The epidemiological transition in The Netherlands

The work reported in this thesis was carried out at the Department of Public Health, Erasmus University Rotterdam, which participates in the Netherlands Institute for Health Sciences (NIHES). NIHES has been recognized by the Royal Netherlands Academy of Arts and Sciences since 1992.

Financial support for the publication of this thesis was provided by the Department of Public Health, Erasmus University Rotterdam

Wolleswinkel-van den Bosch, Judith

The epidemiological transition in The Netherlands / Judith Wolleswinkel-van den Bosch
Thesis Erasmus University Rotterdam. - With Ref. - With summary in Dutch

ISBN 90-9012155-2

Key words: epidemiological transition / mortality / causes of death / trends / determinants / The Netherlands

Cover design: Arjan Schoonhoven, Schiedam
Layout: Bon Mot, Rotterdam
Printed by: Grafisch bedrijf Ponsen en Looijen b.v., Wageningen

The epidemiological transition in The Netherlands

De epidemiologische transitie in Nederland

Proefschrift

ter verkrijging van de graad van doctor
aan de Erasmus Universiteit Rotterdam
op gezag van de rector magnificus
Prof.dr P.W.C. Akkermans M.A.
en volgens besluit van het College voor Promoties.

De openbare verdediging zal plaatsvinden op
woensdag 25 november 1998 om 13:45 uur

door

Judith Heleen Wolleswinkel-van den Bosch

geboren te Ermelo

Promotor	:	Prof.dr J.P. Mackenbach
Overige leden	:	Prof.dr A. Hofman Prof.dr M.J. van Lieburg Prof.dr F.W.A. van Poppel

CONTENTS

The chapters 4 - 9 are based on the following papers and manuscripts:

Chapter 4
Wolleswinkel-van den Bosch JH, Poppel FWA van, Mackenbach JP. Reclassifying causes of death to study the epidemiological transition in The Netherlands, 1875-1992. European Journal of Population 1996, 12: 327-361*

Chapter 5
Wolleswinkel-van den Bosch JH, Poppel FWA van, Tabeau E, Mackenbach JP. Mortality decline in The Netherlands 1850-1992: A turning point analysis. Social Science and Medicine 1998, 47: 429-443†

Chapter 6
Wolleswinkel-van den Bosch JH, Poppel FWA van, Looman CWA, Mackenbach JP. Cause-specific mortality trends in The Netherlands, 1875-1992: A formal analysis of the epidemiological transition. International Journal of Epidemiology 1997, 26: 772-781‡

Chapter 7
Wolleswinkel-van den Bosch JH, Poppel FWA van, Mackenbach JP. The contribution of infectious diseases to mortality decline in The Netherlands, 1875-1970. (submitted for publication)

Chapter 8
Wolleswinkel-van den Bosch JH, Poppel FWA van, Looman CWA, Mackenbach JP. Cultural and economic determinants of mortality decline in The Netherlands, 1875/79 to 1920/24: a regional analysis. (submitted for publication)

Chapter 9
Wolleswinkel-van den Bosch JH, Poppel FWA van, Looman CWA, Mackenbach JP. Determinants of infant and early childhood mortality levels and decline in The Netherlands in the late 19th century. (submitted for publication)

Chapter 1

INTRODUCTION AND OBJECTIVES

MORTALITY HAS DECLINED drastically in many countries throughout the world over the past centuries. Life expectancies doubled, infant and child mortality declined strongly and mortality shifted to older ages. This phenomenon started in the 18th or 19th centuries in western industrialised countries and it was observed in other countries as well, although in a later period of time and at a different pace of decline. This common pattern of mortality decline observed in western countries has led to the formulation of the demographic transition theory and, later, to the formulation of the epidemiological transition theory, which is the subject of this study.

The demographic transition theory as described by Notestein in 1944 and 1953 is conventionally accepted to be the first formulation of the demographic transition theory (Chesnais 1992, Kirk 1996, Notestein *et al.* 1944, Notestein 1953). It explains population changes with the transition of birth and death rates from a stage in which mortality and birth rates are high to a stage in which mortality and birth rates are low. In most countries mortality declines preceded fertility declines (Coale 1986). In 1971, Omran introduced the epidemiological transition theory: a theory of the epidemiology of population change (Omran 1971). One might say that this theory concentrates on mortality decline within the demographic transition theory. Both in the demographic and the epidemiol-

ogical transition theory, 'modernisation' is considered to be the reason for mortality (and fertility) decline.

Omran described three stages of different cause-of-death patterns that accompany mortality change during the transition: 'the age of pestilence and famine', 'the age of receding pandemics' and 'the age of degenerative and man-made diseases'. Like the demographic transition theory, Omran's epidemiological transition theory was based on the experience of mortality decline in western countries (Europe and the United States) in the late 18th to 20th centuries. Omran generalised the epidemiological transition theory to other parts of the world by describing three models for the timing of the epidemiological transition: 'the classical or western model', 'the accelerated model', and 'the contemporary or delayed model' (Omran 1971, 1977a,b, 1983).

The concept of epidemiological transition has proved to be a very attractive concept to public health researchers. The epidemiological transition theory gives a stylised description of historical mortality change in western industrialised countries. In many studies on contemporary health changes, the concept of epidemiological transition is used to give a short description of historical epidemiological changes. The theory of epidemiological transition has also acquired a place in the training of medical and public health students (Van der Maas & Mackenbach 1995). The popularity of the epidemiological transition theory might also be explained by the fact that it is one of the few existing theories in public health. As such, the epidemiological transition theory has two important applications. First, it has been used to explain differences in cause-of-death patterns between countries at certain points in time from different transition timings. Kunst (1997) used the idea of 'delayed transition' to explain differences in socio-economic gradient for ischemic heart disease mortality in southern European countries compared to northern European countries. In the Global Burden of Disease Study, causes of death were classified into three groups. A first group consisted of communicable diseases and maternal, perinatal and nutritional disorders, a second group consisted of non-communicable diseases and a third group of injuries. The epidemiological transition was then described as a shift from group 1 diseases to group 2 diseases. The ratio of group 2 deaths to group 1 deaths has been proposed as an indicator of progress along the path of the epidemiological transition (Murray & Lopez 1997, Frenk *et al.*1989).

Secondly, the epidemiological transition theory has also been used for projections of future health needs in countries that are falling behind in the transition compared to e.g. western industrialised countries. Examples of studies in which future health needs in developing countries are based on historical changes in health patterns in developed countries are, for example, the papers of Schooneveldt *et al.* (1988) and Escovitz (1992). In the first paper, the cause-of-death pattern in Nauru, a Pacific island, is compared to that of the Australian

population. One of the conclusions of the paper is that cancer and cardiovascular mortality levels in Nauru are not yet as high as Australian levels, and therefore a continuing increase of mortality due to those causes is expected. Escovitz's paper points out the future need for internists from western countries in developing countries. The argument is that 'these changes (i.e. demographic and epidemiological changes), which are often accompanied by increasing industrialisation and urbanisation, are creating health problems similar to those in the 'developed' world but are occurring in countries that have far fewer resources' (Escovitz 1992).

Another application of the epidemiological transition theory, related to the ability to project cause-of-death patterns, is the estimation of the cause-of-death structure in developing countries based on the historical mortality experience of western countries (Preston & Nelson 1974, Palloni & Wyrick 1981, Lopez & Hull 1982).

The idea, which arises from Omran's epidemiological transition theory, that mortality decline may differ between populations with respect to timing and pace, but shows equal cause-of-death patterns, offers possibilities for the above-mentioned applications, and probably best explains the attractiveness of the epidemiological transition theory. The theory has, however, also met criticism. Additional phases to the transition have been suggested by some researchers (Olshansky & Ault 1986, Rogers & Hackenberg 1987), and it can be questioned whether the epidemiological transition is a theory, or only a generalisation of mortality experiences in western countries (Smiths 1994, Kirk 1996). On the other hand, it has also been argued that the concept of epidemiological transition should be included in health planning (Philips 1991, 1994). To do so, a good understanding of (historical) epidemiological changes in different populations is necessary, as well as the disentanglement of the determinants of change.

The debate about the determinants of mortality decline is still very much alive. The publications of Thomas McKeown (McKeown & Record 1962, McKeown *et al.* 1972, McKeown 1976a,b) in which he argued that improvements in living standards, particularly nutrition, was the most important factor in mortality decline in the 19th and early 20th centuries, turned the prevailing view on determinants of mortality decline (i.e. medical care and technology were considered to be the most important) upside down. McKeown's view, however, has been challenged by many other researchers (Razzel 1974, Szreter 1988, Mercer 1990). Extensive knowledge of the factors that reduced mortality in countries that went through the epidemiological transition is of great interest to public health. This knowledge could be used to outline public health policy in countries where mortality levels are still relatively high. Besides, understanding the determinants of mortality and morbidity change might also be useful to anticipate future changes in disease and mortality patterns.

The epidemiological transition theory has a prominent place in public health studies, but the theory has also received criticism, and the debate about the determinants of mortality decline is still open. Therefore, it seems to be the right time for a renewed scrutiny. This thesis might contribute to this scrutiny by presenting a detailed analysis of the epidemiological transition in one country, i.e. The Netherlands.

The objective of this thesis is, first, to present a detailed description of the epidemiological transition in The Netherlands with respect to the onset of, and accelerations or decelerations in all-cause mortality decline, with respect to cause-specific mortality trends, and the contribution of infectious diseases to mortality decline. Secondly, this thesis will study the relative importance of determinants of mortality decline in The Netherlands in the late 19th and early 20th centuries. Thirdly, the epidemiological transition theory will be re-examined on the basis of the results for The Netherlands.

An overview of existing views on epidemiological transition theory and on the explanation of mortality decline is given in two further introductory chapters. *Chapter 2* contains an extensive description of the epidemiological transition theory as well as the weaknesses in and criticism on this theory. *Chapter 3* contains an overview of the existing hypotheses on determinants of mortality decline.

In order to present sound results with respect to changing cause-of-death patterns, cause-of-death categories have been constructed that remain constant over time with respect to their nosological content. In The Netherlands, 10 different cause-of-death classifications were used in the period 1875-1992: one Dutch 19th-century classification and nine editions of the International Classification of Diseases and Causes of Death. *Chapter 4* describes the reclassification of these 10 cause-of-death classifications into nosologically continuous cause-of-death groups for the period under study in this thesis.

Chapter 5 presents an analysis of turning points with respect to total, sex-specific and age-specific mortality decline in The Netherlands in the period 1850-1992. In this chapter the onset, phases and end of the epidemiological transition are discussed. Changes in the pace of cause-specific mortality decline are also presented for the phases marked by the turning points in the total mortality trend. Possible determinants of mortality decline are discussed using the information on the trends of cause-specific mortality.

In *Chapter 6*, results are presented of a study to elucidate the changing cause-of-death pattern in The Netherlands in the period 1875-1992. In this chapter clusters of causes of death with the same pattern of rise and decline over time are described. Phases in this changing cause-of-death pattern are suggested, and possible determinants that underlie the course of the distinguished clusters are discussed.

In *Chapter 7*, the contribution of infectious diseases to total mortality decline is analysed for the period 1875-1970 and for four subperiods. The results of this analysis point to the importance of specific infectious diseases in absolute mortality decline in the different periods. Changes in the pace of cause-specific mortality trends from one period to another are presented to find out which determinants might have played a role in the decline of specific infectious diseases. And, relating this information to that on the contribution of infectious diseases to mortality decline, it is discussed which determinants might have contributed most to total mortality decline.

In chapters five to seven, possible determinants of mortality decline are discussed on the basis of the results of descriptive analyses. In chapters eight and nine, however, the determinants of mortality decline in the late 19th and early 20th centuries are studied on the basis of explanatory analyses. The relative importance of economic and cultural determinants of total mortality decline is studied in *Chapter 8*. Studies on mortality decline have very much focused on economic factors, e.g. rising living standards. Several researchers mentioned the role of cultural determinants, but so far these have hardly been studied. *Chapter 9* focuses on the explanation of infant and early childhood mortality (decline) in the late 19th -century. Infant and early childhood mortality played an important role in mortality decline and many studies on determinants of mortality decline specifically addressed these age groups.

In the last chapter of this thesis, the studies for The Netherlands will be placed in a wider context. In *Chapter 10* the results for The Netherlands are discussed in the light of Omran's epidemiological transition theory and a comparison is made with other countries. A reappraisal of the epidemiological transition theory is given based on the results in the other chapters of this thesis and the international literature. Recommendations for further research are given and the policy implications of the epidemiological transition theory are discussed.

Chapter 2

EPIDEMIOLOGICAL TRANSITION THEORY

SINCE OMRAN PUBLISHED his theory of epidemiological transition in 1971, the epidemiological transition has been studied in many countries. The extensive study of the epidemiological transition brought to light weaknesses in the original theory, and additional phases and multiple transitions were suggested by several researchers. In this chapter, the original epidemiological transition theory, the weaknesses in this theory and amendments to the theory are described.

2.1 The epidemiological transition theory as described by Omran

In 1971 Omran introduced what he called the epidemiological transition theory or, in other words, a theory on the epidemiology of population change. He states: 'Conceptually, the theory of epidemiological transition focuses on the complex change in patterns of health and disease and on the interactions between these patterns and their demographic, economic and sociologic determinants and consequences'. Omran formulated five propositions in the epidemiological transition theory (Omran 1971). The exact formulation in his seminal paper of 1971 is cited below. In 1983, Omran published a preliminary update of the

epidemiological transition theory. In the description of the propositions, I will point out any changes in the updated version compared to the original version.

1. 'Mortality is a fundamental factor in population dynamics'.
2. 'During the transition, a long-term shift occurs in mortality and disease patterns whereby pandemics of infection are gradually displaced by degenerative and man-made diseases as the chief form of morbidity and primary cause of death'. This displacement takes place in 3 phases: '*The age of pestilence and famine* in which mortality is high and fluctuating, thus precluding sustained population growth. Average life expectancy at birth is low and variable, vacillating between 20 and 40 years. *The age of receding pandemics* in which mortality declines progressively, the rate of decline accelerates as epidemic peaks become less frequent and eventually disappear, average life expectancy at birth increases steadily from about 30 to 50 years' (55 years in the updated version (Omran 1983)); 'population growth is sustained and begins to describe an exponential curve'. During the latter part of this phase, fertility starts to decline (Omran 1983). '*The age of degenerative and man-made diseases* in which mortality continues to decline and eventually approaches stability at a relatively low level. The average life expectancy at birth rises gradually until it exceeds 50 years' (70 years in the updated version (Omran 1983)). 'It is during this phase that fertility becomes the crucial factor in population growth'.
3. 'During the epidemiological transition the most profound changes in health and disease patterns occur among children and young women'.
4. 'The shifts in health and disease patterns that characterise the epidemiological transition are closely associated with the demographic and socio-economic transition that constitute the modernisation complex'.
5. 'Peculiar variations in pattern, pace, determinants and consequences of population change are differentiated in three basic models of the epidemiological transition: the classical or western model (western countries), the accelerated model (e.g. Japan, eastern European countries), and the contemporary or delayed model (developing countries)'. In the updated version of the transition theory a transitional version of the delayed model (e.g. Singapore, Hong Kong) has been added (Omran 1983). In the classical model mortality started to decline gradually in the 18th or 19th -centuries, but after the turn of the 20th-century mortality decline accelerated. The 'age of degenerative and man-made diseases' is placed in the second and third decade of the 20th-century. Mortality declines were accompanied by fertility declines. In the accelerated model gradual mortality decline started in the second half of the 19th-century. The period taken to complete the transition was much shorter as compared to the classical model. The delayed model describes recent and yet-to-be completed transitions in developing countries. Mortality started to de-

cline in the first half of the 20th -century, but fertility levels remained high. In the transitional delayed model fertility started to decline a few decades after mortality decline (Omran 1971, 1983).

The aim of Omran's paper(s) on the epidemiological transition was twofold. First, he attempted to crystallise and disentangle the determinants and consequences of health and disease changes in a variety of social contexts. Secondly, he aimed to shed light on the population problems of less-developed countries and to provide information needed to treat those problems (Omran 1971, 1977a,b, 1983).

The data Omran used to support his theory were United Nation's model life tables showing mortality patterns at various life expectancy levels (developed and less-developed countries with an overrepresentation of the developed countries). He also used historical and contemporary data from Sweden and England and Wales as well as data from individual countries to illustrate the different transition models. Examples of the classical model are England and Wales and Sweden. Japan is an example of the accelerated model and Chile and Ceylon are examples of the contemporary or delayed model.

In the description of the classical model, the timing of the three phases for western countries is elucidated. The age of pestilence and famine prevailed during pre-modern and early-modern times. Mortality decline was slow and unsteady. More precipitous declines started around the turn of the century. The late phase of the epidemiological transition is placed in the second and third decades of the twentieth century (Omran 1971). Somewhere else in the paper he writes that the gradual shift in disease patterns characteristic of the classical transition can be seen in the steady decline of infectious diseases, such as tuberculosis and diarrhoeal diseases, and the moderate increase in cancer and cardiovascular diseases in England and Wales up to 1920. After the First World War (WWI) the decline of infectious diseases and rise of degenerative diseases is more distinct, and since 1945 the increase in cardiovascular diseases is particularly striking (Omran 1971). In the updated version of the epidemiological transition theory (Omran 1983), he writes that the 'age of degenerative and man-made diseases' started before WWI in western countries, and that it became much more manifest thereafter.

In this updated version, Omran is more explicit about the diseases and causes of death under the heading of degenerative and man-made diseases. Degenerative and man-made diseases include cardiovascular diseases, cancer, stroke, diabetes, metabolic disorders, radiation injury, accidents, occupational hazards, carcinogens in the environment and industry, food additives, stress-related diseases, such as mental illness and drug dependency (Omran 1983).

Although one of the aspects of the aim of the epidemiological transition theory was to disentangle the determinants of health and disease patterns, Omran does not discuss this subject to a great extent. He writes that 'detailed treatment' of determinants of the change in disease pattern is beyond the scope of his paper (Omran 1971, 1983). He mentioned, however, 3 categories of determinants involved in mortality decline viz. ecobiologic determinants (virulence diseases agents, host resistance), socioeconomic/political/cultural determinants (including standard of living, health habits, hygiene, nutrition), and medical and public health determinants (Omran 1971, 1977a,b, 1983). Socioeconomic factors were the primary determinants of the classical transition. These were augmented by the sanitary revolution in the late nineteenth century and by medical and public health progress in the twentieth century (Omran 1971).

In his paper on the preliminary update of the epidemiological transition theory Omran stressed the point that the distinguished models differ not only with respect to timing of the decline of mortality and fertility, but also with respect to the determinants of decline. In the western and accelerated models, modernisation is the key to mortality and fertility decline. In the delayed and transitional delayed models, the health technology input from other countries is the most important determinant of mortality decline (Omran 1983).

2.2 Weaknesses of and amendments to the theory of epidemiological transition

A thorough scrutiny of the epidemiological transition theory shows that the 'ages' in mortality decline are not as clearly defined as they may seem at first sight. In the following sections, we will discuss what we consider to be weaknesses in Omran's theory as well as the amendments to the theory suggested by other researchers (e.g. Olshansky & Ault 1986, Rogers & Hackenberg 1987) to further refine the epidemiological transition theory.

2.2.1 Definition of the 'ages' in mortality decline: cause-of-death pattern and timing

Omran's definition of the 'ages' in mortality decline was rather vague. The names of the phases have been formulated in terms of the prevailing diseases and causes of death, but the description of the characteristics of the 'ages' is given in terms of pace of mortality decline, life expectancy and population growth (Omran 1971, 1983). The timing of the phases is not very accurate either.

The age of pestilence and famine

Omran does not give a starting point for the 'age of pestilence and famine'. He only states that the age of pestilence and famine prevailed during pre-modern and early-modern times (Omran 1971). It suggests that the beginning of the first phase could be dated far back into pre-history, and that mortality had always been high and fluctuating until the onset of the second phase. We do not, however, have mortality data going that far back into history to check this assumption. Wrigley and Schofield showed, on the basis of mortality estimates from parish registrations, that there was a falling trend in life expectancy between the late 16th century and the late 17th century. They also showed that mortality fluctuated considerably during the early 18th century, and that around 1750 life expectancy was at the same level as a century before. In the period 1750-1850 there was an increase in life expectancy, which resulted in a life expectancy at birth in 1850 of only 1.8 years more than the life expectancy around 1600 (Wrigley & Schofield 1981). A similar pattern was reported for Geneva (Perrenoud 1984). These examples show that, at least in England and Geneva, there have been secular swings in the level of mortality in the pre-transitional phase and that the changes since 1750 were not unprecedented in history with respect to scale or level of mortality.

For many western countries we do not know the situation back in the, for example, 18th -century with respect to total mortality, not to mention the availability of data on cause-specific mortality to make well-founded statements on the cause-of-death patterns before the 19th century. Changes in disease and cause-of-death patterns, undoubtedly, also occurred during the pre-transitional phase. One might think of the effects that the change from hunter-gatherer society to agricultural settlements had on disease patterns (McNeill 1976, Lancaster 1990). For the ancient Greek world, for example, changes in mortality and disease patterns as a response to trading, urbanisation, and perception of cleanliness have been described (Grmek 1991).

The age of receding pandemics

This phase is explained as a phase in which epidemics recede and infectious diseases in general, such as tuberculosis and diarrhoeal diseases, decline. This phase is in fact the 'transition phase'. The naming of this phase refers to pandemics, but not all epidemics were pandemics. Cholera, for instance was a pandemic, but many other infectious diseases that played an important role in mortality decline were endemic diseases, e.g. tuberculosis, acute respiratory diseases and dysentery. The timing of this phase is not very clearly defined either. It starts after the 'early modern period'. The end of this phase can be determined from Omran's statements regarding the beginning of the third phase, which was placed in the second and third decades of the 20th century in the classical model. In another

publication, Omran gave a more specific indication of the onset of the third phase viz. before the First World War.

The age of degenerative and man-made diseases

Omran's statements on the onset of the 'age of degenerative and man-made diseases' were not unequivocal, and the description of the cause-of-death pattern was not very accurate either. In the first version of the epidemiological transition theory (1971). Omran does not give a clear definition of the diseases under the heading 'degenerative and man-made diseases'. In the updated version of 1983, he provides a list of diseases which is so general that it seems to include *all* non-infectious diseases. However, there are also non-infectious diseases that declined during a considerable part of the period of mortality decline, for example, stomach cancer and cerebrovascular disease (Whisnant 1983, LaVecchia *et al.* 1992).

Other researchers have tried to specify the causes of death, which increased during the epidemiological transition. Mackenbach (1994) distinguished two types of labels in the literature for the causes of death that increased during the epidemiological transition viz. labels referring to aetiology, pathogenesis or prognosis of these conditions, and labels referring to the presumed wider causes of the rise of those diseases. An example of the first group of labels is Omran's 'degenerative and man-made diseases'.

Examples of the second category of labels are 'civilisation-associated diseases' (Junge & Hoffmeister 1982), diseases of 'modernisation' (Marshall 1991), 'post transition diseases' (Fliess 1991), and 'western diseases'. Trowell and Burkitt introduced the concept of 'western diseases'. Western diseases are disorders that currently have their highest incidence rates in the more affluent western countries and yet they are still rare or even unknown in rural communities throughout the Third World. The diseases were rare or even unknown in western countries before the First World War. (Trowell & Burkitt 1981, Temple & Burkitt 1994). Diseases recognised as western are: diseases with *dietary determinants* such as gastrointestinal diseases (constipation, hiatus hernia, appendicitis, diverticular disease, colorectal polyps, colorectal cancer, haemorrhoids) cardiovascular diseases (chronic heart disease, stroke, essential hypertension, deep vein thrombosis, pulmonary embolism, pelvic phleboliths, varicose veins) metabolic diseases (obesity, diabetes, cholesterol gallstones, renal stones, osteoporosis, gout), cancer (colorectal, breast, prostate, lung, endometrium, ovarian) and other diseases e.g. dental caries. In addition to diseases with dietary determinants there are diseases with an obscure aetiology such as auto-immune diseases (diabetes, thyroiditis), and other diseases (Crohn's, celiac disease, thyrotoxicosis, pernicious anaemia, ulcerative colitis, multiple sclerosis, rheumatoid arthritis) (Temple & Burkitt 1994).

Neither of the categories of labels is satisfying. As far as the label 'degenerative and man-made diseases' is concerned, not all cancers and ischemic heart disease can be seen as age-related biological processes of 'degeneration', and some infectious diseases could be regarded as man-made too, e.g. respiratory and digestive infections induced by bad working and housing conditions. A label such as 'western diseases' suggests that diseases emerging in today's developing countries are the same as those that emerged in developed countries a hundred years ago, which is not necessarily the case (Mackenbach 1994).

2.2.2 The end of the epidemiological transition and recent changes in mortality decline

The onset of the epidemiological transition was not clearly defined, and also the end of the epidemiological transition is just as vague. In the first version of the epidemiological transition theory, Omran states that mortality eventually stabilises, which suggests the end of the transition. In Omran's first version of the epidemiological transition theory he simply could not incorporate the recent (i.e. after 1970) changes in mortality. The fact is that his first paper on the epidemiological transition theory was only published in 1971. However, he published an update in 1983. In the 1983 version he does not propose a new phase, but he describes developments that might occur as the age of degenerative and man-made diseases progresses. He stated that during the third phase other diseases might replace certain degenerative and man-made diseases in importance, depending on changes in medical knowledge, health care, lifestyle and the physical environment. He mentions mortality decline from ischemic heart disease, but argues that (the incidence of) other diseases have increased (e.g. lung cancer) and therefore, he argues, the net gain in life expectancy is small. He writes that mortality decline may come to a standstill or be temporarily reversed. He also mentions that communicable diseases have not totally disappeared. Some may linger on as causes of death e.g. influenza-pneumonia, or new ones may appear (Omran 1983). These speculations about the development of the third phase are rather vague and not totally in accordance with developments in many countries such as a renewed and, in relative terms, considerable mortality decline (Crimmins 1981, Olshansky & Ault 1986).

Other researchers have proposed a fourth phase in the epidemiological transition to incorporate the renewed mortality decline in the epidemiological transition theory. In 1986, Olshansky introduced a fourth phase: *the age of delayed degenerative diseases.* The developments in mortality decline in the 1970s and 1980s were not in concordance with several of Omran's earlier propositions. Rapidly declining death rates were concentrated in advanced ages. Mortality declined at the same pace for males and females, which is discordant with the proposition that females favour over males and children over adults. The age pattern of

mortality by cause remained largely the same as in the third phase, but the age distribution of mortality from degenerative causes had shifted towards older ages. This resulted in a further increase of the life expectancy at birth, albeit less strongly in comparison to the past. Medical technology and public health measures that favoured older over younger people, health care programmes for elderly and poor people, as well as reductions in risk factors were determinants for these epidemiological changes. Olshansky remarked that the described changes in mortality decline have a major effect on the population (Olshansky & Ault 1986). As such it fits in 'the epidemiological transition theory. A theory of the epidemiology of population change' (Omran 1971).

A year later, Rogers and Hackenberg (1987) also introduced a fourth phase in epidemiological transition: *the hybristic phase*. They argued that in this phase mortality is increasingly influenced by life-style and individual behaviour. Rogers and Hackenberg formulated several points of criticism on the original epidemiological transition theory. First, the epidemiological transition theory concentrates on infectious and degenerative diseases as separate groups, but masks interactions such as terminal cancer patients dying of e.g. pneumonia, influenza or septicaemia. Crews showed in a paper on multiple causes of death and the epidemiological transition in American Samoa that the ratio of total mentions to underlying cause of death of infectious diseases increased in the period 1950-1979. This indicates that infectious diseases still play a role in mortality patterns, also in the third phase of the epidemiological transition (Crews 1988). Secondly, the epidemiological transition theory postulates that infectious diseases will decline, but will not be totally eradicated. This was, however, the case for smallpox and diphtheria. Thirdly, the epidemiological transition theory mentions the increase of chronic degenerative diseases, but not the decline of some others e.g. ischemic heart disease. Fourthly, several causes of death have changed significantly over time, but have not been explicitly mentioned in the original theory, e.g. accidents, alcoholism, suicide, homicide, liver cirrhosis (so-called social pathologies). Fifthly, the rise in degenerative diseases is partially related to life-style. Sixthly, the favoured populations with respect to mortality decline have changed (cf. Olshansky): older favour younger people, and women no longer hold a favourable position.

Bah & Rajulton (1991) argued that both views (Rogers and Hackenberg's, and Olshansky's) on the fourth phase of the epidemiological transition are complementary. They describe different aspects of the fourth phase. Olshansky focused on the oldest age groups, while Rogers and Hackenberg consider a broader age range. Social pathologies, for example, occur at young adult ages.

2.2.3 More than one transition, counter-transitions, and epidemiological polarisation

Olshansky and Rogers and Hackenberg suggested to amend the epidemiological transition theory by adding a fourth phase. Others have suggested that the epidemiological transition theory should be formulated in terms of more than one transition (Mackenbach 1992, Horiuchi 1997).

The idea of more than one transition was first proposed with reference to the demographic transition (Lesthaeghe 1986a, Van de Kaa 1987, Cliquet 1991). The principal demographic feature of the second demographic transition is the decline in fertility from slightly above the replacement level of 2.1 birth per woman (this ensures that birth and death remain balanced and populations remain stationary in the long run) to a level well below replacement (Van de Kaa 1987). The start of the second demographic transition has been placed around 1965. The second demographic transition refers to developments in fertility and not so much to developments in mortality.

With respect to developments in mortality, Mackenbach introduced the idea of several transitions instead of one. He argues that the period referred to by Omran as the 'age of pestilence and famine' might be characterised as a period in which there was a shift in pattern of infectious diseases, which resulted in diminishing fluctuations in mortality. The period after 1970 might also be referred to as a separate transition, according to Mackenbach, because by then a totally new epidemiological phenomenon had taken place viz. a decline in degenerative diseases and injuries. Mackenbach divided the mortality change in The Netherlands since 1800 into three epidemiological transitions: 1) the extinction of high fluctuations in mortality, 1800-1875, 2) decline of infectious diseases and other important causes of death from the late 19th century, 1875-1970, and 3) the decline of 'degenerative diseases' and injuries (Mackenbach 1992).

Horiuchi (1997) introduced five transitions, three of which took place in the past and two may take place in the future. Horiuchi defines epidemiological transition as those changes in cause-specific mortality that *lower* the total mortality level. The first transition is called a 'transition from external injuries to infectious diseases'. Among hunter-gatherer populations external causes of death, e.g. war, attacks by carnivores or starvation would have been dominant (Lancaster 1990), while the emergence of agricultural settlements would have increased mortality from infectious diseases through, among other things, increased population density. The second transition, according to Horiuchi, is called the 'transition from infectious diseases to degenerative diseases' and is comparable to the second phase of Omran's epidemiological transition theory. The third transition is called the 'decline of cardiovascular disease mortality'. The latter transition focuses only on cardiovascular disease, while Mackenbach, for example, also includes injuries, which, at least in The Netherlands, also started to decline in

about the same period as cardiovascular diseases declined. Horiuchi (1997) mentioned two other, future transitions viz. 'the decline of cancer mortality' and 'slowing of senescence'. Smoking-related cancers, especially lung cancer, contribute extensively to mortality. Declines in cancer mortality could lower mortality further. In the past few years declines in cancer mortality have already been observed in the United States (Cole & Rodu 1996). Horiuchi argues that if cardiovascular disease mortality and cancer mortality have been significantly reduced, further declines of total mortality would require declines of mortality related to 'old age'. Deaths from 'old-age frailty' should be delayed to older ages: the fifth transition 'slowing of senescence'.

Besides the idea of several consecutive transitions, there is the notion of several transitions happening simultaneously in different subpopulations within a country. The coexistence of 'old' and 'new' health problems and the persistence of wide social and regional disparities with respect to disease patterns and rates of change has been referred to as 'epidemiological polarisation' or 'structural heterogeneity' (Evans *et al.* 1981, Frenk *et al.* 1989, Bobadilla & Possas 1993). Evans *et al.* (1981) argued that this epidemiological diversity makes the health transition process in many developing countries much more complex than the situation in historical developed populations. However, in historical western populations there were differences too in terms of timing and pace of mortality decline between social and regional subpopulations. The studies described in chapters eight and nine, for example, are based on regional differences in mortality decline. The studies on urban-rural differences in mortality are manifold (among others: Preston & Van de Walle 1978, Woods & Woodward 1984, Vögele 1994, Williams & Galley 1995) and differences between social classes have also been studied (Preston & Haines 1991, Williams 1992, Haines 1995).

Another concept that has been suggested to refine the epidemiological transition theory is that of 'counter transition' or 'reverse transition'. In this concept epidemiological transition is not seen as a unidirectional process in which infectious diseases through to degenerative diseases are the dominating causes of death (Frenk *et al.* 1991). Changes in cause-of-death pattern might also take another direction e.g. the recent emergence of infectious diseases of which HIV is the most noticeable example. Horiuchi (1997) defined reverse transition as epidemiological changes that keep mortality levels from declining or as changes that could even increase mortality levels. Such reverse transitions have occurred in history. Horiuchi (1997) mentions two historical reverse transitions: the early phases of industrial revolution and the unhealthy life-styles in prosperous countries. The early phases of industrial revolution in England and Wales were accompanied by deteriorating health conditions, which resulted in a decline of life expectancy (Wrigley & Schofield 1981). An example of the effect of unhealthy life-styles on mortality decline is the increase in ischemic heart disease, which

caused a stagnation of mortality declines in the 1950s and 1960s (Crimmins 1981, see also chapter 5 of this thesis).

2.2.4 Mortality and morbidity transition

In the original theory on the epidemiological transition, Omran stated that 'during the transition, a long term shift in *mortality and disease* patterns takes place'. Not surprisingly, shifts in mortality patterns have been studied more often than morbidity patterns, because mortality is easier to measure.

Riley is one of the few researchers who have studied the epidemiological transition theory and morbidity. He argues that the epidemiological transition may be defined not only as a shift from acute to chronic diseases as leading causes of death, but also as a shift from brief to protracted maladies (Riley 1990). The relationship between morbidity and mortality has been disassociated over time. A parallel association between mortality and morbidity declines would occur if disease incidence declines and case-fatality rates remain relatively stable. Mortality declines in the late 19th and 20th centuries could be ascribed to public health improvements that reduced disease incidence. In the beginning therefore the association between mortality and morbidity might have held, but later on the relationship became disassociated by changes in case-fatality rates. According to Riley, case-fatality rates, for infectious diseases, started to decline around 1930, when chemotherapy was introduced, and later, when antibiotics were introduced. The declines in case-fatality rates and the shift from acute to chronic diseases resulted in a longer duration of disease. The risk of becoming ill declined over time, but the risk of being ill increased (Riley 1989, 1990, Riley & Alter 1989).

Johansson has criticised Riley for not taking into account that cultural changes influenced the increase in morbidity. She argues that, during modernisation, people became less inclined to perceive sickness as a punishment from God, so they were more inclined to seek help. This resulted in an increase in the recordings of sickness (Johansson 1992, Riley 1992). In the discussion on morbidity change, the distinction between self-perceived morbidity and observed morbidity plays an important role. For example, a decrease in disease incidence should lead to a decline in observed morbidity and mortality, but self-perceived morbidity might remain stable, increase or decrease. Cultural determinants might affect self-perceived morbidity without affecting observed morbidity (Johansson 1992, Murray & Chen 1992, Riley 1993, Murray & Chen 1993). According to Murray and Chen (1992), it is premature to conclude that inverse transitions of morbidity (increasing) and mortality (declining) have taken place as most of our knowledge on morbidity change is based on self-perceived morbidity.

The notion that mortality as well as morbidity and disability patterns should also be included in a transition theory is also found in the 'health transition theory' (Caldwell 1989, Frenk *et al.* 1991). The health transition theory has

even a wider scope than changes in health patterns. It also includes a 'health care transition' i.e. 'the organised social response' to changes in mortality, morbidity and disability patterns (Frenk *et al.* 1991).

2.2.5 Last update of the epidemiological transition theory: Omran 1993

Recently, Omran himself published an update of the epidemiological transition theory. It was only a small section in a larger paper on 'the population puzzle in the Middle East', but it differed considerably from his former papers (Omran 1993). The update in his paper of 1983 only differed from the original theory with respect to the transition models that were distinguished. Beside the classical, accelerated and delayed model, a transitional delayed model was added. In the update of 1993, some of the criticism on previous versions of Omran's theory, as discussed in this thesis' introduction, has been incorporated. The most important alterations as compared to former versions are the extension from three phases in epidemiological transition to four, and the extension of the transition models from four (Omran 1983) to six.

The ages of 'pestilence and famine', 'receding pandemics' and 'degenerative and man-made disease' are followed by an age called 'ageing, chronic diseases, emerging new scourges (such as AIDS) and the resurgence of older diseases (such as tuberculosis)' (Omran 1993). This new, fourth phase shows that Omran tried to incorporate the criticism of, among others, Olshansky and Ault, and Rogers and Hackenberg in his theory. It is not clear from the description of the fourth phase in Omran's article of 1993 exactly what the difference in cause-of-death pattern is between the phase of degenerative and man-made diseases and the last phase of ageing, chronic diseases, emerging new scourges and the resurgence of older diseases.

Another important alteration with respect to the phases of the epidemiological transition is that the phases can occur sequentially (industrial countries) or with considerable overlap, because different transitions might take place in subgroups of the population (developing countries) (Omran 1993). This overlap of transitions is also known as 'epidemiological polarisation'.

Originally Omran distinguished 3 models viz. the 'classical model', the 'accelerated variant of the classical model', and the 'delayed model'. In the updated version of 1983 a transitional version of the delayed model was described. In the 1993 update, this transitional variant has been split into three separate variants. Mortality decline started around the end of the Second World War in all countries of the 'transitional version of the delayed model', but those countries differ with respect to fertility decline. Fertility decline may be rapid, the 'rapid transitional variant' (newly industrialised countries like Korea, Taiwan), fertility decline may be gradual, the 'intermediate transitional variant' (Egypt, Mexico) or

fertility decline may be slow, the 'slower transitional variant' in which mortality declines have not been matched by fertility declines (Yemen, Bolivia) (Omran 1993).

2.3 Descriptive analyses of the epidemiological transition

Since Omran's publication, the epidemiological transition has been described for many countries, and in many different ways. Studies differ in their description of the epidemiological transition with respect to method of analysis, cause-of-death selection, the use of life expectancy or mortality rates, and the use of age- and/or sex-specific mortality changes. A brief overview of methods to describe the epidemiological transition is given in the following section.

2.3.1 Descriptive analyses of the epidemiological transition in the literature

A frequently used method to describe the epidemiological transition is to present *the ranking of causes of death*, and the changes in that ranking over time (Omran 1980, Rogers & Hackenberg 1987, Schooneveldt *et al.* 1988, Levison *et al.* 1981, Broudy & May 1983). On the basis of the ranking of primary causes of death it is determined in which phase of the epidemiological transition a country is, in a given period. If cardiovascular diseases and cancer are in the top-three, and they are not immediately followed by an infectious disease, a country is considered to have completed the epidemiological transition; if cardiovascular diseases and cancer are high on the list but certain infectious diseases are still common causes of death, the country is said to be in a transitional phase; if infectious diseases are still the most common primary causes of death, a country is said to be in a pre-transitional phase. Broudy and May, for example, stated that the Navajo Indians were in the transition from phase 2 to 3 in 1975-77, because influenza/pneumonia was still high on the list of most important causes of death, but it was preceded by three man-made diseases (accidents, heart disease and malignant neoplasms) (Broudy & May 1983).

In other studies, the *contribution of causes of death* to change in life expectancy or total mortality over time is used to describe the epidemiological transition (Condran & Cheney 1982, Caselli 1991, Vishnevsky *et al.* 1991). The changing importance of causes of death as underlying cause and as *secondary cause of death* has also been studied to describe the epidemiological transition (Crews 1988). Some studies use the *incidence* of infectious and non-infectious diseases instead of mortality (Vigneron 1989, Young 1988). Most studies on the epidemiological

transition present a selection of infectious and non-infectious diseases, but some only concentrate on so-called 'western diseases' (Gulliford 1996). In some studies the focus has been on changes in age patterns of death instead of changing cause-of-death patterns. The epidemiological transition is described on the basis of changes in the parameters of mortality models with different parameters for childhood mortality, young adult ages and senescence e.g. the Heligman-Pollard model (Gage 1993, Bah & Rajulton 1991).

2.3.2 Descriptive analyses for The Netherlands: clarifying weaknesses in the epidemiological transition theory

The variety in descriptive methods might be due to the fact that the formulation of the phases of epidemiological transition was not unambiguous. In this chapter the weaknesses in Omran's epidemiological transition theory were pointed out. The timing of the phases and the changes in cause-specific mortality were not clearly formulated, and recent changes in mortality were not covered by the original theory. In our analyses conducted for The Netherlands, we try to elucidate these vaguenesses. The analytical methods differ from the frequently used analysis of changes in the ranking of causes of death. In order to determine objectively the timing of the phases and to gain more insight in the changing cause of death pattern, formal statistical methods and a detailed cause of death classification were used. The mortality developments that have occurred since 1970 have also been included in the analyses. The studies cover mortality changes from the mid 19th century to 1992. The phases in the epidemiological transition were described by Omran in terms of all-cause and cause-specific mortality. These two components are analysed separately for The Netherlands. Turning points, separating periods with different paces of mortality decline, are identified in the total mortality trend (chapter five). In chapter six, phases in the epidemiological transition are based on cause-specific mortality changes. In the general discussion the timing of phases determined by both methods will be compared. In both analyses, mortality trends are used instead of the (frequently used) ranking of causes of death. To determine the timing of phases trend analysis is a more appropriate method. Comparing two periods with different rankings of causes of death, and concluding that a country shifted from one phase to the next in the epidemiological transition does not give information about the onset of the different phases. The timing is in fact determined beforehand by choosing specific periods. It also does not reveal the underlying dynamics of changes in cause-of-death trends. The ranking and the shift of a cause of death in this ranking are an outcome of the trend of that specific cause of death, and of the trend of other causes.

As far as the changes in cause-specific mortality are concerned, our studies for The Netherlands differ from other publications on the epidemiological tran-

sition by the level of detail in cause-of-death groups. These groups have been carefully constructed by a reclassification of the cause-of-death classifications that were in use in the period 1875 to 1992 (chapter four). To elucidate the rise and decline of causes of death over time cause-specific mortality trends are studied. The cause-specific mortality trends are clustered, by means of a cluster-analysis, to determine groups of causes of death with equal trends over time (chapter six).

The description of the epidemiological transition for The Netherlands concentrated very much on the analysis of trends and on the use of detailed cause-of-death groups. It was explained before that analysis of trends was more appropriate to clarify the timing, and rise and decline of causes of death as compared to other frequently used methods to describe the epidemiological transition. Another reason why trends are analysed is that trends can be more easily linked to possible determinants of mortality decline than figures of ranking or contribution. Accelerations in the pace of mortality decline of a cause of death indicate the influence of determinants related to that specific cause of death (chapters five, seven). In this way, the mechanism and determinants of health and disease changes might be further 'crystallised and disentangled'. This was, after all, one of the aims of Omran's epidemiological transition theory (Omran 1971, 1983). In the next chapter, current views on determinants of mortality decline will be further explored.

Chapter 3

DETERMINANTS OF MORTALITY DECLINE

OMRAN DID NOT elaborately discuss the determinants of mortality decline in his papers, although the aim of the papers on epidemiological transition was, among others, 'to disentangle the mechanisms and determinants of the change of health and disease patterns over time'. He more or less considered the determinants of mortality decline to be known. Statements made by Omran with respect to determinants of decline are, for example, 'modernisation is the key to mortality and fertility decline in the classical and accelerated model', and 'socio-economic determinants were the primary determinants of the classical transition. These were augmented by the sanitary revolution in the late 19th century and by medical and public health progress in the 20th century' (Omran 1971, 1983). The reasons for mortality decline are, however, still under debate.

Over the years many factors have been put forward as possible determinants of mortality decline. Until the 1950s or 1960s, the prevailing opinion was that public health and improvements in medical care and technology were the most important factors in mortality decline. In the demographic transition theory as well as the epidemiological transition theory 'modernisation' is said to be the factor that induced changes in mortality and fertility (Chesnais 1992, Omran 1971). 'Modernisation' includes economic (e.g. industrialisation, specialisation), political (e.g. large political entities, centralised organisation), social (e.g. urbanisation) and cultural (e.g. rationalisation, individualisation) changes in society. Economic changes have been given most attention in the demographic transition theory (Kirk 1996). The focus on economic factors, and especially improvements in living standards, has long dominated the debate about causes of

mortality decline. One researcher in particular has played a key role in this debate: Thomas McKeown. He stated that improvements in living standards and, predominantly, in nutritional status were important determinants of mortality decline in the late nineteenth and early twentieth centuries, and that medicine was only of minor importance in mortality decline. His so-called 'nutrition-thesis' launched a still ongoing debate about the reasons for mortality decline.

3.1 Thomas McKeown: the nutrition thesis

McKeown showed in his analyses that the majority of the decline in age-standardised mortality in the period 1851-60 to 1891-1900 could be attributed to tuberculosis mortality decline (47%). 23% could be attributed to mortality decline from typhus and typhoid fever, and 20% to scarlet fever mortality decline. Because tuberculosis contributed the most to mortality decline in the second half of the 19th century, McKeown focused on the explanations for the decline of mortality from tuberculosis. According to McKeown the possible causes of mortality decline from infectious diseases could be put under one of the three following headings: 1. preventive or curative therapy, 2. change in balance between the virulence of the infectious organism and the resistance of the host, 3. improvements in environment. According to McKeown, the last option could be broadly divided into changes associated with rising living standards (predominantly changes in diet) and changes that were the result of sanitary reforms (McKeown & Record 1962).

With 'Holmesian' reasoning (Sherlock Holmes: 'When we have eliminated the impossible, whatever remains must be the truth.'), McKeown concluded that the changes associated with living standards had attributed most to tuberculosis-mortality decline and thus to total mortality decline (McKeown & Record 1962). In an article on the modern rise of populations in Europe McKeown extrapolated his findings that improved nutrition was the most important determinant of mortality decline in the 19th century to the 18th century, and he also generalised his findings to other European countries like France, Sweden and Hungary (McKeown *et al.* 1972).

In an analysis of mortality decline in the twentieth century McKeown concluded again that improvements in nutrition must have been the most important factor in mortality decline. The largest contributions to all-cause mortality decline were made by bronchitis/pneumonia/influenza (18.5%) and respiratory tuberculosis (10.8%). Deaths due to these diseases are related to nutritional status of the individual. Because effective medical treatment did not become available until the late 1940s (sulphonamides, streptomycin), McKeown argued that medicine could not account for most of the decline in the twentieth century

(McKeown *et al.* 1975). Change in virulence of the infective organism did not seem to play an important role either.

McKeown's 'nutrition-thesis' has received considerable criticism. In 1974, Razzell criticised the 'interpretation of the modern rise of population in Europe', which had been published by McKeown in 1972. He argued that if food supply had been the crucial variable in mortality decline, reductions in mortality would have almost exclusively concentrated among the poorer sections of a community. As it was, mortality had drastically declined among wealthy people. He also showed that statistics on food consumption per head showed *declines* for bread and meat in the first half of the 19th century. Razzell asked for more consideration of other possible explanations of mortality decline in the 18th and late 19th centuries, such as smallpox inoculation and, more importantly, the increasing importance of personal hygiene and cleanliness (Razzell 1974).

McKeown's conclusions were based on reasoning by exclusion ('Holmesian reasoning'). This method is only valid if all possibilities are taken into account and are examined separately and with equal attention (Woods & Woodward 1984, Szreter 1988). In McKeown's analysis many factors were put under the heading of 'standard of living' without considering the role of factors other than economic ones in improvements of, for example, housing and working conditions (Caldwell 1986, Szreter 1988). According to Szreter, McKeown did not give any consideration to the independent role of socio-political developments that were responsible for improvements in e.g. working and housing conditions, education and various health services. He concluded that, rather than nutritional improvements through higher living standards, the public health movement working through local government should be seen as the true force behind the decline of mortality in the late 19th and early 20th centuries (Szreter 1988).

One reason why Szreter questioned the role of improvements in nutritional status in tuberculosis mortality decline was the counter-acting trends of respiratory tuberculosis (declining in the late 19th century) on the one hand and pneumonia/bronchitis/influenza on the other hand (increasing in the late 19th century). *Both* causes of death are associated with the nutritional status of the individual. In countries other than England and Wales, tuberculosis mortality did not even play an important role in mortality decline. In France, tuberculosis mortality did not decline until after the First World War. According to Preston and Van der Walle 'water support systems' seemed to have had more influence on French mortality figures than 'food support systems' (Preston & Van de Walle 1978). Others explained the late tuberculosis decline in France by referring to the absence of local sanitary measures and national health reforms in the late 19th century (Mitchell 1992).

McKeown's generalisation of the 'nutrition-thesis' to the 18th and early 19th centuries has been criticised by several researchers. The major epidemic diseases in those centuries were plague, smallpox, cholera and typhus. These diseases killed a large number of the population regardless of socio-economic or nutritional status (Mercer 1990). Others showed that patterns of change in food consumption per capita do not match mortality change in the 18th and early 19th centuries (Perrenoud 1984, Fridlizius 1984).

Mercer introduced an alternative determinant of mortality decline viz. the transmission process of infectious-diseases. Fertility declines might have hampered transmission of, particularly airborne, infectious diseases by reducing family size, and by reducing the pool of new susceptibles. This could lead to infection at later ages, which could in turn reduce the severity of infection. There may be other measures that affect the risk of infection, such as better ventilation of houses, which might have had a greater impact on mortality decline from infectious diseases than improvements in nutritional status of the people (Mercer 1990).

Another aspect that should be taken into account in the discussion on determinants of mortality decline is the interrelationship of different (infectious) diseases. The causes of death that are associated with nutritional status, e.g. bronchitis, pneumonia and respiratory tuberculosis, are also common complications of other diseases. Tuberculosis was mentioned as a secondary effect of smallpox infection. Smallpox survivors were more likely to develop tuberculosis (Mercer 1985, 1990). Bronchitis and pneumonia were secondary effects of, for example, measles and whooping cough infection. Therefore, the reduction of childhood diseases, for reasons other than improved nutrition, could also bring about declines in bronchitis, pneumonia and tuberculosis.

The argument of McKeown that medicine did not play an important role in mortality decline has been challenged too. McKeown has been criticised for his narrow definition of medicine. According to McKeown effective medical interventions did not play a role until the introduction of sulphonamides (1930s) and antibiotics (1940s). Before their introduction, most infectious diseases had already drastically declined e.g. tuberculosis. However, some other medical interventions took place before the 1930s and 1940s, such as vaccination against smallpox (Mercer 1985, Rutten 1997), anti-diphtheria serum (Saltet 1909) and health-care measures such as opening tuberculosis and infant clinics (Querido 1968, Sickenga 1980). Besides, medical doctors also played a role in sanitary reforms in the 19th and early 20th centuries.

Mackenbach (1996) tried to quantify the direct contribution of medicine (medical care and technology, drugs, vaccination) to mortality decline, and concluded the contribution might have been 'sizeable and certainly not negligible'. He estimated the contribution of direct medical care to be between 4.7% and

18.5% to mortality decline in The Netherlands in the period from 1875/79 to 1970. The estimation included, among other things, the effect of the introduction of antibiotics on infectious disease mortality decline (Mackenbach & Looman 1988), the improvements in surgery, which probably contributed to the decline in mortality from digestive diseases and improved antenatal and perinatal care (Berry & Malt 1984, Butler & Alberman 1958).

The debate about the determinants of mortality decline is still very much alive. In the past two decades many determinants of mortality decline have been researched and are still being researched. However, it is still not clear what was the contribution of the different determinants to mortality decline. Although we might never completely elucidate this, there is still room for more research on the reasons for mortality decline. Questions such as the relative importance of determinants of mortality decline, and the changing importance of specific determinants over time, are still open to research.

3.2 Conceptual framework of determinants of mortality decline

The determinants that might have affected mortality decline are many. Much research on mortality decline addressed infant and/or childhood mortality. Declines in infant and childhood mortality have greatly contributed to early total mortality declines. Several authors have described frameworks of determinants of mortality (decline) (Mosley & Chen 1984, Kintner 1988a, Frenk *et al.* 1991, Williams & Galley 1995). Determinants in those frameworks are, among others, income, marital fertility, breastfeeding practices, medical care, access to health systems, housing conditions, water supply systems, sewage systems, female education, female employment, urbanisation, population density, and migration.

In this section a simple framework is presented of determinants of mortality decline (figure 3.1). The framework consists of two analytical levels of determinants: a proximate level, and a distal level (comparable frameworks are given by Mosley & Chen 1984 and Frenk *et al.* 1991).

The level of proximate determinants corresponds to *direct associations* of determinants and mortality. There are no intermediary factors in the relationship with the determinant and mortality. Therefore, *change* in the proximate determinants is expected to be directly related to mortality change. The determinants that act on this level have been summarised by the terms living conditions, lifestyle or behavioural factors, medical factors and public health measures. Living conditions refer, for example, to nutritional status, working and housing conditions; behavioural factors include for example marital fertility, breastfeeding practices and childcare; medical factors refer to medical consumption, vaccina-

tion, medical treatment; examples of public health measures are clean drinking water, and sewage systems. An immediate effect on mortality is expected after change in the proximate determinants. Many proximate determinants and their cross-sectional associations between mortality level and level of the determinant have been described in the literature. Improvements due to those determinants are assumed to be related to mortality decline. The remainder of this chapter provides a brief overview of frequently mentioned determinants.

The level of distal determinants of mortality decline on the other hand is *indirectly associated* with mortality decline. The determinants at that level affect mortality through other, more proximate, determinants. The determinants have been summarised by the terms: socio-economic factors (e.g. wealth, education), culture (e.g. religious affiliation), political institutions (e.g. political environment), and ecological setting (e.g. soil type, climate). The determinants at the distal level can initiate change in the proximate determinants and can, consequently, initiate mortality decline. The distal determinants themselves are more stable than the proximate variables, and can in some cases better be perceived as a condition for mortality decline than a determinant of mortality decline (e.g. the ecological settings). The indirect association of the distal determinants and mortality decline can be made clearer with some examples. Wealth, for example, can affect mortality through improvements in e.g. nutrition, housing conditions, and the construction of water supply systems. Education can influence mortality, through knowledge of disease processes and hygiene, which can lead to changes in behaviour such as breastfeeding practices, child-care and personal hygiene. As far as religious affiliation is concerned, it is known that fertility rates were high among Roman Catholics, and that they were less inclined to breast feed the infants.

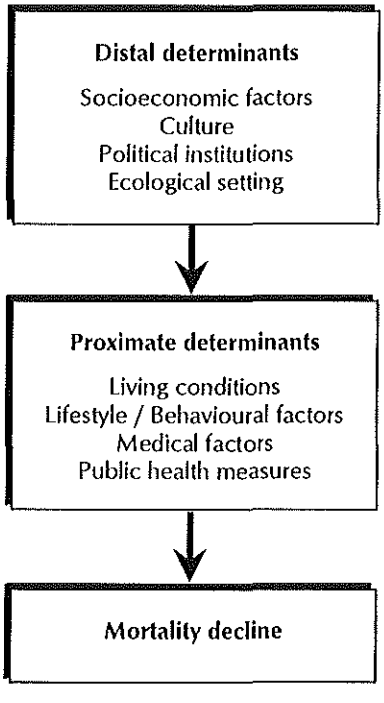


Figure 3.1 – Schematic representation of the analytical levels of determinants of mortality decline.

The scheme presented in figure 3.1 does not try to give an exhaustive representation of the associations between determinants and mortality decline. The scheme presented here suggests, for example, a unidirectional flow of determination. However, there are also feedback loops in the relationships between determinants and mortality decline. Fertility decline (a proximate determinant), for example, is related to mortality decline, but reduced mortality or, in other words, increased survival of children will in turn affect fertility (Coale & Watkins 1986). The determinants at the same analytical level are not necessarily independent. Breastfeeding, for example, leads to birth spacing, and is thus related to fertility. The scheme is designed as a simple device for analysing determinants of mortality decline (see chapters 8 and 9 of this thesis). It can help to structure the way in which the many factors, for which an association with mortality is known from the literature, are related to mortality decline.

In the remainder of this chapter, the mechanisms are discussed through which some, in the literature, frequently mentioned proximate and distal determinants affect mortality (decline). The results of multivariate explanatory studies will be explicitly mentioned. In chapters eight and nine ample consideration will be given to other determinants such as culture, urbanisation and soil type.

3.3 Examples of proximate determinants of mortality decline

3.3.1 Nutrition

Improved nutrition has played a key role in the discussion on reasons for mortality decline, since Thomas McKeown regarded improved nutrition as the most important determinant of mortality decline. McKeown arrived indirectly at his 'nutrition-thesis' viz. through reasoning by exclusion. Opponents of McKeown's thesis were not able to directly challenge the nutrition thesis. They stressed the importance of other factors, which McKeown regarded as unimportant, but they could not directly measure the importance of nutrition in mortality decline. Nutrition is one of the most difficult determinants to measure in historical populations. There are, for example, hardly any data on the amount of calorie-intake for the 19th and early 20th centuries.

As far as The Netherlands are concerned, in the late 19th and early 20th centuries some studies were conducted on the consumption of foodstuffs (Haakma Tresling 1876, Coronel 1876, CBS 1895, 1920). The studies of Haakma Tresling and Coronel show that bread, potatoes and bacon were the most commonly consumed foodstuffs. There were, however, large differences in quantity,

quality and variety of food between social classes. It is not clear from those studies what was the daily calorie-intake in those periods and whether this improved over time. A study concerning the first half of the 19th century presents a calculation of daily protein and calorie-intake on the basis of bread and meat consumption. In the period from 1807 to 1851 no significant rise was noted in protein and calorie-intake for the country as a whole. There were, however, regional differences. Protein and calorie-intake declined in Utrecht, Noord-Holland and Zuid-Holland, while it increased in Overijssel, Gelderland and Noord-Brabant (De Beer 1996).

Because of the difficulties to directly measure nutritional status, other, indirect, ways of measuring have been used such as data on height. Height is considered to be an index of 'net nutritional status'. It measures the net impact of both environment (diseases, workload) and nutritional conditions (Fogel *et al.* 1983, Harris 1994). Fogel stresses that mean final height reflects past nutritional experience of the individual throughout their growing years including the foetal period. So, if final height is used to explain adult mortality, adult nutritional levels do not explain adult mortality, but nutritional levels during infancy and adolescence explain adult mortality (Fogel 1993). Floud *et al.* showed that there was a strong agreement between the timing of the fall in age-specific mortality and the increase in height. Height started to increase in Britain in the birth cohort of 1860. Mortality began to fall in 1870-75 for children under the age of 15, and in 1875-1880 for males aged 15-24 (Floud *et al.* 1990).

The association described above between nutritional status and mortality is concerned with sufficient energy-intake to work and to fight disease. There are other relationships between nutrition and mortality as well. The quality of food is important too. New techniques for food preservation, and pasteurisation, were developed, which reduced food-borne bacterial infections. Another aspect of nutrition and mortality is the composition of people's diet, which can affect disease and cause-of-death pattern over time. During the epidemiological transition causes of death emerged that were associated with changes in the type of food. The so-called 'Western diseases' (Trowell & Burkitt 1981, Temple & Burkitt 1994) have already been mentioned. Trowell and Burkitt regarded dietary changes, such as high saturated fat, sugar and low fibre, as the underlying cause of the emergence of those diseases.

Popkin published an article on the 'nutrition transition'. Analogous to the epidemiological transition theory he described several nutritional patterns that accompany demographic, epidemiological and socio-economic changes (Popkin 1993). The first pattern, called 'collecting food', belongs to the hunter-gatherer economy. The diet is varied and there are few nutritional deficiencies. In the second pattern, called 'famine', cereals predominate in the diet, nutritional deficiency diseases emerge and fat intake is low. This pattern accompanies Omran's

age of pestilence and famine. The third pattern, called 'receding famine', is still characterised by a low variety in diet, but more fruits and vegetables are consumed. Many deficiencies disappear and stature grows. This pattern accompanied the 'age of receding pandemics' in the epidemiological transition theory. The fourth pattern, called 'degenerative diseases', consists of a diet with more animal fat, sugar, processed food and less fibre. This pattern accompanies the 'age of degenerative and man-made diseases'. The fifth and last pattern is called 'behavioural change'. Less fat and more fruits and vegetables are consumed. This pattern accompanies the fourth phase in epidemiological transition, the 'age of delayed degenerative diseases'.

Multivariate explanatory studies that include nutrition as an explanatory variable of mortality (decline) are scarce, especially in historical studies for developed countries. One study, concerned with today's less-developed countries, included calorie consumption per capita in a multivariate causal model for infant mortality. Other variables included education, status of women, health care and economic development. Nutrition had a significant direct effect on the infant mortality rate (Jayachandran & Jarvis 1986).

3.3.2 Housing conditions

Poor housing includes overcrowding as well as bad ventilation, dampness, and bad sanitation. Bad ventilation, dampness and overcrowding are related to an easy transmission of, predominantly, airborne infectious diseases; sanitary conditions are more related to diseases of the digestive system (Burnett 1991, Lowry 1991).

Burnett (1991) argued that it is not possible to isolate the house as a physical structure from the external environment in which it is located. Poor housing also means poor diet, poor sanitation and a poor standard of living in general. Improved housing could even result in an increase in mortality, if higher rents resulted in spending less money on food.

McFarlane argued that the poor housing conditions in Glasgow in the period 1911-51 were the main reason that respiratory tuberculosis mortality failed to decline as rapidly as in other towns in Britain (McFarlane 1989). Condran and Cheney concluded that crowding (persons per dwelling) was the most important factor to explain the respiratory tuberculosis level in Philadelphia in 1880 (Condran & Cheney 1981). This conclusion was based on a multivariate regression analysis including age, ethnic group, number of persons in family, number of homes owned and population density. Infant mortality in Germany in 1890 was also significantly and positively related to 'persons per dwelling'. Other variables in that analysis were urbanisation, income, marital fertility, Catholicism, and doctor density. The association between infant mortality rate and crowding was, however, not significant in the years 1871 or 1910 (Haines & Kintner 1993).

In The Netherlands in the 19th century, medical publications can be found on the relationship between housing conditions and health. For example, the differences in health were examined between people living in a basement and people living on the first floor. Living in basements was associated with higher mortality from diarrhoeal diseases and consumption (i.e. mainly respiratory tuberculosis) (Coronel 1875).

In 1901, the Building Act was introduced in The Netherlands, which stated new requirements for all kinds of newly built buildings. One of the requirements for houses was access to clean drinking water (Vogelzang 1956, Querido 1968).

3.3.3 Working conditions

Bad working conditions are regarded as one of the negative health effects of the early phases of industrialisation. In the late 19th and early 20th centuries several unfavourable working conditions were distinguished: sitting in one posture, too much hard physical labour, noxious chemicals, badly ventilated and/or heated rooms, alcohol consumption, accidents, inhalation of dust particles (Kleipool 1912).

Haines (1991) studied mortality rates for over a hundred occupations in England and Wales in the second half of the 19th century. High mortality was found among innkeepers and servants in inns and hotels. Especially mortality related to excessive alcohol consumption was found for these occupations. Occupations where exposure to dust and noxious substances was commonplace, such as potters, metal workers and chimney sweeps, were also related to high mortality (predominantly respiratory diseases). In The Netherlands, high mortality rates were found among workers in occupations that are comparable to those mentioned by Haines viz. in the glass and diamond industry, pottery, metal industry, and inn and hotel servants in the late 19th century (CBS 1912).

The analysis of the effects of 'work hygiene' on mortality is difficult to assess on the basis of the above-mentioned figures. The negative effects of the urban environment on health or other health-related aspects of social economic status can confound the association.

In The Netherlands, the first steps to improve working conditions were taken in 1889 and 1895 by accepting The Labour Act, which limited working hours, and the Factory Safety Act, in which requirements for machines were established, respectively (Querido 1968).

3.3.4 Fertility

Fertility is an important factor in the demographic transition theory. During this transition, mortality starts to decline and is followed by fertility decline. The ex-

ception from this pattern is France. In France fertility rates declined before mortality rates. The consistent feature of the decline in fertility in different regions in Europe has been the decline in marital fertility (Coale & Watkins 1986).

Fertility declines can be induced by cultural as well as economic factors. Economic circumstances of households could affect fertility. In case of a familial, labour-intensive mode of production, there was less inclination to limit family size. When children became more independent and more occupational structures became available, the economic factor lost importance. However, any sustained decline in fertility required a 'moral acceptability' (Lesthaeghe 1986b). Therefore the secularisation process played an important role too.

High parities and short birth intervals are related to infant and child mortality. High fertility rates can also influence infant and child mortality through infanticide or child neglect (Van de Walle 1986). Forste (1994) showed that higher parities and shorter birth intervals were associated with higher infant and child mortality in a country where economic development is limited, e.g. Bolivia. In this analysis, other variables related to infant mortality were also included such as mother's age, education and perinatal care.

Marital fertility was a significant factor in the explanation of infant mortality in Germany in the late 19th and early 20th centuries (Kinter 1988a, b). This conclusion was based on a multivariate regression analysis including marital fertility, urbanisation, illegitimacy, Catholicism, population density, and access to medical care. In a study of Haines (1995), using data for England and Wales for the period 1895-1911, infant mortality *decline* was regressed on fertility decline, urbanisation, social class and income. Fertility decline turned out to be a significant factor in mortality decline.

Fertility is also related to maternal mortality. It has been shown that increasing parity led to increasing maternal mortality after giving birth three times in the beginning of the 20th century (Loudon 1992).

The reverse relationship i.e. the effect of mortality on fertility has been extensively studied in the context of the demographic transition (mortality declines preceded fertility declines). Several mechanisms of this relationship have been described, of which the child replacement effect is one example (Preston 1978, Van de Walle 1986). The child replacement effect refers to a situation in which high mortality is related to high fertility, because children that die young are replaced by newborn babies.

3.3.5 Breastfeeding

In the late 19th century, marital fertility was relatively high in several provinces of The Netherlands and Germany compared to other European countries. With respect to Germany the high levels of marital fertility have been related to the virtual absence of breastfeeding (Van de Walle 1986). Breastfeeding tends to de-

lay ovulation, which results in larger birth intervals, which is, in turn, related to lower infant mortality. Breastfeeding can also be more directly related to infant and child mortality. Breastfeeding has immunological properties that provide protection against gastro-intestinal and respiratory diseases, it meets all the infant's nutritional requirements for at least the first months of its life, and it is sterile (Huffman & Lamphere 1984, Palloni & Millman 1986).

Data on breastfeeding practices are virtually unavailable for most historical populations. Data on the percentage of 'infants ever been breastfed' are available for Germany. Kintner (1988a, b) showed in explanatory historical studies on German infant mortality that breastfeeding was significantly and negatively associated with infant mortality rates, but less so than with marital fertility, access to health care and illegitimacy. In addition to the above-mentioned variables, the model also consisted of data on urbanisation, Catholicism and population density. That study also showed that the relationship between breastfeeding and infant mortality was quadratic. Prolonged breastfeeding reduced the infant mortality rate further than a short period of breastfeeding. A multivariate study on infant and child mortality in Bolivia, which also included prolonged breastfeeding (after 6 months), showed increased survival of infants being breastfed longer than six months (Forste 1994).

Some Dutch researchers consider differences in lactation patterns, and especially the effect of prolonged breastfeeding on birth spacing, to be important factors for infant mortality decline in The Netherlands in the late 19th and early 20th centuries (Vandenbroecke *et al.* 1983, Lesthaeghe 1983a). Others have argued that other factors such as quality of drinking water and childcare were more important than breastfeeding for the explanation of infant mortality in The Netherlands (Hofstee 1983).

3.3.6 Medical factors

It was mentioned earlier in this section that Mackenbach concluded that the contribution of medicine (medical care and technology, drugs, vaccination) to mortality decline in The Netherlands in the period 1875 to 1970 was not negligible (Mackenbach 1996). Mackenbach included the introduction of antibiotics, surgical innovations and perinatal care in his estimations. Most of these factors apply to the period after 1930.

Although difficult to quantify, medical factors contributed to mortality decline up to the 1930s. In the second half of the 19th century, medical doctors (the so-called 'hygienists') played a central role in matters of public hygiene and public health such as the introduction of water mains, sewage disposal systems and school hygiene, to name a few issues (Houwaart 1991).

Kunitz (1991) stated that medical doctors often served as agents of cultural and behavioural change, because they could be intermediaries between the

upper and working classes. 'Civilised behaviour' could diffuse to lower classes through the physicians.

Another medical factor that could affect mortality, in addition to the effect of medical care and technology and the role of medical doctors in public hygiene and the diffusion of more hygienic behaviour in society, is the availability of and access to medical care. The studies of Kintner (1988a, b) and Haines & Kintner (1993) are the only historical explanatory studies including availability and access to medical care in the analyses. The number of medical doctors per 100,000 inhabitants and infant care centres were significantly associated with lower infant mortality levels in Germany in the late 19th and early 20th centuries.

3.3.7 Public health measures

The introduction of water mains and sewage disposal systems are considered important public health measures for mortality decline. The main routes by which water supply systems have influenced mortality in historical populations are the prevention of water-borne diseases, such as cholera and typhoid, by means of cleaner drinking water, and the reduction of other diarrhoeal diseases by means of improved hygiene (washing of hands, using clean cooking utensils etc.). The introduction of sewage disposal systems reduced the contamination of water resources for drinking water and water for cleaning, such as rivers and canals, as well as of faecal-oral routes other than through ingestion of contaminated water.

