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Clean water technologies and urban mortality in Sweden 1885-1925

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Abstract

Using a newly digitised historical dataset on clean water technologies in Swedish cities, this study tries to find the causal effect (and the magnitude of this effect) of the introduction of clean water technology on mortality in urban areas. Using urban mortality data on a county level, and clean water access data on city level, both OLS, fixed effects, and difference-in-differences models are tried in order to isolate an effect. The result of the models show that it is not possible to capture the mortality (neither general nor disease-specific) variation using this dataset: the general mortality decline in cities during this period is confounded by too many omitted variables apart from clean water technology. This study is a first step in the process, but to come closer to a reliable estimate, sharper variables (both dependent and independent) and closer analysis is needed.

Keywords: mortality decline, clean water technology, epidemiology, urban penalty, Sweden

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1 Introduction

The mortality decline that started in the 18th century, and led to an unprecedented rise in life expectancy in Europe and other parts of the world, is by many considered one of the greatest events in human history. The well-known economic historian Robert Fogel agrees on this, but explains that he's hesitant to call it "one of the greatest human achievements", as he was not sure how much of this development has been under human control (Fogel 1986, p.376). The question is not, he argues, *if* a particular factor (such as economic development, medicine, public health or nutrition) has been involved in the decline, but *how much* it has contributed to the decline. The magnitude and timing of effects on mortality has been, and is, subject of much debate, and this study aims to contribute knowledge to this subject. By looking at a specific angle of the problem (public health interventions in form of clean water technologies), the aim is to try and answer how much this has affected mortality in urban areas in Sweden. The problem that can be solved using these kinds of technologies is the spread of water-borne diseases. These are mostly a thing of the past in developed countries, but continue to be a problem in many other parts of the world today. According to the WHO, 1.8 million people die every year from diarrhoeal diseases related to unsafe water supply, sanitation and hygiene (WHO 2014). The sanitary conditions in several developing countries today are in many ways similar to the disease environment found in western cities in the end of the 19th century (Ferrie and Troesken 2008, p.2), which makes study of the mechanisms behind historical urban mortality relevant also in a contemporary setting. There is, however, a need for more information on the effectiveness of interventions in improving water and sanitation, to distinguish them from other interventions in reducing mortality (Fewtrell et al. 2005, p.42). The research question that guides this analysis is: how much did the implementation of clean water technologies in Swedish cities affect urban mortality between 1885 and 1925?

2 Relevance

As mentioned, the problem of inadequate water and sanitation was not only a problem in 19th and 20th century cities, it is still a problem in many parts of the world today (ibid., p.42). For example, it was estimated by UNICEF that 768 million people relied on "unimproved" water supplies in 2011, and 2.5 billion people lack access to a working sanitation facility (Prüss-Ustün et al. 2014, p.895). In order to know where to place resources for maximum effect in lowering mortality, the costs and benefits of certain projects need to be clear, and this is where learning from history becomes important. As explained by Ferrie and Troesken (2008, p.2): building piped water and sewage is a costly and time-consuming project, compared to vaccinations and information campaigns concerning better household hygiene - therefore we need to be able to anticipate the effects. By estimating an effect of the measures taken in 19th and 20th century cities, some lessons might be learned for how to best lower mortality in water-borne diseases in the future.

3 Background

This section will look closer into the historical and scientific setting before and during the analysis period, and will discuss subjects such as the mortality decline, disease theory, and the environment in a Swedish city during this period.

3.1 The 19th and 20th century mortality decline

The decline in mortality that started in Europe in the 18th century was unprecedented in human history. The decline is often said to have begun in England in the middle of the 18th century, where life expectancy at birth first rose from 37 years in early 18th century to 41 years in 1820 (D. M. Cutler, Deaton, and Lleras-Muney 2006, p.99). Here, it remained stable at around 40 years until 1870, when it began rising steadily and was above 50 years in the early 20th century. This mortality decline has since been repeated in most of the European and Western countries, and also later in various forms and paces in other parts of the world.

3.2 Causes for the mortality decline

The reasons behind the mortality decline in Europe have been widely debated. An important voice in the debate was Thomas McKeown, who sparked a debate by arguing that nutrition and economic growth were the primary cause of mortality decline, rather than medicine and public health improvements. In McKeown, Brown, and Record (1972, p.382), he concludes that the reduction in mortality in 19th and 20th century Europe was "probably due to a significant increase in food supplies", and rules out other factors by comparing their timing to the timing of the mortality decline. This conclusion has been criticised: Aaby (1992, p.174-175) argues that McKeown has greatly exaggerated the importance of nutrition in mortality decline, and also thinks that his "negative exclusion" of other factors is an unsatisfactory method of reaching a reliable conclusion. Easterlin (1999, p.265) also opposes the conclusions of McKeown, by arguing that the time-scale McKeown used in reaching the conclusions is wrong: the take-off dates for economic growth that paved way for better nutrition does not coincide with the time-scale of the mortality decline in the countries that McKeown references to. A more recent opinion is given by Soares (2007, p.254), who notes that although McKeown is right to some extent, there are still substantial parts of the mortality decline that can not be explained by nutrition or economic growth. The main arguments for the different views on the reasons for mortality decline will be presented below.

3.2.1 Nutrition and economic growth

The nutritional argument as a driver of mortality decline is based on the fact that nutrition and the availability of food can have a both a direct and indirect effect on mortality: grave shortage leads to death by starvation, and less than lethal shortage can affect one's ability to resist diseases. McKeown, Brown, and Record (1972, p.379) make the argument that the agricultural sector advanced significantly in Europe just before and during the European mortality decline, but that other factors that could have had a negative effect on mortality were implemented later in time. Fogel (1986) summarises a common criticism against McKeown's case: if food supply was the critical factor, there should have been differences between mortality

reductions in rich and poor classes of a population (since the richer class should have better access to food). This point has been also made by Razzell (1974), who found no such differences: instead, the mortality rate in the English 18th century nobility were as high as in the labouring classes. McKeown's theories have also been disputed using food price variation models on mortality, where many have only found weak and short-term relationships (Fogel 1986, p.374). All in all, according to Fogel (ibid.), most scholars would agree with McKeown that nutrition plays a part in the mortality decline, but that he underestimated the impact of public health and hygiene.

3.2.2 Medicine and vaccination

The effect of medical advances on mortality is not as disputed as the nutritional argument, at least not when it comes to timing. Easterlin (1999, p.269) argues, along with McKeown and Record (1962), that medical advances had little to do with mortality decline until earliest in the late 19th century. From this point on, however, Easterlin (1999, p.273) argues that medical advances such as methods, vaccines and drugs led Europe into the first phase of the epidemiologic transition (as described by Omran (1971)). However, Razzell (1974, p.11), argues that the inoculation for smallpox (which is also mentioned by McKeown) at least should be considered as a factor in the decline of mortality in late 18th/early 19th century. From the late 19th century and onwards, Lee (2003, p.170) sees the progress in medicine as the most important factor in reducing mortality: he mentions that the 20th century is the century when the fatal infectious diseases are brought under control through medical methods, vaccines and drugs. Another medicine-related factor that has been lifted in the debate is the diminished virulence of some critical viruses (the smallpox virus, for example): when viruses for some reason become less aggressive during the 18th and 19th century, mortality is also diminished (PHAS 2005, p.49). This (supposedly spontaneously) decreased virulence would speak against the effect of medicine, and has been argued by Fridlitzius (1985) to be of special importance in the case of Swedish mortality decline during the first demographic transition.

3.2.3 Other public health measures

Besides advances in medicine, general public health measures have also had an impact on a population's susceptibility for disease. Contradicting Lee (2003), Soares (2007, p.254) argues that the decline in infectious diseases in the first half of the 20th century is not attributed to medicine, but to the access to clean water. Easterlin (1999, p.270) also argues along these lines, citing the "sanitation revolution" as being the first step towards reducing mortality in infectious diseases from the mid-19th century and onwards. This "revolution" consists of the introduction of state-sponsored public health systems, such as clean water technologies, waste management, sanitation and meat inspections, to mention a few important measures. Another explanation for the mortality decline is an increasing knowledge and practise of personal hygiene in households and by individuals of a population. Health behaviour campaigns, initiated by authorities, and increasing education of mothers are parts of this phenomenon (D. Cutler and Miller 2005, p.2).

3.2.4 The mortality decline: conclusion

As presented in this section, the reasons for the mortality decline in European and Western countries have been, and are still, subject for debate. The "nutrition" hypothesis presented by McKeown and Record (1962) has been very influential, but has also given rise to criticism and alternative explanations. In this study, the magnitude of a certain aspect of the public health measures (clean water technologies) is investigated, and if reality mirrors the academic debate on the subject - there will be many other factors than clean water technologies affecting the Swedish urban mortality decline. As Cutler (2005, p.2) puts it, the theoretical explanations behind the mortality decline are not mutually exclusive, but it is important to distinguish between them if one wants to draw conclusions.

3.3 Historical background: Sweden

In the beginning of the 19th century, the distribution of people living in urban and rural areas was almost the opposite of what it is today: below 10 % of the population were living in urban areas between 1800-1820, as opposed to almost 85% in 2000 (Klaesson and Pettersson 2009, p.320). The urbanisation process caught momentum first from the 1860s and onwards, which put increasing strain on housing and environment in urban areas. During the 1870s, Sweden experienced an unprecedented industrial growth, which came to drive much of the increase in urbanisation (PHAS 2005, p.396).

During the 19th century, mortality was significantly higher in urban areas than in rural areas (more on this divide in section 7.1.4). The higher mortality was partly due to the spread of infections being facilitated by overcrowding, but also in large parts due to the generally poor domestic hygiene (ibid., p.390). Using the example city of Sundsvall (also included in this analysis), Edvinsson (1992) paints a vibrant and not so attractive picture of the sanitary conditions in a Swedish city at the end of the 19th century: disposal of human and animal excrement (animals were common inside the city at this time) was the responsibility of landlords, and often not their highest priority. Also, this excrement was often gathered to be used as fertilizer in the fields surrounding the city, which meant that piles of manure were kept in apartment building courtyards before being transported away (Edvinsson 1992, p.125). This, combined with the fact that waste also was thrown directly into the streets, meant that large volumes of contaminated water could pour into the groundwater. This groundwater would in many cases be taken up again from drinking wells. Wetterberg and Axelsson (1995, p.14) tell the same story from the city of Gothenburg: waste water containing excrement and garbage was typically left in the streets to be washed away with the rain. The road paving in late 19th century Gothenburg was not as dense as today's cobblestones, which meant that waste trickled down between the rocks and fermented. This created, quoting a contemporary doctor's writing, "horrendous smell in the city" (ibid., p.14). A similar story from Linköping is mentioned by Castensson, Löwgren, and Sundin (1988, p.280), where one of the graveyards was flooded, leaving the run-off water to run into the main river from where drinking water was usually taken. Handling of food was also a problem: production and sales of meat were not regulated, and tales are told of barrels of "yellowish pork, blueish beef and almost transparent meat from sheep" being sold at market (Edvinsson 1992, p.127). In a similar manner, milk of

poor quality was being sold. In general, apart from the problems with clean water, food was also being tampered with, mixed, and re-sold for profit at the expense of hygiene (Edvinsson 1992, p.128).

3.4 Theory of disease spread

The idea of contamination as a physical concept has existed in various forms throughout history: before the entrance of bacteriology in the 1870s, the miasma theory of disease was common (D. Cutler and Miller 2005, p.3). According to this theory, disease was spread through vapours (called "miasmas") that also carried an offensive smell. Cutler (*ibid.*, p.3) notes that this system seems to be based on "a kind of Pavlovian learning", where it was noted that people being exposed to foul odours were more often sick. This notion was confirmed in the classical report by the "father of epidemiology", John Snow: the report mapped a cholera outbreak in London 1854 to a certain drinking well. After the well was closed, the disease outbreak subsided (PHAS 2005, p.402).

The French doctor Jean-Baptiste Fonssagrives published a book on urban hygiene in 1872, in which bad odours in urban areas was theoretically linked to disease spread, according to the miasma theory (Wetterberg and Axelsson 1995, p.35). The different odours of 19th century cities were categorised, and the excrement odour was deemed the most harmful (as it was thought to contain disease). Fonssagrives argued that cities should strive to be odour-free - not by trying to remove or deodorise the odour, but by removing the source of the odour. This view on health and sanitation soon became the prominent view, and was confirmed by the entrance of bacteriology in the 1870s (*ibid.*, p.40). The discovery and isolation of the cholera and tubercle bacteria (by German doctor Robert Koch) in 1872 was a breakthrough in public health innovation, but the exact reasons for the diseases were still disputed by the prominent Bavarian chemist Max von Pettenkofer (Bjur 1988, p.89). Von Pettenkofer argued that the "cholera equation" had three parameters that needed to be fulfilled if the disease was going to infect a person: X (which was presence of the actual bacteria), Y (which was the characteristics of the current time and surroundings) and Z (which was individual characteristics). The somewhat mysterious Y condition was disputed by Koch, who argued that anyone infected with the bacteria could develop the disease. Von Pettenkofer stood by his Y condition so fiercely that he had Koch deliver him a cholera bacteria sample, which he consumed to prove his theory (and did not, reportedly, develop cholera). However, Koch proved his theory by mapping victims of a cholera outbreak in Hamburg in 1892: one city district was almost completely spared of the disease, while the neighbouring district was struck hard. Since the districts were neighbouring, the Y condition should be fulfilled; however, it was found that the difference between the districts was that the spared district was slow-filtering its drinking water through a sand filter (*ibid.*, p.89). Both districts had the same river as water source. The bacteriological view on communicable diseases became, as apparent, the dominant view for both 20th and 21st century medicine.

With this general picture of mortality decline, urban living standards, and theory of disease, we move on to a more specific part of theory that focuses explicitly on clean water technologies.

4 Theory

In this section, the theoretical background relevant for the method used will be presented: this includes a more specific history on the supply of water and relevant previous research.

4.1 Previous research

Studies on the role of public health improvements (and more specifically, clean water technologies) on mortality have been conducted in many geographical settings. A good example is the study by D. Cutler and Miller (2005), which analyses the impact of clean water technologies in 13 American cities on mortality. Mortality in typhoid fever is used as proxy for water-borne diseases, and the results show a "strikingly large and cost-beneficial role of clean water technologies in reducing mortality" (ibid., p.20). While generally stressing the role of an improved diet as the primary force, McKeown and Record (1962, p.120) also conclude that improved public sanitary conditions were an important driver behind the decline in water-borne disease mortality in late 19th century England. On a more general note, Soares (2007) argues that although many scholars link falling mortality to rising income (and what comes with it), many public goods such as early health technologies and infrastructure had a very large part in the mortality reduction. Schultz (1993) finds, on the other hand, that access to water has an insignificant impact on mortality in his model of contemporary low-income urban areas: he instead finds better significance using mother's education level. For Sweden, Castensson, Löwgren, and Sundin (1988, p.287) have made a minor study on four cities, but they find no noticeable connection between the construction of piped water and mortality.

