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Religious differentials in infant and child mortality in Holland, 1855–1912

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Abstract. At least three kinds of hypothesis may be invoked to interpret religious differentials in mortality. They are (i) hypotheses that refer to characteristics, (ii) those that refer to lifestyle, and (iii) those that refer to the social isolation of minorities. This paper tests all three kinds of hypothesis using data on urban child mortality from The Hague just before and during the demographic transition. A hazard analysis suggests that economic and demographic characteristics do not account for much of the variation by religion. An analysis of seasonal mortality suggests that some of the variation may be explained by differences in lifestyle. The third kind of hypothesis is presented here for the first time. We suggest that the social isolation of small religious groups lowered their exposure to certain kinds of infectious disease. We use a simulation study to show that this hypothesis could account for part of the variation.

INTRODUCTION

'To the casual observer, medicine and religion may appear to have little connection except in the names of some hospitals ...' (Numbers and Amundsen 1986, p. 1). In fact, the relationship between religion and mortality is a well-established one, documented for populations before, during, and after the demographic transition. Contemporary religious differentials are known to be due partly to differences in socio-economic characteristics and lifestyle (Jarvis and Northcott 1987 and Troyer 1988) Much less is known about the reasons for these differentials in the past. Usually differences in socio-economic characteristics and lifestyle are quoted as possible explanations (e.g. Schmelz 1973), but only a few studies have ever attempted to check whether these explanations are consistent with the data. Condran and Kramarov (1991) show that socio-economic status and demographic characteristics do not explain the differences in childhood mortality between Christians and Jews. Reid (1997) also shows that the relatively low mortality among Jewish children in England and Wales in the period 1895–1911 cannot be explained away by socio-economic and other characteristics. Most recently, Derosas (2000) has shown the same for the differences in infant mortality between Catholics and Jews in nineteenth-century Venice. McQuillan (1999) has studied differences in infant and child mortality between Catholics and Protestants in Alsace in the period 1750–1870. These differences persist after controlling for socio-economic status and demographic characteristics, especially among infants. Thus, there is a consistent finding that religious differentials largely remain after controlling for socio-economic background. Some have suggested that, given the failure to establish socio-economic explanations, it must be cultural differences in lifestyle that account for most of the religious differentials (e.g. Derosas 2000). However, such a conclusion is unwarranted unless alternative explanations have also been shown to be invalid. Moreover, at least one form of the lifestyle hypothesis can be shown to fit the data poorly.

In the past, most child deaths in today's developed countries were the result of infectious and parasitic diseases. The likelihood of a child contracting such a disease is, among other things, a function of the number and frequency of the child's contacts. Infectious disease will spread evenly through a population if there is homogeneous mixing of individuals. This may not happen when a population consists of several religious groups because transmission rates of disease between these groups may be lower than those within them. In this paper a new hypothesis is developed. It is argued that, in the past, the social isolation of religious minority groups, such as break-away Calvinists (*gereformeerden*), Lutherans, Jews and Mennonites, may have saved lives.

This study uses data on the children of a sample of 3402 couples who married in The Hague in the period 1857–1902. Most of the demographic and socio-economic information was gathered from the population registers, which are available from 1850.

After a discussion of the major mechanisms through which religion is thought to influence mortality and a description of the data and variables we use, we test three kinds of hypothesis that

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may explain religious differentials, using a different method for each kind.

RELIGION AND MORTALITY

Three kinds of hypothesis may be invoked to interpret religious differentials in mortality. They are (i) hypotheses that refer to characteristics, (ii) those that refer to lifestyle, and (iii) those that refer to the social isolation of minorities (compare with Anderson 1986, p. 296).

Religious groups may have different mortality levels because of differences in socio-economic and demographic characteristics. Thus, Bonger (1913, p. 53) attributes part of the higher mortality among Dutch Catholics to their lower level of education. In theory, religious differentials in mortality could also be due to genetic differences, especially when religion coincides with ethnicity. Thus, contemporaries suggested that Jews were more resistant to disease because they were biologically selected for resistance to the diseases associated with slum dwelling (Fishberg 1902). Although genetic factors play a role, they are unlikely to account for much of the variation in mortality (Jacquard 1982, p. 310). Once the proper characteristics have been taken into account, there may still be differences between religious groups. These differences must be due either to lifestyle or to social isolation.

Religion may influence lifestyles and, hence, health-related behaviour. For example, rabbis urged Jews to live healthy lives. The popular book on Jewish law and rituals by the Hungarian Rabbi Shlomo Ganzfried (1975) contains two paragraphs with medical advice. Most of the advice he gave reflects outdated medical practice, but perhaps this and similar books are the source of the greater readiness which is thought to exist among Jews to attend to their health and that of their families. Thus, Jews are thought to have made better use of medical care when available. Goldstein, Watkins and Spector (1994) found that, in the first decades of this century, Jewish mothers in the United States were more likely than Italian mothers to define an ailment as requiring professional attention. An analysis of a smallpox epidemic in the Dutch town of Nijkerk in 1871–72 revealed that Jews were more likely to have been vaccinated in youth (Rutten 1997, pp. 152–53). However, apart from smallpox vaccination there were few preventive therapies or cures available before the end of the nineteenth century (McKeown and Record 1962). Moreover, many physicians were still ignorant of the germ theory at the turn of the century. The most enlightened public health officials 'saw clearly the implications of the germ theory for preventive health care, but they despaired at the difficulties of getting the word across to physicians, let alone parents' (Preston and Haines 1991, p. 200). Hence, many would now agree that around the turn of the century differences in attitude toward professional health care are unlikely to be a major explanation of religious differentials. On the other hand, some continue to argue that 'the influence of medical men on the survival of children was not insignificant. Its effects were indirect, mainly through improvements in hygiene' (e. g. Morel 1991, p. 207).