From the 19th century onwards, publications can be found in both Dutch and international literature on the effect of water supply systems on mortality decline. Onnen wrote in 1895 that the declines in mortality in the town of Dordrecht in the period 1880-1890 should without a doubt be ascribed to the introduction of water supply systems and the closing of canals (Onnen 1895). However, the role of clean drinking water in mortality decline is not that clear. Van Poppel and Van der Heijden (1997) gave an excellent overview of research, in which they attempted to quantify the impact of water supply systems on infant and child mortality. The results of most studies were meagre. It turned out to be very difficult to assess the contribution of water supply systems to mortality decline.

As far as the introduction of public health measures in The Netherlands is concerned, the first water supply system was constructed in Amsterdam in 1853. Before the turn of the century only large towns were able to build water supply systems. In 1909, clean water supply became more common, because the government gave financial support for the implementation of water supply systems in more rural areas (Vogelzang 1956). Swartsenburg (1981) showed that there was a correlation between high expenditure on and an early introduction of water supply systems in towns, and a more rapid mortality decline in The Nether-

lands in the late 19th century. Effective sewage disposal systems only became available in The Netherlands in the beginning of the 20th century (Van Zon 1986).

Public health measures were of course much broader than clean drinking water and sewage disposal. Other public health measures in The Netherlands were for example school hygiene (De Knecht-van Eekelen 1994), tuberculosis consultation clinics (Sickenga 1980), and improvements in housing and working conditions. Private initiatives by charities and denominational organisation were important for the public health reforms in the late 19th and early 20th centuries (Rigter & Rigter 1993, Van der Velden 1996a).

3.4 Examples of distal determinants of mortality decline

3.4.1 Wealth

Improvements in living standards as determinants of mortality have had a prominent place in the discussion on the explanation of mortality decline since McKeown's publications. Economic measures, such as real wage and income, have frequently been used as indicators of living standards and wealth. Increased income can influence mortality in many ways, for example, through nutrition, housing conditions, access to health care, public health measures and access to education.

The relationship between income levels and life expectancy at birth is a-symptotic (Preston 1975). At lower income, life expectancy increases relatively rapidly, and the increase levels off at higher income levels. There is a strong correlation between the logarithm of income and level of mortality. The asymptotic relationship between mortality and income reflects diminishing returns to increases in income. There is a wide variety of dose-response relationships at the individual level that show diminishing returns e.g. nutrition, living conditions in relation to, for example, digestive and respiratory diseases. The asymptotic relationship has shifted upwards over time. Equal life expectancies are reached at lower income levels in later populations e.g. in order to reach a life expectancy between 40 and 60 a nation required an income level approximately 2.6 times higher in the 1930s than in the 1960s (Preston 1975).

Haines (Haines & Kintner 1993, Haines 1995) used income per capita in multivariate explanatory models for historical mortality levels in Germany, and for levels and declines of infant and childhood mortality in Britain in the period 1890-1911. In Germany, income per capita became a significant factor for the level of infant mortality in the early 20th century (1910). Other variables in the

model for Germany were urbanisation, marital fertility, population density, crowding, Catholicism, medical doctor density, migration and population growth (Haines & Kintner 1993). As for Britain, income was significantly associated with infant and childhood mortality levels, but it was virtually unrelated to infant and childhood mortality decline. Other determinants in the model were social class, urbanisation and fertility decline (Haines 1995). Van Poppel (1991, 1992) also found a significant association between income and infant mortality rates in The Netherlands in the early 20th (1928–1933) century but not in the late 19th century. He did find a significant association of income with female mortality in the late 19th century. Ewbank & Preston (1990) also found no relationship of income with infant mortality *decline*. That analysis was based on data for the United States in the period 1903-1923. Other variables in the model were urbanisation, race, and proportion of foreigners.

Other researchers stressed the importance of income *distribution* in the determination of mortality levels (Rodgers 1979, Flegg 1982, Wilkinson 1996).

Height has been proposed as an alternative measure of living standards instead of income or real wages (Tassenaar 1995, De Beer 1996, Brinkman *et al.* 1988). Stature, for example, measures inequality in terms of nutritional deprivation. Average height is not only determined by the level of income, but also by the distribution of income and the consumption of basic necessities by the poor (Steckel 1995). Height is a broader measure of living standards compared to income and real wage, because it also includes working and housing conditions, health and workload (Steckel 1995).

In The Netherlands, the process of 'modern economic growth' started in the second half of the 19th century. Wealth increased in rural as well as in urban areas in that period. An increase in real wages was observed after about 1860 (Van Zanden 1985, 1987).

3.4.2 Female education

Education is indirectly related to mortality decline. Education is said to bring about less adherence to tradition, more open-mindedness to new ideas, and a more rational outlook on life. Female education in particular is an important factor in mortality decline, because of the relationship with infant and child mortality. Caldwell (1979) brought education into the discussion on mortality decline with his article on education in Nigerian mortality decline. Since then, the association between female education and mortality has been studied frequently, predominantly in developing countries (Hobcraft 1993, Bicego & Boerma 1993, Sandiford *et al.* 1995). The main conclusions of this research are that educated women seem more successful at reducing the prevalence of diarrhoeal diseases, they use health care facilities more often, they more often make sure that the children are fully vaccinated, and better educated women marry later in life and

have fewer children. No multivariate explanatory studies were found for historical populations that included education in the analyses.
However, the association between women's literacy and mortality might be confounded by many economic and sociocultural variables, such as fathers' education and occupation as well as income, which might confound the relationship between maternal education and child mortality. Sandiford *et al.* (1995) showed that child survival was significantly higher among children of women who became literate at adult age compared to women who had remained illiterate. The analysis was controlled for household wealth, education of spouse and parents, parity, access to health services, water supply and sanitation.

Boonstra (1993) studied the relationship between illiteracy and several demographic variables for the town of Eindhoven (The Netherlands) and surroundings in the period 1800-1920. Subsequent marriage cohorts showed increasing infant and child mortality for children of illiterate couples, an increase until 1900 followed by a decrease in infant and child mortality for first generation literate couples, and declining infant and child mortality for the second generation of literate couples. An association was not found with respect to the development of birth rate and illiteracy, but there was an association with level of birth rate (Boonstra & Van der Woude 1984).

In the previous sections, the results of multivariate explanatory studies were mentioned when available. Those studies were nearly all cross-sectional and studied total mortality or infant mortality. A further improvement of our understanding of determinants of mortality decline, could, first, be derived from analysis of mortality trends instead of mortality rates, and, secondly, from the study of age-and cause- specific mortality.

Determinants that play a role in the explanation of differences in mortality levels do not necessarily play a role in mortality decline too. In the section on wealth, it was mentioned that income was an important determinant of infant mortality levels, but did not play a role in infant mortality decline in Britain (Haines 1995). Haines' study and a study by Ewbank and Preston (1990) were the few historical studies that analysed mortality decline in a multivariate regression model. In the chapters eight and nine mortality decline in The Netherlands in the late 19th and early 20th centuries is analysed in a multivariate regression analyses including distal and proximate determinants of mortality decline. Analysis of mortality at a lower aggregation level than total mortality, e.g. age-specific and cause-specific mortality, enhances our insight in the association of determinants with mortality (decline). It shows which age and cause-of-death groups are affected by a specific determinant. It is, for example, conceivable that urbanisation affects acute digestive (bad sanitary conditions) as well as acute respiratory diseases (crowding). A cause-specific analysis will elucidate the importance of

either one association. In the same way age-specific analyses can further elucidate the affect of determinants on mortality decline. In chapter nine, determinants of age- and cause-specific mortality (decline) in The Netherlands are analysed in a multivariate regression analysis.

Chapter 4

RECLASSIFYING CAUSES OF DEATH TO STUDY THE EPIDEMIOLOGICAL TRANSITION IN THE NETHERLANDS, 1875–1992

Abstract

Objective: *This article describes a method for reclassifying causes of death in the Netherlands for the period 1875-1992. Two criteria should be met to obtain a useful classification to study the epidemiological transition. First, the categories should be nosologically continuous over the period under study and second, there should be enough detail in causes that are important in the context of the epidemiological transition viz. communicable diseases, non-communicable diseases and external causes of death.*

Data and Methods: *A method developed by Vallin and Meslé (1988a,b), which involves 'dual correspondence tables' and 'fundamental associations', was used to create nosologically continuous categories. These categories were tested for statistical continuity during the transition years of one ICD-revision to the next, using ordinary least squares regression analysis.*

Results: *The reclassification procedure resulted in a nested classification consisting of three levels of refinement of causes of death: 27 causes, 1875-1992; 65 causes, 1901-1992 and 92 causes, 1931-1992. On the basis of this classification, 43% of all deaths in 1875-79 and 98% of all deaths in 1992 could be allocated to either communicable diseases, non-communicable diseases or external causes.*

Conclusion: *Vallin and Meslé's method turned out to be a useful tool to create nosologically continuous cause-of-death categories over time for countries in which bridge-coding is lacking. For The Netherlands, a detailed cause-of-death classification was constructed with a good representation of communicable, non-communicable and external causes of death.*

4.1 Introduction

The shift in the pattern of cause of death is an important feature of the epidemiologic transition. The analysis of trends in cause specific mortality, however, inevitably raises the problem of changing cause of death classifications. This article describes the method we used to construct a continuous and meaningful cause of death classification to study the epidemiologic transition in The Netherlands. The period under study, 1875-1992, covers 9 revisions of the International Classification of Diseases and Causes of Death (ICD) and a pre-ICD classification. The Netherlands adopted the 9 ICD-revisions respectively in the years 1901, 1911, 1921, 1931, 1941, 1950, 1958, 1969 and 1979. The pre-ICD classification was used from 1875 until 1900. The most considerable changes were the transitions from pre-ICD to ICD-1 and from ICD-5 to ICD-6.

We formulated two criteria which the classification to be constructed should meet. First, the nosological content of the causes of death to be distinguished should be as constant as possible over the period under study. The influence of changing medical knowledge and technology, of associated changes in the arrangement of ICD items, and of changing coding habits on mortality trends hampers the realisation of this criterion. We tried to deal with those influences by carefully studying the ICD manuals and testing for statistical continuity of mortality trends during the transition years from one ICD classification to another. The second criterion is that there should be sufficient detail in the causes of death to be distinguished. Broad categories, which cover diseases that are too different from each other anatomically or etiologically, can mask cause-specific dynamics that are important for the description and explanation of the epidemiological transition. Furthermore, the categories should be meaningful in the context of the epidemiological transition. Omran formulated the shift in the pattern of cause of death as a shift from infectious diseases to 'degenerative and man-made diseases' (Omran various years). This latter group of diseases has also been referred to as 'Western diseases' (Trowell & Burkitt 1981). Cardiovascular diseases and cancer are the main exponents of the degenerative diseases and external causes of death of the man-made diseases. In this study, the three groups will be indicated as communicable, non-communicable and external causes of deaths. In constructing cause of death categories we tried to avoid intermingling of these groups in order to be able to study the shift in the pattern of cause of death as accurately as possible in future analyses.

It turned out to be impossible to meet both criteria for all causes of death. Sometimes we had to sacrifice some nosological continuity in order to retain sufficient detail, e.g. in the case of ischemic heart disease. In other cases some detail was sacrificed for nosological continuity, e.g. chronic nephritis was taken together with other kidney diseases. Nevertheless, the overall success of our re-

classification procedure was reasonable. The purpose of this paper is to describe the method we used, and to illustrate its results with a few examples.

4.2 Data and Method

4.2.1 Data

Absolute numbers of deaths, published by the Central Bureau of Statistics, by sex, age and cause were used for the years 1901-1992. We redistributed the number of deaths in the years 1901 and 1902 according to the cause of death ratios from a table with aggregated figures for the years 1901-1904, because only the abridged versions of the ICD-1 were available for the years 1901 and 1902. Data for the years 1875-1900 were published by the Minister of the Interior. The 19th-century tables were quinquennial calendar year publications. The total number of causes of deaths expanded from 34 in the pre-ICD classification to about 5200 in the ninth ICD revision. The number of age categories increased from 8 broad categories (0, 1-4, 5-14, 15-19, 20-49, 50-64, 65-79 and 80 years and above) in the 19th century classification to 5-year age categories up to 80 years and above since ICD-5.

We did not use sex-specific data because it is unlikely that changes in classification schemes will affect male and female mortality differently.

4.2.2 The reclassification procedure: nosological continuity and detail in causes of death

Introduction of the method of Vallin and Meslé

In The Netherlands, as in many other countries, the main difficulty in reclassifying causes of death is the lack of bridge-coding. Coding the deaths of one year according to both the new and old classification would elucidate the transfer of causes of death from one code to another. Vallin and Meslé have made an enormous effort in reconstructing causes of death for France from 1925 to 1991 according to the 9th ICD-revision (Vallin & Meslé 1988a, b, 1993). They have constructed a very systematic and accurate method, consisting of three 'stages': dual correspondence tables, fundamental associations and transition tables. We used the first two stages of Vallin's method to create nosologically continuous groups for the case of the Netherlands. The third stage had to be left out, because in that stage deaths classified according to one ICD-revision were redistributed on the basis of the results of death ratios derived from the first year of the next ICD-revision. We will come back to this in the discussion paragraph.

Dual correspondence tables and fundamental associations method

The dual correspondence tables link the codes of one ICD revision to the following and vice versa by constructing two tables. A first table lists, for example, for each ICD-1 code all the ICD-2 codes that have one or more illnesses or causes of death in common. A second table lists for all ICD-2 codes the corresponding ICD-1 codes or a part of them. These pairs of tables were constructed for all ICD-transitions. Table 4.1 illustrates this by showing a small part of the dual correspondence tables for the ICD-3 to ICD-4 transition. This example shows that ICD-4 code 44 is not only linked to ICD-3 code 25, but also to the codes 12, 13 and 42. ICD-4 code 39b is only linked to code 25.

The procedure followed in the linkage of codes was based on the title of items in the analytical and/or alphabetical manuals of the ICD revisions (Bertillon 1903, General Register Office 1912, Ministère des Affaires Etrangères 1920, CBS 1935, 1940, World Health Organization 1957, 1967, 1977-78).

After the stage of dual correspondence tables, fundamental associations were formed. For example, for every ICD-1 code all connected ICD-2 codes were looked up in the ICD-1/ICD-2 correspondence table, and for those ICD-2 codes the corresponding ICD-1 codes were determined, which could be more than the ICD-1 code one started with. For the newly found ICD-1 codes again the corresponding ICD-2 codes were determined.

The association between certain ICD-1 and ICD-2 codes was complete, when no new ICD-1 or ICD-2 codes were involved. In this way the smallest possible groups of ICD-1 and ICD-2 codes with an identical medical content were created, i.e. the so-called fundamental associations. Such fundamental associations were calculated for all ICD transitions. Table 4.1 gives a fundamental association of ICD-3 codes 12, 13, 25 and 42 with ICD-4 codes 39b and 44. In the last step all fundamental associations of all ICD-revisions, starting with ICD-1, were connected. The fundamental associations of ICD-1/ICD-2 were con-

Table 4.1 – Part of the dual correspondence table of the ICD-3 to ICD-4 transition

ICD-3		ICD-4	
25	other epidemic diseases	39b	other protozoal diseases
		44	other infectious or parasitic diseases
ICD-4		**ICD-3**	
39b	other protozoal diseases	25	other epidemic diseases
44	other infectious or parasitic diseases	12	miliary fever
		13	parotitis epidemica
		25	other epidemic diseases
		42	other infectious diseases

nected to the fundamental associations of ICD-2/ICD-3 via the codes of ICD-2; the fundamental associations of ICD-2/ICD-3 were connected to the associations of ICD-3/ICD-4 via the codes of ICD-3, and so on. The connection of all fundamental associations resulted in 'nosologically continuous' causes of death. The work of Vallin and Meslé was used as a guideline in the case of difficult exchanges between items (Vallin & Meslé 1988b). The tables published by Mackenbach also were helpful for some causes of death in the ICD-2 to ICD-9 period (Mackenbach 1988).

The transition of ICD-5 to ICD-6 was one of the most difficult transitions to create dual correspondence tables for. The classification system changed from a system numbering from 1 onwards to a three digit coding system. The total number of codes increased from 200 to 765 between ICD-5 and ICD-6. The 765 three digit codes were further subdivided in four digit codes. There were many transfers of parts of codes in both directions.

Before the sixth ICD revision, the ICD had only been used for mortality statistics. The sixth revision was considerably expanded with the aim to also make it suitable for morbidity purposes (Dixon 1993). The introduction of morbidity items introduced the possibility that causes of deaths could be associated with a morbidity item. An example is the link of 'epilepsy' and 'mental disorders' via 'mental disorders with epilepsy', ICD-6 item 309.4. Such morbidity links were neglected in the construction of fundamental associations. Some other associations contained both communicable and non-communicable diseases. Those associations were re-examined to see whether it was possible to split them up into associations with only communicable or non-communicable diseases without introducing statistical discontinuity. The assessment of statistical discontinuity in cause of death trends will be discussed later.

Bridging the pre-ICD and ICD-1 classification: using other sources of information

A particularly difficult task was the creation of a dual correspondence table for the transition from the pre-ICD (34 categories) to the first ICD classification (176 categories). The pre-ICD classification was used from 1875-1900 and consisted often of rather vaguely defined categories with a broad medical content. This classification was, in fact, an abridged form of a classification of 1866-1874 consisting of 55 categories. In the late 19th century, the Dutch Inspectors of Public Health complained that the classification was outdated and that it did not even correspond with the medical knowledge of the time in which it had been introduced. Diarrhoea and hydrops were separate categories, but they were hardly mentioned as a cause of death by the medical doctors. Some categories consisted of two or more diseases, which were neither etiologically nor anatomically related, for example, epilepsy and convulsions (Saltet 1895, Saltet 1917,

Evers 1882, Vollenhoven 1889, Inspecteurs van het Geneeskundig Staatstoezicht 1900).

Both the briefness of the classification and the fact that there was no bridge-coding for the pre-ICD and ICD-1 made it very hard to associate the pre-ICD codes to ICD-1 codes. Four sources of information were used to create the most plausible dual correspondence table for the pre-ICD/ICD-1 transition: 1) the classification scheme from 1866, 2) late 19th century medical literature, 3) medical dictionaries and 4) the classification of causes of death for England & Wales. In a letter from the Minister of the Interior to the Inspectors of Public Health of January 21, 1876, a new scheme for classifying the causes of death was introduced. The former 55 categories used in the classification of 1866 were reduced to 34 categories. Instructions on how to classify the codes of the old list of 1866 according to the new list were included (Minister van Binnenlandse Zaken 1876). There were some articles directly addressing the point of cause of death classification, more particularly, which cause of death had to be classified under which heading (Beneke 1875, Saltet 1895, Saltet 1917, Vollenhoven 1889). Saltet mentioned in an article on the mortality of tuberculosis that the classification was mainly anatomical except for the infectious or acute diseases. The various forms of tuberculosis were classified according to the organ affected, except tuberculosis of the lung, which was coded separately. A late 19th century medical dictionary was used to gain insight in the meaning of vague terms like 'debility' and to determine which symptoms of certain diseases were seen as most important in that time (Quain 1883). Finally, the Dutch classification was compared with the much more extensive classification for England & Wales (Registrar-General 1877, 1901). The location of certain diseases in the English classification sometimes gave a clue for its place in the Dutch classification e.g. the classification of leprosy under 'diseases of the skin'.

The result: a nested classification

Much detail in the cause of death classification is lost when linking the pre-ICD codes to the ICD-associations. Furthermore, some causes of death, which are important in studying the epidemiological transition, were missing in the categories for first part of the period 1901-1992, e.g. lung cancer, but could be distinguished from 1931 onwards. Therefore, a nested classification of causes of death was constructed to allow the possibility of analysing mortality using different levels of refinement of causes of death. The classification consisted of 27 categories for the period 1875-1992, which could be split up into 65 categories for the period 1901-1992. These 65 categories could, in turn, be split up into 92 categories for the period 1931-1992. More detailed information about this nested classification will be given in the results paragraph.

We could not completely avoid intermingling of communicable and non-communicable causes of death in the categories of the nested classification. Some categories also remained vaguely defined, especially on the 27 causes level. These vaguely defined groups are less meaningful for the description and explanation of the epidemiological transition, because causes of death within these groups were influenced differently by changes in medical treatment or hygienic measures. The mixed and vaguely defined groups are indicated as 'nosologically not meaningful' in the results paragraph.

Statistical continuity

It is very difficult to assess the influence of changing medical knowledge and changing classifications and coding habits on the content of groups of causes of death. We tried to deal with the influence of the introduction of new classification schemes. Statistical discontinuity in the trend of a certain cause of death at the introduction of a new ICD-revision probably indicates a change in the medical content of that cause. Linear regression analysis was used to assess the continuity of the trend, because this overcomes fluctuations in trends of infectious causes or causes with small numbers of deaths.

Ordinary least squares regression was carried out for every pre-ICD and ICD transition for every nosologically continuous cause of death. The independent variable was the calendar year and the dependent variable was the natural logarithm of the crude death rate. The expected mortality rate and the 95% confidence interval (95%-C.I.) for the first year of the following ICD-revision was calculated based on the regression equation. We checked whether the observed mortality rate for that particular year fell within the 95%-C.I. If this was the case at every transition, that nosologically continuous group was accepted as a separate cause of death. Where the trend hardly fluctuated the confidence interval could, however, be very narrow. This might yield discontinuities, while the absolute or relative difference in mortality rates was, actually, small. Therefore a threshold value of 10% mortality change for discontinuous transition years, i.e. years for which the observed mortality rate did not fall within the 95%-C.I., was introduced. Discontinuities of 10% and below were then accepted as statistically continuous transitions.

The 10% threshold value was calculated in two ways. On the one hand we determined, whether there was an absolute difference between the observed mortality rate of the first year of the 'new' ICD revision (Yobs) compared to the expected mortality rate for that year (Yexp) of more than 10% of the total change in mortality for that specific cause of death in the period under study. On the other hand, the relative difference between Yobs and Yexp was determined for the first year of the 'new' ICD revision. In this case the ratio of Yobs and Yexp should have values between 0.9 and 1.1. Causes of death that had been

determined as continuous either in a relative or in an absolute sense were also accepted as separate causes. Both the absolute and relative mortality differences were taken into account, because future analyses can be based on an additive or a multiplicative model of mortality. If one, for example, wished to determine the contribution of mortality from a certain cause of death to total mortality change, the absolute difference in mortality is of interest. The absolute changes of all causes of death add up to the change in total mortality (additive model of mortality). However, studies concerning the pace of mortality change (multiplicative model of mortality) deal with fractions of mortality change. Relative differences are important in this case.

Causes of death that still had statistically discontinuous transitions after this procedure were either aggregated into broader groups or, nevertheless, accepted as a separate cause for reasons of detail. If groups were split up or aggregated for reasons of detail or discontinuity, the new formed groups were again tested for absolute and relative statistical continuity. By accepting some discontinuous causes as separate causes of death, we tried to prevent as much as possible the intermingling of communicable, non-communicable and external causes of death. Ischemic heart disease, for example, would otherwise be covered by the more general group 'heart diseases', which also contains rheumatic heart disease. Details for discontinuous transition years, especially the percentage discontinuity (absolute and relative) in those years, were documented for every cause of death. So, in future analyses, it is always possible to check whether a certain change in cause-specific mortality might be partly artificial.

4.2.3 Standardisation

Time trends of specific causes of death are presented as an illustration of the reclassification procedure. Those trends are based on directly standardised mortality rates. Eight age groups were used in the standardisation procedure, <1, 1-4, 5-14, 15-19, 20-49, 50-64, 65-79 and 80 years and over. This division in age groups was the highest common factor of all the different age group divisions in the years 1875-1992. In this way we minimised the recalculation of deaths for a certain year based on ratios of later years. The reference population was the average population for the period 1901-1992. This standard was used for all three periods, 1875-1992, 1901-1992 and 1931-1992.

Table 4.2 – The nested classification: 27 causes, 1875-1992; 65 causes, 1901-1992; and 92 causes 1931-1992

1875-1992 (pre-ICD-ICD9)	1901-1992 (ICD1-ICD9)	1931-1992 (ICD4-ICD9)
Congenital malformations	Congenital malformations	Congenital malformations
Cancer	Cancer of the oesophagus, stomach, liver and gallbladder	Cancer of the oesophagus
		Cancer of the stomach
		Cancer of liver and gallbladder
	Cancer of the intestines and peritoneum	Cancer of the rectum
		Cancer of the peritoneum
		Cancer of the small intestines and colon
	Cancer of the skin	Cancer of the skin
	Cancer of the breast	Cancer of the breast
	Cancer of the mouth, female genital organs, and other organs (excl. those already mentioned)	Cancer of the uterus
		Cancer of the ovary and other female genital organs
		Cancer of the pancreas
		Cancer of larynx and lung
		Cancer of mouth, tongue, male genito-urinary system, and other organs (excl. those already mentioned)
Scurvy	Scurvy	Scurvy
Typhus. Typhoid fever.	Typhus	Typhus
Malaria (Including: Intermittent fever. Pernicious fever)	Malaria	Malaria
Smallpox	Smallpox	Smallpox
Scarlet fever	Scarlet fever	Scarlet fever
Measles	Measles	Measles
Cerebrovascular diseases	Cerebrovascular diseases	Cerebrovascular diseases
Brain diseases etc. (Including: Insanity. Diseases of the spinal cord. Paralysis. Syphilis. Convulsions. Trismus. Epilepsy.)	Tuberculosis of the nervous system	Tuberculosis of the nervous system
	Syphilis	Syphilis
	Diseases of the nervous system. Diseases of the thyroid. Basedow's disease	Goitre
		Cretinism
		Basedow's disease
		Other diseases of the thyroid
		Diseases of the nervous system
		M.Parkinson
		Multiple sclerosis
		Epilepsy
		Eye diseases
	Alcoholism	Alcoholism
	Encephalitis/meningitis	Encephalitis/meningitis
	Convulsions	Convulsions
	Poliomyelitis	Poliomyelitis
	Diseases ear	Diseases ear

1875-1992 (pre-ICD-ICD9)	1901-1992 (ICD1-ICD9)	1931-1992 (ICD4-ICD9)
Respiratory tuberculosis (Including: Tuberculosis of the lung and larynx. Haemoptysis)	Respiratory tuberculosis	Respiratory tuberculosis
Diabetes	Diabetes	Diabetes
Diphteria. Croup	Diphtheria	Diphtheria
Whooping cough	Whooping cough	Whooping cough
Acute respiratory diseases	Influenza	Influenza
	Pneumonia	Pneumonia
	Diseases of the pleural cavity	Diseases of the pleural cavity
Chronic respiratory diseases	Diseases of larynx, pharynx, nasal cavity, and oral cavity	Diseases of larynx, pharynx, nasal cavity, and oral cavity
	Chronic bronchitis, asthma, other diseases of the lung	Chronic bronchitis, asthma, other diseases of the lung
Diseases of the circulatory system	Rheumatic fever, chorea	Rheumatic fever, chorea
	Angina pectoris. Ischaemic heart disease	Angina pectoris. Ischaemic heart disease
	Other heart diseases	Diseases of the pericardium
		Acute endocarditis
		Chronic endocarditis
		Acute myocarditis
		Chron.myocarditis, functional heart disease, other heart disease
		Hypertension, hypotension
	Diseases of arteries and veins	Diseases of arteries and veins
Acute diseases of the digestive system. Diarrhoea. Dysentery	Diarrhoea, dysentery, enteritis	Diarrhoea, dysentery, enteritis
	Peritonitis	Peritonitis
	Appendicitis	Appendicitis
Cholera (Including: Asiatic cholera. Cholera nostras)	Cholera	Cholera
Chronic diseases of the digestive system	Tuberculosis of the abdomen	Tuberculosis of the abdomen
	Stomach ulcer	Stomach ulcer
	Diseases of the oesophagus	Diseases of the oesophagus
	Other diseases of the digestive system	Other diseases of the stomach and intestines
		Liver cirrhosis
		Other diseases of liver and gallbladder
		Diseases of the pancreas
		Gallstones
		Intestinal hernia and occlusion
Diseases of the genito-urinary system	Venereal infections (except syphilis)	Venereal infections (except syphilis)
	Acute nephritis	Acute nephritis
	Chronic nephritis. Other kidney diseases	Chronic nephritis. Other kidney diseases
	Diseases of bladder, urethra, and other organs urinary tract	Diseases of bladder, urethra, and other organs urinary tract

1875-1992 (pre-ICD-ICD9)	1901-1992 (ICD1-ICD9)	1931-1992 (ICD4-ICD9)
	Diseases of the prostate. Diseases of other male genital organs	Diseases of the prostate. Diseases of other male genital organs
	Diseases of female genital organs	Diseases of female genital organs
Puerperal diseases	Bleeding and other diseases during pregnancy	Bleeding and other diseases during pregnancy
Puerperal fever	Puerperal fever	Puerperal fever
Other diseases (Including: Debility. Some types of tuberculosis. Scrofula. Rickets. Skin diseases. Abscess. Ulcer. Gangrene. Pyaemia. Haemorrhage. Continuous fever.)	Erysipelas	Erysipelas
	Anthrax	Anthrax
	Disseminated and other tuberculosis	Disseminated and other tuberculosis
	Rheumatism. Arthritis. Rickets. Diseases of locomotion	Rheumatism. Arthritis. Rickets. Diseases of locomotion.
	Adrenal diseases	Adrenal diseases
	Old age. Dementia	Dementia
		Old age
	Diseases of newly born	Diseases of newly born
	Septicaemia. Pyaemia	Septicaemia. Pyaemia
	Other infectious diseases	Other infectious diseases
	Other diseases	Other diseases
Violence	Homicide	Homicide
	Traffic accidents	Traffic accidents
	Other accidents	Other accidents
Suicide (incl. 'by drowning')	Suicide	Suicide
Unknown and ill-defined causes of death. Sudden death. Dropsy	Unknown and ill-defined causes of death	Unknown and ill-defined causes of death

4.3 Results

4.3.1 The nested classification

As shown, the method of constructing fundamental associations, the testing for statistical continuity and the need for detail in causes of death led to a nested classification. Three levels of refinement of causes of death were distinguished which can be studied in different periods of time: 27 (1875-1992), 65 (1901-1992) and 92 causes of death (1931-1992). The cause-of-death categories concerned are shown in table 4.2. The corresponding pre-ICD and ICD codes are given in appendix 1.

Detail in cause of death categories

Table 4.3 shows the number of causes of death of the nested classification belonging to each of the categories communicable, non-communicable and exter-

Table 4.3 – Number of causes of death (COD) distinguished in three periods by three important cause of death categories and meaningfulness.

Period	Total number of COD		Nosologically meaningful			Nosologically not meaningful		
			number of COD	percentage of all deaths covered		number of COD	percentage of all deaths covered	
				start of period	end of period		start of period	end of period
1875-1992	27	communicable diseases	12	35	4	8	57	52
		non-communicable diseases	5	6	40			
		external causes of death	2	2	4			
1901-1992	65	communicable diseases	26	47	6	10	20	13
		non-communicable diseases	25	31	77			
		external causes of death	4	2	4			
1931-1992	92	communicable diseases	27	28	6	12	6	2
		non-communicable diseases	49	62	88			
		external causes of death	4	4	4			

nal causes of death, and it shows how many causes were non-meaningful in the context of the epidemiological transition, i.e. vaguely defined and/or a mixture of communicable and non-communicable diseases.

At the level of 27 causes of death, the number of non-meaningful causes was relatively large (> 50%) due to the broad and vaguely defined categories of the 19th century classification. The category consisting of 'Debility, Some kinds of tuberculosis, Rickets, Some skin diseases, Abscess, Ulcer, Gangrene, Haemorrhage and Continuous fever' is very heterogeneous with respect to the aetiology of the diseases. Besides this, debility, for example, is a very vaguely defined cause of death. At the level of 65 causes (to be distinguished in the period 1901-1992) the number of communicable and non-communicable meaningful causes of death was about the same, and more than 80% of all deaths are covered both in 1901 and 1992 by these causes. The shift from communicable to non-communicable causes of death in this period is shown by the percentage of all deaths covered by communicable diseases at the start and end of the period.

Statistical discontinuities at the pre-ICD and ICD-transitions

For the three levels of refinement of causes of death table 4.4 shows the number of causes with less than 10% discontinuity at the transition years, both in a relative and absolute sense, on the basis of regression analysis of the cause of death categories as defined in the nested classification.

The majority of the causes show discontinuities < 10% for all three levels of refinement of causes of death (except for 92 causes, absolute differences). As far as relative discontinuities are concerned, most discontinuities occurred at the pre-ICD/ICD-1 and ICD-4/ICD-5 transition for the 27 causes level (respec-

Table 4.4 – Number of causes of death with discontinuities < 10% for all pre-ICD and ICD-transitions at three levels of refinement of cause of death. R = relative difference at the transition years. A = absolute difference at the transition years. N.a. = not available.

	27 causes		65 causes		92 causes	
	R	A	R	A	R	A
Pre-ICD - ICD1	19	17	n.a.	n.a.	n.a.	n.a.
ICD1-2	25	24	61	55	n.a.	n.a.
ICD2-3	24	24	53	53	n.a.	n.a.
ICD3-4	25	27	58	60	n.a.	n.a.
ICD4-5	18	23	48	57	69	28
ICD5-6	26	26	64	64	85	38
ICD6-7	25	26	59	62	86	65
ICD7-8	24	24	50	56	71	52
ICD8-9	26	26	56	60	80	66

tively 30% and 33% of the causes), and at the ICD-4/ICD-5 and ICD-7/ICD-8 transitions for the 65 (respectively 26% and 23%) and 92 (respectively 25% and 23%) causes level. The number of causes with absolute discontinuities was large at the pre-ICD/ICD-1 transition (37%), but low at all other transitions (< 15%) for the 27 causes level. It was low at all transitions for the 65 causes level (< 18%) and it was substantial for all transitions at the 92 causes level (all more than 28% of the causes). As mortality decline is much lower in the period 1931-1992 as compared to the periods 1875-1992 and 1901-1992, the difference in mortality rates at the ICD-transitions will sooner lead to absolute discontinuities at the 92 causes level than at the 27 or 65 causes level. The discontinuities in the transition from ICD-4 to ICD-5 may be caused by the Second World War, as the ICD-5 was introduced in 1941. The high level of discontinuities for the transition from ICD-7 to ICD-8 is less clear, because those two revisions are very much alike. Most of the discontinuities fell within a range of 10-20% or 20-40%. Only 7% of all causes at the 27 and 65 causes level had relative or absolute discontinuities of more than 60% at any pre-ICD or ICD-transition. Only 7% of the 92 causes had a relative discontinuity of more than 60%, but as much as 20% of the causes had an absolute discontinuity of more than 60% at the ICD-4 to ICD-5 transition.

Few causes of death were statistically continuous (both relative and absolute) for all ICD-transitions. 7 out of 27 causes were continuous for the whole period 1875-1992. 20 out of 65 causes were continuous for the period 1901-1992 and 18 out of the 92 causes in the period 1931-1992 were completely statistically continuous. Not all discontinuous causes were discontinuous from both a rela-

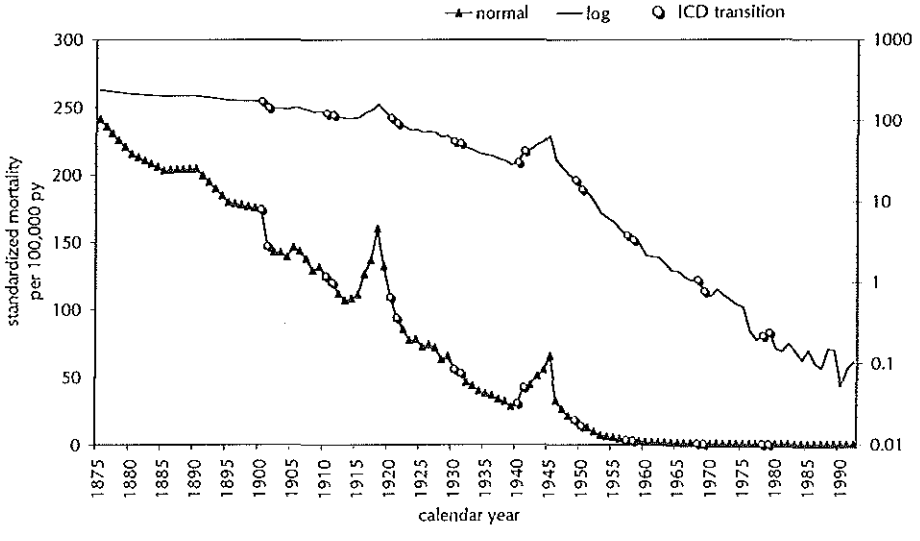


Figure 4.1a – The trend of respiratory tuberculosis with the indications of ICD-transitions.

tive and an absolute point of view e.g. in the period 1901-1992 22 out of 65 causes were only discontinuous in one aspect. This means that for certain types of analysis those causes can be considered as continuous.

Despite of the reported discontinuities, most of the trends were suitable for future analyses. Beside this, enough detail in causes of death was achieved for a thorough investigation of the epidemiological transition in The Netherlands. Some illustrative examples of trends are given in the next paragraph. Trends for all distinguished causes of death are given in the appendices 2a-c.

4.3.2 Illustrative examples

The trends are both plotted on a normal and a logarithmic scale to visualise respectively the absolute and relative differences at the ICD-transitions. The location of the ICD-transition years is indicated with empty squares in the graph.

The trend of respiratory tuberculosis

Respiratory tuberculosis mortality can be studied from 1875 onwards (figure 4.1a). The trend shows an ongoing mortality decline of this cause with a clear period of acceleration of the decline after the Second World War. There is a small discontinuity (absolute 12%, relative 16%) at the transition from the pre-ICD to ICD-1 classification probably caused by changing coding habits. Saltet comes up with a very plausible explanation in his article on the mortality of tuberculosis in the Netherlands. According to Bertillon's nomenclature (ICD-1), respiratory tuberculosis and tuberculosis of other organs occurring simultaneously should be classified under 'respiratory tuberculosis'. Although the Netherlands introduced the ICD-1 in 1901, there were not enough books with coding rules available. Consequently, those tuberculosis cases were classified under the heading 'tuberculosis of two or more organs'. The correct coding rules became available in 1905, which was reflected by a small peak in the trend of respiratory tuberculosis mortality (Saltet, 1917). It is most likely that the apparent discontinuity (relative, 16%) at the transition from ICD-4 to ICD-5 is caused by an increase of respiratory tuberculosis mortality due to deterioration of the physical condition of many people during the Second World War. The discontinuities in the trend of respiratory tuberculosis are very small, which makes this cause suitable for future analyses.

The trend of ischemic heart disease

The trend of ischemic heart disease mortality can be studied from 1901 onwards (figure 4.1b). The trend can be characterised by an enormous increase from about 1930 until 1970, followed by a decline. As far as the pace of mortality change is concerned, there is a rapid increase in mortality from 1930 to 1931. The mortality ratio between these two years is 1.74, which means a relative discontinuity of 74%. The absolute difference in mortality rates is, however,

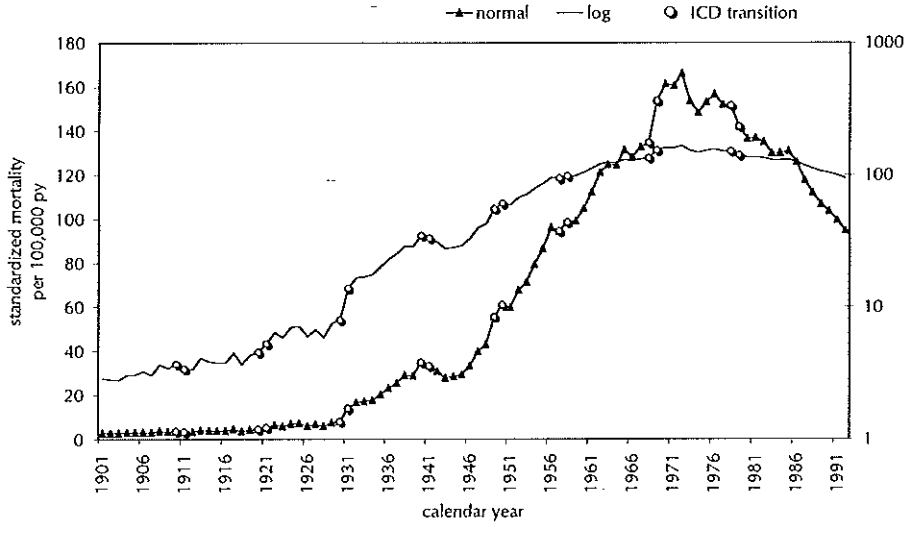


Figure 4.1b – The trend of ischemic heart disease with the indications of ICD-transitions.

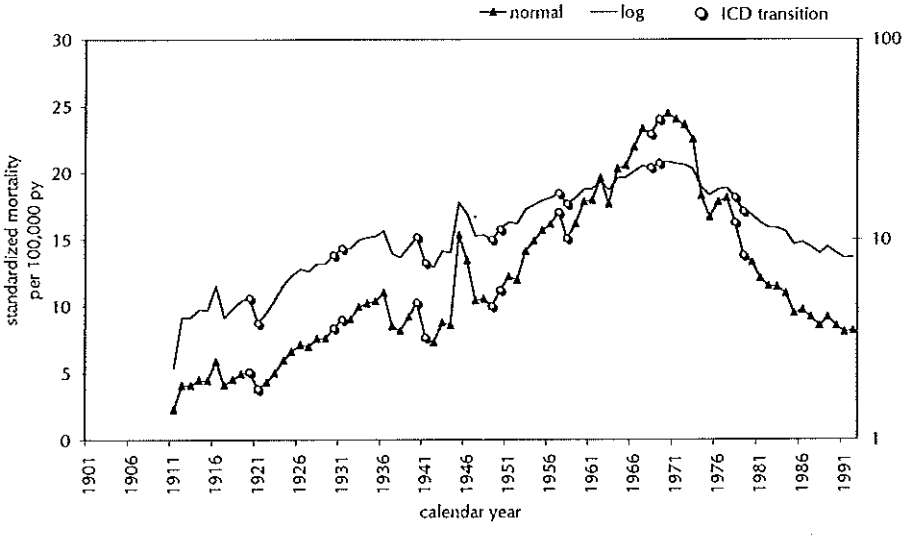


Figure 4.1c – The trend of traffic accidents with the indications of ICD-transitions.

small (5.9 per 100,000 person years) as compared to the total increase of this cause of death (91.9 per 100,000 person years in the period 1901-92, and even 163.36 per 100,000 person years in the period 1901-72) and will hardly affect calculations on the contribution (in this case negative) of this cause of death to total mortality decline. It is not inconceivable, that a great deal of the increase of ischemic heart disease mortality, before 1940, should be attributed to increasing improvement in diagnosis. It is, however, impossible to correct for this type of artefact in cause-specific trends by means of any statistical procedure.

The trend of traffic accidents

The trend of traffic accidents can be studied from 1911 onwards and shows an increase until 1970 and a decline afterwards (figure 4.1c). After 1970, mortality strongly decreased due to a deceleration of the growth rate of traffic mobility and decreases in both injury rate and case fatality (Van Beeck *et al.* 1989). In the context of the decline after 1970 the absolute discontinuity at the years 1978-1979 (32%) is less pronounced than in the context of the decline between 1911 and 1992. The effects of the Second World War are also visible in this trend (absolute discontinuity, 46%; relative discontinuity 33%). The relative and absolute discontinuities at the years 1957-1958 are both small (11% and 24% respectively).

Cancer as an example of enhanced detail by means of a nested classification

There was only one category for cancer mortality in the 27 causes classification. Cancer mortality has been increasing over the whole period 1875-1992, except for a dip just after the Second World War. Neoplasms of the stomach, oesophagus, liver and gallbladder as a group, neoplasms of the intestines and peritoneum, neoplasms of the skin, neoplasms of the breast and neoplasms of other organs including cancer of lung and larynx could be distinguished at the level of 65 causes for the period 1901-1992.

Figure 4.2a shows the trend from 1901-1992 for these groups. The trends show that studying cancer as one group masks the dynamics of the different types of cancer e.g. the declining trend of cancer of the oesophagus, liver and stomach or the elevated mortality of cancer of the intestines and peritoneum during the Second World War.

Figure 4.2b shows the trends for cancers from the group 'other cancers' from figure 4.2a, that could be distinguished at the 92 causes level. Cancer of the uterus, ovary and other female genital organs, pancreas, lung and larynx, and a residual group 'other cancers' including cancer of the prostate could be studied from 1931 onwards. Cancer of the lung and larynx and cancer of the pancreas were strongly increasing. The group 'other cancers', including cancer of the prostate and cancer of the ovary and other female genital organs, also showed an

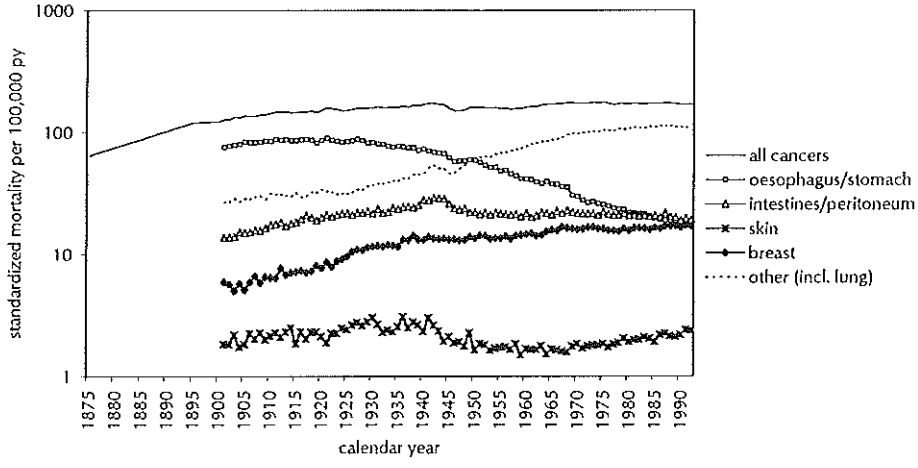


Figure 4.2a – An example of the nested classification: detail in cancer mortality, 1875-1992 and 1901-1992.

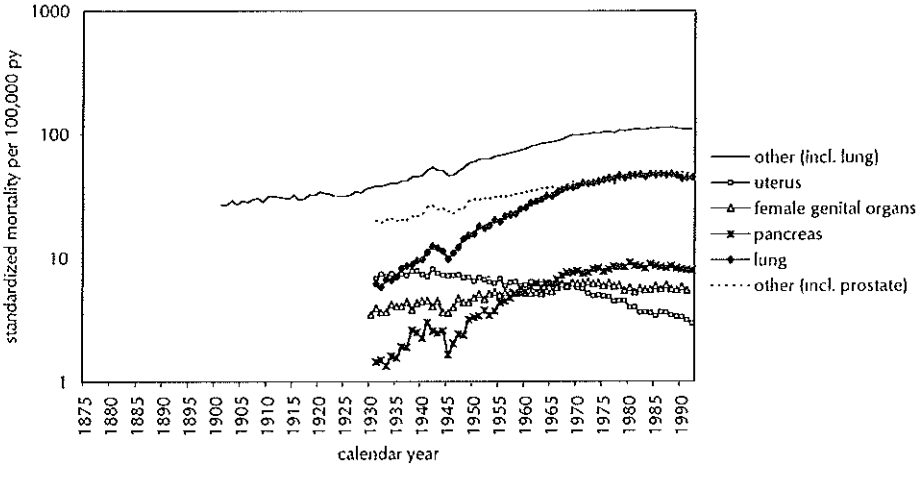


Figure 4.2b – An example of the nested classification: detail in cancer mortality, 1931-1992.

increase. Cancer of the uterus has declined since the Second World War and has shown a progressive decline since 1965. Other cancers, not shown in figure 4.2b, that could be distinguished at the 92 causes level are cancers of the stomach, liver, oesophagus, small intestines and colon, rectum and peritoneum.

4.4 Discussion

4.4.1 Usefulness of the method of Vallin and Meslé

The method of Vallin and Meslé is a good device to create nosologically continuous groups of causes of death for time trend analyses, because of the accurateness of the method. It was, however, not possible to apply directly the associations from Vallin and Meslé's work to the Dutch data. First of all, the period under study was considerably longer in our study (1875-1992) than in the case of France (1925-1991). Second, the associations for France are based on four-digit ICD-items. In The Netherlands, the causes of death have been registered on a four-digit level by the Central Bureau of Statistics, but were only available for analysis on a three-digit level. Therefore the associations could only contain three-digit items. During the construction of the associations we used the titles of four-digit items to determine which items had to be associated, but the end result was on a three-digit level. Third, the statistical bureaux of most countries have made country-specific adjustments to the ICD-revisions as adopted by the WHO. There have been several intra-ICD changes in The Netherlands, which resulted in 22 different 'revisions' in the period 1901-1992. Most of the intra-ICD changes did not influence the construction of associations, but some of them did.

We did not consider the last stage of Vallin's method appropriate for our study. That stage comprises a redistribution of deaths classified according to the previous ICD revision, within an association, using the ratio of deaths classified according to the next ICD revision. The advantage of this step is that the level of detail of the most recent ICD revision is kept over the period under study. It can, however, be doubted whether the underlying assumption of constant ratios of causes of death over one or more ICD periods is correct. Especially when those series are going to be used to study shifting cause of death patterns over time, it seems to be preferable not to impose beforehand a certain cause of death structure on the data.

4.4.2 The two criteria: detail and nosological continuity

The results presented in this article indicate that the criteria of detail and nosological continuity were sufficiently met. The construction of a nested classification enhanced detail and created a good representation of communicable, non-communicable and external causes of death for at least the period 1901-1992. The outcome was less favourable for the 27-causes classification for the period 1875-1992, in which most of the causes were communicable diseases and about one third of all causes were non-meaningful in the context of the epidemiological transition (cf. table 4.4). Nevertheless, 43% of all deaths was covered by a

meaningful cause of death even in 1875, which gives us the opportunity to study a considerable part of the changes in mortality in the 19th century.

As far as the nosological continuity could be quantified, we found that at every ICD-transition large percentages of the causes of death had discontinuities smaller than 10 percent. The definitive nested classification still contained many causes that had a discontinuity in at least, one of the ICD transitions. It should, however, be stressed that some of these are only discontinuous either from an absolute or a relative perspective. This means that for certain types of analyses those causes of death can be considered as continuous for the whole period. Many of the trends were discontinuous for the years 1900-1901 and 1940-1941, which shows the difficulty in bridging the Dutch 19th century classification and the first ICD revision and the influence of the Second World War. The comparatively clearly defined causes of death - mainly infectious - showed no discontinuity for the pre-ICD/ICD-1 transition; e.g. diphtheria, whooping cough, scarlet fever, violence. During the Second World War, there was not only an increase in certain causes of death due to malnutrition and reduced living standards, but there also was an effect on the notification of causes of death, which is shown by the enormous increase in the number of deaths classified as ill-defined or unknown.

As previously stated, by accepting discontinuities the criterion of nosological continuity was violated. One possibility of dealing with the discontinuities would be to combine the causes of death into broader categories. This would, however, be against the criterion of detail. Another possibility of keeping detail would have been the redistribution of numbers of deaths classified according to the previous revision by means of the ratio of deaths classified according to the next revision. This did not seem preferable either, as we discussed before. Therefore, we decided to deal with the remaining discontinuities in the next stage of our project i.e. that of analysing cause of death trends.

4.4.3 Interpreting time trends of nosologically continuous groups

Although the nosologically continuous groups are constructed with the utmost accuracy by comparing titles in ICD-manuals, it is still conceivable that the medical content of certain groups has changed over time. Especially in the case of ill-defined causes of death it might have been the case that a cause of death with the same title in all pre-ICD and ICD revisions had a different medical content in the late nineteenth century than in the twentieth century. The category 'old age' almost certainly included many more diseases in the nineteenth century than in the end of the 20th century, due to lack of medical knowledge and diagnostic devices or changing coding habits. The category 'chronic myocarditis' included 'myodegeneratio cordis' which might have included some types of ischemic heart

disease until ICD-6, although there was a separate category for lesions of the coronary artery in the ICD-revisions.

But even for clearly described causes of death like cancer we still have to be cautious in interpreting the time trends. Cancer mortality can be studied for the whole period 1875-1992 and shows a continuous increase. There was, however, a lot of debate in the late nineteenth and early twentieth century whether this trend was real or artefactual (De Haan 1899, Vollenhoven 1889, Astro 1902, Evers 1882, Drooglever Fortuyn 1924). The influence of ageing of the population and better diagnosis on cancer mortality was discussed. Astro concluded in his thesis on cancer mortality in Utrecht in the period 1872-1902 that the increase in cancer mortality was most likely artefactual, because it was mainly caused by an increase in cancer of the digestive system. The diagnosis of this type of cancer had been considerably improved by the end of the nineteenth century (Astro 1902). It is important to realise whether a trend is artefactual or real, because it affects the interpretation of changes in cause of death pattern.

Changes in registration procedures (e.g. the cause of death certificate) or coding rules might also have influenced the trend of certain causes of death. Important changes in the system of registration were introduced in the years 1926/1927 in The Netherlands. A confidential medical certificate of cause of death was introduced, which included information on the 'main disease' which had caused the patient's death, the 'complication' which had caused the patient's death, and the 'concomitant' causes of death. The confidentiality of the certificate led to an increase in mortality of certain causes of death. Clear increases in mortality in the years 1926/1927 were apparent in the trends of syphilis, diseases of male and female sexual organs and suicide. Also the more specific questions of main cause of death and complications led to changes in registration for several causes of death. Encephalitis and meningitis became less often registered as primary causes of death compared with the period before the new certificate. Septicaemia and pyemia also showed a decrease in mortality, because those causes of death became more often registered as 'complications' instead of 'main diseases'. There was an increase in the trend of diabetes mortality, which became more often registered as the primary cause of death.

The new international cause-of-death certificate as well as the international coding rules to identify the underlying cause of death has been used in The Netherlands since the introduction of ICD-6 in 1950. However we do not expect much effect of this change on cause specific mortality trends, because the distinction between a primary and a secondary cause of death had already been used in the registration of causes of death since 1927.

4.4.4 Developments in medical science and the pre-ICD/ ICD-1 transition

Not only the briefness of the pre-ICD classification but also the developments in medical science of the late 19th century hampered the bridging of the pre-ICD and ICD-1 classification. With the rise of bacteriology for many communicable diseases a causal agent could be defined in the 19th century. Those changes in medical science also affected the cause of death classification. Aetiology became more important in classifying causes of death, instead of anatomy or symptomatology (Fischer-Homberg 1977, Magner 1992). In the 19th century classification, for example, abdominal tuberculosis was classified under 'diseases of the digestive system' and tuberculosis of the nervous system under the heading 'diseases of the nervous system'. Besides classifying causes on the basis of location of the disease, another important characteristic, which determined the classification of was the duration of a disease: acute or chronic. By the time the 19th century classification was developed the tubercle bacillus as the underlying agent of a whole range of clinical patterns had not yet been discovered. This knowledge was available when the first ICD-classification was developed and in that classification the various types of tuberculosis are put under the same heading.

4.5 Conclusion

The first two steps of Vallin and Meslé's method, the construction of dual correspondence tables and fundamental associations, are useful for constructing nosologically continuous data on causes of death. In this study, we modified the method of Vallin and Meslé by creating a nested classification to retain detail instead of redistributing numbers of deaths according to the death ratios of the next classification. The nested classification allows us to study cause-specific mortality at three different levels of refinement of causes of death. The result of the reclassification procedure was satisfactory, as we achieved a good representation of communicable, non-communicable and external causes of death with continuous trends over time. We were able to show trends of several causes of death for a much longer period than previously had been presented.

More detailed studies on the epidemiological transition in other countries are needed to come up with explanations of the transition and to reveal differences between countries. Carefully constructed time series of causes of death are the basis of such analyses. The method described in this paper is easy to apply to other countries and the construction of correspondence tables and fundamental associations could be very useful for any country where bridge-coding is lacking.

Chapter 5

MORTALITY DECLINE IN THE NETHERLANDS IN THE PERIOD 1850–1992: A TURNING-POINT ANALYSIS

Abstract

Objective: *The aim of this paper is to give a detailed and fairly objective description of rapid mortality decline in The Netherlands between 1850 and 1992 with respect to the start, end, and phases of the decline.*

Data and Methods: *Turning points were estimated for the standardised mortality trend, and for age and sex-specific trends between 1850-1992. The used technique was derived from spline functions. The turning points divided the trends into phases with different paces of decline.*

Results: *Standardised mortality started to decline rapidly in The Netherlands around 1880. Four phases in the period of decline could be distinguished: 1880-1917 (1.2% annually), 1917-1955 (1.6%), 1955-1970 (0.4%), 1970-1992 (1.1%). For nearly all age groups, the most rapid decline occurred in a period comparable to 1917-1955. Causes of death which might have shaped the total mortality trend are, among others, respiratory tuberculosis (1917), heart disease (except ischemic) (1955), ischemic heart disease (1970).*

Conclusion: *Causes of death that shaped the mortality trend are related to trends of determinants of mortality decline. The technique used in this paper can also be applied to other trends e.g. fertility decline.*

5.1 Introduction

Over the last two centuries, most Western countries have seen enormous demographic and epidemiologic changes: mortality declined enormously, as did fertility, the population grew and disease patterns shifted. These major changes led to the formulation of two important theories viz. the demographic transition theory and the epidemiologic transition theory. The demographic transition theory explains the population growth in terms of a process in which there is a transition from a stage with high mortality and fertility rates to a stage with low mortality and fertility rates (Beaver 1975, Caldwell 1976, Chesnais, 1992).

In the early 1970s, Omran introduced the epidemiological transition theory. This theory can be regarded as a more detailed description of mortality decline in the demographic transition theory. Omran distinguished three stages in the epidemiologic transition. The first stage is the 'age of pestilence and famine' in which mortality is high and fluctuating. The second stage is the 'age of receding pandemics' in which mortality declines progressively. The rate of decline accelerates as epidemic peaks become less frequent and eventually disappear. The third stage is the 'age of degenerative and man-made diseases' in which mortality first continues to decline and eventually approaches stability at a relatively low level. The dominating causes of death of this stage are cardiovascular diseases,

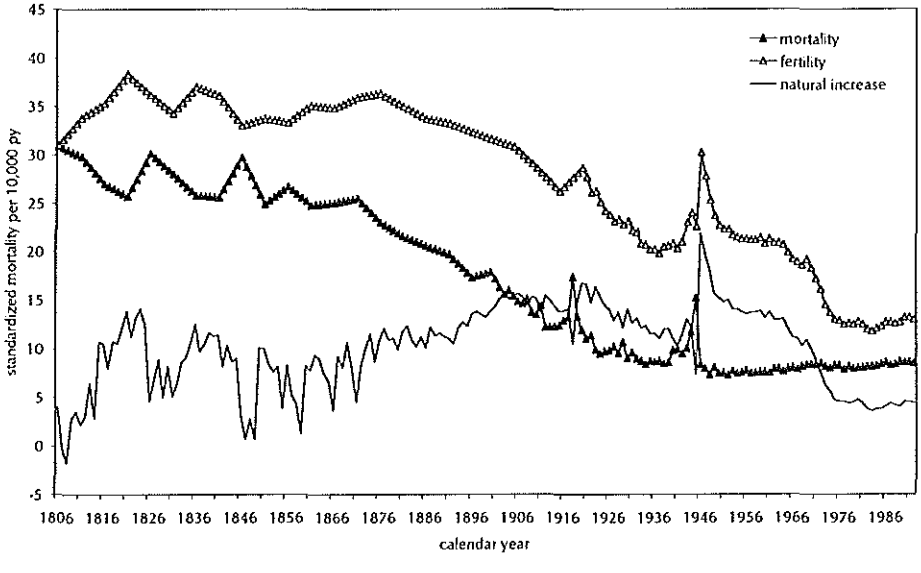


Figure 5.1 – The demographic transition in The Netherlands, 1806-1992. The birth rates are 5-year averages until 1924 (Hofstee 1981), and annual figures derived from Statistics Netherlands thereafter. Crude mortality figures were obtained from the Netherlands Interdisciplinary Demographic Institute.

cancer and external causes of death (Omran 1971, Omran 1983). The demographic and epidemiologic transition have been observed in many countries albeit with differences in start, development and duration of the transition.

Figure 5.1 shows the demographic transition as observed in The Netherlands (Hofstee 1981). It suggests that crude mortality rates had been declining slowly since the beginning of the 19th century and started to decline rapidly around 1875.

The aim of this paper is to give a detailed description of the period of rapid mortality decline in The Netherlands, or more specifically, to describe the start of rapid mortality decline, the phases in the decline in terms of changes in the pace of decline and the end of rapid mortality decline. A description of sex and age-specific mortality changes as well as cause-specific mortality changes is given to elucidate possible sex-specific, age-specific and cause-specific developments in mortality that might have shaped the total mortality trend.

The turning points in the mortality trend described in this paper are not based on a simple visual inspection of the trend, but on a more formal statistical method. A so-called turning point analysis was used to determine phases with different paces of mortality decline. The Netherlands provide a good opportunity for such an analysis because the quality of historical mortality and population data in The Netherlands can be considered as good in comparison to many other Western countries. Ever since 1865 medical doctors have been legally required to issue a medical certificate stating the cause of death, which was then given to the Registrar (Van Poppel 1997).

The advantage of a formal statistical method and reliable data is that these will generate more objective results, and thus provide a sound basis to relate changes in mortality to changes in determinants of mortality.

5.2 Data and Method

5.2.1 Data

Mortality data for the period 1850-1875 were derived from a mortality database constructed by the Netherlands Interdisciplinary Demographic Institute (NIDI) (Tabeau *et al.* 1994). Annual publications of mortality figures by Statistics Netherlands (CBS) were used for the period 1875-1992. Population data for the period 1850-1949 were also provided by the NIDI, and population data for the period 1950-1992 were again obtained from the CBS. The data were used to calculate age- and sex-standardised mortality rates to correct for the effect of changing age and sex distributions on mortality. Direct standardisation was carried by out using the total population from 1901-1992 as the reference population. Eight age groups were used in the standardisation procedure, which was the

greatest common denominator of all age subdivisions used in the study period (1850-1992). The age groups were 0, 1-4, 5-14, 15-19, 20-49, 50-64, 65-79 and 80 years and over.

The influence of 27 causes of death to the turning points in the total standardised mortality trend is presented in this study. The 27 cause of death categories are the result of an extensive reclassification procedure of all cause-of-death classifications in the period 1875-1992, i.e. nine revisions of the International Classification of Diseases, Injuries and Causes of Death (ICD) and one pre-ICD classification. This reclassification procedure has been described elsewhere (Wolleswinkel-van den Bosch *et al.* 1996). Appendix 3 contains the 27 cause-of-death groups together with the corresponding codes of the ninth ICD revision.

5.2.2 Turning point analysis: total mortality and age and sex-specific mortality

In order to identify turning points in the mortality trend that mark periods with a different rate of mortality decline we used a derivation of a spline function. Spline functions are used to describe global movements by estimating turning points that characterise significant changes in that movement. Spline functions may be regarded as a sequence of polynomials each fitted to another part of the study period, which pair-wise have the same values in each knot (Kendall 1973, McNeill *et al.* 1977, Suits *et al.* 1978). The function used in this study is given below (De Beer 1986).

$$\ln Y_t = \alpha_0 + \alpha_1 t + \sum_{i=2}^{j} \alpha_i (t - t_i) D(t - t_i) + \varepsilon_t$$

Y_t is the standardised mortality in year t; t_2 t_3 ... t_i are the turning points; α_0, α_1 ... α_i are the intercept and slopes; ε_t is an error term and $D(t-t_i)$ is a step function,

$D(t-t_i) = 0$ if $t <= t_i$
$D(t-t_i) = 1$ if $t > t_i$
i = turning point

In the model $D(t-t_i)$ is defined as

$$D(t - t_i) = (1 + \sqrt{(t - t_i)^2}\,/(t - t_i)/2$$

The TSP program was used to solve this non-linear least squares regression problem. The number of turning points and initial values for the turning points

and coefficients have to be set to run the model. The optimum solutions for the parameters α_0, α_1 ... α_i and t_i are estimated by minimising the sum of squares of the residuals, which is an iterating process. At every iteration step the values of the parameters are slightly changed, and it is determined whether the sum of squared residuals has decreased. If it has, the changes are adopted and a new iteration step begins. The process continues until the proposed changes are very small in comparison to the parameters themselves (Hall *et al.* 1990). Residual analysis may show that the model is not adequate. A model with a smaller number of turning points was estimated if a significant difference between the slopes before and after the turning point was not found. A model with a larger number of turning points was estimated if there was serious autocorrelation of the residuals (Durbin-Watson statistic) and the sum of squared residuals was relatively high.

The rates of change in the different periods can be calculated as follows:

$$100\{\exp(\sum_{i=1}^{j}\alpha_i)-1\}\%$$

The years 1940-1946 (Second World War) as well as the influenza epidemic of 1918 were excluded from the analyses because these events caused extraordinarily high peaks in the mortality trend, which hampered a good estimation of turning points in most cases.

5.2.3 Contribution of causes of death

Causes of death which greatly contributed to the level of total mortality in the 'turning point years', and which showed a considerable change in the pace of mortality in the period after the turning point compared to the period before the turning point, have probably played a role in the occurrence of the turning points in the total mortality trend. Therefore rates of change of 27 causes of death, which could be studied for the period 1875-1992, were calculated for only those phases in mortality decline for which reliable cause-specific data were available. A simple linear regression analysis with the natural logarithm of mortality as the dependent variable and calendar year as the independent variable was used to calculate the rates of change. In these analyses the years 1918 (influenza epidemic) and 1940-46 were excluded, as was the case in the turning point analyses on total mortality. During the Second World War high peaks in mortality due to diphtheria, tuberculosis, acute digestive diseases and violent death were seen in The Netherlands (Lumey & Van Poppel 1994).

5.3 Results

5.3.1 The onset of rapid mortality decline

Table 5.1 represents the turning points and rates of change for the distinguished periods in the total mortality trend and in sex and age-specific mortality trends for the period 1850-1992. The results indicate that mortality started to decline in 1855, because then a change occurred from a period of increase to a period of decrease in mortality rates. The sex and age-specific results show that mortality

Table 5.1 – Turning points and growth rates for the total mortality trend and sex and age-specific mortality trends in The Netherlands, 1850-1992

	Turning point	Period	Annual growth rate (%)		Turning point	Period	Annual growth rate (%)
Total		1850-1855	4.8	**Age 5-14**		1850-1859	4.6
	1855	1855-1880	-0.8		1859	1859-1890	-2.4
	1880	1880-1917	-1.2		1890	1890-1910	-3.4
	1917	1917-1955	-1.6		1910	1910-1928	-2.0
	1955	1955-1970	-0.4		1928	1928-1951	-4.4
	1970	1970-1992	-1.1		1951	1951-1970	-1.7
Males		1850-1855	4.6		1970	1970-1992	-3.8
	1855	1855-1881	-0.7	**Age 15-19**		1850-1859	3.1
	1881	1881-1917	-1.2		1859	1859-1919	-1.3
	1917	1917-1952	-1.6		1919	1919-1957	-4.7
	1952	1952-1972	0.3		1957	1957-1972	2.3
	1972	1972-1992	-0.8		1972	1972-1992	-3.3
Females		1850-1858	2.8	**Age 20-49**		1850-1858	2.3
	1858	1858-1919	-1.1		1858	1858-1881	-1.3
	1919	1919-1960	-1.8		1881	1881-1920	-1.9
	1960	1960-1992	-1.3		1920	1920-1955	-2.8
Age 0		1850-1871	0.6		1955	1955-1992	-1.1
	1871	1871-1905	-1.2	**Age 50-64**		1850-1855	5.1
	1905	1905-1924	-4.3		1855	1855-1917	-0.9
	1924	1924-1948	-3.1		1917	1917-1957	-1.4
	1948	1948-1957	-5.1		1957	1957-1970	0.7
	1957	1957-1992	-3.0		1970	1970-1992	-1.3
Age 1-4		1850-1859	2.6	**Age 65-79**		1850-1854	4.3
	1859	1859-1888	-1.7		1854	1854-1922	-0.4
	1888	1888-1919	-3.0		1922	1922-1961	-0.9
	1919	1919-1947	-5.8		1961	1961-1970	0.1
	1947	1947-1992	-3.6		1970	1970-1992	-1.0
				Age 80+		1850-1855	3.5
					1855	1855-1927	-0.04
					1927	1927-1992	-0.7

started to decline around 1855 for both sexes and for all age groups, except for the age group 0-1. In this age group mortality started to decline about 15 years later.

5.3.2 Phases in the period of rapid mortality decline

Total mortality

The results of the turning point analysis show five phases with different paces of mortality decline in the period 1855-1992 (table 5.1, figure 5.2). A first acceleration in the decline started in 1880. The most rapid decline (1.6% annually) occurred in the period 1917-1955. This period was followed by a phase (1955-1970) of very slow mortality decline (0.4% annually). Then mortality started to decline rapidly again (1.1%).

Sex-specific mortality

Male and female mortality trends differ with respect to turning points in the pace of mortality decline (table 5.1). Female mortality started to decline in 1858 at a

Figure 5.2 – Total mortality decline in The Netherlands, 1850 to 1992.

Table 5.2 – The contribution of several causes of death to total mortality in turning point years and the rate of change in the periods 1880-1917, 1917-1955, 1955-1970 and 1970-1992.