4.2 History of water supply

Access to water in 19th century urban areas, before the introduction of piped water, often came from public wells (Edvinsson 1992, p.115). Apart from this groundwater supply, surface water (such as nearby rivers and lakes) was also used in households - and both of these types of local water sources created a vulnerability to human-induced water pollution (Castensson, Löwgren, and Sundin 1988, p.280). As described in section 3.3, the groundwater from which the wells were supplied could be contaminated by inadequate waste handling, which led to reoccurring outbreaks of epidemics from water-borne diseases. The first Swedish city to implement piped water was Uppsala, where a wooden pipe system was built in 1649. Usually, a sewage management system was constructed in conjunction with, or not long after, a water-carriage system installed in the city (ibid., p.284).

The introduction of piped water and sewage did not always lead to a direct improvement for the individual citizen's environment. Duffy (1992), cited by D. Cutler and Miller (2005), describe the non-optimal sewage handling system that was first constructed in Philadelphia: the primary sewage outfall was emptied in a river upstream (and close to) the city's primary water intake, which created a "circular water system" (ibid., p.4). As mentioned in section 3.3, auto-contamination of drinking water in Swedish cities was also common. A satirical picture of how the water supply in Stockholm worked (or, didn't work) in the late 19th century can be seen in Figure 1 (the text under the picture says, in translation, "The picture speaks

enough for itself”) - published in a popular satirical paper (Söndags-Nisse 1866).

Figure 1: ”A Stockholm sewer”



Although the knowledge of disease transmission through water was present (see section 3.4), the motivation for introducing piped water in a city was more often something else than improving public health (Edvinsson 1992, p.115). Edvinsson cites, again with the example of Sundsvall, the discussions in city council prior to the installation and expansion of piped water: the question was first raised in 1858, pleading fire safety, industry water supply, and public health as motivation. The suggestion was initially voted down by the council (*ibid.*, p.116). Questions about costs, taxes, and coverage led the question to remain disputed in the council until its acceptance in 1877 (18 years later). Bjur (1988, p.89) tells the same story of political inertia in Gothenburg, and how officials eagerly followed the miasma/bacteriology debate while trying to decide on the implementation of piped water in the city. Debates and delays of implementation of clean water technologies was also common in American cities during this period: D. Cutler and Miller (2005, p.6), citing McCarthy (1987), mentions the city of Philadelphia, where it took 20 years from decision to implementation of water filtering.

Along with the construction of piped water and sewage, the need for purification of the water also arose. D. Cutler and Miller (2005, p.5) mentions three common methods: sand filtering (also known as slow filtering), mechanical filtering (rapid filtering) and chlorination. In Swedish cities, all of these methods have been used, but in different combinations and forms (this data is present in the non-digitised part of this dataset). In Gothenburg, for example, it was discovered in after water pipes had been built in 1890, that the water quality in the water source was unsatisfactory: this meant that the project was delayed so that a water treatment plant could be built (Bjur 1988, p.132). The plant, which used slow filtering technology, started delivering water to the city four years later (*ibid.*, p.134). This type of delays are not well documented in the current dataset, and might cause a bias (more on this in section 7.4).

4.3 Coverage and timing of clean water technologies

A common solution for supplying water to a city was pumping from a nearby lake, as was implemented in Sundsvall: far from all households in the city were covered by the water piping (and were thus forced to use wells), and at year 1900, only 51% of the households were connected (Edvinsson 1992, p.120). The concept of partial treatment within a city is a logical consequence of the natural inertia in implementing large city-scale projects. It is also thereby reasonable to believe that the time-scale of implementation can vary considerably between cities (as seen in Figure 10). Two aspects of implementation of technology are worth noting: firstly, it can be argued that the timing of implementation of clean water technology in a city is exogenous and determined by arbitrary events in a city (D. Cutler and Miller 2005, p.6). This would mean that, for example, city A and city B might be alike and have similar preconditions for implementing clean water technology, but city A might be 10 years slower in implementing because of a different political process. Assuming that this political process does not vary with the current variable of interest (mortality), this difference in timing can be exploited as a quasi-experiment (as is done by Cutler(2005)). Secondly, a city can not implement a full-coverage water/sewage system overnight. As mentioned in the example of Sundsvall, even 18 years after the implementation, only half of the city's population had access to piped water (Edvinsson 1992, p.120). This will, as discussed further in section 7.4, pose a potential bias problem in the following models.

5 Data

The data source for this study is newly digitised official documents from Swedish authorities on population, mortality and clean water technologies. Sweden's historical demographic data is widely regarded for its long record and for its high quality (Bengtsson and Lindström 2003). Still, some areas (like the current) have not yet been used for analysis, which makes this study unique. Data on total mortality (crude death rate, CDR), infant mortality (infant mortality rate, IMR), disease-specific mortality, and clean water technologies have been digitised and gathered into a common format (which means that data collection has been a large part of this analysis). The dataset includes 25 counties and 92 cities, and focuses on the years 1885 to 1925. The chosen starting year of the analysis period was necessary because of 1885 being the first year when mortality data was reported with the urban/rural definition. A 40-year analysis period (ending in 1925, even though data is available further until present day) was chosen in order to keep historical consistency, and also because this is the period where much of the urban mortality decline takes place.

5.1 Mortality data

Several water-borne diseases contributed (among other factors) to a high urban mortality in 19th and 20th century Sweden. The causal effect on general mortality as a dependent variable from different independent variables is not trivial, but it is at the very centre of this study. The mortality data in this analysis has been calculated with the following standard formulas:

$$CDR = \frac{\sum deaths_{year, county(urban)}}{\sum population_{year, county(urban)}} \quad IMR = \frac{\sum deaths, age < 1_{year, county(urban)}}{\sum births_{year, county(urban)}}$$

In raw data, mortality is reported on county level, divided into "rural" and "urban" categories. The CDR and IMR measures are therefore calculated using only urban populations (introduction of piped water and sewage in rural areas was a project that came well after the analysis period in this project).

5.1.1 Infant mortality rate

The IMR measure is, when it comes to historical mortality decline (and water-borne diseases in particular) a more interesting measure than general mortality. Firstly, it is well known that the increase in life expectancy in the 19th and 20th centuries was in large driven by a reduction in IMR (Bloom 2011, p.564), so there is probably a larger effect to be expected here than in a general mortality measure. Secondly, IMR is especially influenced by the water-borne diseases that were present in the 19th and 20th century urban environment (such as diarrhoeal diseases, cholera, and gastroenteritis). It has been shown that the combination of contaminated water and sporadic use of breastfeeding has a large negative impact on infant mortality, which makes the independent variable used in this study interesting in combination with IMR (Habicht, DaVanzo, and Butz 1988, p.456). Still today, diarrhoeal disease is one of the leading causes of infant and child mortality and morbidity in less developed countries (Fewtrell et al. 2005, p.42)

5.1.2 Disease-specific mortality data

Apart from general mortality data, data on mortality from specific diseases on county level has been collected. Specifying the cause of death in mortality data allows us to get rid of the mortality variation that by definition could not be part in the causal chain with our independent variable. In this case, the interesting diseases are water-borne diseases, since clean water technologies are the area of interest, and the diseases that are used in this analysis are: typhoid, cholera, dysentery and gastroenteritis. These four diseases were the most common water-borne diseases in urban areas at the time, and some of them have taken a big toll on human lives in periods (cholera has, for example, been called the "plague of the 19th century" in Sweden). The mortality rates in these diseases separately, and together, will be tested as dependent variables in the models.

5.2 Population data

Population data exists both on city and county level (divided into rural/urban), also broken down in age groups (which makes the IMR possible to calculate). Both mortality and population data comes from the official SCB publications (SCB 1885).

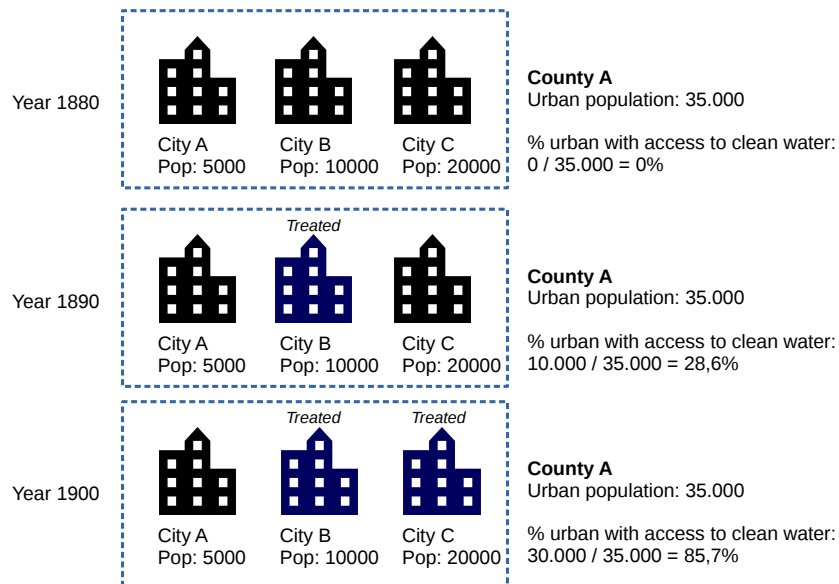
5.3 Independent variable

The independent variable used in this study is, as mentioned, a newly digitised and unique source of historic information on clean water technologies in all Swedish cities (SWEP 1964). The coverage, both in time and completeness, is rare as historical datasets are concerned. The dataset contains timing, quality, characteristics

of most of the water- and sanitation-related systems that gradually were built in Swedish cities as urbanisation gained momentum during the 19th century: water pipes, sewage systems, purification methods and food quality are central parts of the dataset. This study is a first look into the dynamics and effects that all these urban improvements had on life in the cities, and it is the clean water-aspect that is investigated here. From the dataset, timing information (year of implementation) of piped water and sewage is the data being used.

The independent variable for the models has been aggregated and reconstructed as a continuous share of the population with access to piped water (and sewage). The reason for this is the source data design: since it is a city that is treated with access to clean water / sewage, but the mortality which it is supposedly affecting is on county level, there is no way of knowing how much of the mortality that comes from each city. An illustrative example how the independent variables have been calculated can be seen in Figure 2. In the figure, county A consists of three cities (A,B,C) with different population sizes. County A is depicted in three different years (1880, 1890, 1900), and in this example, the population in the cities does not change (in reality, population will of course change over time). In 1880, none of the cities have been treated (i.e. implemented piped water), and therefore the percentage urban population with access to clean water is 0. By 1890, city B has implemented piped water, which gives a county-level urban population access to clean water of 28,6%. By 1900, both city B and C have implemented piped water, which elevates the county-level urban population with access to clean water to 85,7%. This solution is not optimal from a modelling standpoint (having mortality on a city level, where the treatment is implemented, would be preferable), but is a necessity in order to model using this data.

Figure 2: Creation of a continuous independent variable



5.3.1 Data on city level

Only the cities that are present during the whole study period are allowed in the calculation of the percentage stated in 5.3. The reason for this is also connected to the raw data: until a city was given city privileges by the Swedish state, it was not present in population statistics. This means that some cities emerge in data halfway into the study period - which gives a left truncation that makes it impossible to use in the panel. There may be bias introduced by this, but it should in theory be limited: the cities that are given privileges are often very small (relative to already present cities) - this means that the impact these cities' mortality could have on a county level should be minimal.

6 Methods

6.1 Model design

In the most basic setting, one can try to find the effect of clean water technologies on mortality in a basic OLS regression. In this case it would look like:

$$Y_{it} = \alpha + \beta T_{it} + \epsilon_{it}$$

Where Y_{it} is the outcome variable (mortality), T_{it} is the independent variable of interest (share of people in county with access to clean water technology), and ϵ_{it} is the error term (the result of this kind of model in this setting can be seen in section 7.2.1). In this simple case, the effect measured is the general effect on a national level - and it leaves many questions unanswered: how do we know that the effect of people getting access to clean water does not correlate with them getting, for example, better access to food? And if it does, how do we isolate the effect of access to clean water? With this model, that is not possible - since we only measure a general effect of a continuous variable (access to clean water) on an aggregate (national) level.

To get closer to a causal effect, the model can be refined further by defining a treatment variable that is binary - a threshold would indicate that a county is "treated" with access to clean water when the majority (more than 50%) of its urban population has access (more on the problems with such an assumption can be found in section 7.4.1). By doing this, and knowing that not all counties were treated at the start of the analysis period, we will have both treated and untreated counties at different points in time.

6.2 The potential outcomes framework

As described by Angrist and Pischke (2008, p.9), the most credible and intuitively understandable research projects often involve random assignment of some kind. For example, the randomised controlled trial procedure used in medical experiments (where the control group gets a placebo treatment) is a golden standard that has become an ideal also in social sciences. For many reasons, conducting an experiment in social science is often very hard, unethical or very costly. This is why, in most cases, the option is to try and mimic an experiment using quasi-experimental design.

6.2.1 The problem of simple comparison

Going back to the mentioned binary treatment defined above (with a 50 % threshold), this can be formalised as follows: let the variable $D_i = \{0, 1\}$ denote treated / non-treated and Y_i the outcome variable (mortality). The potential outcomes framework is a way of formalising different aspects of an experimental situation, and helps to understand the problems in establishing a causal effect (Angrist and Pischke 2008, p.27). In this case, the example of two counties can be used: county A did not receive treatment (i.e. did not give its urban citizens access to clean water during the study period), but county B did. An initial idea in this situation would be to simply compare the mortality at the end of the study period between county A and B, since only one of them were treated. Formally, the effect would be calculated by: $Y_{1i} - Y_{0i}$ which is the mortality outcome as treated minus the mortality outcome as not treated - the difference is by definition due to the treatment. The problem with this is that we cannot be sure that the counties are comparable, and that what happened with mortality in county A (which was not treated) is the same that would have happened in county B, had it not been treated. The counties may differ in various ways (demographically, politically, economically) that makes the assumption of having county A and a counterfactual "what-if" for county B not a viable assumption. Formally, this is expressed as:

$$(Y_{0i}|D_i = 1) \neq (Y_{0i}|D_i = 0)$$

The expression is read as: the potential outcome as not treated for the county that was treated (which is unobservable, since the county was in fact treated) is not the same as the potential outcome as not treated for the county that was not treated (which is observable). This means that as the model stands, it cannot be used to calculate the causal effect, since the two counties cannot be used as each other's counterfactuals. This is often known as the problem of self-selection, but is basically the same as the problem of not having all available control variables (omitted variable bias) (ibid., p.12).

