree (1996) who argues that BHI increased the demand for health services and the *utilized* capacity of hospitals.

5.3 Evidence from insurance expenditures

To further investigate the channels of the mortality effects, this section presents *prima facie* evidence from health insurance funds' benefits. We draw on newly-digitized data from insurance funds balance sheets reported annually for each district in KSA (1884–1942). The original tables list expenditures for doctor visits, medication, hospitalization, sick pay, maternity benefits, death benefits, and administration in German Mark, as well as the aggregate number of sick days. Combining these data with the annual number of insured in a district, we calculate per capita expenditures by type. Figure 7 presents the evolution of sickness funds' expenditures per insured from 1885 to 1904. While we observe a steady increase in expenditures per insured over the full period of observation, the relative importance of the different kinds of expenditures is remarkably stable. Roughly a third – and thus the largest share – of total expenditures is due to sick pay. Expenditures for doctor visits constitute another 20-25% of total expenditures, followed by expenditures for medication, and hospitalization.

We regress district-level mortality rates on time-varying measures of the various types of expenditures per insured. Expenditures are lagged by one year to avoid most pressing concerns of reverse causality.[65] District fixed effects account for time-invariant heterogeneity between districts, while time fixed effects flexibly capture mortality trends common to all districts. Panel A of Table 5 shows results when using overall mortality as the dependent variable, while panel B shows results when using cause specific mortality as the dependent variable.

Column 1 in Panel A supports the view that knowledge diffusion through physicians played a crucial role in reducing mortality. An increase in expenditures for doctor visits per insured by one standard deviation decreases the mortality rate by 0.11 standard deviations. A similar increase in medication expenditures decreases mortality (column 2) by 0.14 standard deviations, suggesting that medical treatment was an important channel. We, however, do not find a significant response to changes in hospitalization expenditures (column 3).

[64] To capture the development of the supply side, we use time varying district-level information on the number of approbated physicians for the years 1879, 1882, 1887, 1898, and 1901 digitized from KSB HB (1903).

[65] The results are robust to changes in the lag structure. For reasons of consistency, control variables are lagged by one year as well.

Interestingly, mortality does not respond to changes in sick pay, the lion's share of sickness funds' expenditures. The estimated point coefficient is small and far from conventional levels of significance (column 4). It seems that sick pay was not instrumental in reducing (short-run) mortality rates through income smoothing. Sick pay might have been just insufficient to avoid temporary malnourishment and to prevent deterioration of the health status. In fact, sickness was the reason for requesting poor relief in 27% of cases as late as 1913. However, we find a negative coefficient on maternity benefits that reaches marginal significance (column 5). Paid maternity leave for three weeks could smooth family income after birth and give the mother the opportunity for breastfeeding, which both can have positive effects in particular on infant health. In column 6, we find a positive significant relationship between death benefits and the mortality rate. This finding reminds us that we cannot rule out reverse causality completely even though we use lagged explanatory variables.[66] The model yields a negative coefficient of expenditures for administration on mortality in column 7. On the one hand, we expect high administration costs per insured to be a sign of inefficient organization resulting in higher mortality. On the other hand, high administration costs might be indicative of an extensive deployment of sickness inspectors monitoring the hygienic situation of the insured, arguably resulting in lower mortality.

An alternative interpretation of the result in column 1 is that doctor visits are indicative of periods of sick leave. If infectious-disease mortality is a function of interactions with co-workers, staying at home reduces the probability of disease transmission. In column 8, we test this hypothesis using data on sick days per insured as the explanatory variable.[67] We do not find evidence for a significant relationship between the average length of sick leaves and mortality, refuting the hypothesis that isolation from the public was an important driver of the mortality reduction.

Panel B presents results from regressions of cause-specific mortality on sick funds' expenditures. To avoid problems of multicollinearity, we group expenditures into 'health care' (doctor visits, medication, hospitalization), 'compensation' (sick pay, maternity benefits, death benefits), and administration. The results confirm that mortality reductions are associated with health care but not compensation, especially in the field of airborne lung diseases such as tuberculosis.[68]

In sum, this exercise provides further evidence that providing people with access to physicians and health care played a crucial role for the mortality decline. New knowledge on hygiene provided by physicians and embodied in treatment and medication was thus more easily diffused to a population living under poor hygienic condition. This resulted in the prevention of infections from airborne diseases. Furthermore, the admittedly parsimonious regressions provide no evidence in support of the view that sick pay contributed to the BHI-induced mortality decline, despite being responsible for roughly a third of sickness funds' expenditures.

[66] An alternative interpretation is that the death of an insured person is indicative of deterioration of the health prospects of the entire family due to income loss and reduced access to health care.

[67] The data is only available from 1889, hence the lower number of observations.

[68] Including maternity benefits, which were not exclusively pecuniary, into the 'care' category reinforces the findings.

6 Conclusions

We analyze the mortality effect of compulsory health insurance introduced by Otto von Bismarck, Chancellor of the German Empire, in December 1884. Using newly digitized Prussian administrative panel data, we exploit the fact that Bismarck's Health Insurance became mandatory for blue-collar workers but not for other occupations. A difference-in-differences specification that uses occupation-specific mortality rates and a regional fixed effects specification that uses differences in the pre-reform share of blue-collar workers across districts/counties yield consistent results of a negative mortality effect. By the turn of the century, BHI had reduced mortality in the average county by 3.1%, which explains 16.5% of the total mortality decline in this period. A large part of the reduction in mortality is driven by a decline in airborne infectious diseases, in particular tuberculosis, a disease which was not curable at that time. Further empirical analyses support our hypothesis that BHI provided households at the lower end of the income distribution with access to physicians and thus with new knowledge on hygiene that they would not have received without BHI. While we do not find any evidence that sick pay was crucial for the effect of BHI on mortality, it might well be that sick pay affected other outcomes such as workers' long-run morbidity or their political support for Bismarck.

More recent expansions of compulsory health insurance for specific subgroups of the population have been highly effective in increasing access to health care and reducing mortality. In this respect, our findings are in line with studies on major expansions in health insurance coverage in the U.S., such as Medicare for the elderly (see Card et al., 2008, 2009; Finkelstein, 2007; Finkelstein and McKnight, 2008) and Medicaid for the poor (see Currie and Gruber, 1996; Goodman-Bacon, 2018). Yet, there are some key differences between these 20th-century expansions and the introduction of BHI. Present-day health insurance schemes work in an environment of chronic diseases and provide health care by medical treatment. In contrast, BHI was established in an environment of infectious diseases for which cures had not sufficiently developed. As such, our setting might be more readily comparable to the situation in developing countries, where an increasing number of compulsory health insurance schemes are currently introduced (Lagomarsino et al., 2012; Malik, 2014). These schemes are introduced in an environment with a high prevalence of infectious diseases with large parts of the population lacking adequate knowledge on hygiene and disease transmission (Dupas, 2011).

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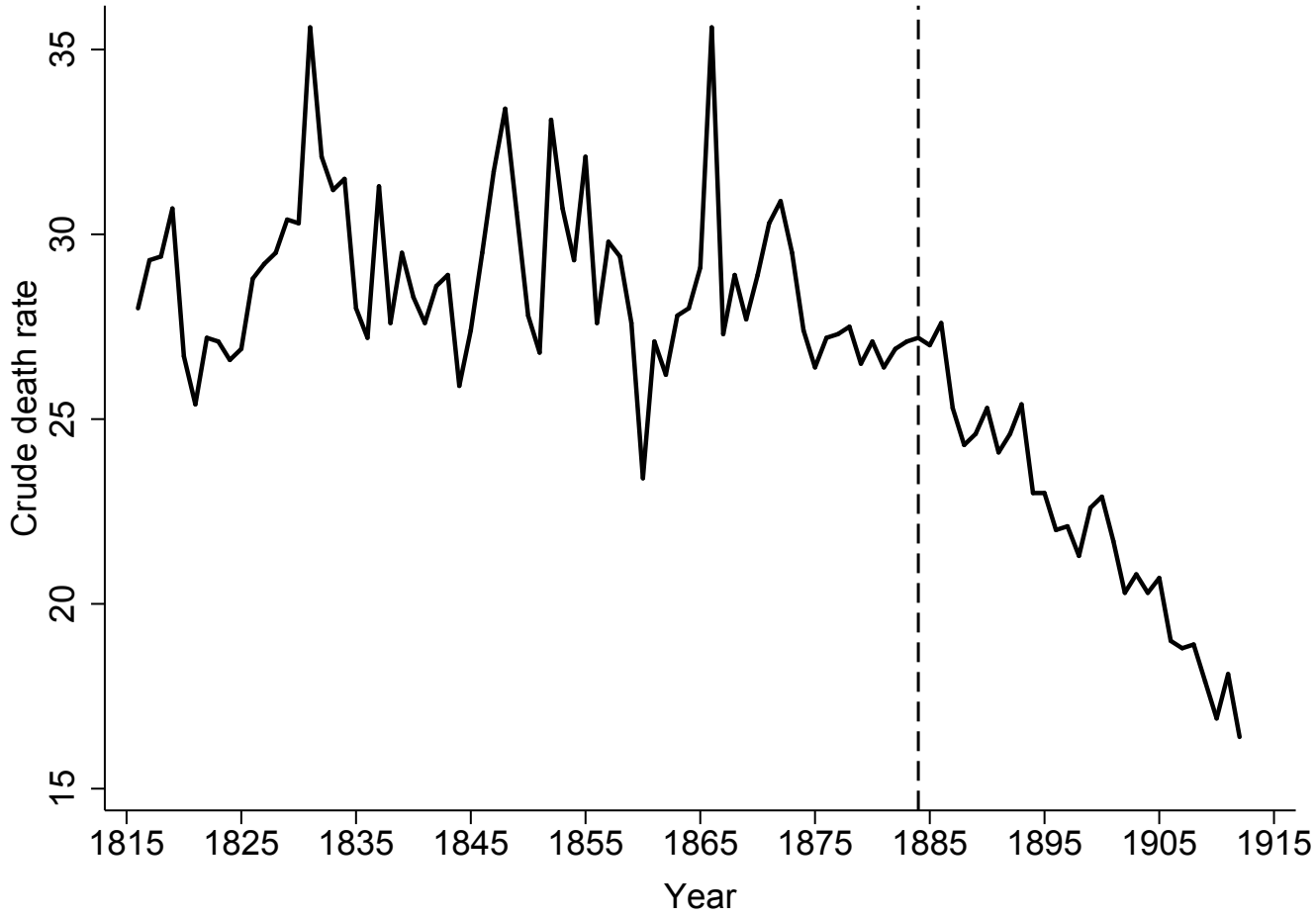

Figure 1: Long-run development of mortality in Prussia. *The figure shows the crude death rate defined as the total number of deaths per year per 1,000 people. The vertical bar indicates the introduction of BHI in 1884. Data source: Hohorst (1978)*

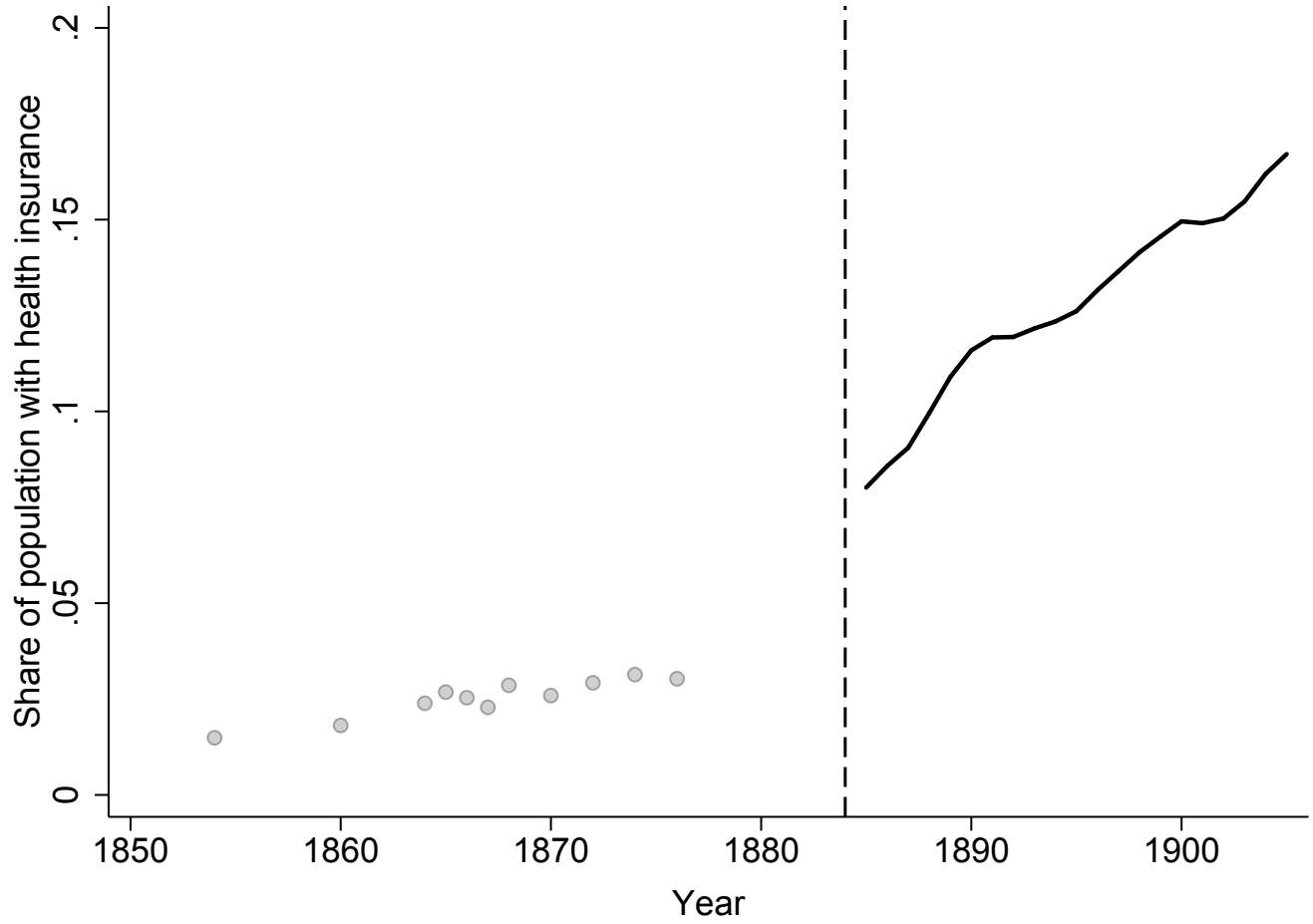

Figure 2: Expansion of Health Insurance in Prussia. *The figure shows the share of health insured in the total population per year. Data refer to Prussia within its respective borders. Insurance benefits vary pre- and post-1884. The vertical bar indicates the introduction of BHI in 1884. Data source: pre 1884 - Handelsministerium (1876); post 1884 - KSA (1884–1942)*