The Polish Rabbi Abraham Zvi Hirsch Eisenstadt (1966) thought that infants were not to be weaned before they were two years old. Of course, there remains the question of to what extent such views reached ordinary people. Jewish women are indeed thought to have breastfed their infants longer (Condran and Kramarow 1991; Van Poppel 1992), but it is not clear whether this was for religious reasons. However, whatever the reason, Woodbury showed in 1926 that standardization for breastfeeding patterns does not remove the advantage in survivorship of Jewish infants in New York at the beginning of this century (Alter 1997, p. 100). While Jews are often thought to have breastfed for longer than average, breastfeeding is believed to have declined among Dutch Catholics in the second half of the nineteenth century (Meurkens 1989). Aalberse argues that the high infant mortality among Dutch Catholics was related to the fact that there were 'Catholic districts in our country where I am aware that it is particularly the Catholics who do not breastfeed because of a misplaced sense of shame or, even worse, because of the tradition of binding young girls' breasts so they won't develop in order that these women are systematically being made unable, in their youth, to feed their own babies ... This terrible habit is probably the result of prudishness which is wrongly equated with morality ...' (1917, p. 354).

Some religious observances and traditional patterns of behaviour among Jews are thought to have been beneficial to health. Personal cleanliness prescribed by religious rituals required handwashing before and after meals and ritual bathing was required of women once a month (Condran and Kramarow 1991, p. 230). It should be noted that in the second half of the nineteenth century, secularization generally led to a decline in religious observance. Thus, it is to be expected that the importance of differences in religious observance as an explanation of religious differences in mortality would decline over time.

The third kind of hypothesis is presented here for the first time. We suggest that the social isolation of small religious groups lowered their exposure to certain kinds of infectious disease. Next to nothing is known about the interaction between children of different religious groups in the past. Simple models of the spread of infectious disease within a population assume the homogeneous mixing of individuals (Anderson and May 1991, p. 65). Individuals are assumed to be equally as likely to interact and become infected by co-religionists as by others. This is unlikely to have been the case in the past. Interaction was probably less frequent and intensive *between* religious groups than *within* them, possibly leading to lower morbidity from airborne and perhaps also from certain other infectious diseases among minority groups.

THE DATA AND VARIABLES

We chose The Hague for a study of religious differentials in mortality. In the second half of the nineteenth century, The Hague evolved from a provincial capital and quiet residential area into a big modern city. In 1850, the city had about 72,000 inhabitants. After 1870, when prosperity increased, the population grew steadily, reaching 206,000 at the turn of the century. More than half of this growth was due to migration. The presence of the Royal Court, Parliament, and government offices attracted large numbers into the service sector. In 1850 this sector made up 42 per cent of the labour force. Another 34 per cent were employed in industry, mostly in the construction sector and the clothing and shoe industries. By 1900 the service sector had declined to less than 37 per cent, while almost 36 per cent were employed in industry by this date (Stokvis 1987, pp. 88 and 149).

The Hague is located in an area of the Netherlands where the highest infant mortality rates were to be found until the 1880s. Before the onset of mortality decline in the 1870s, infant mortality rates usually fluctuated between 200 and 250 per thousand births, while early-childhood mortality rates ($_4m_1$) fluctuated between 40 and 50 per thousand person-years. Just before the decline in the first half of the 1870s, infant and early-childhood mortality peaked. The high infant mortality rates are thought to have been due to the fact that much of the surface and ground water in the western provinces of the Netherlands was heavily contaminated. Canals were used for the disposal of waste, while water from the canals was used for household purposes by the poor. A system of casks for the collection of human excrements was established in The Hague in 1871 (Vogelzang 1956, pp. 73–74) and a system of water pipes in 1874 (Van Zon 1986, pp. 108–109). By 1910 the infant mortality rate had declined to less than 100, while early-childhood mortality had declined to about 10.

Owing to the unbalanced regional distribution of Catholics, a national study of this group might confuse the effect of religion with that of region. Hence, we decided to focus on a city in the western part of the country with adequate numbers of Protestants, Catholics, and Jews. Nevertheless, some form of over-sampling was necessary to ensure the inclusion of a sufficient number of Jews in our study. To identify Jewish marriages, we needed to construct a list of Jewish surnames. We used four different sources: a list of names of Jewish families living in the Jewish quarter of The Hague during the years 1811–1942 (Van Creveld 1989, pp. 214–222); an index of surnames of Jews marrying during the period 1811–1852 (Veldhuijzen 1996); a list of surnames in the Archives of the Sephardic Jewish community of the Hague; and the register of rabbinical marriages in The Hague in the years 1873–1902. Using the list of surnames we searched for couples who married in The Hague during the years 1859–1902. Finally, the population registers of The Hague were consulted to determine whether bride or groom or both were indeed Jewish. In this way a total number of 961 Jewish couples were identified. For all other religious groups, a random sample was drawn from the records of civil marriages contracted in the period 1859–1902. For each year of marriage the sample fraction varied between 4.6 (1902) and 8.2 per cent (1885). The resulting total number of non-Jewish couples was 3005, bringing the grand total to 3966.

Next, the population registers were used to collect socio-economic and demographic information on the couples. Continuous population registers – in the form of bound documents with non-removable pages – were prescribed in the Netherlands by Royal Decree of 22 December 1849. The registers had to record the population residing within the municipality. The returns from the Census of 1849 were copied into the population register, and from then on all changes occurring in the population during the following decade were recorded in the register. For each individual, date and place of birth, relation to the head of the household, sex, marital status, occupation, and religion were recorded.

For the municipality of Tilburg, Janssens (1994) checked the accuracy of the population registers in recording demographic events by checking the registration of births in the population register against the birth registers. She found that 0.2 per cent of births, at most, were not entered in the population register, all such cases being children dying soon after birth. Thus our estimate of mortality in the first month is certainly an underestimate. We have therefore omitted neonatal mortality from our analysis. This allows us to avoid another problem: religious differentials in the definition of stillbirths. Dutch civil law stated that the birth of a child that had died before a birth certificate could be issued was to be reported as a stillbirth in the death register. Death certificates do not differentiate between true stillbirths and children who had died shortly after birth.