6.2.2 Solution to the self-selection problem

If the treated and untreated counties can not be compared because of self-selection / missing control variables, a theoretical solution is if one could (as mentioned above), randomly assign treatment. If treatment is randomly assigned in an experiment, the potential outcomes between treated and not treated are no longer (in theory) different, and thereby the effect on treatment and control groups can be compared to find the causal effect (ibid., p.17). As mentioned, an experimental setting is out of the question in this analysis, so a quasi-experimental method (such as the difference-in-differences method) is the only option.

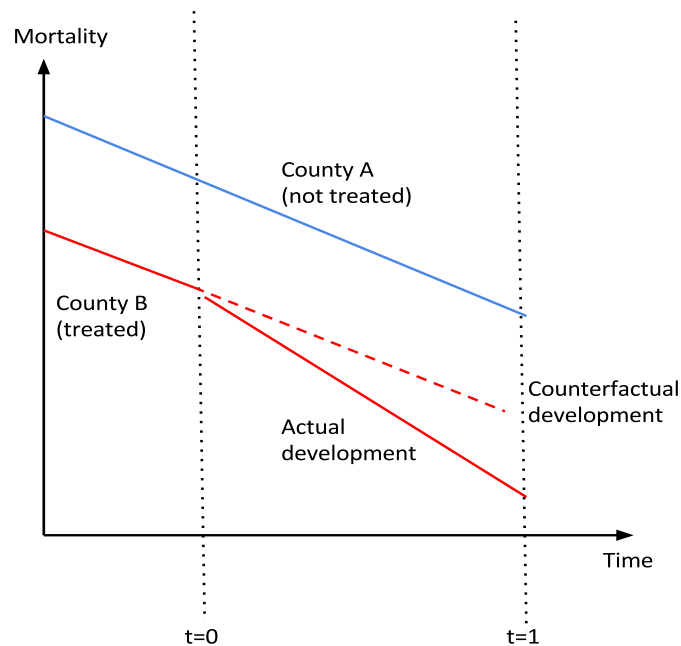
6.3 Difference-in-differences

When analysing panel data on an aggregate level, such as the county level used in this study, the difference-in-differences method is often used. Instead of measuring the difference within a single county at two points in time (within-estimation) or simply comparing the treatment and control group at one point in time (between-subject treatment effect), the difference-in-differences method uses two study units

and at least two points in time to compare. By first measuring the difference within treatment and control group over time, these two differences are then compared to each other (Angrist and Pischke 2008, p.169). The idea behind the design is to get rid of bias that arises when comparing two counties to each other: by using the aggregate level in different points in time, factors that do not vary over time in the county can be disregarded. This means, if certain assumptions hold, that only the effect of the treatment is left. In the case of an epidemiological analysis such as this study, these time-invariant effects on a county level could for example be health practices, climate and institutions (D. Cutler and Miller 2005, p.8).

The difference-in-differences method can be illustrated as in Figure 3. In this figure, county A (as exemplified earlier) is not treated during the study period (between times $t=0$ and $t=1$), and is represented by the blue line. The red lines are county B, which was treated with access to clean water sometime between $t=0$ and $t=1$. The solid red line is the actual (observed) mortality development during and after treatment (which suggests that, in this example, treatment had a negative effect on mortality) - and the dotted line represents the development we assume would have taken place, had county B not been treated with access to clean water.

Figure 3: Illustration of the difference-in-differences function



An important part of the difference-in-differences framework is that it is not the absolute values in mortality that are being compared between county A and B, but the *trend* in mortality.

6.4 Assumptions

Econometrician Joshua Angrist noted, concerning the difference-in-differences method, that: "The difference-in-differences method lives and dies with the parallel trend assumption" (Econtalk.org 2014), and this is probably the best way to describe the potential weaknesses of this method. As noted above, the difference-in-differences

method is based on finding an untreated counterpart for each treated unit, and using the untreated unit as a counterfactual for the treated unit. In order for this to be valid, one must assume that the trend in the dependent variable (in this case, mortality) in the treated county, had it not been treated, would have been the same as the observed trend in the untreated county. This is known as the parallel trend assumption, which is an identifying assumption that by definition cannot be tested (Friedman 2013). Often, this assumption is investigated by comparing pre-treatment trends in both treated and control group, and trends that do not differ in the two groups is of course the more appealing alternative (Mora Villarrubia and Reggio 2012, p.2).

The use of difference-in-differences on an aggregate level (such as the county level in this analysis) is usually due to the variable of interest having its variation locked to this level. A classical implementation is the comparison of state policies in health or labour, where different laws and regulations can be exploited (Angrist and Pischke 2008, p.169). If the aggregate level is where the variable of interest variates, then this is also where the omitted variable bias lies - therefore this is the level to get rid of it (ibid., p.169). In this case, however, the county level is more a necessity because of the structure of data (this issue is discussed in 7.4).

6.4.1 Exogeneity

A potential issue with comparing counties based on their timing of treatment is the possibility that a county may be more prone to adopt clean water technology because of a recent spike in mortality. A completed treatment that comes just after a receding mortality spike could make it look like the treatment is working, when we are in reality observing a general downward trend in mortality. This issue has also been noticed by D. Cutler and Miller (2005, p.10), who argue that, due to the general political inertia and debate on when and how to introduce clean water technologies in cities, the timing can be argued to be exogenous and thereby not interfering with causality. A similar argument can be used in this setting, and is needed in order to make sure that treatment does not vary with endogenous variables.

7 Results

In this section, the results drawn from gathering and modelling of source data is presented. First, the data is presented in descriptive form, and later, the results from statistical modelling are presented. All modelling and calculations have been done using R statistical software (R 2008).

7.1 Descriptive data

7.1.1 Dependent variables

A look at the distribution of IMR and CDR, broken down into years and counties, can be seen in Figures 4, 5 and 6. Beginning with the yearly distribution, a general decline can be seen both in IMR and CDR, with the spread (blue lines represent 95% confidence intervals) being fairly even in both variables. The heterogeneity is bigger in earlier years, and becomes more homogeneous as time goes by. This development goes well in line with the general mortality decline during this period discussed in section 3.1.

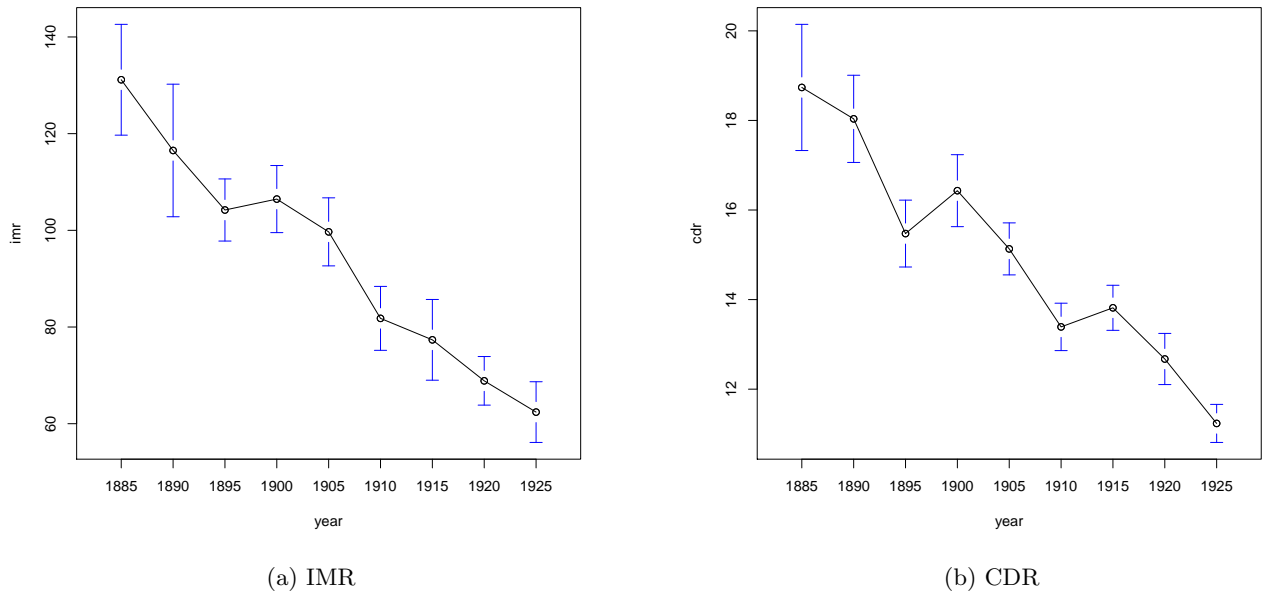


Figure 4: Heterogeneity in mortality across years

The heterogeneity across counties has a fuzzier distribution. The IMR conditional means (Figure 5) spread from under 70 (Värmland county) to over 110 (Norrbotten county), and the confidence intervals are large. The CDR distribution (Figure 6) looks similarly uneven. This indicates a large variation between and within counties, and although the general time trend seems smooth, this might prove to be a challenge when it comes to capturing this variance in a model.

7.1.2 Disease-specific mortality

The disease-specific mortality, as presented in section 5.1.2, has a less general and more diverse distribution than the previous mortality variables. The distribution for cholera and typhoid mortality over time (in urban areas) can be seen in Figure 7. A boxplot, such as Figure 7, is read as follows: the middle line is the median value, the top and bottom of the box is the upper (75%) and lower (25%) quartile (which means that 75% of the values are within that range). The whiskers mark the maximum and minimum values, excluding the outliers (values more/less than 3/2 times of the upper/lower quartiles) - and the outliers are marked by dots. In Figure 7, it can be seen that typhoid seems to become less and less common over time (with some extreme outliers), and cholera peaks around 1905 and then subsides again. Cholera had been raging in Sweden in epidemic forms on and off during the 19th century, but the last violent outbreak came in 1866, which is before this analysis period (PHAS 2005, p.47). It is mentioned by PHAS, that even after these worst outbreaks, local outbreaks (especially in urban areas) were not uncommon, even though they did not reach the levels of the mid-19th century (*ibid.*, p.47). An interpretation of what is seen in the cholera distribution is: a minor surge that shows that there was not full control over epidemics even in the start of the 20th century. The typhoid distribution shows that this disease had a rapid decline in Sweden from the 1870s and onwards, which is confirmed by Edvinsson (1992, p.150).

The other two disease distributions can be seen in Figure 8. The dysentery

Figure 5: IMR heterogeneity across counties

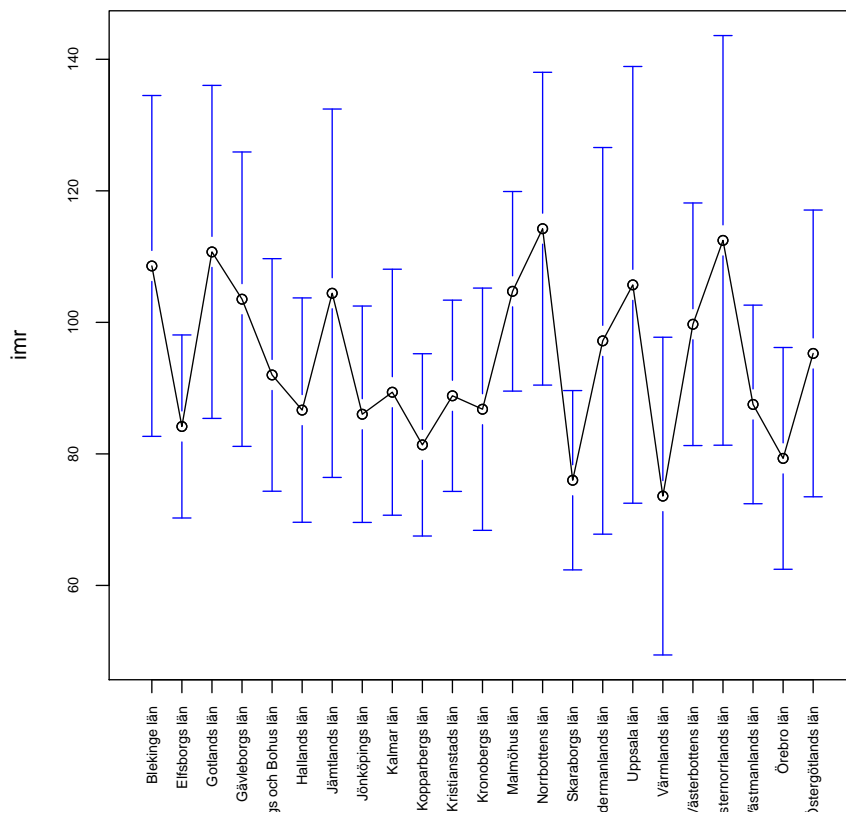
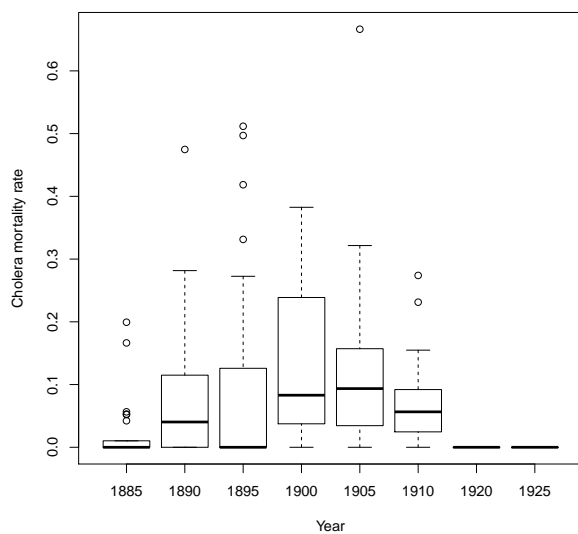
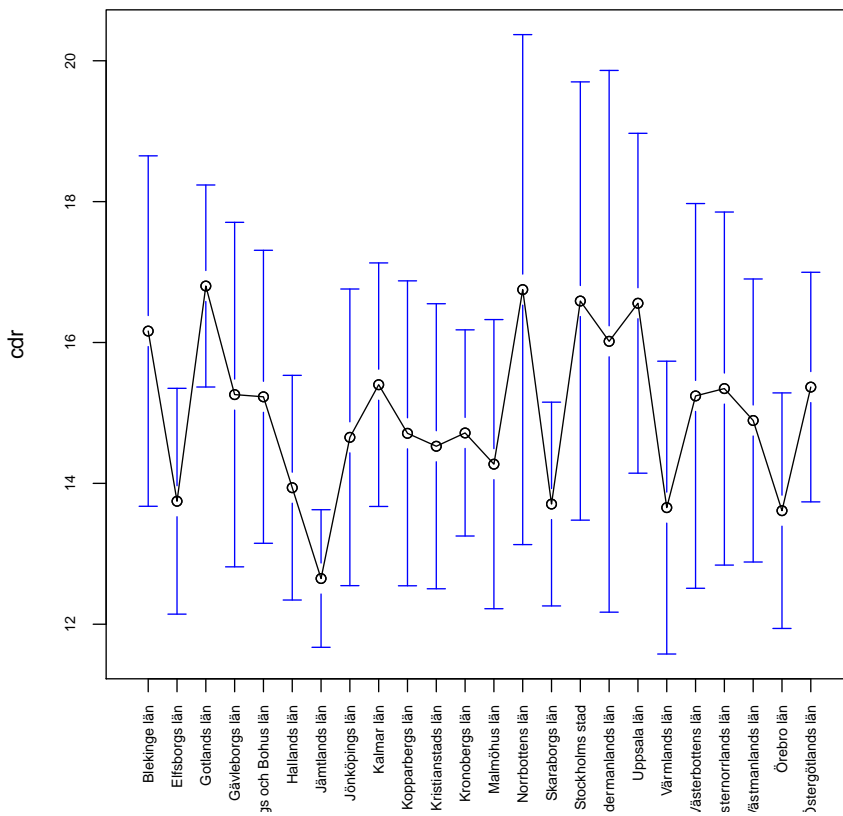
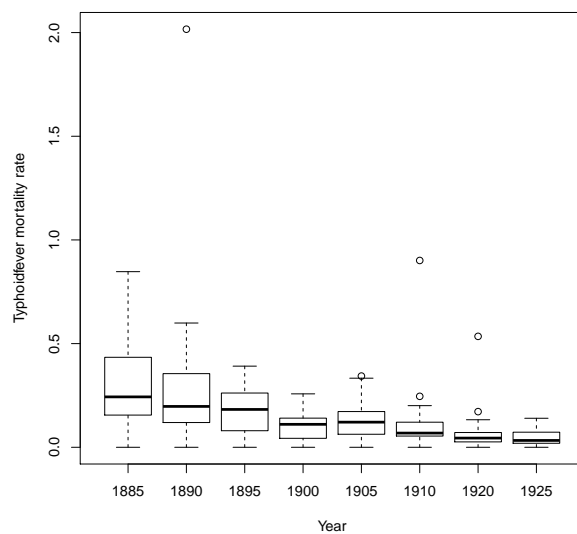