	1880 % total mortality	1880-1917 % annual change	1917 % total mortality	1917-1955 % annual change	1955 % total mortality	1955-1970 % annual change	1970 % total mortality	1970-1992 % annual change
Respiratory tuberculosis	9.68	-1.96	8.71	-6.56	0.70	-13.21	0.09	-10.45
Acute respiratory diseases	9.74	-0.83	11.72	-4.91	4.10	-4.04	3.70	-2.71
Measles	0.97	-1.17	0.49	-9.46	0.04	-7.80	0.02	eradicated
Diphtheria	1.16	-5.78	0.42	-2.61	0.05	eradicated	0.00	eradicated
Whooping cough	1.08	-2.19	0.70	-7.37	0.03	eradicated	0.00	eradicated
Scarlet fever	0.42	-4.07	0.20	-7.05	0.02	-20.64	0.00	eradicated
Brain diseases etc.	8.33	-2.38	5.82	-3.53	2.68	-1.52	2.14	0.09
Acute digestive diseases	6.44	-1.63	4.90	-3.46	1.49	-1.02	1.23	-1.02
Cholera	0.13	-7.07	0.02	eradicated	0.00	eradicated	0.00	eradicated
Typhoid fever	0.93	-4.43	0.29	-9.13	0.01	eradicated	0.00	eradicated
Malaria	0.49	-12.06	0.01	-5.20	0.00	eradicated	0.00	eradicated
Smallpox	0.19	-13.50	0.00	eradicated	0.00	eradicated	0.00	eradicated
Scurvy	0.06	-0.24	0.04	-9.77	0.00	eradicated	0.00	eradicated
Puerperal fever	0.08	-1.84	0.13	-8.16	0.01	-4.81	0.00	eradicated
Other diseases of pregnancy	0.61	-2.72	0.29	-2.97	0.16	-10.29	0.03	-4.77
Chronic digestive diseases	3.64	-2.94	2.08	-1.49	2.02	-0.59	1.96	-0.67
Chronic respiratory diseases	8.33	-3.78	3.58	-2.46	2.72	1.86	3.73	0.58
Genito-urinary diseases	2.16	0.44	4.31	-2.05	2.83	-2.05	2.13	-2.31
Cerebrovascular diseases	3.96	-0.08	6.73	-0.13	13.82	-1.77	11.27	-2.44
Diabetes	0.05	6.85	1.05	-1.48	1.69	-0.05	1.42	2.17
Heart disease (total)	4.05	1.99	12.41	0.56	30.46	0.02	32.85	-1.68
IHD	n.a.	n.a.	0.30	8.31	10.80	3.72	21.41	-2.34
Other heart diseases	n.a.	n.a.	12.11	-0.08	19.66	-4.03	11.44	-0.46
Cancer	3.38	1.80	9.39	0.15	19.62	0.90	22.94	-0.07
Other diseases (incl. 'debility')	24.96	-2.26	17.74	-2.96	7.75	-2.73	5.03	-0.77

Congenital malformations	0.54	-3.08	0.40	2.31	1.67	-1.78	1.38	-1.88
External causes (total)	1.83	-0.75	2.15	1.38	4.49	2.24	6.29	-4.35
Traffic accidents	n.a.	n.a.	0.26	2.91	1.96	3.25	3.23	-5.49
Suicide	0.27	0.63	0.46	-0.45	0.77	0.54	1.02	0.66
Unknown/ill-defined	6.60	-1.20	5.93	-2.57	2.87	-0.45	2.76	-1.04
All causes	100.00	-1.24	100.00	-1.51	100.00	-0.35	100.00	-1.16

rate that was not reached by males until 1881. A turning point near the year 1880 was not found for the female mortality trend. Male mortality increased in the period 1952-1972 while female mortality continued to decline. Contrary to males, female mortality has been declining continuously since 1858 at a fairly high rate.

Age-specific mortality

The turning point in 1880, in the total mortality trend, could only be found in age group 20-49. Other age groups with turning points near the turning point in 1880 are age group 0-1 (1871) age group 1-4 (1888) and 5-14 (1890) (table 5.1).

Turning points near 1917 and 1955 (total mortality decline) were found for most age groups (1-4, 15-19, 20-49, 50-64 and 65-79) (table 5.1). The period between those turning points shows the most rapid decline in the whole period of mortality decline for these age groups.

Total mortality decline decelerated in the period 1955-1970. An increase in mortality was found in age groups 15-19, 50-64 and 65-79 in a period equivalent to 1955-1970 (table 5.1).

The rates of change were generally higher for age groups 0-1 to 15-19 compared to the rates of change of older age groups both in the late 19th and early 20th centuries, and also during the remainder of the 20th century. It was hard to give a reliable estimation of the turning points for the age group 80+, because of the highly fluctuating trend for this age group until the Second World War.

Cause-specific mortality

Table 5.2 shows the importance of the 27 causes of death in terms of contribution to the level of total mortality in the turning point years, and in terms of the rates of change in the phases of the total mortality trend for which cause-specific mortality data were available, i.e. 1880-1917, 1917-1955, 1955-1970 and 1970-1992. A change in the rate of decline of important causes of death might indicate that those causes of death played a role in the changes in the total mortality trend.

Causes of death which contributed largely to the level of mortality in 1880 were 'other diseases (including debility)' (mainly consisting of diseases of the newly born, and 'old age') (25.0%), respiratory tuberculosis (9.7%), acute respiratory diseases (9.7%), 'brain diseases etc.' (mainly meningitis and tuberculosis of the meninges) (8.3%), chronic respiratory diseases (8.3%), unknown and ill-defined causes of death (6.6%), and acute diseases of digestive system (6.4%). The pace of decline was relatively rapid for 'other diseases', 'brain diseases etc.' and chronic respiratory diseases in the period 1880-1917 in comparison to the other important causes of death. However, because of a lack of data for the period 1855-1880 we cannot draw firm conclusions as to which causes of death might be responsible for the acceleration of mortality decline after 1880.

The causes of death mentioned above were still important in 1917, although the percentage of contribution to total mortality had declined. But other causes, viz. cerebrovascular disease (6.7%), heart disease (12.4%) and cancer (9.4%) also became prominent. The most rapid acceleration in mortality decline can be seen for respiratory tuberculosis (from 2.0% to 6.6%), acute respiratory diseases (from 0.8% to 4.9%) and acute digestive diseases (from 1.6% to 3.5%).

Important causes of death in 1955 were heart disease (30.5%), cancer (19.6%), cerebrovascular disease (13.8%), 'other diseases (including debility)' (7.8%), violence (4.5%) and acute respiratory diseases (4.1%). The pace of decline of the, previously rapidly declining, causes of death 'other diseases (including debility)' and acute respiratory diseases slightly decelerated in the period 1955-1970 compared to the period 1917-1955. More important, however, are the trends of heart diseases and cancer. The mortality of heart disease reached stability in the period 1955-1970 (0.02% annual change). The stability of this trend was caused by two complementary trends, viz. the increasing trend of ischemic heart disease and the decreasing trend of other heart diseases. The pace of increase of cancer mortality was not high in the period 1955-1970, but accelerated in this period (0.9%) compared to the period 1917-1955 (0.2%).

Heart diseases and cancer were the most important causes of death in 1970 (32.9% and 22.9% respectively). The trend of both causes of death changed from an increasing to a decreasing trend after 1970. The same change in trend, but more obvious, was apparent for a less important cause of death i.e. external causes of death (6.3%), and more specifically traffic accidents (3.2%).

5.3.3 The end of rapid mortality decline

A deceleration of mortality decline occurred in the period 1955-1970, which might be regarded as the end of a period of rapid mortality decline. However, mortality started to decline rather rapidly again after 1970 (1.1% annually) (table 5.1).

There was even an increase in mortality in the period 1955-1970 for males, but then mortality started to decline again although less progressively than total mortality decline (0.8%). There was no deceleration in female mortality decline that could be labelled as the end of rapid mortality decline (table 5.1).

Periods of increasing mortality could only be found for the age groups 15-19, 50-64 and 65-79 all of which were followed by a period of renewed, relatively rapid, decline (table 5.1).

5.4 Discussion

Mortality decline in The Netherlands in the period 1850-1992 can be divided into five phases with different paces of decline, viz. 1855-1880 (0.8% annually), 1880-1917 (1.2%), 1917-1955 (1.6%), 1955-1970 (0.4%), and 1970-1992 (1.1%). Male mortality figures showed roughly the same phases in mortality decline as total mortality, with the exception of an increase in mortality in the period 1952-1972. Female mortality could only be divided into three periods with different paces of decline viz. 1858-1919 (1.1% decline), 1919-1960 (1.8%), and 1960-1992 (1.3%). The most rapid decline was found in nearly all age groups in the period 1917-1955. Causes of death which played a role in the location of the turning points in the total mortality trend are 'other diseases (including debility)', chronic respiratory diseases, respiratory tuberculosis, 'brain diseases etc.' (turning point 1880), acute respiratory diseases, respiratory tuberculosis, acute digestive diseases (1917), heart diseases (1955), ischemic heart diseases, cancer (1970).

Before we will discuss these results and the relationship with possible determinants of mortality decline, it is necessary to comment briefly on the data and method that were used in this study.

5.4.1 Evaluation of data and methods

Cause-specific mortality

The reliability of the (cause-specific) mortality data was already briefly mentioned in the introduction. An important characteristic of the Dutch cause-of-death registration is that is has been a medical registration since 1865. Although medical knowledge was not as extensive in the late nineteenth century as it is today, the fact that medical doctors were involved in the certification of causes of death in The Netherlands instead of laymen or clergymen, which was the case in many other European countries (Johansen 1993, Kintner 1993, Rogers 1993), improved the reliability of the Dutch cause-of-death statistics. It took, however, some time before cause of death statistics were considered reliable for medical-statistical research. Although doctors were required to issue a medical certificate of cause of death to the registrar (Medical Practitioners Act 1865), the registrar himself was not obliged to request one. After the Burial Act of 1869, in which a medical certificate with the cause of death stated was required in order to bury someone, the quality of the cause of death statistics improved. Since 1875/79, quinquennial mortality statistics were published by age, sex and cause of death, with identical nomenclature until 1895/99. Those statistics were considered extremely useful to anyone involved in medical-statistical research (Van Poppel 1997). Therefore, cause-of-death statistics from 1875/79 onwards are used in this study.

The causes of death used in this study are the result of an extensive reclassification procedure. This reclassification was undertaken to create nosologically continuous categories of causes of death, and to reduce the influence of changes in cause-of-death classification on cause-specific mortality trends to a minimum (Wolleswinkel-van den Bosch *et al.* 1996). However, the nosological content of the cause of death categories is also influenced by the accuracy of the diagnosis made by the medical doctors. Especially the difficulties to distinguish between respiratory tuberculosis and other respiratory diseases have been reported. In the 19th century, respiratory tuberculosis was also known as 'phtisis' or 'consumption'. Phtisis could, however, also occur from other causes than tuberculosis. The accuracy of diagnosing tuberculosis might also have been affected by the stigma attached to tuberculosis in the 19th and early 20th century. Tuberculosis was thought to be hereditary, and family of tuberculosis patients could face problems with their insurance (Hardy 1988, Bryder 1996). Bryder points out that there were still diagnostic problems of respiratory tuberculosis in the 20th century despite of bacteriological tests and X-ray facilities (Bryder 1996).

It is difficult to assess the impact of diagnostic uncertainties on the cause specific mortality results presented in this study. The possibility of exchanges between 'respiratory tuberculosis' and 'chronic respiratory diseases' or 'acute respiratory diseases' (including pneumonia) was mentioned by some Dutch 19th-century authors, but considered as a minor problem by others (Evers 1882, Vollenhoven 1889, Saltet, 1909). There are some reports that cases of acute respiratory and digestive diseases in the late nineteenth century might have wrongly been notified as cases of chronic respiratory and digestive diseases, and vice versa (Evers 1882). The trend of mortality from unknown and ill-defined causes of death might be an indication of development in accuracy of diagnosis. Table 5.2 shows an acceleration in mortality decline of unknown and ill-defined causes of death in the period 1917-1955 compared to 1880-1917. This suggests an improvement in diagnosis in the period 1917-1955. The rates of change for the causes of death that played a role in the turning point in 1917, i.e. respiratory tuberculosis, acute respiratory diseases, and acute digestive diseases, are much more rapid in the period 1917-1955 than the rate of change of unknown and ill-defined causes of death (cf. table 5.2). This suggests that improvements of diagnosis can not explain all of the changes in the trends of those causes of death.

A final remark with respect to cause-of-death categories has to be made on the broad cause-of-death groups present among the 27 causes used in this study. Some of these groups consist of a combination of infectious and non-infectious diseases e.g. 'other diseases (including debility)', 'brain diseases etc.'. It should be kept in mind that the distribution of deaths within these broad groups will change over time from predominantly infectious to non-infectious diseases. The category 'brain diseases etc.' consisted predominantly of (tuberculous)

meningitis and convulsions in the late 19th and early 20th centuries, but over time non-communicable diseases such as Parkinson's disease became more important. With respect to 'other diseases (including debility)' this category consists of a variety of causes (cf. Appendix 1and 3) among which causes related to the 'newly born' and causes related to 'old age'. It is likely that those causes also included infectious diseases. Over time the cause 'diseases of the newly born' will more specifically refer to, for example, perinatal mortality and 'diseases of old age' to, for example, dementia.

Turning point analysis

In this study turning points in mortality trends from 1850 to 1992 were estimated. In particular during the nineteenth century, mortality was highly fluctuating. Such fluctuations can mask the 'real' mortality trend and hamper the estimation of turning points (Suits *et al.* 1978, Perrenoud 1989). Extremely high mortality peaks due to epidemics or other extraneous events such as war can induce turning points that are solely related to such events. For that reason, the years of the influenza epidemic and the Second World War were excluded from the analyses.

The results showed an early turning point around 1855 for most of the analysed trends, which marked the end of a period of increasing mortality (1850-1855) followed by a period of decreasing mortality (1855-1917). However, it is conceivable that the early years of the 1850s were years with relatively low mortality as a reaction to the high mortality rates during the cholera epidemic in 1848/49. The increase in the period 1850-1855 would then be artificial. We checked this by carrying out an additional analysis for total *crude* mortality, starting in the year 1804. The data for this analysis were derived from a publication of the Dutch Statistical Bureau (Oomens 1989), and are only available for total mortality, and not for age and sex-specific mortality. Therefore the analyses presented in this study were based on the period 1850-1992. The years 1847-1849, 1866 and 1871 were excluded from this analysis, because of epidemics in those years viz. cholera (1847-1849), smallpox (1866), and cholera again (1871) (cf. figure 5.1). The result of the additional analysis for crude mortality shows turning points around 1836, 1870, 1917, 1955, and 1970. Mortality declined with 0.4% annually in the period 1804-1836, with 0.1% annually in the period 1836-1870, and with 1.1% annually in the period 1870-1917. The decline in the other periods is similar to the results of the analysis for the period 1850-1992 (cf. table 5.1). This analysis shows that the turning points around 1855 in the total standardised mortality trend and sex, and age-specific mortality trends are likely to be artefacts caused by the preceding cholera epidemic. Therefore, we consider the rapid total standardised mortality decline to have started around 1880 (cf. table 5.1).

Big fluctuations also hampered a good estimation of turning points in the trend of age group 80+. Mortality for this age group was enormously fluctuating from one year to the next until the Second World War. Smoothing the trend by calculating 5-year moving averages did not improve the estimation of the turning points.

Recent changes in the pace of mortality decline, for example changes in the late 1980's, might not have been detected in this type of analysis. The period until 1992 might be too short to detect a turning point. Besides, the rates of change for the most recent period detected in a turning point analysis should not be extended to future years.

5.4.2 The onset and phases of rapid mortality decline: the relationship with determinants of mortality decline

A brief review of the literature on trend and determinants of mortality decline

Secular mortality decline is considered to have started in the mid 18th and early 19th centuries for several European countries e.g. France, England & Wales, Sweden (Wrigley & Schofield 1981, Perrenoud 1984, Fridlizius 1984). Economic changes in the pre-industrial agricultural sector, climatic changes, a changing relationship between host and infective agent (e.g. improved nutritional status of the host) and changing attitudes towards childcare have been put forward as determinants of pre-industrial mortality decline. Mortality decline in the late nineteenth century was more rapid and has mostly been related to industrialisation (Bengtsson *et al.* 1984), or modernisation (Omran 1971) as the cause of mortality decline.

Many determinants of mortality decline, which started in the late nineteenth century, have been described in the literature. In this paper, we will distinguish four sets of determinants viz. socio-economic factors, socio-cultural factors, public health measures and medical factors. *Socio-economic determinants* are regarded as important factors in mortality decline. Industrialisation was already mentioned as a determinant of the onset of mortality decline. The early phases of industrialisation and urbanisation could, however, negatively affect health e.g. the dusty and damp factories and bad urban sanitary conditions. For Britain, it has been described that living standards did not increase during the early stages of industrialisation (Armstrong 1981, Huck 1995), and that bad urban sanitary conditions had negative effects on infant health (Woods *et al.* 1988, 1989). For The Netherlands, however, such strong negative health effects have not been reported (De Jonge 1968). The beneficial effect of industrialisation is the improvement of the economic wealth of a country and consequently the improvement in the standard of living of its inhabitants. Aspects of the standard of living

that influence mortality are, for example, availability of food, housing and working conditions.

Thomas McKeown considered improved nutritional status as the most important factor of nineteenth century and early twentieth century mortality decline (McKeown 1976a,b). Causes of death that are related to nutritional status are e.g. respiratory tuberculosis and acute respiratory diseases (pneumonia). Housing conditions also influence the occurrence of respiratory diseases, but 'diseases of the nervous system', which consists mainly of meningitis (related to crowding and bad hygiene) might have been influenced by housing conditions too. Working conditions might also have had an effect on respiratory diseases, and probably predominantly on males aged 20-49 and 50-64.

Socio-cultural determinants have also been brought up as a factor in mortality decline. The Dutch demographer Hofstee came up with a hypothesis in which mortality decline was related to the extent to which new ideas, especially with respect to hygiene, could diffuse in a society. New ideas would be more easily accepted in modern dynamic societies compared to traditional societies (Hofstee 1979, 1981). The acceptance of certain health ideas would lead to a change in behaviour of the population e.g. changing breastfeeding practices and changes in child care in the late 19th and early 20th centuries, or changes in smoking habits in the late 20th century. The effects of changes in breastfeeding practices and child care are expected to be seen in age groups 0, and 1-4, and probably mostly in the cause-of-death category of acute digestive diseases.

Fertility is a determinant of mortality decline that is (partially) determined by cultural factors. There is an interaction between levels of fertility and mortality. Mortality decline can induce fertility decline because more children will survive to adult ages and thus reduce the need for the replacement of children that died. Fertility decline on the other hand can also induce mortality decline. Birth spacing, for example, is negatively associated with child and maternal mortality (Forste 1994, LeGrand & Philips 1996).

Another set of determinants of mortality are *public health measures*. One of the main criticasters of McKeown's 'nutrition-thesis', Szreter, argued that the public health movement working through local governments, which resulted in the implementation of preventive measures of municipal sanitation and regulation of the urban environment, was the true force behind the decline in mortality in the period 1850-1914 (Szreter 1988). Public health measures such as the construction of sewage and water supply systems predominantly affected mortality from acute digestive diseases, typhoid fever and cholera. The construction of sewage and water supply systems started in The Netherlands in the late 19th century, but a coverage of more than half of the population was not reached until after the turn of the century (Vogelzang 1956, Van Zon 1986). Economic

wealth and the public health movement both played a role in the introduction of these public health measures.

The last set of determinants, *medical care and technology*, became particularly important after the introduction of antibiotics after 1945, but medical determinants might also have played a role before, although it was less important than e.g. economic determinants. Two examples of early effects of medical care are smallpox vaccination and the anti-diphtheria serum. The introduction of smallpox vaccination in the late 18th, early 19th centuries coincided with a considerable decline in smallpox mortality. In the 1870s a resurgence of the epidemic led to an enforcement of vaccination laws in many European countries (Mercer 1985). In The Netherlands, the Law on Communicable Diseases of 1872 stated that a written confirmation of vaccination against smallpox was required from teachers and children to enter the school. Although mortality rates declined, the effects were not as large as compared to countries with compulsory smallpox vaccination (Burgmeijer & Bolscher 1995). The introduction of anti-diphtheria serum in The Netherlands in 1896 might have influenced the decline of mortality from diphtheria in the late 19th and early 20th century (Saltet 1909). The possibility that a diminishing of bacterial virulence played a role in diphtheria mortality decline has, however, also been suggested (Hardy 1993).

The First World War had an impact on medicine viz. the development of health education induced by the need for fit human resources (Rosen 1993). In The Netherlands, tuberculosis control was intensified after the First World War. Special tuberculosis clinics were set up. An important role of these clinics was to investigate the social setting of the patient and to give recommendations on the amount and nature of social aid. Besides, the clinic played an important role in health education (Querido 1968, Sickenga 1980). It is not likely that the introduction of BCG immunisation in 1921 greatly affected mortality due to tuberculosis. In The Netherlands, BCG vaccination has never been applied to the general population. The administration of the BCG vaccine has always been restricted to high-risk groups (Burgmeijer & Bolscher 1995). After the Second World War a new or accelerated mortality decline was found, among other causes of death, for scarlet fever, rheumatic fever, influenza, tuberculosis, bacillary dysentery, which was probably related to the introduction of antibiotics (Mackenbach & Looman 1988).

In the remainder of this paper we will discuss which determinants of mortality decline are most likely to have played a role in the subsequent changes in the pace of mortality decline in The Netherlands, thereby using the results of the sex, age, and cause-specific analyses.

5.4.3 The onset and first phase of rapid mortality decline in The Netherlands: 1880-1917

The pattern of mortality decline in The Netherlands, i.e. slowly declining mortality in the first half of the 19th century and a progressive decline afterwards (cf. figure 5.1), is in accordance with other literature on the start of secular mortality decline in other European countries. The result that rates of change in female mortality were higher during most periods of mortality decline in comparison to males, and that rates of change for younger age groups were generally higher than for adult age groups, are in accordance with Omran's propositions that females were favoured over males and children over adults (Omran 1971, 1983).

Possible determinants of decline

• *Socio-economic determinants* – Industrialisation was mentioned as an important factor in the onset of mortality decline. In The Netherlands, we find similarities in the timing of mortality decline and industrialisation. The first symptoms of industrialisation occurred around 1870, and it really started around 1890 (De Jonge 1971). In the first phase of progressive mortality decline (1880-1917) we then expect to see the effects of industrialisation such as the rise in living standards. Real wages increased in The Netherlands from 1870 onwards until 1930. There was an acceleration in the increase in the period 1901-30 compared to the former years (Van der Spek 1976).

Data on nutrition, e.g. calorie-intake, hardly exist in The Netherlands for the late nineteenth and early twentieth centuries. There are, however, some data about the availability of certain foodstuffs per capita such as wheat, rice, potatoes, sugar, and beef which all increased from 1850 until 1882-1886 and stabilised afterwards until 1916 (CBS 1895, CBS 1920).

Data on housing and working conditions were also hardly available. Measures to improve housing conditions were taken around 1900 (Querido 1968). Data on the average number of persons per room show that there were 1.65 persons/room in 1899, 1.43 in 1909, 0.95 in 1930, and 0.80 in 1956 (CBS 1994). This reduction of the number of persons per room was seen in urbanised as well as rural areas.

Other data that reflect the standard of living are height data of twenty-year-old males that were examined for military service in The Netherlands. Data for the period 1863 to 1941 show a decrease in the percentage of males under 1 metre 55 until 1905, a stabilisation until 1922 and a renewed decrease after 1922 (CBS 1900-1940). Bearing in mind that height at age twenty reflects living standards at young ages, a time lag of about 10 to 20 years should be considered to interpret the data on height as a measure of living standards. The height trend might reflect an increase in living standards until 1885/95, a stabilisation in the

period 1885/95 to 1900/10, and a renewed increase in living standards after 1900/10.

• *Socio-cultural determinants* – Another factor that probably influenced mortality decline in the period 1880-1917 is fertility. Figure 5.1 shows that birth rates started to decline around 1880. Coale determined the onset of marital fertility decline in The Netherlands in 1897 (Coale & Watkins 1986), which is included in the period 1880-1917. Fertility rates declined rapidly until 1930, and a renewed decline set in after the Second World War (CBS 1994).

In the early twentieth century, the percentage of people without religious affiliation sharply increased (CBS 1994). Secularisation is a measure of the culture changes in the modernisation process such as rationalisation, openness to new ideas.

• *Public health measures* – The effects of public health measures are expected to emerge in the first phase of rapid mortality decline. As mentioned before, the introduction of sewage and water supply systems in many towns started in the late nineteenth century. Other research showed that a cluster of causes of death among which typhoid fever, convulsions and acute digestive diseases declined most rapidly in the late nineteenth and early twentieth centuries (Wolleswinkel-van den Bosch et al. 1997).

In this period no large effects of medical factors on mortality decline were expected. Improvements in living standards (probably mainly housing), socio-cultural change (e.g. fertility declines) and public health improvements can all have contributed to mortality decline in the period 1880-1917.

Cause and age-specific mortality decline

Causes of death that greatly contributed to mortality in 1880 are 'other diseases (including debility)', respiratory tuberculosis, acute respiratory diseases, brain diseases etc., chronic respiratory diseases and acute digestive diseases. Among these causes of death 'other diseases (including debility)', brain diseases etc., chronic respiratory diseases and respiratory tuberculosis showed relatively rapid declines in the period 1880-1917 (cf. table 5.2), which could indicate that these causes of death were important in the first phase of rapid mortality decline in The Netherlands.

The decline of respiratory tuberculosis, chronic respiratory diseases and 'brain diseases etc.' (31% meningitis) might all be related to improvements in housing. 'Brain diseases etc.' also covers 'convulsions' (42%). Convulsions can be a symptom of various infectious diseases, but several studies reported a relationship between convulsions and diarrhoeal diseases/dysentery (Kintner 1986, Wolleswinkel-van den Bosch *et al.* 1997). Therefore, the decline in mortality from

'brain diseases etc.' might also be (partially) related to sanitary improvements (public health measures).

The rapid decline in chronic respiratory diseases as well as the turning point in 1880 for age group 20-49 suggest that improved working conditions played a role in mortality decline in the period 1880-1917. However, measures by the central government to improve working conditions were taken as late as 1889. The measures include the Labour Act to reduce working hours, and in 1895, the Factory Safety Act to reduce accidents.

Another cause of death, which showed a rapid decline in the period 1880-1917 was 'other diseases (including debility)' (predominantly deaths under 5 years of age and 'old age'). The mortality trend for age group 1-4 showed a turning point in 1888. According to Evers (1882), the majority of deaths in the category 'other diseases (including debility)' under the age of 5 was due to poor nutrition. However, cultural changes, for example new ideas on childcare, might also have played a role.

We found a turning point around 1875 in the mortality trend for females aged 20-49 (results not shown), and an acceleration of infant mortality decline in 1905. These age-specific mortality declines might be related to the onset of marital fertility decline around 1897 (cf. table 5.1).

5.4.4 The period 1917 - 1955: acceleration of the decline

After 1917 a period of rapid mortality decline started and lasted nearly four decades (excluding the Second World War). The decline was apparent in male and female mortality and in a young age group (1-4), but was most apparent in adult age groups (15-19, 20-49, 50-64 and 65-79) (cf. table 5.1).

Possible determinants of decline

• *Socio-economic determinants* – Incomes increased from 1917 until about 1930, but after that period economic recession set in (CBS 1994). Indicators of living standard more directly related to mortality decline, e.g. housing and nutrition, showed the same pattern as income per head. The number of persons per room continued to decrease until 1930 and stabilised afterwards (CBS 1994). As far as nutrition is concerned there are only data on the availability of specific foodstuffs and not on calorie-intake. The availability of sugar, cheese, margarine and fresh subtropical fruits increased until about 1930 and stabilised thereafter (CBS 1994). In the early 1950s new improvements of the socio-economic factors were visible.

• *Socio-cultural determinants* – The percentage people with no religious affiliation strongly increased in this period. Fertility rates further declined until the mid 1930s, and increased thereafter (CBS 1994).

• *Medical care and technology* – In this period, tuberculosis control became an important issue. After the First World War, tuberculosis clinics were set up. The introduction of antibiotics, after 1945, took place in the last decade of this period. Bearing in mind that the introduction of antibiotics was at the end of the period 1917-1955, the influence of this determinant was probably of minor importance in comparison to other factors in the period 1917-1955.

As far as public health measures are concerned, no important changes occurred in this period.

Cause and age-specific mortality decline

In 1917, the most important causes of death were 'other diseases (including debility)', acute respiratory diseases, respiratory tuberculosis and heart diseases. Respiratory tuberculosis and acute respiratory diseases (predominantly pneumonia) showed the strongest acceleration of mortality decline in the period 1917-1955 compared to 1880-1917, which indicates that those causes might have played an important role in the acceleration of total mortality decline after 1917.

The decline of respiratory tuberculosis is in accordance with the fact that total mortality decline was predominantly apparent in adult ages, the improvements in socio-economic determinants at the onset of this period, and with the more intensified tuberculosis control. The introduction of antibiotics probably played a role too (Mackenbach & Looman 1988). Improvements in housing, nutrition and the introduction of antibiotics are all determinants that might have affected acute respiratory mortality decline too. The role of housing conditions in tuberculosis mortality has been clearly shown by McFarlane in his article tuberculosis mortality in Glasgow in the period 1911-51. Bad housing acted as a brake on the downward trend of tuberculosis mortality in Glasgow (McFarlane 1989).

The fact that mortality declines for infants in the period most comparable to the period 1917-1955 (i.e. 1924-1948 cf. table 5.1) were a deceleration in mortality decline, and that accelerations in mortality declines occurred predominantly in adult age groups, might indicate that the socio-cultural factors, and public health factors were of minor importance for total mortality decline in the period 1917-1955.

5.4.5 The end and a renewal of mortality decline

The end of rapid mortality decline was reached by 1955. After this year total mortality virtually stabilised until 1970. In literature this period has been marked as the end of the demographic and epidemiologic transition. Chesnais (1992), for example, defined the end of the demographic transition as a lasting return (at least five years) to an average rate of natural increase that equals or is less than

that of the period preceding the starting point. Also, life expectancy has to be at least 73 years for females. A female life expectancy of 73 years was reached in The Netherlands in 1952. The natural increase was on average 10 per 100,000 (ranging from 9.5 to 10.3) in the period 1860/70, and was at about the same level in 1970 (9.9 per 100,000). It has been below that level since 1970. According to Omran's epidemiologic transition theory, mortality eventually approaches stability in the last stage of the epidemiologic transition, which is the case in The Netherlands in the period 1955-1970 (Omran 1971, Omran 1983).

However, after 1970 a renewed decline set in, but this decline differed considerably from former periods of decline from an epidemiological point of view. Causes of death that showed an increasing trend before 1970 started to decline from 1970 onwards e.g. ischemic heart disease, some cancers and traffic accidents. These new epidemiological changes have been regarded by some researchers as a fourth stage in the epidemiological transition (Olshansky & Ault 1986, Rogers & Hackenberg 1987).

Stabilisation of mortality and renewed decline: cause-specific mortality and determinants
Mortality decline decelerated in the period 1955-1970. Mortality from heart diseases, an important cause of death in 1955, shows a stabilisation of the trend in the period 1955-1970 in comparison to the period 1917-1955 (cf. table 5.2). This stabilisation was caused by two complementary trends: an increase in ischemic heart disease and a decline in other heart diseases. We did find increasing trends in male mortality as well as in age groups 15-19, 50-64, 65-79 in periods similar to 1955-1970. The increase in male and adult mortality corresponds with the increase in ischemic heart disease.

The increase in age group 15-19 might be related to an increase in mortality from violence, which was a less important but relatively rapidly increasing cause of death in the period 1955-1970 in comparison to 1917-1955 (cf. table 5.2). Van Poppel regarded smoking as the most important determinant related to the increase in male mortality in the period 1955-70. The number of cigarettes smoked per year per person aged 10 years and older increased from 1926 onwards until 1979. He found that trends in food consumption and in composition of nutrients were not compatible with trends in mortality from ischemic heart diseases (Van Poppel 1985).

With respect to the renewed decline after 1970, the decline in ischemic heart disease mortality was important. Changes in life-style such as smoking habits, which could be classified under the heading of socio-cultural factors, and medical factors, diagnostic as well as therapeutic measures, all played a role in reducing the death rate due to ischemic heart diseases (Walker 1977, Kleinman *et al.* 1979, Crimmins 1981, Van Poppel 1985). An example of a medical factor that might have contributed to the decline of heart diseases (excluding ischemic heart

disease) is the introduction of antibiotics after the Second World War. Part of the heart diseases consisted of rheumatic fever and chronic rheumatic heart diseases that are bacterial infections in origin, and therefore could be influenced by the introduction of antibiotics (Mackenbach & Looman 1988).

Another aspect of mortality decline after 1970, that is important in the ongoing decline of female mortality, is the shift of mortality to older age groups. This shift is, among others, due to improvements in medical technology and medical care (Olshansky & Ault 1986).

5.5 Conclusion

Mortality started to decline rapidly around 1880 in The Netherlands and this lasted until 1955-1970. A new period of mortality decline started around 1970. The turning point analysis resulted in a more objective and more sharply defined onset, phases and end of the epidemiologic transition in The Netherlands compared to mere visual inspection of the trend. Turning point analysis is a statistical device to determine periods with different paces of change in long-term processes. As such it is applicable to all types of trend studies e.g. fertility trends.

We were able to point out which causes of death were likely to have influenced the changes in the pace of total mortality decline in The Netherlands. However, to link the changes in cause-of-death mortality to determinants of mortality decline turned out to be a more difficult task. A lower aggregation level than the national level as used in this study would enhance the discriminative power of many determinants. Multivariate regression analyses of e.g. regional data on mortality decline and determinants would be a good statistical method to tie social, economic and medical variables to mortality decline. This paper is part of a larger project on epidemiologic transition in The Netherlands in which results of above-mentioned regression analyses are forthcoming

Chapter 6

CAUSE-SPECIFIC MORTALITY TRENDS IN THE NETHERLANDS, 1875–1992: A FORMAL ANALYSIS OF THE EPIDEMIOLOGICAL TRANSITION

Abstract

Objective: *The objective of this study is to produce a detailed yet robust description of the epidemiologic transition in The Netherlands.*

Data and methods: *National mortality data on sex, age, cause of death and calendar year (1875-1992) were extracted from official publications. For the entire period, 27 causes of death could be distinguished, while 65 causes (nested within the 27) could be studied from 1901 onwards. Cluster analysis was used to determine groups of causes of death with similar trend curves over a period of time with respect to age and sex standardised mortality rates.*

Results: *With respect to the 27 causes, 3 important clusters were found: (1) infectious diseases which declined rapidly in the late 19th century (e.g. typhoid fever), (2) infectious diseases which showed a less precipitous decline (e.g. respiratory tuberculosis), and (3) non-infectious diseases which showed an increasing trend during most of the period 1875-1992 (e.g. cancer). The 65 causes provided more detail. 7 important clusters were found: 4 clusters consisted mainly of infectious diseases, including a new cluster that declined rapidly after the Second World War (WW2) (e.g. acute bronchitis/influenza) and a new cluster showing an increasing trend in the 1920s and 1930s before declining in the years thereafter (e.g. appendicitis). 3 clusters mainly contained non-infectious diseases, including a new cluster that declined from 1900 onwards (e.g. cancer of the stomach) and a new cluster that increased until WW2 but declined afterwards (e.g. chronic rheumatic heart disease).*

Conclusion: *The results suggest that the conventional interpretation of the epidemiologic transition, which assumes a uniform decline of infectious diseases and a uniform increase of non-infectious diseases, needs to be modified.*

6.1 Introduction

The epidemiologic transition theory, as formulated by Omran, describes a shift from infectious to degenerative and man-made diseases in populations throughout time. The age of pestilence and famine, in which mortality is high and fluctuating due to epidemics of infectious diseases, is followed by the age of receding pandemics in which infectious diseases are declining, although they are still important causes of death. In the last age, the age of degenerative and man-made diseases, cardiovascular diseases, cancer, diabetes and other metabolic disorders and diseases introduced by man (such as accidents) predominate as causes of death. The epidemiologic transition has been observed in many countries although time frames are different (Omran 1971, 1977a,b, 1980, 1983).

Omran and many other researchers have described the epidemiologic transition on the basis of broad cause-of-death categories and rather informal analyses, such as the comparison over a period of time of the rank order of causes of death or the contribution of causes of death to all-cause mortality (Broudy & May 1983, Young 1988, Levison *et al.* 1981, Schooneveldt *et al.* 1988). In this paper we will describe the epidemiologic transition in The Netherlands using a more refined cause-of-death classification and a formal statistical method. The aim of this study is to produce a detailed yet robust description of the epidemiologic transition that will form a good starting point for explanatory studies.

A more detailed analysis of causes of death is likely to be more informative for an understanding of the epidemiologic transition than studies that are based on broad cause-of-death categories. In addition, a formal statistical method (cluster analysis) is likely to produce less subjective and more robust results than those gained from informal analyses. The cluster analysis is applied to cause-specific mortality *trends* because time trends are more informative for explanations of the epidemiologic transition than, for example, changes throughout time in the contribution of causes of death to all-cause mortality. This paper presents clusters of causes of death with the same trend over a period of time, irrespective of the level of the mortality rates. In other words, clusters consist of causes of death that decline and increase together throughout time. The results of the cluster analysis will not only provide a detailed description of the pattern of cause-specific mortality trends that underlie the all-cause mortality trend, but they might also contribute to a better understanding of the explanations of the

epidemiologic transition by discussing the results with respect to common determinants of the causes of death within the clusters.

6.2 Data and Methods

6.2.1 Data

Absolute numbers of deaths with respect to sex, age and cause of death were used for the years 1901-1992, which were published by the Dutch Central Bureau for Statistics. Data for the period 1875-1900 were derived from quinquennial figures published by the Dutch Home Office.

6.2.2 Nested Classification

The causes of death had to be reclassified in order to create nosologically continuous groups of causes of death, because the period 1875-1992 covered a very concise 19th century classification as well as 9 revisions of the International Classification of Diseases and Causes of Death (ICD). Causes of death in the new classification were reclassified on the basis of the old classification. Because this results in a classification which is very similar to the concise 19th century classification, a nested classification that consists of three aggregation levels of causes of death was developed so as to keep the level of detail. The construction and validation of this nested classification has been described elsewhere (Wolleswinkel-van den Bosch *et al.* 1996). The study in this paper uses two aggregation levels of causes of death (27 and 65 causes) which could be studied over two different time periods (1875-1992 and 1901-1992 respectively). The causes of death used in this study and the most recent ICD codes have been included in appendix 3.

Most attention will focus on the 65-causes level, because infectious as well as non-infectious causes were equally represented at this level. The 27-causes level, which could be studied for the entire period 1875-1992, is particularly informative with respect to the trends of infectious diseases during the 19th century.

6.2.3 Standardisation

Standardised mortality rates for age and sex were calculated (direct standardisation) to correct for the effect of the changing age and sex distribution on mortality. Eight age groups were used in the standardisation procedure: 0, 1-4, 5-14, 15-19, 20-49, 50-64, 65-79 and 80 years and over, which was the greatest common denominator of all age subdivisions used in the period 1875-1992. The ref-

erence population was the average population in the period 1901-1992. This standard was used for the period 1875-1992 as well as for the period 1901-1992.

6.2.4 Cluster analysis

In this study we wanted to determine which causes of death declined and increased together over a period of time. This means that we were not interested in the general level of causes of death, but in the shape of their trend curves. Therefore, standardised mortality rates for age and sex were standardised to equal means over the period under study for each cause of death.

Many causes of death showed strong interruptions of their trend due to the influenza epidemic of 1918 and the Second World War. Because we were interested in the long-term developments of mortality irrespective of interruptions due to specific extraneous events like war, and because important interruptions in the trend would dominate the results of the cluster analysis the years 1918 and 1940-1946 were excluded from the analyses.

Because of the heuristic nature of cluster methods we used two different methods, each of which aims to minimise the within-cluster-sum-of-squares, i.e. to optimize according to the least squares principle. One is the well-known agglomerative hierarchic cluster method of 'Ward's analysis' (Norusis 1990), the other is a less-known divisive cluster method 'Orbaclan', based on bisecting principal component axes of subsequent clusters (Jongman *et al.* 1995). Both methods were followed by relocation procedures, thereby using the least squares criterion again. Relocation did not lead to many changes, which indicates stable solutions. Because the results of both methods were highly comparable we will only present the results of Ward's analysis. The number of clusters that were presented as the end result was determined on the basis of the agglomeration tree (Norusis 1990).

Sex differentials were studied by relocating the female data using the outcome of the male cluster analysis and vice versa. There were hardly any differences between the clusters for males and females, so the results are not presented according to sex.

6.3 Results

The results of the cluster analysis are represented in tables and figures. The y-axis in the figures represents cluster averages based on mortality rates standardised to equal means (= 1) for the whole period. Clusters consisting of only one or two causes are omitted from the figures for practical reasons.

6.3.1 27 causes, 1875-1992

A division of the 27 causes into 6 clusters provided the best description of differences and commonalities in patterns of changes over the period of time (table 6.1 and figure 6.1). The cluster which shows an increasing trend curve during the period under study consists of all the causes of death which are currently important in developed countries e.g. cancer and cardiovascular diseases (table and figure 6.1, cluster 3). Three causes of death are distinguished as separate clusters in this analysis i.e. cholera, malaria and smallpox (table 6.1, clusters 4-6). Malaria and smallpox had nearly disappeared by the end of the 19th century. The few cases that occurred in the late 19th and early 20th century resulted in enormous peaks in the shape of the trend curve of those causes. Cholera showed a similar trend as cluster 1 (typhoid fever etc.) but the epidemic of the years 1888-1892 caused an enormous peak in the trend resulting in cholera being a separate cluster. Another cluster that consist mainly of other infectious diseases also declined during the whole period under study but showed a more even decline compared to cluster 1 (table and figure 6.1, cluster 2). The cluster with, among others, typhoid fever and scarlet fever declined during the whole period under study but most sharply at the end of the 19th century (table and figure 6.1, cluster 1).

6.3.2 65 causes, 1901-1992

The analysis of 65 causes gives a more detailed insight in the results of the 27 causes of death clustering. The 65 causes are presented as 10 clusters with different trends (table and figure 6.2) of which four clusters predominantly consisted of infectious causes of death (clusters 1-4) and three clusters of non-infectious causes of death (cluster 5-7). In addition, there were three clusters with only one or two causes of death (smallpox, cholera and anthrax, poliomyelitis; clusters 8-10). Cluster 1 is more or less comparable to cluster 1 of the 27 causes clustering; the clusters 2,3 and 4 are more or less comparable to cluster 2, clusters 5 to 7 are more or less comparable to cluster 3, and clusters 8 and 9 are more or less comparable to clusters 5 and 6 of the 27 causes clustering.

Three clusters that consist of infectious causes of death (clusters 1 to 3) differ with respect to the amount of *decline* in the early 20th century. Cluster 1 (typhoid fever etc.) declined the whole 20th century, but most rapidly at the beginning of the century. Cluster 2 (whooping cough etc.) showed a more even decline during 20th century compared to cluster 1. Cluster 3 (acute bronchitis etc.) hardly declined in the early 20th century, but showed a rapid decline after WW2. Cluster 4, which also consisted of infectious diseases (appendicitis etc.), showed, on the contrary, a strong *increase* in the second and third decade of the 20th century followed by a decline after WW2.

Table 6.1 – Clustering of 27 causes of death in the period 1875-1992

Cluster 1: mainly infectious diseases: sharp decline late 19th century, further decline during 20th century
Typhoid fever, scarlet fever, measles, scurvy

Cluster 2: mainly infectious diseases: steady decline during late 19th century and 20th century
Brain diseases etc., respiratory tuberculosis, diphtheria, whooping cough, acute respiratory diseases, chronic respiratory diseases, acute digestive diseases, chronic diseases of the digestive system, diseases of the genito-urinary system, puerperal diseases (exc. puerperal fever), puerperal fever, other diseases (incl. 'debility')*, unknown and ill-defined causes of death

Cluster 3: non-infectious diseases: increase during late 19th and 20th century
Congenital malformations, cancer, cerebrovascular disease, diabetes mellitus, diseases of circulatory system (exc. cerebrovascular disease), external causes of death (exc. suicide), suicide

Cluster 4: rapid decline late 19th century
Malaria

Cluster 5: rapid decline late 19th century with high peaks in 1880 and 1890
Smallpox

Cluster 6: rapid decline late 19th century, epidemic at 1888-1892
Cholera

* This category consists of debility (mainly perinatal causes of death and old age), some types of tuberculosis, scrofula, rachitis, diseases of the skin, abscess, ulcer, gangrene, pyaemia, hemorrhage, continuous fever.

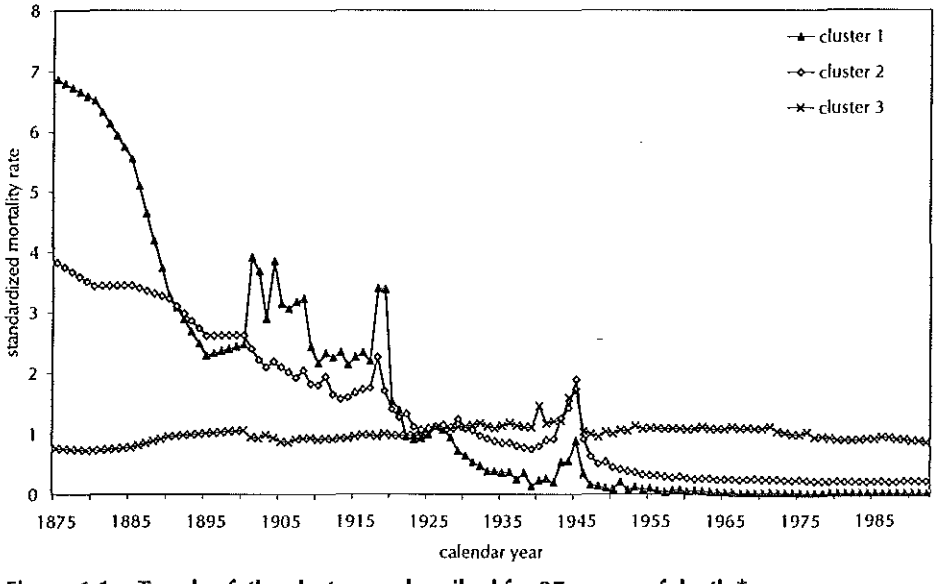


Figure 6.1 – Trends of the clusters as described for 27 causes of death.*
* The y-axis represents cluster averages of cause-specific age-standardized mortality rates standardized to equal means (=1) over the years under study. For an explanation of the content of the clusters see tables 6.1 and 6.2.

As far as the non-infectious-diseases clusters are concerned, cluster 5 (breast cancer etc.) is the only cluster that clearly shows an increase during most of the 20th century. Cluster 6 (stomach cancer etc.) declined relatively fast in the first two decades of this century and has continued to decline since. Cluster 7 (cancer of the intestines and peritoneum) differed from cluster 6 by a slight increase instead of a decline in the first half of this century.

6.3.3 Stages in the epidemiologic transition

We identified several stages in the epidemiologic transition, thereby using the results of the cluster analysis. The years 1918 and 1940-1946 are not taken into consideration, because these years were not included in the cluster analysis. A new stage was assumed to begin when an important change in the trend of one or more clusters occurred. A first stage, 1875-1900, is characterised by an enormous decline of typhoid fever, scarlet fever, scurvy and, to a lesser extent, measles (figure 6.1, cluster 1). The period 1901-1920 is defined as a second stage in which the decline of the above-mentioned causes of death became less progressive, although the decline was still considerable compared to other clusters (figure 6.1, cluster 1 and figure 6.2, cluster 1). The next stage, 1921-WW2, is characterised by an increase in certain infectious diseases e.g. appendicitis (figure 6.2, cluster 4) and a less progressive decline of a number of non-infectious diseases e.g. cancer of stomach/oesophagus/liver/gall-bladder (figure 6.2, cluster 6). There also seems to be a more rapid decline of airborne infectious diseases (figure 6.2, cluster 2). A fourth stage, WW2-1970, is characterised by the strong increase of IHD, several types of cancer and traffic accidents (figure 6.2, cluster 5), a rapid decline of the cluster consisting of, among others, acute bronchitis/influenza (figure 6.2, cluster 3) and the decline of the cluster of infectious diseases which had been on the increase (figure 6.2, cluster 4). The decline of the cluster that includes IHD, cancer and traffic accidents in 1970 marks the last stage (figure 6.2, cluster 5). This last stage corresponds with the extension of Omran's description of the epidemiologic transition with a fourth stage by several researchers (Olshansky & Ault 1986, Rogers & Hackenberg 1987).

6.4 Discussion

6.4.1 Methodological aspects

The results provide global trends and should not be interpreted as detailed information on specific causes of death. Cause-specific trends, which differed only for a small part of the total period under study, were put in the same cluster. For example, IHD and traffic accidents, which increased until 1970 but declined af-

terwards, were put in the same cluster as breast cancer and 'cancer of other organs' (including lung cancer), which continued to increase after 1970. Thus IHD and traffic accidents were not recognised as a separate cluster but were joined with the least differential groups.

Furthermore, the results of the cluster analysis are dependent on the starting point of the period under study. Scarlet fever and typhoid fever, for example, were in the same cluster in the results of the analysis for the period 1875-

Table 6.2 – Clustering of 65 causes of death in the period 1901-1992

Cluster 1: mainly infectious diseases: rapid decline early 20th century
Typhoid fever, malaria, measles, scurvy, diarrhoea/dysentery/enteritis, convulsions

Cluster 2: mainly infectious diseases: less rapid declines early 20th century
Whooping cough, diphtheria, respiratory tuberculosis, tuberculosis of the nervous system, abdominal tuberculosis, disseminated and other tuberculosis, encephalitis/meningitis, scarlet fever, peritonitis, acute nephritis, unknown and ill-defined causes of death

Cluster 3: mainly infectious diseases: most rapid decline after Second World War
Acute bronchitis/influenza, erysipelas, syphilis, rheumatic fever/chorea, old age/dementia, pneumonia, diseases of the pleural cavity, chronic nephritis/other kidney diseases, puerperal fever, bleeding and other diseases during pregnancy/childbirth /puerperium (exc. puerperal fever), septicaemia /pyaemia

Cluster 4: mainly infectious diseases: increase in the 1920s and 1930s
Appendicitis, diseases of prostate and other male genital organs, diseases of female genital organs, venereal infections (exc. syphilis), diseases of the ear (inc. otitis media)

Cluster 5: non-infectious diseases: rapid increase until 1970s
Cancer of breast, cancer of other organs, ischemic heart disease, homicide, traffic accidents

Cluster 6: mainly non-infectious diseases: decline during the 20th century
Cancer of stomach/oesophagus/liver/gall-bladder, adrenal diseases, alcoholism, diseases of larynx/pharynx /nasal cavity/oral cavity, chronic bronchitis/asthma/other respiratory diseases (COPD), diseases of oesophagus, diseases of the newly born

Cluster 7: mainly non-infectious diseases: slight increase until Second World War and a decline afterwards
Cancer of intestines and peritoneum, cancer of the skin, diseases of locomotion/ rheumatism/arthritis/rickets, diabetes mellitus, diseases of the nervous system (exc. M.Parkinson and multiple sclerosis)/diseases of the thyroid gland, stomach ulcer, other diseases of the digestive system, heart disease (exc. ischemic heart disease), cerebrovascular disease, diseases of arteries and veins, diseases bladder/urinary tract, congenital malformations, suicide, other external causes of death, other infectious diseases, other diseases

Cluster 8: highly fluctuating, declining trend early 20th century
Smallpox

Cluster 9: highly fluctuating, declining trend early 20th century
Cholera and anthrax

Cluster 10: epidemic peaks about every 10 years until 1960
Poliomyelitis

1992. In the analysis for the period 1901-1992 scarlet fever was put in a cluster which was less rapid declining compared to the cluster that consisted of typhoid fever. Both causes of death declined tremendously in the last decades of the 19th century. This similarity in trends was more important than differences in later years. However, in the analysis from 1901 onwards, this tremendous decline in the 19th century was not within the scope of the analysis anymore and differences in the shape of the remaining trend became important.

Because we were interested in long term developments of cause-specific mortality, interruptions due to specific extraneous events such as the influenza epidemic in 1918 and the years of the Second World War (1940-46), which would dominate the results of the cluster analysis, were not included in the analyses. If the years 1918 and 1940-46 are included in the analysis, influenza would be a separate cluster, due to the high peak in 1918, and diphtheria and homicide would form a cluster, because of the high peaks in mortality from those causes of death in the Second World War (results not shown in this paper).

A final remark on the cluster analysis concerns the decision how many clusters do best represent the data i.c. the differences and commonalities in cause-specific time trends. The number of clusters is determined on the basis of the increase in the value of the distance measure between clusters as given in the agglomeration tree. Agglomeration is usually stopped as soon as the increase in

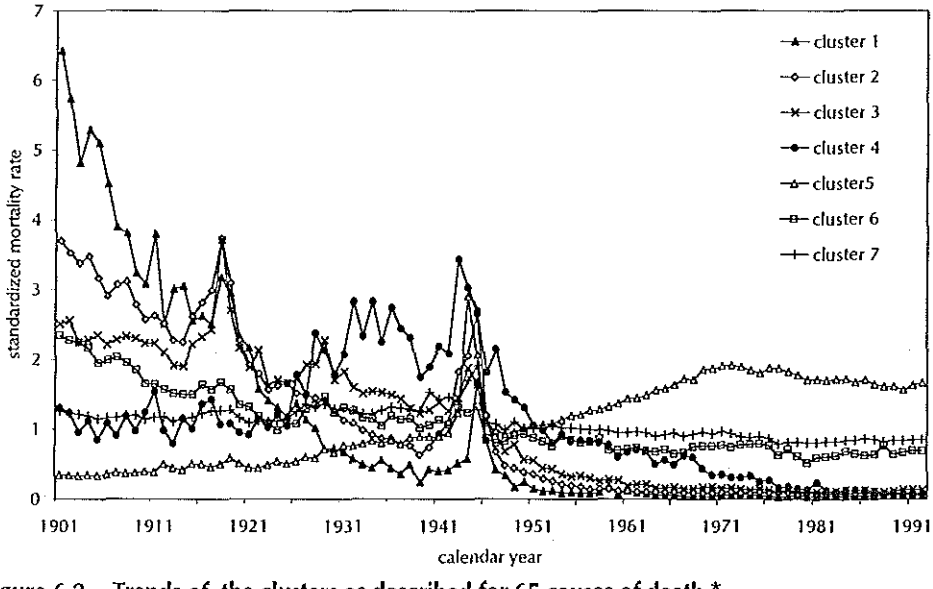


Figure 6.2 – Trends of the clusters as described for 65 causes of death.*

* The y-axis represents cluster averages of cause-specific age-standardized mortality rates standardized to equal means (=1) over the years under study. For an explanation of the content of the clusters see tables 6.1 and 6.2.

distance measure between the clusters that are merged in a next agglomeration step becomes large (Norusis 1990). In the case of 27 causes of death (cf. table 6.1), this led to a division of the cause-specific mortality trends into 6 clusters. In the agglomeration stage of 5 clusters, the causes of death in the clusters 1 and 2 were in the same cluster (cf. table 6.1). There is however a clear distinction in the trend of both clusters: cluster 1 declined sharply late 19th century contrary to cluster 2 (cf. figure 6.1). A division into 7 clusters would lead to a separate cluster of typhoid fever and scarlet fever. The trend curves of those causes of death are, however, similar to the other causes of death in cluster 1 (cf. table 6.1). In the case of 65 causes of death (cf. table 6.2), it was more difficult to determine the point in the agglomeration tree at which the increase in distance measure became large. In the agglomeration stage before the division into 10 clusters, the causes of death in clusters 6 and 7 were in the same cluster. This obscures, however, the fact that cluster 6 represents non-infectious causes of death showing a declining trend throughout the 20th century. In the agglomeration stage after the division into 10 clusters, cholera and anthrax became separate clusters. This did, however, not add to a better understanding of the differences and commonalities in cause-specific mortality trend curves.

In addition to characteristics of the cluster analysis itself there are also characteristics of the data that might have affected the results. Firstly, the age groups that were used for the calculation of standardised mortality rates were rather broad, especially with respect to older ages. This might have biased trends of causes of death at older ages. However, to affect the results of the cluster analysis, the trend should have changed considerably in order to end up in another cluster; this seems not to have happened.

Secondly, the introduction of new cause-of-death classifications, new coding habits and improvements in diagnosis might have affected specific cause-of-death trends and, consequently, the results of the cluster analysis. We have tried to minimise the effects of new classifications and coding habits by conducting an extensive reclassification procedure (Wolleswinkel-van den Bosch *et al.* 1996). However, the introduction of ICD-6 in 1950 might have influenced the inclusion of cerebrovascular accident (CVA) in cluster 7 instead of cluster 6 (cf. table 6.2). CVA mortality had been declining slowly since 1901, but an increase of CVA mortality, probably as a result of changing coding habits, took place in 1950 followed by a renewed rapid decline. We do not expect further serious influences of the introduction of new ICD-revisions on the results of the cluster analysis. It is difficult to deal with the effects of improved diagnosis. The presence of ill-defined causes of death in a declining cluster (cf. table 6.2 and figure 6.2, cluster 2) might indicate that diagnosis and notification improved in the early 20th century. The most direct way to solve this problem would have been to restrict the time frame of the study to, for instance, 1930-1992. In that case, we

would however loose a lot of information on the early period of the epidemiologic transition such as, for example, the strong decline of typhoid fever in the late 19th and early 20th century. Improvements in diagnosis could temporarily change the level and trend of mortality of certain causes of death. Significant changes in the trend curve are however needed to influence the result of the cluster analysis. The IHD trend curve, for example, probably shows a stronger increase in the 1930s than the real increase due to improvements in diagnosis by that time. However, the IHD trend curve is characterised by a strong increase until 1970 and a decrease afterwards. This overall IHD pattern will still hold even if the increase in the early 20th century might not have been as strong as shown by the data.

6.4.2 Changes in cause specific mortality in The Netherlands compared to other countries

The differences in time trend of groups of infectious and non-infectious diseases that can be observed in The Netherlands are not a unique phenomenon. Changes in cause-of-death patterns have been reported for many developed as well as developing countries (Omran 1971, Broudy & May 1983, Young 1988, Levison *et al.* 1981, Schooneveldt *et al.* 1988, Preston & Nelson 1974, Logan 1950, Vallin *et al.* 1990). The decline of certain non-infectious diseases such as stomach cancer and stroke has been reported for many developed countries (Whisnant 1983, Thom *et al.* 1985, Uemura & Pisa 1988, Campbell *et al.* 1980, LaVecchia *et al.* 1992) and the increase in the 1920s and 1930s of certain infectious diseases has been described for England & Wales (Barker 1989a).

In developing countries, CVA was the first cardiovascular disease to emerge as the clinical consequence of high blood pressure, followed by cardiac and renal complications and eventually, angina pectoris and myocardial infarction (Trowell & Burkitt 1981, 1988). The Dutch findings showed an early decline of CVA, an increase of heart disease mortality in the first two decades of this century and an ongoing increase of IHD mortality after WW2 whereas other heart diseases started to decline. An early emergence of appendicitis, the increase of cancer of the colon, breast, ovary and prostate, and an increase in smoking-related diseases as well as traffic accidents have also been reported for developing countries (Marshall 1991, Söderlund & Zwi 1995, Bofetta *et al.* 1993).

6.4.3 The results of the cluster analysis compared to Omran's description of epidemiologic transition

The results indicate that an epidemiologic description of mortality decline in terms of a 'decline of infectious diseases' and a 'rise of non-infectious diseases' is far from satisfactory.

As far as the infectious diseases are concerned four groups with a different time trend could be distinguished. The first group (table 6.1, cluster 1 and table 6.2, cluster 1) might be identified as water and food borne infectious diseases or diseases related to poor hygiene and malnutrition. Mortality from those diseases declined progressively in the late 19th and early 20th century. Typhoid fever, diarrhoea, measles, convulsions, and scurvy belong to this group. The relationship between convulsions and diarrhoea and between measles and diarrhoea has been reported previously (Kintner 1986, Han *et al.* 1990). The construction of water supply systems, which began in the late 19th century, might have contributed to this decline (Vogelzang 1956).

Whooping cough, diphtheria, tuberculosis etc. (table 6.2, cluster 2) have also been declining at least since the turn of the century, though less progressively so. This group might be labelled as airborne infections. An important determinant of the decline in this cluster might have been the improvement in nutritional status. This cluster consists of several types of tuberculosis, which is a disease that is sensitive to nutritional status (McKeown 1976a,b). Overcrowding in houses and factories has probably also been a determinant of the diseases in this cluster. In the late 19th and early 20th century several measures were taken by the national authorities to improve housing and working conditions which might have contributed to the decline of this cluster in the first half of the 20th century (Querido 1968).

The third group of infectious diseases (table 6.2, cluster 3) declined slowly at the beginning of this century. The decline accelerated after WW2. This acceleration was probably partly due to the introduction of antibiotics just after WW2 (Mackenbach & Looman 1988).

The last group of infectious diseases, i.e. appendicitis, otitis media/mastoiditis, venereal infections (except syphilis) (table 6.2, cluster 4) might be identified as temporarily increasing infectious diseases. A possible explanation is Barker's 'hygiene hypothesis'. Around 1920, improvements in hygiene had reduced enteric infections in young children which made them prone to appendicitis and some other infectious diseases e.g. poliomyelitis in adolescence. As hygiene improved further the likelihood to contract an infection at a young adult age also decreased (Barker 1985, 1988, Barker *et al.* 1989, Martyn & Barker 1988).

An increase in mortality has only occurred for part of the diseases that were put under the heading 'degenerative and man-made diseases' by Omran. Three groups could be distinguished, but it is hard to characterise those groups in terms of common aetiology and/or cause of decline. Some causes showed a rapid decline at the beginning of this century e.g. stomach cancer, COPD, alcoholism and 'diseases of the newly born' (perinatal causes of death) (table 6.2, cluster 6). Possible reasons for the decline are e.g. a reduced salt intake due to other food preservation measures (refrigerator) than salting in the case of stom-

ach cancer (Tuyns 1988) and improved obstetric care in the case of perinatal mortality (Loudon 1992).

Another group of causes on average increased slightly at the beginning of the century and declined slowly after WW2 (table 6.2, cluster 7). Heart disease other than IHD had been increasing until 1920 and started to decline in 1950. Some of those heart diseases (e.g. chronic rheumatic heart disease) were infectious in origin and could therefore be affected by changes in hygiene, nutrition and antibiotics, which might explain part of the decline (Campbell 1963, Preston & Nelson 1974).

Only one group of non-infectious diseases has increased during most of the 20th century e.g. IHD, several types of cancer, traffic accidents and homicide (table 6.2, cluster 5). Determinants of these causes e.g. high cholesterol intake, smoking habits and changes in reproductive and health behaviour (Walker 1977, Breslow & Enstrom 1980, Kleinman *et al.* 1979) might be labelled as changes in behaviour that are related to increased affluence.

6.5 Conclusion

This detailed analysis of the epidemiologic transition in The Netherlands has shown the diversity in 'the decline of infectious diseases' and 'the rise of degenerative and man-made diseases'. The results of this study suggest that the conventional interpretation of the epidemiologic transition, which assumes a uniform decline of infectious diseases and a uniform increase of non-infectious diseases, needs to be modified. A more differentiated interpretation of changes in mortality in terms of cause of death is likely to provide a better starting point for explanatory analyses

Chapter 7

THE CONTRIBUTION OF INFECTIOUS DISEASES TO MORTALITY DECLINE IN THE NETHERLANDS, 1875–1970

Abstract

Objective: *To analyse the relative contribution of infectious diseases to total mortality decline in The Netherlands in the period 1875-1970 and four subperiods viz. 1875-1901, 1901-31, 1931-50, 1950-70, and to analyse the pace of infectious disease mortality decline in the four subperiods. The contribution percentage is an indication of the importance of infectious diseases in mortality decline. Analysis of the pace of decline is needed to relate determinants of mortality decline to specific infectious diseases.*

Data and Methods: *Sex, age and cause-specific mortality data and population data were derived from publications by the Dutch Home Office and the Central Bureau of Statistics. To describe the contribution of infectious diseases to mortality decline, the decline of age- and sex-standardised cause-specific mortality was expressed as a percentage of all-cause standardised mortality decline. Linear regression analysis of the logarithm of standardised mortality over time was used to calculate the pace of mortality decline.*

Results: *Respiratory tuberculosis contributed most to mortality decline in the periods 1875-1970 (14.7%), 1875-1901 (16.2%), 1950-70 (14.9%), diarrhoea/dysentery contributed most to the decline in the period 1901-31 (20.3%), and pneumonia in the period 1931-50 (22.0%). A generalised acceleration in mortality decline (due to a decline in water-borne as well as airborne infectious diseases) could be observed in the period 1901-31. In the period 1931-50, accelerations in decline were predominantly observed among airborne infectious diseases.*

Conclusion: *Accelerations in mortality decline (in relation to the contribution of infectious diseases) in the period 1901-31 could be related to accelerations in fertility decline in the same period. In the period 1931-50, improvements in socio-economic and socio-cultural factors decelerated. Medical factors seem to be important contributors to mortality decline in that period.*

7.1 Introduction

Since the late 19th century mortality has drastically declined in The Netherlands. During the mortality decline, the cause-of-death pattern changed substantially from infectious diseases as the main causes of death to non-infectious diseases and external causes. Infectious-disease mortality declined while mortality from non-infectious diseases increased. This shift in cause-of-death patterns became known as the epidemiological transition after Omran published on this subject in the early seventies (Omran 1971). The epidemiological transition has been described for many countries although the timing of this process differed between countries (Omran 1977, Broudy & May 1983, Young 1988, Levison *et al.* 1981, Schooneveldt *et al.* 1988, Wolleswinkel-van den Bosch *et al.* 1997).

Describing mortality decline also raised questions about the determinants of mortality decline. Until the 1950s, the prevailing view was that improvements in medicine and public health measures had been the most influential factors in mortality decline. In 1962, however, Thomas McKeown published a paper in which he argued that improved living standards, and particularly improved nutrition, were the major determinants in 19th-century mortality decline (McKeown & Record 1962). About ten years later, he published a paper in which he reached the same conclusion for the twentieth century (McKeown *et al.* 1975). In these papers, McKeown analysed the contribution of causes of death to mortality decline. Airborne infectious diseases turned out to be the biggest contributors to mortality decline in England & Wales (about 65% in the second half of the 19th century and about 45% in the period 1901-71). Mortality from airborne infectious diseases, particularly respiratory tuberculosis and pneumonia, is related to the nutritional status of the infected person. According to McKeown, other possible determinants did not play an important role in the decline. Increased hygiene, for example, was related to water-borne and food-borne infectious diseases, which did not contribute as much to mortality decline as airborne infectious diseases did. Moreover, medical technology only became available after infectious diseases had already significantly declined (McKeown & Record 1962, McKeown *et al.* 1975).

Using cause-specific data, like McKeown did, is a good starting point to gain insight into possible determinants of mortality decline. Cause- (and age-) specific mortality can be more accurately linked to determinants than to total

mortality. The contribution percentage to mortality decline of a cause of death alone cannot be directly linked to determinants of decline. The contribution to mortality decline is not only determined by the trend of the cause of death or age group itself, but also depends on trends shown by other causes of death. Information on the trends of causes of death can show whether a cause of death that greatly contributed to mortality decline also showed a strong declining trend. Furthermore, trends can be more easily linked to determinants of decline. Accelerations in a trend, for example, suggest the influence of determinants on that cause-specific or age-specific mortality trend. In summary, the percentage of contribution to mortality decline quantifies the importance of a cause of death or age group in mortality decline. Information on cause-specific and age-specific trends is needed to arrive at more well founded conclusions on the role of specific determinants in mortality decline.

The aim of this paper is to analyse the *contribution* of infectious-disease mortality (causes and age groups) to total mortality decline, and to analyse the *pace* of infectious-disease mortality decline in The Netherlands in the period 1875-1970 and in four subperiods viz. 1875-1901, 1901-30, 1930-50 and 1950-70. The paper focuses on the period 1875-1970, because in this period mortality decline is characterised by declines in infectious diseases. From 1970 onwards, declines in ischemic heart disease have determined mortality decline (Wolleswinkel-van den Bosch *et al.* 1997). A detailed cause-of-death classification is used in order to enhance the possibilities to relate causes of death to determinants of mortality decline.

Results are presented for four subperiods, so that we were able to identify causes of death and age groups that were predominantly important in a specific period of time instead of considering the whole period under study. In the discussion section of this chapter, we will try to relate changes in the pace of cause-specific and age-specific mortality decline to trends in determinants of decline.

7.2 Data and Methods

7.2.1 Data

Cause-specific mortality data by sex and age were derived from publications by the Dutch Home Office for the period 1875-1900 and from the Central Bureau of Statistics in The Netherlands (CBS) for the period 1901-1970. Population data were also supplied by the CBS for the whole period from 1875 to 1970.

7.2.2 Reclassification of causes of death

The cause-of-death categories used in this paper are the result of an extensive reclassification procedure of the cause-of-death categories in a Dutch 19th-century classification and 9 revisions of the International Classification of Causes of Death. The reclassification was based on the description of the categories in the ICD-revisions and, for the 19th-century classification, on medical literature published at the time. The result of the reclassification procedure was a nested classification of causes of death in which 27 causes of death could be studied during the period from 1875 to 1992, 65 causes of death could be studied from 1901 to 1992 and 92 causes of death could be studied for the period from 1931 to 1992. The infectious diseases from the above-mentioned 27 and 65 causes are used in this study. The 27 causes-of-death categories reflect the 19th-century classification. This classification includes some vaguely defined cause-of-death categories and categories that cover both infectious and non-infectious diseases. Therefore, results of a more detailed classification (65 causes of death) will be presented too. Appendix 3 contains the cause-of-death labels for the 27 and 65 causes of death as well as the ICD-9 codes for the specific categories. The categories considered to be infectious diseases (in this study) have been marked with an asterisk. A more elaborate description of the reclassification procedure has been published elsewhere (Wolleswinkel-van den Bosch *et al.* 1996, chapter 4).