Couples were divided into four religious categories. The Dutch Reformed constituted the largest group with 1500 couples. The Roman Catholic group had 819 couples, and the Jewish group 910, mostly *Ashkenazim* (709). The residual category (173) comprised a variety of groups: different groups of Calvinists such as the *Christelijk Gereformeerde Kerken* (18) and the *Gereformeerde Kerken in Nederland* (20), small liberal Protestant groups such as the *Evangelisch Luthers Kerkgenootschap* (66) and the *Remonstrantse Broederschap*, and people without religion (27).

The occupational categories used in the vital registration and population registers are rather vague. For example, the sources do not indicate whether a person is self-employed or employed. Any classification based only on occupational designations will necessarily be imprecise and incorporate a variety of ambiguities. This applies in particular to the trade sector. Usually, it is impossible to tell whether a person is a great merchant or just a street vendor.

We used a slightly adapted version of the socio-economic classification by socio-economic group designed by Giele and Van Oenen (1976). Their classification is based on the ideas of contemporaries about the class structure and on the relationship of the individual to the means of production. The categories are:

1. Upper class (employers in industry, professionals, high civil servants; higher military);
2. Petty bourgeoisie (shopkeepers, small entrepreneurs and merchants; self-employed artisans);
3. Intellectuals and lower civil servants (lower level professionals and lower civil servants; foremen and supervisors of various kinds);
4. Farmers and fishermen;
5. Skilled manual workers (craftsmen, skilled labourers, construction workers, service employees and lower military);
6. Unskilled labourers;
7. Without occupation and unknown.

The religious groups have different socio-economic profiles. Jews are over-represented in the trade and retailing sector ('petty bourgeoisie') of The Hague (62.5 per cent compared with 17.8 per cent among Dutch Reformed and 26.5 per cent among Catholics), whereas Catholics and the Dutch Reformed more often have working-class occupations than Jews (48.2 per cent among Catholics and 47.3 per cent among the Dutch Reformed compared with 17.7 per cent among Jews). This should not be interpreted as meaning that Jews were on average better off than others, because many Jews in the trade and retailing sector were just peddlers living in one of the poorest neighbourhoods of The Hague (Van Creveld 1989). Almost all fishermen were Dutch Reformed.

In addition to the individual-level and household-level data, we used published cause-of-death statistics for The Hague. Nation-wide medically certified cause-of-death registration was introduced in the Netherlands on 1 June 1865. Following Wolleswinkel-Van den Bosch and others (1996), groups of causes of death were constructed for the period 1868–1919. The causes were regrouped according to the means of transmission. The category of airborne diseases encompasses by far the largest number of deaths in early childhood (ages 1–4) and is responsible for more than half the deaths in this age group in the nineteenth century. It includes whooping cough, measles, smallpox, scarlet fever, respiratory tuberculosis, disseminated tuberculosis, acute respiratory diseases (influenza, acute bronchitis, pneumonia, diseases of pleural cavity), diphtheria, croup, scarlet fever, rheumatic fever and acute nephritis. The category of water-borne and food-borne diseases includes Asiatic cholera and cholera nostras, typhus, typhoid, acute digestive diseases (diarrhea, dysentery, appendicitis and peritonitis), and abdominal tuberculosis. Airborne, water-borne, and food-borne diseases together account for about 30 to 55 per cent of infant deaths depending on the period.

Some cause-of-death categories, such as convulsions, were omitted from the analysis, even though the number of deaths assigned to them was not small. Convulsions constitute an ill-defined category, and may have included conditions due to airborne as well as water-borne and food-borne diseases. The trend over time in convulsions does not follow the trend in either of these categories, perhaps because much of its trend was determined by improving diagnosis. Our inability to distribute deaths assigned to convulsions among the appropriate cause-of-death categories may be the reason for some of the poor correlations found in our analysis of infant mortality.

RESULTS

The characteristics hypothesis

We used hazard models to examine the impact of religion on the risk of dying in childhood. The data file constructed for the analysis is a child-level file, i.e. each record is that of a child with the mother's and father's information attached. This means there is almost always more than one observation per family. Siblings share genetic traits, environment, resources, and behaviours. For example, some families may have emphasized personal hygiene more than others, and this could have had a substantial impact on mortality. The unmeasured stochastic variation or heterogeneity leading to an imperfect fit between explanatory variables and the outcome of interest partly stems from unmeasured family traits. The statistical effect of ignoring the correlation within families could lead to biased estimates of the parameters and their standard errors. To measure the influence of uncontrolled family traits, a random 'family' effect is included in the statistical models. As well as estimates of risk ratios, we will also present estimates of the standard error (σ) of the random effect using the statistical package *aML* (Lillard and Panis 2000).

We analyzed post-neonatal and early-childhood mortality separately. The first model in Table 1 presents risk ratio estimates for the effect of religion on post-neonatal mortality. Catholic post-neonatal mortality is 23 per cent above that of the reference group, the Dutch Reformed. Jewish infants have a death risk more than 30 per cent below that of the reference group. The mortality of other religious groups is even lower.

In the second model in Table 1 control variables are added. Exposure and resistance to health-threatening factors may have been influenced by variations in socio-economic characteristics. These might have included the availability of food; quality of water, clothing and bedding; size and quality of housing; and access to hygienic and preventive care. As a measure of the socio-economic position of the family, we use the occupational information on the father. In addition, birth cohort is included in the model. In this way we are able to capture the important changes in mortality which took place over time. We also control for several demographic variables. High birth orders and a large number of births often imply short birth intervals, which are associated with neglect and early weaning. Finally, we control for birth season, because infant mortality peaked in the summer.

The main question in which we are interested is whether Jews kept their advantage and Catholics their disadvantaged position compared with the Dutch Reformed after controlling for the above-mentioned factors, and in particular for socio-economic characteristics. The results show that the socio-economic status of the family affects mortality rates. Yet, Jewish death risks are even lower with the controls, while Catholics death risks are almost unchanged. The death risks of other religious groups are slightly higher than in the first model.

The second model also shows that boys have significantly higher death risks than girls. The year in which the child was born (and during which it spent the most risky period of its life) had a strong effect on mortality rates. Because 1855–69 was used here as the period of reference, the time variable indicates that death rates declined over time. The birth order of the child had an effect in the expected direction with high parities showing higher death risks. Infants born in spring had a higher death risk.