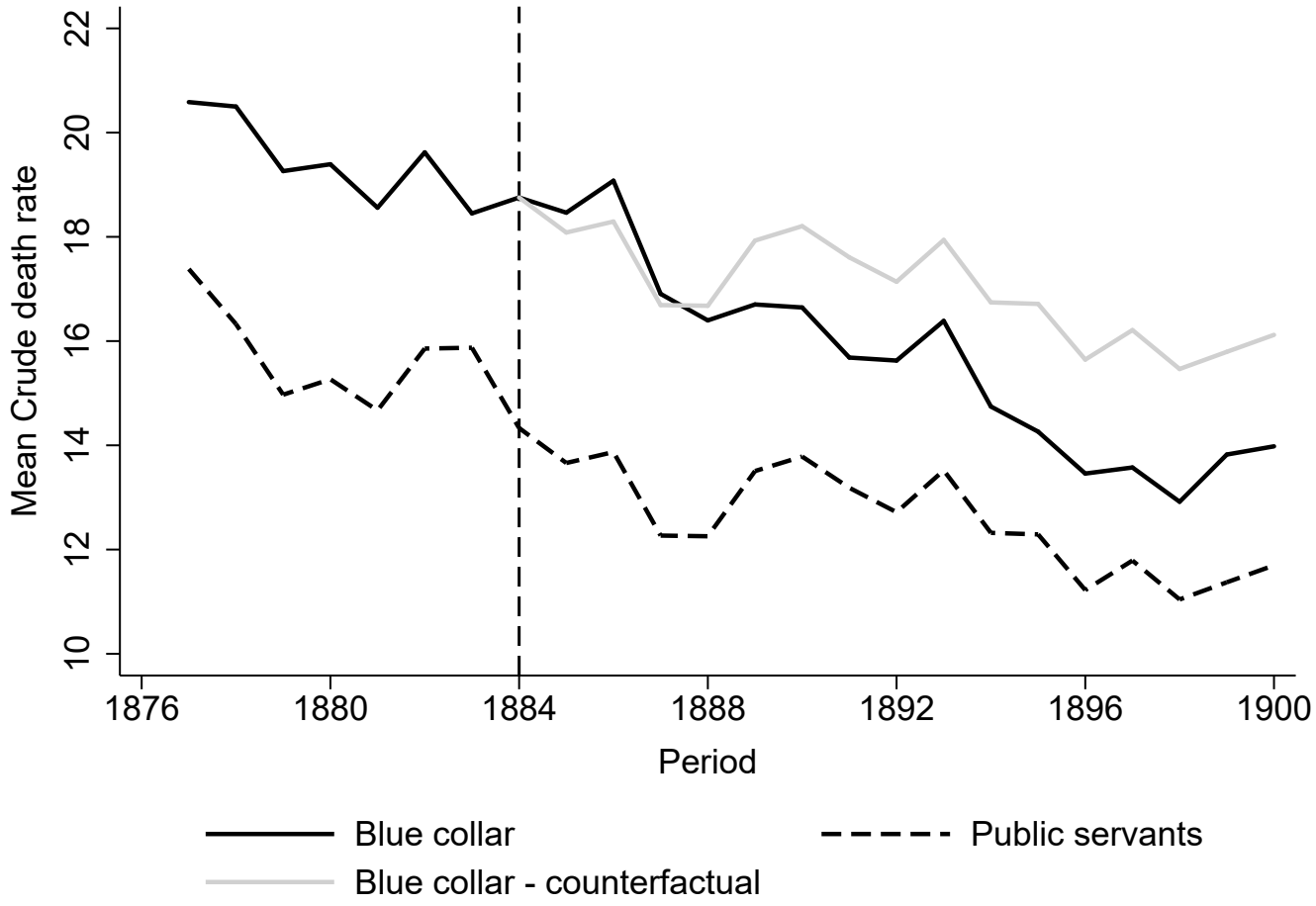

Figure 3: Crude death rates by sector (blue collar and public servants). *The figure shows district-level mean mortality, in deaths per 1,000, across blue-collar occupations and public servants. Following a difference-in-differences logic, the counterfactual is computed by parallel-shifting the trend of the public servants up to the blue-collar workers level in 1884. The vertical bar indicates the introduction of BHI in 1884.*

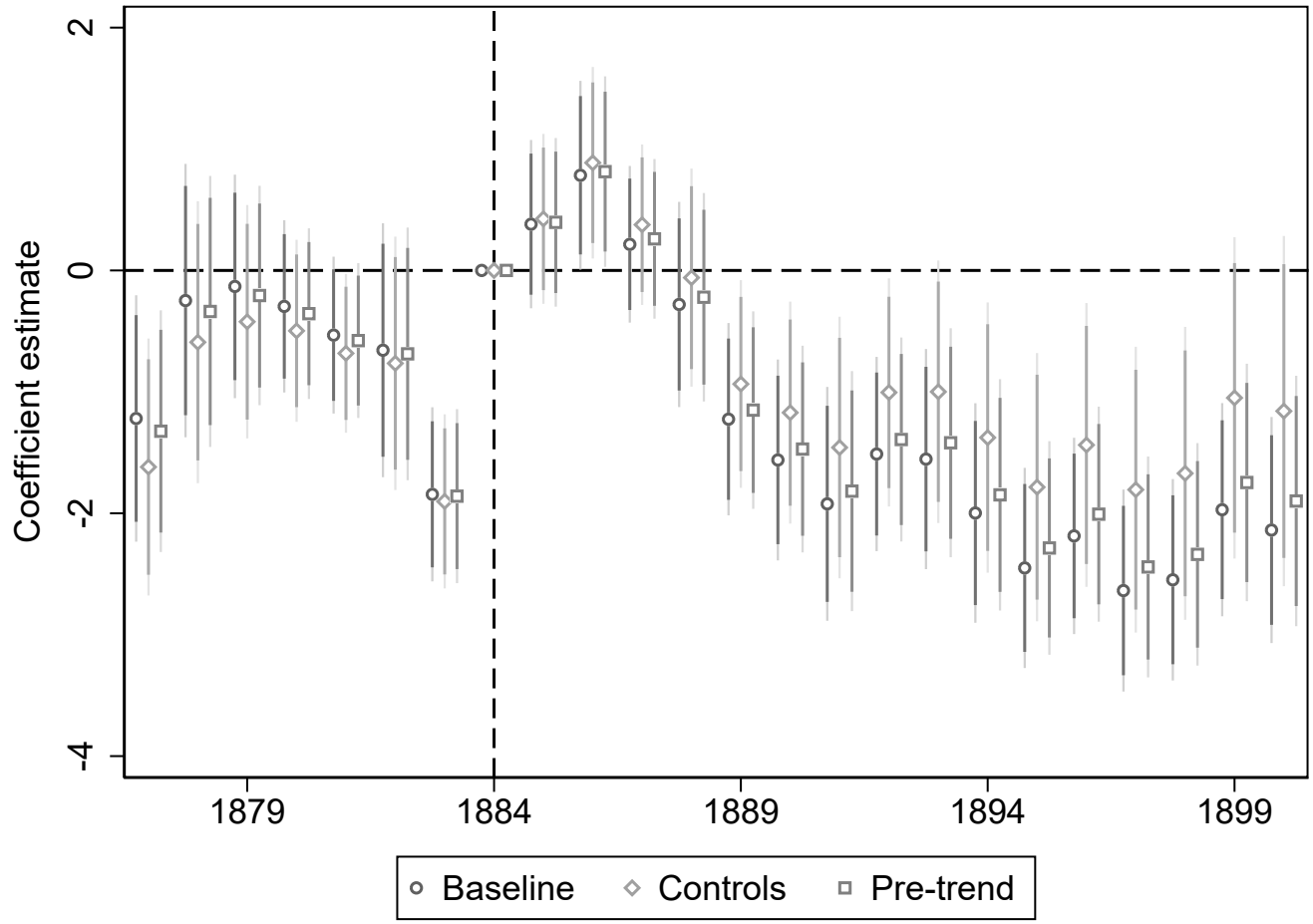

Figure 4: Flexible-DiD estimates. *The figure shows estimated coefficients and confidence intervals (90% and 95%) from a difference-in-differences model similar to Equation 1. The omitted year 1884 is marked by the vertical line and indicates the introduction of BHI.* ∘ *indicate coefficients from a baseline model.* ⋄ *indicate a model that adds control variables, including urbanization, waterworks and sewerage.* □ *correct mortality rates for an occupation* × *district-level pre-trend estimated for the period 1877–1884, extrapolated to 1900.*

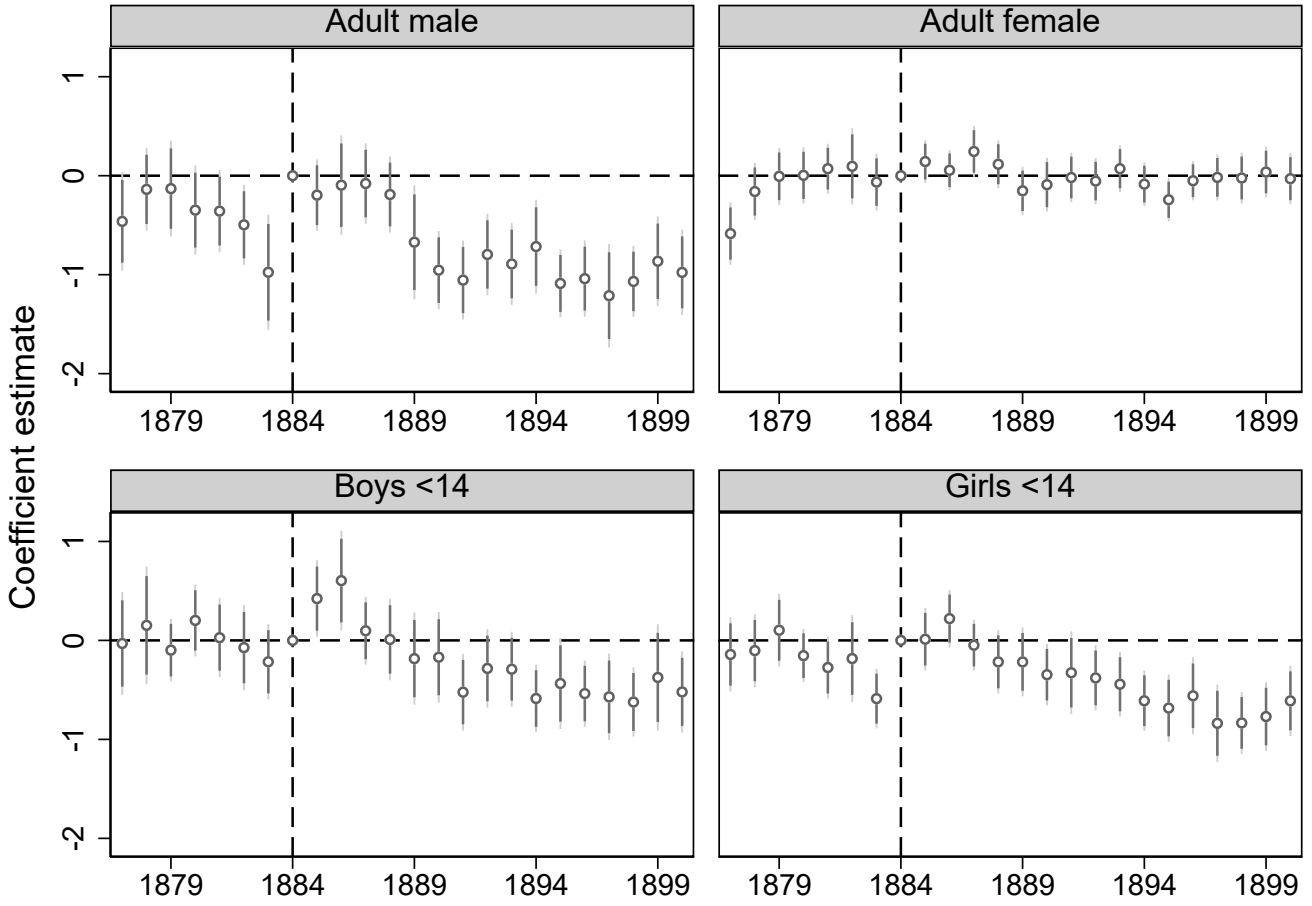

Figure 5: Flexible-DiD estimates. *The figure shows estimated coefficients and confidence intervals (95% and 90%) from a difference-in-differences model similar to Equation 1. The omitted year 1884 is marked by the vertical line and indicates the introduction of BHI. The number of deaths is broken down between gender and adult status whereas the population in the denominator includes all individuals and is the same across specifications. Non-employed adult females and children are classified by the main occupation of their husband, or father respectively. Few adult women and virtually none of the children were employed blue-collar workers and as such eligible for health insurance themselves.*