7.2.3 The contribution of infectious diseases to mortality decline

The contribution of infectious diseases to mortality decline was calculated for the entire period under study (1875-1970), as well as for the four subperiods viz. 1875-1901, 1901-1931, 1931-1950 and 1950-1970. Cause-specific age- and sex-standardised mortality rates were calculated on the basis of the direct method using the age- and sex-distribution of the aggregate population for the period 1901-1992 as the standard. Eight age categories were used viz. 0 years of age, 1-4, 5-14, 15-19, 20-49, 50-64, 65-79 and 80 years and over, which was the greatest common denominator of all age-subdivisions in the period 1875-1992. Standardised cause-specific mortality decline was expressed as a percentage of all-cause standardised mortality decline so as to describe the contribution of specific causes of death to all-cause mortality decline.

7.2.4 Mortality trends of infectious diseases

The pace of mortality decline was calculated for infectious-diseases and age-specific infectious-disease mortality for the four subperiods. A linear regression analysis was performed using the logarithm of the standardised mortality rates as the dependent variable and time as the independent variable.

7.3 Results

7.3.1 The contribution of infectious diseases to mortality decline

Table 7.1 shows the contribution of specific infectious diseases to mortality decline in the period 1875-1970 and the four subperiods. The contribution to the decline in the period 1875-1970 could only be calculated on the 27-causes level. The total (infectious and non-infectious) mortality decline in the period from 1875 to 1970 was 1640.5 deaths per 100,000 person years. The most important infectious diseases that contributed to the decline in this period were respiratory tuberculosis (14.7%), acute respiratory diseases (11.0%), brain diseases etc. (mainly encephalitis/meningitis and convulsions) (11.3%) and acute digestive diseases (mainly diarrhoea and dysentery) (8.3%).

Brain diseases etc. (14.0%) and respiratory tuberculosis (16.2%) played an important role in the period 1875-1901. Three infectious diseases contributed negatively to the decline viz. acute respiratory diseases (-6.9%), measles (-3.4%) and acute digestive diseases (-2.2%), which means that mortality from those causes of death increased in the years 1875-1901.

For the period 1901-31, more detailed information on causes of death is available. The most important findings for this period are the large contribution to mortality decline of acute digestive diseases, particularly diarrhoeal diseases/dysentery (20.3%). Other substantially contributing infectious diseases are brain diseases etc. (11.1%) (including encephalitis/meningitis, 4.4%, and convulsions, 4.6%), respiratory tuberculosis (15.0%), and acute respiratory diseases (13.3%) (including pneumonia, 13.9%).

In the period 1931-50, acute respiratory diseases contributed the most to total mortality decline (37.0%). Influenza/acute bronchitis was responsible for 13.8% and pneumonia for 22.6% of the decline. The contribution of respiratory tuberculosis was also considerable (11.3%).

The absolute decline in total mortality was small in the period 1950-70 due to several opposite cause-of-death trends such as the decline of heart diseases (excluding ischemic heart disease) and the increase of ischemic heart disease. The non-infectious diseases contributed most to the decline in this period, but the contribution of infectious diseases was still considerable viz. 44.8%. Respiratory tuberculosis (14.9%) and pneumonia (8.5%) were the most important contributing infectious diseases.

Table 7.1 – Contribution (in percentages) of infectious diseases to total age- and sex-standardised mortality decline in the period 1875-1970 and in 4 sub-periods. A blank means that figures for that period are not available.

	1875-1970	1875-1901	1901-1931	1931-1950	1950-1970
Water- and food-borne infectious diseases	**10.28**	**1.53**	**23.01**	**4.20**	**4.83**
Cholera	0.15	0.15	0.26	0.00	0.00
Typhoid fever	1.85	3.62	1.33	0.16	0.19
Acute digestive diseases	8.28	-2.24	20.31	3.14	4.09
Diarrhoea/dysentery			*19.33*	*2.01*	*1.60*
Appendicitis			*-0.37*	*0.60*	*2.18*
Peritonitis			*1.35*	*0.53*	*0.31*
Abdominal tuberculosis			1.11	0.90	0.55
Airborne infectious diseases	**29.86**	**9.21**	**40.62**	**53.13**	**33.58**
Respiratory tuberculosis	14.65	16.20	15.01	11.31	14.90
Disseminated tuberculosis			2.52	1.58	3.15
Acute respiratory diseases	10.99	-6.93	13.27	37.04	10.33
Influenza/acute bronchitis			*-1.17*	*13.79*	*1.39*
Pneumonia			*13.94*	*22.57*	*8.48*
Diseases pleural cavity			*0.50*	*0.68*	*0.46*
Diphtheria/croup	1.34	1.44	1.63	0.53	1.78
Whooping cough	1.38	1.13	1.88	0.90	1.29
Scarlet fever	0.39	0.81	0.16	0.16	0.08
Measles	1.11	-3.44	5.39	1.16	0.45
Rheumatic fever			-0.02	0.27	0.75
Acute nephritis			0.78	0.18	0.85
Smallpox	0.10	0.25	0.02	0.00	0.00
Other infectious diseases (mixed etiology)	**13.04**	**18.42**	**11.47**	**8.68**	**8.08**
Brain diseases etc.	11.26	14.04	10.40	6.67	6.78
Tuberculosis of nervous system			*1.44*	*1.81*	*1.54*
Syphilis			*0.10*	*1.12*	*2.73*
Encephalitis/meningitis			*4.42*	*1.34*	*1.87*
Convulsions			*4.60*	*1.95*	*0.00*
Diseases of the ear			*-0.16*	*0.30*	*0.55*
Poliomyelitis			*-0.00*	*0.15*	*0.09*
Puerperal fever	0.46	0.82	0.10	0.57	0.23
Malaria	1.22	3.31	0.13	-0.01	0.09
Erysipelas			0.13	0.53	0.54
Septicaemia/Pyemia			0.43	0.77	0.09
Anthrax			0.04	0.00	0.00
Other infectious diseases			0.22	0.15	0.35
All infectious disease Categories	**53.18**	**29.16**	**75.10**	**66.01**	**46.49**
Total absolute decline	1640.53	578.84	625.65	343.79	92.21
(per 100000py)	100%	100%	100%	100%	100%

7.3.2 Changes in infectious-disease mortality trends

Table 7.2 and 7.3 show the annual percentage decline for infectious-disease categories and age groups (infectious diseases only) in the four subperiods. In the period 1875-1901, the most rapid declines were found for malaria and smallpox. Smallpox mortality was already low by that time, which is reflected by the wide confidence interval. Scarlet fever also showed a relatively rapid decline. With respect to the infectious diseases that contributed most to mortality decline in the period 1875-1901 viz. diseases of the nervous system and respiratory tuberculosis, the pace of decline was 2.0% and 1.5% respectively.

Cholera (9.1% decline annually), diarrhoea/dysentery (7.5%), malaria (7.5%) and typhoid fever (7.3%) declined most rapidly in the period 1901-31. Diarrhoea/dysentery was included in the category acute digestive diseases, which greatly contributed to mortality decline in this period. In the period before, mortality from this cause of death showed no declines. Declines for other causes of death that contributed considerably to mortality decline were less rapid (e.g. respiratory tuberculosis 3.1%, acute respiratory diseases 1.7%), but still represent accelerations in the decline compared to the previous period. Around the turn of the century there was a general acceleration of mortality decline among infectious disease groups with different aetiology (water-borne and food-borne as well as airborne) (table 7.2).

In the period 1931-50, rapid declines for many infectious diseases were found. The ones that declined most rapidly were measles (10.4% annually), puerperal fever (9.9%) and scarlet fever (9.5%). Other, relatively rapidly, declining (7-8%) infectious diseases were convulsions, abdominal tuberculosis and influenza/acute bronchitis. The latter cause of death is part of the category 'acute respiratory diseases', which greatly contributed to the decline in this period. Mortality from acute respiratory diseases accelerated in this period compared to the previous period. Contrary to the period before, acceleration of mortality decline was less general; accelerations were predominantly observed among airborne infectious diseases (table 7.2).

The rapid declines (15-20%) for different types of tuberculosis, viz. tuberculosis of the nervous system, respiratory tuberculosis, disseminated tuberculosis and abdominal tuberculosis, dominate the results for the period from 1950 to 1970. Respiratory tuberculosis greatly contributed to total mortality decline in this period, and mortality declines strongly accelerated in comparison to the preceding period. Other rapid declines were found for e.g. rheumatic fever and erysipelas.

The results for the age-specific trends of infectious-disease mortality show a shift over time with respect to the age groups with the most rapid mortality declines (table 7.3). In the last decades of the nineteenth century mortality decline was most rapid for child infectious disease mortality (ages 1-4 and 5-14)

Table 7.2 – Average annual change (percentages and CI) of cause-specific infectious disease mortality. A blank means that figures for that period are not available.

	1875-1901		1901-1931		1931-1950		1950-1970	
	%	CI	%	CI	%	CI	%	CI
Water- and food-borne infectious diseases								
Cholera	-2.5	[-6.5, 1.4]	-9.1	[-11.4, -5.1]		eliminated		
Typhoid fever	-4.1	[-5.2, -3.0]	-7.3	[-8.8, -5.9]	-1.8	[7.6, 4.0]		eliminated
Acute digestive diseases	0.1	[-0.4, 0.5]	-5.9	[-6.5, -5.1	-0.3	[-3.5, 2.9]	-1.3	[-1.6, -0.9]
Diarrhoea/dysentery			-7.5	[-8.4, -6.6]	0.9	[-3.4, 5.1]	-0.8	[-1.3, -0.3]
Appendicitis			1.9	[1.4, 2.5]	-3.5	[-4.1, -2.9]	-4.6	[-5.6, -3.6]
Peritonitis			-4.8	[5.6, -4.0]	-5.7	[-8.1, -3.3]	-3.8	[-5.1, -2.4]
Abdominal tuberculosis			-4.0	[-4.9, -3.2]	-6.9	[-9.7, -4.1]	-21.3	[-25.1, -17.4]
Airborne infectious diseases								
Respiratory tuberculosis	-1.5	[-2.0, -1.0]	-3.1	[-3.7, -2.5]	-3.4	[-5.9, -0.9]	-15.0	[-15.5, -13.8]
Disseminated tuberculosis			-2.5	[-3.5, -1.6]	-1.7	[-4.2, 0.7]	-11.2	[-13.7, -8.6]
Acute respiratory diseases	1.0	[-0.1, 2.1]	-1.7	[-2.7, -0.6]	-5.6	[-7.5, -3.6]	-4.7	[6.7, -2.8]
Influenza/acute bronchitis			-1.1	[-3.5 1.3]	-6.9	[-11.1, -2.8]	-6.7	[-10.7, -2.6]
Pneumonia			-2.0	[-2.7, -1.3]	-5.5	[-7.1, -3.8]	-3.9	[-5.3, -11.5]
Diseases pleural cavity			-2.2	[-2.7, -1.7]	-3.6	[7.2, -0.0]	-7.1	[-9.7, -4.5]
Diphtheria/croup	-3.2	[-5.8, -0.8]	-3.9	[-5.2, -2.7]	8.1	[-1.3, 17.6]		eliminated
Whooping cough	-1.5	[-2.1, -0.8]	-3.3	[-4.1, -2.5]	-4.9	[-8.1, -1.9]		eliminated
Scarlet fever	-6.3	[-9.7, -2.9]	-3.5	[-5.9, -1.1]	-9.5	[-14.3, -4.6]		eliminated
Measles	0.5	[-3.0, 3.9]	-5.8	[-7.2, -4.5]	-10.4	[-15.2, -5.5]	-9.4	[-13.4, -5.4]
Rheumatic fever			1.0	[0.2, 1.7]	-0.3	[-3.3, 2.6]	-13.8	[-16.7, -10.9]
Acute nephritis			-3.5	[-4.2, -2.7]	-1.2	[-3.4, 1.1]	-8.7	[-10.8, -6.9]
Smallpox	-11.5	[-19.3, -3.6]		eliminated				

Other infectious diseases(mixed etiology)								
Brain diseases etc.	-2.0	[-2.2, -1.8]	-2.7	[-3.0, -2.4]	-2.0	[-3.2, -0.8]	-1.8	[-2.3, -1.3]
Tuberculosis of the nervous system			-2.1	[-2.8, -1.5]	-6.2	[-8.7, -3.7]	-21.1	[-24.8, -17.3]
Syphilis			-0.4	[-0.8, 0.0]	-2.8	[-3.9, -1.7]	-8.2	[-9.0, -7.5]
Encephalitis/meningitis			-4.4	[-4.9, -3.9]	-1.3	[-3.7, -0.4]	-3.1	[-3.6, -2.6]
Convulsions			-5.4	[-6.1, -4.8]	-7.9	[-9.8, -5.9]		
Diseases of the ear			2.9	[1.7, 4.0]	-2.3	[-5.0, 0.5]	-6.6	[8.4, -4.6]
Poliomyelitis			4.4	[-8.3, 17.2]	-2.4	[-11.1 6.3]	-15.6	[-20.6, -10.6]
Puerperal fever	-2.1	[-5.8, 1.6]	-0.3	[-0.9, 0.4]	-9.9	[-13.0, -6.8]		eliminated
Malaria	-12.4	[-13.5, -11.4]	-7.5	[-8.9, -6.1]	1.8	[-3.6, 7.1]		eliminated
Erysipelas			-1.1	[-2.2, 0.1]	-5.5	[-8.9, -2.1]	-10.5	[-13.6, -7.8]
Septicaemia/Pyemia			-0.6	[-1.3, 0.2]	-4.9	[-7.8, -2.1	-3.0	[5.1, -0.8]
Anthrax			-9.1	[-11.4, -6.8]		eliminated		eliminated
Other infectious diseases			-1.1	[-1.9, -0.2]	0.2	[-2.4, 2.7]	-2.7	[-3.5, -1.8]
All infectious disease categories	-0.7	[-1.2, -0.3]	-2.9	[-3.6, -2.3]	-3.3	[-5.4, 1.2]	-3.8	[-4.9, -2.9]
Total mortality(all causes of death)	-0.9	[-1.2, -0.6]	-1.4	[-1.7, -1.1]	-0.9	[-2.1, 0.3]	-0.6	[0.8, -0.4]

Table 7.3 – Average annual change (percentages and CI) of age-specific infectious disease mortality.

	1875-1901		1901-1931		1931-1950		1950-1970	
	%	CI	%	CI	%	CI	%	CI
0 years of age	-0.5	[-0.5, -0.4]	-5.3	[-5.8, -4.7]	-5.6	[-7.5, -3.7]	-7.1	[-9.0, -6.1]
1-4	-2.0	[-2.4, -1.6]	-4.4	[-5.3, -3.5]	-7.5	[-8.1, -6.8]	-7.5	[-8.3, -6.7]
5-14	-2.3	[-2.6, -1.9]	-3.0	[-3.9, -2.1]	-7.2	[-8.4, -6.1]	-6.3	[-7.3, -5.2]
15-19	-1.3	[-1.4, -1.2]	-3.1	[-4.2, -2.0]	-8.1	[-9.3, -6.9]	-5.3	[-7.1, -3.5]
20-49	-1.6	[-1.8, -1.4]	-2.7	[-3.7, -1.7]	-6.1	[-6.9, -5.4]	-6.7	[-7.6, -6.0]
50-64	-0.6	[-0.9, -0.3]	-2.6	[-3.2, -2.0]	-4.2	[-5.0, -3.4]	-4.5	[-5.2, -3.8]
65-79	1.8	[1.3, 2.4]	-1.2	[-1.8, -0.6]	-3.7	[-4.9, -2.4]	-3.2	[4.2, -2.2]
80+	3.9	[3.2, 4.5]	0.1	[-0.6, 0.9]	-3.3	[-5.0, -1.7]	-1.8	[-3.2, -0.6]
Total mortality (all causes of death)	-0.9	[-1.2, -0.6]	-1.4	[-1.7, -1.1]	-0.9	[-2.1, 0.3]	-0.6	[-0.8, -0.4]

Infant infectious disease mortality declined most rapidly in the first decades of the twentieth century. In the periods 1931-50 and 1950-70 rapid declines were also found for adults. Increasing infectious-disease mortality trends were found for the oldest age groups in the period 1875-1901.

Accelerations in infectious-disease mortality decline were found for all age groups in the period 1901-31 in comparison to the period 1875-1901. The same holds for the period 1931-50 in comparison to 1901-31, except for infant mortality. However, infant mortality decline due to infectious diseases showed an acceleration in the period 1950-70 in comparison to 1931-50, whereas this acceleration was not found for the other age groups (table 7.3).

7.4 Discussion

Respiratory tuberculosis (14.7%), diseases of the nervous system (including meningitis, convulsions) (11.3%), acute respiratory diseases (11.0%), and acute digestive diseases (8.3%) were the infectious diseases that contributed most to mortality decline in the period 1875-1970. Large contributions to mortality decline were found for acute digestive diseases (20.3%) in 1901-31 and acute respiratory diseases (37.0%) in the period 1931-50. Changes in the pace of infectious-disease mortality decline were also studied. Respiratory tuberculosis mortality, for example, declined in 1875-1901, and the decline accelerated in 1901-31; acute digestive diseases mortality showed no decline in the period 1875-1901, but mortality declined rapidly in the period 1901-31. In this section, we will try to relate the changes in cause- and age-specific infectious-disease trends to trends in determinants of mortality decline, but first we will discuss the quality of the data and make an international comparison of the results.

7.4.1 The quality of cause-specific mortality data

Dutch cause-specific mortality data are of a high quality compared to data of other countries, because causes of death have been medically registered in The Netherlands since 1865 i.e. only medical doctors were allowed to certify deaths. In many other European countries in the 19th and early 20th centuries, deaths were certified by clergymen or laymen (Van Poppel 1997, Rogers 1993, Kintner 1993).

Despite the good standard of the Dutch cause-of-death classification system, there were some reports about inaccuracies with respect to the certification of chronic or acute respiratory or digestive diseases, and with respect to possible exchanges between 'respiratory tuberculosis' and 'chronic respiratory diseases' (Evers 1882, Saltet 1909, Vollenhoven 1889). A vaguely defined cause of death which had a huge contribution to late 19th-century mortality decline (55%) was

'other diseases (including debility)' (results not shown). This cause of death was not considered an infectious disease (see appendix 3). On the basis of the reclassification of 19th-century categories to ICD-1 categories (Wolleswinkel-van den Bosch *et al.* 1996), we could argue that about 30% of the cases of 'other diseases (including debility)' died from 'diseases of the newly born' (predominantly congenital debility, icterus and scleroma of neonates, atelectasis of newly born), about 50% of the cases died from 'old age' (senility, dementia), about 10% was due to defined infectious diseases (some types of tuberculosis) and 10% of the cases was due to other diseases. In other words, at least 10% of deaths due to 'other diseases (including debility)' might be classified as an infectious disease. Assuming that all causes of death within the category 'other diseases (including debility)' declined at the same pace as the category as a whole, about 5 to 6 percent could be added to the contribution of 'all infectious disease categories' to mortality decline in the period 1875 to 1901; about 4 percent could be added to the contribution of this category for the period 1875-1970 (table 7.1).

However, possible exchanges have been reported between the categories 'other diseases (including debility)' and 'acute digestive diseases' or other childhood infectious diseases (Onnen 1895) in the 19th century. Considering the vagueness of the category 'other diseases (including debility)' (and some other categories) it is not possible to determine the exact contribution of all infectious diseases to mortality decline in the period 1875-1901 or 1875-1970. The figures in table 7.1 with respect to the contribution of infectious diseases to mortality decline should be perceived as a lower limit of the contribution of infectious diseases to mortality decline.

The causes of death used in this study are the result of an extensive reclassification procedure of all cause-of-death classifications that have been used in the period 1875-1970. In this procedure we determined, among other things, whether the gap between mortality rates in the last year of an old classification and the first year of a new classification was larger than expected on the basis of the trend in preceding years (Wolleswinkel-van den Bosch *et al.* 1996, chapter 4). In other words, we determined whether there were any large and rapid artificial (e.g. changed coding practices) declines or increases during the transition from one classification to the next. The transition from the brief nineteenth-century classification (1875-1900) to the first ICD-classification (1901-1910) showed relatively large 'gaps' in the trend for about one-third of the causes of death (not all of these are infectious diseases).

With respect to this study, important causes of death that showed a large gap were respiratory tuberculosis, acute digestive diseases and measles. Acute digestive diseases and measles showed a negative contribution to mortality decline in the period 1875-1901 (cf. table 7.1). This increase might be partially artificial. The results in table 7.2 already showed that the increasing trends for those

causes of death were not statistically significantly different from no increase at all.

With respect to respiratory tuberculosis, we know that in 1901, cases in which both respiratory tuberculosis and tuberculosis of another organ were present, were coded as 'tuberculosis of more than one organ' instead of respiratory tuberculosis (which was the coding rule) (Saltet 1917). If we assume

Table 7.4 – Comparison of McKeown's results for England & Wales for the contribution of causes of death in the period 1901-1971 with the results for The Netherlands in the period 1901-1970. The cause-of-death categories are McKeown's (McKeown et al. 1975).

	England & Wales 1901-1971	The Netherlands 1901-1970	
Airborne infectious diseases	45.3	55.0	
Bronchitis/pneumonia/influenza*	18.5	26.3	(20.8)
Respiratory tuberculosis	10.8	13.8	
Scarlet fever	1.2	0.2	
Rheumatic fever/rheumatic heart disease†	3.4	2.8	
Nephritis*	3.0	5.1	(0.59)
Whooping cough	2.7	1.5	
Measles	2.4	3.6	
Diphtheria	2.4	1.3	
Smallpox	0.1	0.0	
Infections ear/larynx/pharynx	0.8	0.4	
Water- and food-borne infectious diseases	16.4	17.9	
Diarrhoea, dysentery	10.4	12.2	
Tuberculosis (non-respiratory)	4.7	4.8	
Typhoid fever	1.3	0.9	
Other infectious diseases	11.7	10.0	
Convulsions/teething	5.6	3.3	
Syphilis	1.4	0.7	
Appendicitis/peritonitis	0.7	1.2	
Puerperal fever	0.5	0.3	
Other‡	3.5	4.5	
Non-infectious diseases	26.6	17.1	
Total mortality decline	1157.4	1061.7	

* McKeown included chronic bronchitis and chronic nephritis in these categories. The figures for The Netherlands also include chronic bronchitis and nephritis although in the rest of the paper those categories are not included in the category of infectious diseases. The figures between brackets give the contribution to the decline excluding chronic diseases.

† The figure for The Netherlands is composed of 'rheumatic fever' and 'heart disease (except ischemic heart disease)'. Rheumatic heart diseases could not be separated from the 'heart disease' group.

‡ The figure for The Netherlands consists of all other infectious disease groups that could be distinguished in the 65 cause-of-death categories for the period 1901-1992.

that in 1901 all cases of 'tuberculosis of more than one organ' were in fact cases of respiratory tuberculosis, and that such cases were correctly coded as respiratory tuberculosis in the period 1875-1900, the contribution of respiratory tuberculosis to mortality decline in the period 1875-1901 would have been 11% instead of 16% (cf. table 7.1).

7.4.2 International comparison of the results

We were able to compare the results for The Netherlands with McKeown's study on England and Wales in the period 1901-1971 (table 7.4) (McKeown *et al.* 1975). The contribution of specific causes of death to mortality decline in that period is very similar in both countries. McKeown's results for the second half of the nineteenth century (1851-60 to 1891-1900) showed a contribution of 44% of respiratory tuberculosis, and 22% contribution of typhoid and typhus (McKeown & Record 1962). Especially the contribution of tuberculosis is considered to be exceptional in comparison to other countries (Preston & Nelson 1974, Mercer 1990). These results could not be compared to a similar period for The Netherlands.

A study in the city of Philadelphia for the period 1870-1900 showed contributions to total mortality decline of 26.8% for respiratory tuberculosis, -21.4% for influenza/pneumonia and 19.8% for diarrhoeal diseases (Condran & Cheney 1982). Respiratory tuberculosis and diarrhoeal diseases mortality declines are both considerably higher than the figures for The Netherlands in the period 1875-1900 (cf. table 7.1). This may be due to the higher death rates of tuberculosis and diarrhoeal diseases in urban areas (i.e. Philadelphia) as compared to rural areas, or due to differences in nutrition, quality of drinking water or coding differences. In The Netherlands we also found a negative contribution of influenza/pneumonia to mortality in the late nineteenth century although this contribution was less drastic.

A comparison of this study's results with other studies might be hampered by differences in the periods under study, differences in the content of the cause-of-death categories or by differences in the proportion of ill-defined causes of death.

7.4.3 Possible determinants of mortality decline and their relationship to infectious-disease mortality

There have been many studies on the determinants of mortality decline, and many determinants of mortality decline have been hypothesised. A study of Frenk et al. gives a good overview of all factors involved in mortality decline (Frenk *et al.* 1991). We roughly divide those determinants into socio-economic

(e.g. income), socio-cultural (e.g. breastfeeding practices), public health (e.g. water supply system) and medical factors (e.g. antibiotics).

Socio-economic factors, such as income, can affect mortality in various ways. Examples of socio-economic factors are nutrition, housing and education opportunities, the construction of water supply and sewage systems, and access to medical care (Preston 1975, Blane 1990, Flegg 1982, Rodgers 1979). Nutrition and crowded housing conditions are two socio-economic determinants that are more directly related to mortality rates than income. Causes of death mentioned in the literature that are particularly related to nutritional status are respiratory tuberculosis and pneumonia. Improved nutritional status will generally improve resistance to infections and thus also affect mortality due to other infectious diseases. Causes of death related to overcrowding can be all kinds of airborne diseases e.g. diphtheria, whooping cough, acute respiratory diseases, measles. Meningitis might also be related to overcrowding due to bad hygiene in crowded housing conditions.

Socio-cultural factors might have played a role in mortality decline too. The acceptance in a society of new ideas on, for example, health behaviour, would be partly determined by the 'openness' of that society (Hofstee 1981, Ewbank & Preston 1990). Secularisation and education can be indicators of this 'openness'. Especially new ideas on infant hygiene and childcare, e.g. breastfeeding, will influence mortality. Examples of infectious hygiene-related diseases are acute digestive diseases (including diarrhoea/dysentery), convulsions, meningitis and typhoid fever. Mortality among children under 5 is most likely to be affected by socio-cultural determinants. A more direct determinant of mortality decline that is at least partially culturally determined, is fertility. The positive association between fertility and infant mortality has been described in the literature (Forste 1994, Coale & Watkins 1986).

The construction of water supply systems and sewage systems is an important *public health factor*. Causes of death that are related to these public health factors are water-borne diseases such as typhoid, cholera and acute digestive diseases (diarrhoea/dysentery). In the second half of the nineteenth century the first water supply systems were constructed in The Netherlands (Vogelzang 1956, Groote 1995). Several public health acts viz. the 'Factory Safety Act' and the 'Building Act' were introduced in The Netherlands around the turn of the century to regulate working and housing conditions (Querido 1968). Causes of death that are related to working conditions are e.g. respiratory tuberculosis and acute respiratory diseases.

As far as *medical factors* are concerned, early examples of the role of medicine in mortality decline are the prevention of smallpox cases through vaccination (late 18th century) (Mercer 1985, Rutten 1997). and the influence on the case-fatality of a diphtheria infection through anti-diphtheria serum (late 19th

century) (Saltet 1909). Important medical factors were the introductions of sulphonamides in the 1930s and antibiotics in the late 1940s. Causes of death that are likely to be affected by antibiotics are puerperal fever, acute respiratory diseases (influenza, pneumonia), scarlet fever, dysentery, rheumatic fever, erysipelas, otitis media, syphilis, respiratory tuberculosis and rheumatic heart disease (Mackenbach & Looman 1988). In addition, surgical (appendicitis) and diagnostic improvements (tuberculosis) were introduced around 1930 and in 1949 respectively (Mackenbach 1996). Vaccination of young children against diphtheria, whooping cough, poliomyelitis and tetanus started in The Netherlands in 1952 (Burgmeijer & Bolscher 1995).

7.4.4 Relating changes in determinants to changes in the pace of infectious-disease mortality decline

So far, we have shown which infectious diseases contributed most to mortality decline, and which accelerations in trends of infectious diseases and age groups were observed for specific periods. We also discussed possible determinants of mortality decline and the infectious diseases and age groups that were most likely to be affected by those determinants.

In this section, we will try to relate mortality declines to changes in specific determinants of decline. Table 7.5 presents percentages of annual change for several possible determinants of infectious-disease mortality decline for which data were nationally available for periods comparable to 1875-1901, 1901-1931, 1931-1950 and 1950-1970 (Groote 1995, Van der Spek 1870, CBS 1895, CBS 1920, CBS 1994, CBS 1900-1917, Mandemakers 1996, Van der Velden 1996a, b). Unfortunately, historical data on determinants of mortality decline are scarce in The Netherlands. Data were not available for all determinants for the periods under study, and sometimes the percentage of decline could only be based on the first and last years of a specific period (cf. table 7.5). It should be noted that relative changes in determinants are related to relative mortality changes, and that no time lags are considered in the effect of the determinants on mortality decline.

As far as *socio-economic determinants* are concerned, data were available on real wages, crowding and nutrition (i.e. consumption of several foodstuffs). Real wages increased in The Netherlands in the period 1875-1901. This trend continued in the period 1901-31, followed by a stagnation of real wages in the period 1931-50 and a renewed increase thereafter. None of the infectious diseases follow a comparable course of mortality decline over the whole period from 1875 to 1970. Infectious diseases that show a continuous mortality decline in the periods 1875-1901 and 1901-31 are diphtheria and 'brain diseases etc.' (table 7.2).

Data on persons per room were only available from 1900 onwards. Infectious diseases that are related to crowding, i.e. encephalitis/meningitis and diphtheria, showed rapid declines in 1901-31 and a deceleration of the decline in 1931-50 (tables 7.2 and 7.5). The strong increase in diphtheria is most likely an effect of the Second World War.

The trends of the consumption of several foodstuffs give a general picture of increasing consumption in the late 19th century and a decline or less rapid increase after the turn of the century. However, for most infectious diseases, particularly for those related to nutritional status (e.g. respiratory tuberculosis, acute

Table 7.5 – Percentage annual change of determinants of mortality decline for periods comparable to 1875-1901, 1901-31, 1931-50 and 1950-70 if data were available. Percentage annual change was calculated using regression analysis on the logarithm for fertility, all food products, female education, medical doctors, water supply system, and was calculated manually for real wage, crowding and secularization, because in the latter cases only two figures for each period were available.

Real wages
(Dutch guilders per capita)
1880-1901: 2%
1901-1930: 3%
1930-1950: 0%
1950-1970: 4%

Crowding
(persons per room)
1900-1931: -1%
1931-1947: 0%
1947-1973: -1%

Wheat
(hectoliter per capita)
1875-1901: 1.8% [1.2, 2.4]
1901-1916: 1.8% [0.2, 3.6]

Potatoes
(hectoliter per capita)
1875-1901: 0.7% [-0.2, 1.7]
1901-1916: -1.9% [-2.7, -1.1]

Rice
(kilogram per capita)
1875-1901: 1.9% [0.8, 3.0]
1901-1916: -1.9% [-5.8, 1.8]

Sugar
(kilogram per capita)
1875-1901: 3.3 [3.1, 3.5]
1901-1916: 2.9 [2.0, 3.9]

Beef
(kilogram per capita)
1875-1901: 0.7% [0.1, 1.3]
1901-1916: 0.2% [-0.4, 0.8]

Secularisation
(% people without religious affiliation)
1875-1899: 23%
1899-1930: 19%
1930-1947: 1%
1947-1971: 2%

Female education
(girls aged 12-18 per 1000 receiving education)
1879-1899: 2.9% [2.7, 3.1]
1899-1930: 3.5% [2.3, 4.8]

Total fertility rate
1879-1901: -0.4% [-0.6, -0.2]
1901-1931: -1.4% [-1.5, -1.2]
1931-1950: 0.6% [-0.8, 2.1]
1950-1970: -0.9% [-1.6, -0.2]

Medical doctors
(inhabitants per doctor)
1879-1899: 3.3% [-9.2, 15.7]
1899-1930: -8.0% [-10.3, -5.7]

Water supply system
(% population access to piped water)
1875-1901: 4.3% [3.2, 5.4]
1901-1914: 1.8% [1.7, 1.9]

respiratory diseases) we found an acceleration in the decline after 1901 instead of a deceleration.

With respect to *socio-cultural determinants*, the percentage of people with no church affiliation shows a similar increase in the two periods 1875-1901 and 1901-31 (table 7.5). Such a pattern does not emerge for infectious diseases or for the age groups expected to be most closely related to this variable e.g. acute digestive diseases and infant mortality (table 7.2).

A more direct cultural variable is total fertility. Total fertility shows a slow decline in the period 1875-1901 followed by rapid declines in 1901-31 (table 7.5). This pattern emerged for acute digestive diseases (predominantly occurring at very young ages), for infant and early childhood mortality, as well as for some other childhood diseases such as whooping cough, scarlet fever, diseases of the nervous system and acute respiratory disease (tables 7.2 and 7.3).

As far as *public health factors* are concerned, a rapid increase in the percentage of people with access to water supply systems was observed for the years 1875-1901, followed by a less rapid increase in 1901-31 (table 7.5). However, the strongest declines were found for water-borne infectious diseases in the period 1901-31 (table 7.2). No decline at all was apparent for acute digestive diseases in the period 1875-1901 (table 7.2). The trend of 'access to clean drinking water' was based on data from the year a water supply system was introduced in a particular town. It was assumed that from that year onwards all inhabitants had access to water mains. However, predominantly the wealthier people had access to clean drinking water in the 19th century. They were able to pay for good quality water, and had more knowledge of the importance of clean drinking water than the poor. After the turn of the century good quality drinking water gradually became available to larger groups of the population. The Housing Act of 1901, for example, required a connection to a water supply system for every newly built house (Vogelzang 1956). Such measures, which were introduced after 1900, might explain the acceleration in mortality of water-borne infectious diseases in the period 1901-31

With respect to *medical factors*, data on the number of inhabitants per medical doctor were available for the periods 1879-1899 and 1899-1930. After the turn of the century the number of inhabitants per doctor decreased or, in other words, medical doctor density increased. The early decades of the twentieth century saw a lot of changes in the Dutch health care system. This is best exemplified by the hospital transformation from a nursing home for the indigent sick into an institution for the healing of all (Van der Velden 1996b). Such changes are general and cannot be directly related to specific infectious diseases.

It is likely that the introduction of sulphonamides in the 1930s and antibiotics in the 1940s, as well as the national vaccination programme in the 1950s affected infectious-disease mortality. Examples of infectious diseases that

showed a strong acceleration of decline in the period 1930-50 are influenza/ acute bronchitis, pneumonia, scarlet fever, measles, tuberculosis of the nervous system, puerperal fever, erysipelas and septicaemia/pyemia (table 7.2) (Mackenbach & Looman 1988). The deceleration in the trends of other determinants (e.g. real wage, overcrowding) (table 7.5) supports the idea of a probably important role of medical factors in the period 1931-50.

In the period 1950-70 a diffusion of the use of antibiotics might have further accelerated the decline of some infectious diseases. Different types of tuberculosis as well as rheumatic fever showed rapid declines in the period 1950-70 (table 7.2), which might be related to the use of antibiotics (Mackenbach & Looman 1988). In 1952 the national vaccination programme was started, which included vaccinations against diphtheria and whooping cough. These infectious diseases were eliminated in the period 1950-70 (table 7.2). We also found strong declines for the youngest age groups in the period 1950-70, which might also be related to the introduction of the vaccination programme (table 7.2).

7.5 Conclusion

After the turn of the century a generalised acceleration in infectious disease mortality decline (water-borne as well as airborne) could be observed. After 1931, the acceleration in mortality decline could be predominantly observed among airborne infectious diseases. We tried to relate changes in trends of determinants to changes in infectious-disease-specific mortality trends in order to identify possible determinants for specific infectious diseases. This, combined with the results on the contribution of infectious diseases to mortality decline, might provide an indication of the contribution of specific determinants to mortality decline. Such an analysis is hampered by the fact that different determinants of mortality decline happen to be collinear, and because different determinants might act on the same cause of death.

Nevertheless, it may be possible to draw some conclusions. Fertility decline showed a strong acceleration in the period 1901-31, an acceleration that was also observed for several infant and early childhood diseases such as acute digestive diseases, acute respiratory diseases and whooping cough. Acute digestive and respiratory diseases contributed largely to mortality decline in the period 1901-31. Medical factors (sulphonamides, antibiotics) probably played an important role in the acceleration of mortality decline from airborne infectious diseases in the period 1931-50. The absence of such accelerations in other possible determinants such as socio-economic and socio-cultural factors supports this hypothesis.

Other types of analysis, such as multivariate analysis with variation in the data with respect to the exposure to determinants of mortality decline (e.g. re-

gional analyses), will be needed to further disentangle the complexity of the determinants of mortality decline.

Chapter 8

THE ROLE OF CULTURAL AND ECONOMIC DETERMINANTS IN MORTALITY DECLINE IN THE NETHERLANDS, 1875/79 TO 1920/24

Abstract

Objective: *To determine the relative importance of cultural and economic factors in mortality decline in The Netherlands in the periods 1875/79-1895/99 and 1895/99-1920/24.*

Data and Methods: *Mortality data by region, age, sex and cause of death as well as population data were derived from Statistics Netherlands for the years 1875/79, 1885/89, 1895/99, 1910/14, 1920/24. Regional mortality declines were estimated on the basis of Poisson-regression models. In a multivariate analysis the estimated declines were associated with economic (wealth tax) and cultural variables (Roman Catholicism and secularisation) corrected for confounders (soil type, urbanisation).*

Results: *In the period from 1875/79 to 1895/99, Roman Catholicism was significantly associated with all-cause mortality decline and with mortality decline from diseases other than infections diseases. Mortality declined less rapidly in Roman Catholic areas. Secularisation was significantly associated with infections-disease mortality decline. In areas with a high percentage of people without a religious affiliation, mortality declined more rapidly. In the period from 1895/99 to 1920/24, wealth tax was significantly associated with all-cause and infections-disease mortality decline. Mortality declined more rapidly in wealthy areas. Intermediary factors in the relationship between cultural factors and mortality decline were fertility decline, but more importantly, the number of medical doctors per 100,000 inhabitants. No intermediary factors were found for the association between the economic variable and mortality decline.*

Conclusion: *Cultural and economic factors both played an important role in mortality decline in The Netherlands, albeit in different periods of time. The analysis of intermediary factors*

suggests that the acceptance of new ideas on hygiene and disease processes was an important factor in the association between culture and mortality decline in the late 19th century.

8.1 Introduction

Mortality started to decline rapidly in The Netherlands around 1875. Many other western populations have also experienced rapid mortality declines in the nineteenth century, although the timing was different between countries. The analogous experience in different countries led to the formulation of the demographic transition theory (for an extensive description see Chesnais 1992) and the epidemiological transition theory (first described by Omran (1971)).

In both the demographic and epidemiological transition theories, mortality decline and shift in cause-of-death pattern have been related to 'modernisation' (Chesnais 1992, Omran 1971). Modernisation encompasses processes of economic, political, social and cultural change in society. Socio-economic changes are, for example, industrialisation and specialisation. The percentage of people working in the agrarian sector decreased. Production became more and more mechanised and commercialised. Work became more specialised, which induced a differentiation of occupations and of education. During the modernisation process not only industrial productivity increased, but also consumption by the population. Cultural changes characteristic of the modernisation process are, for example, rationalisation and secularisation. Secularisation refers to a diminishing influence of traditional institutions in society. Related to this is rationalisation, which can be interpreted as an application of scientific knowledge in diverse areas of society.

In literature on the demographic transition and on mortality decline in general the focus was mostly on socio-economic changes. The most well known example is the work of McKeown, who considered improvements in living standards and particularly improvements in nutrition to be the most important factors in 19th and 20th-centuries' mortality decline (McKeown & Record 1962, McKeown *et al.* 1975). Other determinants through which an increase in living standards might have affected mortality decline are, for example, improvements in housing conditions, access to water supply systems, and access to medical care (Preston 1975, Flegg 1982, Kintner 1988a, McFarlane 1989). However, negative health effects of socio-economic changes, especially those related to industrialisation and urbanisation, have been observed too (Woods *et al.* 1988, Huck 1995).

Cultural processes of modernisation might influence health too. Secularisation and rationalisation can be related to an increased literacy, including an increased knowledge of disease processes and an increased openness to new ideas,

for example with respect to (personal) hygiene (Hofstee 1981, Ewbank & Preston 1990, Preston & Haines 1991, Vögele 1994). Fertility decline, which is strongly related to infant and early childhood mortality decline, is also partly determined by culture. Sustained fertility decline requires 'moral acceptability' (Lesthaeghe 1986b).

Cultural factors are much more difficult to study in comparison to economic factors, which probably explains the relatively minor attention for such factors. Culture can be defined as the set of beliefs and attitudes shared by a group of people. Attitudes and beliefs are hard to measure, even more so in a historical setting and at aggregate level. There are, however, variables that can be used as cultural indicators at an aggregate level. Secularisation, for example, is generally measured as the increasing non-adherence to a religious denomination. This variable could be used as an indicator of the cultural changes in the modernisation process.

The pace and timing of the modernisation can differ between groups in society. In The Netherlands, it was reported that Roman Catholics were less inclined to accept new ideas on disease processes and hygiene in comparison to Protestants (Philips 1980). Others described the Roman Catholic population (in particular those that lived in rural areas) as being very obedient to authorities, living within the bonds set by the Roman Catholic clergy (Wichers 1965). Related to these characteristics are the strong adherence to folk medicine (Philips 1980, Rutten 1985), the tendency not to breastfeed infants, and the (persistence of) high fertility rates among Roman Catholics (Van Poppel 1992). In The Netherlands there is a clear regional distinction between Roman Catholics and Protestants. Mortality differences between Roman Catholics and Protestants have also been observed (a high percentage of Roman Catholics is related to high mortality levels) (Van Poppel 1992).

Recently, cultural factors have received more attention in mortality research. Research that addresses the health transition, for instance, explicitly includes cultural factors such as rising female education, and declining fertility (Frenk *et al.* 1991, Cleland & Van Ginneken 1988). Those studies are usually carried out for developing countries. In historical studies of developed countries, cultural factors are more and more mentioned as possibly important determinants of mortality (decline) (Preston & Haines 1991, Vögele 1994, Corsini & Viazzo 1997).

Historical studies in which cultural factors are analysed in a multivariate design, and which address mortality decline instead of mortality levels are still scarce. The aim of this study is to determine the relative importance of both cultural and economic factors in mortality *decline* in The Netherlands in the periods 1875/79-1895/99 and 1895/99-1920/24. The relative importance of the determinants is analysed with a multivariate regression model, in which the re-

gional variation in the prevalence of determinants is related to mortality decline in 27 regions in The Netherlands. Two periods are analysed, because for many regions, mortality decline accelerated after the turn of the century. Results of multivariate analyses are presented and intermediary factors in the association of cultural and economic factors of mortality decline are discussed.

8.2 Data and Method

8.2.1 Data

Mortality and population data

All-cause and cause-specific mortality data by age and sex were obtained from publications of the Dutch Home Office (which provided data for quinquennial periods) for the years 1875/79, 1885/89 and 1895/99. Data for the years 1910/14 and 1920/24 were derived from annual publications of Statistics Netherlands. Mortality data as well as data on explanatory variables were obtained for the (by that time) 11 Dutch provinces and 16 towns (each having more than 20,000 inhabitants) (figure 8.1). Figures for 11 rural areas were calculated by subtracting the values for the 16 towns from the 11 provinces in which they were situated.

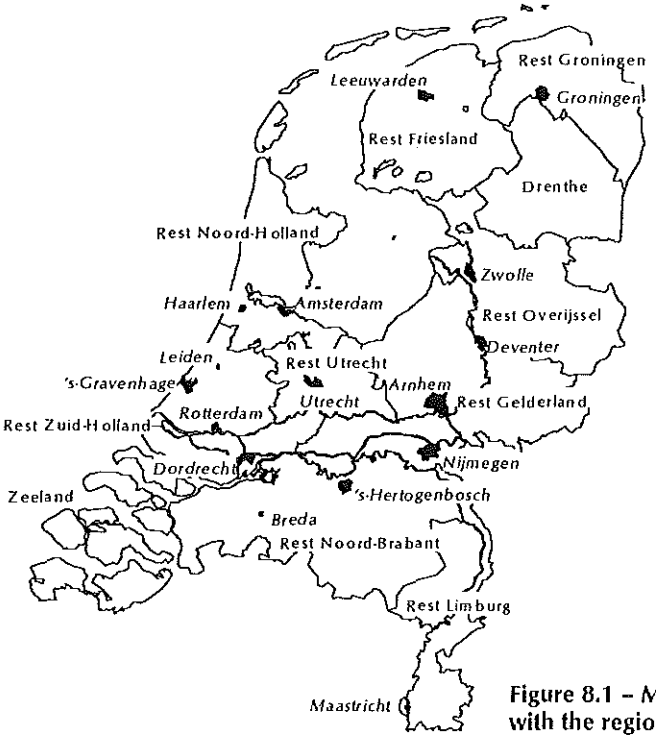


Figure 8.1 – Map of The Netherlands with the regions selected for this study.

The causes of death were divided into two broad groups: infectious diseases and other diseases. The period from 1875/79 to 1920/24 covers one 19th-century cause-of-death classification and 3 editions of the International Classification of Diseases and Causes of Death. Therefore cause-of-death groups had to be reclassified into the different classifications in order to create nosologically continuous cause-of-death groups over time. A detailed description of the reclassification procedure has been published elsewhere (Wolleswinkel-van den Bosch *et al.* 1996). The following causes of death from the 19th-century classification are included in the group 'infectious diseases': syphilis, abscess/ulcer/pyemia, typhus/typhoid fever, continuous fever, malaria, smallpox, scarlet fever, measles, diseases of the skin (i.e. mainly erysipelas), convulsions, brain diseases (i.e. mainly tuberculous meningitis, other meningitis and encephalitis), respiratory tuberculosis, diphtheria/croup, whooping cough, acute respiratory diseases, diarrhoea/dysentery, cholera and puerperal fever. The group 'other diseases' includes all other causes of death.

Determinants of mortality decline

Data on determinants were obtained for 16 towns and 11 rural areas. The economic variable in the analysis is an indicator of wealth viz. wealth tax (in guilders per capita). This tax consisted of 6 components: tax on rental value of houses and other buildings, tax on the number of 'doors and windows connected to open air', tax on the number of fireplaces, tax on furniture, tax on servants and maids, and tax on horses (Blok & De Meere 1978). Two different cultural variables were examined viz. the percentage of population without religious affiliation (parameterisation of secularisation) and the percentage of population with a 'Roman Catholic' affiliation (parameterisation of Roman Catholicism).

The effect on mortality decline of the, distal, economic (wealth) and cultural determinants (secularisation and Roman Catholicism) is indirect (cf. Chapter 3). The association with mortality decline is mediated by other, more proximate, determinants of mortality decline. Wealth can influence health by improvements at household level, such as better food or housing and access to medical care, but wealth can also have a beneficial effect on health because of increased government expenditure on, for example, water supply systems and sewage systems. The association between secularisation or Roman Catholicism and mortality decline is mediated by behavioural factors such as breastfeeding practices, marital fertility, and medical consumption.

The intermediary or proximate determinants used in this study are persons per dwelling, population percentage with access to water supply system, medical doctor density and marital fertility. The number of persons per dwelling is a parameterisation of housing conditions, in particular overcrowding. Overcrowding was often associated with bad sanitary conditions and lack of ventilation. Water-

and food-borne as well as airborne infectious diseases, such as typhus, typhoid fever, cholera, respiratory tuberculosis, and childhood diseases (measles, whooping cough, diphtheria) can be easily transmitted in overcrowded dwellings (Burnett 1991).

Access to clean drinking water will affect mortality from water-borne infectious diseases, such as cholera, typhoid fever, diarrhoeal diseases, and dysentery. In the late 19th century, the construction of water supply systems was carried out by private enterprises in The Netherlands, and depended on the wealth and size of a town. Government interference began after the turn of the century. The Housing Act of 1901, for example, required access to water mains for every new building (Vogelzang 1956).

The influence of medical doctors on mortality decline, with respect to curative care, was limited in the late 19th century. During the early decades of the 20th century more efficacious health care services became available, which was best exemplified by the hospital transformation from a nursing home for the indigent sick into an institution for the health of all (Van der Velden 1996a). It has been argued that medical doctors often served as agents of cultural and behavioural change in the late 19th century because they were intermediaries between the upper class and working class. Medical doctors could introduce to lower classes hygienic behaviour and childcare practices that had been accepted earlier by the upper classes (Kunitz 1991). In the 19th century, medical doctors also played a significant role in the sanitary improvements (Houwaart 1991).

Marital fertility predominantly affects infant and early childhood mortality. High parities and short birth intervals are associated with high infant and early childhood mortality levels (Van de Walle 1986).

Two other, distal, determinants that could confound the association between the economic (wealth) and cultural (Roman Catholicism and secularisation) variables and mortality decline were taken into account in the analyses viz. soil type and urbanisation. In The Netherlands, there are big differences between regions with respect to soil type. Regions can be roughly divided into sandy regions and regions of mainly clay soil. The type of soil affected economic development of the region. The unfavourable sandy soil resulted in mixed farming (to spread risks) and inhabitants were more self-supportive as compared to the clay areas. In the clay regions, the economy was more market-oriented (Jobse-van Putten 1990). The market-oriented regions were also the more open-minded regions (regarding new ideas on disease processes and hygiene). Apart from economic or cultural associations, soil type could also affect mortality in other ways. Clay soil was more brackish than sandy soil, which made groundwater in those areas less suitable for drinking water. As a result, the more polluted surface water was used for drinking and cleaning. This in turn resulted in higher levels of mortality from water-borne infectious diseases (Hofstee 1981).

Urbanisation is related to both economic and cultural variables. In towns, there was more differentiation of occupations as compared to rural areas, and production was market-oriented. Urbanisation is often related to industrialisa-

Table 8.1 – Distal and proximate determinanants used in the analyses: parameterisation, data source and years.

	Parameterisation	Data Source	Years in analysis
Distal determinants			
Soil type	Region consists predominantly of sandy soil: yes/no	De Grote Bosatlas, 1968	1875/79, 1895/99
Urbanisation	Town yes/no	See figure 8.1	1875/79, 1895/99
Roman Catholicism	Percentage population with 'Roman Catholic' as religious affiliation	Historical Ecological Database (HED)*	1875/79, 1895/99
Secularisation	Percentage population without religious affiliation	HED	1875/79, 1895/99
Wealth	Wealth tax in guilders per capita	HED	1875/79, 1895/99
Proximate determinants			
Marital fertility	Ig-index †	Calculated on the basis of figures derived from the HED and Lesthaeghe, 1977	1875/79, 1885/89, 1895/99, 1910/14, 1920/24
Housing conditions	Number of persons per dwelling	Bijdragen tot de Statistiek van Nederland (CBS, 1879, 1889, 1899, 1910, 1920)	1875, 1889, 1899, 1910, 1920
Clean drinking water	Percentage of population living in town with piped water	De Grootte, 1995	1879, 1889, 1899, 1914
Medical care	Number of medical doctors per 100,000 inhabitants	Verslagen aan de koning van de bevindingen en handelingen van het geneeskundig staatstoezicht in het jaar 1875-1899, Verslagen en mededeelingen betreffende de volksgezondheid 1923	1875/79, 1885/89, 1895/99, 1923

* The Historical-Ecological Database is managed by Stichting Beleidsondersteunend Ruimtelijk Onderzoek (BRON), which is related to the University of Amsterdam.

† The Ig-index is the age-standardised ratio of the annual number of legitimate births occurring to married women of childbearing age (15-49), that would occur if they were subject to natural fertility (i.e. age-specific marital fertility rates of the Hutterite population) (Lesthaeghe 1977).

Table 8.2 – Range of the values of the cultural and economic variables.

	Percentage population Roman Catholics		Percentage population without religious affiliation		Wealth tax Guilders per capita	
	1875/79	1895/99	1875/79	1895/99	1875/79	1895/99
Mean	38.1	37.5	0.4	2.2	2.6	2.3
Max	98.5	98.3	1.2	7.9	5.4	4.3
Min	5.2	4.8	0.0	0.0	0.7	0.8

tion, but there was not such a strong urban-industrial complex in The Netherlands compared to other western European countries (Knippenberg 1980). Towns were a melting pot of people with all kinds of socio-cultural backgrounds. Social relationships more easily crossed boundaries of kinship and traditional alliances (Van der Woude *et al.* 1990). In such an environment new ideas on hygiene, disease processes and childcare, were more easily accepted than in rural areas. Towns had a better infrastructure than rural areas, including better access to education and medical care. Urbanisation can also have an effect on health independent of wealth or culture. In rural areas fresh food was more readily available, the price of milk was lower, the transport of food was less time-consuming, and the quality of food was better. There were also negative health effects in towns. The high population density facilitated the transmission of, particularly airborne, infectious diseases.

In addition, the tax on rental value (part of the wealth tax variable used in the analyses) differed according to the size of the town. Other components such as tax on horses or servants were not dependent of town size. When urbanisation is added to the model this partly corrects for the effect of differential tax values by town size. Table 8.1 presents the determinants used in the analyses in this study, their parameterisation, the years under study, and data source. In table 8.2 the range of the values of wealth tax, Roman Catholicism and secularisation are given.

8.2.2 Methods

The pace of mortality decline in the regions was estimated on the basis of a log-linear regression analysis (Poisson regression). The model used is described below.

$$E(Y_{ijrt}) = N_{ijrt} e^{\alpha+\beta_i+\gamma_j+\delta_r+(\varepsilon+\varepsilon_r)T}$$

$E(Y_{ijrt})$= expected number of deaths per age-group (i), sex (j), region (r) and quinquennium (t)

N_{ijrt} = population numbers per age-group, sex, region and quinquennium
α = intercept (i.e. log regional mortality rate 1875/79 men, age 0, region Amsterdam)
β_i = log relative risk sex-category i (men reference category)
γ_j = log relative risk age-category j (age 0 reference category)
δ_r = log relative risk regional mortality rate (Amsterdam reference category)
ε = log annual mortality decline (region Amsterdam)
ε_r = difference in annual mortality decline between region$_r$ and Amsterdam
T = year since 1875/79 (0 for 1875/79, 10 for 1885/89, 20 for 1895/99)

Percentage regional mortality decline per decade = $(1 - e^{(\varepsilon+\varepsilon_r).10})*100$

Mortality declines were estimated for two periods: 1875/79 to 1895/99 and 1895/99 to 1920/24. The same model as described above was used for the second period with 1895/99 as T=0. Most regional mortality trends were non-linear over the entire period from 1875/79 to 1920/24. By splitting up the period from 1875/79 to 1920/24 into a period covering the late 19th century and a period covering the early 20th century, linear trend estimation was made possible. The estimates for the logarithm of mortality decline in the 27 regions ($\varepsilon+\varepsilon_r$) were used in a multivariate linear regression analysis in order to relate mortality decline to cultural (percentage of Roman Catholics or percentage of people without religious affiliation) and economic (wealth tax) variables corrected for possibly confounding factors.

The level of wealth and the percentages for secularisation or Roman Catholicism at the onset of a period of decline were related to mortality decline in the periods under study. Wealth and Roman Catholicism or secularisation were considered to be variables of which no immediate effect on mortality was expected, but which would induce change in more proximate variables of mortality. The next step in the analyses was to add the more proximate determinants of mortality decline to the multivariate model. In doing so, we tried to determine which factors mediated between the cultural and economic associations (if present) and mortality decline, and whether the proximate factors showed an independent significant association with mortality decline. The proximate variables were expected to have an immediate effect on mortality. Therefore, change in the proximate variables in the period under study was related to change in mortality (i.e. mortality decline). The absolute change over time of the proximate variables was calculated using linear regression analyses. Results of the multivariate analyses are presented for all-cause mortality, infectious-disease mortality and 'other' causes of death.

In order to determine whether the regional mortality declines differed significantly, we performed a likelihood ratio test; all tests were significant. To pre-

sent significant differences in decline from Amsterdam for individual regions (i.e. ε_r differs significantly from zero), results of T-tests will be presented.

8.3 Results

8.3.1 Regional variation in mortality decline

Figure 8.2 shows the regional differences in all-cause, infectious-disease, and 'other disease' mortality decline in the periods from 1875/79 to 1895/99 and from 1895/99 to 1920/24. Exact figures are provided in table 8.3. In both periods, the decline in the majority of the regions differed significantly from the decline in Amsterdam, which was taken as a reference (table 8.3). The first period is characterised by rapid declines in the rural areas of the southwestern provinces (Zuid-Holland, Zeeland) and northern provinces (Friesland, Groningen) of The Netherlands. Mortality declines are slow in the rural areas and most towns in the southern provinces (Noord-Brabant, Limburg) and eastern provinces (Gelderland, Drenthe). In many provinces, mortality declines were equally rapid in towns and rural areas in the period from 1875/79 to 1895/99. With respect to infectious-disease mortality decline in this period, a similar pattern to that for total mortality decline could be observed i.e. more rapid declines in the southwestern and northern parts of The Netherlands. The declines seem to be more

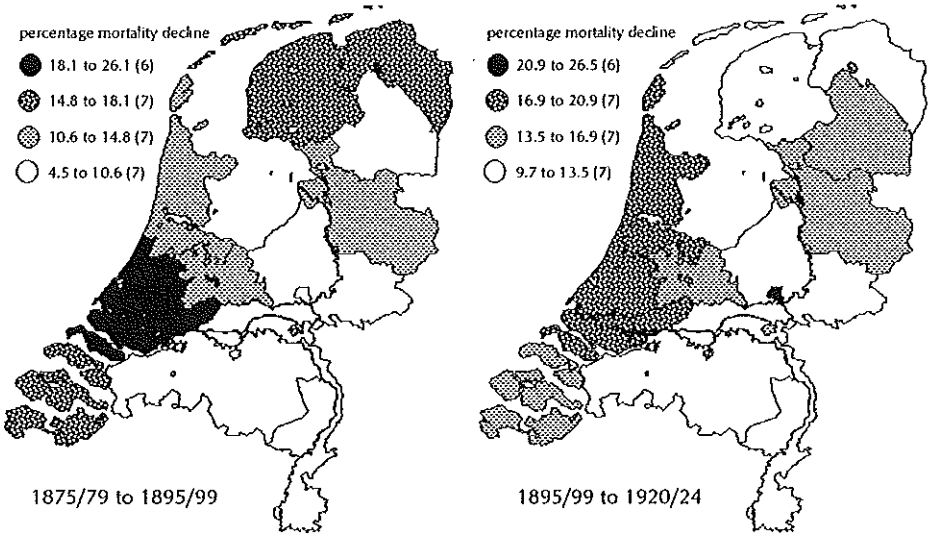


Figure 8.2a – Percentage all-cause mortality decline per decade, 1875/79 to 1895/99 and 1895/99 to 1920/24

rapid in towns as compared to rural areas. In the case of 'other diseases', declines were most rapid in the rural areas and towns of Zuid-Holland and Overijssel.

In all regions, the pace of all-cause mortality decline was more rapid in the early decades of the 20th century than in the late 19th century (figure 8.2 and table

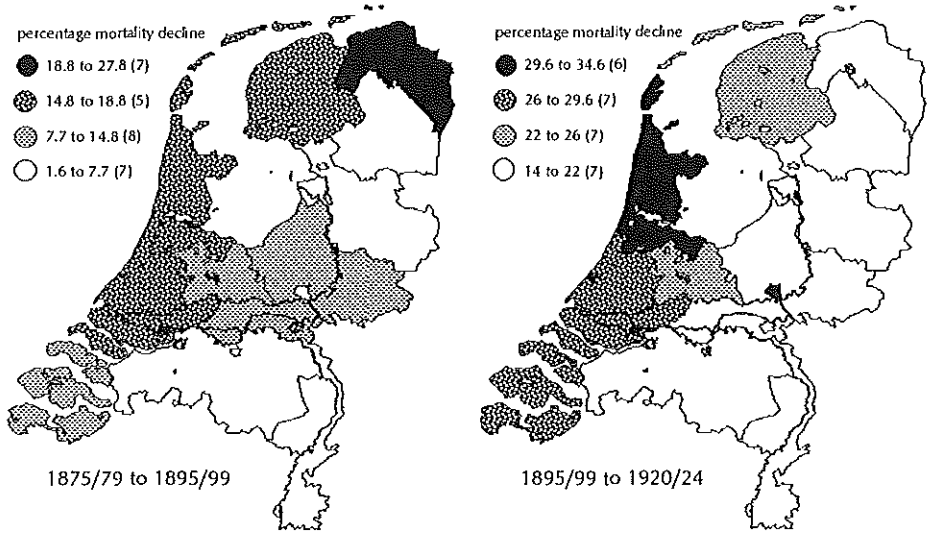


Figure 8.2b – Percentage infectious diseases mortality decline per decade, 1875/79 to 1895/99 and 1895/99 to 1920/24

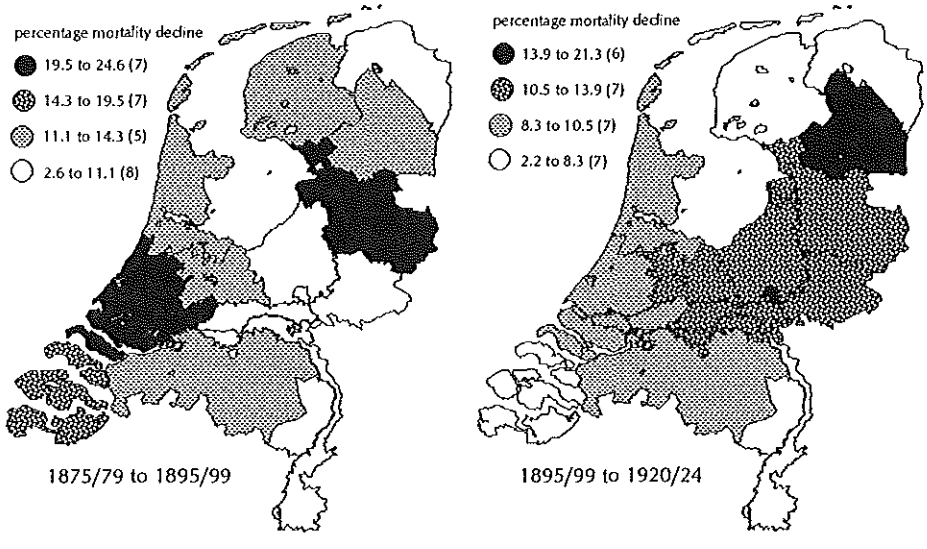


Figure 8.2c – Percentage mortality decline from other diseases than infectious diseases per decade, 1875/79 to 1895/99 and 1895/99 to 1920/24

8.3). This was especially due to rapid infectious-disease mortality declines. The pace of decline of 'other diseases' mortality did not accelerate.

The regional pattern changed compared to the former period. The northern provinces, for example, no longer show the relatively rapid declines. As for all-cause mortality, declines seem to be more rapid in towns compared to their rural surroundings in the period from 1895/99 to 1920/24. Remarkable are the rapid decline of infectious-disease mortality in the rural areas of the province

Table 8.3 – Poisson regression estimates of percentages mortality decline per decade 1875/79 to 1895/99 and 1895/99 to 1920/24. 16 towns and 11 rural areas (= provinces minus selected towns). All-cause mortality, infectious disease mortality and mortality from other causes of death.

	All-cause mortality decline		Infectious diseases decline		Other causes of death decline	
	1875/79 to 1895/99	1895/99 to 1920/24	1875/79 to 1895/99	1895/99 to 1920/24	1875/79 to 1895/99	1895/99 to 1920/24
Towns						
Amsterdam	17.6	16.2	19.6	24.8	15.2	8.9
Arnhem	4.5	23.0	6.3	32.5	2.6	16.1
Breda	8.9	18.1 ns	10.7	28.5 ns	7.3	9.8 ns
Deventer	15.5 ns*	12.9	7.7	24.2 ns	24.6	2.2
Dordrecht	24.1	26.5	23.6	33.9	24.3	21.3
Groningen	18.7 ns	16.9 ns	21.2 ns	28.6 ns	15.3 ns	7.6 ns
Haarlem	13.2	16.7 ns	19.1	24.7	6.8	10.9 ns
's-Hertogenbosch	4.9	20.8	5.9	28.5	4.2	13.2
Leeuwarden	14.2	13.3	14.4	24.1 ns	14.3 ns	6.5 ns
Leiden	26.1	20.9	27.8	30.8	24.5	13.9
Maastricht	10.6	21.5	17.4 ns	22.5 ns	3.4	20.4
Nijmegen	16.0 ns	13.5	17.6 ns	14.4	14.3 ns	12.7
Rotterdam	18.1	20.9	13.4	28.0	22.5	14.5
's-Gravenhage	16.2	17.9	13.3	29.6	19.1	8.7 ns
Utrecht	18.5 ns	18.8	18.8 ns	26.0 ns	18.1	12.6
Zwolle	10.7	20.9	3.5	34.6	19.5	8.3 ns
Rural areas of provinces						
Friesland	14.8	11.8	17.7	22.3	12.3	5.3
Groningen	15.1	11.7	20.4 ns	18.8	9.0	5.9
Drenthe	8.2	15.9 ns	4.9	16.9	11.1	15.1
Overijssel	11.8	16.5 ns	4.1	21.2	19.9	11.6
Gelderland	8.8	12.3	8.2	14.0	9.4	10.9
Utrecht	11.8	15.7 ns	9.6	22.0	13.8 ns	10.5 ns
Noord-Holland	14.4	19.7	15.1	31.1	13.1	10.3
Zuid-Holland	18.7	17.9	14.8	27.5	22.5	10.1 ns
Zeeland	16.9 ns	16.3 ns	14.4	28.2	19.3	6.7
Noord-Brabant	7.1	11.7	1.6	14.7	11.4	9.3 ns
Limburg	6.2	9.7	2.6	14.3	9.4	5.6

* ns means that the decline in that region did not differ significantly from the decline in Amsterdam ($p<0.05$)

of Noord-Holland and the rapid decline of 'other-diseases' mortality in the eastern provinces of Gelderland, Overijssel and particularly Drenthe.

8.3.2 Correlation between mortality decline and determinants of decline

Before presenting the results of the multivariate analyses, simple univariate correlations will be discussed between possible determinants of mortality decline and the estimated all-cause, infectious- disease, and 'other- diseases' mortality decline in the periods from 1875/79 to 1895/99 and from 1895/99 to 1920/24 (table 8.4).

The cultural factor Roman Catholicism is significantly correlated with all-cause mortality decline and mortality decline from 'other diseases' in the period from 1875/79 to 1895/99. A high percentage of Roman Catholics is associated

Table 8.4 – Pearson's correlation coefficients between determinants and mortality decline in the periods 1875/79 to 1895/99 and 1895/99 to 1920/24.*

	All-cause mortality decline		Infectious diseases decline		Other diseases decline	
	1875/79 to 1895/99	1895/99 to 1920/24	1875/79 to 1895/99	1895/99 to 1920/24	1875/79 to 1895/99	1895/99 to 1920/24
Distal determinants						
Sandy soil	0.55‡	0.29	0.69‡	0.40†	0.16	0.08
Urbanisation	-0.25	-0.52‡	-0.33	-0.51‡	-0.07	-0.28
% Roman Catholics	0.55‡	0.04	0.37	0.28	0.51‡	-0.19
% Without religious affiliation	-0.38†	0.06	-0.53‡	-0.25	-0.05	0.28
Wealth tax	-0.28	-0.64‡	-0.33	-0.71‡	-0.11	-0.29
Proximate determinants						
Marital fertility	0.47†	0.47†	0.50‡	0.37	0.26	0.32
Persons per dwelling	0.29	-0.09	0.18	-0.08	0.28	-0.02
% Access to water supply system	-0.10	0.14	-0.16	0.12	-0.03	0.06
Medical doctor density	0.52‡	-0.06	0.39†	-0.17	0.42†	0.09

* As far as distal determinants of mortality decline are concerned, the level of the determinants at the onset of the period (i.e. 1875/79 or 1895/99) is related to percentage mortality decline; as far as proximate determinants are concerned, absolute change in the determinants in the period under study is related to percentage mortality decline. A negative correlation means that a high level of the determinant is correlated with (rapid) mortality decline, or in case of proximate determinants, an increase in the level of the determinant is related to (rapid) mortality decline.

† significance at the 95% level.

‡ significance at the 99% level.

with less rapid mortality declines. With respect to infectious-disease mortality, the cultural factor secularisation is significantly associated with mortality decline. A high percentage of people without religious affiliation is associated with rapid mortality declines.

Other variables that showed significant correlations in the late 19th century are soil type (total and infectious-disease mortality), changes with regard to medical doctors (total mortality) and change in marital fertility (infectious diseases). Sandy soil, an increase in doctor density and an increase in marital fertility were all associated with less rapid mortality declines.

In the period from 1895/99 to 1920/24, a significant correlation between the cultural variables and mortality decline had disappeared. The wealth indicator (wealth tax), on the other hand, was strongly associated with all-cause and infectious-disease mortality decline. Greater wealth was associated with more rapid mortality declines. Other significant correlations were found between urbanisation and mortality decline from all-causes and infectious diseases. Mortality declines were generally less rapid in rural areas.

8.3.3 Multivariate analysis of cultural and economic determinants of mortality decline

Table 8.5 shows the results of the multivariate analyses to study the relative importance of cultural and economic determinants of mortality decline. The economic and cultural variables were analysed together corrected for confounding variables. Separate analyses were conducted for the variables Roman Catholicism and secularisation.

The results of the univariate correlations showed that the cultural factor is more important in the late 19th century than the economic factor, and that in the early 20th century the economic factor is more important than the cultural factor (table 8.4). In a multivariate analysis, after correction for confounders, this conclusion remains valid (table 8.5). In case of all-cause and 'other-disease' mortality decline, the variable Roman Catholicism showed a significant association. The association for mortality decline from other diseases was somewhat stronger than that for infectious diseases. Secularisation showed a significant association in the case of infectious-disease mortality decline. This association was less significant than the association between Roman Catholicism and mortality decline.