Table 2 presents the results of a hazard analysis of early-childhood mortality. By early childhood, Catholic children have about the same risk as Dutch Reformed children. Jewish children have a risk that is 18 per cent below that of Dutch Reformed. The risk of other religious groups is even lower. When we control for other variables, the difference between Catholics and the Dutch Reformed remains insignificant. The risk of Jewish children is even lower and that of other religious groups rises slightly. We also analyzed death risks among children aged 5–14. The results are very similar to those among children ages 1–4 and are therefore not presented here.

Thus it appears that differences in socio-economic and demographic characteristics do not explain much of the religious variation in post-neonatal and early-childhood mortality. If differences in socio-economic and demographic characteristics do not explain the religious differentials, differences in lifestyle or social isolation must do so.

The lifestyle hypothesis

In this section we test versions of the lifestyle hypothesis. The results are mixed. We first test the hypothesis that religious differentials in breastfeeding patterns explain part of the variation by religion in *infant* mortality. Unfortunately, without data on breastfeeding patterns, we are unable to test this hypothesis directly. However, some support for the breastfeeding hypothesis can be found by analysing seasonal mortality among infants by religion. In this section, the residual category of 'other' religious groups has been omitted because of insufficient numbers.

Table 1. *Hazard analysis of post-neonatal mortality, The Hague 1855–1912*

	Model 1		Model 2	
Variable	Risk ratio	*t*-statistic	Risk ratio	*t*-statistic
Religion				
Dutch Reformed (reference)	–	–	–	–
Jewish	0.641	–5.01	0.607	–5.01
Roman Catholic	1.213	2.45	1.200	2.23
Other religions	0.567	–2.88	0.643	–2.20
Sex				
Male			1.309	4.60
Occupational group				
Upper class			0.559	–3.06
Petty bourgeoisie			0.894	–0.88
Professionals and lower civil servants			0.657	–2.21
Farmers and fishermen			0.952	–0.27
Skilled labourers			0.818	–1.65
Unskilled labourers (reference)			–	–
Without occupation and Unknown			0.705	–2.58
Birth cohort				
1855–1869 (reference)			–	–
1870–1879			0.880	–0.94
1880–1889			0.987	–0.10
1890–1899			0.867	–1.07
1900–1909			0.575	–3.84
1910–1912			0.219	–4.41
Parity				
1 (reference)			–	–
2–5			1.436	4.50
6+			1.940	6.98
Seasonality of birth				
January-March			1.069	0.80
April-June			1.228	2.59
July-September			1.135	1.54
October-December (reference)			–	–
σ	0.703	13.80	0.666	11.46
Log Likelihood				
Initial	–11233.20		–11233.20	
Final	–11145.42		–11078.44	

Source: Population Registers, The Hague.

As explained above, we are forced to limit the analysis of infant mortality to the post-neonatal period. Figure 1 presents religious differentials in post-neonatal mortality rates by period. Religious differentials seem to have converged to a large extent by the early decades of the twentieth century. Among Catholics there was a temporary upsurge in the 1890s. The reasons for this upsurge are unclear. Superimposed on the trends in *post-neonatal* mortality by religion are trends in cause-specific *infant* mortality for two major categories of cause of death – water-borne and food-borne diseases grouped together and airborne diseases. Figure 1 shows that trends in Catholic and Protestant post-neonatal mortality reflect trends in infant mortality caused by water-borne and food-borne diseases to a certain extent, while there is much less of a resemblance between trends in Jewish post-neonatal mortality and trends in mortality from those diseases. In particular, the upsurge in Catholic post-neonatal mortality occurred at about the same time as a rise in mortality from water-borne and food-borne diseases among infants. The sensitivity of Catholic infants to water-borne and food-borne diseases could be due to a decline in breastfeeding. There are at least three mechanisms by which breastmilk benefits infant and child health: (i) it has immunological properties that provide protection against

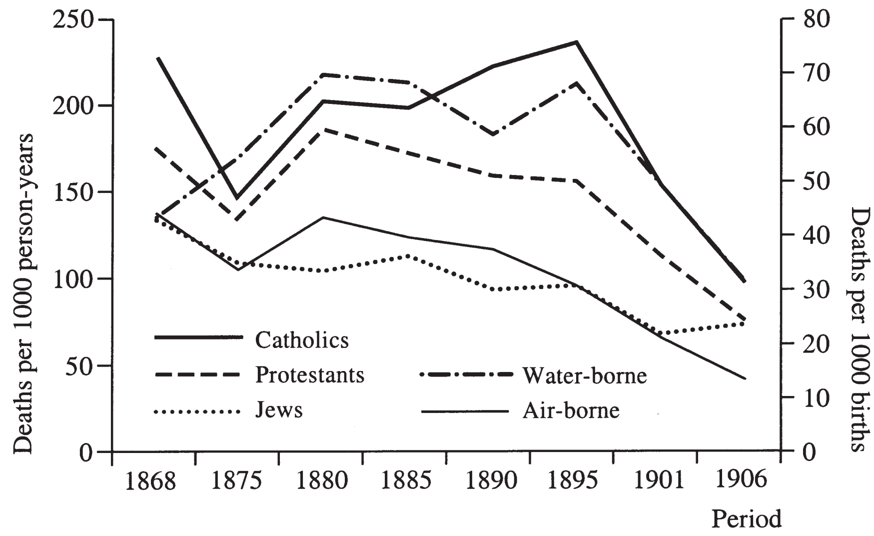
Table 2. *Hazard analysis of early-childhood mortality (1–4), The Hague 1855–1912*

