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Descriptive Finding

Exploring the mortality advantage of Jewish neighbourhoods in mid-19th century Amsterdam

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Exploring the mortality advantage of Jewish neighbourhoods in mid-19th century Amsterdam

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Abstract

BACKGROUND
Many studies have observed that religion plays an important role in determining inequalities in mortality outcomes before the mortality decline in late 19th century Europe. Yet, it is difficult to pinpoint what exactly caused the mortality advantage observed for Jewish populations before the start of the demographic transition.

OBJECTIVE
To explore an alternative approach to the observed Jewish mortality advantage by comparing differences and similarities in various cause-specific mortality rates in Amsterdam’s 50 neighbourhoods in the mid-19th century.

RESULTS
Jewish neighbourhoods had an overall mortality advantage, which was reflected in lower infant, respiratory, diarrhoeal, and smallpox death rates. Only in the cholera epidemic did the Jewish neighbourhoods not experience this health advantage.

CONCLUSION
Before the mortality decline, individual (and community) behaviours could already have been having an important impact on inequalities in health, although not for all diseases.

CONTRIBUTION
The neighbourhood approach is a useful alternative when individual-level data is not available to demonstrate how variation in social, economic, and disease environments may have resulted in health inequalities. In addition, the comparison of various cause-specific mortality rates in Jewish and non-Jewish neighbourhoods helps to disentangle which determinants might explain why Jewish mortality rates were lower.

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1. Introduction

Many studies have observed that religion plays an important role in determining inequalities in mortality outcomes before the mortality decline in late 19th century Europe (van den Boomen 2021; Kemkes-Grottenthaler 2003; Walhout 2019; Wolleswinkel-van den Bosch et al. 2000). Jewish communities in particular had lower general mortality and infant mortality rates than other religious groups. Moreover, these differences in mortality persist even when socioeconomic status and income level are taken into account (Connor 2017; Derosas 2003; Haeusermann 2016; Sawchuk, Tripp, and Melnychenko 2013). Various studies on the Netherlands (van Poppel 1992; van Poppel, Schellekens, and Liefbroer 2002) and Amsterdam (Ekamper and van Poppel 2019; Snel and van Straten 2006, 2018; Verdoorn 1981: 62–67) point in the same direction. Several sometimes contradictory factors have been suggested to explain the relatively low mortality of Jews: mothers providing better care for their children (e.g., longer breastfeeding), willingness to embrace modern medicine (e.g., smallpox vaccination), a better life style (e.g., hygiene practices, better diet and nutrition), the low prevalence of syphilis, the lack of alcohol abuse, and favourable local air, soil, and water conditions in their neighbourhoods (Coronel 1864; Egeling 1863; Israëls 1862; Pinkhof 1907; Rutten 1997; Teixeira de Mattos 1865: 209–210). Overall, it seems difficult to pinpoint what exactly caused the mortality advantage observed for Jewish populations before the start of the demographic transition.

In this study we have explored an alternative approach to the observed Jewish mortality advantage by comparing differences and similarities in various cause-specific mortality rates in Amsterdam’s 50 neighbourhoods in the mid-19th century. We compare neighbourhoods instead of individuals for two reasons. First, no information is available at the individual level to estimate the population at risk, which makes it impossible to calculate individual-level mortality chances. Second, information available at the neighbourhood level allows us to take a broader approach and explore how variation in the social, economic, and disease environments may result in health inequalities. We compare cause-specific mortality from several infectious diseases to explore if such analysis indicates why neighbourhoods with a large percentage of Jewish inhabitants have better cause-specific mortality rates for some diseases but not for others. In short, our descriptive findings suggest that our exploratory approach is able to provide a more detailed account of the factors determining general mortality outcomes and that using cause-specific mortality rates can demonstrate which underlying mechanisms are responsible.
2. Sources, data, and methods

The Amsterdam Cause-of-death Database (hereafter, ACD) is the main source for our analysis. This dataset contains individual death records between 1853 and 1926 with information on the day of death, age, gender, cause of death, residential and/or institutional address, and in some cases occupation. The city authorities initiated the registers in 1853 in response to the increasing fear amongst the population of being buried alive. Hence, it was decided that all deaths had to be verified by a doctor, resulting in a systematic registration of all deceased individuals together with the cause they died of from 1854 onwards (Neurdenburg 1929). When the Public Health Inspectorate Act and the Medical Practitioners Act were introduced, it became mandatory for medical doctors to provide a medical certificate for every death. This in turn increased the accuracy and completeness of the cause-of-death registers (van Poppel and van Dijk 1997). In general, from 1865 onwards the Amsterdam cause-of-death registration is very complete and reliable compared to other sources of information (Muurling, Riswick, and Buzasi 2021).