Figure 6: CDR heterogeneity across counties



(a) Cholera mortality rate over time



(b) Typhoid mortality rate over time

Figure 7: Disease-specific mortality rates

distribution, as seen, is almost not present during this study period, apart from some outliers. This, as with typhoid, follows a trend of this disease that is also mentioned by Edvinsson (1992, p.150). The distribution of gastroenteritis is one of general decline, as with the CDR/IMR rates. Worth noting is the magnitude of the rates: gastroenteritis has an average rate of around 1/1000 in the beginning of the period, while cholera and typhoid are well below 0.5/1000 (comparing with CDR, which starts at around 17/1000 in 1885).

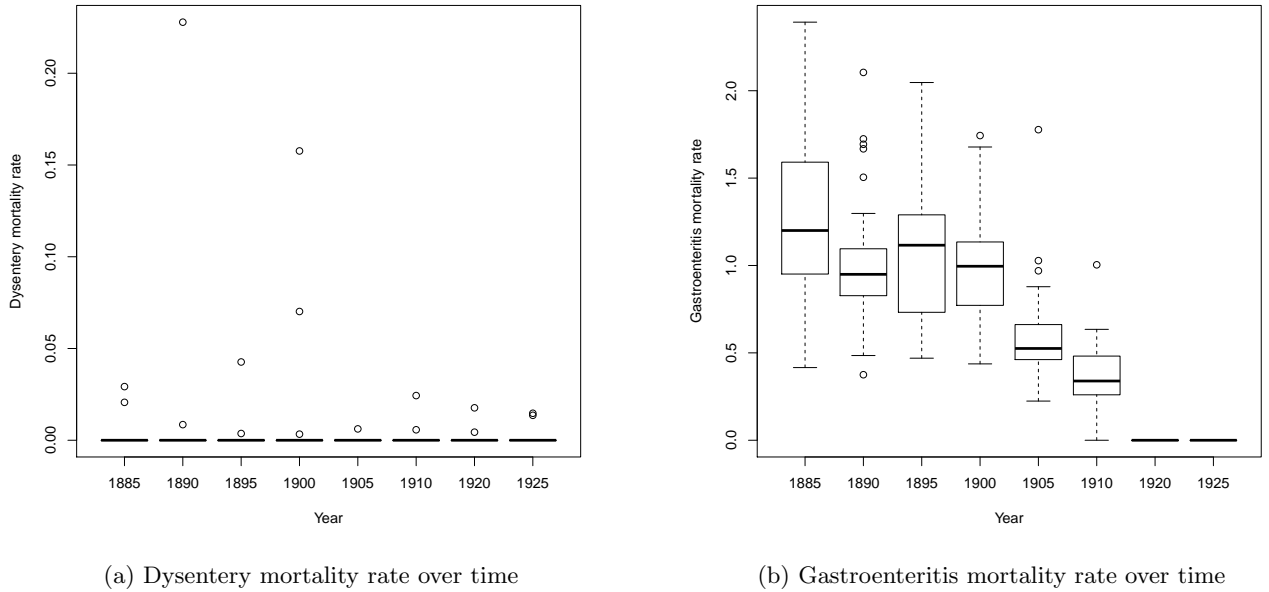


Figure 8: Disease-specific mortality rates

The overall water-borne mortality rate (all four diseases) over time can be seen in Figure 9. As shown above, a large part of this combined variable consists of gastroenteritis mortality, and it also follows the same pattern of general decline.

7.1.3 Time of implementation of clean water technology

A cumulative graph of the cities and their technology implementation times can be seen in Figure 10. It shows that up until the start of the 20th century, the majority of cities had not constructed water or sewage systems, but that the development rapidly spread after this. It is also noteworthy that the construction of water and sewage systems seem to more or less follow each other: constructing only one of them would not be a complete solution.

7.1.4 The urban penalty

It is well known that urban mortality in the 19th century (and good parts of the 20th century) in developed countries was considerably higher than rural mortality (which is sometimes referred to as the "urban penalty"). Although not used other than descriptively in this analysis, mortality data for the analysis period is also displayed for rural areas. In order to show the difference in mortality at the time, the distribution of IMR and CDR on rural / urban areas over time in the current dataset can be seen in Figures 11 and 12. In general, both averages and distributions

Figure 9: Mortality in water-borne diseases in total over time

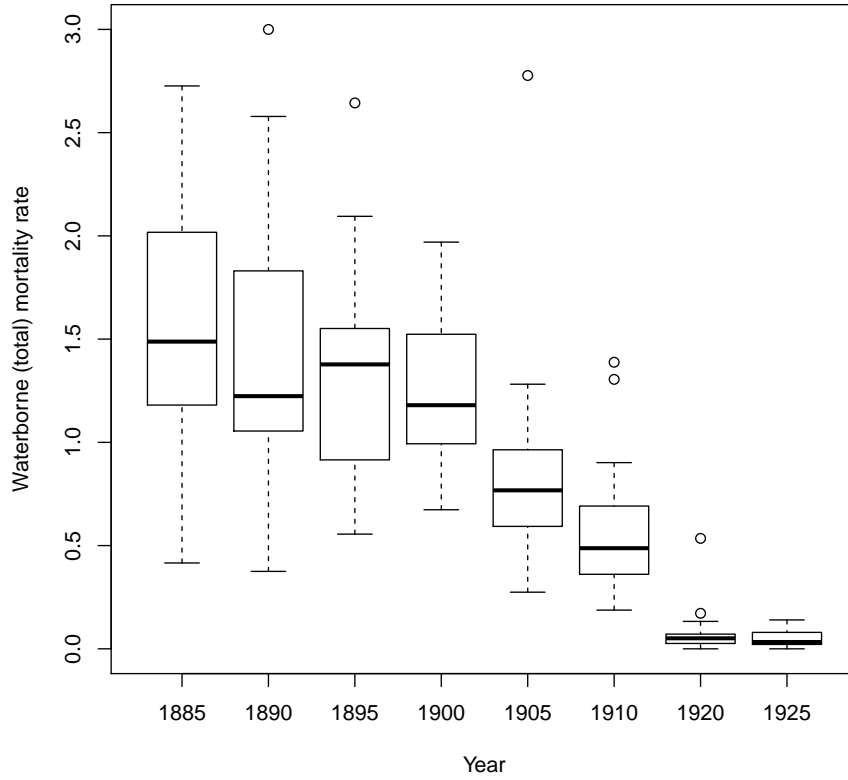
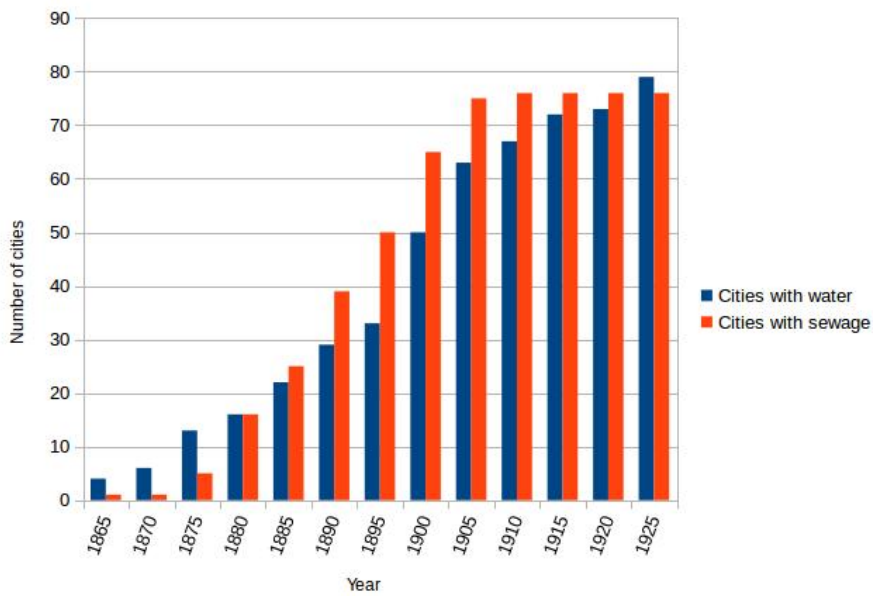
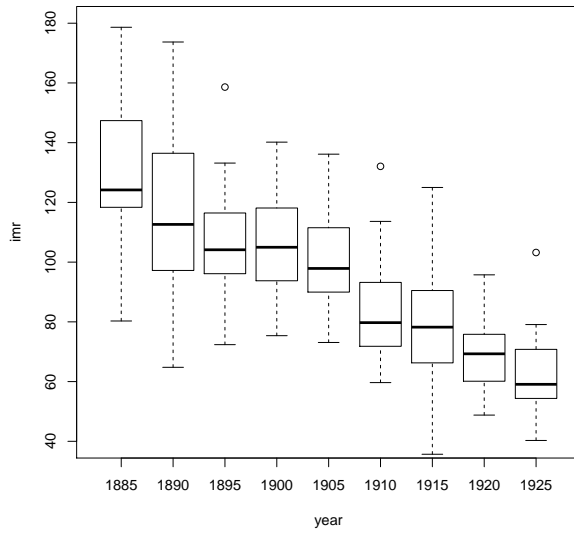


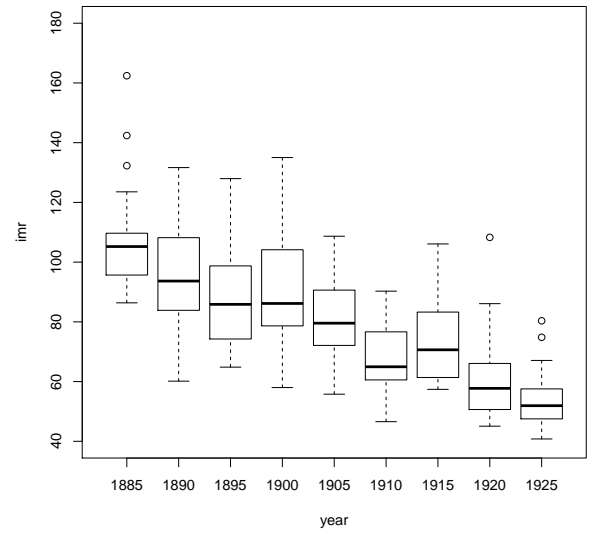
Figure 10: Cumulative number of cities that implemented piped water/sewage



go higher in urban areas, both for CDR and IMR. IMR in particular is a lot lower in rural areas, and this has been linked specifically to hygiene connected to drinking water in a time when breastfeeding was not common (Brändström 1984).