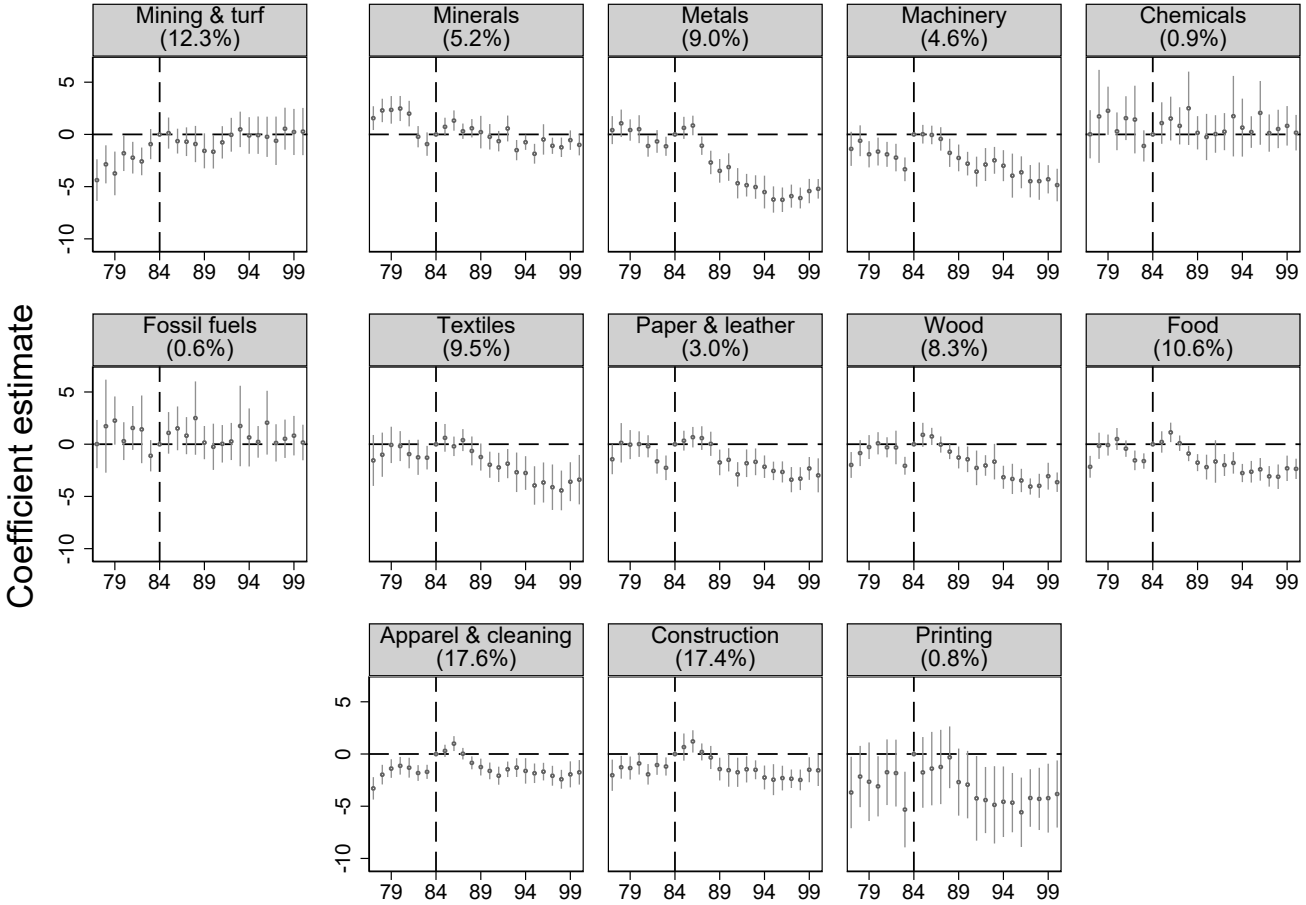

Figure 6: Flexible-DiD estimates by industry. *The figure shows estimated coefficients and 95% confidence intervals from a difference-in-differences model similar to Equation 1 across blue-collar occupations. Brackets indicate the size of each occupation relative to the total blue-collar labor force in 1882. The omitted year 1884 is marked by the vertical lines and indicates the introduction of BHI.*

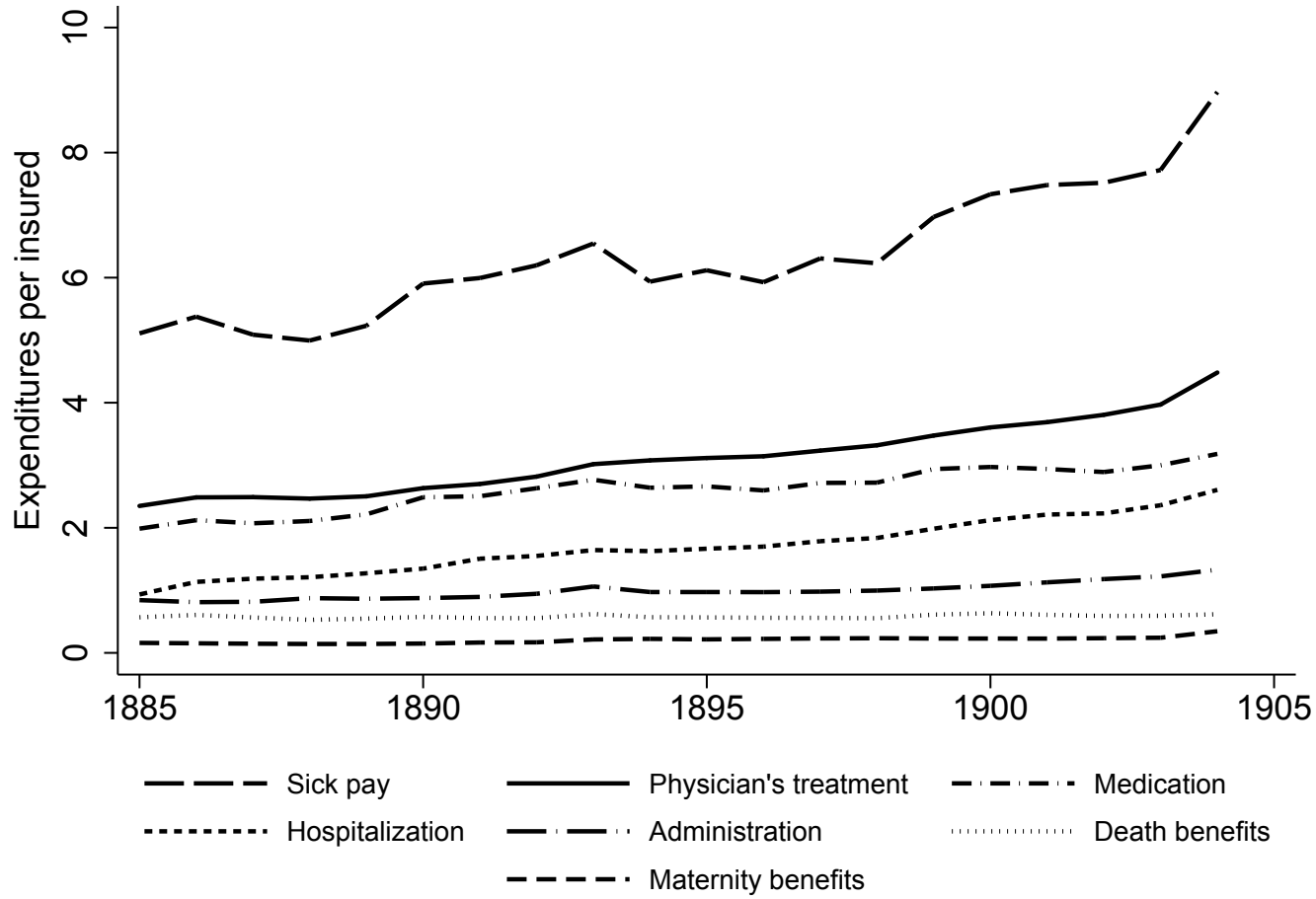

Figure 7: Sickness funds' expenditures per insured in Marks. *The figure shows types of sickness funds' expenditures per insured in Marks over time. Data source: KSA (1884–1942)*

Table 1: County fixed effects model, using the 1882 blue-collar workers' share

Dep. var.: Crude death rate	Baseline Spec. (1)	Control Variables (2)	Male Mortality (3)	Female Mortality (4)	Infant Mortality (5)	Legit. Infants (6)	Illegit. Infants (7)	Self-empl. Placebo (8)	Publ. S. Placebo (9)
Treatment × 1875	1.891	1.365	-6.215***	0.394	-20.608*	-23.233*	-1.949	7.116*	1.542
	(1.623)	(1.642)	(1.694)	(1.789)	(11.694)	(12.226)	(56.694)	(3.785)	(4.000)
Treatment × 1885	-7.768***	-7.308***	-7.133***	-6.553***	1.751	-0.839	30.701	-0.953	2.405
	(1.693)	(1.691)	(1.974)	(1.596)	(11.286)	(11.788)	(45.929)	(4.099)	(3.259)
Treatment × 1890	-4.754**	-3.841*	-6.484***	-6.379***	-24.694	-36.884**	48.046	9.085	0.350
	(2.041)	(2.041)	(2.345)	(2.050)	(15.003)	(15.423)	(53.929)	(5.703)	(4.164)
Treatment × 1895	-8.631***	-7.464***	-5.345**	-8.277***	-40.802**	-62.828***	80.179	1.845	-0.364
	(2.080)	(2.113)	(2.557)	(2.161)	(20.313)	(20.654)	(76.660)	(6.347)	(4.761)
Treatment × 1900	-12.330***	-11.176***	-15.543***	-12.883***	-50.970**	-79.880***	106.128	-0.859	4.750
	(2.399)	(2.431)	(2.845)	(2.545)	(25.452)	(25.507)	(75.658)	(7.337)	(5.573)
Controls	No	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
County FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Time FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Observations	2645	2645	2645	2645	2645	2645	2645	2645	2645
Counties	441	441	441	441	441	441	441	441	441
Periods	6	6	6	6	6	6	6	6	6
R-squared	0.75	0.75	0.74	0.74	0.23	0.26	0.03	0.75	0.75

Notes: The table reports county-level fixed effects estimates. All variables are averaged over five year periods from 1875 to 1904. The omitted period is 1880-84. The dependent variable measures crude death rates using total deaths per alive population in thousands. Dependent variable in columns 1, 2, 8, and 9: total mortality; column 3: male mortality; column 4: female mortality; column 5: infant mortality (< 1 year) per 1,000 births; column 6: infant mortality (< 1 year) born in wedlock per 1,000 births in wedlock; column 7: infant mortality (< 1 year) born out of wedlock per 1,000 births out of wedlock. Treatment variables in columns 1–7: blue-collar workers' population share in 1882, interacted with time dummies; column 8: self-employed industrial workers' population share in 1882, interacted with time dummies; column 9: public servants' population share in 1882, interacted with time dummies. Controls: urbanization rate, waterworks per capita, sewerage per capita. Standard errors, clustered at the county level, in parentheses. * 10%, **5%, *** 1% confidence level.