Variable	Model 1 Risk ratio	Model 1 *t*-statistic	Model 2 Risk ratio	Model 2 *t*-statistic
Religion				
Dutch Reformed (reference)	–	–	–	–
Jewish	0.808	–1.98	0.711	–2.69
Roman Catholic	1.022	0.22	1.030	0.29
Other religions	0.542	–2.21	0.622	–1.75
Sex				
Male			1.179	2.11
Occupational group				
Upper class			0.603	–2.10
Petty bourgeoisie			0.882	–0.78
Professionals and lower civil servants			0.748	–1.21
Farmers and fishermen			1.059	0.25
Skilled labourers			0.912	–0.62
Unskilled labourers (reference)			–	–
Without occupation and Unknown			0.642	–2.51
Birth cohort				
1855–1869 (reference)			–	–
1870–1879			0.705	–2.37
1880–1889			0.609	–3.47
1890–1899			0.403	–5.80
1900–1909			0.239	–8.04
1910–1912			0.284	–3.41
Parity				
1 (reference)			–	–
2–5			1.624	4.39
6+			2.269	6.43
σ	0.667	9.18	0.535	6.22
Log Likelihood				
Initial	–7516.41		–7516.41	
Final	–7495.50		–7426.80	

Source: Population Registers, The Hague.

gastro-intestinal and respiratory disease; (ii) it meets all the infant's nutritional requirements for at least the first six months of life; and (iii) it is sterile (Forste 1994, pp. 497–98). Being sterile, breastmilk is particularly important in protecting infants from water-borne and foodborne diseases. This supports the suggestion of Aalberse (1917) that the upsurge in Catholic post-neonatal mortality was the result of the decline in breastfeeding among Catholics.

We do not have cause-of-death data by religion. Instead we computed post-neonatal mortality rates by season by religion. Previous research has shown that much can be learned about causes of death from an analysis of the seasonality of mortality, because gastric disease is more common in the summer, while respiratory disease is more common in the winter (see for example Landers 1993). Figure 2 presents the number of post-neonatal deaths in each season per 1000 person-years lived by religious group. The total for the four seasons adds up to the post-neonatal mortality rate for the whole year. In order to stress seasonal peaks, five seasons are shown, the first and the last being identical. Figure 2 shows that religious differentials are largest in the summer months. Catholic and Protestant post-neonatal mortality peak in the summer, while Jewish post-neonatal mortality peaks in the winter. This suggests that Catholic and Protestant infants suffered more from gastric disease. Below-average breastfeeding among Catholics and above-average breast-feeding among Jews could explain these findings. The religious differentials in the seasonal pattern of deaths are probably not simply the result of religious differentials in the seasonal pattern of



Note: the length of periods, which are indicated by the first year, changes slightly over time.

Figure 1. Post-neonatal deaths per 1000 person-years by religion and cause-specific infant mortality rates (per 1000 births), The Hague 1868–1910.
Source: Cause-of-death statistics from the Netherlands Central Bureau of Statistics and Ministry of the Interior.

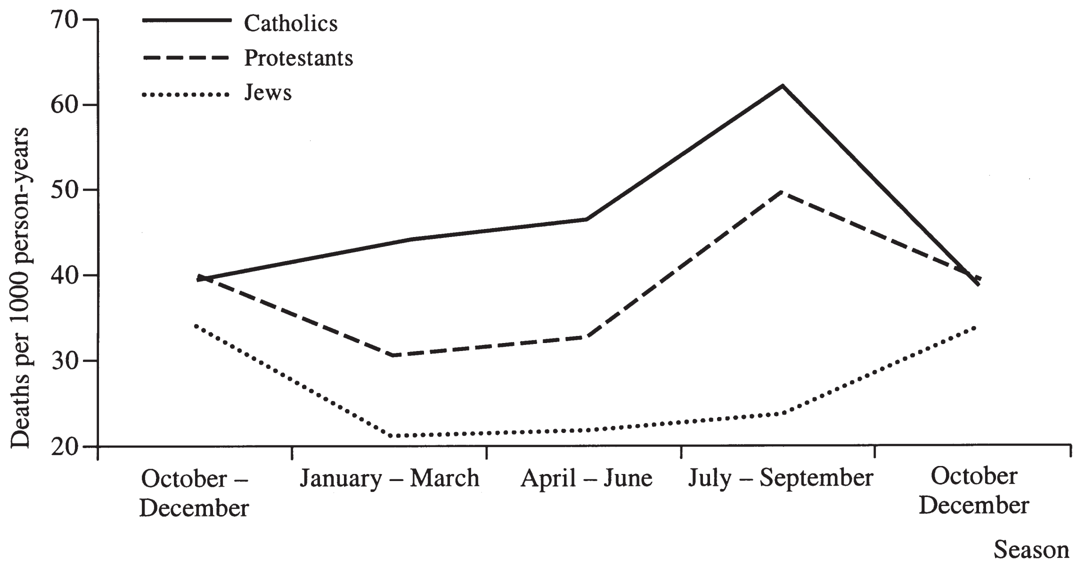
births, since a comparison of the models in Table 1 shows that seasonality of birth does not account for much of the religious variation in post-neonatal mortality. Thus our analysis of religious differentials in post-neonatal mortality by season suggests that the breastfeeding hypothesis is consistent with the data.

After the first year, breastmilk benefits the child's health much less (Forste 1994, p. 509). Therefore, we now turn to the hypothesis that personal hygiene explains some of the religious differentials in mortality after infancy. If personal hygiene explains the low mortality among Jews, then we would expect Jewish mortality from water-borne and food-borne diseases to be lower than that among religious groups, because habits of personal cleanliness, such as hand-washing before meals and ritual bathing, should particularly affect mortality from these diseases.

Figure 3 presents estimates of early-childhood mortality ($_4m_1$) by religion. After the first year very small differences remain between Catholics and Protestants. The main difference in this age group is between Jews and these two groups combined. Mortality in early childhood declines over time in all religious groups. Thus, by the beginning of the twentieth century little is left of the relative advantage of Jews.