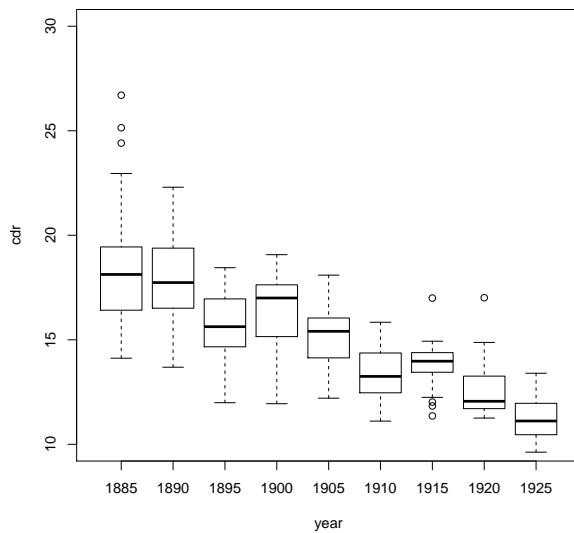


(a) IMR, urban areas

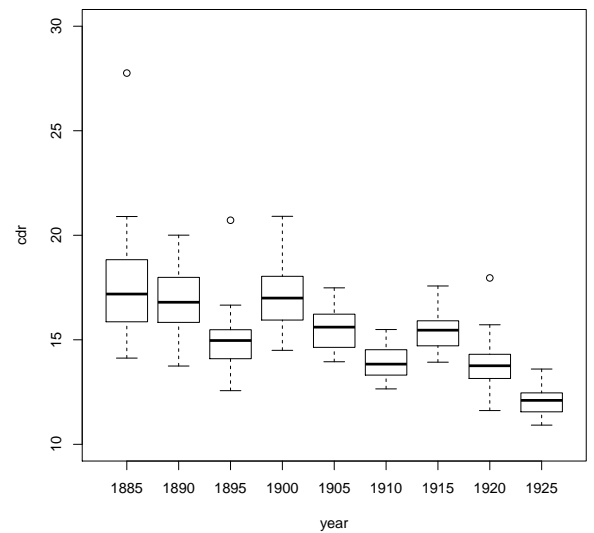


(b) IMR, rural areas

Figure 11: IMR in urban/rural areas over time



(a) CDR, urban areas



(b) CDR, rural areas

Figure 12: CDR in urban/rural areas over time

7.1.5 Independent variables

The continuous variables presented in section 5.3 are computed per county (examples of these variable distributions per county can be seen in Figures 18, 19 and 20). The distribution of these variables per year can be seen in Figures 13 (water) and 14 (sewage). The "water access" variable has a low average county coverage to begin with, and slowly makes it way up to a high average in the beginning of the 20th century. Still, the spread continues to hang on until 1910, but with a very high average coverage. An interpretation of this is that if there is any variance to exploit in this independent variable, it is better used sooner than later in the analysis period. When approaching the 20th century, most of the counties already have high coverage of piped water: if we are not using lagged effects, we need to make use of the differences before they disappear. The "sewage coverage" variable is even steeper in its rising coverage, and by 1910, all included counties have a complete sewage coverage (complete, as in that cities have been noted to have implemented - whether it is a de facto 100% coverage for all citizens is not certain). Again, this means that without extensive use of lagged variables, the variation needs to be exploited early in the analysis period.

Figure 13: % urban population with access to water, distribution per year

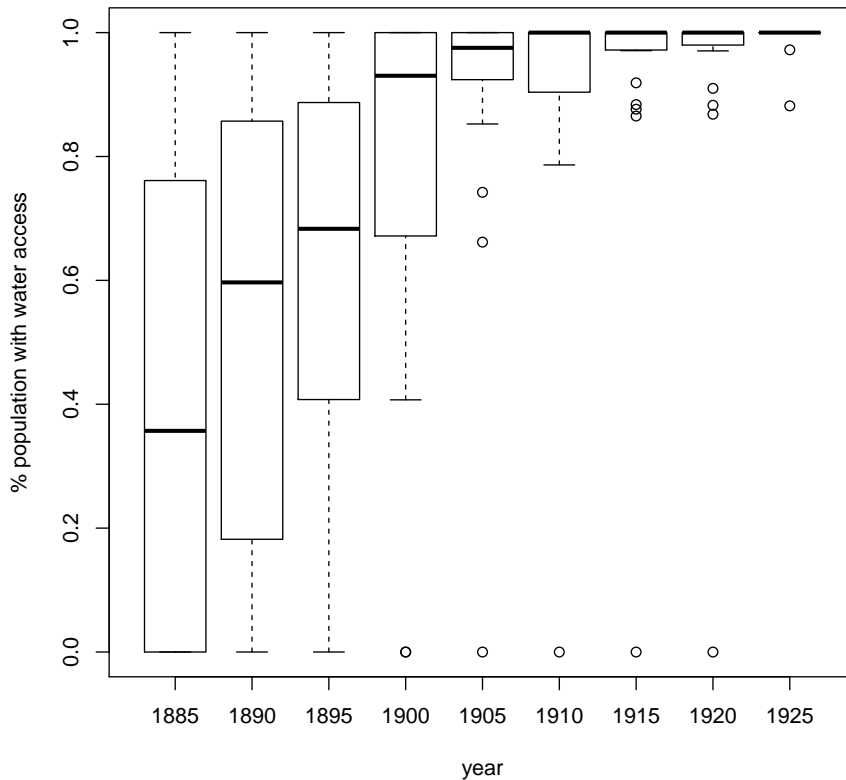
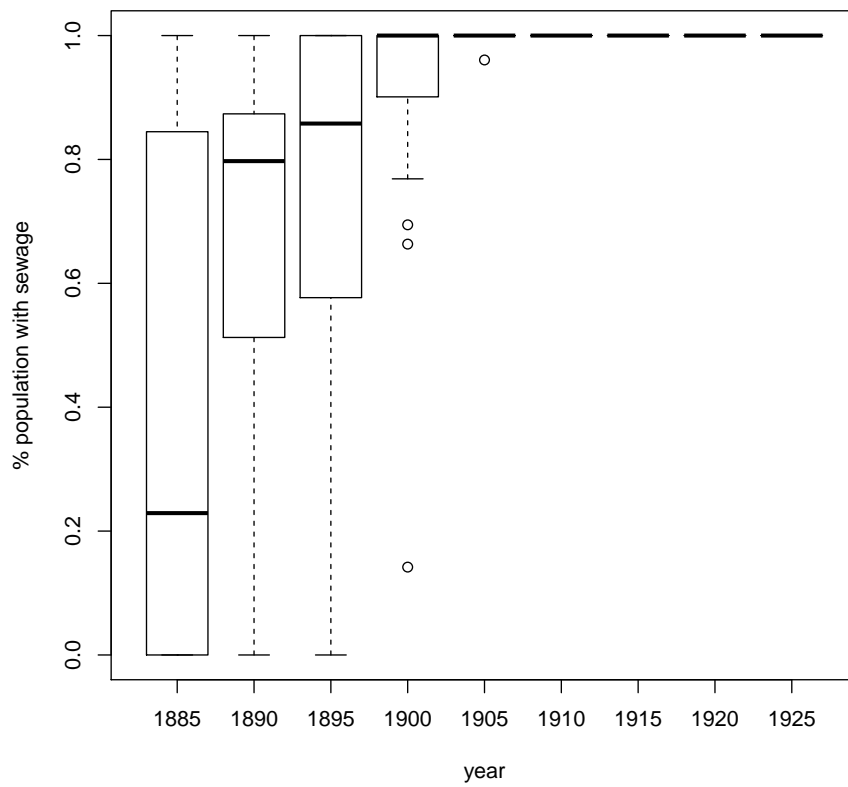


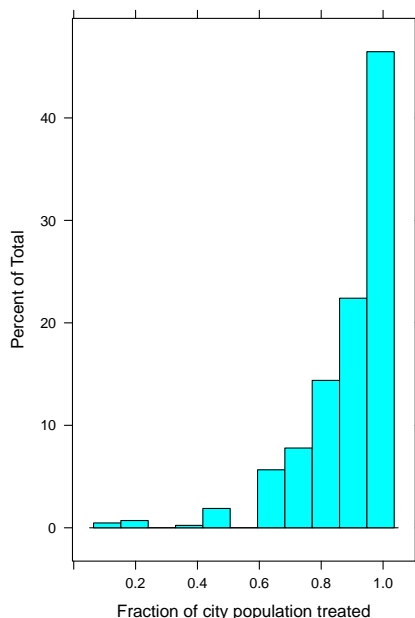
Figure 14: % urban population with access to sewage, distribution per year



7.1.6 Population in treatment

The data on how large a portion of the city population that had actual access to the clean water technology that has been implemented (indicated by a year of implementation in raw data) is not present in most of the cities' data posts (this issue is discussed further in section 7.4.2). By judging from the years when this data is available, it was not collected until late 1920 and onwards (which creates a skew in itself, since treatment population probably increases following a non-complete implementation). It is, however, present for some data posts, and the distribution of "treatment population" as a fraction of total city population can be seen in Figure 15. In this figure, we see that over 80% of the city observations that include a "treatment population" have over 80% of its citizens covered with access to piped water. The validity of this bias estimation can be compromised, as mentioned, by the fact that these observations arrive later in time - so the exact extent of this bias remains unknown.

Figure 15: Fraction of city population actually treated with access to piped water

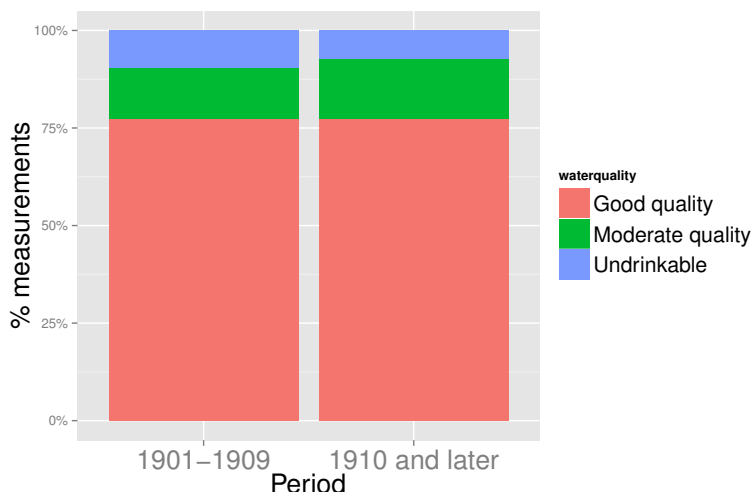


7.1.7 Water quality

Although not part of the model as a control variable (due to insufficient coverage), water quality data has been collected from 1901 and onwards, and is here used to illustrate what effect the clean water technologies might have had on the measured water quality. The measurement seem to be sporadic in cities, and starts just after 1900 (when around 50% of the cities already had installed piped water and sewage, see Figure 10). A difference is made before and after 1910, and the results can be seen in Figure 16. The original data consists of 15 different quality categories that have been aggregated to three, and as seen in the figure, there is very little difference between the two periods (it should be noted that the last observation in this variable is from 1939, so later historic measurements do not influence too much).

It is interesting to see that the portion of "Undrinkable" water measurements is just marginally lower in the later period - this can lead to the interpretation that the positive effect of implementing the technologies on the actual water quality was relatively slow (examples of these slow implementation processes and failures have been mentioned in section 4.2). There could of course be measurement irregularities affecting the result, but in general, they point in the direction of slow effects on water quality and/or confounding factors.

Figure 16: Water quality measurement distribution - aggregated count of unique city measurements



As a note, it can be mentioned that the poor water quality in Stockholm poked fun at in Figure 1 from 1866 can neither be verified nor ruled out: the first water quality measurement for Stockholm in this dataset is from 1901 - and this year, the quality was given the highest grade.

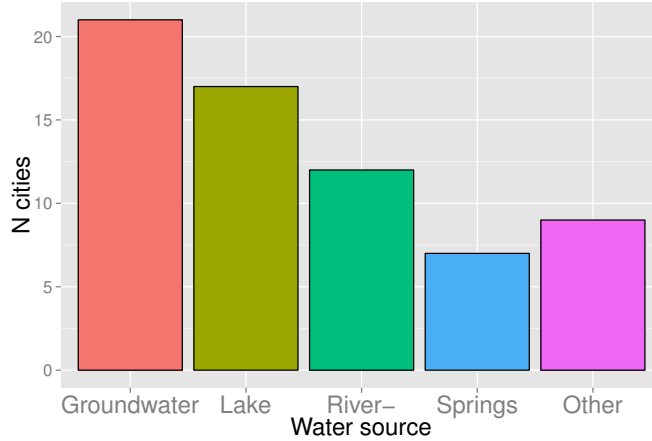
7.1.8 Water sources

Another descriptive variable that might be interesting in this context is the source from which water is taken, both before and after piped water has been introduced. Apart from the five categories in Figure 17, some less used water sources (such as ponds and caves) have been collected in "Other". As seen in the figure, groundwater is the most common source, followed by natural water surfaces such as lakes, rivers, and springs. When it comes to sensitivity to auto-contamination, groundwater has frequently been mentioned as being contaminated by run-off and surface water (see section 3.3) - an interpretation of this might be that high usage of groundwater as source leads to greater risk of contaminated drinking water. If this is the case in cities with piped water, it will introduce a confounding factor in the causal chain from clean water technology to lower mortality.

7.2 OLS model output

In all models run, both CDR and IMR have been tested as dependent variable, and all combinations of with and without "percentage population with water/sewage access" have been tried. In general, IMR is the more interesting mortality measure in this setting (see section 5.1.1), so in some output tables only IMR is reported,

Figure 17: Water source distribution - count of unique city observations



unless the CDR result not was of particular interest. The disease-specific mortality data is also tested in its own model. It should be noted that counties that have 100% coverage of water/sewage for all of the analysis period have been excluded from the models. Outputs from the different models will be interpreted and commented in the sections below.

7.2.1 Model 1: simple OLS

The first model (Models 1-4 in Table 1) is a simple OLS regression without any controls. The model is specified as:

$$Y_{IMR} = \alpha + \beta_1 W_{it} + \beta_2 S_{it} + \epsilon_{it}$$

Where Y_{IMR} is the outcome mortality variable (IMR), α is the intercept, $\beta_1 W_{it}$ is the independent variable ”% with access to clean water”, $\beta_2 S_{it}$ is the independent variable ”% with access to sewage” and ϵ_{it} is the error term.

The results indicate, at this simplified level, that a growing percentage of urban population with access to water and/or sewage has a strong and statistically significant negative effect on both IMR and CDR. According to output from the first model (column 1), for example, a 10 unit increase in the percentage (which equals a 10% increase) of urban population with access to piped water yields: $\exp(-0.0041274 * 10) = 0.9595662$, which means a 4% decrease in IMR. Similarly, 50% increase in clean water access would give a 19% decrease in IMR ($1 - \exp(-0.0041274 * 50) = 0.186$). The effect on CDR from water access (column 2) is somewhat smaller than the one on IMR. The effects from sewage access (columns 3 and 4) are very similar to the effects from water access. Judging by the two final models (columns 5 and 6), this is due to sewage access co-varying with water access, since much of the effect from sewage (and its significance) disappears when both independent variables are included in the model.

The effects from these models are, since no county dummies are included, to be interpreted on a national level - and one interpretation is that this shows how complicated effects on mortality are to measure. Obviously, there is a huge omitted variable bias in this model (since there are no external controls), and the statistical significance does not give any reassurance that these independent variables would be the only thing affecting mortality in 19th century Sweden. Of course, there are

	log(IMR)	log(CDR)	log(IMR)	log(CDR)	log(IMR)	log(CDR)
Intercept	4.806 (0.048)***	2.914 (0.028)***	4.867 (0.060)***	2.943 (0.036)***	4.860 (0.059)***	2.937 (0.034)***
% water	-0.004 (0.001)***	-0.003 (0.000)***			-0.003 (0.001)**	-0.003 (0.001)***
% sewage			-0.004 (0.001)***	-0.003 (0.000)***	-0.002 (0.001)	-0.001 (0.001)
R ²	0.207	0.298	0.196	0.255	0.228	0.333
Adj. R ²	0.203	0.295	0.192	0.251	0.219	0.325
Num. obs.	198	198	180	180	180	180

*** $p < 0.001$, ** $p < 0.01$, * $p < 0.05$

Table 1: Model 1 output

many more concepts affecting mortality negatively during this period in time (some of which discussed in section 3.1), and this model indicates that access to clean water probably correlates with many of them. This makes, in theory, the effect from access to clean water harder to isolate (in absence of perfect control variables, which is something that is practically impossible).