In the period from 1895/99 to 1920/24 the wealth indicator, wealth tax, is the most important factor in all-cause and infectious-diseases mortality decline. The association was strongest for infectious- disease mortality.

The variables in the model with respect to the percentage of Roman Catholics explained 50% of the variation in total mortality decline, 55% of the variance in mortality decline from infectious diseases, and 28% of mortality decline from 'other diseases' in the period from 1875/79 to 1895/99. When the

percentage for people without religious affiliation was used as cultural variable, the percentage variance decreased in case of all-cause and 'other-disease' mortality decline. In the models for the period from 1895/99 to 1920/24 the percentages variance explained were about similar.

Table 8.5 – Association between level of economic determinants (wealth tax) and cultural determinants (% Roman Catholics or % without religious affiliation) in the periods 1875/79 and 1895/99 and mortality decline in the periods 1875/79 to 1895/99 and 1895/99 to 1920/24.

	Change of percentage mortality decline per decade per unit difference in the independent variable (percent points)		
	All-cause mortality	**Infectious disease mortality**	**Mortality from other causes of death**
1875/79 to 1895/99			
Wealth tax *(1 guilder per capita higher)*	-0.8	-1.3	-0.4
% Roman Catholics *(10 percent points higher)*	-0.9‡	-0.6	-1.3‡
R^2	0.50	0.55	0.28
Wealth tax *(1 guilder per capita higher)*	-0.3	-1.1	+0.4
% Without religious affiliation *(1 percent point higher)*	+3.7	+7.6†	-0.5
R^2	0.34	0.57	0.03
1895/99 to 1920/24			
Wealth tax *(1 guilder per capita higher)*	+3.3†	+5.9‡	+1.6
% Roman Catholics *(10 percent points higher)*	+0.0	-0.0	+0.3
R^2	0.41	0.56	0.14
Wealth tax *(1 guilder per capita higher)*	+3.5†	+6.4‡	+1.5
% Without religious affiliation *(1 percent point higher)*	-0.6	+0.0	-0.9†
R^2	0.47	0.52	0.24

Independent variables in the model: wealth tax, % Roman Catholics (or % withous religious affiliation), urbanisation, soil type
+ means more rapid decline, - means less rapid decline.
† means significance at the 95% level, and ‡ at the 99% level.

8.3.4 Analysis of proximate determinants of the associations of economic and cultural variables with mortality decline

Besides the distal determinants wealth tax and percentages of Roman Catholics or people without religious affiliation, there are more proximate determinants of mortality decline, e.g. housing conditions, medical doctor density, access to clean

Table 8.6a – Analysis of intermediary factors (proximate determinants) in the significant associations between level of cultural (% Roman Catholics, % without religious affiliation) and economic (wealth tax) determinants in 1875/79, and mortality decline in 1875/79 to 1895/99.

	Change of percentage mortality decline per decade per unit difference in the independent variables (percent points)		
	All causes	**Infectious disease mortality**	**Mortality from other causes of death**
% Roman Catholics *(10 percent points higher)*			
Confounders only*	-0.94‡	n.s.a.	-1.29†
Confounders + marital fertility	-0.83†		-1.26†
Confounders + persons per dwelling	-0.83†		-1.15†
Confounders + access to water supply system	-0.94‡		-1.30‡
Confounders + medical doctor density	-0.71†		-1.06†
% Without religious affiliation *(1 percent point higher)*			
Confounders only*	n.s.a.	+7.60†	n.s.a.
Confounders + marital fertility		+6.20	
Confounders + persons per dwelling		+8.00†	
Confounders + access to water supply system		+7.55	
Confounders + medical doctor density		+6.20	
Wealth tax *(1 guilder higher)*			
Confounders only§	n.s.a.	n.s.a.	n.s.a.

n.s.a. = no significant association
+ means more rapid decline, - means less rapid decline
* Wealth tax, urbanisation, soil type
† significance at the 95% level
‡ significance at the 99% level
§ % Roman Catholics (or % without religious affiliation), urbanisation, soil type

drinking water and marital fertility. These determinants might mediate the association between the above-mentioned cultural and economic determinants and mortality decline. The proximate determinants might also have an independent significant association with mortality decline.

The addition of absolute change in marital fertility to the model, as presented in table 8.5, reduced the association between the percentage of Roman Catholics and mortality decline in the period from 1875/79 to 1895/99 by 12% (table 8.6a). An increase in marital fertility was related to a less rapid mortality decline. The number of persons per dwelling also turned out to be a mediator between the association of Roman Catholics with total mortality decline.

Table 8.6b – Analysis of intermediary factors (proximate determinants) in the significant associations between level of cultural (% Roman Catholics, % without religious affiliation) and economic (wealth tax) determinants in 1895/99, and mortality decline in 1895/99 to 1920/24.

	Change of percentage mortality decline per decade per unit difference in the independent variables (percent points)		
	All causes	Infectious disease mortality	Mortality from other causes of death
Wealth tax *(1 guilder higher)*			
Confounders only*	+3.3†	+5.9‡	n.s.a.
Confounders + marital fertility	+3.2†	+5.7‡	
Confounders + persons per dwelling	+3.3†	+5.9‡	
Confounders + access to water supply system	+3.3†	+5.9‡	
Confounders + medical doctor density	+4.1‡	+6.7‡	
% Roman Catholics *(10 percent points higher)*			
Confounders only§	n.s.a.	n.s.a.	n.s.a.
% Without religious affiliation *(1 percent point higher)*			
Confounders only§	n.s.a.	n.s.a.	n.s.a.

n.s.a. = no significant association
\+ means more rapid decline, - means less rapid decline
* % Roman Catholics, urbanisation, soil type. The results for secularisation instead of Roman Catholicism as confounding variable were highly comparable
† significance at the 95% level
‡ significance at the 99% level
§ Wealth tax, urbanisation, soil type

Changing the number of persons per dwelling in the model also reduced the association between percentage of Roman Catholics and mortality decline by 12%. An increase in the number of persons per dwelling was related to less rapid mortality decline. A third proximate determinant that mediated the association between percentage of Roman Catholics and mortality decline was medical doctor density. The addition of this variable to the model reduced the association by 24%. However, an increase in the number of medical doctors per 100,000 inhabitants was associated with less rapid mortality decline whereas an association with more rapid mortality decline was expected.

With respect to infectious-disease mortality, marital fertility and medical doctor density (both 18% reduction) mediated the cultural association. For mortality decline from diseases other than infectious diseases, changes in the number of persons per dwelling in medical doctor density reduced the cultural associations by 11% and 18% respectively in the period from 1875/79 to 1895/99. The same contra-intuitive association between medical doctor density and mortality decline was found for infectious-disease mortality decline and mortality decline from diseases other than infectious diseases.

With respect to the period from 1895/99 to 1920/24, in which wealth tax turned out to be an important determinant of all-cause and infectious-disease mortality decline, none of the proximate variables seemed to be an intermediate for the association between tax and mortality decline (table 8.6b). The addition of medical doctor density made the association even stronger.

Independent significant associations of proximate determinants and mortality decline were found neither in the period from 1875/79 to 1895/99 nor in the period from 1895/99 to 1920/24.

8.4 Discussion

Cultural and economic factors played a role in mortality decline in The Netherlands in the late 19th and early 20th centuries. Cultural factors were relatively more important in the late 19th century and economic factors in the early 20th century. Factors that mediated the relationship between the cultural factor and mortality decline seem to be changes in marital fertility, persons per dwelling, and medical doctor density. Intermediary factors could not be identified for the association between tax and total or infectious-disease mortality decline. Before discussing these results some comments on the used data and methods will be made first.

8.4.1 Data and method

Reliability of historical mortality data

In the analyses a distinction is made between infectious-disease mortality decline and mortality decline from diseases other than infectious diseases. However, it is conceivable that exchanges took place between categories of infectious diseases and other diseases. For example, exchanges may have taken place between respiratory tuberculosis (infectious diseases) and chronic respiratory diseases (other diseases), or acute respiratory diseases (infectious diseases) and chronic respiratory diseases (other diseases). Some researchers mentioned the possibility of such exchanges in the Dutch literature, but others did not consider this to be a major problem (Evers 1882, Vollenhoven 1889).

Exchanges like these would have affected the results of the analyses if the inaccuracies in coding were differential between the regions. In other words, in some regions, certain coding inaccuracies occurred only or significantly more than in other regions. If the coding inaccuracies were non-differential between regions, the estimates of the infectious disease and 'other diseases' mortality decline would be inaccurate, although the analysis of regional differences in mortality decline would not be affected.

There is not much literature about regional differences in coding. Onnen (1895) mentioned the occurrence of exchanges between the categories 'debility' (a 19th-century cause-of-death category consisting predominantly of infant and early childhood diseases, and old age) and acute digestive diseases. The 'debility'-category is included in the 'other diseases' group in this analysis, while acute digestive diseases are included in the infectious-diseases group. Onnen's paper shows that mortality from 'debility' and acute digestive diseases fluctuated strongly in several large towns in The Netherlands in the periods 1880/85 and 1885/90. Such fluctuations over time within towns will lead to less accurate estimations of mortality decline from that specific cause of death. However, this paper only includes results for the broad groups 'infectious diseases' and 'other diseases', which will have reduced the effect of those inaccuracies on the results of the analyses in this paper.

Methodology

The analysis of the relative importance of cultural and economic determinants in mortality decline was conducted in two steps. First, regional mortality declines were estimated using a Poisson-regression model. Secondly, those estimates were related to a set of independent variables in a multivariate linear regression analysis. By using the estimates of mortality decline in the multivariate regression analysis, the estimated declines are used as point-estimates. Information on the accuracy of the estimates of regional mortality decline is lost during the second step of the analysis. Analysis of the standard errors showed that they were on

average about 18% of the differences between the estimates of mortality decline. This means that there was extra variation in the estimates of mortality decline, which is not taken into account in the multivariate analysis. This variation was, however, not sizeable. Besides, strong associations (significance at the 99%-level) between the economic and cultural variables and mortality decline were found in several instances (table 8.5).

If variables in a multivariate model are strongly correlated we cannot distinguish their separate associations with the dependent variable i.e. mortality decline. This is known as the multicollinearity problem. The variance-covariance matrix of the parameters of the model was calculated for the periods 1875/79 to 1895/99 and 1895/99 to 1920/24 (Norusis 1990). Relatively strong correlations were only found for urbanisation and wealth tax. The correlation coefficients were 0.7 and 0.8 respectively in the two periods. Although the correlation coefficients are not extremely high, it should be taken into account that, especially in the period from 1895/99 to 1920/24, urbanisation and wealth tax are to some extent related.

Absolute change in proximate determinants was related to mortality decline in the analysis presented in this paper. Using relative changes did not change the results so that other proximate determinants turned out to be intermediary factors in the associations between the economic and cultural variables and mortality decline.

8.4.2 Relationship between mortality level and mortality decline

Figure 8.2 shows the differences in mortality decline in the periods from 1875/79 to 1895/99 and from 1895/99 to 1920/24. It could be that regions with the most rapid mortality declines were also the regions with the highest mortality levels at the onset of a period of decline. Mortality levels might have been high in urban areas due to, among other things bad sanitation, but mortality could also decline more rapidly in those areas due to, for example, early fertility declines or early introduction of water supply systems. Correlation coefficients were calculated for the logarithm of regional levels of mortality ($\alpha+\gamma_r$ in the Poisson-regression model) at the onset of a period of decline and the logarithm of regional mortality declines ($\varepsilon+\varepsilon_r$) in that period. A significant association (95%-level) between levels and decline was found for the period 1875/79 to 1895/99 (-0.49), but not for the period 1895/99 to 1920/24 (-0.36). In the period 1875/79-1895/99, areas with the highest mortality in 1875/79 tended to show the most rapid mortality declines.

In order to find out whether regions with relatively high mortality levels still had relatively high mortality levels at the end of a period of decline, correlation coefficients were calculated for mortality levels in 1875/79 and 1895/99, and in 1895/99 and 1920/24. Both correlation coefficients were significant at the

95%-level (0.53 and 0.79 respectively). The correlation between mortality levels in 1875/79 and 1895/99 was less strong as compared to 1895/99 and 1920/24. Certain towns with the highest mortality in 1875/79, e.g. Leiden and Dordrecht, showed low mortality levels in 1895/99.

8.4.3 Cultural determinants of mortality decline

This study shows that cultural factors played a relatively important role in late 19th-century mortality decline in The Netherlands. In the literature, the cultural factor in mortality decline has been discussed in terms of increased attention for personal health care, knowledge of disease processes and acceptance of new ideas on hygiene (Ewbank & Preston 1990, Preston & Haines 1991, Vögele 1994). In this paper two different cultural variables have been used viz. secularisation and Roman Catholicism. Secularisation refers probably more directly to cultural changes related to the modernisation process, such as the acceptance of new ideas, than Roman Catholicism does. Secularisation refers to a decreasing adherence to any religious denomination. Roman Catholicism refers to specific characteristics of the Roman Catholic populations as compared to other religious subgroups of the population. These characteristics include high fertility rates and a reluctance to accept new ideas, and also specific practices with respect to infant care such as breastfeeding, which was not common among Roman Catholics (Van Poppel 1992, Kintner 1988b).

With respect to all-cause mortality decline and mortality decline from 'other diseases' the cultural factor of percentage of Roman Catholics was associated with mortality decline, while percentage of people without religious affiliation was associated with mortality decline from infectious diseases. The findings that percentage without religious affiliation (secularisation) was only significantly associated with infectious-disease mortality decline might indicate the importance of the acceptance of new ideas on hygiene, personal health care, and knowledge of disease processes for infectious-disease mortality decline. As for mortality decline from 'other diseases' other factors also seem to play a role.

The two cause-of-death groups 'infectious diseases' and 'other diseases' differ with respect to age distribution. In 1875, about one-third of both groups consisted of mortality at age 0. With respect to infectious-disease mortality, about 20% of the cases occurred in age group 1-4, while this was only about 6% for mortality from diseases other than infectious diseases. With respect to mortality at older ages, only 15% of the cases occurred for people over 50 years of age, while in the case of mortality from other diseases this was about 40%.

Marital fertility decline was a more important proximate determinant that mediated the association between percentage without religious affiliation and infectious-diseases mortality (reduction of the association by 18%), than it was in the association between percentage of Roman Catholics and mortality decline

from other diseases (reduction of the association by 11%). This may be due to a stronger association between secularisation and marital fertility than between Roman Catholicism and fertility, and to the fact that the category 'infectious diseases' consisted for a large part of early childhood mortality as well as infant mortality.

The absolute change in the number of persons per dwelling turned out to be a proximate determinant in the association between percentage of Roman Catholics and total mortality decline and mortality decline from other diseases. An age-specific analysis showed that persons per dwelling only played a role at old ages (ages 65 to 79) (results not shown). The group 'other diseases' consisted of, among other causes of death, chronic digestive diseases, chronic respiratory diseases, and 'consumption of old age' (excluding respiratory tuberculosis). The latter is an important category. It is conceivable that poor housing conditions (parameterised as persons per dwelling) such as damp houses affect chronic respiratory disease. There are at least reports on tuberculosis mortality and housing (MacFarlane 1989, Condran & Cheney 1982). It is not clear why Roman Catholicism is related to the older age groups. Most of the literature on Roman Catholicism is related to breastfeeding practices and fertility, both of which are determinants of mortality at very young ages.

The association between Roman Catholicism and mortality decline from other diseases might indicate another association. Roman Catholicism is strongly confined to the south of The Netherlands. Other variables with the same regional distribution might account for the association between Roman Catholicism and mortality decline, such as mediation through housing conditions. The most likely variables to be related to housing conditions are urbanisation and wealth. Indicators of both variables had already been included in the model.

An important proximate determinant in the association between the cultural factor and mortality decline was the change in the number of medical doctors per 100,000 inhabitants (table 8.6a). However, an increase (or a less strong decline) in medical doctor density was associated with less rapid mortality decline, which was not as expected. In the analysis presented in this paper, absolute change of the determinant was related to percentage of mortality decline. If relative change was used, the same contra-intuitive association between medical doctor density and mortality decline was observed. Relating change in medical doctor density to mortality decline assumes a direct effect of the supply of medical care on health. This might be the case for curative health care. In the late 19th century, curative effects of health care were, nevertheless, limited.

Earlier, we pointed out that medical doctors could play a role in the diffusion of new ideas on health (Kunitz 1991). When considering this role of medical doctors, no immediate effect of medical doctors on mortality decline is expected. An analysis was conducted, in which not the change in medical doctor density

but the level of medical doctor density at the onset of the period of decline was added to the model, so as to investigate medical doctors' role. In the period from 1875/79 to 1895/99, medical doctor density in 1875/79 was negatively associated with mortality decline. A large number of medical doctors per 100,000 inhabitants in 1875/79 was related to rapid mortality decline. This association was significant with respect to all-cause mortality and mortality from infectious diseases. The association between the percentage of Roman Catholics and mortality decline was reduced by 38% in the case of all-cause mortality and by 24% in the case of mortality from 'other diseases'. The association between percentage without religious affiliation and infectious disease mortality decline was also reduced by 24%. These results suggest that medical doctors in the late 19th century had an important role as 'agents of cultural and behavioural change' (Kunitz 1991), i.e. health behaviour, rather than a curative role.

8.4.4 Economic determinants of mortality decline

We were not able to identify proximate determinants that mediated the association between wealth tax and mortality in the period from 1895/99 to 1920/24. The way in which different proximate determinants have been operationalised might be related to these negative results. Housing conditions, for example, were operationalised as 'persons per dwelling'. This was probably not specific enough to measure overcrowding, bad ventilation and bad sanitary conditions. More specific data such as 'persons per room' or 'rooms connected to open air' were, however, only available at a higher aggregation level. The variable 'wealth tax' is an indicator of wealth in general including, for example, income and nutrition. However, the nature of the wealth tax variable (tax on 'doors and windows connected to open air', tax on fireplaces), also suggests that housing conditions might have played a role in the relationship between wealth and mortality decline.

As far as the operationalisation of the availability of water mains is concerned, it was assumed that the whole population had access to clean drinking water since the construction of a water supply system. This may not have been the case, which could have affected the results. However, another Dutch study showed that before 1895 expenditure of municipalities was more strongly related to wealth than after 1895. Expenditure includes public safety (including costs for drinking water) and other public measures (including measures concerned with infrastructure). Wealthier areas started earlier with all sorts of public measures than less wealthy areas. In the early 20th century, the less wealthy areas begin to catch up with the other areas, which weakened the association between wealth and expenditure on public measures (Knippenberg 1980).

Adding the change in the number of medical doctors per 100,000 inhabitants to the model made the association between tax and mortality decline

stronger. It could also be that the wealthiest areas already had sufficient medical doctors, and that less wealthy areas (with less rapid mortality declines) still had room for an increase in medical doctor density in the early 20th century.

8.5 Conclusion

This study has shown, on the basis of a multivariate analysis or regional data, that both cultural and economic determinants played important roles in mortality decline, but in different periods of time. The results suggest that the acceptance of new ideas was an important element of the cultural determinant. This element was predominantly related to infectious disease mortality. Fertility declines could only explain part of the association between cultural factors and mortality decline in the late 19th century. The economic determinant was also strongly related to infectious disease mortality decline.

Chapter 9

DETERMINANTS OF INFANT AND EARLY CHILDHOOD MORTALITY LEVELS AND DECLINE IN THE NETHERLANDS IN THE LATE NINETEENTH CENTURY

Abstract

Objective: *To study the relative importance of various determinants in total and cause-specific infant and early childhood mortality rates and decline in The Netherlands in the period 1875/79 to 1895/99.*

Data & Methods: *Mortality and population data were derived from publications of Statistics Netherlands for 16 towns and 11 rural areas. Mortality levels and decline were estimated on the basis of a Poisson-regression model. The associations of the estimated levels and declines, and determinants of infant and early childhood mortality were analysed using multivariate linear regression analysis. The causes of death studied were infectious diseases contributing largely to infant mortality (convulsions, acute digestive diseases, acute respiratory diseases) and early childhood mortality (brain diseases (i.e. predominantly encephalitis/meningitis), acute respiratory diseases, measles).*

Results: *Infant mortality rates were high in the southwestern part of The Netherlands in 1875/79. Due to fast declines in the western regions, this pattern changed into a north-south gradient in 1895/99. Early childhood mortality showed an urban-rural gradient in mortality levels in 1875/79. Mortality was high in towns. This gradient had largely disappeared in 1895/99, due to rapid declines in towns. Roman Catholicism and marital fertility were significantly associated with infant mortality in 1875/79 and 1895/99. The association of Roman Catholicism with infant mortality was stronger in 1895/99, because mortality declines were less rapid in Roman Catholic areas in 1875/79 to 1895/99. Urbanisation was significantly*

associated with early childhood mortality in 1875/79 and 1895/99. This association weakened over time, due to the rapid mortality declines in towns. Crowding (persons per dwelling) was significantly associated with early childhood mortality from acute respiratory diseases in 1875/79 and 1895/99, but not in mortality decline.
Conclusion: *Cultural factors (Roman Catholicism) were predominantly associated with infant mortality and diarrhoeal diseases. In case of early childhood mortality on the other hand, urbanisation played the most important role and was predominantly associated with acute respiratory diseases. So, different determinants of mortality (decline) play a role in both age groups, and they affect different causes of death. Therefore, infant and childhood mortality should be studied separately. It remains important that explanatory studies of mortality (decline) are carried out for different countries. Findings with respect to determinants of mortality (decline) for one country do not necessarily apply to other countries as well. The results for The Netherlands with respect to infant mortality differed from England and Wales.*

9.1 Introduction

Over the years a considerable amount of literature has been published on determinants of infant and childhood mortality. Infant and childhood mortality decline contributed largely to the early stages of the demographic and epidemiological transition that started in Western countries in the 18th or 19th century. Murray and Chen (1984) defined five sets of factors that can directly influence child mortality: maternal factors (e.g. age, parity), environmental contamination (e.g. food, water), nutrient deficiency (e.g. calories), injuries (accidental or intentional) and personal illness control (e.g. personal preventive measures). Most of these factors cannot be studied directly. Determinants of infant mortality that have been studied are, for example, income, marital fertility, illegitimacy, feeding practices, infant care, access to the health care system, housing conditions, female education, female employment, urbanisation (Among others: Kintner 1988b, Williams & Galley 1995, Graham 1994, Haines 1995, Huck 1995, Kok *et al.* 1997). It is difficult to assess the relative importance of all those determinants described in the literature, because many studies are descriptive, and the multivariate explanatory studies do not (and cannot) cover all determinants. Beside that, the importance of the different factors seems to differ by country. We will briefly describe a few important studies in the field of infant and childhood mortality (decline), that have contributed largely to the existing ideas on determinants of infant and child mortality.

Woods *et al.* (1988, 1989) published two papers on the causes of rapid infant mortality decline in England and Wales in the period 1861-1921. Infant mortality rates were high in 19th-century urban areas in England and Wales. This was called the 'urban-sanitary-diarrhoeal-effect'. This effect stands for higher

infant mortality in towns in hot summer in which, due to bad sanitary conditions, high mortality of diarrhoeal diseases occurred. The deteriorating effects of bad sanitary conditions on infant mortality in towns have also been published by others (Williams 1992, Williams & Galley 1995, Brandstrom 1988, Watterson 1988).

Woods *et al.* (1988, 1989) also concluded that social class and income did have significant effect on the *level* of infant mortality but not so much on the timing and pace of infant mortality decline. This conclusion has been confirmed by Haines (1995) in a multivariate explanatory analysis (including also urbanisation and fertility) for the period 1895-1911.

In late 19th and early 20th centuries Germany, the urban environment was associated with high infant mortality, but other factors played a more important role in infant mortality. Kintner (1988a,b) analysed German infant mortality using multivariate analyses and a large set of determinants of infant mortality (industrialisation, fertility, urbanisation, illegitimacy, Catholicism, breastfeeding practices, access to health care, access to piped water). It turned out that marital fertility explained most of the variation in infant mortality, followed by the variables illegitimacy and medical doctors density. That study is also one of the few historical studies in which data for breastfeeding practices were available.

In the United States, childhood mortality was among the highest of the world around 1900, while income levels were also among the highest. The inequality in childhood mortality by social class was much lower in the United States as compared to England and Wales. The most advantaged social classes enjoyed no advantage in mortality. On the basis of those findings Preston and Haines argued that 'lack of knowledge rather than lack of resources was principally responsible for foreshortening life' (Preston & Haines 1991). Another finding from Preston and Haines' work was that ethnicity was a more important factor in mortality than social class, urbanisation and income. The idea that cultural factors such as the diffusion of new ideas on disease processes and health behaviour have been important determinants of infant and childhood mortality (decline) has been put forward by many authors, and has been suggested as a new direction of research on infant and childhood mortality decline (Preston & Haines 1991, Ewbank & Preston 1990, Vögele 1994, Corsini & Viazzo 1997).

The aim of this study is to determine the relative importance of various determinants of all-cause and cause-specific infant and early childhood mortality rates and decline in The Netherlands in the period 1875/79 to 1895/99. Mortality started to decline rapidly in The Netherlands around 1875/79. The timing and pace of mortality decline differed between age groups and causes of death. Early childhood mortality declined stronger as compared to infant mortality decline (Wolleswinkel-van den Bosch *et al.* 1998). So far, only a few studies have analysed *cause-specific* mortality (declines). Analysis of cause-specific mortality can

further elucidate the relationship between determinants and mortality, because of the differences in aetiology between the cause-of-death groups. In this paper results will be presented for three important causes of infant and early childhood mortality. With respect to the determinants of mortality (decline), two types of determinants will be distinguished viz. determinants directly related to infant and early childhood mortality (proximate determinants, e.g. marital fertility), and determinants that acted, indirectly, through proximate determinants on infant and early childhood mortality (distal determinants, e.g. female labour participation, urbanisation). The relative importance of the determinants is analysed with a multivariate regression model, in which the regional variation in the prevalence of determinants is related to mortality levels and decline in 27 regions in The Netherlands. In the late nineteenth century, there was a large geographical variation in the prevalence of determinants in The Netherlands. These differences are used to elucidate the determinants of mortality (decline). The results for The Netherlands will be discussed in an international perspective.

9.2 Data and Method

9.2.1 Data

Mortality and population data

Total sex- and cause-specific infant (age 0-1) and early childhood mortality (age 1-4) data were obtained from publications of the Dutch Home Office (which provided data for quinquennial periods) for the years 1875/79, 1885/89 and 1895/99. Data on live births and population figures for the years 1875/79, 1885/89, 1895/99 were also obtained from publications of the Dutch Home Office. Data on live births were not available by sex. Mortality and population data as well as data on explanatory variables were obtained for the (by that time) 11 Dutch provinces and 16 towns (with more than 20,000 inhabitants) (figure 9.1). Figures for 11 rural areas were calculated by subtracting the values for the 16 towns from the 11 provinces in which they were situated.

Beside total mortality, three infectious diseases that contributed relatively largely to infant or early childhood mortality in the late 19th century were included in the study. In case of infant mortality, these causes of death were convulsions (about 15% contribution), acute respiratory diseases (10%) and acute digestive diseases (17%); in case of early childhood mortality these causes of death were acute respiratory diseases (about 25%), measles (5%), and brain diseases (10%). The category 'acute digestive diseases' did not include cholera, typhoid fever or abdominal tuberculosis, but other diarrhoeal diseases; the category 'acute respiratory diseases' did not include whooping cough, scarlet fever, measles, diphtheria, but mostly influenza and pneumonia. The category brain

diseases consisted predominantly of encephalitis and meningitis. One classification of causes of death consisting of 34 categories was used during the period 1875/79 to 1895/99, so no reclassification of cause-of-death categories was needed for this study (Wolleswinkel-van den Bosch 1996).

Determinants of mortality

Two types of determinants were used viz. determinants directly related to infant and early childhood mortality (proximate determinants), and determinants that acted, indirectly, through proximate determinants on infant and early childhood mortality (distal determinants). The proximate, direct determinants used in this study are persons per dwelling, percentage people with access to water supply system, medical doctor density, midwife density, and marital fertility. Wealth tax, female labour participation, urbanisation, soil type and Roman Catholicism are the distal, indirect, determinants in this study.

Persons per dwelling is a parameterisation of housing conditions, in particular overcrowding. Overcrowding was associated with bad sanitary conditions and lack of ventilation (Burnett 1991). Therefore, water- and foodborne as well as airborne diseases can be related to this determinant.

Marital fertility is an important determinant of infant mortality (Kintner 1988a,b). High parities and short birth intervals are related to high infant mortal-

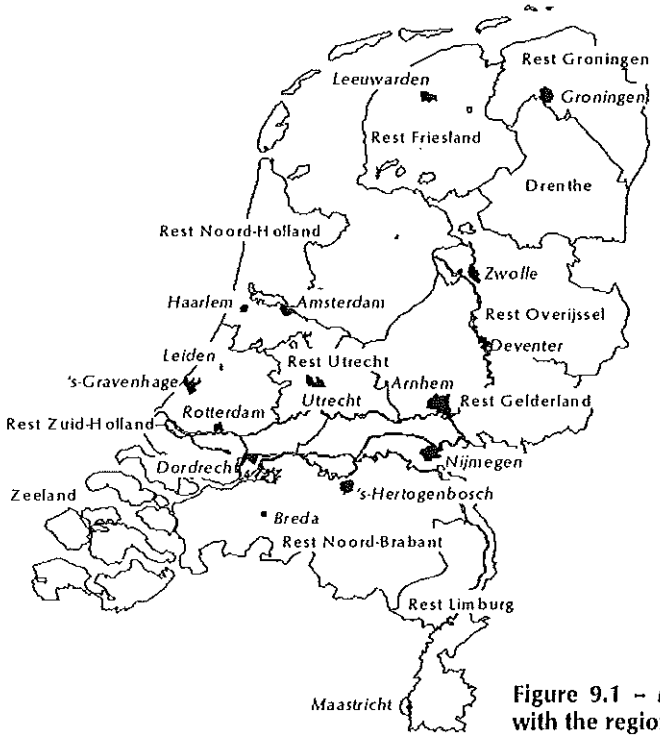


Figure 9.1 - Map of The Netherlands with the regions selected for this study.

ity rates (Van de Walle 1986). Marital fertility rates could affect other children in the family too, because longer birth intervals meant more care for, especially, the youngest child (Coale & Watkins 1986). According to Reves (1985), the major factor in infant and early childhood mortality decline in England and Wales was the alteration of the family size and age structure, leading to an increase of the mean age at infection for most infectious diseases, thereby lowering the case-fatality rate.

Access to clean drinking water will especially affect infant and early childhood mortality from waterborne diseases such as diarrhoeal diseases (acute digestive diseases). In the late 19th century, the construction of water supply systems was for a great deal carried out by private enterprises in The Netherlands. Whether piped water was available depended on the size and wealth of a town (Vogelzang 1956).

The role of medical doctors with respect to curative care was limited in the late 19th century. Medical doctors could, however, serve as intermediaries between the upper class and working class. In that way they could convey ideas on hygienic behaviour and child care, that had been accepted earlier by the upper class, to the working classes (Kunitz 1991). Beside that, medical doctors played an important role in the sanitary movement in The Netherlands in the 19th century (Houwaart 1991). In The Netherlands, the midwives were well trained, and mostly from low social classes. They were only allowed to attend normal pregnancies. There were large differences in the density of midwives. The midwife density was low in the provinces of Noord Brabant and Limburg as compared to, for example, the provinces of Noord- en Zuid-Holland (Marland 1995, Van Lieburg & Marland 1989).

Wealth tax is used as an indicator of wealth. Wealth can influence health by improvements at the household level, such as better food or housing, access to medical care, on the other hand wealth can also have a beneficial indirect effect on health by increased governmental expenditures on, for example, water supply and sewage systems. The wealth tax variable used in this study consisted of 6 components: tax on rental value of houses and other buildings, tax on the number of 'doors and windows connected to open air', tax on the number of fireplaces, tax on furniture, tax on servants and maids, and tax on horses. The tax on rental value (part of the wealth tax variable used in the analyses) differed according to the size of the town. Other components such as tax on horses or servants were not dependent of town size (Blok & De Meere 1978). Adding urbanisation to the multivariate regression models, however, partly corrects for the effect of differential tax values by town size.

Another determinant that is indirectly related to infant and early childhood mortality (decline) is female labour participation. The effect on mortality can either be positive or negative. Labour participation of the mother can in-

crease the level of wealth of the household by the input of a second wage. On the other hand, it could also mean less good care for the children such as no breastfeeding (or with shorter duration) for the infants (Brändström 1988, Graham 1994).

Cultural factors can influence mortality (decline) through determinants related to health behaviour (cf. chapter 8). In this paper, Roman Catholicism is used as a cultural factor. It has been reported that breastfeeding was less common among Roman Catholics as compared to other religious denominations (Van Poppel 1992). High marital fertility levels were also related to Roman Catholicism, there was a strong adherence to folk medicine among Roman Catholics, and they were less inclined to accept new ideas (Wichers 1965, Philips 1980, Rutten 1985).

In the introduction, the important role that urbanisation has played in the literature on child mortality has been pointed out. Urbanisation can be related to infant and early childhood mortality (decline) in various ways (positive and negative). The bad sanitary urban environment has mostly been related to infant mortality. High urban infant mortality rates have been related to the negative health affects of industrialisation (Huck 1994, 1995, Woods *et al.* 1988, 1989). In The Netherlands there was not such a strong urban-industrial complex as in, for example, England and Wales (Knippenberg 1988). The high population density facilitated the transmission of, predominantly, airborne infectious diseases. The urban society was more open to new ideas (including those on hygiene and childcare) as compared to rural areas. People with different socio-cultural backgrounds lived together in the cities, which facilitated the acceptance of other opinions on a number of things (Van der Woude *et al.* 1990). Other ways in which city life could affect health are the good infrastructure with respect to schooling and medical care as compared to rural areas.

A last determinant that could indirectly affect mortality is the type of soil. In The Netherlands, strong differences exist between regions with respect to soil type. Regions can be roughly divided into sandy regions and regions of mainly clay soil. The type of soil affected economic development of the region. The unfavourable sandy soil resulted in mixed farming (to spread risks) and the inhabitants were more self-supportive as compared to the clay areas. In the latter regions, the economy was more market-oriented (Jobse-van Putten 1990). The market-oriented regions were also the more open-minded regions. Apart from these economic and cultural associations with mortality (decline), the soil type could also have a direct association with mortality. Clay soil was more brackish than sand soil, which made groundwater less suitable as drinking water. As a result of that, the more polluted surface water was used in those areas for drinking and cleaning. This resulted in higher levels of waterborne infectious disease mortality (Hofstee 1981).

Table 9.1 gives the variables used in the analyses in this study, their parameterisation, years in the study and data source.

Table 9.1 – Distal and proximate determinants used in the analyses: parameterisation, data source and years.

	Parameterisation	Data Source	Years in analysis
Distal determinants			
Soil type	Region consists predominantly of sandy soil: yes/no	De Grote Bosatlas, 1968	1875/79, 1895/99
Urbanisation	Town yes/no	See figure 9.1	1875/79, 1895/99
Roman Catholicism	Percentage population with 'Roman Catholic' as religious affiliation	Historical Ecological Database (HED)*	1875/79, 1895/99
Wealth	Wealth tax in guilders per capita	HED	1875/79, 1895/99
Female labour participation	Percentage working women	HED	1875/79, 1895/99
Proximate determinants			
Marital fertility	Ig-index †	Calculated on the basis of figures derived from the HED and Lesthaeghe, 1977	1875/79, 1885/89, 1895/99
Housing conditions	Number of persons per dwelling	Bijdragen tot de Statistiek van Nederland (CBS, 1879, 1889, 1899)	1875, 1889, 1899
Clean drinking water	Percentage population with access to piped water	De Grootte, 1995	1879, 1889, 1899
Medical care	Number of medical doctors per 100,000 population	Verslagen aan de koning van de bevindingen en handelingen van het geneeskundig staatstoezicht in het jaar 1875-1899	1875/79, 1885/89, 1895/99
	Number of midwives per 1,000 women aged 15-49	Idem	

* The Historical-Ecological Database is managed by Stichting Beleidsondersteunend Ruimtelijk Onderzoek (BRON), which is related to the University of Amsterdam.

† The Ig-index is the age-standardised ratio of the annual number of legitimate births occurring to married women of childbearing age (15-49) that would occur if they were subject to natural fertility (i.e. age-specific marital fertility rates of the Hutterite population) (Lesthaeghe 1977).

9.2.2 Methods

The mortality level and decline were estimated on the basis of a log-linear regression model (Poisson regression) designed to estimate mortality levels for the years 1875/79 and 1895/99, and mortality decline in the period 1875/79 to 1895/99. Separate models were used for infant mortality and early childhood mortality. The model used is described below.

$$E(Y_{irt}) = N_{irt} e^{\alpha+\beta_i+\gamma_r+(\delta+\delta_r)T}$$

$E(Y_{irt})$= expected number of deaths per sex category (i), region (r) and quinquennium (t)
N_{irt} = population numbers per sex category, region and quinquennium
α = intercept (i.e. log regional mortality rate 1875/79 men, region Amsterdam)
β_i = log relative risk sex-category i (men reference category)
γ_r = log relative risk regional mortality rate (Amsterdam reference category)
δ = log annual mortality decline (region Amsterdam)
δ_r = difference in annual mortality decline between region$_r$ and Amsterdam
T = year since 1875/79 (0 for 1875/79, 10 for 1885/89, 20 for 1895/99)

Regional mortality rate in 1875/79 for men = $e^{\alpha+\gamma_r}$
Percentage regional mortality decline per decade = $(1 - e^{(\delta+\delta_r).10})*100$

In case of infant mortality the population numbers were live births, and no distinction between sex groups could be made. In case of early childhood mortality the population numbers were the number of children aged 1-4 years by sex group. The estimates for the logarithm of infant and early childhood mortality levels in the 27 regions for the quinquennia 1875/79 ($\alpha+\gamma_r$) and 1895/99 ($(\alpha+\gamma_r)+(\delta+\delta_r).20$) and the estimates for the logarithm of mortality decline in the period 1875/79 to 1895/99 ($\delta+\delta_r$) were used in a multivariate linear regression analysis in order to relate those estimates to determinants of mortality (decline).

The multivariate regression analyses were conducted for two different levels of determinants of mortality: determinants indirectly related to mortality (decline) (distal determinants), and determinants directly related to mortality (decline) (proximate determinants). In the analyses of the distal determinants of mortality, urbanisation, Roman Catholicism, wealth tax, soil type and female labour participation were put together in a model. The proximate determinants were analysed separately, each direct determinant corrected for a set of basic and distal determinants (urbanisation, wealth tax, Roman Catholicism, and soil type). In the analyses concerning infant and early childhood mortality decline, absolute changes in marital fertility, persons per dwelling, access to piped water and medical doctor or midwife density were associated with mortality decline.

In order to determine whether the regional mortality rates and declines differed significantly, we performed a likelihood ratio test; all tests were significant. To present significant differences in rates and decline from Amsterdam for individual regions (i.e. γ_r and δ_r differs significantly from zero), results of T-tests will be presented.

9.3 Results

9.3.1 Regional variation in infant and early childhood mortality levels and declines

Figure 9.2a shows maps for The Netherlands for the years 1875/79 and 1895/99 with regional differences in infant mortality levels. In table 9.2 figures for the different towns and rural areas are given. In 1875/79, *infant mortality* rates were higher in the southwest of The Netherlands as compared to the northeast. In the southwestern part towns do not always show higher mortality rates as compared to the surrounding rural areas; in the northeastern part mortality rates are mostly higher in towns. In 1895/99, the highest infant mortality rates were found in the rural areas and towns of Zuid-Holland, Zeeland, Noord-Brabant and Limburg. The southwest and northeast difference between higher and lower infant mortality that was apparent in 1875/79 has rather become a south-north difference in 1895/99.

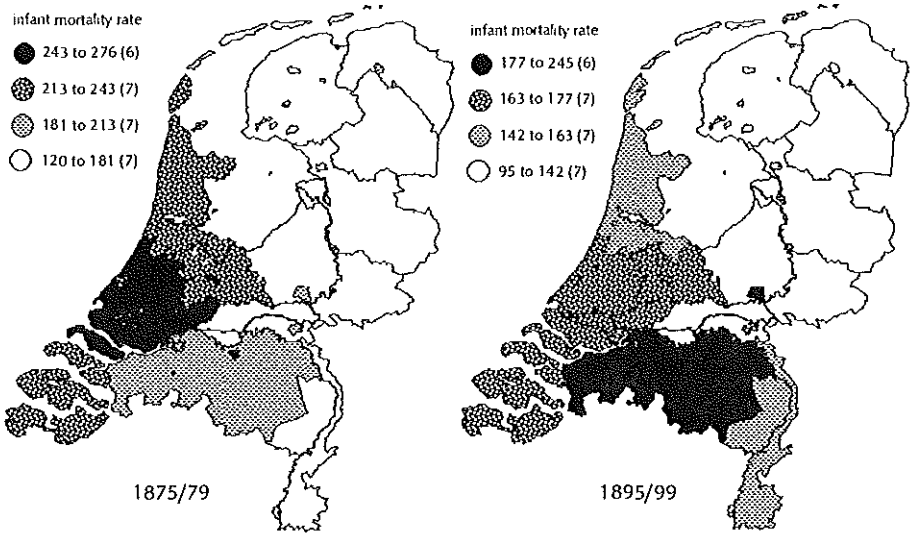


Figure 9.2a – Regional differences in infant mortality levels, 1875/79 and 1895/99.

Table 9.2 – Poisson regression estimates of infant mortality (per 1,000 live births) and early childhood mortality (per 1,000 persons-years) for males* in 1875/79 and 1895/99, and percentage infant and early childhood mortality decline per decade in the period 1875/79 to 1895/99.

	Infant mortality			Early childhood mortality		
	rate 1875/79	rate 1895/99	% decline per decade	rate 1875/79	rate 1895/99	% decline per decade
Towns						
Amsterdam	216	152	16	44	23	27
Arnhem	186	183	1	30	23 ns	12
Breda	250	225	4	39	22 ns	25 ns
Deventer	187	170	5	40	23 ns	23 ns
Dordrecht	256	155 ns	22	56	19	42
Groningen	197	119	22	36	19	27 ns
Haarlem	212 ns	150 ns	16 ns	31	20	19
's-Hertogenbosch	243	244	0	41 ns	30	14
Leeuwarden	167	115	17 ns	34	19	25 ns
Leiden	253	147 ns	23	56	19	41
Maastricht	215 ns	245	+7	51	24 ns	32
Nijmegen	204 ns	171	8	42 ns	23 ns	26 ns
Rotterdam	232	177	12	44 ns	21	30
's-Gravenhage	237	166	16 ns	31	20	19
Utrecht	248	163	19	40	22	26 ns
Zwolle	181	147 ns	10	45 ns	26	24 ns
Unweighted average towns	218	171	12	41	23	26
Rural areas of provinces						
Friesland	137	95	16 ns	27	14	26 ns
Groningen	135	110	9	25	16	21
Drenthe	120	111	4	29	17	16
Overijssel	137	124	5	33	21	20
Gelderland	146	137	3	26	19	14
Utrecht	224	173	12	27	20	13
Noord-Holland	213 ns	142	18	27	19	16
Zuid-Holland	276	170	21	34	20	22
Zeeland	219	165	13	25	14	25
Noord-Brabant	204	196	2	25	20	10
Limburg	146	159	+5	24	17	17
Unweighted average rural areas	178	144	9	27	18	18
Observed for The Netherlands	197	153	12	30	19	23

* Only the figures for males are given in this table. Due to the nature of the model the relative differences between regions are the same for males and females

\+ means increase in mortality

ns means that the rate or % decline did not differ significantly from the value for Amsterdam ($p<0.05$)

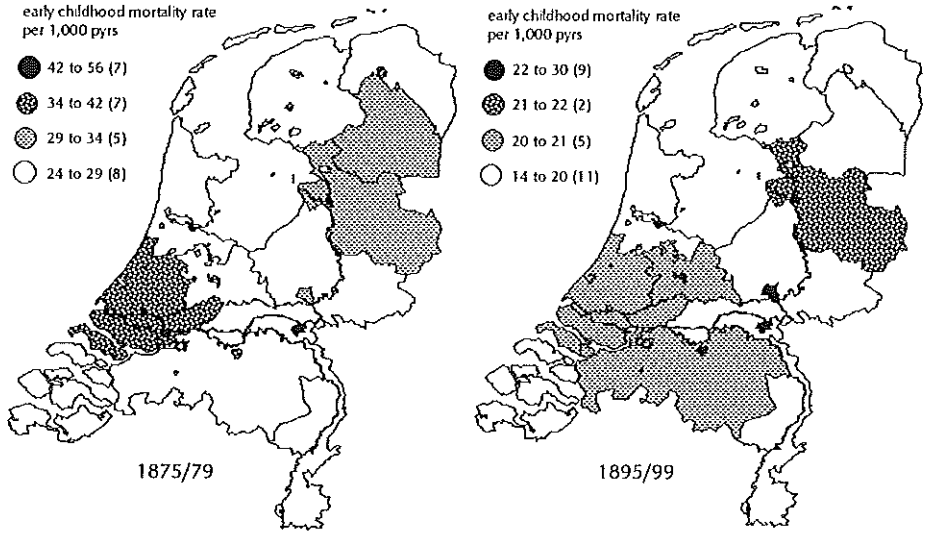


Figure 9.2b – Regional differences in early childhood mortality levels, 1875/79 and 1895/99.

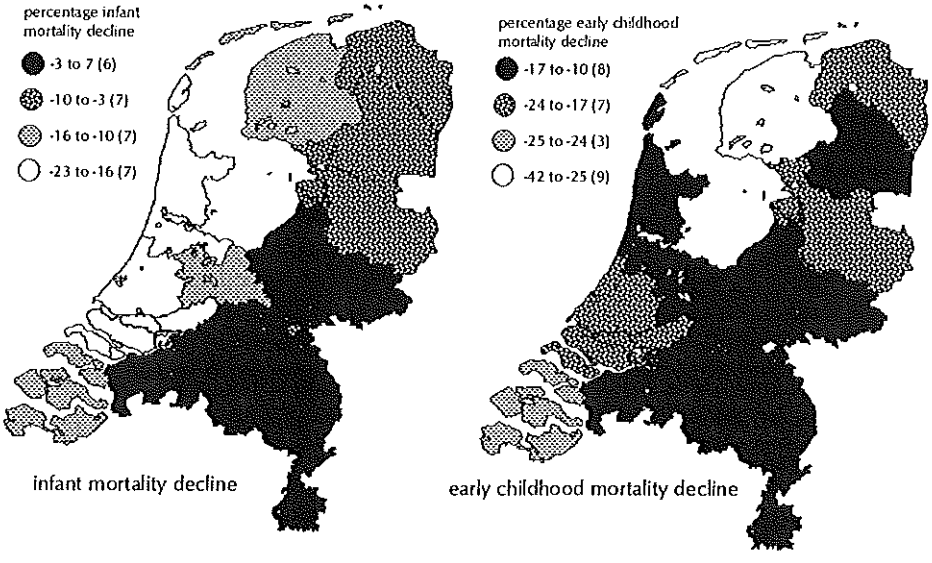


Figure 9.2c – Regional differences in infant and early childhood mortality decline, 1875/79 to 1895/99.

With respect to *early childhood* mortality levels (figure 9.2b), there is no clear division between southwest and northeast in 1875/79 as was found for infant mortality levels. The geographical pattern for early childhood mortality shows a clear urban-rural gradient. Early childhood mortality levels were higher in towns as compared to the surrounding rural areas. In towns mortality ranged from 30 to 56 per 1000 person years; in rural areas mortality ranged from 24 to 34 per 1000 person years. In 1895/99, this urban-rural gradient had for a large part disappeared.

Infant mortality declines were slower in the period 1875/79 to 1895/99 as compared to early childhood mortality declines (table 9.2). In the rural areas of Limburg and the city of Maastricht infant mortality even increased in this period. As far as infant mortality is concerned, there was more a difference in western versus eastern provinces as compared to a rural-urban difference (figure 9.2c). *Early childhood mortality declines* were especially strong in the southwestern towns as compared to their surroundings (figure 9.2c).

Infant mortality rates in 1875/79 and 1895/99 were strongly related. The same holds for early childhood mortality in both years. High infant mortality rates in 1875/79 were associated with rapid infant mortality declines, albeit less strong than the association between early childhood mortality rates in 1875/79 and the subsequent mortality decline. Regions with rapid infant mortality declines were mostly regions with rapid early childhood mortality declines too (table 9.3).

Table 9.3 – Pearson's correlation coefficients between infant and early childhood mortality levels and declines in 1875/79, 1895/99 and the period 1875/79 to 1895/99.

	Infant mortality 1875/79	Infant mortality 1895/99	Infant mortality decline	Child mortality 1875/79	Child mortality 1895/99	Child mortality decline
Infant mortality 1875/79	1.00	0.60†	-0.40†	0.52‡	0.36	-0.37
Infant mortality 1895/99		1.00	0.40‡	0.32	0.69‡	0.06
Infant mortality decline			1.00	-0.30	+0.33	0.50‡
Child mortality 1875/79				1.00	0.60‡	-0.76‡
Child mortality 1895/99					1.00	0.07
Child mortality decline						1.00

† means significance at the 95% level, and ‡ at the 99% level.
- means that a high mortality level is associated with a low mortality level, or that a high mortality level is correlated with (rapid) mortality decline.

9.3.2 Multivariate analyses of the association between determinants and (cause-specific) mortality

Distal determinants of mortality levels and decline

In tables 9.4a and b, results of the multivariate analyses of the distal determinants that could affect infant and early childhood mortality (decline) are presented. The

Table 9.4a – Multivariate analyses of level of distal determinants and (cause-specific) infant mortality levels and decline.

	All causes	Con-vulsions	Acute respiratory diseases	Acute digestive diseases
Distal determinants 1875/79	***Percentage change of infant mortality 1875/79 per unit difference in the independent variables***			
Sand soil *(sand vs. clay)*	-18%	+4%	-11%	-57%
Urbanisation *(town vs. rural area)*	2%	23%	15%	-52%
% Roman Catholics *(10 percent point higher)*	+3%†	+17%‡	-0%	+13%†
Wealth tax *(1 guilder per capita higher)*	+8%	-2%	-4%	+44%†
% Female labour participation *(1 percent point higher)*	-1%	-4%†	+1%	-2%
R^2	0.50	0.68	0.31	0.41
Distal determinants 1895/99	***Percentage change of infant mortality 1895/99 per unit difference in the independent variables***			
Sand soil *(sand vs. clay)*	-8%	+23%	-2%	-18%
Urbanisation *(town vs.rural area)*	-0%	42%†	4%	-29%
% Roman Catholics *(10 percent point higher)*	+7%‡	+18%‡	+2%	+14%‡
Wealth tax *(1 guilder per capita higher)*	+6%	-8%	+1%	+29%
% Female labour participation *(1 percent point higher)*	+0%	-4%	-0%	-1%
R^2	0.68	0.74	0.17	0.49
Distal determinants 1875/79	***Change of percentage infant mortality decline per decade in 1875/79 to 1895/99 per unit difference in the independent variables (in percent points)***			
Sand soil *(sand vs. clay)*	-6	-8	-6	-33†
Urbanisation *(town vs.rural area)*	+1	-17	+8	-6
% Roman Catholics *(10 percent point higher)*	-2‡	-1	-1	-0
Wealth tax *(1 guilder per capita higher)*	+1	+3	-4	+4
% Female labour participation *(1 percent point higher)*	-0	-0	+1	-0
R^2	0.58	0.25	0.30	0.34

† means significance at the 95%-level, ‡ means significance at the 99%-level.
+ means an increase in mortality level, or a more rapid mortality decline
- means a decrease in mortality level, or a less rapid mortality decline

variables soil type urbanisation, Roman Catholicism, wealth tax, and female labour participation have been analysed together in the regression model.

With respect to *infant mortality* (table 9.4a), Roman Catholicism turned out to be significantly associated with higher infant mortality rates in 1875/79, and this association became stronger in 1895/99. Predominantly infant mortality

Table 9.4b – Multivariate analyses of level of distal determinants and (cause-specific) early childhood mortality levels and decline.

	All causes	Measles	Brain diseases (encephalitis/ meningitis)	Acute respiratory diseases
Distal determinants 1875/79	***Percentage change of early childhood mortality 1875/79 per unit difference in the independent variables***			
Sand soil *(sand vs. clay)*	-1%	+10%	-11%	-14%
Urbanisation *(town vs. rural area)*	42%‡	74%‡	61%‡	39%‡
% Roman Catholics *(10 percent point higher)*	-1%	-6%	-4%	+1%
Wealth tax *(1 guilder per capita higher)*	-8%	-4%	-8%	-7%
% Female labour participation *(1 percent point higher)*	+0%	-5%†	-0%	+0%
R^2	0.66	0.71	0.75	0.68
Distal determinants 1895/99	***Percentage change of early childhood mortality 1895/99 per unit difference in the independent variables***			
Sand soil *(sand vs. clay)*	+4%	-15%	-23%‡	+10%
Urbanisation *(town vs.rural area)*	21%†	47%	45%‡	15%
% Roman Catholics *(10 percent point higher)*	+4%†	-15%‡	+0%	+6%‡
Wealth tax *(1 guilder per capita higher)*	-0%	-42%†	-1%	+14%
% Female labour participation *(1 percent point higher)*	-1%	+4%	-0%	-2%
R^2	0.53	0.41	0.81	0.52
Distal determinants 1875/79	***Change of percentage early childhood mortality decline per decade in 1875/79 to 1895/99 per unit difference in the independent variables (in percent points)***			
Sand soil *(sand vs. clay)*	-4	-7	+3	-12†
Urbanisation *(town vs.rural area)*	+15†	+38†	-16	+17†
% Roman Catholics *(10 percent point higher)*	-2†	+5	-2†	-2†
Wealth tax *(1 guilder per capita higher)*	-4	-2	-2	-8†
% Female labour participation *(1 percent point higher)*	+1	-2	-0	+1
R^2	0.47	0.45	0.33	0.57

† means significance at the 95%-level, ‡ means significance at the 99%-level.
+ means an increase in mortality level, or a more rapid mortality decline
- means a decrease in mortality level, or a less rapid mortality decline

from convulsions and acute digestive diseases were associated with Roman Catholicism in both years. The stronger association of Roman Catholicism and high infant mortality levels in 1895/99 could be explained from the less rapid declines (or increases) in infant mortality in Roman Catholic regions in the preceding decades. No such significant association between infant mortality decline and Roman Catholicism could, however, be found for convulsions and acute digestive diseases. Urbanisation was not significantly associated with high infant mortality from all-causes in 1875/79. However, infant mortality from convulsions was significantly higher in cities than in rural areas in 1895/99. Declines in infant mortality from convulsions were less fast in the cities in the period 1875/79 to 1895/99, albeit that this association was not significant.

The variance explained by the variables in the model was larger for infant mortality levels in 1895/99 as compared to 1875/79 (except for acute respiratory diseases), and was the largest for infant mortality from convulsions in both years. In general, the variance explained was larger in case of levels of infant mortality than in case of declines.

With respect to *early childhood mortality* (table 9.4b), urbanisation was significantly associated with mortality from all-causes, measles, brain diseases (i.e. mainly encephalitis/meningitis) and acute respiratory diseases. Rural life was healthier than urban life in 1875/79, and in 1895/99. In 1895/99 the association was, however, only significant for all-cause mortality and mortality from brain disease. In general, early childhood mortality declined significantly faster in cities as compared to rural areas in the late nineteenth century, except for brain diseases. Early childhood mortality declined less rapidly in Roman Catholic areas, just like infant mortality declines. This resulted in positive associations between early childhood mortality from all causes and from acute respiratory diseases and Roman Catholicism.

The variance explained by the variables in the model for early childhood mortality was higher in 1875/79 than in 1895/99. The variables in the model explained most of the variance in early childhood mortality from brain diseases. As far as early childhood mortality declines are concerned, the variables explained best the variance in acute respiratory disease mortality. In general, the variance in early childhood mortality decline was better explained than the variance in infant mortality decline.

Proximate determinants of mortality levels and decline

The relationship of proximate determinants and mortality levels and decline was analysed, corrected for possibly confounding factors: soil type, urbanisation, Roman Catholicism, and wealth tax (tables 9.5a, b). High marital fertility rates were strongly associated with high *infant mortality* levels in 1875/79, and, although less strongly so, in 1895/99. This association was especially strong for acute di-

Table 9.5a – Multivariate regression analyses of the level of proximate determinants and (cause-specific) infant mortality (1875/79 and 1895/99), and the absolute change of proximate determinants and infant mortality decline (1875/79 to 1895/99).

	All causes	Con-vulsions	Acute respiratory diseases	Acute digestive diseases
Proximate determinants 1875/79	*Percentage change of infant mortality 1875/79 per unit difference in the independent variables*			
Marital fertility *(Ig-index 0.1 higher)*	+23%‡	+1%	-5%	+99%‡
Persons per dwelling *(1 person more)*	-1%	+8%†	+1%	-3%
% Access to piped water *(1 percent point higher)*	+0%	+1%‡	+0%	-0%
Medical doctor density *(1 doctor per 100,000 more)*	+1%‡	+0%	+0%	+2%
Midwives density *(1 midwife per 1,000 more)*	-5%	+3%	-2%	-0%
Proximate determinants 1895/99	*Percentage change of infant mortality 1895/99 per unit difference in the independent variables*			
Marital fertility *(Ig-index 0.1 higher)*	+12%†	-7%	+1%	+35%†
Persons per dwelling *(1 person more)*	+1%	+3%	+4%†	+1%
% Access to piped water *(1 percent point higher)*	+0%	+0%	+0%	+0%
Medical doctor density *(1 doctor per 100,000 more)*	+0%	-1%	+1%	+2%
Midwives density *(1 midwife per 1,000 more)*	+5%	+39%	+9%	+21%
Absolute change in proximate determinants 1875/79 to 1895/99	*Change of percentage infant mortality decline per decade in 1875/79 to 1895/99 per unit difference in the independent variables (in percent points)*			
Marital fertility *(Ig-index increase with 0.1 per decade)*	-8	+4	-7	-31
Persons per dwelling *(increase no. of persons with 1 per decade)*	+1	-1	+7	+6
% Access to piped water *(increase with 1 percent point per decade)*	+0	-0	+0	+0
Medical doctor density *(increase doctors per 100,000 with 1 per decade)*	-7†	+5	+1	-6
Midwives density *(increase midwives per 1,000 with 1 per decade)*	-3†	+0	+1	-1

Confounding variables of the proximate determinants: urbanisation, soil type, wealth tax, Roman Catholicism.

† means significance at the 95%-level, ‡ means significance at the 99%-level.

\+ means an increase in mortality level, or a more rapid mortality decline

\- means a decrease in mortality level, or a less rapid mortality decline

Table 9.5b – Multivariate regression analyses of the level of proximate determinants and (cause-specific) early childhood mortality (1875/79 and 1895/99), and the absolute change of proximate determinants and early childhood mortality decline (1875/79 to 1895/99).

	All causes	Measles	Brain diseases ((encephalitis/ meningitis)	Acute respiratory diseases
Proximate determinants 1875/79	***Percentage change of early childhood mortality 1875/79 per unit difference in the independent variables***			
Marital fertility *(Ig-index 0.1 higher)*	+7%	+24%	+7%	+2%
Persons per dwelling *(1 person more)*	+3%	-5%	+3%	+4%†
% Access to piped water *(1 percent point higher)*	+0%	-0%	+0%	+0.3%†
Medical doctor density *(1 doctor per 100,000 more)*	+0%	+1%	+0%	+0%
Midwives density *(1 midwife per 1,000 more)*	n.a.	n.a.	n.a.	n.a.
Proximate determinants 1895/99	***Percentage change of early childhood mortality 1895/99 per unit difference in the independent variables***			
Marital fertility *(Ig-index 0.1 higher)*	+12%†	+48%	-2%	+15%
Persons per dwelling *(1 person more)*	+2%	-1%	+1%	+6%†
% Access to piped water *(1 percent point higher)*	+0%	-0%	+0%	+0%
Medical doctor density *(1 doctor per 100,000 more)*	+0%	+1%	-0%	-0%
Midwives density *(1 midwife per 1,000 more)*	n.a.	n.a.	n.a.	n.a.
Absolute change in proximate determinants 1875/79 to 1895/99	***Change of percentage early childhood mortality decline per decade in 1875/79 to 1895/99 per unit difference in the independent variables (in percent points)***			
Marital fertility *(Ig-index increase with 0.1 per decade)*	+2	-2	+10	-5
Persons per dwelling *(increase no. of persons with 1 per decade)*	-11	+20	-2	-2
% Access to piped water *(increase with 1 percent point per decade)*	+0	+1	-0	-0
Medical doctor density *(increase doctors per 100,000 with 1 per decade)*	-10†	-12	+3	-8
Midwives density *(increase midwives per 1,000 with 1 per decade)*	n.a.	n.a.	n.a.	n.a.

Confounding variables of the proximate determinants: urbanisation, soil type, wealth tax, Roman Catholicism.

† means significance at the 95%-level, ‡ means significance at the 99%-level.

+ means an increase in mortality level, or a more rapid mortality decline

- means a decrease in mortality level, or a less rapid mortality decline

gestive disease mortality in 1875/79, a cause of death very common among infants. In case of convulsions, another common cause of death among infants, the association with marital fertility was, however, not present. As far as change in the proximate determinants and infant mortality decline was concerned, only significant associations were found with the change in medical doctor density and midwives density. These associations were, however, not as expected: an increase in medical doctor or midwives density was associated with an increase of infant mortality (table 9.5a).

In 1875/79 and 1895/99, a relatively large number of persons per dwelling was significantly associated with high levels of *early childhood mortality* from acute respiratory diseases. In 1895/99, marital fertility was, just like infant mortality, significantly associated with early childhood mortality levels. With respect to early childhood mortality decline only the change in medical doctors density was significantly associated with mortality change. The association was, however, not as expected (table 9.5b).

The variance explained in infant mortality from all causes and acute digestive diseases, was relatively high in the models with marital fertility for 1875/79 and 1895/99 (R^2 = 0.70 and 0.75 respectively for all-causes, 0.61 and 0.58 for acute digestive diseases). In case of early childhood mortality levels, the variance explained was relatively high for the model with marital fertility in 1895/99 (R^2 = 0.60), and, in case of acute respiratory diseases, for the model with the number of persons per dwelling in 1875/79 and 1895/99 (R^2 = 0.74 and 0.58 respectively).

9.4 Discussion

In the late 19th century, Roman Catholicism was significantly associated with all-cause infant mortality levels and decline; urbanisation was most significantly associated with early childhood mortality levels and decline. As far as more proximate variables are concerned, marital fertility was significantly associated with infant and early childhood mortality in The Netherlands in 1875/79 and 1895/99. The number of persons per dwelling, another proximate determinant, was predominantly associated with early childhood mortality from acute respiratory diseases. After a discussion of the data and method used in this study, the role of the different determinants in infant and early childhood mortality (decline) are discussed and the results for The Netherlands are compared to findings for other countries.

9.4.1 Data and Methods

Cause-specific mortality data

In this study, specific causes of death (acute digestive diseases, acute respiratory diseases, convulsions, brain diseases (i.e. mainly meningitis/encephalitis) and measles) were studied beside all-cause infant and early childhood mortality. The reliability of historical cause-specific mortality data has been the subject of much debate. As far as the causes of death studied in this paper are concerned, 'convulsions' is not a clearly defined cause of death. Convulsions might be a symptom of a variety of diseases with fever (Hardy 1988). Others have argued that the category 'convulsions' mainly covered diarrhoeal diseases with cramps (Kintner 1986).

In case of acute respiratory diseases, exchanges with tuberculosis and chronic respiratory disease have been suggested by several authors (Evers 1882, Saltet 1909, Van Vollenhoven 1889). We do not consider such exchanges a big problem in this study, because we only studied age groups 0 and 1-4. This makes exchanges with chronic respiratory disease and tuberculosis, causes of death that occurred mostly at adult ages, less likely.

With respect to acute digestive diseases, exchanges with the vague 19th-century cause-of-death category 'debility' have been reported in a Dutch article on health in the city (Onnen 1895). Onnen presented mortality figures that showed considerable swings between the quinquennia 1880/85 and 1885/90 for acute and chronic digestive diseases combined in one direction and for 'debility' in the other direction. The city 'Maastricht', for example, recorded 130 deaths from debility in 1880/85 and 444 deaths from digestive diseases in that year, while in 1885/90 591 deaths from debility were recorded and only 89 deaths from digestive diseases. This suggests that in case of acute digestive diseases regional differences in coding or diagnosis existed in the late 19th century. Therefore, it should be kept in mind that part of the regional variation in the estimates of mortality from acute digestive diseases in this study could be caused by differences in coding and classification. The associations between infant mortality from acute digestive diseases and Roman Catholicism and marital fertility are, however, so strong that it is not likely that the incorrect coding could explain the complete association.

Regression models

The analysis of determinants of infant and early childhood mortality levels and decline was conducted in two steps. First, regional mortality levels and declines were estimated using Poisson-regression models. Secondly, those estimates were related to a set of independent variables in a multivariate linear regression analysis. By using the estimates of mortality decline in the multivariate regression analysis, the estimated declines are used as point-estimates. Information on the

accuracy of the estimates of regional mortality levels and declines is lost during the second step of the analysis. In order to determine whether residual variation in the estimates was large, the average variation around the point-estimates was expressed as a percentage of the variation between the point-estimates. Residual variation was not sizeable in most cases. Considerable residual variation was present in case of infant mortality from acute respiratory diseases in 1875/79 and 1895/99, for early childhood mortality from diphtheria in 1875/79 and 1895/99, and in case of early childhood mortality from acute respiratory diseases and measles in 1895/99.

If variables in a multivariate model are strongly correlated we cannot distinguish their separate associations with the dependent variable i.c. mortality rate or decline. This is known as the multicollinearity problem. The variance-covariance matrices of the parameters of the models used in this study were calculated (Norusis 1990). Relatively high correlations were found for urbanisation and wealth tax, and for marital fertility level and Roman Catholicism (both correlations around 0.70) in 1875/79 and 1895/99. Change in marital fertility was not strongly correlated with Roman Catholicism.

The number of independent variables that can be analysed in the regression models is limited by the number of objects in the analysis. In this study the 27 regions are the objects of analysis. In order to get valid estimates of the parameters the number of independent variables should not exceed the square root of the objects in the analysis (being 5 independent variables in the analyses presented in this study).

In the analyses of the change in proximate determinants and mortality decline, the results for absolute change in the determinant were presented. Using relative changes did not alter the results so, that other proximate determinants came to the fore as important determinants of infant and early childhood mortality decline.

9.4.2 Determinants of infant and early childhood mortality levels and decline

Roman Catholicism and marital fertility

Roman Catholicism and marital fertility were important determinants of infant mortality (decline) in The Netherlands in 1875/79 and 1895/99 (tables 9.4a and 9.5a). The importance of marital fertility for the level of infant mortality was also found in late 19th and early 20th centuries Germany (Kintner 1988a,b). The association of Roman Catholicism and infant mortality became stronger over time, because infant mortality declined less rapidly in Roman Catholic regions in the period 1875/79 to 1895/99. Marital fertility did not seem to play a large role in the explanation of this slow decline in Roman Catholic regions. The change in marital fertility was not significantly associated with infant mortality decline (ta-

ble 9.5a). High marital fertility rates were, however, not the only characteristics of the Roman Catholic population in The Netherlands. Roman Catholics were less inclined to accept new ideas on disease processes and hygiene as compared to Protestants, in The Netherlands (Philips 1980). The Roman Catholic population (particularly those living in rural areas) has been described as strongly obedient to authorities, living within the bonds set by the Roman Catholic clergy (Wichers 1965). Related to these characteristics are the strong adherence to folk medicine (Philips 1980, Rutten 1985), and a lesser tendency to breastfeed infants (which is also related to high fertility rates) (Van Poppel 1992).

Convulsions and acute digestive diseases were the causes of death most strongly related to Roman Catholicism. Acute digestive disease mortality was strongly related to marital fertility too, but convulsions was not. The association of infant mortality from acute digestive diseases and marital fertility might be related to a common determinant viz. breastfeeding. Breastfeeding is related to longer birth intervals, and it provides adequate nutrition for the infant (sterile food, immunisation) (Huffman 1984, Palloni & Millman 1986). There are however no data on breastfeeding available for the regions in this study.

It might be that the largest part of the category of convulsions consisted of other diseases with fever than of acute digestive diseases with cramps, and that therefore no association with marital fertility was found. Overcrowding was, for example, related to high levels of infant mortality from convulsions (table 9.5a). This suggests that probably a relatively large part of the category 'convulsions' consisted of airborne instead of water-and food borne infectious diseases.

In 1895/99, Roman Catholicism and marital fertility turned out to be significant factors for early childhood mortality as well (Table 9.4b and 9.5b) Reductions in fertility can affect early childhood mortality as well, because a reduction in family size will improve care and resources for the older children (Coale & Watkins 1986).

Urbanisation

There have been many publications on the deteriorating effects of the urban environment on infant mortality. Infant mortality levels would especially increase by the bad sanitary conditions in the towns. In summer, these sanitary conditions would lead to high mortality from diarrhoeal diseases in urban regions (the urban-sanitary-diarrhoeal-effect). This has been described for England and Wales, but also for other European countries such as France and Sweden (Preston & Van de Walle 1978, Nelson & Rogers 1994).

In The Netherlands, however, urbanisation was not a significant factor in *infant* mortality in the late 19th century, and acute digestive disease mortality was even lower in towns as compared to rural areas (cf. Table 9.4a). Roman Catholicism and marital fertility were important determinants of infant mortality instead.