The time period under scrutiny covers the years between the two largest epidemics in the Netherlands in the 19th century: cholera in 1866 and smallpox in 1871. We conduct descriptive and OLS regression analyses using various neighbourhood variables that are constructed from a range of primary sources from a time period as close as possible to the constructed mortality rate. The dependent variables in the regressions are average crude death rate, average infant death rate, average respiratory death rate, and average diarrhoeal death rate (all concerning 1867–1870, and all based on deaths per 10,000 people), as well as the cholera death rate of 1866 and the smallpox death rate of 1871. The diarrhoeal death rate consists of typhoid fever, infantile cholera, diarrhoea, dysentery, gastric catarrh, gastroenteritis, enteritis, teething, and dyspepsia. Tuberculosis, pneumonia, acute bronchitis, whooping cough, scarlet fever, and croup are counted in the respiratory death rate (see Janssens 2021 for more information about the coding practices of historical causes of death). The death rate is obtained by dividing the number of (cause-specific) deaths by the population in each neighbourhood multiplied by 10,000. The population figures were calculated using interpolation between the censuses (Centraal Bureau voor de Statistiek 1999) as shown in the Github repository (Buzasi 2022).

Because ACD data lacks sufficient information on the socioeconomic status of the deceased, we use an alternative variable to take the impact of wealth on mortality into account, namely the categorisation of all 50 neighbourhoods into ‘poor’, ‘rather poor’, ‘rather wealthy’, and ‘wealthy’ by the Amsterdam physician Abraham Hartog Israëls (1862), based on his knowledge of the city. Although this variable precedes the studied periods of mortality, we assume that the relative wealth position of neighbourhoods was fairly stable in the 19th century. This assumption is supported by comparing two alternative wealth indicators: the correlation coefficient between the mean rental values
(Lesger and van Leeuwen 2012) from the 1832 land register and the share of high tax revenues in 1897/98 (Het Bureau van Statistiek der Gemeente 1909) is high (0.893, 95% CI: 0.814, 0.939). Additional neighbourhood variables include housing density, birth rate (Israëls 1862), presence of a hospital in 1871, and, importantly, a variable for the four Jewish neighbourhoods where 50% or more of the household heads in the neighbourhood were Jewish. With just under 18,000 inhabitants, these Jewish neighbourhoods made up around 7% of the Amsterdam population. Housing density is the average number of people by inhabitable building by neighbourhood. The number of buildings is obtained from the 1869 census. For more information on the sources, data, and variables, see Muurling, Riswick, and Buzasi (2021).

3. Descriptive findings

In the 19th century, substantial socioeconomic differences existed across Amsterdam’s urban landscape. Figure 1 demonstrates the crude death rate by neighbourhood and where the Jewish neighbourhoods were located. This gives an impression of the lower general mortality of the Jewish neighbourhoods compared to their non-Jewish counterparts in Amsterdam during the mid-19th century. The bar chart in Figure 2A confirms earlier studies as well as observations documented by contemporaries: most neighbourhoods with a relatively large share of Jewish inhabitants had lower mortality in general despite their wealth status (Egeling 1863; Israëls 1862; Teixeira de Mattos 1865). The Jewish advantage in all-cause mortality is confirmed in the regression models, even after including controls (Model 1 and 7 in Table 1). Because differences in age structure between neighbourhoods are not taken into account in the crude death rate, the infant death rate is visualized in Figure 2B. Although the simple bar chart might not be convincing, the more favourable position of Jewish neighbourhoods in terms of infant mortality becomes clear once we control for their lower wealth status and other socioeconomic factors (Model 8 in Table 1). This finding is in line with observations that the circumstances for infants were more favourable because of better childcare (e.g., hygiene practices in general and concerning food preparation in particular), more and longer breastfeeding, and longer birth intervals (van Poppel, Schellekens, and Liefbroer 2002; Walhout 2010).