7.2.2 Model 2: added time dimension

In model 2 (columns 1 and 2 in Table 2), factor variables for each included 5-year period have been included. The model is specified as:

$$Y_{IMR} = \alpha + \beta_1 W_{it} + \beta_2 S_{it} + \beta_3 T_{it} + \epsilon_{it}$$

Where Y_{IMR} is the outcome mortality variable (IMR), α is the intercept, $\beta_1 W_{it}$ is the independent variable "% with access to clean water", $\beta_2 S_{it}$ is the independent variable "% with access to sewage", $\beta_3 T_{it}$ is the time fixed effect and ϵ_{it} is the error term.

For IMR, both the magnitude, significance and also the direction (sewage yields a positive effect on IMR) of the effect from the independent variables disappear when time dummies are added. For CDR, there is still a small but significant negative effect from water access. The yearly dummies (using year 1885 as reference category) all have a large and significantly negative effect on both IMR and CDR, and it is hard to interpret this any other way than it being a general time trend (and thus, probably omitted variable bias). Theorists give different interpretations as to why mortality declined during this period in different parts of the world - but if we assume that is due to some more tangible concept than just "the times" themselves, there seems to be missing control variables (might be demographic, economic, social) that can account for this variance. Another interpretation could of course be that the included independent variables (water/sewage access) have not been optimally configured or collected.

7.2.3 Model 3: added county dummies

Adding a dummy for each included county makes the model account for even more of the variation (higher R^2), and makes room for larger effects from the year dummies (columns 3 and 4 in Table 2). The model is specified as:

$$Y_{IMR} = \alpha + \beta_1 W_{it} + \beta_2 S_{it} + \beta_3 T_{it} + \beta_4 C_{it} + \epsilon_{it}$$

Where Y_{IMR} is the outcome mortality variable (IMR), α is the intercept, $\beta_1 W_{it}$ is the independent variable "% with access to clean water", $\beta_2 S_{it}$ is the independent

	M2:log(IMR)	M2:log(CDR)	M3:log(IMR)	M3:log(CDR)
Intercept	4.856 (0.054)***	2.923 (0.029)***	4.970 (0.075)***	2.999 (0.041)***
% water	-0.001 (0.001)	-0.002 (0.000)***	0.002 (0.001)*	-0.001 (0.001)
% sewage	0.001 (0.001)	0.001 (0.001)	-0.001 (0.001)	0.000 (0.001)
Year 1890	-0.155 (0.069)*	-0.029 (0.037)	-0.147 (0.057)*	-0.027 (0.031)
Year 1895	-0.214 (0.072)**	-0.160 (0.039)***	-0.210 (0.060)***	-0.160 (0.033)***
Year 1900	-0.203 (0.078)*	-0.092 (0.042)*	-0.220 (0.068)**	-0.099 (0.037)**
Year 1905	-0.271 (0.083)**	-0.156 (0.044)***	-0.307 (0.073)***	-0.169 (0.040)***
Year 1910	-0.443 (0.083)***	-0.288 (0.044)***	-0.484 (0.074)***	-0.302 (0.040)***
Year 1915	-0.534 (0.083)***	-0.245 (0.044)***	-0.581 (0.074)***	-0.261 (0.041)***
Year 1920	-0.622 (0.083)***	-0.327 (0.044)***	-0.671 (0.074)***	-0.344 (0.041)***
Year 1925	-0.737 (0.083)***	-0.451 (0.045)***	-0.803 (0.075)***	-0.473 (0.041)***
Elfsborg county			-0.238 (0.081)**	-0.154 (0.045)***
Gotland county			0.128 (0.098)	0.007 (0.054)
Gävleborg county			-0.038 (0.081)	-0.060 (0.045)
Halland county			-0.220 (0.081)**	-0.138 (0.045)**
Jämtland county			-0.054 (0.082)	-0.232 (0.045)***
Jönköping county			-0.237 (0.082)**	-0.090 (0.045)*
Kalmar county			-0.160 (0.084)	-0.053 (0.046)
Kopparberg county			-0.268 (0.083)**	-0.097 (0.045)*
Kristianstad county			-0.165 (0.082)*	-0.108 (0.045)*
Malmöhus county			-0.000 (0.082)	-0.127 (0.045)**
Norrbottn county			0.083 (0.085)	0.009 (0.047)
Skaraborg county			-0.341 (0.082)***	-0.155 (0.045)***
Södermanland county			-0.145 (0.082)	-0.032 (0.045)
Uppsala county			-0.075 (0.082)	0.033 (0.045)
Värmland county			-0.451 (0.082)***	-0.170 (0.045)***
Västerbotten county			-0.051 (0.082)	-0.071 (0.045)
Västmanland county			-0.175 (0.082)*	-0.087 (0.045)
Örebro county			-0.311 (0.081)***	-0.163 (0.045)***
Östergötland county			-0.152 (0.082)	-0.034 (0.045)
R ²	0.569	0.674	0.745	0.797
Adj. R ²	0.543	0.655	0.695	0.758
Num. obs.	180	180	180	180

*** $p < 0.001$, ** $p < 0.01$, * $p < 0.05$

Table 2: Model 2 and 3 output

variable ”% with access to sewage”, $\beta_3 T_{it}$ is the time fixed effect, $\beta_4 C_{it}$ is the county fixed effect and ϵ_{it} is the error term.

The resulting county effects are diverging, some counties have positive effects on mortality (but none of these are significant) - and others have strong, significant, negative effects on mortality. With the addition of county effects, even more of the effect from our independent water/sewage variables disappear and become insignificant, and the effect from the yearly dummies becomes stronger. Also, the effect on IMR from water access actually becomes positive and significant on the 5% level, which is surprising and counter-intuitive. Värmland and Örebro counties have noteworthy individual effects on mortality that are strongly negative and significant. Same as when interpreting model 2, the results from model 3 suggest that there are other variables at play than water/sewage access. Since we are not controlling for these, the variance seems to gather in time and county dummies.

7.2.4 Model 4: added treatment dummies

For model 4 (result in Table 3), a county-specific fixed effect of treatment has been added, using the binary treatment indicator (introduced in section 6.1). The model is specified as:

$$Y_{IMR} = \alpha + \beta_1 W_{it} + \beta_2 S_{it} + \beta_3 T_{it} + \beta_4 C_{it} + \beta_5 D_{it} + \beta_6 D_{it} C_{it} + \beta_7 P_{it} + \epsilon_{it}$$

Where Y_{IMR} is the outcome mortality variable (IMR), α is the intercept, $\beta_1 W_{it}$ is the independent variable ”% with access to clean water”, $\beta_2 S_{it}$ is the independent variable ”% with access to sewage”, $\beta_3 T_{it}$ is the time fixed effect, $\beta_4 C_{it}$ is the county fixed effect, $\beta_5 D_{it}$ is the general treatment effect, $\beta_6 D_{it} C_{it}$ is the county-specific treatment interaction effect, $\beta_7 P_{it}$ is the county population variable, and ϵ_{it} is the error term.

Introducing a treatment dummy does not, compared to model 3, seem to make much of a difference. County population has been added as a control to this model, but does not give a consistent effect. The general treatment effect (”Treatment=1”) is negative for IMR, positive for CDR, but neither of them is significant. Looking closer at the county-specific treatment effects (missing counties are due to a constant treatment dummy of 1 for all of the study period - i.e. more than 50% of the urban population had water access when the analysis period started), the results are counter-intuitive. Only two counties (Gotland and Kopparberg) have a significant effect from treatment on IMR, and this turns out to be positive (i.e. more access to water yields higher IMR). For CDR, no significant county-specific treatment effects have been found.

An interpretation of these results is that the 50% treatment dummy probably might be a bit too arbitrary and simplistic to capture anything of value. A closer look at the two significant treatment counties gives some insight to this: Figure 18 shows how the ”water access”-variable and IMR variate over time in Gotland county. The significant positive effect of treatment in this county seems to stem from the last time period (1925) when the county goes from 0 to 100% in water access, at the same time as IMR makes a slight increase. Judging from the development of IMR in Gotland county, access to water did not have much to do with the general decline (it should be noted that Visby, the only city on the island of Gotland, was at the time small city, which makes it less reliable as a carrier of water-borne mortality). Figure

19 shows the corresponding numbers for Kopparberg county - here we see a sharp rise in water access, and a variation in IMR at the same time that seems unrelated. From 1900 and onwards, the county is fully treated, but IMR fluctuates, leaving a slight positive slope in average (which is seen in the aforementioned coefficient). All in all, the results from this full OLS model with county-specific treatment dummies are rather weak, and some of the effects are contrary to what was expected, based on theory and previous research.

Figure 18: % urban population with access to water and IMR, Gotland county

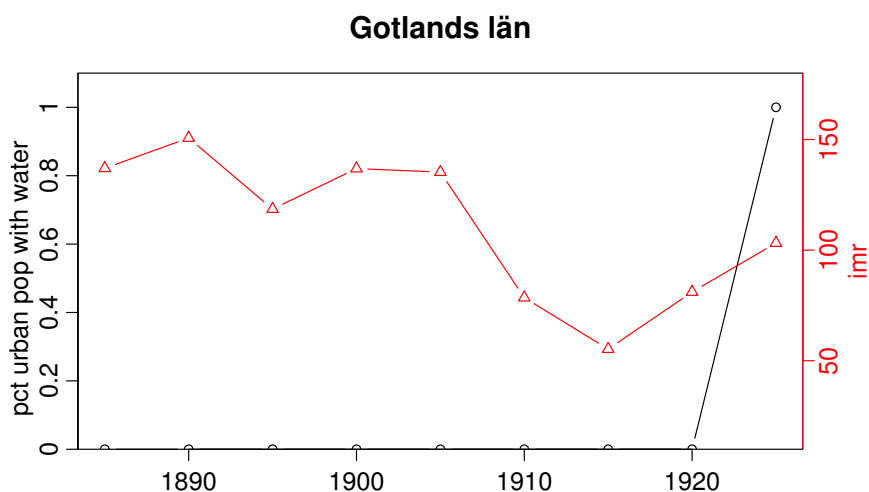
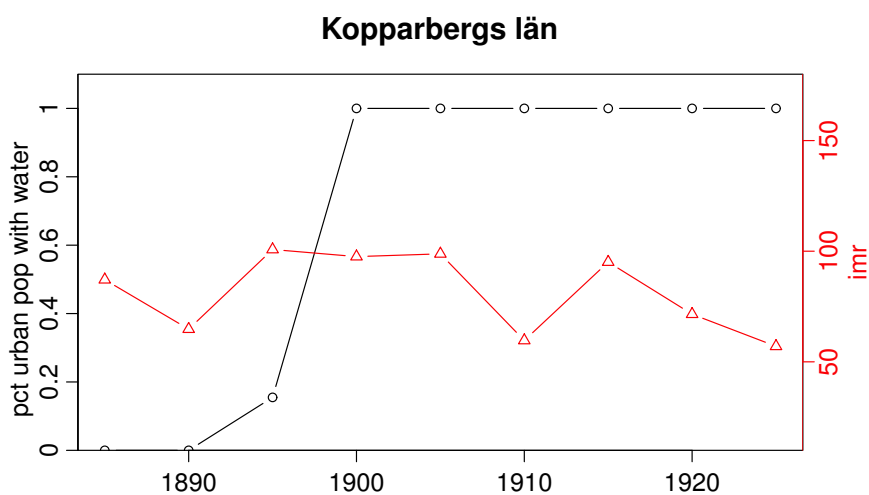


Figure 19: % urban population with access to water and IMR, Kopparberg county



	M4:log(IMR)	M4:log(CDR)
Intercept	4.866 (1.197)***	4.315 (0.623)***
% water	0.000 (0.002)	-0.002 (0.001)
% sewage	-0.001 (0.001)	0.000 (0.001)
log(population)	0.031 (0.118)	-0.127 (0.062)*
Year 1890	-0.116 (0.061)	0.001 (0.032)
Year 1895	-0.172 (0.066)*	-0.134 (0.034)***
Year 1900	-0.189 (0.081)*	-0.052 (0.042)
Year 1905	-0.263 (0.091)**	-0.100 (0.048)*
Year 1910	-0.442 (0.100)***	-0.214 (0.052)***
Year 1915	-0.538 (0.108)***	-0.158 (0.056)**
Year 1920	-0.632 (0.122)***	-0.218 (0.063)***
Year 1925	-0.780 (0.128)***	-0.340 (0.067)***
Elfsborgs län	-0.230 (0.082)**	-0.156 (0.043)***
Gotlands län	-0.122 (0.256)	-0.210 (0.133)
Gävleborgs län	-0.043 (0.086)	-0.028 (0.045)
Hallands län	-0.206 (0.089)*	-0.174 (0.046)***
Jämtlands län	-0.252 (0.291)	-0.600 (0.151)***
Jönköpings län	-0.218 (0.084)*	-0.085 (0.044)
Kalmar län	-0.323 (0.215)	-0.108 (0.112)
Kopparbergs län	-0.638 (0.241)**	-0.292 (0.126)*
Kristianstads län	-0.147 (0.120)	-0.206 (0.063)**
Malmöhus län	-0.276 (0.292)	0.081 (0.152)
Norrbottnens län	-0.125 (0.241)	-0.105 (0.126)
Skaraborgs län	-0.412 (0.263)	-0.286 (0.137)*
Södermanlands län	-0.178 (0.258)	0.161 (0.135)
Uppsala län	-0.046 (0.090)	0.016 (0.047)
Värmlands län	-0.536 (0.236)*	-0.319 (0.123)*
Västerbottens län	-0.214 (0.297)	-0.313 (0.155)*
Västmanlands län	-0.389 (0.223)	-0.122 (0.116)
Örebro län	-0.313 (0.090)***	-0.182 (0.047)***
Östergötlands län	-0.151 (0.112)	0.061 (0.058)
Treatment=1	-0.137 (0.218)	0.049 (0.114)
Gotland treatment	0.590 (0.275)*	0.046 (0.143)
Jämtland treatment	0.271 (0.233)	0.226 (0.121)
Kalmar treatment	0.117 (0.224)	0.067 (0.116)
Kopparberg treatment	0.502 (0.224)*	0.116 (0.117)
Malmöhus treatment	0.269 (0.234)	-0.020 (0.122)
Norrbotten treatment	0.243 (0.223)	-0.057 (0.116)
Skaraborg treatment	0.068 (0.263)	0.115 (0.137)
Södermanland treatment	0.022 (0.259)	-0.230 (0.135)
Värmland treatment	0.071 (0.234)	0.138 (0.122)
Västerbotten treatment	0.230 (0.225)	0.036 (0.117)
Västmanland treatment	0.221 (0.227)	0.022 (0.118)
R ²	0.768	0.834
Adj. R ²	0.696	0.783
Num. obs.	180	180

*** $p < 0.001$, ** $p < 0.01$, * $p < 0.05$

Table 3: Model 4 output

7.2.5 Model 5: disease-specific mortality as outcome

In this last OLS model, the four disease-specific mortality variables (presented in section 5.1.2) are used as dependent variables instead of the general mortality measurements CDR and IMR. The idea behind why this is preferable to general mortality is intuitive: all the mortality that is not related to water-borne diseases (and thereby can not have been caused by lack of access to clean water) is not present in this model - thereby there should be less confounding factors. This model is the same as in model 3, with time and county dummies but no treatment dummy (see Table 2). The model is specified as:

$$Y_{Disease-specific} = \alpha + \beta_1 W_{it} + \beta_2 S_{it} + \beta_3 T_{it} + \beta_4 C_{it} + \epsilon_{it}$$

Where $Y_{Disease-specific}$ is the outcome mortality variable (for the specific disease), α is the intercept, $\beta_1 W_{it}$ is the independent variable " % with access to clean water", $\beta_2 S_{it}$ is the independent variable " % with access to sewage", $\beta_3 T_{it}$ is the time fixed effect, $\beta_4 C_{it}$ is the county fixed effect and ϵ_{it} is the error term.