Comparable findings were reported for the Dutch neighbouring country Germany by Kintner (1988a). She pointed out that marital fertility was a far more important factor in 19th century infant mortality than urbanisation. Others also reported that, in the case of Germany, there was no clear urban-rural difference in infant mortality levels, but rather a regional difference (Vögele 1994). The Dutch situation with respect to urbanisation and infant mortality was more comparable to Germany than to England and Wales. Infant mortality from convulsions in 1895/99 was, however, higher in towns as compared to rural areas. The analysis of infant mortality decline century suggests that this was caused by the slow decline of infant mortality from convulsions in towns in the period 1875/79 to 1895/99 (table 9.4a).

Early childhood mortality, on the other hand, was significantly higher in towns. In 1875/79, measles mortality, mortality from encephalitis/meningitis, and acute respiratory diseases were all significantly higher in towns as compared to rural areas. Early childhood mortality from acute digestive diseases (results not shown) was also higher in towns, but this association was not significant. The deteriorating effect of urban life on early childhood health in The Netherlands seemed to act predominantly through airborne infectious diseases instead of acute digestive diseases.

In 1895/99, the urban-rural difference for early childhood mortality had diminished. Mortality declined more rapidly in towns in the period 1875/79 to 1895/99, so rural and urban mortality levels converged, except for early childhood mortality from brain diseases (table 9.4b). This convergence of urban and rural mortality is in concordance with findings for England and Wales and the United States during comparable periods of time (Watterson 1988, Preston & Haines 1991). In the United States, mortality decline was most rapid in the, by then, ten largest cities, and has been related to the earlier introduction of all kinds of public health measures in those cities (Preston & Haines 1991). Simple correlations from another study for The Netherlands, showed that towns which spent a relatively large amount of money on water supply and sewerage facilities had relatively strong mortality declines (Swartsenburg 1981).

Other determinants related to specific causes of death

Roman Catholicism, urbanisation and marital fertility were, in the analyses presented in this paper, significantly associated with all-cause infant and early childhood mortality (decline) and with some specific causes of death as well. Some other determinants, such as wealth tax, female employment, and crowding were only significantly related to specific causes of death, and not to all-cause infant or early childhood mortality.

Research for England and Wales showed that income was an important determinant of levels of infant mortality, but that it was less important in case of

infant mortality decline (Woods *et al.* 1988, Haines 1995). In the analyses presented in this paper, *wealth tax* was significantly associated with infant mortality from acute digestive diseases in 1875/79, but the association was not as expected (high tax levels were associated with high mortality rates). In the case of early childhood mortality, however, high wealth tax levels (i.e. wealthy areas) were associated with significant lower mortality rates from measles in 1895/99. Measles is a cause of death strongly related to living standards, especially nutrition, and probably also overcrowding (Hardy 1993, Aaby 1984). Like the results for England and Wales, we did not find a significant association for infant or early childhood mortality *decline* and wealth. Another study for The Netherlands has shown that wealth tax did not play a role in all-cause mortality decline in the late 19th century, but it did play a role in the early 20th century (chapter eight). These findings may be in line with a recent publication by Szreter (1997), who hypothesised that economic growth always has negative consequences: 'a sequential model of four Ds of disruption, deprivation, disease and death'. Whether death is the final outcome depends on, among other things, the political and social situation in the country (Szreter 1997). The finding that wealth was associated with high mortality from acute digestive diseases, and that mortality decline was not significantly associated with mortality decline in the late 19th century might be explained by the fact that there were some negative health affects of processes that caused the wealthy situation, which came under control in the early 20th century.

Female employment has been indicated as a possible determinant of infant mortality (Brändström 1988, Graham 1994). Simple correlations showed associations of high female employment levels with high infant mortality levels (results not shown). However, if other variables related to infant and childhood mortality were added to the model, female employment was, in most cases, associated with low infant and early childhood mortality. This association was significant in case of infant mortality from convulsions and early childhood mortality from measles in 1875/79 (table 9.4a,b). These results suggest that the positive effect of female employment viz. an increased household income was more important than the negative effects such as less breastfeeding or reduced child care in The Netherlands in that period. The finding that especially measles mortality was reduced in regions with high percentages of female labour participation would fit in the explanation of an increased household income. Most recent studies on female employment conclude that other determinants (e.g. fertility) were more important determinants of infant mortality than female employment (Preston & Haines 1991, Graham 1994, Brändström 1988). In The Netherlands, Roman Catholicism and urbanisation were also more important determinants of infant mortality and early childhood mortality levels than female employment (table 9.4a,b).

The number of *persons per dwelling* had a significant effect (corrected for possible confounding variables) on infant mortality from convulsions in 1875/79 and on early childhood mortality from acute respiratory diseases in 1875/79 and 1895/99 (table 9.4a,b). A high number of persons per dwelling was related to a high mortality rate. Overcrowding can affect mortality in different ways viz. facilitation of the transmission of airborne diseases, and an effect on water- and foodborne diseases due to the often bad sanitary conditions related to overcrowding (Burnett 1991). As far as early childhood mortality is concerned, the transmission of airborne diseases seemed to be the most important mechanism, considering the significant association with mortality from acute respiratory diseases. In case of infant mortality it is less clear. The significant association with mortality from convulsions might indicate that bad sanitary conditions related to overcrowding play a role too. Other results of the analyses presented in this paper suggested however that the category 'convulsions' consisted probably for a large part of airborne infectious diseases.

As far as *access to piped water* is concerned, infant and early childhood mortality showed not the expected association of a high percentage of the population with access to clean drinking water and low mortality rates. An increase in the percentage population with access to piped water was however associated with more rapid mortality declines. This association was significant for early childhood mortality in case of univariate correlations (results not shown). The association was not significant anymore in the multivariate analysis (table 9.5b). Other research has shown that it is difficult to find a significant association between the introduction of water supply systems and improvements in health. A big problem is that data for other factors that are strongly related to mortality (predominantly from acute digestive diseases) are often lacking. Examples of these factors are improvements in hygiene and sanitation (personal hygiene), and breastfeeding practices (Van Poppel & Van der Heijden 1997).

Other variables did not show the expected association with mortality (decline). The results for *medical care factors* were either not significant or the association was not in the expected direction. The density of midwives did not seem to play a role in mortality (decline). None of the associations were significant. Albeit that, a high density of midwives was related to low infant mortality levels in 1875/79. In the period 1875/79 to 1895/99, medical doctor and midwife densities tended to decline or remain stable, among other things, due to changes in the educational system for medical doctors (Van der Velden 1996b). Apparently those declines did not negatively affect mortality rates. It seems that the density of medical doctors and midwives remained sufficient for the demand in the different regions.

9.5 Conclusion

Cultural factors (Roman Catholicism) were predominantly associated with infant mortality and diarrhoeal diseases. With respect to early childhood mortality on the other hand, urbanisation played the most important role and was predominantly associated to acute respiratory diseases. So, different determinants of mortality (decline) play a role in both age groups, and they are related to different causes of death. Therefore, studies on the explanation of child mortality levels and decline should consider both age groups separately. This has also been argued in a recent publication on the decline of infant and child mortality in Europe (Woods 1997). With respect to determinants of infant mortality, the results for The Netherlands differed from the findings for England and Wales. Urbanisation did not play an important role in infant mortality (no 'urban-sanitary-diarrhoeal-effect'), but was important in case of early childhood mortality. Roman Catholicism and marital fertility were the key determinants for infant mortality. As far as infant mortality is concerned, The Netherlands shows a pattern comparable to Germany. Because a lot of literature on infant mortality stems from England and Wales (because of their abundance of data), these findings tend to shape our understanding of the determinants of infant mortality. However, those findings do not necessarily apply to other countries as well. Several years ago, Preston and Haines (1991) argued already, that the 'model of understanding' of the relationships between determinants of mortality (decline) and child mortality based on the British experience needed an alteration for the American situation, at least with respect to the role of social class.

Just like age-specific mortality, using cause-specific mortality in the analyses will add to a better understanding of the determinants of mortality (decline). Unfortunately, studies using cause-specific mortality are still rare. The use of cause of death categories with a uniform aetiology is preferred, although this is not always possible considering the often vaguely defined historical cause-of-death categories. Examples from this paper with respect to cause-specific mortality are the association of Roman Catholicism with acute digestive diseases instead of acute respiratory diseases (also an important cause of infant mortality), and the role of crowding in case of acute respiratory diseases and not acute digestive diseases. More explanatory studies using age- and cause-specific mortality and from different countries are needed to further improve our understanding of the underlying determinants of mortality (decline).

Chapter 10

GENERAL DISCUSSION: THE EPIDEMIOLOGICAL TRANSITION THEORY RE-EXAMINED

10.1 Summary of the results

The objective of this thesis was, first, to present a detailed description of the epidemiological transition in The Netherlands with respect to the onset of and accelerations or decelerations in all-cause mortality decline, with respect to cause-specific mortality trends, and the contribution of infectious diseases to mortality decline. Secondly, this thesis will study the relative importance of determinants of mortality decline in The Netherlands in the late 19th and early 20th centuries. Thirdly, the epidemiological transition theory will be re-examined on the basis of the results for The Netherlands.

In order to study cause-specific mortality trends over more than a century, the cause-of-death categories of the different cause-of-death classifications had to be reclassified. The method and result of this effort were presented in chapter four. A nested cause-of-death classification was created in which 27 causes could be distinguished from 1875 to 1992, 65 causes from 1901-1992 and 92 causes from 1931-1992. The 27 and 65 causes were used in the analyses presented in chapter five to seven.

In chapter five, the onset of and phases in mortality decline in The Netherlands were determined. The beginning of rapid or accelerated mortality decline

was determined in 1880. In 1917 mortality decline accelerated. This acceleration phase lasted until 1955. The period 1917-1955 was the period with the most rapid mortality decline. Mortality levels stabilised from 1955 to 1970. A renewed decline started in the year 1970. In terms of Omran's epidemiological transition theory, the last phase of the epidemiological transition could be placed in The Netherlands in the period 1955-1970.

In chapter six, changes in cause-specific mortality rates in The Netherlands were studied. Causes of death with the same trend curve over time were clustered. The results of the cluster analysis showed that a description of the epidemiological transition theory in terms of a 'decline of infectious diseases and increase of non-infectious diseases' is far from satisfactory. The conventional view of a uniform decline of infectious diseases and a uniform increase of non-infectious diseases should be modified. Some infectious diseases started only to decline considerably after WWII (e.g. influenza, syphilis, rheumatic fever). Others were even temporarily rising (e.g. poliomyelitis). As far as non-infectious diseases are concerned, a cluster could be identified that consisted of causes of death that had been declining since the beginning of this century (e.g. stomach cancer, COPD, perinatal causes of death).

The contribution of infectious diseases to mortality decline in the period 1875 to 1970, and four subperiods was studied in chapter seven. Respiratory tuberculosis contributed largely to mortality decline in the period 1875-1970 (15%). Some infectious diseases contributed mostly to mortality decline in a specific subperiod e.g. acute digestive diseases in the period 1901-31 (20%) and acute respiratory diseases in the period 1931-50 (37%).

In the descriptive studies presented in the chapters five to seven determinants of mortality decline were discussed by comparing cause-specific mortality trends and trends in determinants of mortality decline. In the chapters eight and nine, results of multivariate explanatory analyses of determinants and mortality decline (and levels), have been presented. In those analyses the regional variation in the prevalence of determinants that existed in The Netherlands in the late 19th and early 20th centuries were related to mortality levels and declines in that period. Cultural factors turned out to be relatively important in the early phase of mortality decline (the late 19th century). Economic factors, on the other hand, were more important in the first quarter of the 20th century (chapter eight).

The analysis of determinants of infant and early childhood mortality (chapter nine) showed that different determinants played a role in those age groups. Roman Catholicism and marital fertility turned out to be especially associated with infant mortality and acute digestive diseases, while urbanisation and crowding were associated with early childhood mortality and acute respiratory diseases. With respect to the role of urbanisation in infant mortality, The Netherlands was comparable to Germany in the late 19th century, but differed from

England and Wales. In the latter country, late-19th-century infant mortality (instead of early childhood mortality) has been related to the negative effects of urbanisation.

The results of the studies presented in this thesis have already been discussed in the preceding chapters. In this chapter, the epidemiological transition in The Netherlands will be discussed in a wider perspective. The reliability of the Dutch historical mortality data will be discussed, and the timing and pace of mortality decline in The Netherlands will be compared to other European countries. The weaknesses of Omran's epidemiological transition theory will be re-examined using the findings for The Netherlands together with the international literature. Elements for revision of the epidemiological transition theory will be presented. Finally, recommendations for further research and the policy implications of the epidemiological transition theory will be discussed.

10.2 Reliability of historical cause-specific mortality data

In The Netherlands, causes of death have been registered since 1755 (in The Hague), but until 1865, when the Medical Practitioners Act was adopted, provincial or local committees (in large towns) were in charge of the registration of causes of death. An attempt to adopt an act to establish a national cause of death registration in 1804 failed (Van Dijk 1982). Beside the Medical Practitioners Act (1865), the Burial Act (1869) was important for the Dutch cause-of-death registration system too.

The Medical Practitioners Act stated that medical doctors had to state as accurately as possible on a medical certificate the cause of death when one of their patients died. This medical certificate had to be given to the Registrar (of the registry of birth, deaths and marriages). The Registrar could only give permission to bury a person when a completely filled out medical certificate was given by the medical doctor (Burial Act, 1869). The medical certificate had to be filled out by the doctor who had attended the patient during his or her illness. Otherwise, an expert appointed by the municipal council issued the medical certificate after post-mortem examination (Van Poppel 1997).

Inventories of death were sent by each municipality to the provincial statistical offices and, after processing of these inventories, codes (according to the prevailing cause-of-death classification) were assigned to the causes of death at the public health inspectorate. The final processing of cause-specific mortality statistics was the responsibility of the Dutch Home Office. Later (1901) the final processing came in hands of a statistical bureau (CBS), and in the 1920s the

coding of causes of death became centralised and in the hands of a medical statistical officer too (Van Poppel 1997).

In summary, in the process of events of death until the cause-of-death statistics there are several steps that could influence under which code the death occurred in the statistic. The diagnosis (medical doctor) and the coding (Public Health Inspector or Medical Statistical Officer) are the most important steps in this process.

10.2.1 Diagnosis

In the period under study, 1875-1992, the availability of diagnostic devices increased drastically. Especially in the late 19th and early 20th centuries there was an enormous progress in the diagnosis of infectious as well as non-infectious diseases. The discovery of the infective agent of several infectious diseases in the late 19th century (e.g. tuberculosis 1882, cholera 1883, diphtheria 1884, pneumococcus 1886 (Rosen 1993)) made the development of diagnostic tests (e.g. a sputum test in case of tuberculosis) possible. The invention of X-ray technology (1895) was another step in the improvement of diagnosis, especially in case of tuberculosis. In the early decades of the 20th century, radiology became an important diagnostic device in the early detection of tuberculosis (Groen 1966). In the early twentieth century, diagnosis of non-infectious diseases improved too. In the 1920s, blocking of the coronary artery could be detected with electrocardiography, and in the same period heart catheterisation was invented. The combination of X-ray technology and the administration of contrast fluid by a catheter further improved the diagnosis of heart diseases (Berreklouw 1966). Only a few examples have been mentioned here, but diagnosis improved for many diseases in the early decades of the twentieth century (Van Dongen 1966).

Beside these important improvements in diagnostic devices, other factors could affect the diagnosis in the 19th century. The medical doctor who had attended the deceased person in his or her last period of illness had to issue the medical certificate. However, the density of medical doctors differed considerably across the country, so that in regions with a low medical doctor density it was more likely that no medical doctor had attended the ill person. The medical doctor density was much higher in the northwestern regions of The Netherlands as compared to the southeast. In 1875/79, there was one medical doctor for 5840 inhabitants in the province of Limburg, while there was one medical doctor for 1704 inhabitants in Zuid-Holland (Van der Velden 1996b). Although in absence of a medical doctor an expert had to examine the dead body, it has been reported that this was sometimes done by unqualified people. It also happened, if a medical doctor was asked to come after the death of a person, that the doctor just put the cause of death as told by the family or other attendants on the

medical certificate without examining the body (Van Poppel 1997). Such practices undoubtedly affected the reliability of the diagnosis in the 19th century.

10.2.2 Coding

The Netherlands was divided into four districts, each with a public health inspector who was in charge of the coding of causes of death. The public health inspectors were trained medical doctors, and they had agreed to abide by various coding rules. The decentralised coding of causes of death, however, gave the possibility of differences in coding between the inspectors. The director of the CBS complained in a letter to the Central Committee of Statistics in 1904, that there were differences in coding between public health inspectors, and even the individual inspectors were not always consistent in the way they coded the causes of death (Unpublished letter 1904).

Acute pneumonia and broncho-pneumonia in childhood were coded under 'diseases of the newly born' by one of the public health inspectors. The same inspector also coded convulsions and tuberculosis of the nervous system under 'diseases of the newly born'. In other words, many causes of death occurring at young ages were put in the category of diseases of the newly born by this inspector. The other three inspectors coded those causes of death according to the correct code of the International Classification of Causes of Death. There were also some differences in the coding of diseases at older ages. Degeneratio cordis was coded under 'diseases of old age' by one inspector instead of under heart disease. Pulmonary tuberculosis was coded under 'tuberculosis of two or more organs' instead of under respiratory tuberculosis by another inspector. The same inspector also coded tuberculosis of the intestines under 'tuberculosis of two or more organs', while there was a separate code for that type of tuberculosis too (Unpublished letter 1904). The problems of differences in coding were solved by providing the public health inspectors with more extensive guidelines for coding (Van Poppel 1997).

It took however until the 1920s before centralised coding was introduced. Changes in coding over time could still occur when, for example, a new Medical Statistical Officer became in charge of the coding or due to changes in policy of coding of causes of death as primary or secondary. A recent example of the way in which coding can affect the cause-of-death statistics is the coding of diabetes mellitus (Mackenbach *et al.* 1991).

Beside the aforementioned steps in the process of cause-of-death statistics there is one other important aspect that determines the eventual code i.e. the actual cause-of-death classification that is used. Until 1901, the Dutch Home Office had been in charge of the decision which classification was used, later this was determined by the statistical bureau (CBS). If a new edition of the International

Classification of Causes of Death was developed, it was a country's decision when to implement the new classification. In chapter four it has been described, how we dealt with the changes in classifications in The Netherlands.

10.2.3 Effect of inaccurate diagnosis and coding on the findings for The Netherlands in this thesis

In chapter four, the reclassification of causes of death over a pre-ICD and nine ICD-revisions has been described. In this way, cause-of-death categories were constructed that were nosologically continuous over time. This procedure took care of changing classifications, but changes in diagnosis or incorrect coding by the public health officer or the medical statistical officer are not covered by such a reclassification.

The effect of incorrect coding and diagnosis on the cause-specific mortality data has probably been the largest in the period 1875 to 1901. Only a brief classification of 34 cause-of-death categories was used in the late 19th century. Many categories of the 19th-century classification were broadly defined, e.g. brain diseases, skin diseases, diseases of pregnancy, violence. Although this hampers the interpretation of the causes of death included in such a broad category, it does not automatically mean that the diagnosis and coding were inaccurate in case of those categories. Other categories in the 19th-century classification were clearly defined, and can be considered as being accurate e.g. smallpox, scarlet fever, measles, whooping cough, cholera, malaria, puerperal fever. In case of two categories, viz. 'debility' and respiratory tuberculosis, inaccuracies in diagnosis and coding have explicitly been mentioned in the literature. Exchanges between respiratory tuberculosis, and acute or chronic respiratory diseases have been reported, as well as exchanges between 'debility' and acute digestive diseases (Evers 1882, Vollenhoven 1885, Saltet 1909, Onnen 1895).

Incorrect diagnosis and coding occurred, but it is not clear to what extent. There are reports in the literature that it happened, but there are no unanimous statements about the magnitude of the problem. However, the possible effect of inaccuracies on the results in this thesis can, of course, be discussed. The effect depends on the type of analysis (trend or level of mortality), the time period of the study (the late 19th century was most inaccurate), aggregation level of the study (national or regional), and whether the inaccuracies are differential.

In case of the analysis of trends, inaccurate diagnosis (or coding) would not be a big problem if the proportion of inaccurately diagnosed (or coded) cases within a certain cause-of-death category would remain the same over time. In that case the overall trend of the cause of death also reflects the trend of the 'real' cases. Diagnosis improved strongly in the late 19th and early 20th centuries, so the proportion accurately and inaccurately diagnosed (or coded) cases within a cause of death category changed over time. However, the declines in tuberculosis

mortality, 'debility' and chronic respiratory disease were so rapid that it is unlikely that the decline could completely be explained from inaccurate diagnosis or coding. In the period 1880-1917, for example, the percentage annual decline of all-cause mortality was 1.24, while this percentage was 2.26 in case of other disease (including 'debility'), 1.96 in case of respiratory tuberculosis, and 3.78 in case of chronic respiratory diseases (cf. Chapter five).

Whether the inaccurate diagnosis or coding is differential will also affect the results. In case of respiratory tuberculosis mortality decline, it was probably more likely that respiratory tuberculosis was coded as 'debility' or chronic respiratory diseases in the 19th century, than the other way around. It was mentioned before that tuberculosis of two or more organs was sometimes coded under 'debility' in the 19th-century classification. Beside that, it has been argued that, because of the stigma the diagnosis tuberculosis brought to the family of the deceased, intentionally the wrong diagnosis was put on the medical certificate (Bryder 1996, Hardy 1988). If this type of differential misclassification occurred, and assuming that accurate coding improved over time, the results presented in this thesis are an underestimation of respiratory tuberculosis mortality decline. As far as other disease (including 'debility') is concerned, the results are more likely to be an overestimation, considering the reports of coding of respiratory tuberculosis and acute digestive diseases as 'debility'.

If the real cause-specific mortality levels are of interest, misclassification of causes of death will lead to the wrong results. Whether the subject of study is national mortality or regional mortality does, however, play a role too. If the proportion of misclassified causes is the same in all regions under study, differences in level of cause-specific mortality between regions will remain the same. This was, however, not always the case. It is known that in some towns 'debility' was coded as acute digestive disease, while in other places it was the other way around (Onnen 1895). In chapter nine, regional acute digestive disease mortality levels have been used in an analysis of the determinants of mortality. Inaccurate mortality figures could in that case lead to incorrect inferences on the association of specific determinants with mortality from acute digestive diseases. In chapter nine, however, the associations were so strong, that the conclusions in that chapter are still considered valid.

10.2.4 Quality of the Dutch cause-of-death statistics in comparison with other European countries

In The Netherlands, cause-of-death registration has been a national concern since 1865. Several other European countries started a national registration earlier. In Sweden and Norway, a national cause-of-death registration came into use as early as the second half of the 18th century. Causes of death in the countryside were by then diagnosed and recorded by the clergy. In 1860 a statistical bureau

took over the registration of causes of death in Sweden, but it was not until 1971 that a death certificate issued by a physician became compulsory for all deaths (Rogers 1993). In England and Wales, the Registration Act of 1837 ordered a national cause-of-death registration. In the early years of the registration, notice of a death was to be given to the local registrar, and was normally done by a close relative of the deceased. This 'informant' provided details with respect to the cause of death too. From 1845, the certification of the cause of death by a medical doctor was encouraged by the Registrar-General. The proportion of medically uncertified deaths was as low as four percent for England and Wales as a whole (Williams 1996).

The Dutch 19th-century classification of causes of death was brief in comparison to, for example, England and Wales, Sweden, Norway, and France, all of which had classifications with more than a hundred causes of death in the late 19th century (Beneke 1875). During the period 1867 to 1874 a classification system of fifty-five causes of death was used in The Netherlands. The reliability of the cause-of-death statistics was highly questionable in the early years of the national cause-of-death classification and registration. The percentage cases in the category 'unknown' was high and there were no adequate checks on the correctness of the data. The Burial Act of 1869 led to a large improvement in the cause of death statistics. The number of cases classified as 'unknown' or 'without medical attendance' declined strongly. In the period 1875 to 1899, quinquennial reports were published with identical nomenclature in all publications. These were considered by contemporaries as extremely useful for medical-statistical research (Van Poppel 1997). Unfortunately, in those publications the number of cause-of-death categories had been reduced to thirty-four.

Despite the briefness of the classification, the fact that the cause-of-death registration was a medical registration positively distinguished the Dutch cause-of-death classification from those in other countries. The German Beneke (1875), who published an overview of the organisation of cause-of-death registrations in several European countries in the second half of the 19th century, started his chapter on The Netherlands with a remark on the excellent medical regulations in The Netherlands, referring to the Public Health Inspectors in case of coding of causes of death. In Sweden, for example, the clergy was involved in diagnosis of the cause of death, in rural areas, until the early 20th century (Rogers 1993), and in some German states, diagnosis by laymen remained common until the early 20th century (Kintner 1993).

The quality of the cause-of-death data, with respect to the diagnosis of the cause of death, should be considered high in The Netherlands as compared to other European countries. In the 19th century this advantage is, unfortunately, partly reduced by the briefness of the classification. However, The Netherlands adopted the first ICD-classification already in 1901(in some other countries this

was much later e.g. Denmark around 1930, and Germany around the 1940s), which means that from that year a medical registration was combined with detail in the classification.

10.2.5 Detailed reclassification of causes of death

In chapter four, a method was presented to reclassify the cause-of-death groups of 9 ICD-revisions and a pre-ICD classification to create nosologically continuous cause-of-death groups that could be studied for the period 1875/79 to 1992. Those cause-of-death groups had to be useful to study the epidemiological transition. Therefore we aimed at a good representation of communicable, non-communicable and external causes of death, and we succeeded to do so. The procedure described in chapter four was, however, very labour intensive, and others have studied the epidemiological transition using more crude methods to link 19th-century cause-of-death categories to more recent ICD-categories.

Mackenbach (1993a,b) used a crude reclassification method to compare cause-of-death groups in 1875/79 and 1970 in order to compute the contribution of those causes of death to mortality decline. Beside some well-defined infectious diseases groups, such as smallpox, scarlet fever/measles, whooping cough/diphtheria, respiratory tuberculosis, pneumonia/acute bronchitis/ influenza, broad cause of death groups were used in his analysis, such as diseases of the respiratory system, diseases of the digestive system. A comparison of the crude and detailed reclassification method shows that there are differences between the crude method used by Mackenbach and the detailed method described in this thesis. For example, the contribution of respiratory tuberculosis to male mortality decline in the period 1875/79 to 1970 was 13% on the basis of the crude reclassification, and 15% on the basis of the detailed classification. These percentages were 12% and 13% respectively in case of acute respiratory diseases. The results of the percentage contribution of measles/scarlet fever and diphtheria/whooping cough were identical: 3% and 2% respectively for both reclassification methods. However, as far as the contribution of *all* infectious diseases to mortality decline is concerned, the difference between the crude and the detailed classification was substantial. The results of the detailed reclassification method were 6 to 7 percent points higher (53% males and females combined) as compared to the crude reclassification method (46% males, 45% females).

The detailed reclassification has added value for research of the epidemiological transition in particular and for historical mortality research in general. As far as the epidemiological transition is concerned, first, the contribution of infectious diseases to mortality decline was higher according to the detailed classification. This could partly be explained from the broad category 'brain diseases etc.' which consisted mainly of infectious diseases (predominantly tuberculosis of the nervous system and encephalitis/meningitis) in the late 19th century. This cate-

gory was not considered an infectious disease category in the crude reclassification, but was considered to be so in the detailed reclassification. Secondly, part of the objective of this thesis was to give a detailed description of cause-specific mortality changes in The Netherlands. Such a detailed study of the epidemiological transition requires more detailed categories of causes of death than those used by Mackenbach. The reclassification method presented in chapter four resulted in such a detailed classification. Thirdly, the reclassification method presented in chapter four was derived from the work of Vallin and Meslé (1988, 1990), who produced nosologically continuous groups of causes of death for the period 1925 to the 1990s in France. In Italy a similar project has been started too (Vallin & Meslé 1996). So, in several countries detailed classifications are becoming available. This creates the possibility of international comparison of the epidemiological transition on a more detailed level than has been done so far.

With respect to historical mortality research in general, the detailed reclassification procedure resulted in trends for many causes of death that have never been studied for the years before 1950. Until now, mortality data were only available on disk from the 1950s onwards. However, for the studies presented in this thesis, all age-, sex-, and cause-specific mortality figures (on a 3 digit level) for all ICD-revisions and the pre-ICD classification have been entered in a database.

The Dutch cause-of-death statistics proved to be useful to study the epidemiological transition in The Netherlands. The cause-of-death classification in The Netherlands in the late 19th century was rather brief, and there were inaccuracies in coding and diagnosis of specific causes of death, but the effects of these inaccuracies on the conclusions presented in this thesis were limited. Although the 19th-century classification was brief, diagnosis by a medical doctor has been required in The Netherlands since 1865, which was earlier than in several other European countries. Besides, the detailed first International Classification of Causes of Death was introduced right away in The Netherlands in 1901. The reclassification procedure initially designed by Vallin and Meslé (1988, 1990) and applied for The Netherlands in chapter four made detailed analysis of the epidemiological transition possible, and yielded a valuable database for historical cause-specific mortality research.

10.3 Mortality decline in The Netherlands in a European perspective

10.3.1 The onset of mortality decline

Chesnais (1992) grouped countries according to the onset of crude mortality decline:

1. Mortality rates started to come down in France, Czechoslovakia and Scandinavian countries in the late 18th and early 19th century, and mortality decline accelerated in those countries around 1870.
2. Around 1870 mortality rates started to decline across Europe. Starting in the northwestern countries (England and Wales, Belgium, *The Netherlands*), in central Europe (Switzerland, Germany, Hungary) and in Poland and Russia. About 10 years later mortality started to decline in Italy and former Yugoslavia as well.
3. Around 1890/1900 mortality declines started in the southwest (Spain, Portugal) and southeast (Bulgaria, Romania) of Europe.

The pattern of European mortality decline is one from northwest to the southeast.

Vallin (1991) studied mortality decline in Europe in the period 1720 to 1914. Beside France and the Nordic countries (cf. Chesnais 1992), he also considered England and Wales an example of early mortality decline (i.e. starting in the 18th or early 19th centuries). Among the early starters there were different patterns of decline. The Nordic countries experienced a regular mortality decline since the middle of the 18th century. France, on the other hand, experienced a clear crude mortality decline in the period 1750 to 1845, followed by a stagnation of mortality decline in the period 1845 to 1890. According to Vallin, crude mortality in England and Wales had been fairly constant from the mid-18th century until about 1830. However, the trend of life expectancy at birth presented by Wrigley and Schofield (1981) for the period 1551 to 1861, shows an improvement in life expectancy from 1750 until about 1830.

Considering the findings for The Netherlands with respect to the onset of mortality decline (chapter five), the start of rapid mortality decline in The Netherlands was determined around 1880 (1.2% annually). Mortality had already been declining slowly from the beginning of the 19th century (0.4% annually until 1836, and 0.1% annually thereafter until 1870/80). In the early 19th century, the large swings in mortality could easily mask this slow decline. There are no national mortality data for The Netherlands before 1804. Data for the 18th century are only available for specific towns such as Amsterdam. The crude mortality

trend for Amsterdam showed an increase in mortality from 1774 until about 1820 and a decline thereafter (Jansen & De Meere 1982).

According to Schofield (1981), the early mortality declines that could be studied for a limited number of European countries should not be seen as a precursor of the massive fall of mortality that could be observed in most European countries in the late 19th and early 20th centuries. The mortality trend in the 18th and early 19th centuries was characterised by large swings of mortality, and the observed declines were not unique in history. Vallin considers the mortality decline starting in the late 19th century of a different order than earlier mortality declines too. He speaks about the onset of a new mortality regime, which started in the second half of the 19th century. 'A mortality regime in which crisis mortality does not shape the trend anymore' (Vallin 1991). This 'new mortality regime' is what we know as the transitional phase of the demographic transition theory, and the 'age of receding pandemics' in Omran's epidemiological transition theory.

Considering Chesnais' grouping of countries, England and Wales should also be put in the group of early starters, and The Netherlands possibly as well considering the onset of decline in 1804. A good comparison of countries with respect to the onset of mortality decline is, however, not possible. Only for a limited number of countries data on mortality for the 18th and early 19th centuries are available. If countries are classified according to the onset of 'the new mortality regime', The Netherlands would surely belong to the group of European countries in which this 'new mortality regime' started firstly viz. around 1870/80.

Table 10.1 – Life-expectancy at birth (males) for several European countries in the years 1870/80, 1910, 1950 and 1990. N.a. = not available.

Country	Life expectancy at birth 1870	Life expectancy at birth 1910	Life expectancy at birth 1950	Life expectancy at birth 1990
Sweden	45	55.2	69.1	74.8
Norway	47	55.0	70.2	73.4
France	41	48.5	62.8	72.8
Czechoslovakia	n.a.	40.0	63.6	67.3
England & Wales	39	51.5	66.5	73.6
The Netherlands	35	52.9	70.3	73.7
Germany	37	47.4	64.2	71.8
Hungary	n.a.	39.1	59.6	65.1
Russia	n.a.	34.7	57.3	64.2
Italy (1880)	33	44.3	62.9	73.6

Sources: Vallin, 1991; Haines and Kintner, 1993; Caselli, 1991 and 1996; Van Poppel, 1996

10.3.2 The level of mortality at the onset of mortality decline

Table 10.1 gives life expectancies from different sources for different countries starting with the year 1870. In all selected countries, mortality started to decline rapidly around 1870. Life expectancies are presented instead of crude mortality rates in order to avoid effects of differences in age structure of the selected populations.

Table 10.1 shows that there are considerable differences between countries in life expectancy at the onset of rapid mortality decline. If we compare The Netherlands to Norway, there is a difference of 10 years in 1870. A first explanation for this difference could be the difference in pre-transitional mortality decline. In Norway mortality started to decline earlier as compared to The Netherlands. However, in France mortality decline started early too, but mortality rates have always been higher in France as compared to Norway (Vallin 1991).

The differences in life-expectancy at birth at the onset of 'the new mortality regime' are more likely a mix of differences in pre-transitional mortality decline and other factors that can determine the level of mortality. Climatic differences between countries might, for example, have played a role. In southern European countries diarrhoeal infectious diseases were more easily spread as compared to northern countries. Diarrhoeal diseases were an important cause of death for infants. Higher infant mortality rates in turn affect the life expectancy at birth. A factor that might explain part of the difference between Norway and The Netherlands is, for example, the high fertility rates (which are related to high infant mortality levels) in The Netherlands as compared to other northwestern European countries (Coale & Treadway 1986).

Table 10.2 – Relative increase in life-expectancy at birth (men) for several European countries in the years 1870/80-1910, 1910-1950 and 1950-1990

Country	e(0)1910 / e(0) 1870	e(0) 1950 / e(0) 1910	e(0) 1990 / e(0) 1950
Sweden	1.23	1.25	1.08
Norway	1.17	1.27	1.05
France	1.18	1.29	1.16
Czechoslovakia	n.a.	1.59	1.06
England & Wales	1.32	1.29	1.11
The Netherlands	1.51	1.32	1.05
Germany	1.28	1.35	1.12
Hungary	n.a.	1.52	1.09
Russia	n.a.	1.65	1.12
Italy	1.34	1.42	1.17

10.3.3 The pace of mortality decline

The last column of table 10.1 shows that differences in life expectancy at birth have diminished over the years. Life expectancy in The Netherlands and Norway differed 10 years in 1870 while this was only 0.3 years (in favour of The Netherlands) in 1990. A convergence of mortality in the European countries has taken place during the epidemiological and demographic transition. Table 10.2 shows the relative increase of life expectancy in the periods 1870 to 1910, 1910 to 1950 and 1950 to 1990.

The increase in life expectancy in the period 1870 to 1910 was large in The Netherlands as compared to other northwestern European countries. As far as Norway and France are concerned, those were early starters with respect to mortality decline, which might explain their relatively small increase in life expectancy as compared to The Netherlands. England and Wales, on the other hand, had a comparable timing of the epidemiological transition as The Netherlands, but showed less rapid increases in life expectancy. In The Netherlands, a variety of public health measures were taken in the late 19th and early 20th centuries e.g. the introduction of piped water in the cities, measures to improve the safety in factories, building requirements. Besides, improvements in living standards and fertility declines have also been suggested to have contributed to mortality decline in that period (cf. chapter five). The combined improvements of economic, cultural and public health factors have probably caused the relatively rapid declines in The Netherlands in the late 19th and early 20th centuries.

The period 1910 to 1950 is characterised by the rapid increases in life expectancy in Central-European countries. According to Caselli (1996) mortality variation between European countries diminished in this period due to the introduction of sulphonamides, antibiotics and vaccinations, and due to socio-economic improvements in the central European countries. The relative increases in life expectancy differed not much among the northwestern European countries, but the increase in The Netherlands was again relatively strong.

In the last period, 1950 to 1990 improvements in life expectancy were less rapid as compared to the former period. The relative improvements in life expectancy in The Netherlands in that period are, contrary to the former periods, among the lowest in Europe. The Netherlands started off with a relatively low life expectancy in 1870, but rapidly made up the difference with the Nordic countries. The Netherlands had already achieved mortality levels comparable to Norway in 1930, while England and France only reached 'Norwegian' life expectancy levels around the 1970s and 1990s (Caselli 1996).

10.3.4 Cause-specific mortality

Not only the pace of mortality decline differed among countries in which the 'new mortality regime' started in the late 19th century, but also the cause-of-death pattern. The cause of death pattern differed with respect to the contribution of causes of death to mortality as well as the pace of specific mortality decline.

Caselli (1991) compared the cause-of-death pattern of England and Wales (in 1870) and Italy (in 1880) at the onset of the transitional phase of the epidemiological transition. The contribution of diarrhoeal diseases to mortality was about twice as high in Italy (11%) as compared to England and Wales (6%). Respiratory tuberculosis, on the other hand, contributed twice as much to mortality in England and Wales (10%) as compared to Italy (6%) at the onset of the transition. In The Netherlands, acute digestive diseases contributed 6% to mortality at the onset of the transitional phase, and respiratory tuberculosis contributed 10% (cf. table 5.2). Higher mortality from diarrhoeal diseases compared to The Netherlands and England and Wales remained a characteristic of Italian mortality throughout the transition. Differences in cause-of-death pattern between countries might occur because of differences in timing of the transition, and this idea has also been used to explain differences in mortality patterns between countries. However, comparison of the cause-of-death pattern at the onset of decline shows that there were differences by that time too, which should be explained from differences in determinants of mortality.

Not only the contribution of causes of death differed among countries with comparable onsets of mortality decline, but the pace of cause-specific mortality differed as well. Mortality from diarrhoeal diseases (at age 0-4) declined less rapidly in Italy as compared to northwestern European countries in the early 20th century. Mortality from acute respiratory diseases did not show such a clear north-south gradient (Caselli 1996).

The Netherlands can, together with other northwestern countries e.g. England and Wales, be considered a country in which accelerated mortality decline started early, i.e. around 1870. The Netherlands distinguished itself from other countries with respect to the rapid increase in life expectancy in the late 19th and early 20th centuries. With respect to cause-specific mortality there were differences between northwestern and southern European countries too. Acute digestive diseases were more important contributors to mortality, and declined less rapid in southern countries (e.g. Italy) than in north-western countries (e.g. The Netherlands). More research on cause-specific mortality decline would be needed to draw more soundly based conclusions on differences in the epidemiological transition with respect to cause-specific mortality change.

10.4 The epidemiological transition theory: weaknesses of Omran's theory re-examined

In the introduction of this thesis, an overview was given of some weaknesses in and criticism on Omran's epidemiological transition theory. Especially, the definition of the stages of the epidemiological transition by Omran was unclear with respect to cause of death pattern and timing of the stages. In the former chapters, the epidemiological transition has been studied in detail for The Netherlands. In the light of these results, Omran's epidemiological transition theory will be re-examined with respect to the changes in cause-specific mortality, and the timing of the stages. Only the 'age of receding pandemics', the 'age of degenerative and man-made diseases' and the 'age of ageing, chronic diseases, emerging new scourges and the resurgence of older diseases' will be taken into consideration. The pre-transitional phase, 'the age of pestilence and famine' has not been studied for The Netherlands.

10.4.1 Cause-specific mortality change

In chapter six, the changes in cause-specific mortality in The Netherlands in the period 1875-1992 were studied. In that chapter, it was pointed out that the prevailing perception of the epidemiological transition as a decline of infectious diseases and an increase of non-infectious diseases is too simple (e.g. the early declines of stomach cancer, cerebrovascular diseases, and the rise of appendicitis and poliomyelitis). The description of the stages of the epidemiological transition in Omran's original epidemiological transition theory with respect to changes in cause-specific mortality was rather vague, and did not leave much room for another interpretation of the changing cause-of-death pattern than that of a decline of infectious diseases and an increase of non-infectious diseases (cf. chapter two). However, it should also be mentioned that, in the last update of his theory in 1993, Omran did mention the possibility of a renewed increase of infectious diseases ('emerging new scourges' e.g. AIDS, and the 'resurgence of older diseases' e.g. tuberculosis).

Several researchers have proposed labels for the increasing causes of death e.g. civilisation associated diseases, diseases of modernisation, Western diseases. Those concepts relate the increase of diseases to civilisation, modernisation and affluence. During modernisation certain causes of death indeed increased, but the same causes of death started to decline later in the process of modernisation. Appendicitis came up during the 1920s and declined in the 1930s, ischemic heart disease and traffic accidents showed a strong increase after WWII but have been declining since the 1970s (cf. chapter six). So, the effect of modernisation on the course of disease is not uniform over time. Another example of this non-uni-

form effect is the deteriorating effect of industrialisation on health in Britain's industrial cities in the second and third quarter of the 19th century, while these deteriorating effects diminished in the last quarter of the 19th century (Szreter 1997). In order to label causes of death according to their course over time, more specific concepts should be considered than concepts such as modernisation, civilisation or affluence, which are too general.

It would be interesting to know if the clusters of rising and declining causes of death as were found for The Netherlands are also found for other countries. The increase of appendicitis in the 1920s has been reported for England and Wales (Barker 1989a). Considering the study of Caselli (1996) on differences in cause-of-death trends (e.g. diarrhoeal diseases) in England and Wales and Italy, we might indeed expect differences between countries in the trend curves of causes of death.

10.4.2 Timing

The timing of the stages in the epidemiological transition was not accurately defined by Omran either. The onset of the 'age of receding pandemics' was located after the 'early modern period', and the onset of the 'age of degenerative and man-made diseases' was located in a rather broad time period, viz. just after the first world war, in the 1920s or 1930s. The different 'ages' in the epidemiological transition were characterised by Omran on the basis of total mortality change and cause-specific mortality change. In chapter five of this thesis, phases in the epidemiological transition in The Netherlands were determined on the basis of total mortality decline, and in chapter six on the basis of changes in cause-specific mortality.

In table 10.3, the phases distinguished in the analyses for The Netherlands are compared to Omran's 'ages'. The analyses of the epidemiological transition in The Netherlands resulted in more phases than the four 'ages' defined by Omran. For The Netherlands, some of Omran's 'ages' have to be split into subphases in order to make timing comparable.

According to Omran, the 'age of degenerative and man-made diseases' started just after the First World War. The results for The Netherlands show that this 'age' could be split into two subphases (table 10.3). It could be argued, based on the results for The Netherlands, that the second subphase, starting after the Second World War, marked more clearly a new 'age' than the mortality changes that started just after the First World War. After the Second World War, all-cause mortality decline strongly decelerated, and the rise of ischemic heart disease and traffic accidents is most striking. The mortality changes just after the First World War are still characterised by strong mortality declines and declines in infectious diseases mortality.

In the last update of the epidemiological transition theory Omran added a fourth stage to the transition, 'the age of ageing, chronic diseases, emerging new scourges and resurgence of old diseases' (Omran 1993). This had earlier been proposed by others (Olshansky & Ault 1986, Rogers & Hackenberg 1987). Omran gave, however, no indication of the timing of this stage. In The Netherlands, a renewed decline of mortality was observed after 1970. This decline was characterised by the decline of ischemic heart disease and traffic accidents. Renewed

Table 10.3 – Comparison of phases in the epidemiological transition in The Netherlands and the 'ages'described in the epidemiological transition theory by Omran

Omran (1971, 1983, 1993)	The Netherlands: Phases based on total mortality decline (chapter five)	The Netherlands: Phases based on cause-specific mortality change (chapter six)
The 'age of pestilence and famine' Onset: not defined Mortality is high and fluctuating	could not be studied	could not be studied
The 'age of receding pandemics' Onset: after the early modern period Mortality declines progressively, steady decline of infectious diseases such as diarrhoeal diseases and tuberculosis, and a moderate increase in cancer and cardiovascular disease	Onset: 1880 Progressive mortality decline started	Onset: 1875 2 subphases 1875-1901: characterised by decline of water-and food-borne diseases 1901-1921: characterised by decline of water- and food-borne as well as airborne infectious diseases
The 'age of degenerative and man-made diseases' Onset: after the First World War, or in the 1920s or 1930s Mortality continues to decline and eventually approaches stability, the decline of infectious diseases and rise of degenerative diseases is more distinct and, since 1945, the increase in cardiovascular disease is particularly striking.	Onset: 1917 2 subphases: 1917-1955: acceleration of mortality decline, period with the most rapid mortality decline 1955-1970: hardly any mortality decline	Onset: 1921 2 subphases: 1921-WW2: characterised by decline of airborne infectious diseases, and the rise and decline of other infectious diseases WW2-1970: characterised by the increase of ischemic heart disease and traffic accidents
The 'age of ageing, chronic diseases, emerging new scourges and the resurgence of older diseases' Onset: not defined Not further described	Onset: 1970 Renewed progressive mortality decline	Onset: 1970 Decline of ischemic heart disease and traffic accidents

increases in infectious disease mortality were no characteristic of that phase in The Netherlands.

In the previous paragraphs we have discussed Omran's stages in the epidemiological transition on the basis of the findings for The Netherlands. The changes in cause-specific mortality rates should be described more specifically as compared to a decline in infectious diseases and an increase in degenerative and man-made diseases. The effects of modernisation or affluence on cause-specific mortality have not been uniform over time. Many causes of death showed an increase followed by a decline (e.g. ischemic heart disease). With respect to timing of stages in the epidemiological transition, it seemed to be more logical to place the onset of the 'age of degenerative and man-made diseases' after the Second World War instead of in the 1920s or 1930s. On the basis of all-cause and cause-specific analysis, more phases in mortality decline were defined than the three (or four (Omran 1993)) stages defined by Omran.

A more general notion with respect to the stages of the epidemiological transition is that the epidemiologic transition theory should be considered more a theory of succession of stages than a transition from one stage to another. The transition from one stage to another suggests a certain equilibrium or steady state before and after the transitional period (Smith 1994). In the demographic transition there is a sort of equilibrium before and after a transitional period i.e. high mortality and high fertility to low mortality and low fertility. In the epidemiological transition there is no such equilibrium. The cluster-analyses of cause-of-death trends provides a good example that epidemiological change is an ongoing process of rise and decline of groups of causes of death instead of a transition of one steady state to the next.

10.5 Revision of the epidemiological transition theory

The epidemiological and demographic transition theories have been criticised for being non-theories, because they cannot explain or predict changes in mortality (and fertility) or changes in cause-of-death patterns for other countries or other periods of time (Chesnais 1992, Kirk 1996). Several researchers have proposed alternatives to Omran's original theory. In this section, we will discuss whether the epidemiological transition theory can be considered a theory or just an empirical generalisation of the mortality experience in various countries, and, if revision of the epidemiological transition theory is needed, which elements will be necessary in a revised version.

10.5.1 Natural sciences and the construction of theories

The philosophy of natural sciences describes theories as follows. Theories are constructed when research of certain phenomena shows regularities that can be described in empirical laws. Theories try to explain those regularities and try to give a more profound and more accurate understanding of the phenomena. The observed phenomena are manifestations of underlying entities or processes. Those fundamental entities or processes that are postulated by the theory must be described clearly and precisely in order to be able to explain and predict. The theory should not only be able to explain the empirical phenomena observed before, but should also be feasible to predict new phenomena of the same order (Hempel 1966).

Considering the definition of scientific theories as used in natural sciences, the criticism that the epidemiological transition theory is not a theory, seems to be justified. The theory as described by Omran is more a description of regularities - mortality decline is accompanied by specific cause-specific mortality changes - than a formulation of fundamental processes that explain those regularities. Omran did formulate a proposition on the determinants of mortality decline in his theory, but in general terms: 'in countries in which the epidemiological transition started before the 20th century, the main determinant of mortality decline was the rise in living standards, while in countries that experienced the epidemiological transition in the 20th century it was medical care and technology' (Omran 1983). This formulation is, however, to general to be able to explain the changes in cause-specific mortality in countries, or the variety in the decline between countries, or to predict cause-specific mortality changes. The last update of Omran's theory (1993) was also more an extended description of 'regularities' (i.c. a fourth phase in the transition, and extra models with respect to timing and pace of mortality decline) than a refined formulation of underlying processes that constitute total mortality change and change in cause-specific mortality.

Several researchers have used Omran's 'description of regularities' to estimate cause-specific mortality (for countries for which these data are scarce) on the basis of the relationship between cause-specific mortality rates and total mortality in western countries (Preston & Nelson 1974, Palloni & Wyrick 1981, Lopez & Hull 1982). Bulatao (1993) estimated cause-specific mortality for the period 1970 to 2015 for industrial countries, non-market economies, Latin American countries and Asia. Broad cause-of-death categories were estimated, such as infections, neoplasms, circulatory diseases, and more specific categories, such as diarrhoea, acute respiratory diseases, ischemic heart disease, cerebrovascular accident. The accuracy of the estimations was evaluated by comparing the predicted percentage mortality from specific causes of death to the reported percentage in 1985. This comparison showed that differences in predicted and reported percentages ranged from 5 to 12 percent points for the broad cause of death groups (such dif-

ferences were also found in a study by Bah (1995) for Ghana and Kenya), from 12 to 34 for specific infectious diseases, and from 8 to 24 for specific diseases of the circulatory system. The accuracy of the estimations differed also by type of country. The predictions of the broad cause of death groups were least reliable for non-market economies (mortality from diseases of the circulatory system was predicted 38%, while the actual percentage was 50). In summary, as far as broad cause-of-death groups are concerned the epidemiological transition theory leads to quite precise estimations, but in case of more specific causes of death the estimated and real values differ considerably.

10.5.2 Alternative conceptions of science

As far as the demographic transition theory is concerned, the issue of theory or non-theory has been discussed more extensively. Recently, Szreter published a so-called 'critical intellectual history' of the demographic transition theory in which he argued that 'the idea of demographic transition was itself the product of a particular conception of social science as a guide for policy, a science employing a positivistic methodology that was simultaneously investigative and predictive' (such as in natural sciences – *W-wdB*) (...) 'such a conception does not today correspond to the only accepted notion of 'science': there are other alternatives, especially for social and historical problems' (Szreter 1993).

The alternatives are other schools of social science that consider the aim of scientific explanation and its methods mere understanding, and not prediction or control. As Szreter puts it: 'social phenomena occur as an irreversible flow or complex sequence of events. All that is available for study is their scientific, historical reconstruction, from which can come an understanding of how that irreversible history happened' (Szreter 1993). Szreter pleas for a study of fertility change in specific communities without the preoccupation of a 'fertility transition'.

Not all researchers agree with Szreter on this subject. The demographic transition is a very complex phenomenon considering the enormous amount of determinants involved and the variety in transitions among countries, but the differences are not that big, they argue. The transition may be accelerated or delayed but the transition itself is inescapable is their argument (Kirk 1996).

It is indeed true that historical or social phenomena are open systems contrary to the closed system of the experiment in natural sciences. In order to be able to explain historical or social phenomena it is necessary to continuously include new information. This makes explanation of such phenomena complex, and predictions can in most cases only be made on the short term. It has been argued, however, that it is in principle not impossible in historical and social sciences to formulate theories to explain and predict historical and social phenomena (Wesley 1982). Referring to the subject of study, there are differences

between populations with respect to timing and pace of mortality decline, and cause-specific mortality changes, but this does not mean that there might not be underlying processes that explain those differences. A revised epidemiological transition theory should formulate those underlying processes. Before we will further explore the possibility of the formulation of a revised epidemiological transition theory, alternative theories to Omran's theory that have been published in the literature will be discussed.

10.5.3 Alternatives to Omran's epidemiological transition theory

The 'health transition theory' and the 'theory on assets for health' will briefly be discussed here. Both the 'health transition theory' and the 'theory on assets for health' focus on underlying processes of mortality decline and epidemiological change.

Health transition theory

The health transition can be divided into two components: the epidemiological transition and the health care transition. The epidemiological transition is the long-term process of change in health of a society (including mortality, morbidity and disability); the health care transition is the change in the patterns of the organised social response to health conditions (Frenk *et al.* 1991).

Some argue that there is in principle no difference between health transition and epidemiological transition. 'Both are concerned with the distribution of death and disease among population groups; with their determinants and consequences; and with how these factors change over time. The difference lies in that the health transition theory emphasises the social, cultural and behavioural determinants of health' (Cleland 1989). Others consider this emphasis on social, cultural and behavioural determinants so important that the term 'health transition' is justified beside 'epidemiological transition' (Caldwell 1989).

Frenk *et al.* (1991) gave an extensive formulation of health transition theory. They distinguished three major underlying mechanisms involved in the epidemiological transition: fertility decline, changes in risk factors, and improvement in case-fatality rates. Changes in various risks, biological, environmental, social, behavioural, occupational, can change the incidence of disease and cause-specific mortality. Changes in those risks can be induced by social, economic, and cultural processes. As far as the rapid mortality declines in historical Western countries are concerned, those processes are summarized in the term modernisation. Those social, economic and cultural processes result not only in a reduction of certain risks (and consequently a reduction of specific diseases), e.g. environmental sanitation and a reduced risk of diarrhoeal diseases, but also induce new risks, e.g. nutrition and cardiovascular disease. Changes in the case-fatality

rate also contribute to mortality decline. Improvements in effective treatment of many diseases have resulted in a reduction of the case-fatality rate. Together with other factors such as changes in life-style this mechanism has played an important role in the mortality decline after the 1970s (Olshansky & Ault 1986, Crimmins 1981).

Fertility decline was mentioned by Frenk et al. as a mechanism that influences the age-distribution of the population. Changes in the age-distribution can change the absolute number of cases while the age-specific mortality rates remain the same. This mechanism plays more a role in health planning; the other two mechanisms are important for the formulation of underlying processes of mortality decline and cause-specific mortality change.

Theory of assets for health

Murray and Chen (1993) proposed a new theory for the explanation of mortality decline: a theory of assets for health. According to Murray and Chen, existing theories (and hypotheses) on mortality decline (the McKeown-thesis, modern health technology hypothesis, and the emphasis on culture and behaviour as is the case in the health transition theory) are 'static and overly concentrated on the short-term. (...) mortality may be regulated by longer-term cumulative processes related to the build-up or depletion of a society's 'health stocks". Health-relevant assets can be physical assets, e.g. infrastructure of the health care system, schools, housing, water supply, and social assets, e.g. education, health related perceptions and behaviour, social institutions (social security, health insurance). Indicators of the health assets are, for example, the capacity and quality of the health care system, public infrastructure, household environment, literacy, and education.

An accumulation of health assets may reach a certain threshold at which mortality decline accelerates. The accumulation of health assets can make a secular mortality decline possible, which will be only temporarily disturbed by crises. After the crisis, mortality will rapidly return to pre-crisis levels (Caldwell 1986, Murray & Chen 1993). Physical assets (e.g. infrastructure) may depreciate over time and require constant re-investments. Social assets (e.g. knowledge, health behaviour) are less likely to depreciate over time (Murray & Chen 1993). In the context of the health transition theory it has also been argued that especially changes in perceptions and behaviour of people may well be irreversible, while improvements in the public field, for example environmental sanitation, infrastructure, might be more vulnerable to deterioration (Van de Walle 1989).

The theory of assets for health seems to be an attractive proposal for a new theory on mortality decline. The onset of a 'new regime of mortality decline' in the 19th century in Western countries is linked to the modernisation process. Modernisation consists of changes in different fields of society: economic, cul-

tural, political and social. All these different processes (e.g. industrialisation, rationalisation) might be interpreted as an accumulation of health assets, which could lead to accelerated and secular mortality decline. Another example in favour of the theory of assets for health can be found in the mortality decline in The Netherlands. Mortality levels indeed rapidly returned to pre-war after the Second World War (figure 5.2). It is, however, more difficult to explain the acceleration in mortality decline in The Netherlands in 1917, which lasted until 1955, in terms of acquired assets of health or a threshold of health assets that was reached.

10.5.4 Elements for revision of the epidemiological transition theory

It was shown that Omran's original epidemiological transition theory did not accurately describe detailed cause-specific mortality changes (such as described for The Netherlands in chapter six). With respect to broad categories the original theory could, to some extent, be used to estimate cause-specific mortality changes in countries in which the transition started later as compared to western countries. In order to be useful for more accurate predictions of cause-specific mortality, the theory should be revised. The authors of the alternative theories discussed before provided elements for a new theory to understand mortality decline and/or epidemiological change. The health transition theory and the health assets theory tried to describe underlying processes of mortality decline and/or cause-specific mortality change, while the original epidemiological transition theory only described the observed regularities in mortality decline in different countries. In this section we will discuss whether the alternative theories are adequate to explain and predict cause-specific mortality changes, and if they are not, what other elements should be included in a revised epidemiological transition theory.

The phenomena or regularities that a revised epidemiological transition theory should be able to explain and predict are cause-specific mortality changes accompanying accelerated mortality decline. The theory should only be able to explain and predict phenomena of the same order as those that are subject of the theory.

Should 'pre-transitional' and 'post-transitional' mortality decline be included in the theory?

The first phase of the original epidemiological transition theory, 'the age of pestilence and famine', refers to the period of pre-transitional mortality. I would argue that that phase should not be included in a revised epidemiological transition theory. A theory should be able to explain and predict phenomena of the same order. Other researcher have argued that the so-called pre-transitional

mortality decline should not be considered as early stages of modern rapid mortality decline. Vallin (1991) spoke of a new mortality regime in which chance factors no longer play as large a part as before. Schofield (1981) argued that the accelerated mortality decline in the 19th century was unprecedented in history, while pre-transitional mortality decline (in the 18th century) was not.

Omran (1993) included a fourth phase, the 'age of ageing, chronic diseases, emerging new scourges and the resurgence of older diseases' in the last update of the epidemiological transition theory. Olshansky and Ault (1986), and Rogers and Hackenberg (1987) had suggested a fourth phase in the epidemiological transition theory too to include mortality changes that occurred after the 1970s. Should those relatively recent mortality changes be included in the epidemiological transition theory? I would argue that, just like the pre-transitional mortality changes, these post-transitional mortality changes should not be included in the theory. The transitions of countries, that followed the epidemiological transition (in terms of a decline of infectious diseases and a rise in non-communicable diseases) before, began to show deviating transitions around the 1960s. Western mortality developments from the 1960s or 1970s onwards appeared to be not a general phenomenon. Russia, for example, had followed the transition as observed in western countries, but after 1960 the (cause-specific) mortality started to differ considerably. Mortality decline from cardiovascular diseases was not found in Russia and in some other central- and eastern European countries (Chen *et al.* 1996, Feachem 1994).

Underlying processes of mortality decline and cause-specific mortality change

The theory of assets for health provides only an explanation for secular mortality decline and not for cause-specific mortality changes. The latter should, however, be included in a revised theory of epidemiological transition. The way the health transition theory has been formulated seems to provide more points for departure to formulate a revised epidemiological transition theory.

The changes in cause-specific mortality over time show rises and declines of specific causes of death. This was clearly shown by the results of the cluster-analysis presented in chapter six. Causes of death such as appendicitis and poliomyelitis increased during the1920s and 1930s and decreased afterwards; ischemic heart disease and traffic accidents increased until 1970 and decreased thereafter; causes of death such as chronic rheumatic heart disease slightly increased until the Second World War and declined thereafter. But also causes of death that have been declining since the late 19th century have shown increases in earlier periods of time. Smallpox, and 'fevers', for example, increased in the 17th and 18th centuries and declined thereafter (Mercer 1990).

The underlying mechanism that explains these courses of rise and decline might be formulated as follows. Mortality from specific causes of death rises in

response to the emergence of new risk factors, e.g. new nutritional patterns and smoking in case of ischemic heart disease, and increased motorised traffic in case of traffic accidents. Mortality can decline again when the risk factor is eliminated or reduced, or when the case fatality rate is reduced (cf. the mechanisms described by Frenk *et al.* (1991)). The mechanisms to reduce mortality can be perceived as 'adaptation mechanisms' of a population to the emerging health risk (Van Beeck 1998). Adaptation to new health risks can occur in many different ways, such as public health measures, medical treatment, and behavioural changes. Szreter (1997) presented an example of this pattern of emergence of and adaptation to health risks. According to Szreter, economic growth has negative consequences that may be conceptualised as 'a sequential model: the four D's of disruption, deprivation, disease, and death'. Whether the final D of death occurred depended on the country's 'political, ideological, social and institutional history'. Nineteenth century industrialisation (economic growth) led to increases in mortality (predominantly among infants and young children) in Britain's industrial cities. This negative association between economic growth and health turned into a positive association when new political developments made adequate public health measures possible (Szreter 1997).

A formulation of the epidemiological transition in terms of rise and decline of causes of death, and in terms of a disruption of health by emerging health risks and adaptation to health risks is quite different from the epidemiological transition theory presented by Omran. Although this formulation might still be too general to be considered a new theory, it gives the epidemiological transition theory a new focus viz. the underlying mechanisms, and the cause-specific mortality changes as a result of that, instead of a description of epidemiological transition as a decline of infectious diseases and a rise of non-infectious diseases with little reference to underlying determinants. In a revised epidemiological transition theory, the underlying processes of mortality change should be described more specific with reference to causes of death, to 'accelerated' mortality decline, to the factors that caused the disruption of health, and to the way in which populations adapted to the disruption of health. In other words, the theory should be formulated more in terms of determinants of causes of death that have emerged and declined over time. An example of the formulation of an underlying process of cause-specific mortality change could be Barker's 'hygiene hypothesis' (Barker 1985, Barker *et al.* 1988). This hypothesis states that as hygiene improves, enteric infections in young children will be reduced, which in turn make those children prone to appendicitis and some other infections such as poliomyelitis in adolescence. When hygiene further improves, the likelihood of contracting an infection as a young adult decreases too. The 'hygiene hypothesis' explains part of the sequence of rise and decline of cause-specific mortality that could be observed during the epidemiological transition viz. first a decline in

acute digestive diseases and later an increase in appendicitis and poliomyelitis (cf. chapter six).

The current state of knowledge on historical cause-specific mortality change is still too patchy to formulate a theory that will provide an integrated formulation of the underlying processes of cause-specific mortality change. Beside that, some results suggest that the same determinant is related to different causes of death in different countries. In chapter nine, it has been argued that urbanisation had especially negative effects on mortality from acute respiratory diseases in The Netherlands as compared to mortality from acute digestive diseases in England and Wales. This would indicate that it might not be possible to formulate a theory for detailed cause-specific mortality change. So, at the moment a complete formulation of a revised epidemiological transition theory is not possible. The theory of assets for health, the health transition theory, and the perception of cause-specific mortality changes in terms of disruption of health and adaptation to health risks, enhance the understanding of mortality decline and cause-specific mortality decline, but are, in their current formulations, too general to explain or predict cause-specific mortality change. Omran's original theory can be used to estimate cause-specific mortality changes for broad cause-of-death groups in countries in which mortality decline started later than in western countries. However knowledge on determinants should be incorporated in the theory (and models) to improve projections of cause-specific mortality decline for developing countries. More research is needed on detailed cause-specific mortality change in various countries and on the determinants of mortality decline.

10.5.5 Further insight in underlying determinants of (cause-specific) mortality decline

The studies presented in this thesis can contribute to a further elucidation of the underlying processes of rise and decline of causes of death. The studies for The Netherlands focussed on mortality decline (instead of level), and on cause-specific mortality (instead of all-cause mortality). Both aspects of mortality have hardly been studied using explanatory analyses for other historical western countries. With reference to the findings for The Netherlands, three issues will be discussed in more detail: the changing importance of determinants over time, culture as a determinant of mortality decline, and cause-specific mortality decline.

The descriptive studies presented in this thesis showed that mortality decline since 1875/79 can be divided in several subphases. These phases could not only be characterised in terms of accelerations or decelerations in the pace of mortality decline, but also in terms of causes of death that dominated those phases. The results of the descriptive analysis presented in chapter seven

showed, for example, that different causes of death dominated different phases of mortality decline. In the period 1901-31 a generalised acceleration of cause-specific mortality decline could be observed, while in the period 1931-50 the most rapid accelerations in mortality were observed among airborne infectious diseases. In terms of absolute mortality decline, acute digestive diseases contributed most to mortality decline in the period 1901-31, while acute respiratory diseases contributed most in the period 1931-50. Although respiratory tuberculosis contributed most to absolute mortality decline if the whole period 1875/1992 was taken into account, in different subphases of the decline other causes of death contributed more to mortality decline. The fact that different causes of death dominate the subphases of mortality decline indicates that different *determinants* played a role in those subphases.

The idea that different determinants dominated different phases of decline came also to the fore from the explanatory analysis presented in chapter eight. Cultural factors were more important in the early years of 'the new mortality regime' (1875/79 to 1895/99), while economic factors became more important after the turn of the century. Others have also described the changing importance over time of specific determinants. Kintner (1988b) reported that the significance of illegitimacy as a determinant of infant mortality increased in the period 1871 to 1901 and declined thereafter. This changing relationship might be explained from improved care for illegitimate infants after the turn of the century. With respect to marital fertility, the significant association with infant mortality declined in Germany in the period 1871 to 1900 (Kintner 1988b). In case of Spain, changing associations of income and mortality over time have been reported. No significant (negative) associations of income with mortality were found for the late 19th century, while such associations were present in the early 20th century (Reher, unpublished paper 1998).

Another interesting finding with respect to determinants of mortality decline is, that cultural factors played a role in mortality decline. In chapter eight it was shown that Roman Catholicism and secularisation were significantly associated with mortality decline in the first decades of rapid mortality decline. There are not many historical studies for western countries in which cultural factors are taken into consideration. Studies for Germany showed that Roman Catholicism was a significant factor of infant mortality levels in Germany (Kintner 1988a, b, Vögele 1994). It has been argued that cultural factors as possible determinants of mortality decline, and fertility decline have had more attention in European countries as compared to the United States (Kirk 1996). It is true that, as far as The Netherlands is concerned, decades ago, Hofstee (1979, 1981) came up with the hypothesis that mortality decline was related to the extent to which new ideas, especially with respect to hygiene, could diffuse in a society. New ideas would be more easily accepted in modern dynamic societies compared to tradi-

tional societies. Cultural factors have also been extensively studied by the Belgian demographer Lesthaege (1983). However, cultural factors are getting more attention in research on mortality decline in other countries as well. Preston and Haines (1991), and Ewbank and Preston (1990) came up with indirect evidence of a role of cultural factors in mortality decline in the United States in the late 19th and early 20th centuries as well. In a multivariate analysis it turned out that child mortality declined faster among children of professionals and managers than among the general population. These declines turned out not to be related to higher incomes, urbanisation or race. The researchers conclude that these fast declines are consistent with a faster acceptance of improved hygiene and child-care practices among the upper class (Ewbank & Preston 1990).

Studies of determinants of mortality decline using a lower aggregation level than total mortality, e.g. age-specific or cause-specific mortality, can contribute to gain more insight in the underlying mechanisms of mortality decline. Considering multivariate explanatory studies, age-specific studies (particularly studies on infant mortality) have been conducted for a variety of countries. Cause-specific mortality decline has hardly been studied for historical western countries using multivariate explanatory analyses. In chapter eight and nine, the results of such analyses were presented for The Netherlands. The results of those analyses show that determinants of total mortality decline were significantly associated with specific underlying causes of death. The analyses in chapter eight showed, for example, that wealth tax was significantly associated with infectious diseases mortality decline in the first decades of the 20th century, but not with mortality decline from other causes of death. The two cultural variables Roman Catholicism, and secularisation were associated with different cause-of-death groups in the late 19th century. Roman Catholicism was associated with other causes of death than infectious diseases, and secularisation was associated with infectious diseases mortality decline. In chapter nine, it was shown that Roman Catholicism was significantly associated with mortality from convulsions and acute digestive diseases, and not with acute respiratory diseases, another common cause of infant mortality. In case of early childhood mortality decline, it turned out that urbanisation was significantly associated with measles and acute respiratory diseases, but not with brain diseases (i.e. mainly encephalitis/meningitis). Such cause-specific analyses enhance our understanding of the determinants of total mortality decline, and of cause-specific mortality changes.

10.6 Recommendations for further research

Four fields of research, which can further improve our understanding of the underlying processes of mortality decline in The Netherlands, and the epidemiological transition theory in general, will briefly be discussed.

10.6.1 Determinants of mortality decline in The Netherlands

Only a selection of determinants of mortality decline could be analysed in the studies presented in the former chapters. There were statistical reasons why the number of determinants in the models was limited, and for some determinants there were no adequate data available on the regional level or for the time period under study. The fact that certain determinants were not used in the models, does of course not mean that their role in mortality decline is considered limited. Possible important determinants of mortality decline, which could not be implemented in the models are, for example, female education and breastfeeding. Consistent time series of data on, for example, socio-economic status (occupation, education) are not available for the 19th and early 20th centuries in The Netherlands.

In this thesis, the analyses of determinants of mortality decline were confined to the period 1875/79 to 1920/24, the early phases of the 'new mortality regime'. Mortality decline in the period after 1920/24 until 1950 would be interesting for further research. The most rapid mortality declines were observed in The Netherlands for that period, and analyses for that period would result in a series of studies on determinants of mortality decline covering the late 19th until the late 20th centuries. As far as the period after 1950s is concerned several studies have already been conducted (Mackenbach *et al* 1988, Mackenbach *et al* 1989, Kunst *et al.* 1990).