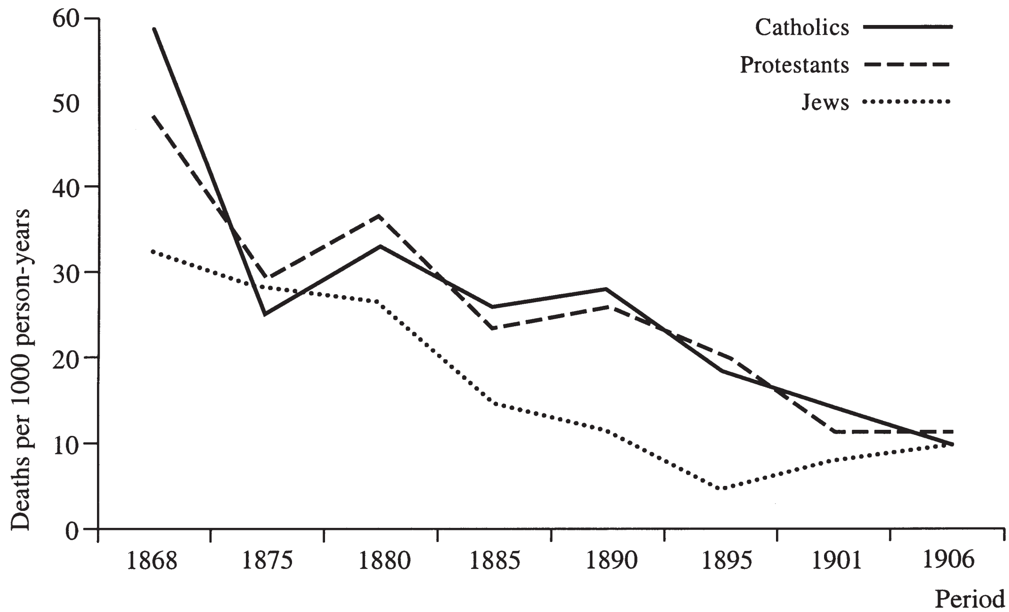
Again, we do not have cause-specific mortality rates by religion. However, since Protestants and Catholics together make up the vast majority of the population, we do have a good idea of the relative importance of water-borne and food-borne diseases in early childhood. Figure 4 compares early-childhood mortality among Protestants with early-childhood mortality from the combined category of water-borne and food-borne diseases in the population as a whole. This comparison suggests that air-borne disease explains much of the trend in mortality in this age group among Protestants. The conclusion for Catholic children is very similar (Figure not shown). Mortality from water-borne and food-borne diseases may have been close to zero among Jewish children aged 1–4. However, this would not have been enough to explain the low mortality among Jewish children in this age group because mortality in this cause-of-death category among other children was not very high either.

Further evidence for the marginal role played by water-borne and food-borne diseases in religious differentials in early-childhood mortality is provided by an analysis of seasonal mortality. As we mentioned before, gastric disease spread by contaminated food and water peaks in the summer months. Thus, if personal hygiene matters, one would expect religious differentials to be largest during the months of July, August, and September (Landers 1993, pp. 215–217). Figure 5 presents religious differentials in early-childhood mortality by season. Among none of the religious groups does early-childhood mortality peak in the summer months, and differences between Jews and Protestants in



Note: the last quarter, October-December, has been presented twice.

Figure 2. Post-neonatal mortality by religion and season, The Hague 1855–1910.
Source: Population Registers, The Hague.

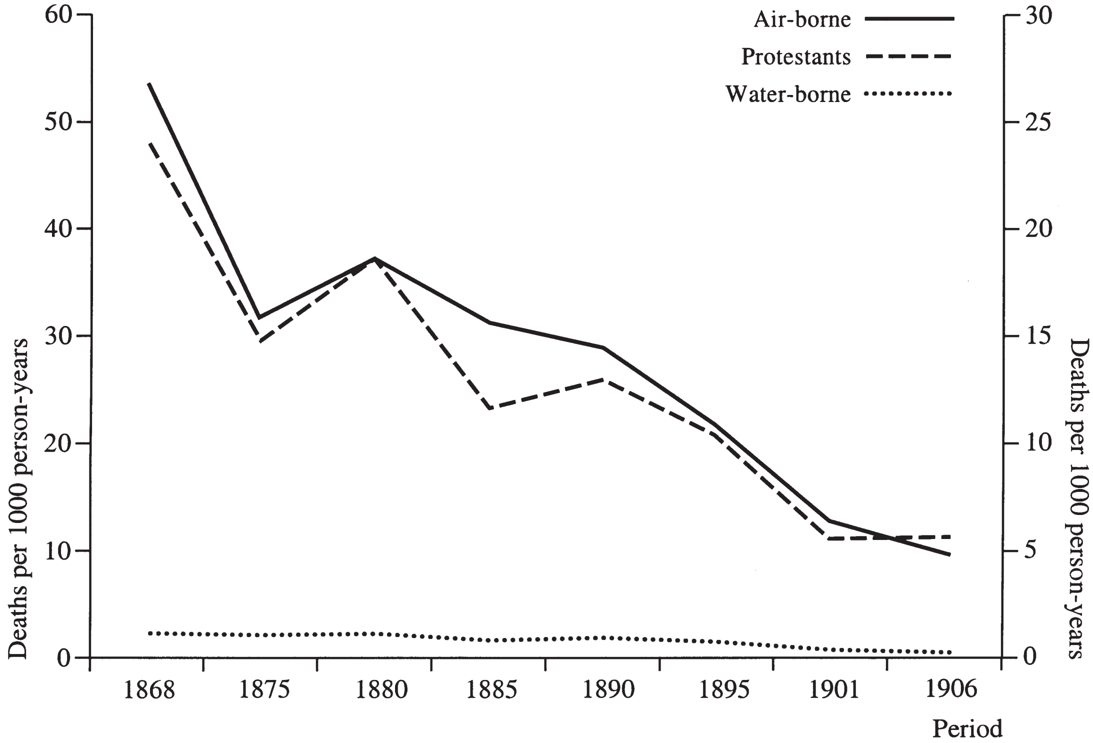


See note under Figure 1.

Figure 3. Trends in early-childhood mortality ($_4m_1$), The Hague 1868–1910.
Source: Population Registers, The Hague.

early-childhood mortality are smallest in the summer months.

In short, we found mixed results for the lifestyle hypothesis. The breastfeeding hypothesis seems to be consistent with data on post-neonatal mortality and therefore could account for some of the religious differentials in the first year. However, after infancy the breastfeeding hypothesis is unlikely to explain much of the religious variation. The same applies to the personal hygiene hypothesis in this age group.