Table 2: Robustness tests using district-level data

	Initial blue collar workers (1882)				Initial insured (1885)			
Dep. var.: Crude DR	Controls	Average age	Age groups	SPD vote	Controls	Average age	Age groups	SPD vote
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Treatment × 1875	2.34	2.93	-13.41**	1.81	5.17	5.01	-8.85**	4.83
	(5.08)	(5.19)	(5.53)	(4.89)	(4.46)	(4.42)	(4.22)	(4.28)
Treatment × 1885	-13.21**	-14.23**	-12.15***	-13.04**	-15.46**	-15.17**	-12.40***	-15.06**
	(5.48)	(5.68)	(3.87)	(5.45)	(6.14)	(6.12)	(4.39)	(6.15)
Treatment × 1890	-10.58	-12.17	-12.33	-8.90	-12.45	-12.05	-10.22	-11.25
	(8.91)	(9.12)	(8.34)	(8.37)	(10.38)	(10.45)	(8.56)	(10.44)
Treatment × 1895	-15.31*	-16.76**	-14.77*	-13.45*	-17.65**	-17.70*	-12.02*	-16.72*
	(8.14)	(7.87)	(7.35)	(7.73)	(8.16)	(8.75)	(6.64)	(9.03)
Treatment × 1900	-19.36**	-20.97**	-20.56**	-17.27**	-22.01***	-22.26**	-18.44**	-21.15**
	(8.72)	(8.17)	(8.54)	(8.33)	(7.47)	(8.30)	(8.00)	(8.60)
Standard controls	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Average age	No	Yes	No	No	Yes	No	No	No
Age groups	No	No	Yes	No	No	No	Yes	No
SPD vote	No	No	No	Yes	No	No	No	Yes
District FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Time FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Observations	216	216	216	216	216	216	216	216
Districts	36	36	36	36	36	36	36	36
Periods	6	6	6	6	6	6	6	6
R-squared	0.88	0.88	0.92	0.88	0.89	0.89	0.92	0.89

Notes: The table reports district-level fixed effects estimates. All variables are averaged over five year periods from 1875 to 1904. The omitted period is 1880-84. The dependent variable measures crude death rates using total deaths per alive population in thousands. Treatment variable in columns 1–4: blue-collar workers' population share in 1882, interacted with time dummies; in columns 5–8: share of the population covered by health insurance in 1885, interacted with time dummies. Standard controls: urbanization rate, waterworks per capita, sewerage per capita. Columns 2 and 6: add average age of a district's population; columns 3 and 7: add seven time-varying variables capturing the size of age cohorts as a fraction of the total population (cohorts: 1-9, 10-19, 20-29, 30-39, 40-49, 50-59, 60-69, 70 plus); columns 4 and 8: add vote share of the workers' party SPD (and its predecessors) measured at the latest general elections. Standard errors, clustered at the district level, in parentheses. * 10%, **5%, *** 1% confidence level.

Table 3: District fixed effects: causes of death

		Infectious						
Dep. var.: Crude DR	Accident	Waterborne	Airborne	Lung	TB+Scrofula	Non-infectious	Maternal	Unknown
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Treatment × 1875	0.111	3.123*	-5.157**	-3.098**	0.476	-0.564	0.039	1.484
	(0.211)	(1.694)	(2.263)	(1.459)	(0.866)	(0.886)	(0.100)	(1.501)
Treatment × 1885	-0.141	2.799	-10.273***	-2.591	-1.462*	-1.495***	-0.247***	0.325
	(0.208)	(2.095)	(2.973)	(1.626)	(0.760)	(0.533)	(0.088)	(1.753)
Treatment × 1890	0.276	2.511	-7.315	-6.283**	-5.333***	-2.011**	-0.260**	3.170
	(0.178)	(3.516)	(4.559)	(2.740)	(1.254)	(0.771)	(0.124)	(2.554)
Treatment × 1895	0.309	0.977	-7.026*	-9.829***	-8.288***	-2.637**	-0.226*	5.038*
	(0.186)	(4.904)	(3.938)	(2.638)	(1.927)	(1.117)	(0.123)	(2.856)
Treatment × 1900	0.490**	0.672	-10.658***	-13.585***	-10.502***	-4.572***	-0.067	5.000
	(0.238)	(5.034)	(3.150)	(2.383)	(2.615)	(1.445)	(0.159)	(2.975)
Controls	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
District FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Time FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Observations	216	216	216	216	216	216	216	216
Districts	36	36	36	36	36	36	36	36
Periods	6	6	6	6	6	6	6	6
R-squared	0.56	0.25	0.80	0.62	0.84	0.23	0.92	0.79

Notes: The table reports district-level fixed effects estimates. All variables are averaged over five year periods from 1875 to 1904. The omitted period is 1880-84. The dependent variable measures total deaths by cause per alive population in thousands. Treatment variable: blue-collar workers' population share in 1882, interacted with time dummies. *Accident* is death from accidents; *Waterborne* is death from typhus, typhoid fever, and three types of diarrheal diseases; *Airborne* is death from smallpox, scarlet fever, measles, diphtheria, pertussis, scrofula, tuberculosis, tracheitis, pneumonia and other lung diseases; *Lung* is death from pertussis, scrofula, tuberculosis, pneumonia and other lung diseases; *TB+Scrofula* is death from scrofula and tuberculosis; *Non-infectious* is death from cancer, edema, stroke, heart disease, brain disease, and kidney disease; *Maternal* is maternal death related to childbirth; *Unknown* is unknown or unspecified cause of death. Controls: urbanization rate, waterworks per capita, sewerage per capita. Standard errors, clustered at the district level, in parentheses. * 10%, **5%, *** 1% confidence level.

Table 4: County fixed effects: the supply of health

Dep. var.: Crude death rate	Supply of health services		Supply of health knowledge	
	Medical professionals 1882	Approbated doctors 1882	Distance to university	University dummy
	(1)	(2)	(3)	(4)
Treatment × 1875	2.462	-8.394	3.442	-1.078
	(3.131)	(6.272)	(13.209)	(5.451)
Treatment × 1885	-3.411	4.931	-41.395***	-7.719**
	(3.066)	(6.579)	(7.409)	(3.543)
Treatment × 1890	10.855**	32.872***	-84.904***	-0.308
	(4.196)	(9.376)	(10.371)	(6.070)
Treatment × 1895	1.492	21.846***	-61.803***	-5.739
	(4.171)	(7.586)	(10.549)	(4.567)
Treatment × 1900	1.454	18.952*	-85.695***	-8.090
	(4.658)	(9.750)	(14.826)	(5.015)
Treatment × Health supply × 1875	-1.499	15.220	-0.170	14.527
	(1.968)	(16.465)	(1.205)	(9.169)
Treatment × Health supply × 1885	-3.081	-43.081**	3.158***	-22.854**
	(1.872)	(17.805)	(0.698)	(9.131)
Treatment × Health supply × 1890	-12.801***	-138.460***	7.463***	-40.780**
	(2.939)	(27.628)	(0.976)	(16.873)
Treatment × Health supply × 1895	-7.658***	-113.731***	5.049***	-34.555**
	(2.832)	(31.017)	(0.978)	(16.139)
Treatment × Health supply × 1900	-10.838***	-125.202***	6.903***	-41.553**
	(2.982)	(38.516)	(1.376)	(17.864)
Controls	Yes	Yes	Yes	Yes
County/District FE	Yes	Yes	Yes	Yes
Time FE	Yes	Yes	Yes	Yes
Observations	2645	216	2645	216
Counties	441	36	441	36
Periods	6	6	6	6
R-squared	0.76	0.92	0.76	0.91

Notes: The table reports county-level fixed effects estimates. All variables are averaged over five year periods from 1875 to 1904. The omitted period is 1880-84. The dependent variable measures crude death rates using total deaths per alive population in thousands. Treatment variable: blue-collar workers' population share in 1882, interacted with time dummies. The supply of health services is measured as number of medical professionals per 1,000 people in 1882 (column 1) or approbated doctors per 1,000 people in 1882 (column 2). The supply of health knowledge is measured as log(1+Euclidean distance in km) to closest university with a medical school (column 3) or a dummy that is unity if the district had a university with a medical school (column 4). All lower order interaction terms between period dummies, the treatment, and health supply variables are included. Standard errors, clustered at the county level, in parentheses. * 10%, **5%, *** 1% confidence level.

Table 5: Mortality and health expenditures

Panel A - Dependent variable: Crude death rate; Explanatory variable as indicated in column head

	Health care expenditures			Compensation expenditures			Administration expenditures	Days of sick leave
	Doctor visits	Medication	Hospitalization	Sick pay	Maternity ben.	Death ben.		
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
L.Expenditure(std)	-0.109*	-0.141**	-0.040	-0.004	-0.064	0.070*	-0.072	-0.030
	(0.055)	(0.041)	(0.073)	(0.071)	(0.038)	(0.039)	(0.063)	(0.035)
Controls	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
District FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Time FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Observations	684	684	684	684	684	684	684	576
Districts	36	36	36	36	36	36	36	36
Periods	19	19	19	19	19	19	19	16
R-squared	0.81	0.81	0.80	0.80	0.80	0.80	0.80	0.79

Panel B - Dependent variable: Crude death rate by cause of death as indicated in column head; Explanatory variable as indicated

			Infectious					
	All	Accident	Waterborne	Airborne	Lung	TB+Scrofula	Non-infectious	Maternal
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
L.Health care(std)	-0.128*	0.066	-0.218	-0.031	-0.264**	-0.301***	-0.175*	0.106
	(0.071)	(0.106)	(0.149)	(0.113)	(0.110)	(0.102)	(0.101)	(0.066)
L.Compensation(std)	0.012	0.055	-0.029	0.059	0.041	0.032	-0.062	-0.051
	(0.054)	(0.109)	(0.077)	(0.070)	(0.094)	(0.086)	(0.066)	(0.059)
L.Administration(std)	-0.019	0.058	-0.160	-0.084	-0.126	-0.031	0.097	0.050
	(0.050)	(0.093)	(0.145)	(0.101)	(0.101)	(0.104)	(0.079)	(0.077)
Controls	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
District FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Time FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Observations	612	612	612	612	612	612	612	612
Districts	36	36	36	36	36	36	36	36
Periods	17	17	17	17	17	17	17	17
R-squared	0.75	0.17	0.52	0.69	0.60	0.78	0.26	0.70

Notes: The table reports district-level fixed effects estimates. The dependent variable measures crude death rates using total annual deaths per 1,000 (panel A) or annual death rates by cause of death per alive population in thousands (panel B). *Accident* is death from accidents; *Waterborne* is death from typhus, typhoid fever, and three types of diarrheal diseases; *Airborne* is death from smallpox, scarlet fever, measles, diphtheria, pertussis, scrofula, tuberculosis, tracheitis, pneumonia and other lung diseases; *Lung* is death from pertussis, scrofula, tuberculosis, pneumonia and other lung diseases; *TB+Scrofula* is death from scrofula and tuberculosis; *Non-infectious* is death from cancer, edema, stroke, heart disease, brain disease, and kidney disease; *Maternal* is maternal death related to childbirth. Explanatory variables are sickness funds' expenditures per insured or duration of sick leave per insured, lagged by one year. All variables are normalized with mean zero and a standard deviation of one. Controls: urbanization rate, waterworks per capita, sewerage per capita, each lagged by one year. Standard errors, clustered at the district level, in parentheses. * 10%, **5%, *** 1% confidence level.

Appendix (for online publication only)

Appendix A Time-series and cross-country statistics

At the end of the 19th century, mortality rates declined across all industrializing countries. Figure A.1 shows the crude death rates for Prussia and various European countries against years from 1875 to 1913.[A.1] In line with Figure 1, we find that Prussia had experienced high levels of mortality in the early 19th century. This 'German penalty' was still evident in the 1870s but vanished by 1900 when Germany's mortality had approached the levels of other countries. However, it is not before the mid-1880s that we observe a distinct break in the Prussian mortality trend. From 1885 to 1913, the crude death rate in Prussia declined from about 27 to about 17 deaths per 1,000 people, corresponding to a substantial drop of almost 40%.

Accordingly, the decline of Prussian mortality rates from 1884 onwards seems to be noticeably more accentuated than in other Western countries, which might be related to the introduction of BHI. To highlight the comparatively steeper mortality decline in Prussia, we plot the difference in the mortality rate of Prussia and every other country by year, while normalizing the respective mortality difference in 1884 to zero. Figure A.2 shows that the mortality decline for Prussia was indeed considerably stronger than the mortality decline in all other countries during this period. The comparison also suggests a *less* pronounced mortality decline prior to 1884 in Prussia than in most other countries. Thus, using the introduction of BHI in 1884 as a reference point, we find the Prussian mortality decline to be largest in comparison with other Western and Northern European countries.

Although remarkable, these findings from simple time-series and cross-country statistics should not be interpreted as evidence for a causal effect of BHI on mortality. If Prussia experienced structural changes that other countries did not experience simultaneously and if these changes affect mortality and happen to coincide with the introduction of BHI, this might explain Prussia's comparatively strong mortality decline at the end of the 19th century.