A promising initiative, which will improve the availability of data on certain determinants, is the Historical Sample of the Population of The Netherlands (HSN) (Mandemakers 1995). The main objective of the HSN is the construction of a random sample of 0.5 percent of all men and women born in The Netherlands between 1812 and 1922 and subsequently the collection of data concerning their life-course. The strength of the HSN will be its national character. Former initiatives were, and comparable databases in other countries are, mostly regional projects, which makes generalisations to the total population difficult. The database will contain data on several determinants of mortality decline e.g. religion, marital status, occupation, female education, and will provide us with consistent data for those determinants over time. The HSN will also make analysis for the early 19th century possible, for which only studies for some large cities are available (Jansen & Meere 1982).

10.6.2 Cohort-analysis

In the studies on mortality decline for The Netherlands no attention was paid to the fact that part of the observed (cause-specific) mortality change might be explained by cohort effects. It would be interesting to analyse mortality decline with respect to period-effects and cohort-effects. Several publications have

shown that cohort effects do play a role in recent changes in cancer mortality. Cohort effects played a role in trends of stomach cancer (together with period effects), colorectal cancer, lung cancer, breast cancer, and prostate cancer (Caselli 1996, Evstifeeva *et al.* 1997). Others have observed cohort-effects in the trend of ischemic heart disease mortality. According to the 'early programming hypothesis' of Barker *et al.* (1989b), risks on ischemic heart disease in adult life are already determined in utero.

The declines in ischemic heart disease and cancer mortality are recent mortality changes. It is however likely that cohort effects also played a role during earlier mortality declines. If, for example, the time trend of height of army recruits aged twenty in The Netherlands is considered, a constant increase can be observed from 1863 onwards (Van Wieringen 1986). Height is predominantly determined in early childhood and adolescence, and is the net result of diseases, nutrition, and workload. The increasing height might, for example, reflect more favourable disease environments of consecutive cohorts. Although cohort-effects might explain part of the historical decline, period effects will have determined an important part of mortality decline. The life expectancy changes have been enormous in the past century. Life expectancy doubled in about 140 years, which is in only about 5 generations. Cohort effects should also be considered in the explanation of differences in cause- of-death pattern between countries. There were differences between Western and Eastern European countries. The cohort effect for colorectal cancer was not apparent in Western European countries, and was less strong for breast and prostate cancer in those countries as compared to the Eastern European countries (Evstifeeva *et al.* 1997).

10.6.3 International comparison of the epidemiological transition

In former sections is has been argued that countries differ with respect to cause-specific mortality change. Until now, however, a lot of (historical) research has addressed international differences in total mortality decline, but cause-specific mortality decline has been studied less frequently. It has been suggested before that it would be interesting to compare, for example, the results of the clustering of cause-specific mortality trends in The Netherlands with, to begin with, other European countries. The analysis of long-term cause-specific mortality series requires the construction of nosological cause-of-death categories as was done for The Netherlands (chapter four). In other countries but The Netherlands there have been initiatives to reclassify causes of death on a detailed level (e.g. France, Italy). Differences can be expected, considering the trends on cause-specific mortality decline presented by Caselli (1996) which showed slower declines of diarrhoeal diseases as compared to Norway and England. There might be a geographical (e.g. north-south) gradient with respect to the cause-specific mor-

tality pattern, and there might also be differences between, for example, The Netherlands and the Scandinavian countries or England. Cause-specific mortality trends should be also be analysed with respect to the change in risk factors and the adaptation mechanisms that resulted in the observed course of cause-specific mortality.

The analysis of so-called 'deviant transitions', such as the trends of ischemic heart disease in Eastern European countries is also of interest for the understanding of determinants of mortality change. Those countries followed at first the epidemiological transition of the Western European countries, but have recently shown deviations from that trend.

10.6.4 Morbidity transition

The epidemiological transition theory and the health transition theory both include morbidity and disability change too. However, a lot of research is still needed in this field before general statements on the development of morbidity and disability can be incorporated in a theory. Empirical data on the morbidity transition are lacking (or have not been studied) for many countries. Riley (1989) is one of the few researchers, who studied historical morbidity data, predominantly from England and Wales. He studied, among other things, the duration of sickness, changing disease profiles, and case-fatality rates. He concluded that mortality decline in the past hundred years was accompanied by an increase in the risk of being sick (Riley 1989). In case of The Netherlands, there has been little quantitative research on historical morbidity patterns and trends. There might be several sources worth exploring in The Netherlands, for example sickness funds. In the early 20th century there were sickness funds which were mainly located in the provinces of North and South Holland. Large private enterprises such as Philips started their own sickness funds in the early 20th century. Around the First World War, the sickness funds also spread more and more into rural areas. Other sources than health insurances, could be the municipal health services with, for example, data on child health from the school health services, or the 'cross societies' (*Kruisverenigingen*) (Van der Velden 1996a).

10.7 Policy implications

Several researchers have argued that the epidemiological transition theory should be incorporated in health care planning (Philips 1991, 1994, Bobadilla & Possas 1993). Philips (1991, 1994) stated that the concept of epidemiological transition might be used in a predictive sense viz. to anticipate future health changes. It has been shown in former sections that the epidemiological transition theory has been used to predict changes in broad cause of death groups e.g. infectious dis-

eases, cardiovascular diseases, cancer, accidents. Although the estimations are not completely accurate, projections until the year 2015 show a further increase in mortality rates from cancer and cardiovascular diseases in developed and, more strongly so, in developing countries (Bulatao 1993). Beside epidemiological changes, demographic changes are important for health policy too. Most causes of death are strongly age related, so changes in the age structure of the population will affect the burden of cause-specific mortality in a population. Murray and Lopez (1996) presented projections of the ratio of numbers of non-communicable to communicable deaths for different groups of countries. These ratios were used to indicate the progress of epidemiological and demographic transition in those countries. This ratio remains relatively stable until the year 2020 for the 'established market economies' (including The Netherlands), but will increase strongly in the 'former socialist economies' as well as in China, and there will also be an increase in other countries of the world in the next century, except for 'sub-Saharan Africa'. Such projections of the epidemiological transition are important for the planning of health services, health promotion and prevention programmes for specific diseases.

Beside the role of the epidemiological transition theory in the prediction of cause-specific mortality change, Omran's publications triggered an enormous amount of research, which in turn has improved our understanding of these changes. This knowledge can help to adapt to newly emerging health risks. So far, in the past century western countries have managed to adapt sooner or later to the newly emerging health risks. The rise and decline of causes of death is an ongoing process, so in the future we can expect new health risk to emerge to which populations have to adapt.

Especially the re-emergence of 'old' infectious diseases is gaining attention. Recently, an article has been published that infectious diseases (as primary causes of death) have been increasing in the United States in recent years (Pinner *et al.* 1996). AIDS has been on the increase in young adults, and pneumonia has been an important increasing cause of death in older age groups. Reasons for the re-emergence of infectious diseases are manifold, such as behavioural changes (e.g. intravenous drug use, medical techniques, norms and values), increased mobility (e.g. aeroplanes, war), demographic change (e.g. population density/urbanisation), exploitation of the physical environment (e.g. change in agricultural and industrial techniques), decreased attention to infectious diseases (e.g. breakdown of infrastructure, loss of knowledge), and changes in viruses (e.g. mutation, recombination). It has been argued that the ecological effects of human acting, e.g. disruption of the habitat of apes, played a role in the emergence of AIDS in the human population (Goudsmit 1997). Especially increased mobility is considered to be an important factor (Osterhaus 1997). Also changes in non-infectious disease patterns can be expected. In the United States cancer in-

cidence for children under age 15 has been increasing since the early 1970s, which has been ascribed to toxic chemicals in the environment (Horiuchi 1997).

As far as The Netherlands is concerned, on the basis of mortality developments in recent years, a further increase of mortality from prostate cancer for males and an increase of mortality from lung cancer and CARA can be expected in the near future (Tabeau & Huisman 1997). With respect to communicable diseases, resistance of microorganisms to antibiotics is worldwide an increasing problem. In The Netherlands, it is still a minor problem, because of a restrictive policy of antibiotics use. However, the immigration of patients from countries in which resistance is prevalent might enlarge the problem in the future. Other possible increasing infectious diseases are malaria and typhoid fever, due to travelling, and hospital infections and sepsis, due to invasive diagnostic en therapeutic techniques and resistance to antibiotics (Van den Berg Jeths *et al.* 1997).

In order to be able to adapt adequately to newly emerging diseases early recognition of diseases that are on the increase, as well as knowledge of the determinants of the disease is necessary. With respect to infectious diseases, surveillance of the resistance of infectious diseases to antibiotics is important. Possibly increasing infectious diseases, such as malaria, typhoid fever and tuberculosis are currently carefully surveyed in The Netherlands (Van den Berg Jeths *et al.* 1997). Another example of the surveillance of infectious diseases is the national registration system of hospital infections that has been set up. Monitoring of known risk factors of causes of death is also important to be able to adequately respond to, or prevent a renewed increase of causes of death. Smoking behaviour and alcohol consumption among teenagers, which has both increased in recent years, will lead to an increase of cardiovascular and respiratory diseases in the future if there is no adequate response to this trend. Epidemiologists can play an important role in contributing to the adaptation to new health risks by unravelling the determinants of diseases and causes of death, as well as research on adaptation strategies such as early diagnosis (e.g. screening on breast cancer), and health promotion (e.g. life style factors).

REFERENCES

Aaby P, Bukh J, Lisse IM, Smiths AJ. Overcrowding and intensive exposure as determinants of measles mortality. American Journal of Epidemiology 1984, 120: 49-63

Armstrong WA. The trend of mortality in Carlisle between the 1780s and the 1840s: A demographic contribution to the standard of living debate. Economic History Review 1981, 94-114

Astro PV. *Statistiek der carcinoomsterfte in de gemeente Utrecht gedurende het tijdvak 1872-1902.* Wentzel & Co., Utrecht 1902

Bah SM, Rajulton F. Has Canadian mortality entered the fourth stage of epidemiological transition? Canadian Studies in Population 1991, 18: 18-41

Bah SM. Indirect Estimation of cause of death structure in Africa and contemporary theories of mortality. Social Biology 1995, 42 (Fall-Winter): 247-255

Barker DJP. Acute appendicitis and dietary fibre: an alternative hypothesis. Br Med J 1985, 290: 1125-27

Barker DJP, Osmond C, Golding J, Wadsworth MEJ. Acute appendicitis and bathrooms in three samples of British children. Br Med J 1988, 296: 956-958

Barker DJP. Rise and fall of Western diseases. Nature 1989, (338) March: 371-372

Barker DJP, Osmond C, Winter PD, Margetts B. Weight in infancy and death from ischaemic heart disease. Lancet 1989, 577-580

Beaver S.E. *Demographic transition theory reinterpreted.* Lexington Books, Lexington (Mass) 1975

Beneke FW. *Vorlagen zur Organisation der Mortalitäts-Statistik in Deutschland.* N.G. Elwertsche Verlagsbuchhandlung, Marburg 1875

Bengtsson T, Fridlizius G, Ohlsson R. *Pre-Industrial Population Change.* Almquist and Wiksell International, Stockholm 1984

Berreklouw S. De ontwikkeling van de cardiologie in de laatste zestig jaren. In: Van Dongen JA (ed.). *De vooruitgang van de geneeskunde in onze eeuw.* De Bussy, Amsterdam 1966

Berry B, Malt RA. Appendicitis near its centenary. Annals of Surgery 1984, 200: 567-575

Bertillon J. *Nomenclatures des maladies arretées par la Commission international chargée de reviser les nomenclatures nosologiques (18-21 aout 1900) pour etre en usage à usage pairir de 1er janvier 1901 avec notices et annexes.* Imprimerie typographique de l'école d'Alembert, Montévain 1903

Bicego GT, Boerma JT. Maternal education and child survival. A comparative study of survey data from 17 countries. Social Science and Medicine 1993, 36: 1207-1227

Blane D. Real wages, the economic cycle, and mortality in England and Wales, 1870-1914. International Journal of Health Services 1990, 20: 43-52

Blok L, De Meere JMM. Welstand, ongelijkheid in welstand en censuskiesrecht in Nederland omstreeks het midden van de 19e eeuw. Economisch- en Sociaal-historisch Jaarboek 1978, 41: 175-290

Bobadilla JL, Possas C. Health policy issues in three Latin American countries: implications of the epidemiological transition. In : Gribble JN, Preston SH (eds.). *The epidemiological transition. Policy and planning implications for developing countries.* National Academy Press, Washingtion DC 1993

Bofetta P, La Vecchia C, Levi F, Lucchini F. Mortality patterns and trends for lung cancer and other tobacco related causes in the America's, 1955-1989. Int J Epid 1993, 22: 377-384

Boonstra OWA, Van der Woude AM. Demographic transition in The Netherlands. A statistical analysis of regional differences in the level and development of the birth rate and of fertility, 1850-1890. AAG Bijdragen 1984, 24: 1-57

Boonstra OWA. *De waardij van eene vroege opleiding.* Landbouw Universiteit/Uitgeverij Verloren, Wageningen/Hilversum 1993

Brändström A. The impact of female labour conditions on infant mortality: A case study of the parishes of Nedertorneå and Jokkmokk, 1800-96. Social History of Medicine 1988, 1: 329-358

Breslow L, Enstrom JE. Persistence of health habits and their relationship to mortality. Prev Med 1980, 9: 469-483

Brinkman HJ, Drukker JW, Slot B. Lichaamslengte en reëel inkomen: een nieuwe schattingsmethode voor historische reeksen. Economisch en Sociaal Historisch Jaarboek 1988, 51: 35-79

Broudy DW, May PA. Demographic and epidemiologic transition among the Navajo Indians. Social Biology 1983, 30: 1-16

Bryder L. 'Not always one and the same thing': The registration of tuberculosis death in Britain, 1900-1950. Social History of Medicine 1996, 9: 253-265

Bulatao RA. Mortality by cause 1970 to 2015. In: Gribble JN, Preston SH (eds.). *The epidemiological transition. Policy and planning implications for developing countries.* National Academy Press, Washington DC 1993

Burgmeijer R, Bolscher N (eds). *Vaccinaties bij kinderen. Uitvoering en achtergronden van het rijksvaccinatieprogramma en andere vaccinaties bij kinderen.* Van Gorcum, Assen 1995

Burnett J. Housing and the decline of mortality. In: Schofield R, Reher D, Bideau A (eds.). *The decline of mortality in Europe.* Clarendon Press, Oxford 1991

Butler NR, Alberman ED. *Perinatal problems; the second report of the 1958 British Perinatal Mortality Survey.* Livingstone, Edinburgh 1958

Caldwell JC. Towards a restatement of demographic transition theory. Population Development Review 1976, 23: 321-366

Caldwell JC. Education as a factor in mortality decline. An examination of Nigerian data. Population Studies 1979, 33: 395-413

Caldwell JC. Routes to low mortality in poor countries. Population Development Review 1986, 12: 205-

Caldwell JC. Introductory thoughts on health transition. In: Caldwell JC et al. (eds). *What we know about health transition: the cultural, social and behavioural determinants of health. The proceedings of an international workshop.* Canberra, May 1989

Campbell H, Chiang R, Hansluwka H. Cancer mortality in Europe. Patterns and Trends, 1955-1974. World Health Stat Q 1980, 33: 152-184

Campbell M. Death rates from diseases of the heart: 1876 to 1959. Br Med J 1963, 31 August: 528-535

Caselli G. Health transition and cause-specific mortality. In: Schofield R Reher D, Bideau (eds.). *The decline of mortality.* Clarendon Press, Oxford 1991

Caselli G. National differences in the health transition in Europe. Historical Methods 1996, 29: 107-125

Centraal Bureau voor de Statistiek (CBS). *Bijdragen tot de Statistiek van Nederland. Onderzoek naar het gebruik van sommige voedings- en genotmiddelen.* Van Weelden en Mingelen, The Hague 1895.

Centraal Bureau voor de Statistiek (CBS). *Bijdragen tot de statistiek van Nederland. Uitkomsten der zesde, zevende, achtste, negende en tiende volkstelling in de jaren 1879, 1889, 1899, 1910, 1920.* Belinfante, Den Haag.

Centraal Bureau voor de Statistiek (CBS). *Jaarcijfers voor het Koninkrijk der Nederlanden. Rijk in Europa.* Belinfante, The Hague 1900-1917

Centraal Bureau voor de Statistiek (CBS). *Statistiek van de sterfte onder mannen met onderscheiding naar het beroep en in verband met leeftijden en doodsoorzaken.* Bijdragen tot de statistiek van Nederland no. 172., 's-Gravenhage 1912

Centraal Bureau voor de Statistiek (CBS). *Maandschrift van het Centraal Bureau voor de Statistiek. Onderzoek naar het verbruik van sommige voedings- en genotmiddelen 1892-1918.* N.V. Drukkerij Trio, The Hague 1920

Centraal Bureau voor de Statistiek (CBS). *Jaarcijfers voor het Koninkrijk der Nederlanden.* Belinfante/SDU uitgeverij, The Hague 1900-1940.

Centraal Bureau voor de Statistiek (CBS). *Groote internationale lijst van doodsoorzaken 1935.* Unpublished report. Centraal Bureau voor de Statistiek, Den Haag 1935

Centraal Bureau voor de Statistiek (CBS). *Groote internationale lijst van doodsoorzaken 1938.* Unpublished report. Centraal Bureau voor de Statistiek, Den Haag 1940

Centraal Bureau voor de Statistiek (CBS). *Vijfennegentig jaren statistiek in tijdreeksen.* SDU uitgeverij, The Hague 1994

Chen LC, Wittgestein F, McKeon E. The upsurge of mortality in Russia: causes and policy implications. Population and Development Review 1996, 22: 517-530

Chesnais J-C. *The demographic transition. Stages, patterns and economic implications.* Clarendon Press, Oxford 1992

Cliquet RL. De tweede demografische transitie: feit of fictie?. Bevolking en Gezin 1991, 2: 1-42

Cleland J, Van Ginneken J. Maternal education and child survival in developing countries: the search for pathways of influence. Social Science and Medicine 1988, 27: 1357-1368

Cleland J. The idea of the health transition. In: Caldwell JC et al. (eds.). *What we know about health transition: the cultural, social and behavioural determinants of health. The proceedings of an international workshop.* Canberra, May 1989

Coale AJ, Cotts Watkins S (eds.). *The decline of fertility in Europe.* Princeton University Press, Princeton New Yersey 1986

Coale AJ, Treadway R. A summary of the changing distribution of overall fertility, marital fertility and the proportion married in the provinces of Europe. In: Coale AJ, Cotts Watkins S. *The decline of fertility in Europe.* Princeton University Press, Princeton New Jersey 1986

Coale AJ. The decline of fertility in Europe since the eighteenth century as a chapter in demographic history. In: Coale AJ, Cotts Watkins S. *The decline of fertility in Europe.* Princeton University Press, Princeton New Jersey 1986

Cole P, Rodu B. Declining cancer mortality in the United States. Cancer 1996, 78: 2045-2048

Condran GA, Cheney RA. Mortality trends in Philadelphia: age-and cause-specific death rates 1870-1930. Demography 1982, 19: 97-123

Coronel S. Over den invloed der woningruimte op de gezondheid der bewoners. Nederlands Tijdschrift voor Geneeskunde 1875, 11: 201-202

Coronel S. De volksvoeding in Friesland. Nederlands Tijdschrift voor Geneeskunde 1876, 12 (II): 103-129

Corsini CA, Viazzo PP (eds.). *The decline of infant and childhood mortality. The European experience: 1750-1990.* Martinus Nijhoff Publishers, Dordrecht 1997

Crews DE. Multiple cause of death and the epidemiologic transition in American Samoa. Social Biology 1988, 35: 198-211

Crimmins EM. The changing pattern of American mortality decline, 1940-1977, and its implications for the future. Population Development Review 1981, 7: 229-254

De Beer JAA. Omslagpunten in demografische trends, Maandstatistiek van de Bevolking 1986, 3: 29-33

De Beer JJA. De levensstandaard in Nederland. Voeding en gezondheid in de eerste helft van de negentiende eeuw. Tijdschrift voor Sociale Geschiedenis 1996, 22: 24-52

De Grote Bosatlas, 46e druk. Wolters-Noordhoff, Groningen 1968

De Knecht-van Eekelen A. Ontwikkeling van de schoolgeneeskunde in Nederland (1796-1904). Gewina 1994, 17: 219-234

De Haan J. De toeneming der sterfte aan kanker, Nederlands Tijdschrift voor Geneeskunde 1899, 35: 963-968

De Jonge JA. Industrialisatie in Nederland tussen 1850 en 1914. Thesis, 1968

De Jonge JA. The role of the outer provinces in process of Dutch economic growth in the nineteenth century. In: Bromley JS, Kossmann EH (eds.). *Britain and The Netherlands.* Volume IV p. 208-225. Den Haag 1971

Dixon H. The work of WHO on the ICD in the Postwar Era, In: *The Centenary of the International Classification of Diseases (1893-1993).* ISI Occasional paper series 1993, nr.3: 17-22

Drooglever Fortuyn HJW. De sterftelijn in de laatste driekwart eeuw, Nederlands Tijdschrift voor Geneeskunde 1924, 60: 53-63

Escovitz GH. The health transition in developing countries: a role for internists from the developed world. Annals of Internal Medicine 1992, 116: 499-504

Evans J, Lashman Hall K, Warford J. Shattuck lecture - Health care in the developing world: Problems of scarcity and choice. New England Journal of Medicine 1981, 305: 1117-1127

Evers JCG. Bijdrage tot de bevolkingsleer van Nederland. Belinfante, The Hague 1882

Evstifeeva TV, MacFarlane GJ, Robertson C. Trends in cancer mortality in central European countries. European Journal of Public Health 1997, 7:169-176

Ewbank DC, Preston SH. Personal health behaviour an d the decline in infant and child mortality: the United States, 1900-1930. In: Caldwell JC et al. (eds.). *What we know about health transition. Health Transition* Series 1990, no 2., volume I

Feachem R. Health decline in Eastern Europe. Nature 1994, 367: 313-314

Fischer-Homberg E. Geschichte der Medizin. Springer Verlag, Berlin 1977

Flegg AT. Inequality in income, illiteracy and medical care as determinants of infant mortality in underdeveloped countries. Population Studies 1982, 36: 441-453

Fliess KH. Mortality transition among the Wends of Serbin, Texas, 1854-1884: change in pattern of death. Social Biology 1991, 38: 266-276

Floud R, Wachter K, Gregory A. *Height, health and history. Nutritional status in the United Kingdom, 1750-1980.* Cambridge University Press, Cambridge 1990

Fogel RW, Engerman SL, Floud R, Friedman G, Margo RA, Sokoloff K e.a. Secular changes in American and British stature and nutrition. Journal of Interdisciplinary History 1983, XIV: 445-481

Fogel RW. New sources and new techniques for the study of secular trends in nutritional status, health, mortality and the process of aging. Historical Methods 1993, 26: 5-43

Forste R. The effects of breastfeeding and birth spacing on infant and child mortality in Bolivia. Populations Studies 1994, 48: 497-511

Frankenberg E. The effects of access to health care on infant mortality in Indonesia. Health Transition Review 1995, 5: 143-163

Frenk J, Bobadilla J, Sepulveda J, Lopez-Cervantes M. Health transition in middle-income countries: New challenges for health care. Health Policy and Planning 1989, 4: 29-39

Frenk J, Bobadilla JL, Stern C, Frejka T, Lozano R. Elements for a theory of the health transition. Health transition review 1991, 1: 21-38

Fridlizius G. The mortality decline in the first phase of the demographic transition: Swedish experiences. In: Bengtsson T, Fridlizius G, Ohlsson R (eds.). *Pre-industrial population change.* Almquist and Wiksell International, Stockholm 1984

Gage TB. The decline of mortality in England and Wales 1861 to 1964: decomposition by cause of death and component mortality. Population Studies 1993, 47: 47-66

General Register Office. *Manual of the International list of Causes of Death - second revision.* Her Majesty's Stationery Office, London 1912

Goudsmit J. *Vrijend virus. Over aard en oorsprong van het Aidsvirus.* Contact, Amsterdam 1997

Graham D. Female employment and infant mortality: some evidence from British towns, 1911, 1931 and 1951. Continuity and Change 1994, 2: 313-346

Grmek M. *Diseases in the ancient Greek world.* John Hopkins University Press, Baltimore 1991

Groen AS. Zestig jaar longziekten. In: Van Dongen JA (ed.). *De vooruitgang der geneeskunde in onze eeuw.* De Bussy, Amsterdam 1966

Grootte PD. *Kapitaalvorming in infrastructuur in Nederland, 1800-1913.* Labyrinth Publications, Capelle a/d IJssel 1995

Gulliford MC. Epidemiological transition in Trinidad and Tobago, West Indies 1953-1992. International Journal of Epidemiology 1996, 25: 357-365

Haakma Tresling T. Onderzoek naar de volksvoeding in Nederland. Nederlands Tijdschrift voor Geneeskunde 1876, 12 (II): 1-53

Haines MR. Conditions of work and the decline of mortality. In: Schofield R, Reher D, Bideau A (eds.). *The decline of mortality in Europe.* Clarendon Press, Oxford 1991

Haines MR. Socio-economic differentials in infant and child mortality during mortality decline: England and Wales, 1890-1911. Population Studies 1995, 49: 297-315

Haines MR, Kintner HJ. The mortality transition in Germany, 1869-1935. Paper presented at the International Population Conference Montreal 1993, Liege IUSSP

Hall RE, Johnston J, Lilien DM. *Micro TSP User's Manual.* Quantitative Micro Software, Irvine California 1990

Han AM, Khin MM, Aye T, Hlaing T. Measles-associated diarrhoea in the infectious diseases hospital, Rangoon. J Tropic Med Hygiene 1990, 93: 205-209

Hardy A. Diagnosis, death and diet: the case of London, 1750-1909. Journal of Interdisciplinary History 1988, 19: 387-401

Hardy A. *The epidemic streets. Infectious disease and the rise of preventive medicine, 1856-1900.* Clarendon Press, Oxford 1993

Harris B. Health, height and history: an overview of recent developments in anthropometric history. Social History of Medicine 1994, 7: 297-320

Hempel CG. *Filosofie van dn natuurwetenschappen.* Het Spectrum, Utrecht 1966

Hobcraft J. Women's education, child welfare and child survival: a review of the evidence. Health Transition Review 1993, 3: 159-175

Hofstee EW. *De demografische ontwikkeling van Nederland in de eerste helft van de negentiende eeuw.* Van Loghum Slaterus, Deventer 1979

Hofstee EW. *Korte demografische geschiedenis van Nederland van 1800 tot heden.* Fibula-Van Dishoeck, Haarlem 1981

Hofstee EW. Geboorten, zuigelingenvoeding en zuigelingensterfte in hun regionale verscheidenheid in de 19e eeuw. Bevolking en Gezin 1983, supplement: 7-60

Horiuchi S. Epidemiological transitions in developed countries: past, present and future. Paper presented at the Symposium on Health and Mortality. Brussels, November 1997

Houwaart ES. *De hygiënisten. Artsen, staat en volksgezondheid in Nederland 1840-1890.* Historische Uitgeverij Groningen, Groningen 1991

Huck P. Infant mortality in nine industrial parishes in Northern England, 1813-1836. Population Studies 1994, 48: 513-526

Huck P. Infant mortality and living standards of English workers during the industrial revolution. Journal of Economic History 1995, 55: 528-550

Huffman S, Lamphere B. Breastfeeding performance and child survival. In: Mosley WH, Chen LC (eds.). *Child survival. Strategies for research.* Population Development Review 1984, 10 supplement p. 93-116

Inspecteurs van het geneeskundig staatstoezicht. *Jaarverslag 1898.* Belinfante, Den Haag 1900

Jansen PC, De Meere JMM. Het sterftepatroon in Amsterdam 1774-1930. Een analyse van doodsoorzaken. Tijdschrift voor Sociale Geschiedenis 1982, 26: 180-223

Jayachandran J, Jarvis GK. Socio-economic development, medical care, and nutrition as determinants of infant mortality in less-developed countries. Social Biology 1986, Fall-Winter: 301-315

Jobse-van Putten J. De zelfvoorziening in ruimtelijk perspectief. Een cultuurhistorische vergelijking van regionale indelingen in Nederland. Volkskundig Bulletin 1990, 16: 21-47

Johansen HC. The Development of Reporting Systems for Causes of Death in Denmark. Paper presented at the Conference on the History of Registration of Causes of Death. November 11-14. Bloomington, Indiana 1993

Johansson S. Ryan. Measuring the cultural inflation of morbidity during the decline in mortality. Health Transition Review 1992, 2: 78-89

Jongman RHJ, Ter Braak CJF, Van Tongeren OFR (eds.). *Data analysis in community and landscape ecology.* Cambridge University Press, Cambridge 1995

Junge B, Hoffmeister H. Civlization-associated diseases in Europe and industrial countries outside Europe: regional differences in trends in mortality. Preventive Medicine 1982, 11: 117-130

Kendall MG. *Time-Series.* Griffin, London 1973

Kintner HJ. Classifying causes of death during the late nineteenth and early twentieth centuries: the case of German infant mortality. Historical Methods 1986, 19: 45-54

Kintner HJ. The impact of breastfeeding patterns on regional differences in infant mortality in Germany, 1910. European Journal of Population 1988a, 2: 223-261

Kintner HJ. Determinants of temporal and areal variation in infant mortality in Germany, 1871-1933. Demography 1988b, 25: 597-609

Kintner HJ. Recording the epidemiologic transition in Germany, 1816-1934. Paper presented at 'The conference on the history of registration of causes of death'. November, Bloomington 1993

Kirk D. Demographic transition theory. Population Studies 1996, 50: 361-387

Kleinman JC, Feldman JJ, Monk MA. The effects of Changes in Smoking Habits on Coronary Heart Disease Mortality. Am J Public Health 1979, 69: 795-802

Kleipool CM. *Een kritische beschouwing over beroepsziekte- en beroepssterfte-statistiek.* Thesis, 1912

Knippenberg H. De toenemende rol van de overheid in Nederland, 1850-1940. Een analyse van de regionale verschillen in de uitgaven van gemeentelijke overheden. Unpublished paper. Sociaal Geografisch Instituut, Universiteit van Amsterdam 1980

Knippenberg H, De Pater B. *De eenwording van Nederland. Schaalvergroting en integratie sinds 1800.* SUN, Nijmegen 1988

Kok J, Van Poppel F, Kruse E. Mortality among illegitimate children in mid-nineteenth-century The Hague. In: Corsini CA, Viazzo PP (eds.). *The decline of infant and child mortality. The european experience: 1750-1990.* Martinus Nijhoff Publishers, Dordrecht 1997

Kunitz SJ. The personal physician and the decline of mortality. In: Schofield R, Reher D, Bideau A (eds.). *The decline of mortality in Europe.* Clarendon Press, Oxford 1991

Kunst AE, Looman CWN, Mackenbach JP. Socio-economic mortality differences in The Netherlands in 1950-1984: a regional study of cause-specific mortality. Social Science and Medicine 1990, 31: 141-152

Kunst AE. *Cross-national comparisons of socio-economic differences in mortality.* Thesis, Rotterdam 1997

Lancaster HO. *Expectations of life: A study in the demography, statistics and history of world mortality.* Springer-Verlag, New York 1990

LaVecchia C et al. Trends of cancer mortality in Europe, 1955-1989: I. Digestive sites. Eur J Cancer 1992, 28: 132-235

LeGrand TK, Philips JF. The effect of fertility reductions on infant and child mortality: evidence from Matlab in Rural Bangladesh. Population Studies 1996, 50: 51-68.

Lesthaeghe RJ. *The decline of Belgian fertility, 1800-1970.* Princeton 1977

Lesthaeghe R. A century of demographic and cultural change in Western Europe. An exploration of underlying dimensions. Population and Development Review 1983, 9: 411-

Lesthaeghe R. De borstvoeding als verklaring voor regionale verschillen in vruchtbaarheid en zuigelingensterfte: Nederland en Belgie in het midden van de XIXe eeuw. Bevolking en Gezin 1983, supplement: 61-84

Lesthaeghe R, Van de Kaa DJ. 'Twee demografische transities?'. In: Van de Kaa DJ, Lesthaeghe R (eds.). *Bevolking: Groei en Krimp.* Van Loghum Slaterus, Deventer 1986

Lesthaeghe R, Wilson C. Modes of production, secularization and the pace of fertility decline in Western Europe, 1870-1930. In: Coale A, Watkihs S. *The decline of fertility in Europe.* Princeton University Press, New Jersey 1986

Levison CH, Hastings DW, Harrison JN. The epidemiologic transition in a Frontier Town - Manti, Utah: 1849-1977. American Journal Physical Anthropology 1981, 56: 83-93

Logan WPD. Mortality in England and Wales from 1848 to 1947. Pop Studies 1950, 4: 132-178

Lopez AD, Hull TH. A note on estimating the cause of death structure in high mortality populations. Population Bulletin United Nations 1982, 14

Loudon I. *Death in childbirth. An international study of maternal care and maternal mortality 1800-1950.* Clarendon Press, Oxford 1992

Lowry S. *Housing and Health.* British Medical Journal, London 1991

Lumey LH, Van Poppel FWA. The Dutch Famine of 1944-45: mortality and morbidity in past and present generations. Social History of Medicine 1994, 7: 229-246.

Mackenbach JP. *Mortality and Medical Care.* Thesis, Leiden 1988

Mackenbach JP, Kunst AE, Looman CWN, Habbema JDF, Maas PJ van der. Regional differences in mortality from conditions amenable to medical intervention in The Netherlands: a comparison of four time-periods. Journal of Epidemiology and Community Health 1988, 42: 325-332

Mackenbach JP, Looman CWN. Secular trends of infectious disease mortality in The Netherlands, 1911-1978: Quantitative estimates of changes coinciding with the introduction of antibiotics. International Journal of Epidemiology 1988, 17: 618-624

Mackenbach JP, Looman CWN, Kunst AE. Geographical variation in the onset of decline of male ischemic heart disease mortality in The Netherlands. American Journal of Public Health 1989, 79: 1621-1627

Mackenbach JP, Snels IAK, Friden-Kill LM. Diabetes mellitus als doodsoorzaak. Ned Tijdschr Geneeskd 1991, 135: 1492-1496

Mackenbach JP. *De veren van Icarus. Over de achtergronden van twee eeuwen epidemiologische transities in Nederland.* Bunge, Utrecht 1992

Mackenbach JP. De epidemiologische transitie in Nederland. Ned Tijdschr Geneeskd 1993a, 137: 132-138

Mackenbach JP. De achtergronden van de sterftedaling tussen 1875/79 en 1970 in Nederland. T Soc Gezondheidsz 1993b, 71: 192-197

Mackenbach JP. The contribution of medical care to mortality decline: McKeown revisited. Journal of Clinical Epidemiology 1996, 49:1207-1213

Mackenbach JP. The epidemiologic transition theory (editorial). Journal of Epidemiology and Community Health 1994, 48: 329-331

Magner LN. *A History of Medicine.* Marcel Dekker inc., New York 1992

Mandemakers K, Boonstra O (eds.). *De levensloop van de Utrechtse bevolking in de 19e eeuw.* Van Gorcum, Assen1995

Mandemakers CA. *Gymnasiaal en middelbaar onderwijs. Ontwikkeling, structuur, sociale achtergrond en schoolprestaties, Nederland 1800-1968.* Thesis, Rotterdam 1996

Marland H. Questions of competence: the midwife debate in The Netherlands in the early twentieth century. Medical History 1995, 39: 317-337

Marshall M. The second fatal impact: cigarette smoking, chronic disease, and the epidemiological transition in Oceania. Social Science and Medicine 1991, 33: 1327-1342

Martyn CN, Barker DJP. Motoneuron disease and past poliomyelitis in England and Wales. Lancet 1988, June 11: 1319-1321

McFarlane N. Hospitals, housing and tuberculosis in Glasgow, 1911-51. Social History of Medicine 1989, 2: 59-85

McKeown T, Record RG. Reasons for the decline of mortality in England and Wales during the nineteenth century. Population Studies 1962, 16: 94-122

McKeown T, Brown RG, Record RG. The modern rise of population in Europe. Population Studies 1972, 26: 345-382

McKeown T, Record RG, Turner RD. An interpretation of the decline of mortality in England and Wales during the twentieth century. Population Studies 1975, 29: 391-422

McKeown ThF. *The modern rise of population.* Edward Arnold, London 1976a

McKeown ThF. *The role of medicine - dream, mirage or nemesis?* London: Nuffield Provincial Hospitals Trust, 1976

McNeill WH. *Plagues and Peoples.* Anchor press, Garden City New York 1976

McNeill DR, Trussell TJ, Turner JC. Spline interpolation of demographic data. Demography 1977, 14: 245-252.

Mercer A. *Disease, mortality and population in transition.* Leicester University Press, Leicester 1990

Mercer AJ. Smallpox and epidemiological-demographic change in Europe: the role of vaccination. Population Studies 1985; 39: 287-307

Minister van Binnenlandse Zaken. Brief aan de Inspecteurs van het Geneeskundig Staatstoezicht. Den Haag 1876

Ministère des affaires etrangères. *Nomenclatures internationales des maladies et causes de décès - troisième session.* Imprimerie Coquemard, Paris 1920

Mitchell A. An inexact science: the statistics of tuberculosis in late nineteenth-century France. Social History of Medicine 1990, 3: 387-403

Mitchell A. Tuberculosis statistics and the McKeown thesis: A rebuttal to David Barnes. Socal History of Medicine 1992, 5: 291-296

Mosley WH, Chen LC. An analytical framework for the study of child survival in developing countries. In:

Mosley WH, Chen LC (eds.). *Child survival. Strategies for research.* Population Development Review 1984, 10 supplement p.25-45

Murray CJL, Chen LC. Understanding morbidity change. Population Development Review 1992, 18: 481-503

Murray CJL, Chen LC. Understanding morbidity change: reply to Riley. Population Development Review 1993, 19: 812-815

Murray CJL, Chen LC. In search of a contemporary theory for understanding mortality change. Social Science and Medicine 1993, 36: 143-155

Murray CJL, Lopez AD. Mortality by cause for eight regions of the world: Global Burden of Disease Study. Lancet 1997, 349: 1269-1276

Murray CJL, Lopez AD (eds.). *The global burden of disease.* Chapter 7. Harvard University Press 1996

Nelson MC, Rogers J. Cleaning up the cities: application of the first comprehensive public health law in Sweden. Scandinavian Journal of History 1994, 19: 17-39

Norusis MJ. *SPSS Base System User's Guide.* SPSS inc. 1990

Notestein FW et al. *The future population of Europe and the Soviet-Union: Population projections, 1940-1970.* Geneva, 1944

Notestein FW. Economic problems of population change. In: Proceedings of the Eighth International Conference of Agriculture Economists. New York, 1953

Olshansky SJ, Ault AB. The fourth stage of the epidemiologic transition: the age of delayed degenerative diseases. Millbank Memorial Fund Quaterly 1986, 64: 355-391

Omran AR. The epidemiologic transition; a theory of the epidemiology of population change; Millbank Memorial Fund Quarterly 1971, 49: 509-538

Omran AR Epidemiologic transition in the United States: The health factor in population change, Population Bulletin 1980, 32: whole issue

Omran AR. The epidemiologic transition theory. A preliminary update, Journal of Tropical Pediatrics 1983, 29: 305-316

Omran AR. A century of epidemiologic transition in the United States. Preventive Medicine 1977a, 6: 30-51

Omran AR. Epidemiologic transition in the United States; the health factor in population change. Population Bulletin 1977b, 32: 3-42

Omran AR. The epidemiologic transition theory. In: Population Bulletin United Nations 1993, 48 no. 1

Onnen PW. De gezondheidstoestand der 12 grootste gemeenten van Nederland. Geneeskundige Bladen 1895, 2e serie: 237-264

Oomens C.A. *De loop der bevolking van Nederland in de negentiende eeuw.* CBS statistische onderzoekingen M35. SDU, The Hague 1989

Osterhaus ADME. Virologie in Rotterdam: een blijvende uitdaging. Mediator 1997, 8: 10-12

Palloni A, Millman S. The effects of breastfeeding and pace of childbearing on mortality at early ages. Demography 1986, 23: 31-52

Palloni A, Wyrick R. Mortality decline in Latin America: changes in the structure of causes of death, 1950-1975. Social Biology 1981, 28: 187-216

Perrenoud A. The mortality decline in a long-term perspective. In: Bengtsson T, Fridlizius G, Ohlsson R (eds.). *Pre-industrial population change.* Almquist and Wiksell International, Stockholm 1984

Perrenoud A. Atténuation des Crises et Déclin de la Mortalité, Annales de Démographie Historique, 1989

Philips DR. Problems and potential of researching epidemiological transition: examples from southeast asia. Soc.Sci.Med. 1991, 33: 395-404

Philips DR. Does epidemiological transition have utility for health planners? Soc.Sci.Med. 1994, 38: vii-x

Philips JFR. Gezondheidszorg in Limburg. Groei en acceptatie van de gezondheidsvoorzieningen 1850-1940. Van Gorcum, Assen 1980

Pinner RW, Teutsch SM, Simonsen L, Klug LA, Graber JM, Clarke MJ, Berkelman RL. Trends in infectious diseases mortality in the United States. JAMA 1996, 275: 189-193

Popkin BM. Nutritional patterns and transitions. Population and Development Review 1993, 19: 138-157

Preston S, Nelson VE. Structure and changes in causes of death: an international summary. Population Studies 1974, 28: 19-31

Preston SH. The changing relation between mortality and level of economic development. Population Studies 1975, 29: 231-48

Preston SH, Van de Walle E. Urban french mortality in the nineteenth century. Population Studies 1978, 32: 275-297

Preston SH. (ed.). The effects of infant and child mortality on fertility. Academic Press, New York 1978

Preston SH, Haines MR. Fatal years. Child mortality in late nineteenth-century America. Princeton University Press, Princeton 1991

Quain R. *A dictionary of Medicine.* London, 1883

Querido A. *The development of socio-medical care in The Netherlands.* Routledge & Kegan Paul Ltd, London 1968

Razzell PE. 'An interpretation of the modern rise of population in Europe' - A critique. Population Studies 1974, 28: 5-17

Registrar-General. *38th Annual Report of Birth, Death and Marriages in England (abstracts of 1875).* Her Majesty's Stationery Office, London 1877

Registrar-General. *64th Annual Report of Birth, Death and Marriages in England (abstracts of 1901).* Her Majesty's Stationery Office, London 1901

Reves R. Declining fertility in England and Wales as a major cause of the twentieth century decline in mortality. American Journal of Epidemiology 1985, 122: 112-126

Rigter H, Rigter RBM. Volksgezondheid: een assepoester in de Nederlandse politiek. Gewina 1993, 16: 1-17

Riley J, Alter G. The epidemiologic transition and morbidity. Annales de Démographie Historique 1989: 199-213

Riley JC. *Sickness, recovery and death: A history and forecast of ill health.* University of Iowa Press, Iowa City 1989

Riley J. The risk of being sick: morbidity trends in four countries. Population and Development Review 1990, 16: 403-432

Riley J. From a high mortality regime to a high morbidity regime: is culture everything in sickness? Health Transition Review 1992, 2: 71-78

Riley JC. Understanding morbidity change: comment on an article by Murray and Chen. Population Development Review 1993, 19: 807-811

Rodgers GB. Income and inequality as determinants of mortality: an international cross-sectional analysis. Population Studies 1979; 33: 343-51

Rogers J. Reporting causes of death in Sweden 1750-1930: the relationship between the development of a classification system and its practical implementation. Paper presented at 'The conference on the history of registration of causes of death'. November, Bloomington 1993

Rogers RG, Hackenberg R. Extending epidemiologic transition theory: A new stage. Social Biology 1987, 34: 234-43

Rosen G. *A History of Public Health.* John Hopkins University Press, Baltimore/London 1993

Rutten W. *'De vreselijkste aller harpijen.' Pokkenepidemieen en pokkenbestrijding in Nederland in de achttiende en negentiende eeuw: een sociaal-historische en historisch-demografische studie.* A.A.G. Bijdragen nr. 36, Wageningen 1997

Rutten WJMJ. Mortaliteit en medicalisering. Een regionaal differentiele analyse van de sterfte zonder geneeskundige behandeling in Nederland (ca. 1870-

1900). In: *Holland en de historische demografie.* Historische Studien 16, Dordrecht 1985

Saltet RH. Einiges über die Statistik der Todesursachen in den Niederlanden, Hygienische Rundschau 1895, 5: 493-505

Saltet RH. De sterfte aan besmettelijke ziekten in Nederland en in Amsterdam van 1866-1905. Nederlands Tijdschrift voor Geneeskunde, 1909: 83-92

Saltet RH. De sterftestatistiek der tuberculose in Nederland. Nederlands Tijdschrift voor Geneeskunde 1917, 53: 2038-2040

Sandiford P, Cassel J, Montenegro M, Sanchez G. The impact of women's literacy on child health and its interaction with access to health services. Population Studies 1995, 49: 5-17

Schofield R. Population growth in the century after 1750: the role of mortality decline. In: Bengtsson T et al. (eds.). *Pre-industrial population change. The mortality decline and short-term population movements.* Almquist and Wiksell International, Stockholm 1981

Schooneveldt M, Songer T, Zimmet P, Thoma K. Changing mortality patterns in Nauruans: an example of epidemiological transition. Journal Epidemiology Community Health 1988, 42: 89-95

Sickenga FN. *Korte geschiedenis van de tuberculosebestrijding in Nederland 1900-1960.* Koninklijke Nederlandse Centrale Vereniging tot Bestrijding der Tuberculose, The Hague 1980

Smith LR. Reflections on the next stage of the epidemiological transition. In: Mathers C, McCallum J, Robine J-M (eds.). *Advances in health expectancies: Proceedings of the 7th Meeting of the International Network on Health Expectancy (REVES).* Canberra, February 1994

Söderlund N, Zwi AB. Traffic-related mortality in industrialized and less developed countries. Bull WHO 1995, **73**: 175-182

Steckel RH. Stature and the standard of living. Journal of Economic Literature 1995, 33: 1903-1940

Suits DB, Mason A. Chan L. Spline functions fitted by standard regression methods. Review of Economics and Statistics 1978, 60: 132-139

Swartsenburg LA. Sterfte in de stad, 1869-1910. Unpublished paper, 1981

Szreter S. The idea of demographic transition and the study of fertility change: a critical intellectual history. Population and Development Review1993, 19: 659-701

Szreter S. The importance of social intervention in Britain's mortality decline c.1850-1914: a re-interpretation of the role of public health. Social History of Medicine 1988, 1: 5-37

Szreter S. Economic growth, disruption, deprivation, disease and death: On the importance of the politics of public health for development. Population and Development Review 1997, 23: 693-728

Tabeau E, Van Poppel FWA, Willekens F. *Mortality in The Netherlands: the data base.* NIDI report no. 36. Netherlands Interdisciplinary Demographic Institute, The Hague 1994

Tabeau E, Huisman C. Trendextrapolatie van de sterfte naar doodsoorzaken 1994-2015. In: *Volksgezondheid Toekomst Verkenning, deel IV Gezondheid en zorg in de toekomst.* RIVM, Bilthoven 1997

Tassenaar V. Regional differences in standard of living in the Netherlands, 1800-1875. A study based on anthropometric data. Research Memorandum GD-22, RU Groningen 1995

Temple NJ, Burkitt DP. *Western diseases. Their dietary prevention and reversibility.* Humana Press, New Jersey 1994

Thom TJ, Epstein FH, Feldman JJ, Leaverton PE. Trends in total mortality and mortality from heart disease in 26 countries from 1950-1978. Int J Epid 1985, 14: 510-520

Trowell HC, Burkitt DP (eds.). *Western diseases - Their emergence and prevention.* Edward Arnold, London 1981

Trowell HC, Burkitt DP. Blood-pressure rise with age - A Western disease? Lancet 1981, 2 (8248): 693-4

Tuyns AJ. Salt and gastrointestinal cancer. Nutr Cancer 1988, 11: 229-232

Uemura K, Pisa Z. Trends in cardiovascular disease mortality in industrialized countries since 1950. World Health Stat Q 1988, 41: 155-178

Unpublished letter. Manager Central Bureau of Statistics to Central Statistical Committee. Den Haag 1904

Vallin J, Souza SD, Palloni A eds. *Measurement and analysis of mortality. New approaches.* Oxford 1990

Vallin J. Mortality in Europe from 1720 to 1914: Long-term trends and changes in patterns by age and sex. In: Schofield R et al. (eds.). *The decline of mortality in Europe.* Clarendon Press, Oxford 1991

Vallin J, Meslé F. *Les causes de décès en France de 1925 à 1978. Travaux et Documents. Cahier 115.* INED Presses Universitaire de France, Paris 1988a

Vallin J, Meslé F. *Les causes de décès en France de 1925 à 1978. Travaux et Documents. Annexes 1-4 du Cahier 115.* INED Presses Universitaire de France, Paris 1988b

Vallin J, Meslé F. Reconstructing long term series of causes of death: the case of France, Unpublished Paper presented at the conference on 'The History of Registration of Causes of Death', Bloomington, Indiana USA, 1993

Vallin J, Meslé F. *Comment suivre l'évolution de la mortalité par cause malgré les discontinuités de la statistique? Le cas de la France de 1925 à 1993.* Dossiers et Recherches 53, INED 1996

Van Beeck EF, Mackenbach JP, Van Oortmarssen GJ, Barendregt JJM, Habbema JDF, Van der Maas PJ. Scenarios for the future development of accident mortality in The Netherlands. Health Policy 1989, 11: 1-17

Van Beeck E. Injuries. *A continuous challenge for public health.* Academic Thesis, Rotterdam 1998

Van den Berg Jeths A, Gijsen R, Maas IAM. Toekomstige trends in incidentie en prevalentie van aandoeningen. In: *Volksgezondheid Toekomst Verkenning, deel IV Gezondheid en zorg in de toekomst.* RIVM, Bilthoven 1997

Van Dijk JP. Doodsoorzakenclassificaties van 1750 tot 1900. Tijdschrift voor Geschiedenis, Natuurwetenschappen, Wiskunde en Techniek 1982, 5: 145-157

Van Dongen JA. *De vooruitgang der geneeskunde in onze eeuw.* De Bussy, Amsterdam 1966

Van de Kaa DJ. Europe's second demographic transition. Population Bulletin 1987, 42 no 1.

Van Lieburg, MJ, Marland H. Midwife regulation, education, and practice in The Netherlands during the nineteenth century. Medical History 1989, 33: 296-317

Van der Maas PJ, Mackenbach JP (eds.). *Volksgezondheid en gezondheidszorg.* Bunge, Utrecht 1995

Van Poppel FWA. The development of cause-of-death registration in The Netherlands: 1865-1955. Continuity and Change 1997, 12: 265-287

Van Poppel FWA. Reversal of declining mortality - the case of The Netherlands in the period 1950-80, Maandbericht Gezondheid (Statistics Netherlands) 1985, november: 5-19

Van Poppel F, Van der Heijden C. The effects of water supply on infant and childhood mortality: a review of historical evidence. Health Transition Review 1997, 7: 113-148

Van Poppel F. Regionale sterfteverschillen in Nederland 1850-1930. Continuiteit en verandering. Amsterdams Sociologisch Tijdschrift 1991, 18: 34-72

Van Poppel F. Religion and health: Catholicism and regional mortality differences in nineteenth century Netherlands. Social History of Medicine 1992, 5: 229-253

Van Poppel F, Tabeau E, Willekens F. Trends and sex-differentials in Dutch mortality since 1850: insights from a cohort- and period perspective. Genus 1986, 52: 107-134

Van der Spek J. Lonen en prijzen vanaf 1870. Economisch-Statistische Berichten 1976, november: 1144-1147

Van der Velden H. Overvloed en schaarste. De verspreiding van geneeskundige hulp in Nederland in de negentiende eeuw. Gewina 1996; 19: 210-230

Van der Velden H. The Dutch health services before compulsory health insurance, 1900-1941. Social History of Medicine 1996; 9: 49-68

Van de Walle F. Infant mortality and the European demographic transition. In: Coale AJ, Cotts Watkins S. *The decline of fertility in Europe.* Princeton University Press, Princeton New Jersey 1986

Van de Walle E. How do we define the health transition? In: Caldwell JC et al. (eds.). *What we know about health transition: the cultural, social and behavioural determinants of health. The proceedings of an international workshop.* Canberra, May 1989

Van Wieringen JC. Secular growth changes. In: Falkner F, Tanner JM (eds.) *Human growth.* Plenum, New York 1986

Van Zanden JL. *De economische ontwikkeling van de Nederlandse landbouw in de negentiende eeuw, 1800-1914.* Thesis 1985

Van Zanden JL. *Economische groei in Nederland in de negentiende eeuw. Enkele nieuwe resultaten.* Economisch en Sociaal Historisch Jaarboek 1987, 50: 51-76

Van Zon H. *Een zeer onfrisse geschiedenis. Studies over niet-industriële vervuiling in Nederland, 1850-1920.* Thesis, 1986

Vandenbroecke C, Van Poppel F, Van der Woude AM. De zuigelingen- en kindersterfte in Belgie en Nederland in seculair perspectief. Bevolking en Gezin 1983, supplement: 85-115

Verslag aan den koning van de bevindingen en handelingen van het geneeskundig staatstoezicht in het jaar..... Van Weelden en Mingelen, 's-Gravenhage 1875-1899

Verslagen en mededeelingen betreffende de volksgezondheid. Staatsuitgeverij, 's-Gravenhage 1923

Vigneron E. The epidemiological transition in an overseas territory: disease mapping in French Polynesia. Social Science and Medicine 1989, 29: 913-922

Vishnevsky AG, Shkolnikov V, Vassin SA. Epidemiological transition in the USSR as mirrored by regional differences. Genus 1991, 47: 79-101

Vögele J. Urban infant mortality in imperial Germany. Social History of Medicine 1994, 7: 401-425

Vogelzang I. *De drinkwatervoorziening in Nederland voor de aanleg van de drinkwaterleidingen.* Thesis. Gouda: Joh Mulder publishers, 1956

Vollenhoven H van. Twee vijfjarige overzichten. Vergelijkende beschouwingen van sterfte in Nederland 1875-1885. Nederlands Tijdschrift voor Geneeskunde 1889, 25: 597-616

Walker WJ. Changing United States life-style and declining vascular mortality: cause or coincidence?. New England Journal of Medicine 1977, 297: 163-165.

Watterson PA. Infant mortality by father's occupation from the 1911 census of England and Wales. Demography 1988, 25: 289-306

Wesley PGE. *Elementaire wetenschapsleer.* Boom, Meppel 1982

Whisnant JP. The role of the neurologist in the decline of stroke. Ann Neurol 1983, 14: 1-7

Wichers AJ. *De oude plattelandsbeschaving. Een sociologische bewustwording van de 'overheerigheid'.* Centrum voor Landbouwpublikaties en Landbouwdocumentatie, Wageningen 1965

Wilkinson RG. Unhealthy societies. The afflictions of inequality. Routledge, London 1996

Williams N, Galley C. Urban-rural differentials in infant mortality in Victorian England. Population Studies 1995, 49: 401-420

Williams N. Death in its season: class, environment and the mortality of infants in nineteenth-century Sheffield. Social History of Medicine 1992, 5: 71-94

Williams N. The reporting and classification of causes of death in the mid-nineteenth century: The example of Sheffield. Historical Methods 1996, 29: 58-71

Wolleswinkel-van den Bosch JH, Poppel FWA van, Mackenbach JP. Reclassifying causes of death to study the epidemiologic transition in The Netherlands, 1875-1992. European Journal of Population 1996, 12: 327-361

Wolleswinkel-van den Bosch JH, Poppel FWA van, Looman CWN, Mackenbach JP. Cause-specific mortality in The Netherlands, 1875-1992: a formal analysis of the epidemiologic transition. International Journal of Epidemiology 1997, 26: 772-781

Wolleswinkel-van den Bosch JH, Poppel FWA van, Tabeau E, Mackenbach JP. Mortality decline in The Netherlands in the period 1850-1992: a turning point analysis. Social Science and Medicine 1998, 47: 429-443

Woods RI, Watterson PA, Woodward JH. The causes of rapid infant mortality in England and Wales, 1861-1921 Part I. Population Studies 1988, 42: 343-366

Woods RI, Watterson PA, Woodward JH. The causes of rapid infant mortality in England and Wales, 1861-1921 Part II. Population Studies 1989, 43: 113-132

Woods RI, Woodward JH (eds.). *Urban disease and mortality in nineteenth century England.* St Martin's Press, New York 1984

Woods R, Williams N, Galley C. Differential mortality patterns among infants and other young children: The experience of England and Wales in the nineteenth century. In: Corsini CA, Viazzo PP (eds.). *The decline of infant and child mortality. The european experience: 1750-1990.* Martinus Nijhoff Publishers, Dordrecht 1997

World Health Organization. Manual of the international statistical classification of diseases, injuries and causes of death - Seventh revision. World Health Organization, Geneva 1957

World Health Organization. Manual of the international statistical classification of diseases, injuries and causes of death - Eighth revision. World Health Organization, Geneva 1967

World Health Organization. Manual of the international statistical classification of diseases, injuries and causes of death - Ninth revision. World Health Organization, Geneva 1977/78

Woude A van der, Hayami A, Vries J de (eds.). *Urbanization in history. A process of dynamic interactions.* (p.44) Clarendon Press, Oxford 1990

Wrigley EA, Schofield RS. *The population history of England, 1541-1871: a reconstruction.* Edward Arnold, London 1981

Young TK. Are Subarctic Indians undergoing the epidemiologic transition? Social Science and Medicine 1988, 26: 659-671

SUMMARY

IN 1971, OMRAN published the 'epidemiological transition theory'. This theory gives a stylised description of mortality decline in developed countries. According to Omran, mortality decline followed three phases: first, *the age of pestilence and famine* in which mortality was high and fluctuating, and average life expectancy at birth was low and variable, vacillating between 20 and 40 years; secondly, the *age of receding pandemics* in which mortality declined progressively, the rate of decline accelerated as epidemic peaks became less frequent and eventually disappeared, average life expectancy at birth increased steadily from about 30 to 50 years; thirdly, *the age of degenerative and man-made diseases* in which mortality continued to decline and eventually approached stability at a relatively low level

Omran's theory was based on the mortality experience in western countries in the late 18th to 20th centuries. He generalised the epidemiological transition to other parts of the world. Other countries could differ with respect to onset and pace of the decline, but mortality decline went through the same phases of cause-specific mortality. The concept of epidemiological transition theory has widely been used in public health. It offers possibilities for the explanation of differences in cause-of-death pattern from differences in timing of the transition, and for projections of cause-specific mortality change in developing countries based on the historical experience of western countries. The original epidemiological transition theory has, however, met criticism too and has been refined by others. The original theory has been accused by some researchers of being

merely an empirical generalisation of mortality experiences in western countries; others have added a fourth phase to the transition theory in order to include recent mortality changes. There is also still a debate going on about the determinants of mortality decline. Considering all this, it seemed to be the right time for a renewed scrutiny of the epidemiological transition theory. This thesis might contribute to this scrutiny by presenting a detailed analysis of the epidemiological transition in The Netherlands.

The objective of this thesis is, first, to present a detailed description of the epidemiological transition in The Netherlands with respect to the onset of, and accelerations or decelerations in all-cause mortality decline, with respect to cause-specific mortality trends and the contribution of infectious diseases to mortality decline. Secondly, this thesis will study the relative importance of determinants of mortality decline in The Netherlands in the late 19th and early 20th centuries. And thirdly, the epidemiological transition theory will be re-examined on the basis of the results for The Netherlands.

After the introduction and objectives described in **chapter 1,** Omran's original publications on the epidemiological transition are presented in **chapter 2.** Other literature in which weaknesses in Omran's theory were pointed out or amendments to the theory were suggested are reviewed as well. The main weakness of Omran's original theory was the inaccurate description of the phases of the transition with respect to timing and cause-specific mortality changes. Beside that, the epidemiological transition theory by Omran does not adequately describe determinants of the epidemiological transition.

In **chapter 3** the literature on determinants of mortality decline is reviewed, and a scheme of analytical levels of determinants of mortality decline is presented. Two analytical levels were distinguished: the level of proximate determinants, which consists of determinants that are directly associated with mortality decline (e.g. crowding, nutritional status), and the level of distal determinants, which are indirectly related to mortality decline, and affect mortality decline through the proximate determinants (e.g. wealth, religious affiliation). This scheme was used in the analyses of determinants of mortality decline to structure the many determinants involved.

In chapters 4 to 9 descriptive and explanatory studies of the epidemiological transition in The Netherlands are presented. **Chapter 4** describes a method for reclassifying causes of death in the Netherlands for the period 1875-1992. Two criteria had to be met to obtain a useful classification to study the epidemiological transition. First, the categories should be nosologically continuous over the period under study and second, there should be enough detail in causes, which are important in the context of the epidemiological transition viz. communicable diseases, non-communicable diseases and external causes of death. The newly created categories were tested for statistical continuity during

the transition years of one ICD-revision to the next. The reclassification procedure resulted in a nested classification consisting of three levels of refinement of causes of death: 27 causes, 1875-1992; 65 causes, 1901-1992; and 92 causes, 1931-1992. On the basis of this classification, 43% of all deaths in 1875/79 and 98% of all deaths in 1992 could be allocated to communicable diseases, non-communicable diseases or external causes. The level of refinement of 27 and 65 causes were used in the descriptive analyses of the epidemiological transition in the Netherlands.

In **chapter 5** the epidemiological transition is described on the basis of all-cause mortality. A detailed and fairly objective description of rapid mortality decline in The Netherlands between 1850 and 1992 with respect to the start, end, and phases of the decline is given. Turning points were estimated for the total mortality trend, and for age and sex-specific trends between 1850-1992. The turning points divided the trends into phases with different paces of decline. Standardised mortality started to decline rapidly in The Netherlands around 1880. Four phases in the period of decline could be distinguished: 1880-1917 (1.2% annually), 1917-1955 (1.6%), 1955-1970 (0.4%), 1970-1992 (1.1%). For nearly all age groups, the most rapid decline occurred in a period comparable to 1917-1955. Causes of death which might have shaped the standardised mortality trend were, among others, respiratory tuberculosis (1917), heart disease (except ischemic) (1955), ischemic heart disease (1970). Causes of death that shaped the mortality trend are related to trends of determinants of mortality decline.

A description of the epidemiological transition based on cause-specific mortality trends was given in **chapter 6.** Analyses were conducted for the period 1875 to 1992. Cluster analysis was used to determine groups of causes of death with similar trend curves over a period of time with respect to age- and sex-standardised mortality rates. In case of the analysis for 27 causes (studied for the period 1875-1992), 3 important clusters were found: (1) infectious diseases which declined rapidly in the late 19th century (e.g. typhoid fever), (2) infectious diseases which showed a less precipitous decline (e.g. respiratory tuberculosis), and (3) non-infectious diseases which showed an increasing trend during most of the period 1875-1992 (e.g. cancer). The 65 causes (1901-1992) provided more detail. 7 important clusters were found: 4 clusters consisted mainly of infectious diseases, including a new cluster that declined rapidly after the Second World War (WW2) (e.g. acute bronchitis/influenza) and a new cluster showing an increasing trend in the 1920s and 1930s before declining in the years thereafter (e.g. otitis media). 3 clusters mainly contained non-infectious diseases, including a new cluster that declined from 1901 onwards (e.g. cancer of the stomach) and a new cluster that increased until WW2 but declined afterwards (e.g. chronic rheumatic heart disease).

The decline in infectious diseases is an important characteristic of the epidemiological transition. In **chapter 7** the relative contribution of infectious diseases to total mortality decline in The Netherlands in the period 1875-1970 and four subperiods viz. 1875-1901, 1901-31, 1931-50, 1950-70 was analysed, as well as the pace of infectious disease mortality decline in the four subperiods. Respiratory tuberculosis contributed most to the decline in the periods 1875-1970 (14.7%), 1875-1901 (16.2%), 1950-70 (14.9%), diarrhoea/dysentery in 1901-31 (20.3%), and pneumonia in the period 1931-50 (22.0%). A generalised acceleration in mortality decline (waterborne as well as airborne infectious diseases) could be observed in the period 1901-31. In the period 1931-50 accelerations in decline were predominantly observed among airborne infectious diseases. The accelerations in mortality decline (from infectious diseases which contributed largely to mortality decline) in the period 1901-31 could be related to accelerations in fertility decline in the same period. In the period 1931-50, improvements in socio-economic and socio-cultural factors decelerated. Medical factors seemed to be important contributors to mortality decline in that period.

In chapters 5 to 7, trends in determinants are related to cause-specific mortality trends to get insight in possible determinants of mortality decline. To further elucidate the determinants of mortality decline multivariate explanatory analyses are needed instead of univariate descriptive analyses. In chapters 8 and 9 mortality levels and declines in the late 19th and early 20th centuries in The Netherlands are analysed using multivariate regression analyses. The regional variance in mortality and determinants, which was clearly apparent at that time in The Netherlands, was used in these analyses. In **chapter 8** the relative importance of economic and cultural determinants of mortality decline have been studied for the periods 1875/79-1895/99 and 1895/99-1920/24. Regional mortality declines were estimated using Poisson-regression models. In a multivariate analysis the estimated declines were associated with economic (wealth tax) and cultural variables (Roman Catholicism and secularisation) corrected for confounders (urbanisation and soil type). In the period 1875/79 to 1895/99, Roman Catholicism was significantly associated with all-cause mortality decline and with mortality decline from other diseases than infectious diseases. Secularisation was significantly associated with infectious-disease mortality decline. In the period 1895/99 to 1920/24, wealth tax was significantly associated with all-cause and infectious disease mortality decline. Intermediary, more proximate, factors in the relationship between cultural factors and mortality decline were fertility decline, and the number of medical doctors per 100,000 population. No intermediary factors were found for the association between the economic variable and mortality decline. Cultural and economic factors both played an important role in mortality decline in The Netherlands, but in different periods of time.

In **chapter 9** determinants of (cause-specific) infant and early childhood mortality (decline) in the late 19th century are studied. Mortality levels and decline were estimated with a Poisson-regression model. The associations of the estimated levels and declines, and determinants of infant and early childhood mortality were analysed using multivariate linear regression analysis. The causes of death studied were, in case of infant mortality, convulsions, acute digestive diseases, acute respiratory diseases, and, in case of early childhood mortality, encephalitis/meningitis, acute respiratory diseases, and measles. Roman Catholicism and marital fertility were significantly associated with high infant mortality (total, acute digestive diseases and convulsions). The association for Roman Catholicism was stronger in 1895/99 as compared to 1875/79 due to less rapid mortality declines in Roman Catholic areas. Urbanisation was significantly associated with high early childhood mortality (total, acute respiratory diseases, encephalitis/meningitis). The strength of this association decreased over time, because of faster mortality declines in towns. The findings for The Netherlands seemed to be more comparable with findings for Germany than with findings for England and Wales.

In **chapter 10** of this thesis, the epidemiological transition theory is reexamined based on the studies presented in chapters 4 to 9 and the international literature. First, the reliability of the Dutch cause-of-death statistics, on which the results in chapter four to nine are based, and the comparability of the epidemiological transition in The Netherlands with other European countries is discussed. The cause-of-death classification in The Netherlands in the late 19th century was rather brief, and there were inaccuracies in coding and diagnosis of specific causes of death, but the effects of these inaccuracies on the conclusions presented in this thesis were limited. Although the 19th-century classification was brief, diagnosis by a medical doctor has been required in The Netherlands since 1865, which is earlier than in several other European countries. Besides, the detailed first International Classification of Causes of Death was introduced right away in The Netherlands in 1901.

The Netherlands can, together with other northwestern countries e.g. England and Wales, be considered a country in which accelerated mortality decline started early, i.e. around 1870/80. The Netherlands distinguished itself from other countries with respect to the rapid increase in life expectancy in the late 19th and early 20th centuries. With respect to cause-specific mortality there was probably also a difference between northwestern and southern European countries. Acute digestive diseases were more important contributors to mortality, and declined less rapid in southern countries (e.g. Italy) than in north-western countries (e.g. The Netherlands) More research on cause-specific mortality decline would be needed to draw more soundly based conclusion with respect to cause-specific mortality.

The analyses of the weaknesses in Omran's original theory with respect to the changes in cause-specific mortality and the timing of the epidemiological transition, showed that the changes in cause-specific mortality rates should be described more specifically than a 'decline in infectious diseases and an increase in degenerative and man-made diseases'. The transition seemed to be more an ongoing rise and decline of causes of death than a transition of one stage of causes of death to another. On the basis of all-cause and cause-specific mortality analyses for The Netherlands, more phases in mortality decline could be defined than the stages defined by Omran.

Omran's original epidemiological transition theory did not accurately describe detailed cause-specific mortality changes, nor did it accurately describe the determinants of the transition. In **chapter 10** elements for a renewed formulation of the epidemiological transition theory were suggested. The epidemiological transition theory should be described in terms of rise and decline of causes of death, and in terms of a disruption of health by emerging health risks and adaptation to health risks. This gives the epidemiological transition theory a new focus, viz. the underlying mechanisms, and the cause-specific mortality changes as a result of that, instead of a description of epidemiological transition as a decline of infectious diseases and a rise of non-infectious diseases with little reference to underlying determinants.

The publication of the epidemiological transition theory has triggered an enormous amount of research on epidemiological changes, including on the determinants of those changes. Considering the historical rise and decline of causes of death, newly emerging health risks can be expected in the future (for example the re-emergence of 'old' diseases such as tuberculosis). Epidemiologists can play an important role in contributing to the adaptation to new health risks by unravelling the determinants of diseases and causes of death, as well as research on adaptation strategies, such as early diagnosis (e.g. cancer screening) and health promotion.