See note under Figure 1.

Figure 4. Trends in Protestant early-childhood mortality (left Y-axis) and early-childhood mortality from airborne disease (right Y-axis), The Hague 1868–1910.
Source: As Figure 1.

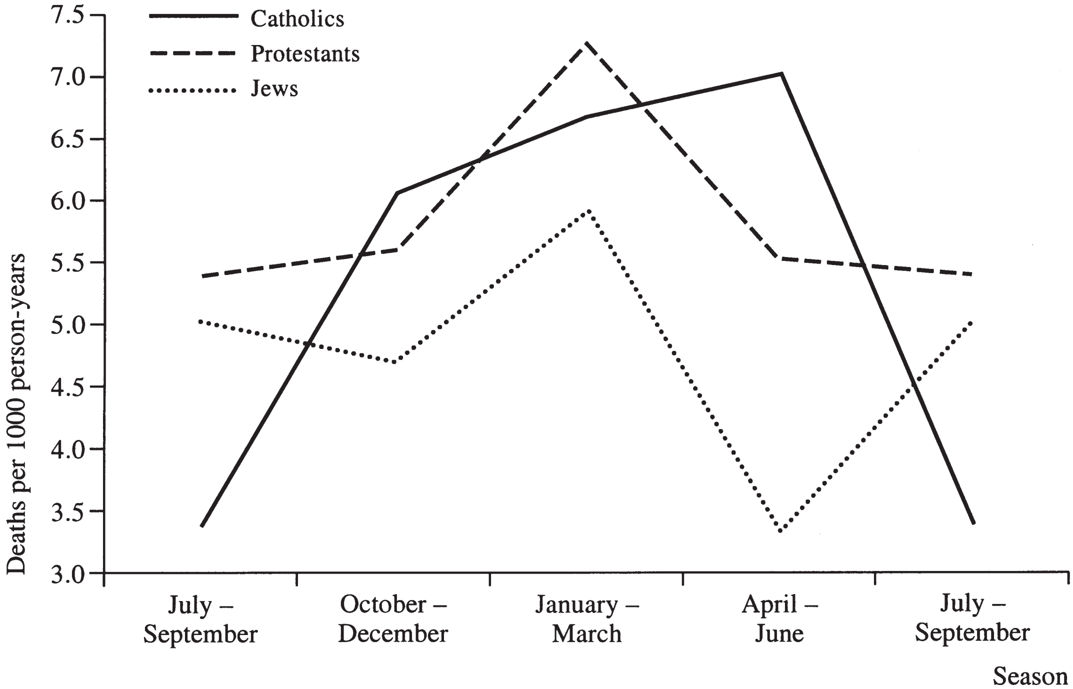
Social isolation

It is impossible to test the social isolation hypothesis with the kind of data we have. Instead of real data we use a simulation study to show that this hypothesis should not be overlooked in attempts to explain the significantly lower mortality of small and relatively isolated religious groups. The simulation study was performed on an initial cohort of 1000 individuals all of whom are susceptible to an airborne disease, which is introduced at the beginning of the study. A larger cohort size would make the assumption of homogeneous mixing within groups too unrealistic. A certain fraction of the population belongs to a minority religious group.

We start the simulation with two primary cases only, one in each group, and follow the spread of disease week by week to the time when the disease has disappeared. Thus, all other cases are secondary. We assume a simple compartmental model in which infected individuals pass on the disease to uninfected and, hence, susceptible individuals and all infected individuals either recover and obtain lifetime immunity or die.

We borrowed from a model with spatial heterogeneity, substituting religious groups for urban and rural areas. In this model secondary cases are created by contact with infected individuals at an infection rate $\lambda_i = \beta_{i1} .Y_1 + \beta_{i2}.Y_2$, where Y_i is the number of infected individuals in group i and β_{ij} is a transmission parameter between individuals in group i and j (Anderson and May 1991, p. 307). The transmission parameter is not dependent on density, because we are interested in religious groups that are urban and we assume that both groups have about the same population density. We assume that interaction between children in neighbourhoods of different density is relatively rare. For example, we assume that Jewish children, many of whom lived in a high-density neighbourhood, mostly interacted with other children who lived in the same neighbourhood or adjacent neighbourhoods with a similar density. Because lower-density neighbourhoods are often wealthier than higher-density ones, our assumption implies few contacts between children belonging to different socio-economic classes.

Our model states that when individuals belong to the same group $\beta_{11} = \beta_{22} = \beta$, but when they belong to different groups $\beta_{12} = \beta_{21} = \varepsilon\ \beta$, where ε is an isolation parameter. In other words, our model assumes that individuals have less contact with individuals from the opposite group. Our model implies



Note: the third quarter, July-September, has been presented twice.

Figure 5. Early-childhood mortality by religion and season, The Hague 1855–1910.
Source: Population Registers, The Hague.

that individuals in the minority group have contact with fewer total persons than those in the majority group. We did not include a segregation parameter explicitly, although Jews tended to concentrate in one of the poorer high-density neighbourhoods (Van Creveld 1989). Instead, we assume that segregation influences the infection rate through social isolation. For most infections the direct measurement of the transmission parameter is essentially impossible (Anderson and May 1991, p. 63). Hence, the transmission parameter has been set at $\beta = 0.0024$ pro hoc. Thus, we assume that there are no differences in within-group infection rates, unlike the lifestyle hypothesis, which assumes that $\beta_{11} \neq \beta_{22}$. We have assumed that one out of ten children is still infected after a week, which implies an infectious period of 7-8 days or about one day more than measles (Anderson and May 1991, p. 31).