[A.1] The data come from a range of national sources that are collected and made available by the team of the Human Mortality Database, a joint project of the University of California, Berkeley (USA) and the Max Planck Institute for Demographic Research (Germany). For details, please visit http://www.mortality.org or www.humanmortality.de

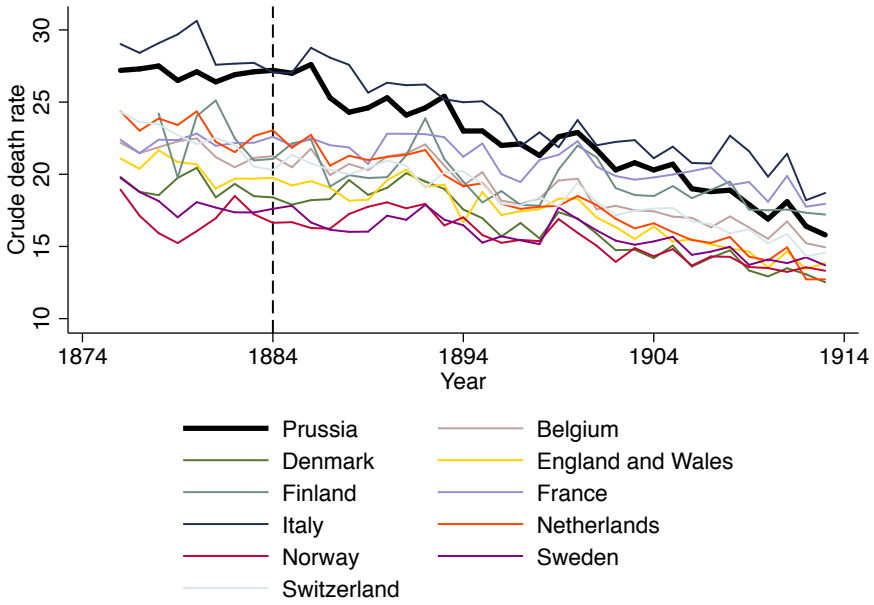

Figure A.1: Mortality decline across Western European countries. *The figure shows the number of deaths per year per 1,000 people for several countries. The vertical bar indicates the introduction of BHI in 1884. Data source: Human Mortality Database (http://www.mortality.org)*

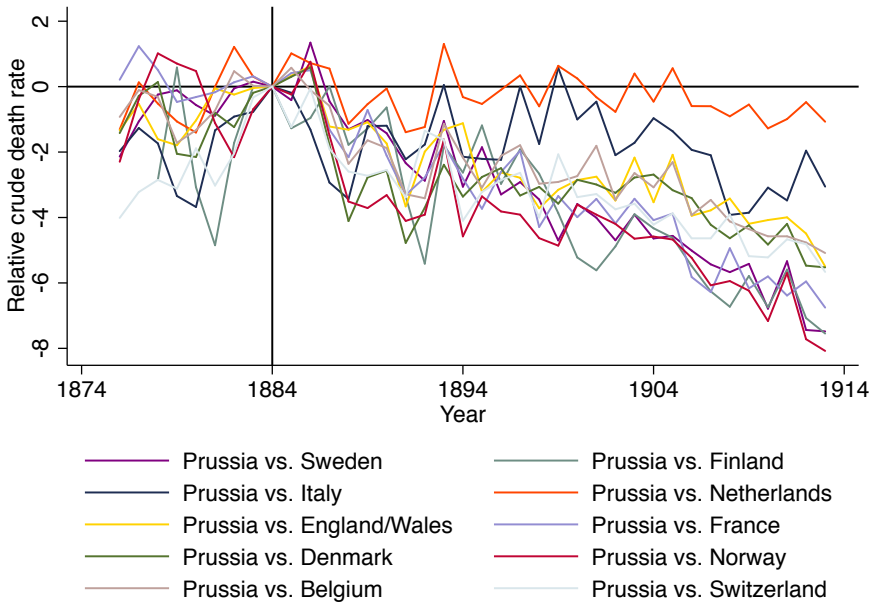

Figure A.2: International mortality decline relative to Prussia. *The figure shows the number of deaths per 1,000 people in Prussia minus the number of deaths per 1,000 people in the respective other country over time. The variable is normalized to zero in 1884. The vertical bar indicates the introduction of BHI in 1884. Data source: Human Mortality Database (http://www.mortality.org)*

Appendix B Descriptive Statistics

Table B.1: Expansion of health insurance

Year	Population	Health Insured	% Insured Population
1854	17,077	254	1.5%
1860	18,136	329	1.8%
1864	19,149	458	2.4%
1868	24,069	688	2.9%
1870	24,403	632	2.6%
1872	24,801	725	2.9%
1874	25,352	795	3.1%
1876	25,922	785	3.0%
1885	28,232	2,263	8.0%
1890	29,819	3,457	11.6%
1895	31,697	3,998	12.6%
1900	34,254	5,123	15.0%
1905	37,058	6,192	16.7%

Notes: The table shows descriptive statistics on the expansion of health insurance in Prussia within its respective borders. Population and the number of insured are expressed in thousands. Insurance benefits vary pre- and post-1884.

Table B.2: Population by sectors

	1882	1895	1907
Panel A:		Labor force in total population	
Total population	27,279	31,855	37,293
Industrial sector labor force	3,651	4,756	6,688
	13.4%	14.9%	17.9%
Public sector labor force	587	823	1,027
	2.2%	2.6%	2.8%
Panel B:		Sectoral population in total population	
Total population	27,279	31,855	37,293
Industrial sector population	9,394	12,196	16,244
	34.4%	38.3%	43.6%
Public sector population	1,306	1,672	2,043
	4.8%	5.2%	5.5%
Panel C:		Labor force	
Total labor force	11,704	13,242	18,038
Industrial sector labor force	3,651	4,756	6,688
	31.2%	35.9%	37.1%
Public sector labor force	587	823	1,027
	5.0%	6.2%	5.7%
Panel D:		Female labor force	
Total female labor force	3,349	3,412	5,763
Industrial sector female labor force	585	761	1,078
	17.5%	22.3%	18.7%
Public sector female labor force	61	97	166
	1.8%	2.8%	2.9%

Notes: The table reports descriptive statistics based on the occupation censuses conducted in the German Empire in 1882, 1895, and 1907. Population is reported in thousands. Counts of the total *sectoral population* in Panel B include children and non-employed family members, assigned according to the occupation of the father or husband, respectively. The industrial sector refers to the official sector B (Industry); the public sector refers to the official sector E (Public).

Figure B.1: Worker's income and food consumption *The figure shows the average household income (all sources), male labor income and food consumption (excluding luxury items) of blue-collar workers within five year periods from 1870–1910. Marker size indicates number of observations. The data is based on individual-level data from household accounts as reported by Fischer (2011).*

Appendix C Descriptive statistics and robustness tests for the DiD specification

Table C.1: Variable definitions for DiD estimates in Figures 4 and 5

1. Crude death rate:	Total number of individuals in an occupational group (non-employed women and children categorized by occupation of husband and father, respectively), who died (excl. stillbirths) in a district in a year, per 1,000 individuals (men, women, and children) in that occupational group (denominator interpolated between occupation censuses of 1882, 1895, and 1907).
2. Adult male death rate:	Total number of adult men in an occupational group, who died in a district in a year, per 1,000 individuals (men, women, and children) in that occupation (denominator interpolated between occupation censuses of 1882, 1895, and 1907).
3. Adult female death rate:	Total number of adult women in an occupational group (non-employed women categorized by occupation of husband), who died in a district in a year, per 1,000 individuals (men, women, and children) in that occupational group (denominator interpolated between occupation censuses of 1882, 1895, and 1907).
4. Boys < 14 *death rate*:	Total number of boys younger than 14 years in an occupational group (categorized by occupation of father), who died in a district in a year, per 1,000 individuals (men, women, and children) in that occupational group (denominator interpolated between occupation censuses of 1882, 1895, and 1907).
5. Girls < 14 *death rate*:	Total number of girls younger than 14 years in an occupational group (categorized by occupation of father), who died in a district in a year, per 1,000 individuals (men, women, and children) in that occupational group (denominator interpolated between occupation censuses of 1882, 1895, and 1907).
6. Urbanization (share):	Total population in cities (with legal city rights) in a district in a year, per total population (denominator and enumerator interpolated between quinquennial population censuses).
7. Waterworks (p.c.):	Total population in cities with waterworks in a district in a year, per total population (denominator and enumerator interpolated between quinquennial population censuses).
8. Sewerage (p.c.):	Total population in cities with sewerage in a district in a year, per total population (denominator and enumerator interpolated between quinquennial population censuses).
9. Average age:	Mean age of the total population in a district in a year (denominator and enumerator interpolated between quinquennial population censuses).
10. SPD (share):	Share of votes for the SPD and its predecessor parties in the general elections in a district in a year (interpolated between elections of 1874, 1878, 1881, 1884, 1887, 1890, 1893, 1898, and 1903)
11. Wheat yields:	Average yield of summer wheat in kilograms per hectare in a district in a year (available for 1878–1900, extrapolated to 1877, missing for the district of Berlin)
12. Protestant (share):	Total number of Protestants in a district in a year, per total population (denominator and enumerator interpolated between quinquennial population censuses).

Sources: Denominators in variables 1-5 and variables 9 and 11 digitized from various volumes of KSBB (1861–1934), enumerators in variables 1-5 and variables 6, 10, and 12 from Galloway (2007), information in variable 7 and 8 derived from Grahn (1898-1902) and Salomon (1906-1907).

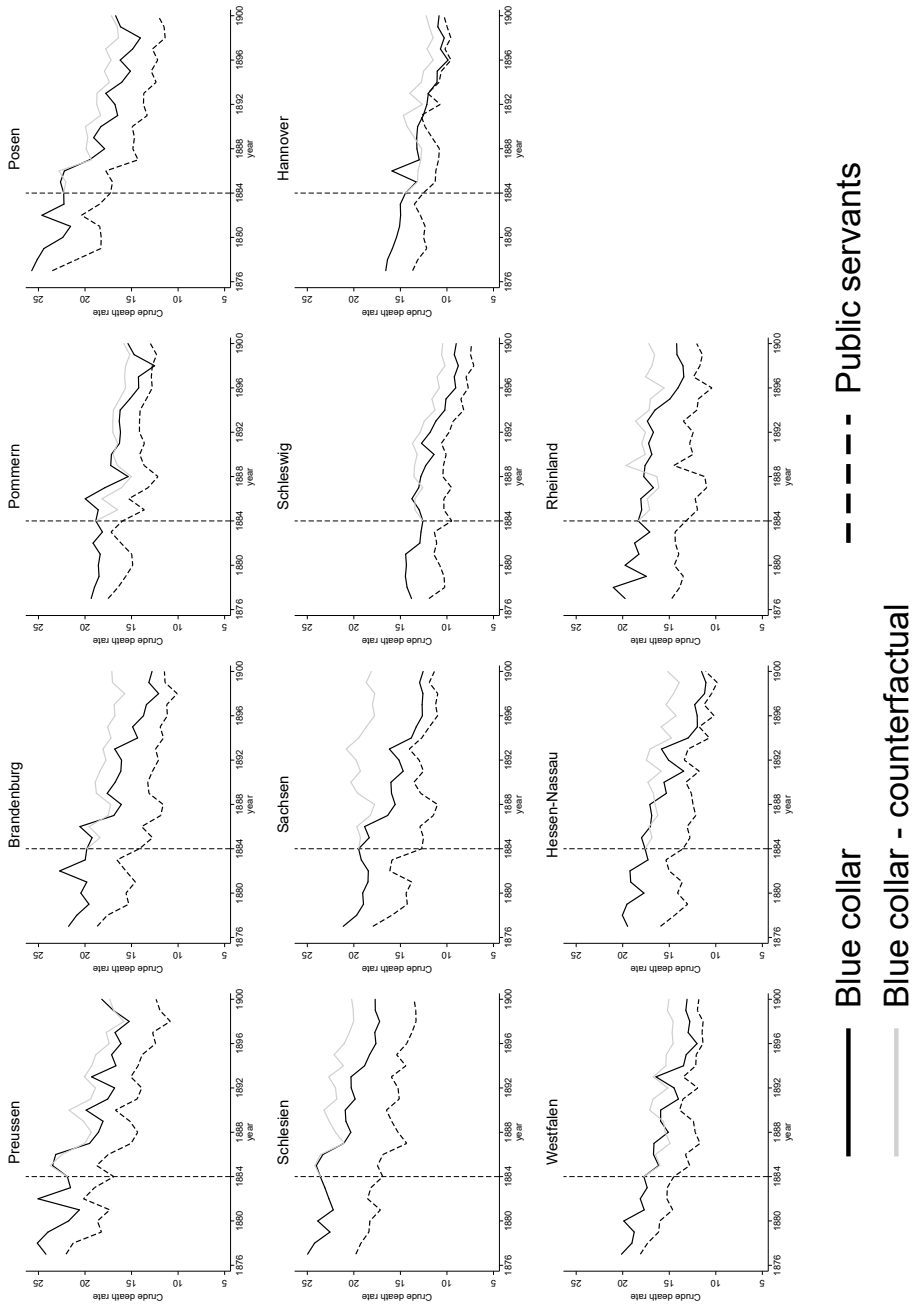

Figure C.1: Crude death rates by sector (blue-collar groups and public servants) and province. *The figure shows district-level mean mortality, in deaths per 1,000, across blue-collar occupations and public servants for each Prussian province. Following a difference-in-differences logic, the counterfactual is computed by parallel-shifting the trend of the public servants up to the blue-collar workers level in 1884. The vertical bars indicate the introduction of BHI in 1884.*

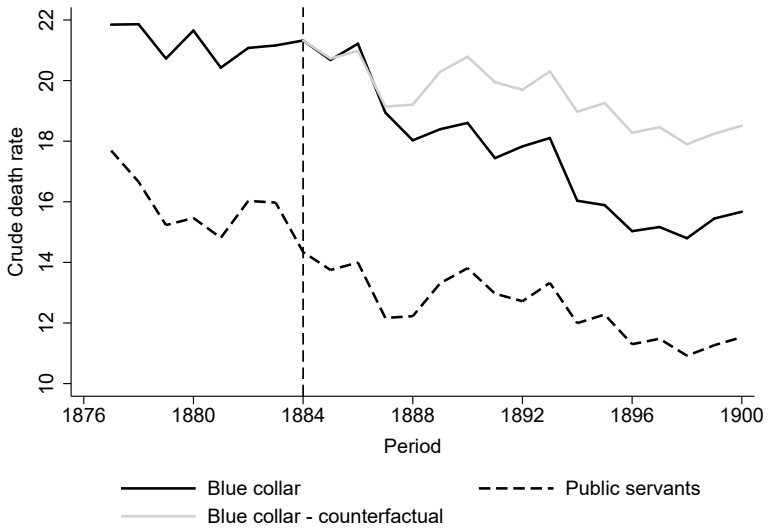

Figure C.2: Crude death rates by sector (blue-collar groups and public servants). *The figure shows the number of deaths per 1,000 people by sectoral groups, after collapsing blue-collar mortality and public servants mortality into two separate time series. Following a difference-in-differences logic, the counterfactual is computed by parallel-shifting the trend of the public servants up to the blue-collar workers level in 1884. The vertical bars indicate the introduction of BHI in 1884.*