The output from model 5 can be seen in Table 4. To begin with, the only significant effect from our independent variables of interest are in the dysentery model - and there, the effects are very small. The effect in the dysentery model can probably be traced back to the very subtle distribution of this mortality variable (as seen in Figure 8, section 7.1.2) - if there is an effect picked up in this small distribution, it is likely to be local and/or sporadic. In the other models, the pattern of strong yearly coefficients and sporadic county coefficients being strong is similar to the previous multiple regression models. In theory, it should be easier to explain variation in disease-specific mortality than in general mortality using "access to clean water" as an independent variable (the rest of the model being equal, as is now), but the results from model 5 does not seem to support this. Apparently, putting general confounding factors and omitted variables aside (which should be the same for both general and disease-specific models), the disease-specific mortality is seemingly as hard to explain using clean water technology-variables as is the general mortality. An interpretation of this could be that the heterogeneity introduced at the aggregated (county) level has a large effect also on the disease-specific mortality: the dynamics of epidemics and transmission of disease maybe does not allow themselves to be aggregated and simplified up from the level where they normally operate (which in this case would be the city/sub-city level).

7.3 Difference-in-differences

The OLS models run so far do not give any reassurance that the difference-in-differences model should be capturing all the county-specific effects and thus showing a causal effect. This is due to the fact that the effect of the treatment variable seems to be taken over by more general variables (suggesting that we have omitted variables in the OLS models) - and there is no ground for arguing that this independent variable would earn a greater significance in another type of model.

In their classical difference-in-differences example, Card and Krueger (1993) look closer at the impact of a minimum wage increase in one state on state employment. The data they use are sub-units (restaurants and their employment outcomes) aggregated to state level, where the independent variable (state-controlled minimum wage) has its effect. In this analysis, the sub-units (cities) do not own the outcome

	Typhoid	Dysentery	Cholera	Gastroenteritis	Total waterborne
Intercept	0.273 (0.085)**	0.005 (0.008)	-0.004 (0.036)	1.305 (0.120)***	1.579 (0.161)***
% water	-0.000 (0.001)	-0.000 (0.000)**	-0.001 (0.000)	-0.002 (0.001)	-0.003 (0.002)
% sewage	0.001 (0.001)	0.000 (0.000)*	0.001 (0.000)	0.002 (0.001)	0.003 (0.002)
Year 1890	0.017 (0.060)	0.008 (0.006)	0.040 (0.025)	-0.164 (0.084)	-0.099 (0.113)
Year 1895	-0.111 (0.062)	-0.002 (0.006)	0.038 (0.026)	-0.158 (0.088)	-0.234 (0.118)*
Year 1900	-0.198 (0.070)**	0.002 (0.007)	0.084 (0.029)**	-0.136 (0.098)	-0.247 (0.131)
Year 1905	-0.182 (0.075)*	0.001 (0.007)	0.088 (0.032)**	-0.517 (0.106)***	-0.610 (0.142)***
Year 1910	-0.189 (0.075)*	0.002 (0.007)	0.043 (0.032)	-0.749 (0.107)***	-0.892 (0.142)***
Year 1920	-0.234 (0.076)**	0.003 (0.007)	-0.019 (0.032)	-1.117 (0.107)***	-1.368 (0.143)***
Year 1925	-0.257 (0.078)**	0.005 (0.007)	-0.016 (0.033)	-1.107 (0.110)***	-1.375 (0.147)***
Elfsborgs county	-0.076 (0.095)	-0.002 (0.009)	-0.011 (0.040)	-0.224 (0.134)	-0.313 (0.179)
Gävleborgs county	0.056 (0.095)	0.002 (0.009)	0.029 (0.040)	0.088 (0.135)	0.175 (0.180)
Göteborgs och Bohus county	-0.068 (0.096)	0.002 (0.009)	-0.004 (0.041)	-0.118 (0.136)	-0.188 (0.181)
Gotlands county	0.118 (0.109)	-0.017 (0.010)	0.102 (0.046)*	0.066 (0.154)	0.269 (0.206)
Hallands county	0.241 (0.095)*	-0.002 (0.009)	0.021 (0.040)	-0.296 (0.134)*	-0.036 (0.179)
Jämtlands county	-0.121 (0.096)	-0.002 (0.009)	0.006 (0.041)	-0.161 (0.135)	-0.278 (0.180)
Jönköpings county	0.008 (0.095)	0.001 (0.009)	0.005 (0.040)	-0.352 (0.135)*	-0.338 (0.180)
Kalmar county	0.025 (0.098)	-0.007 (0.009)	-0.017 (0.042)	-0.266 (0.138)	-0.265 (0.185)
Kopparbergs county	-0.048 (0.097)	-0.003 (0.009)	0.010 (0.041)	-0.381 (0.137)**	-0.421 (0.182)*
Kristianstads county	-0.037 (0.095)	-0.005 (0.009)	-0.010 (0.040)	-0.174 (0.135)	-0.226 (0.180)
Kronobergs county	-0.049 (0.095)	-0.004 (0.009)	-0.018 (0.040)	-0.297 (0.135)*	-0.368 (0.180)*
Malmöhus county	-0.007 (0.095)	-0.002 (0.009)	-0.017 (0.040)	-0.050 (0.135)	-0.075 (0.180)
Norrbottnens county	-0.042 (0.099)	-0.005 (0.009)	-0.000 (0.042)	-0.022 (0.141)	-0.069 (0.188)
Örebro county	-0.095 (0.095)	-0.002 (0.009)	-0.009 (0.040)	-0.271 (0.134)*	-0.377 (0.179)*
Östergötlands county	-0.019 (0.096)	-0.001 (0.009)	0.143 (0.041)***	-0.115 (0.135)	0.008 (0.180)
Skaraborgs county	-0.031 (0.095)	-0.002 (0.009)	-0.024 (0.040)	-0.413 (0.135)**	-0.470 (0.180)**
Södermanlands county	0.051 (0.095)	-0.002 (0.009)	0.038 (0.040)	-0.110 (0.135)	-0.023 (0.180)
Uppsala county	0.001 (0.096)	-0.001 (0.009)	0.000 (0.041)	-0.028 (0.135)	-0.027 (0.180)
Värmlands county	-0.044 (0.096)	-0.002 (0.009)	-0.003 (0.041)	-0.198 (0.136)	-0.247 (0.181)
Västerbottnens county	-0.103 (0.096)	0.022 (0.009)*	0.032 (0.041)	-0.205 (0.135)	-0.254 (0.181)
Västmanlands county	-0.058 (0.096)	0.003 (0.009)	0.093 (0.041)*	-0.215 (0.136)	-0.177 (0.181)
R ²	0.322	0.197	0.411	0.778	0.751
Adj. R ²	0.182	0.031	0.289	0.732	0.699
Num. obs.	176	176	176	176	176

*** $p < 0.001$, ** $p < 0.01$, * $p < 0.05$

Table 4: Model 5 output

variable (mortality), so a pre-post scenario of treatment boils down to four data points (for reference, see Figure 3) that can be subtracted from one another to find the differences (mortality after treatment period minus mortality before treatment period), and then the difference in differences (treated minus non-treated trend). This makes a regression difference-in-differences model less needed than in the case of Card and Krueger (1993) (where conditional means over all restaurants are needed to find the average effect). Here, a regression models will still be run, using the following setup. The pre-post differences can be calculated by:

$$DiD = (\bar{Y}_{post}^T - \bar{Y}_{pre}^T) - (\bar{Y}_{post}^C - \bar{Y}_{pre}^C) = \Delta\bar{Y}^T - \Delta\bar{Y}^C$$

Where \bar{Y}^T is the sample mean for the outcome variable in the treated county (and with the C superscript for control county). In a regression setup, the difference-in-differences estimator can then be obtained by:

$$DiD = \alpha + \beta_1 D_i$$

Where $\beta_1 D_i$ is a treatment dummy indicator (and the input data consists of $\Delta\bar{Y}^T$ and $\Delta\bar{Y}^C$. As mentioned, in this example, the effect might as well be calculated by hand (since the sample means at aggregate level are already given).

7.3.1 Kronoberg and Kalmar counties

The first difference-in-differences example is between Kronoberg and Kalmar counties. They have been chosen as each other's counterfactuals because they are adjacent and not too far away from each other in population size. Kalmar county did not reach the treatment threshold until 1905, which means that the period between 1885 and 1900 was untreated - Kronoberg, however, reached the treatment threshold already in 1890 - so by looking at the period 1885 - 1900, a diff-in-diff can be calculated. The counties' development in dependent and independent variables can be seen in Figure 20, where we can see the treatment variable progression. The general slope of the IMR variable also looks similar, and unfortunately, it is not possible to investigate the parallel trend assumption any further back than this (since data is missing). The assumption can not, as mentioned in section 6.4, be formally tested anyway - but a look further back in time would have been reassuring.

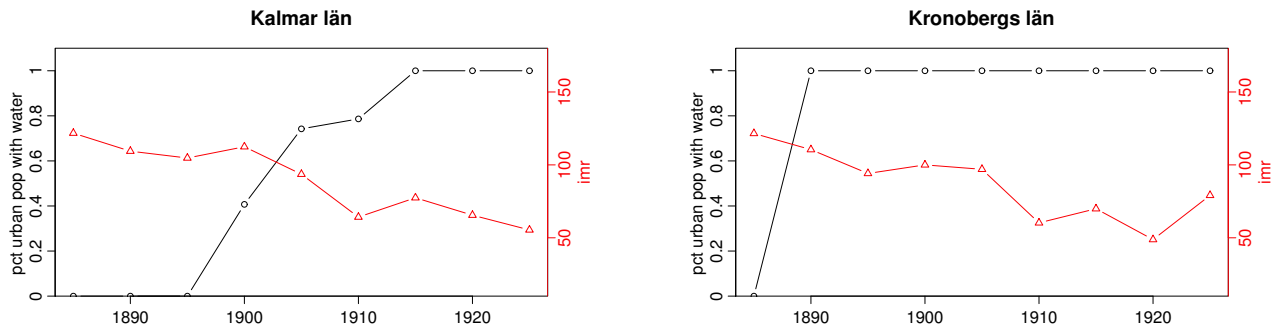


Figure 20: IMR and % urban population with water access

The result from the rudimentary regression model gives a DiD-coefficient of treatment of -12.215, which is to be interpreted as that the average IMR in Kronoberg

county was lowered by -12.215 because of passing the treatment threshold (if we assume that the development in Kronoberg otherwise would have had the same trend as in Kalmar county). In general, the difference-in-differences framework is set to capture effects at the aggregate level (as mentioned in section 6.3), leaving the treatment to be the only difference. With that in mind, answering whether or not this diff-in-diff analysis is believable is hard, but with the experiences from the OLS models above, it is likely that there is omitted variable bias (and what we're seeing is not the actual treatment effect).

7.3.2 Norrbotten and Jämtland counties

The second difference-in-differences example is between Norrbotten and Västerbotten counties - they are close to each other (but not adjacent) in the far north of Sweden. The urban population of Jämtland is in general about half of that in Norrbotten, and this may be a source of bias, but in general I consider these to be a reasonable counterfactual match. The dependent and independent variable progression in the two counties can be seen in Figure 21. As seen in the figures, treatment arrives later in Norrbotten (1905) than in Jämtland (1895), and the IMR variation seems to be greater in Jämtland than Norrbotten (Jämtland having a IMR spike that coincides with the county becoming fully treated with water access). As with the last example, it is hard to verify the parallel trend assumption since treatment comes early in the process.

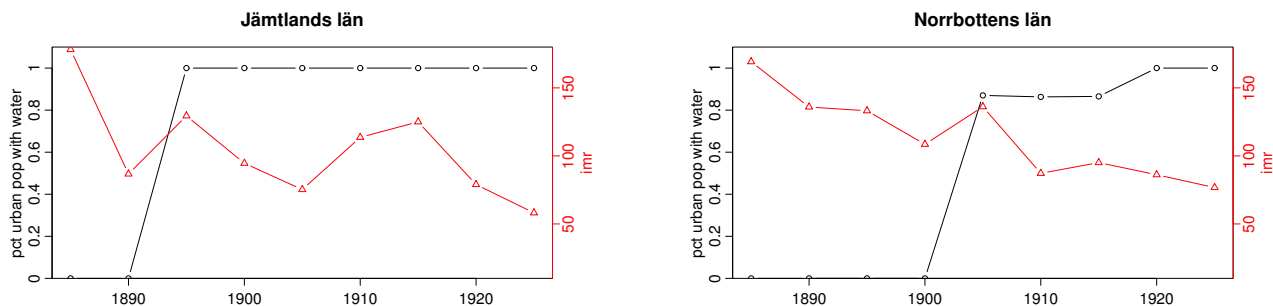


Figure 21: IMR and % urban population with water access

The result from the diff-in-diff regression model gives a coefficient of treatment of -22.86, which is to be interpreted as that the average IMR in Jämtland county was lowered by -22.86 because of passing the treatment threshold (assuming parallel trends). Being an average, it is a fairly high effect from treatment. As with the previous diff-in-diff example, one can question whether or not most of the variation is captured at the county level, or if the effect in the treated county also is reflecting omitted variables.