Time is discrete in our model. If X_{it} is the number of susceptibles in group i in week t, then the number of infected in the same group in week t will be $Y_{it} = \lambda_i X_{it}$. Note that λ_i is a function of the number of those infected in the previous week in both groups. The remaining number of susceptibles in the next week is obtained by simply subtracting the number of infected from the number of susceptibles. Eventually, the number of susceptibles declines and with it the number infected. The remaining number of susceptibles is much less likely to interact with the dwindling number of infected than with the rising number of recovered and immune individuals. At a certain stage the rate of transmission declines below unity and the spread of the disease stops. Figure 6 shows an example of one simulation, when the isolation parameter $\varepsilon = 0.3$ and the fraction belonging to the minority group $f = 0.3$. It shows the percentage 'ever-infected' or the percentage either dead or recovered every week by group. In the majority group, the percentage of ever-infected reaches a maximum of 89 percent, while among the minority group it is 68 percent. It should be noted that this result is independent of the group to which the primary cases belong. In the simulation the spread of the disease in the cohort was assumed to have started with two primary cases, in each group. When the simulation starts with only one primary case in either of the two groups, the results are very similar. In this example, the ratio of the percentage 'ever-infected' in the minority group to that in the majority group is 0.77.

The ratio of the percentage 'ever-infected' in the minority group to that in the majority group may be still smaller. Table 3 shows the ratio of the percentage 'ever-infected' in the minority group to that in the majority group as a function of the isolation parameter ε and the fraction belonging to the

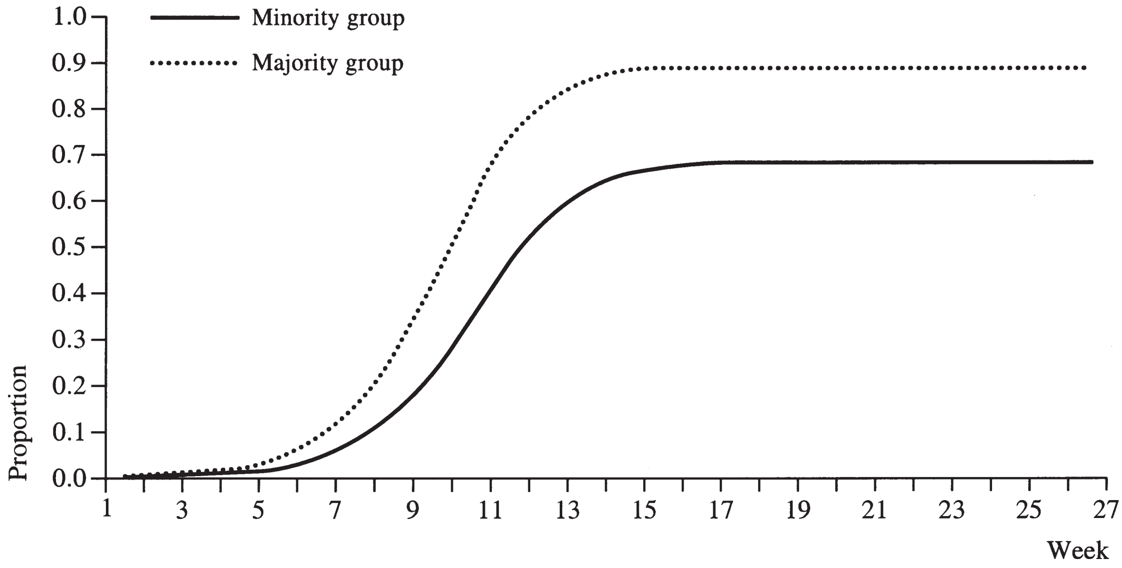


Figure 6. Proportion 'ever-infected' in two hypothetical subpopulations as a function of time elapsed (in weeks) since the introduction of the first cases.

Table 3. *Simulation study of the spread of an infectious disease in a minority group: ratio of 'ever-infected' in minority group relative to those in majority group by isolation parameter (ε) and fraction of initial cohort belonging to minority group.*

	Fraction of initial cohort belonging to minority group			
ε	0.1	0.2	0.3	0.4
0.1	0.296	0.330	0.529	0.735
0.2	0.498	0.558	0.638	0.790
0.3	0.628	0.693	0.769	0.871
0.4	0.746	0.796	0.846	0.921
0.5	0.824	0.858	0.900	0.946

minority group. The simulation study shows that both parameters have a strong effect on relative morbidity. When f = 0.1 and ε = 0.1, the ratio is less than 0.30. The simulation study also shows that the effect of isolation is small to non-existent when the fraction of the initial cohort belonging to the minority group reaches 50 percent or more.

CONCLUSION AND DISCUSSION

Like previous studies, our hazard analyses show that socio-economic and demographic characteristics do not explain any of the religious differentials in infant or child mortality. This leaves two kinds of hypothesis to explain the differentials. We found that one version of the lifestyle hypothesis is consistent with data for the first year. Differences in breastfeeding patterns may explain the mortality differences found among infants. Unfortunately, we were unable to incorporate any of the Protestant minority groups into our analysis of seasonal mortality to test whether part of their relatively low infant mortality was due to breastfeeding. After infancy, we did not find any mortality differences between the two major religious groups, Catholics and the Dutch Reformed. In this age group, the major difference was between the two larger denominations, Catholics and the Dutch Reformed, on the one hand, and other smaller denominations on the other. Of course, breastfeeding is unlikely to account for differences after infancy. We tested one other lifestyle hypothesis: that differences in personal hygiene protected Jews from disease. Provided that differences in seasonal mortality reflect underlying differences in causes of death to a certain extent, our analysis of seasonal mortality suggests that this hypothesis is unlikely to account for much of the mortality variation after infancy. This leaves us with an unknown version of the lifestyle hypothesis and the social isolation hypothesis as possible explanations of the religious differentials after infancy, provided the influence of physicians on the survival of children was insignificant. If the isolation of the Jewish community and, in particular, the partial isolation of its infants and young children from other religious groups is a possible explanation for their relatively low mortality, as suggested by our simulation, then other socially isolated religious minority groups should also have enjoyed relatively low mortality. Such groups are underrepresented in our sample and make up our category of 'other religious groups'. The hazard analysis shows that their mortality is very low, that it resembles that of the Jewish population, and that it cannot be explained away by the characteristics hypothesis. Perhaps Christian minority groups had a lifestyle that protected them from infectious disease to a certain extent. If so, we do not know the features of this lifestyle, apart from social isolation.

NOTES

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