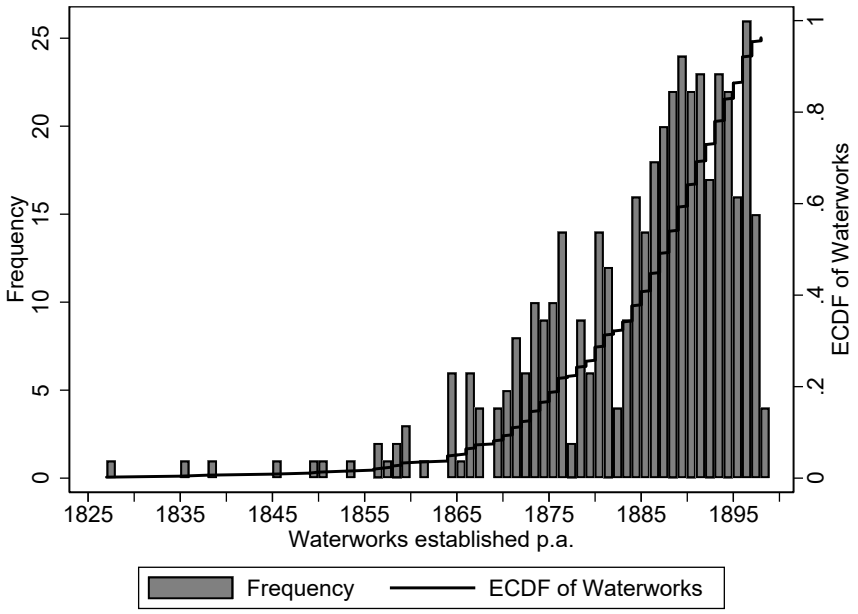

Figure C.3: The roll-out of waterworks in Prussia. *The figure shows the roll-out of waterworks in Prussian cities over time. The bars indicate the annual number of new waterworks put into service. The line indicates the cumulative distribution function. Data source: Grahn (1898-1902)*

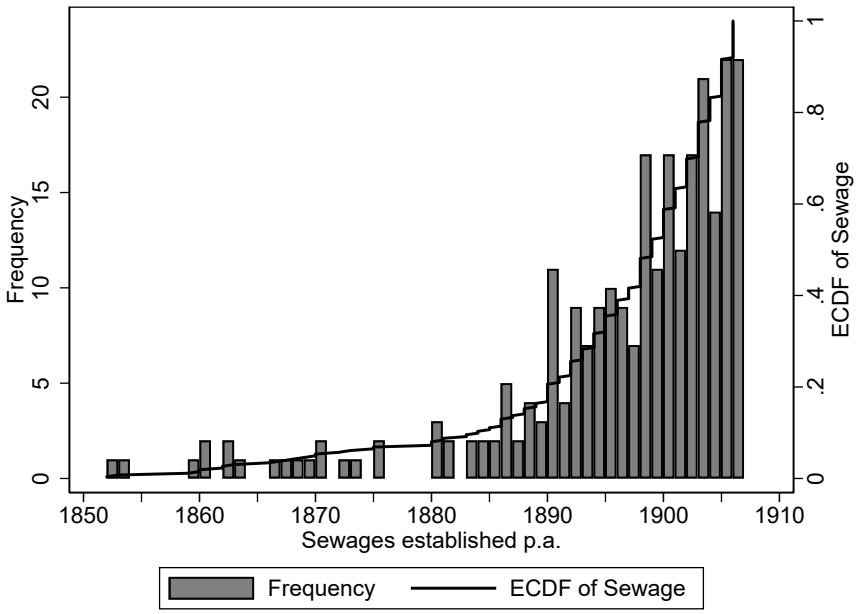

Figure C.4: The roll-out of sewerage in Prussia. *The figure shows the roll-out of sewerage systems in Prussian cities over time. The bars indicate the annual number of new sewerage put into service. The line indicates the cumulative distribution function. Data source: Salomon (1906-1907)*

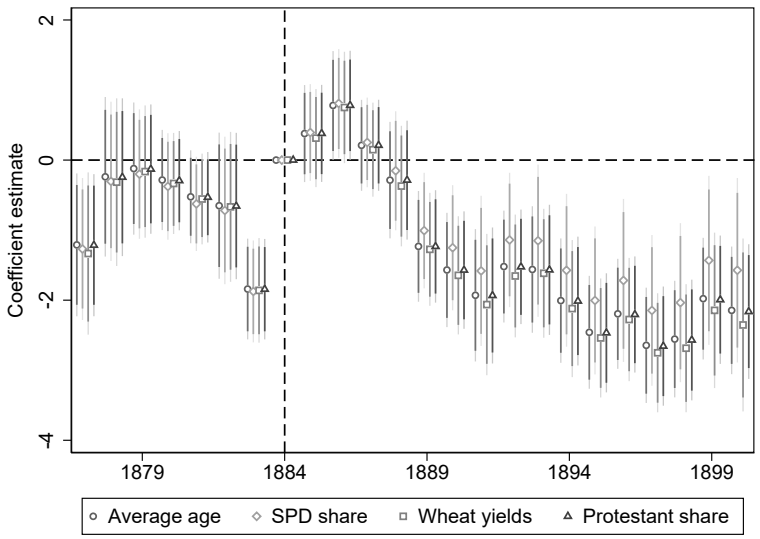

Figure C.5: Flexible-DiD estimates with additional covariates. *The figure shows estimated coefficients and confidence intervals (95% and 90%) from a DiD model similar to Equation 1. The omitted year 1884 is marked by the vertical line and indicates the introduction of BHI. We plot the coefficients from a baseline model that controls for the average age of the population (○), the SPD vote share (◇), wheat yields (□), or the Protestant share (△).* See Table C.1 for variable definitions.

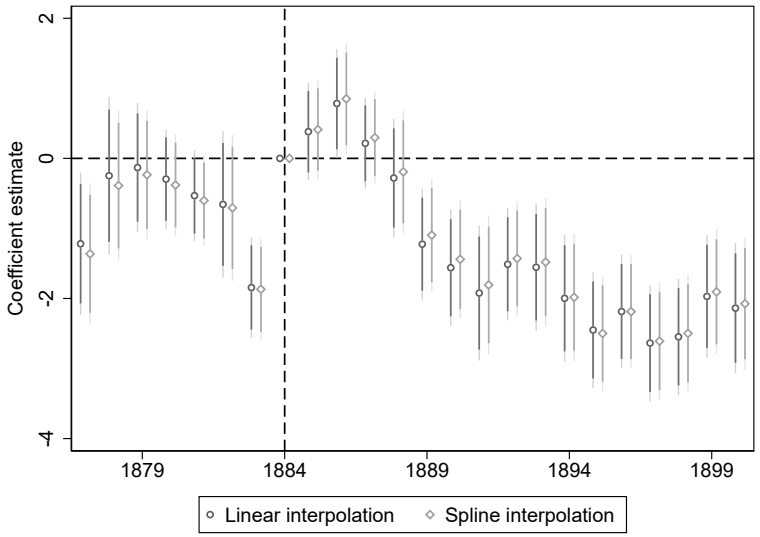

Figure C.6: Flexible-DiD estimates with spline interpolation. *The figure shows estimated coefficients and confidence intervals (95% and 90%) from a DiD model similar to Equation 1 where the denominator of the dependent variable is interpolated using linear or spline interpolation.*

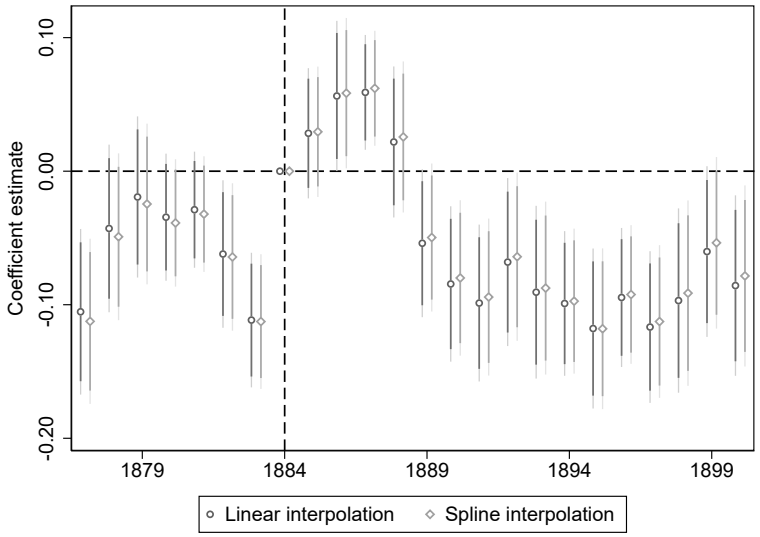

Figure C.7: Flexible-DiD estimates in log-specification. *The figure shows estimated coefficients and confidence intervals (95% and 90%) from a DiD model similar to Equation 1 where the dependent variable is log total death and log population enters on the right hand side of the equation using either linear or spline interpolation.*

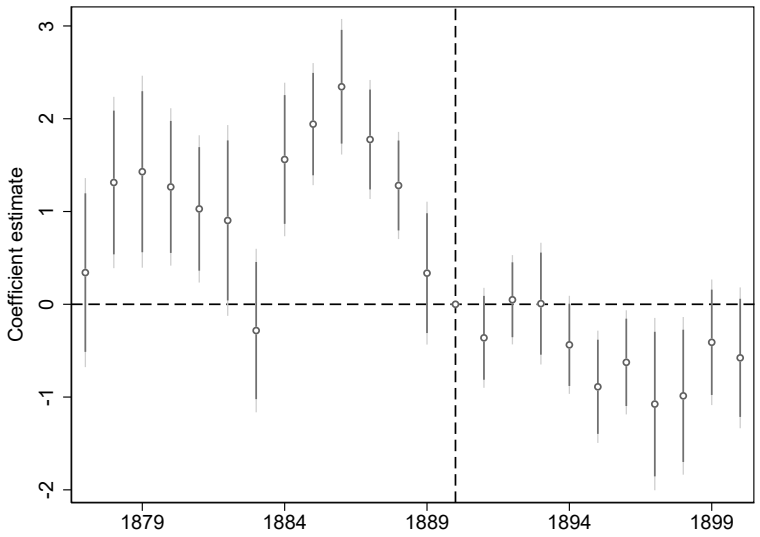

Figure C.8: Flexible-DiD estimates using 1890 as reference. *The figure shows estimated coefficients and confidence intervals (95% and 90%) from a DiD model similar to Equation 1. The omitted year 1890 is marked by the vertical bar and indicates the introduction of the old-age pension system.*

Table C.2: Flexible-DiD estimates after collapsing mortality

Dep. var.: Deaths per occ. Pop.	Baseline	Urbanization	Waterworks	Sewerage
	(1)	(2)	(3)	(4)
Blue Collar x 1877	-0.1617	-0.3155	-0.3789*	-0.2407
	(0.2272)	(0.2411)	(0.2259)	(0.2283)
Blue Collar x 1885	1.0627***	1.2015***	1.2670***	1.1444***
	(0.1604)	(0.1499)	(0.1681)	(0.1578)
Blue Collar x 1889	-0.9193***	-0.6619**	-0.3785	-0.7339**
	(0.2761)	(0.2561)	(0.3300)	(0.2803)
Blue Collar x 1893	-1.6119***	-1.2189***	-0.8212**	-1.3139***
	(0.2747)	(0.2856)	(0.3975)	(0.3038)
Blue Collar x 1897	-1.9076***	-1.3317***	-0.8834**	-1.4946***
	(0.2869)	(0.4149)	(0.4248)	(0.3646)
Urbanization × Sector	No	Yes	No	No
Waterworks × Sector	No	No	Yes	No
Sewerage × Sector	No	No	No	Yes
District × Sector FE	Yes	Yes	Yes	Yes
District × Time FE	Yes	Yes	Yes	Yes
Observations	432	432	432	432
R-squared	0.94	0.94	0.94	0.94

Notes: The table reports estimates from a DiD model similar to Equation 1. Mortality is collapsed by sector and into four year periods from 1877 to 1900. The dependent variable measures crude deaths rates by sector of the household head per alive occupational population (including dependents) in thousands. The omitted period is 1881-84. Controls: urbanization rate (column 2), waterworks per capita (column 3), sewerage per capita (column 4). Standard errors, clustered at the sector-by-district level, in parentheses. * 10%, **5%, *** 1% confidence level.