Investigating cause-specific mortality rates, however, may give some clues as to the reasons for this pattern. Does taking a closer look at mortality caused by respiratory diseases, diarrhoea, cholera, and smallpox offer any clues as to which determinants caused Jewish neighbourhoods to do better? Did they have a health advantage regardless of the infectious disease under scrutiny? Or did this differ when differentiating between endemic and epidemic diseases?
Figure 1: Map of the crude death rate and Jewish neighbourhoods in Amsterdam, 1867–1870

Note: Own estimates based on Amsterdam Cause-of-death database.
Figure 2: Death rates in Amsterdam’s neighbourhoods

Note: Own estimates based on Amsterdam Cause-of-death database.
The Jewish neighbourhoods had a health advantage in both respiratory and diarrhoeal endemic infectious diseases, even though these have very different determinants (see Figures 2C and 2D and Models 3, 4, 9, and 10 in Table 1). Respiratory infectious diseases, such as tuberculosis, can be best viewed as diseases that slowly weaken individuals before eventually resulting in death. Therefore, they are often understood in relation to the nutritional status or labour circumstances of individuals, as the majority of respiratory deaths concerned adults: 60% of individuals in this cause-of-death category are above 14 years of age (McKeown, Record, and Turner 1975; Wolleswinkel-van den Bosch 1998). Yet, even though respiratory infections are believed to be fostered by a concentration of poor, malnourished people in unhealthy physical and social environments, the Jewish neighbourhoods did comparatively well regardless of their impoverished state. Some scholars have argued that a healthier lifestyle due to Jewish dietary and other ritual laws and relative social isolation may explain the Jewish mortality advantage regarding respiratory diseases (Blom and Cahen 2017: 235; van Poppel, Schellekens, and Liefbroer 2002: 279). By contrast, the diarrhoeal death rate mostly represents the number of infants and young children who got sick and eventually died from drinking contaminated water. In this cause-of-death category 53% were infants and 70% were below 10 years of age. The fact that the Jewish neighbourhoods fared much better may therefore be proof that breastfeeding was much more common, thereby avoiding the risks of infants ingesting contaminated water.

While people saw the detrimental effects of the described endemic mortality rates on a daily basis, epidemic mortality is often viewed as a societal shock (Alfani 2021). The comparison of the results of the two studied epidemics, cholera and smallpox, is particularly insightful (see Figure 2E and 2F). While the Jewish neighbourhoods were among the most affected in the 1866 cholera outbreak, they did remarkably well during the smallpox epidemic five years later. This is also confirmed by the regression analysis (Models 5, 6, 11, and 12 in Table 1). To explain these findings we turn to what we know about differences in the causes and transfer of these two epidemic diseases.

As a diarrhoeal and predominantly waterborne disease, the transmission of cholera is closely linked to inadequate access to clean water because of infected lakes, canals, and sanitation facilities such as waste disposal and sewerage. Moreover, even clean water can become infected by poor hygiene of the persons handling the water outside and inside the household. In addition to being transmitted through water, consumption of contaminated food can also increase the likelihood of getting cholera (Taylor et al. 2015). As opposed to other diarrhoeal diseases, cholera affects age groups that normally have low mortality, and does not mainly affect infants. The fact that Jewish neighbourhoods fared so badly during the cholera epidemic suggests that their sewage and water quality was similar to, or worse than, other areas in the city. Neither better hygiene and lifestyle practices in these communities nor possible favourable air and soil conditions seem to
have resulted in lower cholera mortality in Jewish neighbourhoods. Thus, the reason for the high cholera death rate might have been that the disease could not be avoided by individual behaviour but was contingent on the availability and quality of the water and disposal systems in the neighbourhoods (Davenport, Satchell, and Shaw-Taylor 2019; Snel and van Straten 2018; Walhout and Beekink 2021). This result reflects the general impression of contemporaries, who wondered what determined who got infected with cholera: “Could it have been due to the lack of (sufficiently close) standpipes with clean water in the poorer parts of the city, which also include the Jewish neighbourhoods?” (Von Baumhauer 1866).