7.4 Limitations

7.4.1 Partial treatment

The nature of the data included in this analysis does not let us know, on a city level, how many people that in reality have access to clean water and/or sewage (as

mentioned in section 5.3). This limitation arises from data lacking data on coverage and introduces a potential confounding of the effect of clean water technology on mortality. The problem lies in the fact that a city is assumed (more on this in section 7.4.2) to be completely treated (i.e. have access) from the year in which implementation is said to have started. This means that there might be an overestimation of the effects of the technologies, as it is assumed that all of the a city's population has access from the given year. If data had existed on actual treatment coverage, it could have been used as an instrument to regulate the output - since the current situation is similar to an intention-to-treat situation, where all that are offered treatment do not take up on the offer (Angrist and Pischke 2008, p.122). An attempt to estimate the magnitude of this bias has been presented in section 7.1.6.

A further dimension of this problem is the binary treatment threshold (defined in section 6.1): choosing a 50% threshold is intuitively logical when thinking about majorities, but in this complex context, it is of course arbitrary. Theoretically, it is not more arbitrary than any other numerical threshold, but it is hard to verify if it is the right level for deciding treatment. Depending on how access to water varies with mortality, it might be both an upwards or downwards bias.

7.4.2 Timing and duration of introduction

As discussed in section 4.3, the timing with which a city takes a decision to implement clean water technology can be considered to be exogenous, which makes the basis for comparison in a difference-in-differences framework. Treatment (year of implementation start) is (by necessity) assumed to cover the whole city directly from the start, and this "overnight" introduction of a city-wide complicated technology is not likely, not even in theory. Cutler (2005, p.8) had the same challenge, and has solved this by using a treatment year that corresponds to the year when "the majority of the municipal population were first served by these interventions". Unfortunately, such information is not available for this analysis, which is why the assumption of complete and direct treatment is needed. Another form of delay is the one mentioned in section 4.2, in which piped water/sewage is finished in construction, but not actually in operation because of technical problems. These limitations in relation to the results found are discussed in section 8.

In study similar to this analysis, by Castensson, Löwgren, and Sundin (1988), the year of construction of water pipes /sewage is linked to mortality measures in four Swedish cities (between 1860 and 1920). They conclude that the mortality data shows "no changes that are noticeably connected to the years of construction", and that further research is needed to study the spread of these innovations (ibid., p.286). They hypothesise that the effect from these systems might be subtle, such as that the water-borne epidemics would become less pronounced after the technology had been implemented - but this would not be directly visible in data. While other authors (such as D. Cutler and Miller (2005)) have seen clear and large effects, others have not - which makes further research such as this needed, but also opens up for the possibility that apparent mechanisms might be hard-pressed to find.

7.4.3 Data levels

As discussed in sections 6.4 and 5.3, the mortality data exists on an aggregate level, while the independent treatment variable exists on a sub-level (county vs.

city). Compared to a study like D. Cutler and Miller (2005), where both treatment (access to clean water) and outcome (mortality) data is available on city level, this is a limitation. The results obtained using a continuous treatment variable are confounding by definition, since we in theory cannot know if the effect of access to clean water on mortality is the same in every city. In practise, however, it is relatively same to assume that the basic epidemiological mechanisms work the same way in every city - it is more a question of the reliability of the independent variable data: if a city had not, in practise, given its citizens access to clean water a certain year (even though raw data says it has), the effect of clean water on mortality would be underestimated. However, despite counting on the effect of the independent variable being the same in all cities, the basic confounding problem of not knowing which mortality comes from which city still remains. Combined with the potential timing differences in getting access to the majority of a city's population, this might lead to large parts of the variation going unexplained.

8 Discussion

To recap the research question for this analysis: how much did the implementation of clean water technologies in Swedish cities affect urban mortality between 1885 and 1925?

The answer to this question is, that a consistent estimate of the magnitude of the effect of clean water technologies on urban mortality in Sweden cannot be given based on this analysis. Judging from the general models, the percentage of urban population with access to clean water seems to have an inverse relation with mortality, but as the models add controls and fixed effects, much of this variation is instead captured by general time- and county-specific effects. Castensson, Löwgren, and Sundin (1988, p.287) conclude after their similar study of Swedish cities, that "there are too many confounding factors that conceal the possible effects of sanitary improvements" - and that conclusion might also hold true for this analysis. As cited in the beginning of this paper, D. Cutler and Miller (2005) conclude that although the various reasons for the mortality decline are not mutually exclusive, it is important to distinguish between them - and this distinguishing of effects has not been possible with this method and data.

The difference-in-differences approach seems to, in its simplicity, make sense judging by the examples presented in this analysis. However, the parallel trend assumption is hard to comment on, as treatment often occurs early in the analysis period. The lack of much-needed but not available control variables (to match the theories on why urban mortality dropped - no researcher ever suggested it was only due to clean water technology) makes the observed outcome of the difference-in-differences examples harder to believe.

Summarising comments and findings from the analysis: firstly, the effects found show, as the models progress, increased signs of omitted variable bias, taking the form of general time/county trends in mortality. Mortality did, which has been established several times in this analysis and elsewhere, drop significantly during this study period - but the independent variables used in this analysis are not sharp enough to explain their part in the variance in mortality. Using disease-specific mortality with water-borne diseases (which are usual suspects in the causal chain between access to clean water and mortality) does not help make the picture any

clearer. Secondly, the aggregate level (county, consisting of sub-units carrying the independent variable data) may be obscuring effects, due to partial treatment (since a total treatment of each city had to be assumed). The aggregate level that was necessary in order to use the independent variable of interest (% with access to clean water) together with the county-level mortality data might have smoothed out differences and relationships that would have been visible on the level where the independent variable operates: the city level. Thirdly, using a treatment dummy based on majority of a continuous treatment variable seems to be too simplistic to capture any variance. Finally, the timing and possible lagged effect of treatment might obscure treatment effects. As has been noted by several researchers (see section 4.2), the implementation and expansion of a city water system can be a long and tedious political process. If the "treatment year" reported in statistics does not coincide with when a majority of the population was given access, this might introduce bias and make it harder to capture mortality variation in a model.

The result from this study is, from my own point of view, an introduction to this newly digitised dataset and a starting point for further research. As much as we know of the general mortality decline in 19th and 20th century Europe, there are still knowledge gaps that could be filled, and this could also contribute with valuable input to policies for handling current problems of the same type, in other parts of the world.

8.1 Future research

As mentioned in the Discussions-section, there are many ways in which a future project might come closer to a causal effect of clean water technologies on urban mortality. The following points would be interesting to look at in a future analysis:

- working on a lower data level (i.e. city level, both for dependent and independent variables) would remove some of the confounding factors that lie in aggregation, and allow the independent and dependent variables to variate closer to the level where they actually operate (i.e. through water sources, organised by city officials).
- making use of data on clean water technologies that have not been fully digitised yet, more control variables (for example on water purification technologies, water quality, and sewage technology) could help capture more of the mortality variation, and also throw light on which kinds of technologies had the best effect.
- if they can be located, more general demographic and economic control variables could be put to good use in the models, to further try and isolate the "water" variables from the, as of now, omitted variables.
- instead of using a majority treatment threshold (such as the "50% treated" threshold used in this analysis), a continuous treatment variable could be exploited to get a more dynamic picture of the relationship between access to clean water and mortality.
- since it has been established that timing and coverage of treatment might have a confounding effect, more advanced and individualised lagged independent variables might help capture further variation that is now unexplained.

- a subject that has been briefly mentioned in this analysis, but is also interesting, is the differentials in access to clean water that were present at the time: patterns of mortality broken down in, for example, socio-economic status, would be an interesting angle to look closer into.

References

- Aaby, Peter (1992). “Lessons for the past: Third World evidence and the reinterpretation of developed world mortality declines”. In: *Health Transition Review*, pp. 155–183.
- Angrist, Joshua D and Jörn-Steffen Pischke (2008). *Mostly harmless econometrics: An empiricist’s companion*. Princeton university press.
- Bengtsson, Tommy and Martin Lindström (2003). “Airborne infectious diseases during infancy and mortality in later life in southern Sweden, 1766–1894”. In: *International Journal of Epidemiology* 32.2, pp. 286–294.
- Bjur, Hans (1988). *Vattenbyggnadskonst i Göteborg under 200 år*. Göteborgs VA-verk.
- Bloom, David E (2011). “7 billion and counting”. In: *Science* 333.6042, pp. 562–569.
- Brändström, Anders (1984). ” *De kärlekslösa mödrarna*”: spädbarnsdödligheten i Sverige under 1800-talet med särskild hänsyn till Nedertorneå. Universitetet i Umeå.
- Card, David and Alan B Krueger (1993). *Minimum wages and employment: A case study of the fast food industry in New Jersey and Pennsylvania*. Tech. rep. National Bureau of Economic Research.
- Castensson, Reinhold, M Löwgren, and Jan Sundin (1988). “Urban water supply and improvement of health conditions”. In: *Society, Health and Population during the Demographic Transition*, pp. 273–298.
- Cutler, David M, Angus S Deaton, and Adriana Lleras-Muney (2006). *The determinants of mortality*. Tech. rep. National Bureau of Economic Research.
- Cutler, David and Grant Miller (2005). “The role of public health improvements in health advances: the twentieth-century United States”. In: *Demography* 42.1, pp. 1–22.
- Duffy, John (1992). *The sanitarians: a history of American public health*. University of Illinois Press.
- Easterlin, Richard A (1999). “How beneficent is the market? A look at the modern history of mortality”. In: *European Review of Economic History* 3.03, pp. 257–294.
- Econtalk.org (2014). *EconTalk Episode with Joshua Angrist, hosted by Russ Roberts*. URL: http://www.econtalk.org/archives/2014/12/joshua_angrist.html.
- Edvinsson, Sören (1992). “Den osunda staden: sociala skillnader i dödlighet i 1800-talets Sundsvall”. In:
- Ferrie, Joseph P and Werner Troesken (2008). “Water and Chicago’s mortality transition, 1850–1925”. In: *Explorations in Economic History* 45.1, pp. 1–16.
- Fewtrell, Lorna et al. (2005). “Water, sanitation, and hygiene interventions to reduce diarrhoea in less developed countries: a systematic review and meta-analysis”. In: *The Lancet infectious diseases* 5.1, pp. 42–52.
- Fogel, Robert W (1986). “Nutrition and the decline in mortality since 1700: some preliminary findings”. In: *Long-term factors in American economic growth*. University of Chicago Press, pp. 439–556.
- Fridlitzius, Gunnar (1985). “The mortality decline in the first phase of the demographic transition: Swedish experiences”. In:
- Friedman, Jed (2013). *The often (unspoken) assumptions behind the difference-in-difference estimator in practice*. URL: <http://blogs.worldbank.org/impactevaluations/often-unspoken-assumptions-behind-difference-difference-estimator-practice>.
- Habicht, Jean-Pierre, Julie DaVanzo, and William P Butz (1988). “Mother’s milk and sewage: their interactive effects on infant mortality”. In: *Pediatrics* 81.3, pp. 456–461.
- Klaesson, Johan and Lars Pettersson (2009). “Urban–rural development in Sweden”. In: *New Directions in Regional Economic Development*. Springer, pp. 317–330.
- Lee, Ronald (2003). “The demographic transition: three centuries of fundamental change”. In: *The journal of economic perspectives* 17.4, pp. 167–190.

- McCarthy, Michael P (1987). *Typhoid and the politics of public health in nineteenth-century Philadelphia*. Vol. 179. American Philosophical Society.
- McKeown, Thomas, RG Brown, and RG Record (1972). “An interpretation of the modern rise of population in Europe”. In: *Population studies* 26.3, pp. 345–382.
- McKeown, Thomas and RG Record (1962). “Reasons for the decline of mortality in England and Wales during the nineteenth century”. In: *Population studies* 16.2, pp. 94–122.
- Mora Villarrubia, Ricardo and Iliana Reggio (2012). *Treatment effect identification using alternative parallel assumptions*. Tech. rep. Universidad Carlos III, Departamento de Economía.
- Omran, Abdel R (1971). “The epidemiologic transition: a theory of the epidemiology of population change”. In: *The Milbank Memorial Fund Quarterly*, pp. 509–538.
- PHAS, Public Health Agency of Sweden (2005). *Svenska folkets hälsa i historiskt perspektiv*. Statens folkhälsoinstitut R 2005:8.
- Prüss-Ustün, Annette et al. (2014). “Burden of disease from inadequate water, sanitation and hygiene in low-and middle-income settings: a retrospective analysis of data from 145 countries”. In: *Tropical Medicine & International Health* 19.8, pp. 894–905.
- R (2008). *R: A Language and Environment for Statistical Computing*. ISBN 3-900051-07-0. Vienna, Austria: Development Core Team, R Foundation for Statistical Computing. URL: <http://www.R-project.org>.
- Razzell, PETER E (1974). “‘An interpretation of the modern rise of population in Europe’—A critique”. In: *Population Studies* 28.1, pp. 5–17.
- SCB, Statistiska Centralbyrån (1885). *Befolkning 1851–1910 (BiSOS A)*. URL: http://www.scb.se/sv_/Hitta-statistik/Historisk-statistik/Statistik-efter-serie/Bidrag-till-Sveriges-officiella-statistik-BiSOS-1851-1917/Befolkning-1851-1910-BiSOS-A/.
- Schultz, T Paul (1993). “Mortality decline in the low-income world: causes and consequences”. In: *The American Economic Review*, pp. 337–342.
- Soares, Rodrigo R (2007). “On the determinants of mortality reductions in the developing world”. In: *Population and Development Review* 33.2, pp. 247–287.
- Söndags-Nisse (1866). *En kloak – i Stockholm*. Söndags-Nisse – Illustrerad Veckoblad för Skämt, Humor och Satir, nr 34.
- SWEP (1964). *Statistiska uppgifter över vatten- och avloppsverk*. Swedish water and waste water works association.
- Wetterberg, Ola and Gunilla Axelsson (1995). *Smutsguld & dödligt hot*. Göteborgs renhållningsverk: Stadsbyggnad Chalmers tekniska högskola,
- WHO (2014). *Water Sanitation Health: Burden of disease and cost-effectiveness estimates*. URL: http://www.who.int/water_sanitation_health/diseases/burden/en/.