Appendix D Descriptive statistics and robustness tests for the FE specification

Table D.1: Summary statistics for county-level estimates in Tables 1 and 4

	Mean	SD	Min	Max	Observations
Crude death rate	24.391	3.921	15.093	40.319	2645
CDR (male)	26.350	4.553	15.835	44.646	2645
CDR (female)	23.237	3.471	13.871	36.906	2645
CDR (infants)	222.488	51.409	115.469	478.142	2645
CDR (legitimate infants)	220.656	50.712	119.877	487.012	2645
CDR (illegitimate infants)	367.704	89.490	121.212	771.739	2645
Blue collar workers in 1882 (p.c.)	0.074	0.046	0.011	0.258	2645
Self-employed industrial in 1882 (p.c.)	0.042	0.018	0.011	0.157	2645
Public servants in 1882 (p.c.)	0.019	0.019	0.005	0.162	2645
Urbanization (share)	0.294	0.214	0.000	1.000	2645
Waterworks (p.c.)	0.091	0.216	0.000	1.000	2645
Sewerage (p.c.)	0.043	0.171	0.000	1.000	2645
Medical professionals 1882 (p.m.)	1.346	1.033	0.230	10.511	2645
(ln) Distance to university	10.579	1.859	0.000	12.324	2645

Notes: The table reports summary statistics of variables used to estimate the models in Tables 1 and 4.

Table D.2: Variable definitions for county-level estimates in Tables 1 and 4

1. Crude death rate:	Five year average of deaths (incl. stillbirths) in a county, per 1,000 people (denominator interpolated between quinquennial population censuses).
2. CDR (male) :	Five year average of male deaths (incl. stillbirths) in a county, per 1,000 men (denominator interpolated between quinquennial population censuses).
3. CDR (female):	Five year average of female deaths (incl. stillbirths) in a county, per 1,000 women (denominator interpolated between quinquennial population censuses).
4. CDR (infants):	Five year average of deaths of children less than 1 year old in a county, per 1,000 births (both incl. stillbirths).
5. CDR (legitimate infants):	Five year average of deaths of legitimate children less than 1 year old in a county, per 1,000 legitimate births (both incl. stillbirths).
6. CDR (illegitimate infants):	Five year average of deaths of illegitimate children less than 1 year old in a county, per 1,000 illegitimate births (both incl. stillbirths).
7. Blue collar workers in 1882 (p.c.):	Total number of employed blue-collar workers (sector B) in 1882 in a county, per total population in 1880.
8. Self-employed industrial in 1882 (p.c.):	Total number of self-employed industrial workers (sector B) in 1882 in a county, per total population in 1880.
9. Public servants in 1882 (p.c.):	Total number of public servants (sector E) in 1882 in a county, per total population in 1880.
10. Urbanization (share):	Five year average of population in cities (with legal city rights) in a county, per per total population (denominator and enumerator interpolated between quinquennial population censuses).
11. Waterworks (p.c.):	Five year average of population in cities with waterworks in a county, per total population (denominator and enumerator interpolated between quinquennial population censuses).
12. Sewerage (p.c.):	Five year average of population in cities with sewerage in a county, per total population (denominator and enumerator interpolated between quinquennial population censuses).
13. Medical professionals 1882 (p.m.):	Total number of medical professionals in 1882 in a county, per 1,000 people in 1880.
14. (ln) Distance to university:	Euclidean distance to the closest university with a medical school in the German Empire in kilometers.

Sources: Variables 1-10 from Galloway (2007), information in variable 11 and 12 derived from Grahn (1898-1902) and Salomon (1906-1907), variable 13 from Becker et al. (2014), variable 14 from own calculations using ArcGIS.

Table D.3: Summary statistics for district-level estimates in Tables 2 and 3

	Mean	SD	Min	Max	Observations
Crude death rate	24.108	3.435	16.587	32.676	216
Blue collar workers in 1882 (p.c.)	0.083	0.038	0.030	0.182	216
Insured in 1885 (p.c.)	0.070	0.034	0.014	0.177	216
Urbanization (share)	0.349	0.153	0.077	1.000	216
Waterworks (p.c.)	0.039	0.163	0.000	1.000	216
Sewerage (p.c.)	0.034	0.164	0.000	1.000	216
Average age	26.345	1.171	23.310	29.097	216
Age group 1-9 (share)	0.227	0.018	0.161	0.271	216
Age group 10-19 (share)	0.209	0.012	0.164	0.233	216
Age group 20-29 (share)	0.164	0.017	0.128	0.270	216
Age group 30-39 (share)	0.132	0.011	0.115	0.184	216
Age group 40-49 (share)	0.105	0.006	0.091	0.127	216
Age group 50-59 (share)	0.081	0.008	0.060	0.108	216
Age group 60-69 (share)	0.053	0.008	0.030	0.073	216
Age group 70 plus (share)	0.028	0.006	0.014	0.043	216
SPD vote (share)	0.111	0.121	0.000	0.595	216
CDR accidents	0.395	0.076	0.212	0.639	216
CDR waterborne	1.359	0.748	0.358	5.663	216
CDR airborne	7.390	1.302	4.799	10.419	216
CDR lung	5.514	1.051	3.338	8.482	216
CDR TB and scrofula	2.762	0.938	1.412	5.417	216
CDR non-infectious	2.967	0.609	1.637	4.778	216
CDR maternal	0.183	0.064	0.075	0.373	216
CDR unknown	2.564	1.171	1.078	6.613	216
Approbated doctors (p.m.)	0.358	0.157	0.096	1.234	216
Hospital patients (p.c.)	0.013	0.008	0.002	0.047	144

Notes: The table reports summary statistics of variables used to estimate the models in Tables 2 and 3.

Table D.4: Variable definitions for district-level estimates in Tables 2 and 3

1. Crude death rate:	Five year average of deaths (incl. stillbirths) in a district, per 1,000 people (denominator interpolated between quinquennial population censuses).
2. Blue collar workers in 1882 (p.c.):	Total number of employed blue-collar workers (sector B) in 1882 in a district, per total population in 1880.
3. Insured in 1885 (p.c.):	Total number of insured in 1885 in a district, per total population in 1885.
4. Urbanization (share):	Five year average of population in cities (with legal city rights) in a district, per total population (denominator and enumerator interpolated between quinquennial population censuses).
5. Waterworks (p.c.):	Five year average of population in cities with waterworks in a district, per total population (denominator and enumerator interpolated between quinquennial population censuses).
6. Sewerage (p.c.):	Five year average of population in cities with sewerage in a district, per total population (denominator and enumerator interpolated between quinquennial population censuses).
7. Average age:	Five year average of the mean age of the total population in a district (denominator and enumerator interpolated between quinquennial population censuses).
8. Age group 1-9 (share):	Five year average of population aged 1-9 in a district, per total population (denominator and enumerator interpolated between quinquennial population censuses).
9. Age group 10-19 (share):	Five year average of population aged 10-19 in a district, per total population (denominator and enumerator interpolated between quinquennial population censuses).
10. Age group 20-29 (share):	Five year average of population aged 20-29 in a district, per total population (denominator and enumerator interpolated between quinquennial population censuses).
11. Age group 30-39 (share):	Five year average of population aged 30-39 in a district, per total population (denominator and enumerator interpolated between quinquennial population censuses).
12. Age group 40-49 (share):	Five year average of population aged 40-49 in a district, per total population (denominator and enumerator interpolated between quinquennial population censuses).
13. Age group 50-59 (share):	Five year average of population aged 50-59 in a district, per total population (denominator and enumerator interpolated between quinquennial population censuses).
14. Age group 60-69 (share):	Five year average of population aged 60-69 in a district, per total population (denominator and enumerator interpolated between quinquennial population censuses).
15. Age group 70 plus (share):	Five year average of population older than 70 in a district, per total population (denominator and enumerator interpolated between quinquennial population censuses).
16. SPD vote (share):	Share of votes for the SPD and its predecessor parties in the general elections of 1874, 1878, 1884, 1890, 1893 and 1898 in a district.
17. CDR accidents:	Five year average of deaths from accidents in a district, per 1,000 people (denominator interpolated between quinquennial population censuses).
18. CDR waterborne:	Five year average of deaths from typhus, typhoid fever, and three types of diarrheal diseases in a district, per 1,000 people (denominator interpolated between quinquennial population censuses).
19. CDR airborne:	Five year average of deaths from smallpox, scarlet fever, measles, diphtheria, pertussis, scrofula, tuberculosis, tracheitis, pneumonia, and other lung diseases in a district, per 1,000 people (denominator interpolated between quinquennial population censuses).
20. CDR lung:	Five year average of deaths from pertussis, scrofula, tuberculosis, pneumonia, and other lung diseases in a district, per 1,000 people (denominator interpolated between quinquennial population censuses).
21. CDR TB and scrofula:	Five year average of deaths from scrofula and tuberculosis in a district, per 1,000 people (denominator interpolated between quinquennial population censuses).
19. CDR non-infectious:	Five year average of deaths from cancer, edema, stroke, heart disease, brain disease, and kidney disease in a district, per 1,000 people (denominator interpolated between quinquennial population censuses).
20. CDR maternal:	Five year average of deaths related to childbirth in a district, per 1,000 people (denominator interpolated between quinquennial population censuses).
21. CDR unknown:	Five year average of unknown or unspecified causes of death in a district, per 1,000 people (denominator interpolated between quinquennial population censuses).
22. Approbated doctors (p.m.):	Total number of approbated doctors (1876, 1879, 1882, 1887, 1898, 1901) in a district, per 1,000 people (denominator interpolated between quinquennial population censuses).
23. Hospital patients (p.c.):	Five year average of hospital patients in a district, per total population (denominator interpolated between quinquennial population censuses).

Sources: Variables 1-2, 4, and 16 from Galloway (2007), information in variable 4 and 5 derived from Grahn (1898-1902) and Salomon (1906-1907), all other variables digitized from various volumes of KSBB (1861–1934).

Table D.5: Summary statistics for insurance expenditure estimates in Table 5

	Mean	SD	Min	Max	Observations
Crude death rate	22.763	3.270	15.766	33.078	684
Doctor visits (p.i.)	3.133	0.790	1.147	6.621	684
Medication (p.i.)	2.540	0.675	1.339	5.321	684
Hospitalization (p.i.)	1.497	0.680	0.373	4.250	684
Sick pay (p.i.)	5.662	2.126	1.852	14.136	684
Maternity ben. (p.i.)	0.176	0.127	0.009	0.694	684
Death ben. (p.i.)	0.510	0.192	0.121	1.096	684
Administration (p.i.)	0.926	0.337	0.370	2.190	684
Sick days (p.i.)	6.050	1.174	0.452	11.198	612
Urbanization (share)	0.378	0.156	0.122	1.000	684
Waterworks (p.c.)	0.239	0.183	0.000	1.000	684
Sewerage (p.c.)	0.157	0.180	0.000	1.000	684

Notes: The table reports summary statistics of variables used to estimate the models in Panel A of Table 5.

Table D.6: Variable definitions for insurance expenditure estimates in Table 5

1. Crude death rate:	Total number of deaths (incl. stillbirths) in a district in a year, per 1,000 people (denominator interpolated between quinquennial population censuses).
2. Doctor visits (p.i.):	Total insurance fund expenditures on doctor visits in Mark in a district in a year, per insured.
3. Medication (p.i.):	Total insurance fund expenditures on medication and remedies in Mark in a district in a year, per insured.
4. Hospitalization (p.i.):	Total insurance fund expenditures on hospital care and board in Mark in a district in a year, per insured.
5. Sick pay (p.i.):	Total insurance fund expenditures on sick pay to members and dependents in Mark in a district in a year, per insured.
6. Maternity ben. (p.i.):	Total insurance fund expenditures on maternal care in Mark in a district in a year, per insured.
7. Death ben. (p.i.):	Total insurance fund expenditures on death benefits in Mark in a district in a year, per insured.
8. Administration (p.i.):	Total insurance fund expenditures on administration in Mark in a district in a year, per insured.
9. Sick days (p.i.):	Total number of sick days of members in a district in a year, per insured.
10. Urbanization (share):	Total population in cities (with legal city rights) in a district in a year, per total population (denominator and enumerator interpolated between quinquennial population censuses).
11. Waterworks (p.c.):	Total population in cities with waterworks in a district in a year, per total population (denominator and enumerator interpolated between quinquennial population censuses).
12. Sewerage (p.c.):	Total population in cities with sewerage in a district in a year, per total population (denominator and enumerator interpolated between quinquennial population censuses).

Sources: Variable 1 and 10 from Galloway (2007), information in variable 11 and 12 digitized from Grahn (1898-1902) and Salomon (1906-1907), variables 2-9 digitized from various volumes of KSA (1884–1942).