In contrast to cholera, smallpox is an airborne infectious disease that was mainly spread through prolonged close face-to-face contact with an infected person, or contact with the pus from pustules of an infected person. Alternatively, it could be transmitted through direct contact with contaminated objects, such as bedding or clothing (Crosby 2008; Milton 2012). In Amsterdam during the peak year of the smallpox epidemic, 1871, the largest burden of death fell on children below the age of 4 (Muurling, Riswick, and Buzasi 2021). Previous studies have contended that factors such as housing density, public health policies and control interventions, individual prevention, and vaccination influenced who was affected by smallpox (Davenport, Satchell, and Shaw-Taylor 2018; Hardy 1993; Krylova and Earn 2020). While differences in wealth and housing density appear to have been decisive in determining the smallpox mortality across most parts of the city, they fail to explain why Jewish neighbourhoods did so well. Instead, the Jewish smallpox mortality advantage suggests that individual behaviours and community norms and values played an important role. Research has contended that Jewish communities were often relatively isolated socially and in terms of labour relations. Having fewer contacts outside of their community and neighbourhood may have reduced their chances of coming into contact with infected people (Mercier 2006; van Poppel, Schellekens, and Liefbroer 2002).

Perhaps more pertinent is the characterization of the Jewish population as more willing to embrace modern medical insights and take preventative measures. This may in part be due to the prominent role of Jewish doctors in the 19th century public health movement and their active support for vaccination (Nieuw Israelietisch Weekblad 1871; Blom and Cahen 2017). Although we lack neighbourhood vaccination rates, it is likely that the Jewish population vaccinated their children against smallpox more than the general population (Rutten 1997). Vaccination was available before the epidemic of the 1870s, but the city government noticeably intensified its vaccination campaign from the onset of the smallpox outbreak, actively encouraging its inhabitants to get vaccinated free of charge (Muurling, Riswick, and Buzasi 2021). The relative success of the Jewish neighbourhoods during the smallpox epidemic, in spite of being poorer and overcrowded, suggests the importance of vaccination uptake. This may just be one example of Jews in
Amsterdam adapting to medical knowledge and health innovations faster and in greater numbers than the general population.

Table 1: Regression analyses without and with controls, comparing death rates in Jewish and non-Jewish neighbourhoods

|                      | av. CDR | av. IDR | av. resp. DR | av. diarrh. DR | cholera DR | smallpox DR |
|----------------------|---------|---------|--------------|---------------|------------|-------------|
| **Without controls** |         |         |              |               |            |             |
| Jewish               | -40.024 | -9.574  | -22.167      | -11.569       | 77.206     | -46.032     |
| rob. std. error      | 17.588  | 9.482   | 3.380        | 1.115         | 35.750     | 6.923       |
| p-value              | 0.027   | 0.318   | <0.001       | <0.001        | 0.036      | <0.001      |
| Adj. $R^2$           | 0.082   | 0.004   | 0.285        | 0.366         | 0.339      | 0.070       |
| **With controls**    |         |         |              |               |            |             |
| Jewish               | -76.058 | -16.464 | -30.285      | -14.777       | 54.656     | -90.445     |
| rob. std. error      | 25.043  | 6.131   | 4.994        | 2.184         | 33.251     | 17.754      |
| p-value              | 0.004   | 0.010   | <0.001       | <0.001        | 0.108      | <0.001      |
| Adj. $R^2$           | 0.420   | 0.575   | 0.413        | 0.487         | 0.460      | 0.562       |

*Note:* Own estimates based on Amsterdam Cause-of-death Database. The number of observations is 50 in each model. Control variables: wealth, housing density, birth rate, presence of a hospital.

4. Discussion

In our study we have investigated whether examining various cause-specific mortality rates provides insight into why the neighbourhoods with a large percentage of Jewish inhabitants generally had lower mortality than other neighbourhoods. Two main conclusions can be drawn. First, when individual-level data is not available the neighbourhood approach is a useful alternative to demonstrate how variation in social, economic, and disease environments may result in health inequalities. Second, the comparison of various cause-specific mortality rates in Jewish and non-Jewish neighbourhoods helps to disentangle which determinants might explain why Jewish mortality was lower. Our analysis demonstrates that Jewish neighbourhoods had an overall mortality advantage, which was reflected in the lower infant, respiratory, diarrhoeal, and smallpox death rates. Only when faced with the cholera epidemic did Jewish neighbourhoods not experience this health advantage. This suggests that even before the mortality decline, individual and community behaviours could have been having an important impact on health inequalities, although this was not true for all diseases. Future studies could use a similar approach to investigate whether similar insights can be obtained by studying other social groups, a greater variety of geographical areas, and a larger number of cause-specific mortality outcomes. Doing so will enable us to better understand the underlying mechanisms that caused inequality in mortality outcomes in populations where mortality is not declining.
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