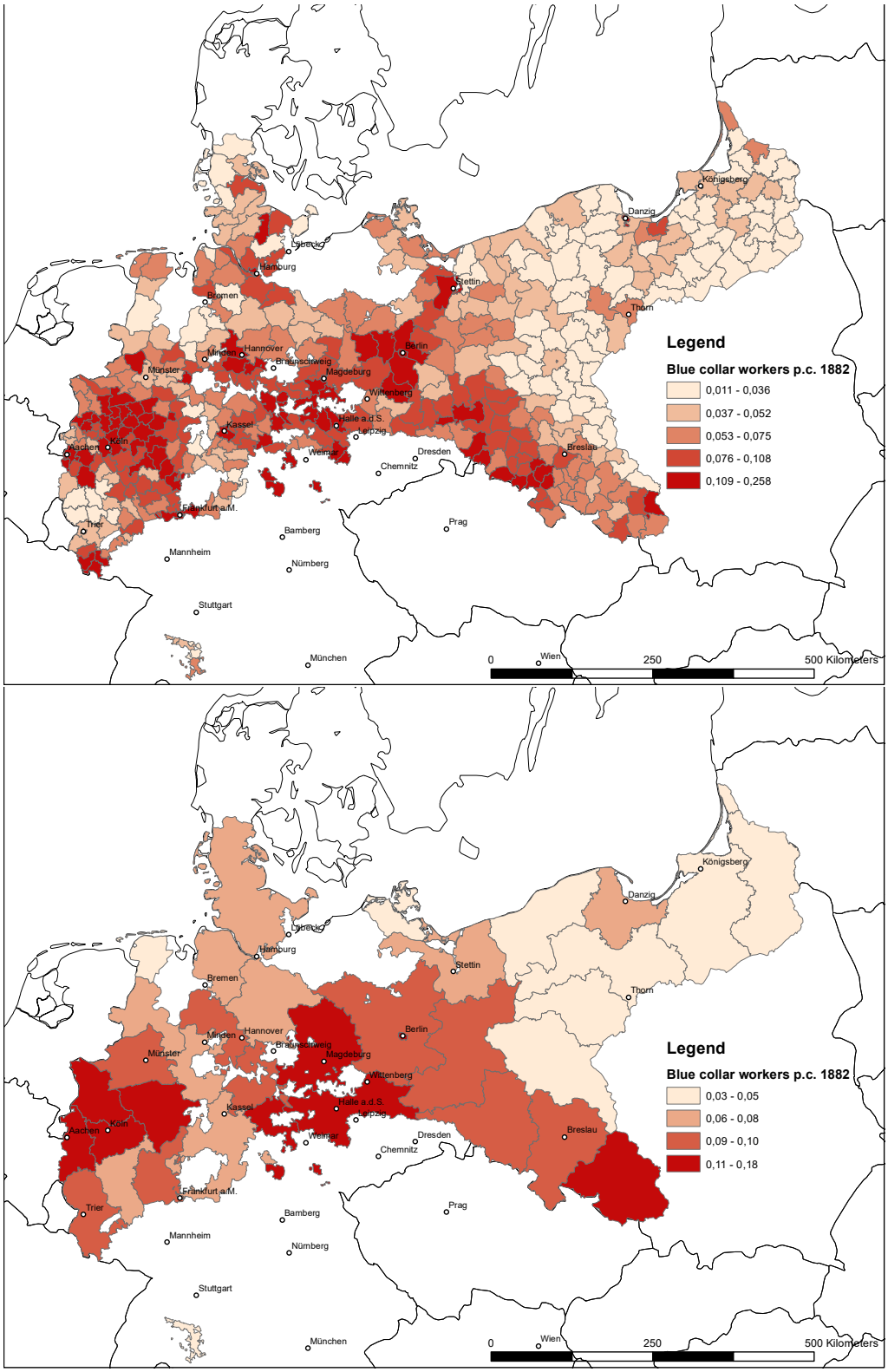

Figure D.1: Cross-sectional variation in the blue-collar worker share in 1882. *The figure shows a map of Prussian counties (top) and districts (bottom) in the borders of 1880. Darker colors indicate a higher share of blue-collar workers in the total population.*

Table D.7: County fixed effects model, accounting for baseline controls

Dep. var.: Crude death rate	Baseline Spec.	Control Variables	Male Mortality	Female Mortality	Infant Mortality	Legit. Infants	Illegit. Infants	Self-empl. Placebo	Publ. S. Placebo
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
Treatment × 1875	1.891	1.209	-5.774***	-1.244	-8.386	-14.398	100.781	7.876*	0.055
	(1.623)	(2.027)	(2.022)	(2.170)	(13.294)	(13.931)	(64.676)	(4.247)	(5.028)
Treatment × 1885	-7.768***	-7.573***	-7.614***	-7.399***	-1.982	-9.295	54.231	2.120	13.891***
	(1.693)	(2.022)	(2.313)	(1.837)	(13.077)	(13.634)	(56.638)	(4.138)	(5.250)
Treatment × 1890	-4.754**	-0.632	-3.104	-3.412	-11.998	-28.614*	94.573	17.394***	17.294***
	(2.041)	(2.274)	(2.549)	(2.216)	(15.991)	(16.397)	(67.271)	(5.785)	(5.805)
Treatment × 1895	-8.631***	-6.364**	-4.049	-8.182***	-59.419***	-82.634***	75.727	7.549	13.013**
	(2.080)	(2.472)	(2.916)	(2.458)	(22.614)	(23.090)	(81.886)	(6.128)	(6.321)
Treatment × 1900	-12.330***	-10.860***	-14.257***	-12.817***	-52.439*	-82.909***	128.922	4.884	19.833***
	(2.399)	(2.770)	(3.184)	(2.845)	(27.972)	(28.094)	(86.156)	(6.901)	(7.378)
Controls × Time FE	No	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
County FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Time FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Observations	2645	2645	2645	2645	2645	2645	2645	2645	2645
Counties	441	441	441	441	441	441	441	441	441
Periods	6	6	6	6	6	6	6	6	6
R-squared	0.75	0.75	0.74	0.74	0.23	0.26	0.03	0.75	0.75

Notes: The table reports county-level fixed effects estimates. All variables are averaged over five year periods from 1875 to 1904. The omitted period is 1880-84. The dependent variable measures crude death rates using total deaths per alive population in thousands. Dependent variable in columns 1, 2, 8, and 9: total mortality; column 3: male mortality; column 4: female mortality; column 5: infant mortality (< 1 year) per 1,000 births; column 6: infant mortality (< 1 year) born in wedlock per 1,000 births in wedlock; column 7: infant mortality (< 1 year) born out of wedlock per 1,000 births out of wedlock. Treatment variables in columns 1-7: blue-collar workers' population share in 1882, interacted with time dummies; column 8: self-employed industrial workers' population share in 1882, interacted with time dummies; column 9: public servants' population share in 1882, interacted with time dummies. Controls: urbanization rate, waterworks per capita, and sewerage per capita, all held fixed for 1880–1884 and interacted with time dummies. Standard errors, clustered at the county level, in parentheses. * 10%, **5%, *** 1% confidence level.

Table D.8: County fixed effects model, accounting for mean reversion

Dep. var.: Crude death rate	Baseline Spec.	Control Variables	Male Mortality	Female Mortality	Infant Mortality	Legit. Infants	Illegit. Infants	Self-empl. Placebo	Publ. S. Placebo
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
Treatment × 1875	1.891	1.564	-5.347***	1.591	-11.771	-13.709	42.499	5.041	-0.227
	(1.623)	(1.620)	(1.641)	(1.736)	(10.852)	(11.562)	(52.590)	(3.841)	(4.069)
Treatment × 1885	-7.768***	-6.855***	-5.896***	-4.957***	3.388	1.491	71.517*	-2.546	1.087
	(1.693)	(1.644)	(1.921)	(1.511)	(11.359)	(11.991)	(42.556)	(4.490)	(3.063)
Treatment × 1890	-4.754**	-2.979	-3.946*	-3.786*	-21.154	-31.820**	95.852*	6.128	-2.102
	(2.041)	(2.005)	(2.253)	(2.012)	(14.906)	(15.512)	(49.285)	(6.159)	(3.664)
Treatment × 1895	-8.631***	-6.591***	-2.594	-5.562***	-31.405*	-50.268***	132.818*	-1.133	-2.982
	(2.080)	(2.062)	(2.470)	(2.145)	(18.803)	(19.316)	(70.765)	(6.960)	(4.155)
Treatment × 1900	-12.330***	-10.018***	-11.889***	-9.312***	-35.527	-61.718***	181.488***	-5.258	0.941
	(2.399)	(2.300)	(2.658)	(2.489)	(22.228)	(22.612)	(61.601)	(7.795)	(4.963)
Controls	No	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Baseline dep. var. × Time FE	No	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
County FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Time FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Observations	2645	2645	2645	2645	2645	2645	2645	2645	2645
Counties	441	441	441	441	441	441	441	441	441
Periods	6	6	6	6	6	6	6	6	6
R-squared	0.75	0.77	0.76	0.76	0.26	0.29	0.10	0.77	0.76

Notes: The table reports county-level fixed effects estimates. All variables are averaged over five year periods from 1875 to 1904. The omitted period is 1880-84. The dependent variable measures crude death rates using total deaths per alive population in thousands. Dependent variable in columns 1, 2, 8, and 9: total mortality; column 3: male mortality; column 4: female mortality; column 5: infant mortality (< 1 year) per 1,000 births; column 6: infant mortality (< 1 year) born in wedlock per 1,000 births in wedlock; column 7: infant mortality (< 1 year) born out of wedlock per 1,000 births out of wedlock. Treatment variables in columns 1-7: blue-collar workers' population share in 1882, interacted with time dummies; column 8: self-employed industrial workers' population share in 1882, interacted with time dummies; column 9: public servants' population share in 1882, interacted with time dummies. Controls: urbanization rate, waterworks per capita, sewerage per capita, and baseline mortality (1880-84) interacted with time dummies. Standard errors, clustered at the county level, in parentheses. * 10%, **5%, *** 1% confidence level.

Table D.9: District fixed effects using blue-collar workers and insured population

	Initial blue collar workers (1882)					Initial insured (1885)				
Dep. var.: Crude DR	Base	Controls	Male	Female	LegInf	Base	Controls	Male	Female	LegInf
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)
Treatment × 1875	1.91	2.34	-6.87	1.06	12.29	5.04	5.01	-3.60	3.90	20.34
	(5.37)	(5.08)	(5.23)	(5.71)	(42.20)	(4.30)	(4.42)	(4.12)	(5.60)	(42.70)
Treatment × 1885	-13.33**	-13.21**	-14.20**	-14.20**	-22.92	-15.01**	-15.17**	-15.49**	-15.95**	-52.28
	(5.36)	(5.48)	(5.93)	(5.46)	(43.79)	(6.11)	(6.12)	(6.90)	(6.07)	(43.38)
Treatment × 1890	-9.67	-10.58	-15.63	-16.20	-107.93	-11.27	-12.05	-19.03	-18.19	-151.34*
	(8.80)	(8.91)	(12.05)	(9.86)	(81.41)	(10.65)	(10.45)	(14.02)	(11.42)	(77.35)
Treatment × 1895	-13.89*	-15.31*	-15.03	-18.90**	-185.53	-16.73*	-17.70*	-19.68	-22.29**	-232.41**
	(8.17)	(8.14)	(11.60)	(8.98)	(114.02)	(9.16)	(8.75)	(12.75)	(9.39)	(103.72)
Treatment × 1900	-18.41**	-19.36**	-24.59**	-24.09**	-204.16*	-21.83**	-22.26**	-29.03**	-28.18***	-238.17**
	(8.81)	(8.72)	(12.01)	(9.30)	(117.51)	(9.00)	(8.30)	(12.36)	(8.83)	(103.43)
Controls	No	Yes	Yes	Yes	Yes	No	Yes	Yes	Yes	Yes
District FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Time FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Observations	216	216	216	216	216	216	216	216	216	216
Districts	36	36	36	36	36	36	36	36	36	36
Periods	6	6	6	6	6	6	6	6	6	6
R-squared	0.88	0.88	0.85	0.86	0.51	0.88	0.89	0.86	0.87	0.52

Notes: The table reports district-level fixed effects estimates. All variables are averaged over five year periods from 1875 to 1904. The dependent variable in columns (1), (2), (6), and (7) is crude death rates measured as total deaths per alive population in thousands. The dependent variable in columns (3) and (8) is male mortality, the dependent variable in columns (4) and (9) is female mortality, and the dependent variable in columns (5) and (10) is infant mortality (< 1 year) born in wedlock per 1,000 births in wedlock. The treatment variable in columns (1)-(5) is blue-collar workers' population share observed in 1882 interacted with time-period dummies. The treatment variable in columns (6)-(10) is the share of the population covered by health insurance in 1885 interacted with time-period dummies. The omitted period is 1880-84. Controls include the urbanization rate, waterworks per capita, and sewerage per capita. Standard errors are clustered at the district level and given in parentheses. * 10%, **5%, *** 1% confidence level

Table D.10: The supply of health services

	Initial blue collar workers (1882)	
Dep. var.:	Approbated doctors	Hospital patients
	(1)	(2)
Treatment × 1875	-0.07	
	(0.21)	
Treatment × 1885	-0.16**	0.01*
	(0.07)	(0.01)
Treatment × 1890	-0.07	0.04***
	(0.15)	(0.01)
Treatment × 1895	0.28	0.05***
	(0.41)	(0.01)
Treatment × 1900	0.42	
	(0.57)	
Controls	Yes	Yes
District FE	Yes	Yes
Time FE	Yes	Yes
Observations	216	144
Districts	36	36
Periods	6	4
R-squared	0.70	0.81

Notes: The table reports district-level fixed effects estimates. The treatment variable is blue-collar workers' population share observed in 1882 interacted with time-period dummies. The dependent variable in column (1) is the number of approbated doctors per capita. The dependent variable in column (2) is the number of inpatients in hospitals (unavailable prior to 1880). Controls include the urbanization rate, waterworks per capita, and sewerage per capita. The omitted period is 1880-84. Standard errors are clustered at the district level and given in parentheses. * 10%, **5%, *** 1% confidence level