SAMENVATTING

OMRAN PUBLICEERDE IN 1971 zijn 'epidemiologische transitietheorie'. Deze theorie geeft een beschrijving van de sterftedaling in ontwikkelde landen. Deze sterftedaling verloopt volgens Omran in drie fasen. Ten eerste was er 'het tijdperk van pest en hongersnood' *(the age of pestilence and famine)*, waarin de sterfte hoog was en sterk fluctueerde en de levensverwachting bij geboorte laag en variabel was, tussen de 20 en 40 jaar. Ten tweede was er 'het tijdperk van terugtredende pandemieën' *(the age of receding pandemics)*, waarin de sterfte sterk daalde. Deze daling werd sterker als de epidemieën minder vaak voorkwamen. De levensverwachting bij de geboorte nam gestaag toe van 30 tot 50 jaar. Ten derde was er 'het tijdperk van de degeneratieve en door de mens veroorzaakte ziekten' *(the age of degenerative and man-made diseases)*. In dit tijdperk daalde de sterfte verder en bereikt uiteindelijk stabiliteit op een relatief laag niveau.

Omrans theorie was gebaseerd op sterfteveranderingen in westerse landen vanaf het eind van de 18e tot aan de 20e eeuw. Hij generaliseerde de epidemiologische transitietheorie ook naar andere delen van de wereld. Landen konden verschillen wat betreft het tijdstip waarop de sterke sterftedaling begon en de snelheid van de sterftedaling, maar de sterftedaling volgde wel dezelfde fasen van doodsoorzaakspecifieke sterfte. De epidemiologische transitietheorie is één van de weinige theorieën binnen de volksgezondheid. Zij biedt mogelijkheden om verschillen in doodsoorzaakpatronen tussen landen te verklaren vanuit verschillen in timing van de epidemiologische transitie. Daarnaast biedt zij mogelijk-

heden voor het voorspellen van toekomstige doodsoorzaakpatronen in landen waarin de epidemiologische transitie minder ver gevorderd is. Er is echter ook kritiek dat de epidemiologische transitietheorie geen theorie is, maar slechts een generalisatie van sterfteveranderingen in verschillende landen. Andere onderzoekers hebben voorgesteld fasen toe te voegen aan de epidemiologische transitie om meer recente sterfteveranderingen in de theorie in te passen. Daarnaast is het nog steeds niet geheel duidelijk wat de oorzaken zijn geweest van die sterke sterftedaling in westerse landen in de afgelopen eeuw(en). Gegeven de populariteit van de epidemiologische transitietheorie, de kritiek en de voortdurende discussie over de determinanten van sterftedaling, lijkt de tijd rijp voor een hernieuwde evaluatie van de epidemiologische transitietheorie van Omran. Dit proefschrift wil aan deze evaluatie bijdragen door gedetailleerde analyses van de epidemiologische transitie in Nederland te presenteren.

Het doel van dit proefschrift is, ten eerste, om een gedetailleerde beschrijving van de epidemiologische transitie in Nederland te geven wat betreft het begin van de sterftedaling, versnellingen en vertragingen in de sterftedaling en wat betreft doodsoorzaakspecifieke sterfte-ontwikkelingen. Een tweede doel is het relatieve belang te bepalen van determinanten van sterftedaling in Nederland in de laatste decennia van de 19e en de beginjaren van de 20e eeuw. Een laatste doel, ten slotte, is om de epidemiologische transitie theorie te evalueren, gebruikmakend van de resultaten voor Nederland.

Na de introductie en doelstellingen beschreven in **hoofdstuk 1**, wordt in **hoofdstuk 2** een overzicht gegeven van de originele publicaties over de epidemiologische transitietheorie door Omran en andere literatuur waarin kritiek en aanvullingen op de theorie zijn gegeven. De beschrijving van de fasen in de transitie wat de timing en de doodsoorzaakspecifieke veranderingen betreft bleken onnauwkeurig in Omran's epidemiologische transitietheorie. Daarnaast besteedde Omran weinig aandacht aan de determinanten van de epidemiologische transitie.

In **hoofdstuk 3** wordt literatuur over determinanten van sterftedaling besproken en worden deze determinanten gepresenteerd in een schema met twee analyseniveaus: proximale en distale determinanten. Proximale determinanten zijn direct aan sterfte gerelateerd (bv. voeding); distale determinanten zijn indirect aan sterftedaling gerelateerd (bv. godsdienst) en hebben effect op de sterftedaling via de proximale determinanten.

In de hoofdstukken 4 tot en met 9 worden de beschrijvende en verklarende studies van de epidemiologische transitie in Nederland gepresenteerd. In **hoofdstuk 4** wordt een methode beschreven om de verschillende classificaties van sterfte naar doodsoorzaak die in de periode van studie (1875-1992) zijn gehanteerd te reclassificeren. Dit is nodig om doodsoorzaakcategorieën te creëren die zoveel mogelijk nosologisch constant zijn door de tijd. Om de

epidemiologische transitie te bestuderen is gestreefd naar een goede vertegenwoordiging van infectieziekte-categorieën, niet-infectieziekte-categorieën en categorieën met externe doodsoorzaken. De uiteindelijke categorieën werden getest op statistische continuïteit over de verschillende overgangen van de ene classificatie naar de andere. Het uiteindelijke resultaat van de reclassificatie-methode zijn drie niveaus van detail in doodsoorzaken: 27 doodsoorzaken voor de periode 1875-1992, 65 voor de periode 1901-1992, en 92 voor de periode 1931-1992. Op grond van deze indeling kon 43% van de sterfte in 1875/79 en 98% van de sterfte in 1992 toegekend worden aan een duidelijk omschreven categorie van infectieziekten, niet-infectieziekten of externe doodsoorzaken. De niveaus van 27 en 65 doodsoorzaken worden in de analyses in de hoofdstukken 5 tot en met 7 gebruikt.

In **hoofdstuk 5** wordt een gedetailleerde en objectieve omschrijving gegeven van de epidemiologische transitie in termen van totale sterfte. Het begin van en fasen in de transitie worden beschreven. Omslagpunten die perioden markeren met een versnelling of een vertraging in de sterftedaling werden bepaald in de totale sterftetrend en in leeftijds- en geslachtsspecifieke sterfte-trends voor de periode 1850-1992. De totale sterftetrend begon in Nederland sterk te dalen in 1880. Vervolgens konden vier fasen in die sterftedaling onderscheiden worden op basis van de snelheid van de daling: 1880-1917 (1.2% daling per jaar), 1917-1955 (1.6%), 1955-1970 (0.4%), 1970-1992 (1.1%). Voor mannen en vrouwen en in bijna alle leeftijdsgroepen daalde de sterfte het sterkst in de periode 1917-1955. Doodsoorzaken die de totale sterftetrend gevormd zouden kunnen hebben zijn, onder andere, respiratoire tuberculose (omslagpunt 1917), hartziekten (behalve ischemische hartziekten) (omslagpunt 1955) en ischemische hartziekten (omslagpunt 1970). Deze doodsoorzaken werden vervolgens aan determinanten gerelateerd.

In **hoofdstuk 6** wordt een beschrijving van de epidemiologische transitie in Nederland gegeven op grond van trends in doodsoorzaakspecifieke sterfte. De analyses zijn uitgevoerd voor de periode 1875-1992. Met behulp van cluster-analyse werden groepen doodsoorzaken bepaald met een zelfde trend door de tijd. In de analyse van 27 doodsoorzaken, voor de periode 1875-1992, konden drie belangrijke clusters onderscheiden worden: (1) infectieziekten die snel daalden aan het eind van de 19e eeuw (bv. typhus), (2) infectieziekten die een minder snelle daling lieten zien (bv. respiratoire tuberculose) en (3) niet-infectieziekten die een stijgende trend gedurende het grootste gedeelte van de periode 1875-1992 lieten zien (bv. kanker). De analyse met 65 doodsoorzaken, voor de periode 1901-1992 gaf een meer gedetailleerd inzicht in de doods-oorzaakspecifieke trends. Zeven belangrijke clusters werden onderscheiden. Vier clusters bestonden voornamelijke uit infectieziekten, inclusief een cluster waarbij een sterke daling na de Tweede Wereldoorlog optrad (bv. acute bronchitis/

influenza) en een nieuw cluster infectieziekten dat een stijgende trend liet zien in de jaren '20 en '30, gevolgd door een daling (bv. otitis media). Drie clusters bestonden voornamelijk uit niet-infectieziekten, inclusief een nieuw cluster dat een dalende trend liet zien sinds 1901 (bv. maagkanker) en een cluster dat een lichte stijgende trend had tot aan de Tweede Wereldoorlog en daarna daalde (bv. chronisch rheumatische hartziekten).

De daling in infectieziekten is een belangrijk element van de epidemiologische transitie. In **hoofdstuk 7** is het relatieve aandeel van infectieziekten aan de sterftedaling in de periode 1875-1970 en vier subperioden (1875-1901, 1901-1931, 1931-1950, 1950-1970) beschreven. Daarnaast is ook de snelheid van de daling van de infectieziekten voor die vier subperioden geanalyseerd. Respiratoire tuberculose droeg het meest bij aan de daling in de perioden 1875-1970 (14.7%), 1875-1901 (16.2%) en 1950-1970 (14.9%). Diarree/dysenterie droeg het meeste bij aan de daling in de periode 1901-1931 (20.3%) en pneumonie in de periode 1931-1950 (22.0%). Een algemene versnelling in de sterftedaling van infectieziekten (door water en voedsel overgedragen en door lucht overgedragen) deed zich voor in de periode 1901-1931. In de periode 1931-1950 deden zich met name versnellingen in de sterftedaling van via lucht overgedragen infectieziekten voor. Versnellingen in de sterftedaling van infectieziekten die veel bijdroegen aan de sterftedaling in 1901-1931 kon gerelateerd worden aan versnellingen in vruchtbaarheidsdaling in dezelfde periode. De versnellingen in de periode 1931-1950 konden niet gerelateerd worden aan verbeteringen van sociaal-economische of culturele factoren. In die periode leken medische factoren de belangrijkste determinanten voor de sterftedaling.

In de hoofdstukken 5 tot en met 7 werden doodsoorzaakspecifieke trends gerelateerd aan trends in determinanten om inzicht te krijgen in mogelijke determinanten van sterftedaling. Voor een verdergaand inzicht in de determinanten van sterftedaling zijn multivariate analyses nodig. In de hoofdstukken 8 en 9 zijn determinanten van de sterfte(daling) aan het eind van de 19e en begin 20e eeuw in Nederland geanalyseerd met behulp van multivariate regressie-analyse. De variatie in de prevalentie van verschillende determinanten in 27 regio's in Nederland wordt in die analyses gerelateerd aan de sterfte(daling). In **hoofdstuk 8** wordt het relatieve belang van economische en culturele variabelen van sterftedaling in de periode 1875/79-1895/99 en 1895/99-1920/24 onderzocht. Regionale sterftedaling werd geschat met behulp van Poisson-regressie analyse. In een multivariate analyse werden de geschatte sterftedalingen gerelateerd aan economische ('personele belasting') en culturele variabelen (rooms-katholicisme en secularisatie) gecorrigeerd voor confounders (grondsoort en urbanisatie). In de periode 1875/79-1895/99 was rooms-katholicisme significant geassocieerd met totale sterftedaling en sterftedaling van niet-infectieziekten. De sterfte daalde minder snel in rooms-katholieke regio's. Secularisatie was significant geassocieerd

met de daling van de sterfte aan infectieziekten. De infectieziektensterfte daalde sneller in regio's met een hoog percentage van de bevolking zonder godsdienstige affiliatie. In de periode 1895/99-1920/24, was personele belasting significant geassocieerd met sterftedaling aan infectieziekten en totale sterfte. De sterfte daalde sneller in meer welvarende gebieden. Meer proximale factoren die de associatie tussen de culturele factoren en sterftedaling zouden kunnen verklaren waren huwelijksvruchtbaarheid en de artsendichtheid. Er werden geen intermediaire factoren gevonden voor de associatie tussen welvaart ('personele belasting') en sterftedaling. Culturele en economische factoren speelden beide een rol in de sterftedaling in Nederland maar waren in verschillende perioden meer of minder belangrijk.

In **hoofdstuk 9** worden determinanten van (doodsoorzaakspecifieke) zuigelingensterfte en sterfte van 1- tot 4-jarigen in het einde van de 19e eeuw bestudeerd. Sterfteniveaus en sterftedaling werden geschat met een Poisson-regressie model. Deze schattingen werden in een multivariate lineaire regressie-analyse aan determinanten van sterfte gerelateerd. De doodsoorzaken die voor zuigelingen geanalyseerd werden zijn acute spijsverteringsziekten, acute respiratoire aandoeningen en stuipen; voor de sterfte van 1- tot 4-jarigen werden hersenziekten (vnl. encephalitis en meningitis), acute respiratoire aandoeningen en mazelen geanalyseerd. Rooms-katholicisme en huwelijksvruchtbaarheid waren significant geassocieerd met hoge zuigelingensterfte (totale sterfte, acute spijsverteringsziekten en stuipen). De associatie met rooms-katholicisme was sterker in 1895/99 dan in 1875/79 vanwege minder sterke sterftedaling in rooms-katholieke gebieden in de periode 1875/79-1895/99. Urbanisatie was significant geassocieerd met hoge sterfte op de leeftijd 1-4 jaar (totale sterfte, acute respiratoire aandoeningen en hersenziekten). De mate van deze associatie nam af door de tijd, omdat de sterfte in steden juist sneller daalde dan op het platteland. Deze bevindingen ten aanzien van determinanten van zuigelingen- en jonge-kindersterfte komen overeen met resultaten voor Duitsland, meer dan met resultaten voor Engeland en Wales.

In het laatste hoofdstuk van het proefschrift, **hoofdstuk 10**, worden de resultaten van de studies voor Nederland in een bredere context bediscussieerd en wordt Omran's epidemiologische transitietheorie geëvalueerd. Eerst wordt in dit hoofdstuk de betrouwbaarheid van de Nederlandse doodsoorzakenstatistiek bediscussieerd en wordt de epidemiologische transitie in Nederland vergeleken met die in andere Europese landen. De doodsoorzakenclassificatie in de 19e eeuw was relatief kort en er waren enige onnauwkeurigheden in het coderen en in de diagnose van specifieke doodsoorzaken, maar de effecten hiervan op de conclusies van dit proefschrift zijn beperkt. Hoewel de 19e-eeuwse classificatie kort was, was een belangrijk voordeel van de Nederlandse classificatie dat diagnose van de doodsoorzaak door een arts al sinds 1865 vereist was. Dit is eerder

dan in verschillende andere Europese landen. Daarnaast is het zo dat in Nederland direct in 1901 de eerste, en uitgebreide, Internationale Classificatie van Doodsoorzaken werd ingevoerd.

Nederland kan samen met andere Noordwest-Europese landen, zoals Engeland en Wales, beschouwd worden als een land waarin de epidemiologische transitie vroeg begon, nl. rond 1870/80. Nederland onderscheidde zich van andere landen waarin deze versnelde sterftedaling vroeg begon door de relatief snelle daling in voornamelijk het einde van de 19e en het begin van de 20e eeuw. Wat betreft doodsoorzaakspecifieke sterftedaling waren er verschillen tussen Noordwest-Europese en Zuid-Europese landen. Acute spijsverteringsziekten bijvoorbeeld droegen niet alleen méér bij aan de sterfte in Zuid-Europese landen (bv. Italië) dan in Noordwest-Europese landen (bv. Nederland), maar de sterfte aan deze ziekten daalde ook minder snel in Zuid-Europese landen.

De analyses van de kritiekpunten op Omran's originele theorie wat de veranderingen in doodsoorzaakspecifieke sterfte en de timing van de epidemiologische transitie betreft, laten zien dat de weergave van de epidemiologische transitie als een daling van infectieziekten en een stijging van niet-infectieziekten te eenvoudig is. De epidemiologische transitie is meer een voortdurende stijging en daling van doodsoorzaken dan een transitie van het ene doodsoorzaakpatroon naar het andere. Daarnaast brachten de analyses van totale en doodsoorzaakspecifieke sterftetrends in Nederland voor de periode 1875-1992 meer fasen in de epidemiologische transitie aan het licht dan die door Omran genoemd worden.

In Omran's oorspronkelijke epidemiologische transitietheorie werden de doodsoorzaakspecifieke verandering niet gedetailleerd beschreven en werd slechts zijdelings ingegaan op de determinanten van sterftedaling. Een gedetailleerde doodsoorzaakanalyse is echter wel van belang om de onderliggende mechanismen van de steftedaling te kunnen doorgronden. In hoofdstuk 10 worden elementen voor een hernieuwde formulering van de epidemiologische transitie voorgesteld. Hierbij is gebruik gemaakt van bestaande theorieën, zoals de 'health transition theory'. De epidemiologische transitie zou omschreven moeten worden in termen van stijging en daling van doodsoorzaken en in termen van verstoring van de gezondheid door opkomende gezondheidsrisico's en aanpassing aan die risico's. Dit geeft de epidemiologische transitietheorie een nieuwe focus, nl. die van de onderliggende mechanismen en de doodsoorzaakspecifieke verandering als het resultaat daarvan. Dit in tegenstelling tot de huidige omschrijving in termen van een daling van infectieziekten en een stijging van niet-infectieziekten zonder verwijzing naar de determinanten. Gezien de historische trend van stijging en daling van verschillende doodsoorzaken, kan de opkomst van nieuwe ziekten en doodsoorzaken voor de toekomst verwacht worden. Een voorbeeld hiervan is bijvoorbeeld de opkomst van 'oude' infectieziekten zoals

tuberculose. Epidemiologisch onderzoek kan een belangrijke bijdrage leveren aan het aanpassen aan opkomende gezondheidsrisico's door onder andere onderzoek van determinanten, surveillance van (infectie)ziekten, vroege opsporing van ziekten en gezondheidsvoorlichting.

Appendix 1

PRE-ICD AND ICD CODES BELONGING TO THE CAUSE-OF-DEATH CATEGORIES OF THE NESTED CLASSIFICATION

1875-1992		1901-1992								
Cause of death	19th century classification	ICD-1 (1901-1910)	ICD-2 (1911-1920)	ICD-3 (1921-1930)	ICD-4 (1931-1940)	ICD-5 (1941-1949)	ICD-6/7 (1950-1968)	ICD-8 (1969-1978)	ICD-9 (1979-1992)	Cause of death
Congenital malformations	1	150	150	159	157a-c	157a-j	750-759	740-749, 750-759	740-749, 750-759	Congenital malformations
Cancer	5	40	40	44a-b	46a	46a	150	150	150	Cancer of the oesophagus
					46b	46b	151	151	151	Cancer of the stomach
					46d	46e	155-156	155-156	155-156	Cancer of the liver and gallbladder
		41	41	45a-b	46c	46d	154	154	154	Cancer of the rectum
					46f	46g	158	158	158	Cancer of the peritoneum
					46g	46c	152-153	152-153	152-153	Cancer of the colon and small intestines
		44	44	48	52	53	190-191	172-173	172-173	Cancer of the skin
		43	43	47	50	50	170	174	174-175	Cancer of the breast
		39, 42, 45	39, 42, 45	43a-b, 46a-c, 49a-c	48	48a-b	171-174	180-182	179-182	Cancer of the uterus
					49a-b	49	175-176	183-184	183-184	Cancer of the ovary and other female genital organs
					46e	46f	157	157	157	Cancer of the pancreas
					47a-b	47a-c	161-165	161-163	161-165	Cancer of lung and larynx

1875-1992		1901-1992								
Cause of death	19th century classification	ICD-1 (1901-1910)	ICD-2 (1911-1920)	ICD-3 (1921-1930)	ICD-4 (1931-1940)	ICD-5 (1941-1949)	ICD-6/7 (1950-1968)	ICD-8 (1969-1978)	ICD-9 (1979-1992)	Cause of death
					46h, 51, 53	46h, 51a-c, 52, 54a-b, 55a-d	142, 159-160, 177-181, 192-200, 202-203, 205-207	142, 159-160, 170-171, 185-200, 202-203	142, 159-160, 170-171, 185-200, 202-203	Cancer of other organs
Scurvy	6.2	49	49	53	60a-b	67a-b	282	264	267	Scurvy
Typhoid fever. Paratyphoid fever	7	1	1	1a-b	1, 2a-b	1, 2a-c	040-041	001-002	002	Typhoid fever. Paratyphoid fever
Malaria (including: intermittent fever, pernicious fever)	9	4a-b	4a-b	5a-b	38a-b	28a-d	110-117	084	084	Malaria
Smallpox	10	5	5	6	6a-c	34a-b	084	050	050	Smallpox
Scarlet fever	11	7	7	8	8	8	050-051	034	034	Scarlet fever
Measles	12	6	6	7	7	35	085	055	055	Measles
Cerebrovascular diseases	15	64-65	64-65	74a-b, 83	82a-b	83a-b	330-334	430-438	430-438	Cerebrovascular diseases
Brain diseases etc. (including insanity, diseases of the spinal cord, paralysis, syphilis, convulsions, trismus, epilepsy)	3.2, 14, 16, 17	28	30	32	24	14	010	013	013	Tuberculosis of the nervous system
		36, 62, 67	37, 62, 67	38a-e, 72, 76	34a-c, 80, 83	30a-d4	020-029	090-097	090-097	Syphilis
		51, 63, 66, 69-70, 74-75, 89	51, 63, 66, 69-70, 73-75, 88	60a-b, 61, 73, 75a-b, 78-79, 82a-b, 84, 85a-e	66a	63a	250	240	240	Goitre
					66c	63c	253	243-244	243-244	Cretinism
					66b	63b	252	242	242	Basedow's disease
					66d-e	63d-e	251, 254, 271	241, 245-246, 252	241, 245-246, 252	Other diseases of the thyroid

					81, 82c, 87b,e	82, 83c, 87b,e	351-352, 354-357, 360-369	330-333, 341, 343-344, 346-358	330-331, 333-337, 341-344, 346-359	Other diseases of the nervous system
					87c	87c	350	342	332	M.Parkinson
					87d	87d	345	340	340	Multiple Sclerosis
					85	85	353	345	345	Epilepsy
					88	88	095, 370-379, 380-389	076-077, 360-369, 370-379	076, 360-379	Eye diseases
		56	56	66	75	77	307, 322	291, 303	291, 303	Alcoholism
		60, 61a-b	60, 61a-b	23-24, 70-71	17-18, 78a-b, 79	6, 37a-c, 80a-b, 81a-b	057, 082-083, 340-344	036, 045-046, 062-066, 320-324	036, 046-049, 062-064, 320-326	Encephalitis. Meningitis
		71	71	80	86	86	-	-	-	Convulsions
		-	-	22	16	36	080-081	040-044	045, 138	Poliomyelitis
		76	76	86	89a-b	89a-b	390-398	380-389	380-389	Diseases of the ear
Respiratory tuberculosis (including: tuberculosis of the lung and larynx, haemoptysis)	18.1	26-27	28	31	23	13a-b	001-008	010-012	010-012	Respiratory tuberculosis
Diabetes mellitus	18.2	50	50	57	59	61	260	250	250	Diabetes mellitus
Diphtheria. Croup	19, 25	9a-b	9a-b	10	10	10	055	032	032	Diphtheria. Croup
Whooping cough	20	8	8	9	9	9	056	033	033	Whooping cough

1875-1992		1901-1992								
Cause of death	**19th century classification**	**ICD-1 (1901-1910)**	**ICD-2 (1911-1920)**	**ICD-3 (1921-1930)**	**ICD-4 (1931-1940)**	**ICD-5 (1941-1949)**	**ICD-6/7 (1950-1968)**	**ICD-8 (1969-1978)**	**ICD-9 (1979-1992)**	**Cause of death**
Acute respiratory diseases	21	10, 90	10, 89	11a-b, 99a	11a-b, 106a	33a-b, 106a	480-483, 500	466, 470-474	466, 487	Influenza, acute broncitis
		92-93	91-92	100, 101a-b	107-109	107-109	490-493	480-486	480-486	Pneumonia
		94	93	102	110	110a-b	518-519	510-511	510-511	Diseases of the pleural cavity
Chronic respiratory diseases	22	87-88, 100-101	86-87, 99-100	97-98, 108a-b, 109	104-105, 115	104a-b, 105, 115a-d	470-475, 510-514, 516-517, 530-538	460-465, 500-504, 506, 508, 520-529	460-465, 470, 472-476, 478, 520-529	Diseases of larynx, pharynx, nasal cavity, oral cavity
		91, 95-99	90, 94-98	99b-d, 103-106, 107a-b	106b-c, 111-113, 114a-b	106b-c, 111a-c, 112-113, 114a-e	240-241, 465, 501-502, 520-527	450, 490-493, 507, 512-519	415, 477, 490-496, 500-508, 512-519	Chronic bronchitis. Asthma. Other diseases of the lung
Diseases of the circulatory system. Rheumatism. Arthritis.	23, 24	47, 73	47, 72	51, 81	56, 87a	58a-d, 87a	400-402	390-392	390-392	Rheumatic fever. Chorea
		80	80	89	94	94a-b	420	410-414	410-414	Angina pectoris. Ischemic heart disease.
		77-79, 85-86	77-79, 85a-85b	87-88, 90, 95-96	90	90a-b	432	393, 420-423	393, 420, 423	Diseases of the pericardium
					91	91a-c	430	421	421	Acute endocarditis
					92	92a-c	410-414	394-397, 424	394-397, 424	Chronic endocarditis
					93a	93a	431	422	422	Acute myocarditis

					93b-c, 95a-b	93b-d, 95a-c	415-416, 422, 433-434	398, 425-429	398, 416-417, 425-429	Chronic myocarditis. Functional heart disease. Other heart disease
					102a-b, 103	102-103	440-447, 467	400-404, 458	401-405, 458-459	Hypertension. Hypotension
		81-83, 142	81-83, 142	91a-c, 92-93, 151	96-97, 99-100, 98a-b	96-99, 100a-b	450-456, 460-464, 466	445, 451-456	445, 451-456	Diseases of arteries and venes.
Acute diseases of the digestive system. Diarrhoea. Dysentery.	26.1, 26.2, 28	14a-b, 105a-b, 106	14, 104-105	16a-c, 111b, 113-114	13a-c, 117b, 119, 120a-b	27a-c, 117b, 119, 120a-b, 123a	045-048, 541, 571-572	004, 006-009, 532, 561-563	004, 006-009, 532, 555-558, 562	Diarrhoea. Dysentery. Enteritis.
		116	117	126	129	129	576-577	567	567	Peritonitis
		118	108	117	121	121	550-553	540-543	540-543	Appendicitis
Cholera (including: Asiatic cholera, cholera nostras)	27.1, 27.2	12-13	12-13	14-15	12	4	043	000	001	Cholera
Chronic diseases of the digestive system	29	29	31	33	25	15a-b	011	014	014	Abdominal tuberculosis
		103	102	111a	117a	117a	540, 542	531, 533-534	531, 533-534	Stomach ulcer
		102	101	110	116	116	539	530	530	Diseases of the oesophagus
		104, 108-110, 112-114, 117	103, 109-111, 113-115, 118	112, 118a-b, 119a-b, 120, 122a-b, 123-125, 127	118, 123	118, 123b	543-545, 573-575, 578	535-537, 564-566, 568-569	535-537, 564-566, 568-569, 578-579	Other diseases stomach and intestines
					124a-b	124a-b	581	571	571	Liver cirrhosis
					125, 127	125a-b, 127a-b	580, 582-583, 585-586	570, 572-573, 575-576	570, 572-573, 575-576	Other diseases liver and gallbladder

1875-1992		1901-1992								
Cause of death	19th century classification	ICD-1 (1901-1910)	ICD-2 (1911-1920)	ICD-3 (1921-1930)	ICD-4 (1931-1940)	ICD-5 (1941-1949)	ICD-6/7 (1950-1968)	ICD-8 (1969-1978)	ICD-9 (1979-1992)	Cause of death
					128	128	587	577	577	Diseases of the pancreas
					126	126	584	574	574	Gallstones
					122a-b	122a-b	560-561, 570	550-553, 560	550-553, 560	Abdominal hernia and intestinal occlusion
Diseases of the genito-urinary system	30	37-38	38	39, 40a-b	35a-c	25, 44a	030-039	098-099	098-099	Venereal infections (syphilis excluded)
		119	119	128	130	130	590	580	580	Acute nephritis
		120-121	120-122	129-131	131-132, 133a-b	131a-c, 132, 133a-b	591-594, 600, 601, 603	581-584, 590, 591, 593	581-591, 593	Chronic nephritis. Other kidney diseases
		122-124	123-125	132-133, 134a-b	134a-c, 135a-b, 136a-b	134a-c, 135a-b, 136a-b	602, 604-609	592, 594-599	592, 594-599	Diseases of the bladder, urethra, and other organs urinary tract
		125-126	126-127	135-136	137-138	137a-b, 138	610-617	600-607	600-608	Diseases of the prostate. Diseases of other male genital organs
		127-133	128-133	137-140, 141a-b, 142	54a, 55a, 139a-d	56a-c, 57a-c, 139a-d	214-217, 233-235, 620-626, 630-637	218-221, 234-236, 610-616, 620-629	218-221, 610-611, 614-629	Diseases of female genital organs

Puerperal diseases	31.1	134-136, 138-141	134-136, 138-141	143a-c, 144-145, 147-150	141-143, 144a-b, 145b, 146-147, 148a-b, 149-150	141a-b, 142, 143a-c, 144a-d, 145, 146a-d, 147c-d, 148a-d, 149a-b, 150a-c	640-650, 652, 660, 670-678, 680, 682-689	630-645, 650-662, 671-678	630-639, 640-648, 650-669, 671-676	Bleeding and other diseases of pregnancy
Puerperal fever	31.2	137	137	146	140, 145a	140a-b, 147a-b	651, 681	670	670	Puerperal fever
Other diseases (including: debility, some types of tuberculosis, scrofula, rickets, skin diseases, abscess, ulcer, gangrene, pyaemia, haemorrhage, continuous fever)	2, 3.1, 4, 8, 13	18	18	21	15	11	052	035	035	Erysipelas
		22	22	27	20	7	062	022	022	Anthrax
		30-35	29, 32-35	34-35, 36a-e, 37a-b	26-31, 32a-c	16, 17a-c, 18-20, 21a-b, 22a-c	012-019	015-019	015-018, 137	Disseminated and other tuberculosis
		48, 146-149	36, 48, 146-149	52, 56, 156-158	57a-b, 58, 63-64, 154-155, 156a-b	59a1-a3, 59b, 60, 66a, 70, 154a-c, 155, 156a-b	283-285, 288, 720-727, 730-738, 740-749	265, 274, 710-718, 720-738	268, 274, 710-739	Arthritis. Rickets. Diseases of locomotion
		52	52	63	68	65a-b	274	255	255	Adrenal diseases
		154	154	164	162b	162b	794	794	797	Old age
					162a	162a	304	290	290	Dementia
		151-153	151-153	160, 161a-b, 162-163	158-159, 160a-b, 161a-c	158-159, 160a1-b2, 161a-e	770-776	760-779	760-779	Diseases of newly born
		20	20	41	36a-b	24a-c	053-054, 063	038	038	Septicaemia. Pyaemia

1875-1992		1901-1992								
Cause of death	19th century classification	ICD-1 (1901-1910)	ICD-2 (1911-1920)	ICD-3 (1921-1930)	ICD-4 (1931-1940)	ICD-5 (1941-1949)	ICD-6/7 (1950-1968)	ICD-8 (1969-1978)	ICD-9 (1979-1992)	Cause of death
		2-3, 11, 15-17, 19, 21, 23-24, 72, 107, 111, 144	2-3, 11, 15-17, 19, 21, 23-25, 106-107, 112, 144	2-4, 12-13, 17a-d, 18-20, 25-26, 28-30, 42, 115, 116a-f, 121, 153	3-5, 14a-d, 19a-b, 21-22, 33, 37, 39a-b, 40, 41a-b, 42-44, 152	3, 5, 12, 23, 26a-c, 29, 31, 32a-b, 38a-f, 39-43, 44c-d, 152	042, 044, 058-061, 064, 070-074, 086-094, 096, 100-108, 120-124, 135-138, 691-693	003, 020-021, 023-027, 030-031, 037, 039, 051-054, 056-057, 060-061, 067-068, 070-075, 078-083, 085-089, 100-104, 110-117, 120-136,681-682	003, 020-021, 023-027, 030-031, 037, 039-044, 051-054, 056-057, 060-061, 065-066, 070-075, 077-083, 085-088, 100-104, 110-118, 120-136, 139, 681-682	Other infectious diseases
		25, 46, 53-55, 68, 84, 115, 143, 145	26-27, 46, 53-55, 68, 84, 116, 143, 145	50, 54-55, 58a-b, 59, 62, 64, 65a-b, 69, 77, 94, 152, 154a-b, 155	54b, 55b, 61-62, 65, 67, 69, 70a-b, 71a-b, 72a-c, 73-74, 84a-b, 101, 151, 153	44b, 56d-e, 57d-e, 62, 64, 66b, 68-69, 71, 72a-c, 73a-d, 74a-b, 75, 76a-d, 84a-d, 101, 151, 153	201, 204, 210-213, 218-232, 236-239, 242-245, 270, 272-273, 275-277, 280-281, 286-287, 289-303, 305-306, 308-318, 320-321, 324-	201, 204-217, 222-228, 230-233, 237-239, 251, 253-254, 256-258, 260-263, 266-273, 275-289, 292-302, 305-315, 457, 505, 680, 683-	201, 204-208, 210-217, 222-239, 251, 253-254, 256-266, 269-273, 275-289, 292-302, 306-319, 471, 680, 683-686, 690-698, 700-709	Other diseases

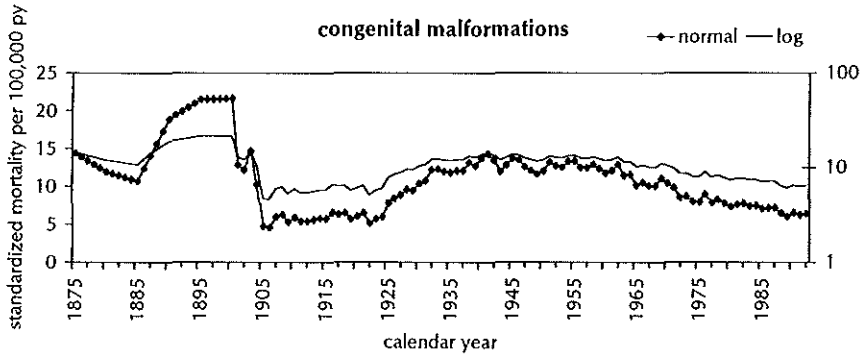
							326, 468, 515, 690, 694-698, 700-716	686, 690-698, 700-709		
Violence	32.1, 33.1	-	182-184	197-200	172a-b, 173-175	165-168	E980-983, E964	E960-969	E960-969	Homicide
		-	175	188b	186a1-a4	169, 170a-c, 171a-b	E800-802, E810-825, E830-835, E840-845, E960	E800-807, E810-823, E825-827, E940	E800-807, E810-829	Traffic accidents
		57a-b, 58-59, 164-172, 174-176	57a-b, 58-59, 164-174, 176, 178-181, 185-186	67-68, 175-180, 181a-b, 182-187, 188a, 189-191, 193-196, 201a-c, 202-203	76a-b, 77a-b, 176-177, 178a-b, 179-185, 186a5-a6, b1-b3, 187-188, 190-193, 194a-b, 195-198	78a-b, 79a-b, 172-174, 175a-c, 176-188, 191-194, 195a, b1-b2, c-e, 196a-b, 197-198	049, 323, E850-858, E860-866, E870-888, E890-895, E900-904, E910-932, E934-936, E940-946, E950-959, E961-962, E965, E984-985, E990-999	005, 304, E830-838, E840-845, E850-877, E880-903, E905-936, E941-949, E970-978, E980-999	005, 304-305, E830-838, E840-848, E850-876, E878-888, E890-903, E905-949, E970-978, E980-999	Other external causes of death
Suicide	32.2, 33.2	155-163	155-163	165-174	163-171	163a1-b3, 164a-g	E963, E970-979	E950-959	E950-959	Suicide
Unspecified and ill-defined or unknown causes of death. Sudden death. Dropsy.	6.1, 34.1, 34.2	173, 177-178, 179a-b	177, 187-188, 189a-b	192a-b, 204-205, 206a-b	189a-b, 199-200	189, 199, 200a-c	780-793, 795, E933	780-793, 795-796, E904	780-796, 798-799, E904	Unknown and ill-defined causes of death. Sudden death.

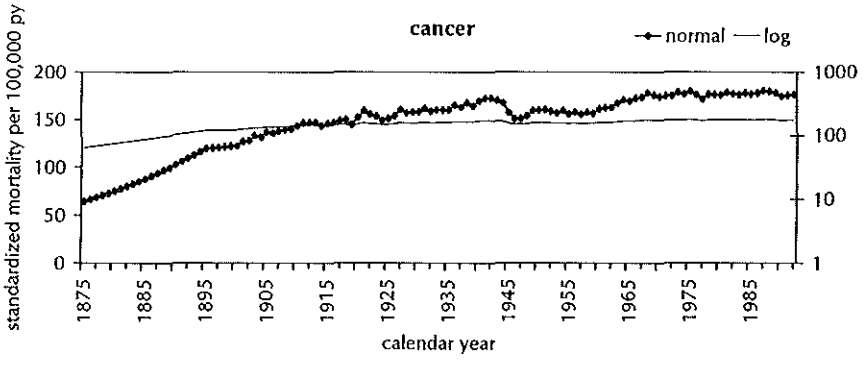
Appendix 2a

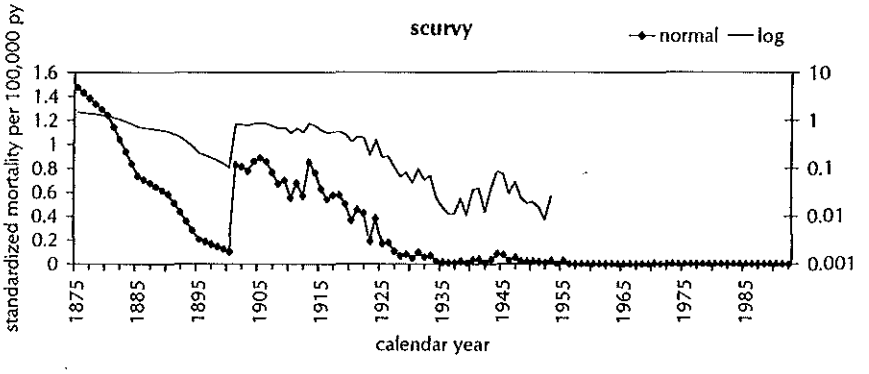
TRENDS OF CAUSES OF DEATH THAT COULD BE STUDIED FOR THE PERIOD 1875/79 TO 1992*

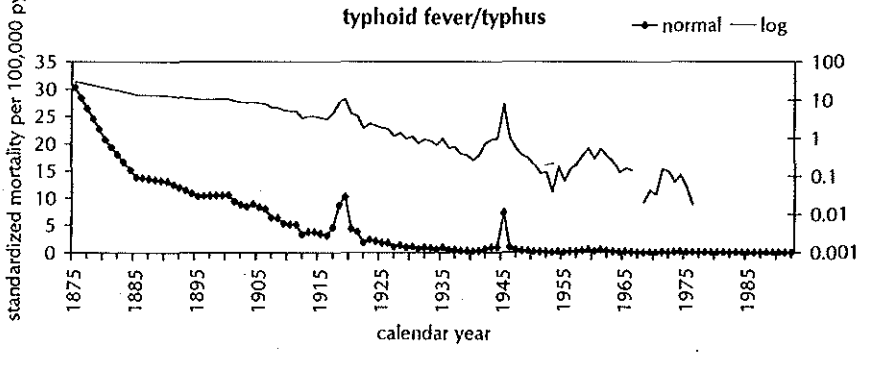
See appendix 1 for the corresponding ICD codes

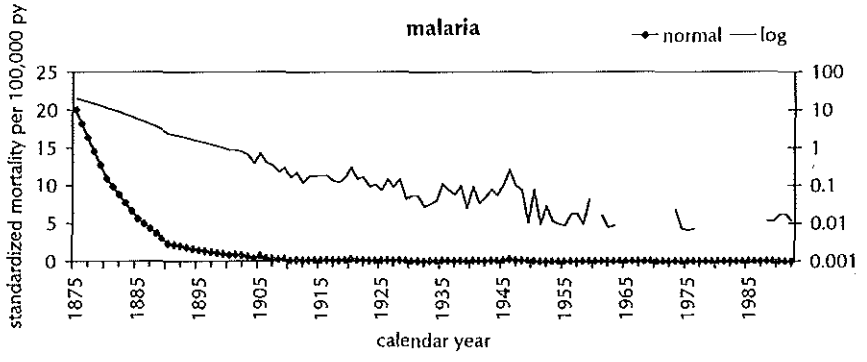
* Age- and sex-standardised mortality rates. Direct standardisation with the average population of 1901-1992 as the reference population.
1875/79 to 1895/99 are quinquennial figures. Figures for individual years have been interpolated.
Trends on the log-scale will be interrupted if the mortality rate is zero.

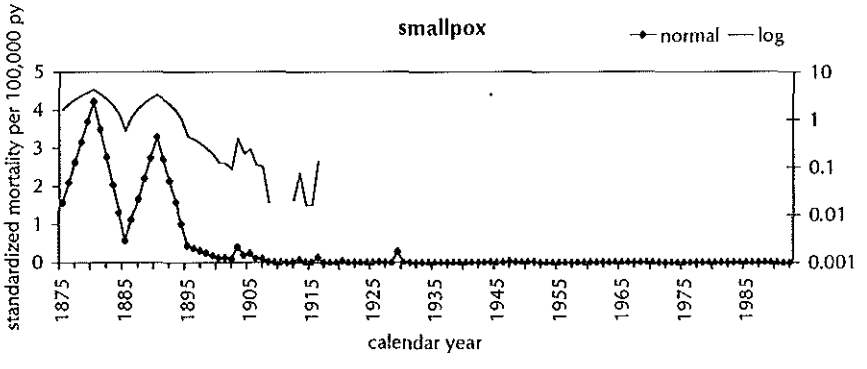

congenital malformations
normal
log
standardized mortality per 100,000 py
25
20
15
10
5
0
100
10
1
1875
1885
1895
1905
1915
1925
1935
1945
1955
1965
1975
1985
calendar year

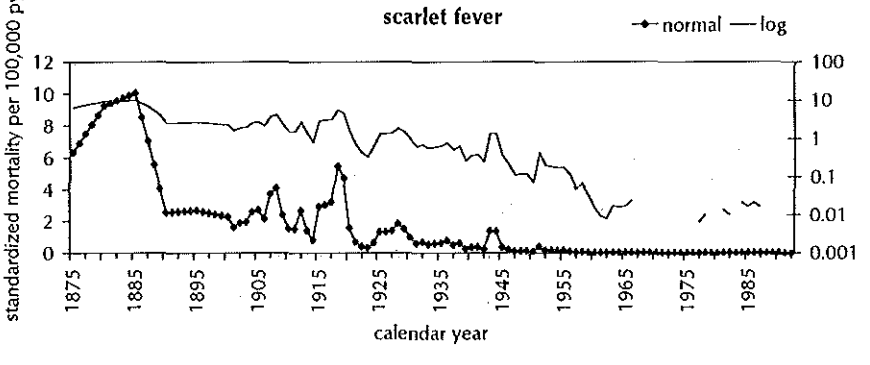

cancer
normal
log
standardized mortality per 100,000 py
200
150
100
50
0
1000
100
10
1
1875
1885
1895
1905
1915
1925
1935
1945
1955
1965
1975
1985
calendar year

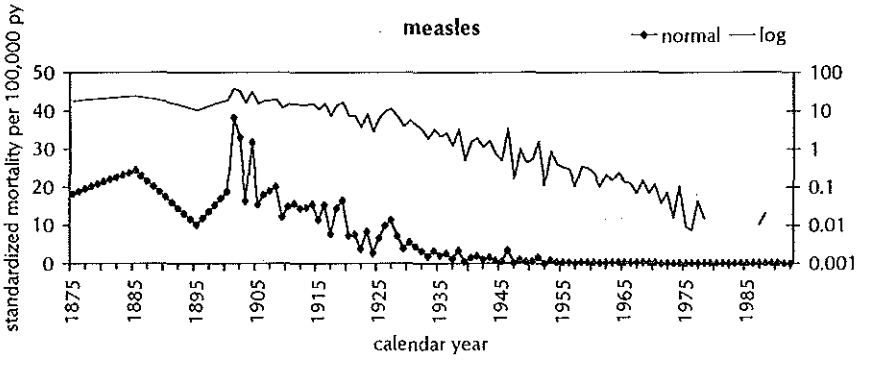

scurvy
normal
log
standardized mortality per 100,000 py
1.6
1.4
1.2
1
0.8
0.6
0.4
0.2
0
10
1
0.1
0.01
0.001
1875
1885
1895
1905
1915
1925
1935
1945
1955
1965
1975
1985
calendar year

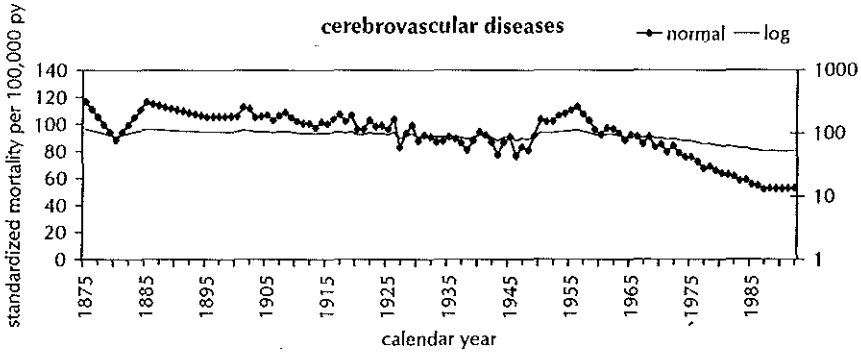

typhoid fever/typhus
normal
log
standardized mortality per 100,000 py
35
30
25
20
15
10
5
0
100
10
1
0.1
0.01
0.001
1875
1885
1895
1905
1915
1925
1935
1945
1955
1965
1975
1985
calendar year

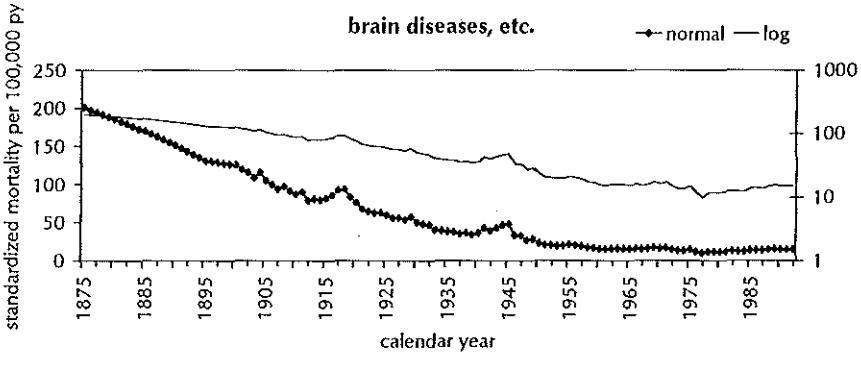

malaria
normal
log
standardized mortality per 100,000 py
25
20
15
10
5
0
100
10
1
0.1
0.01
0.001
1875
1885
1895
1905
1915
1925
1935
1945
1955
1965
1975
1985
calendar year

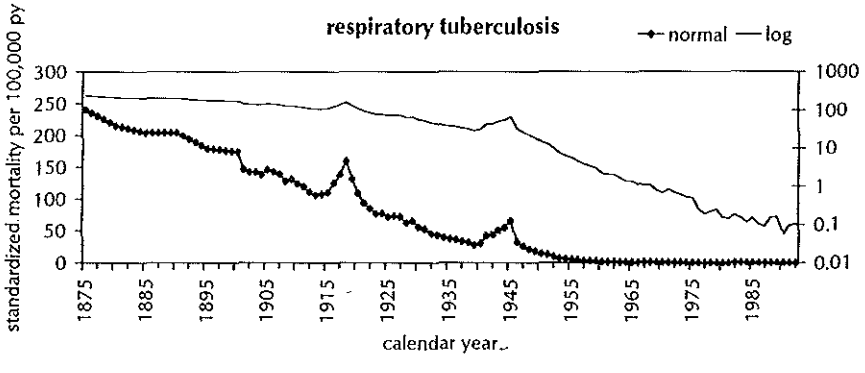

smallpox
normal
log
standardized mortality per 100,000 py
5
4
3
2
1
0
10
1
0.1
0.01
0.001
1875
1885
1895
1905
1915
1925
1935
1945
1955
1965
1975
1985
calendar year

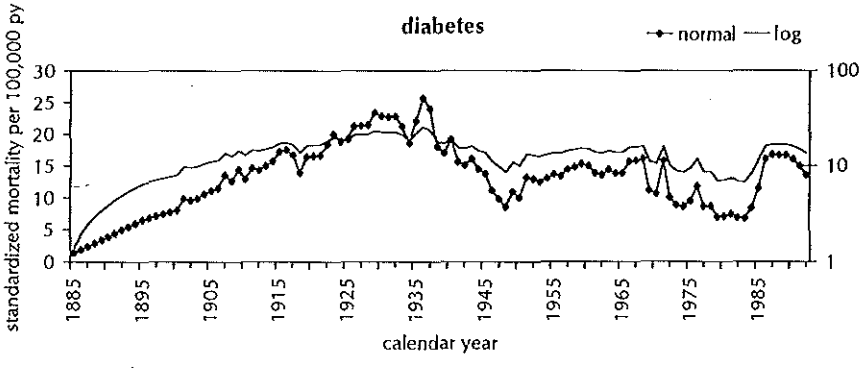

scarlet fever
normal
log
standardized mortality per 100,000 py
12
10
8
6
4
2
0
100
10
1
0.1
0.01
0.001
1875
1885
1895
1905
1915
1925
1935
1945
1955
1965
1975
1985
calendar year

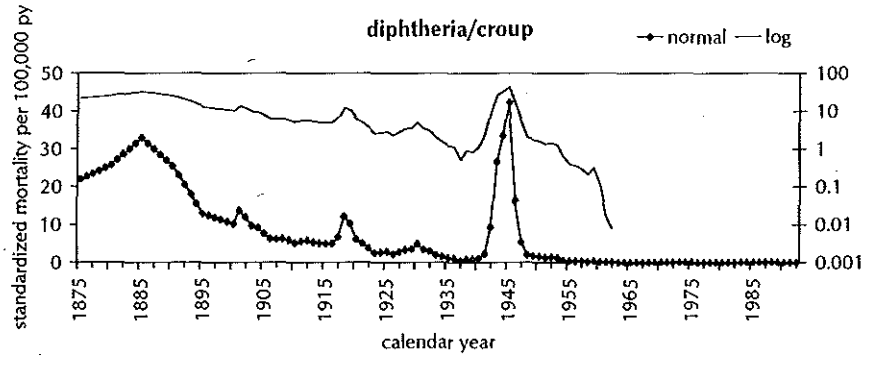

measles
normal
log
standardized mortality per 100,000 py
50
40
30
20
10
0
100
10
1
0.1
0.01
0.001
1875
1885
1895
1905
1915
1925
1935
1945
1955
1965
1975
1985
calendar year

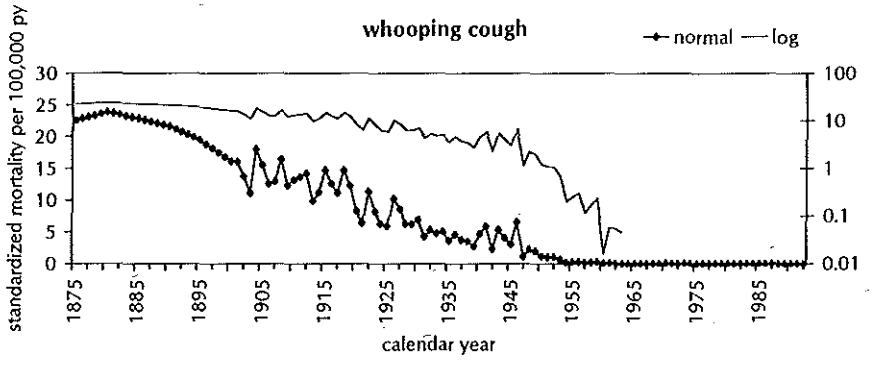

cerebrovascular diseases
normal
log
standardized mortality per 100,000 py
140
120
100
80
60
40
20
0
1000
100
10
1
1875
1885
1895
1905
1915
1925
1935
1945
1955
1965
1975
1985
calendar year

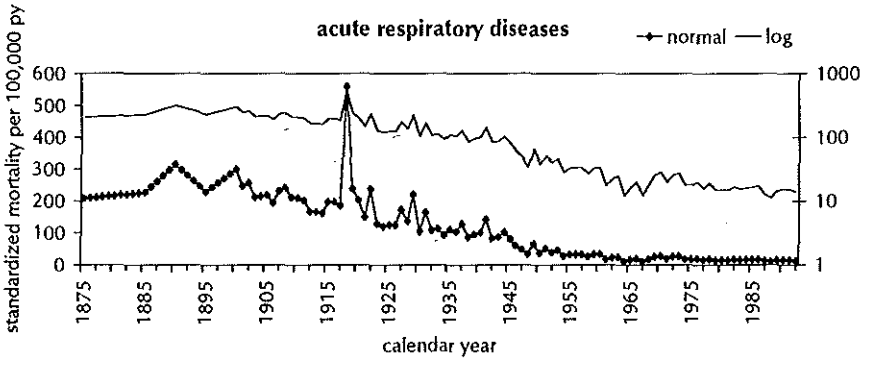

brain diseases, etc.
normal
log
standardized mortality per 100,000 py
250
200
150
100
50
0
1000
100
10
1
1875
1885
1895
1905
1915
1925
1935
1945
1955
1965
1975
1985
calendar year

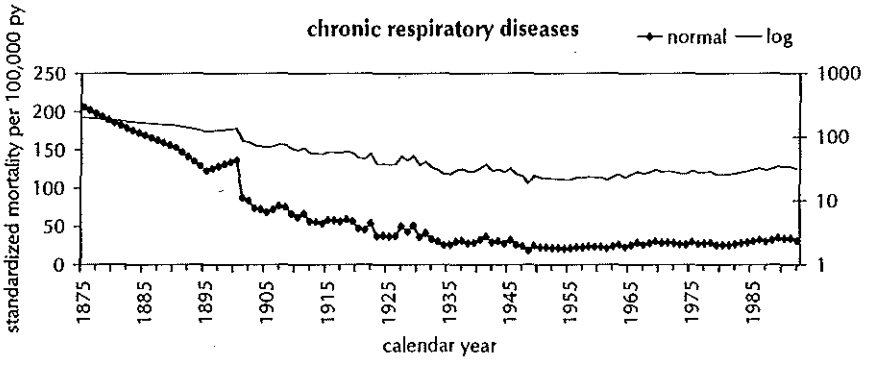

respiratory tuberculosis
normal
log
standardized mortality per 100,000 py
300
250
200
150
100
50
0
1000
100
10
1
0.1
0.01
1875
1885
1895
1905
1915
1925
1935
1945
1955
1965
1975
1985
calendar year

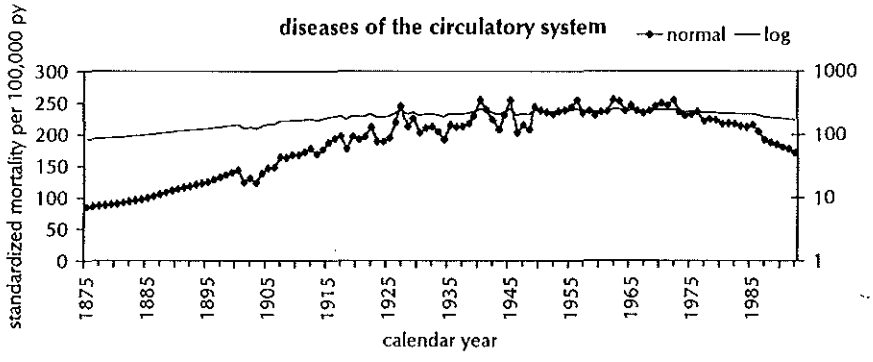

diabetes
normal
log
standardized mortality per 100,000 py
30
25
20
15
10
5
0
100
10
1
1885
1895
1905
1915
1925
1935
1945
1955
1965
1975
1985
calendar year

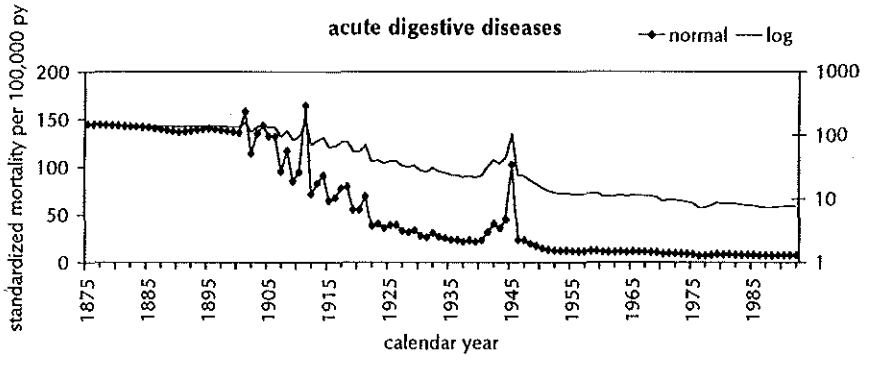

diphtheria/croup
normal
log
standardized mortality per 100,000 py
50
40
30
20
10
0
100
10
1
0.1
0.01
0.001
1875
1885
1895
1905
1915
1925
1935
1945
1955
1965
1975
1985
calendar year

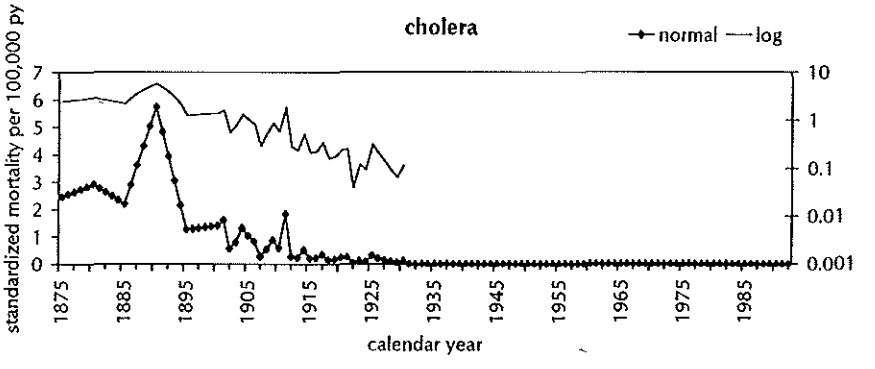

whooping cough
normal
log
standardized mortality per 100,000 py
30
25
20
15
10
5
0
100
10
1
0.1
0.01
1875
1885
1895
1905
1915
1925
1935
1945
1955
1965
1975
1985
calendar year

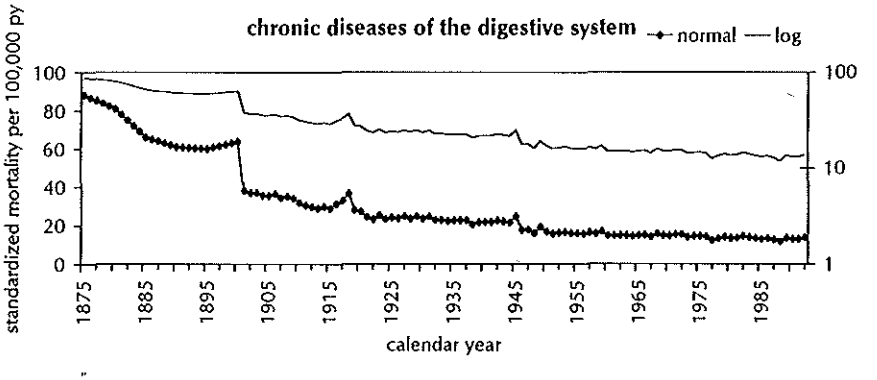

acute respiratory diseases
normal
log
standardized mortality per 100,000 py
600
500
400
300
200
100
0
1000
100
10
1
1875
1885
1895
1905
1915
1925
1935
1945
1955
1965
1975
1985
calendar year

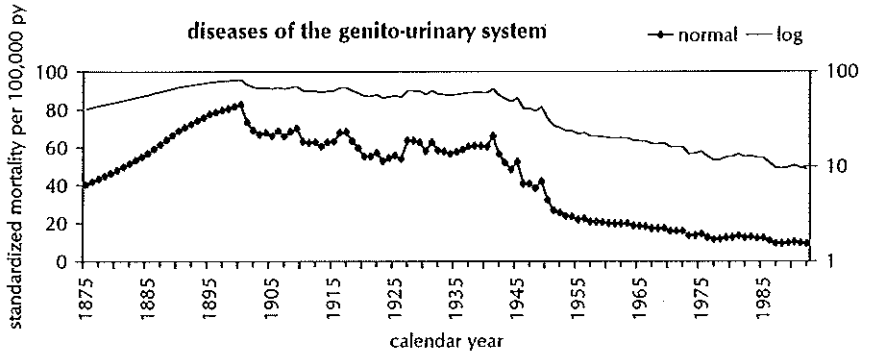

chronic respiratory diseases
normal
log
standardized mortality per 100,000 py
250
200
150
100
50
0
1000
100
10
1
1875
1885
1895
1905
1915
1925
1935
1945
1955
1965
1975
1985
calendar year

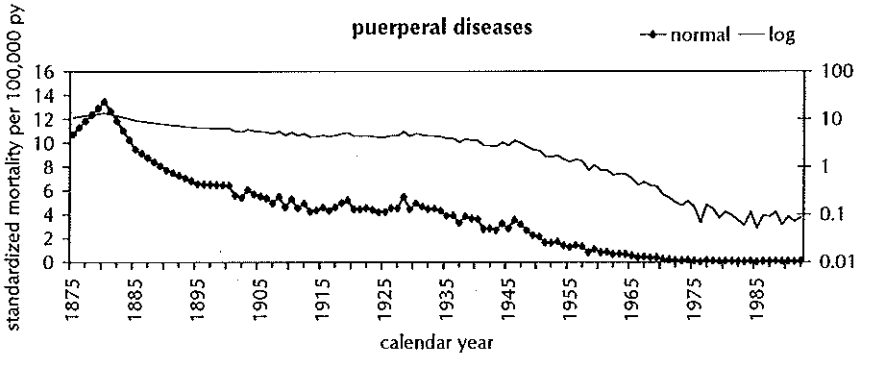

diseases of the circulatory system
normal
log
standardized mortality per 100,000 py
300
250
200
150
100
50
0
1000
100
10
1
1875
1885
1895
1905
1915
1925
1935
1945
1955
1965
1975
1985
calendar year

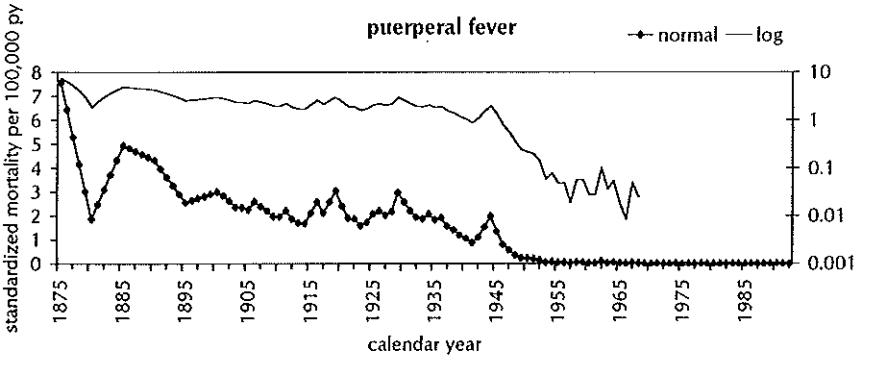

acute digestive diseases
normal
log
standardized mortality per 100,000 py
200
150
100
50
0
1000
100
10
1
1875
1885
1895
1905
1915
1925
1935
1945
1955
1965
1975
1985
calendar year

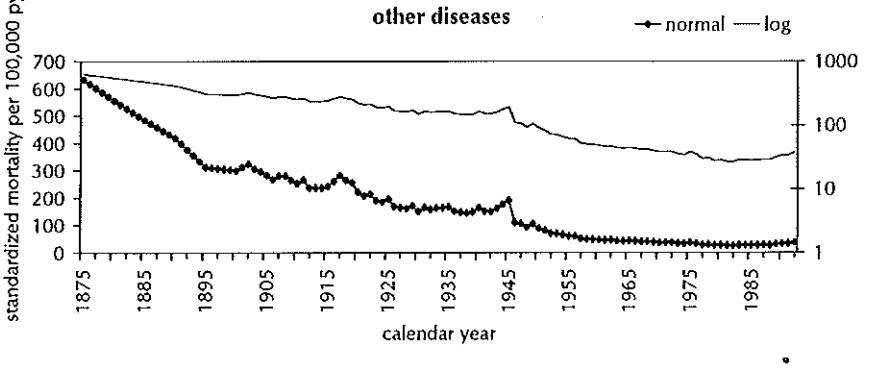

cholera
normal
log
standardized mortality per 100,000 py
7
6
5
4
3
2
1
0
10
1
0.1
0.01
0.001
1875
1885
1895
1905
1915
1925
1935
1945
1955
1965
1975
1985
calendar year

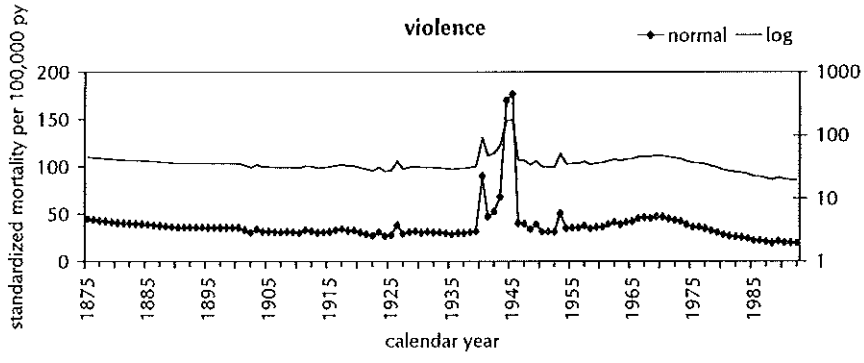

chronic diseases of the digestive system
normal
log
standardized mortality per 100,000 py
100
80
60
40
20
0
100
10
1
1875
1885
1895
1905
1915
1925
1935
1945
1955
1965
1975
1985
calendar year

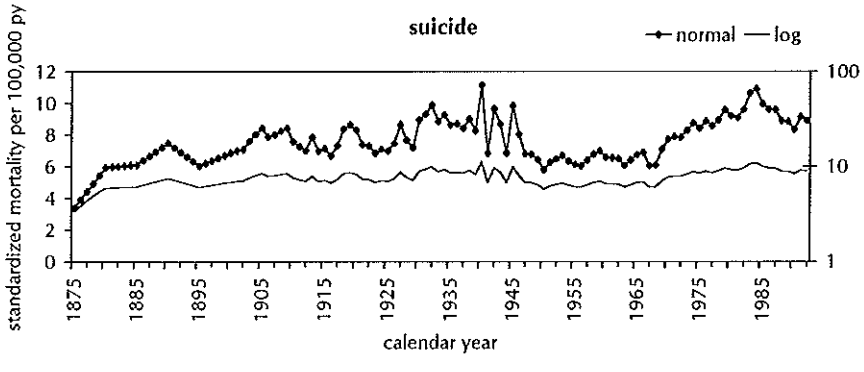

diseases of the genito-urinary system
normal
log
standardized mortality per 100,000 py
100
80
60
40
20
0
100
10
1
1875
1885
1895
1905
1915
1925
1935
1945
1955
1965
1975
1985
calendar year

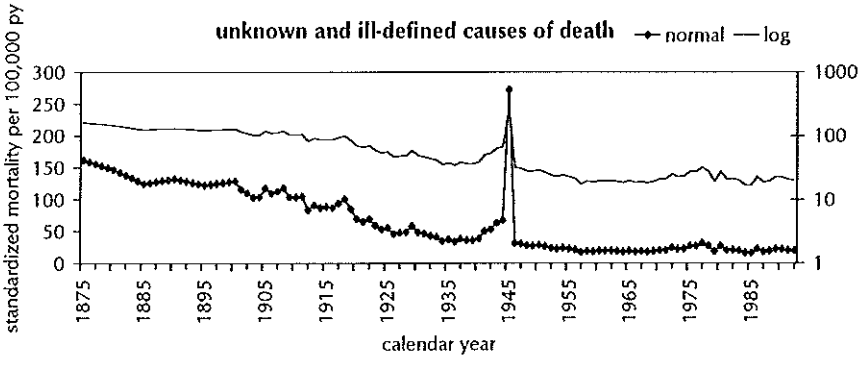

puerperal diseases
normal
log
standardized mortality per 100,000 py
16
14
12
10
8
6
4
2
0
100
10
1
0.1
0.01
1875
1885
1895
1905
1915
1925
1935
1945
1955
1965
1975
1985
calendar year


puerperal fever
normal
log
standardized mortality per 100,000 py
8
7
6
5
4
3
2
1
0
10
1
0.1
0.01
0.001
1875
1885
1895
1905
1915
1925
1935
1945
1955
1965
1975
1985
calendar year


other diseases
normal
log
standardized mortality per 100,000 py
700
600
500
400
300
200
100
0
1000
100
10
1
1875
1885
1895
1905
1915
1925
1935
1945
1955
1965
1975
1985
calendar year


violence
normal
log
standardized mortality per 100,000 py
200
150
100
50
0
1000
100
10
1
1875
1885
1895
1905
1915
1925
1935
1945
1955
1965
1975
1985
calendar year


suicide
normal
log
standardized mortality per 100,000 py
12
10
8
6
4
2
0
100
10
1
1875
1885
1895
1905
1915
1925
1935
1945
1955
1965
1975
1985
calendar year

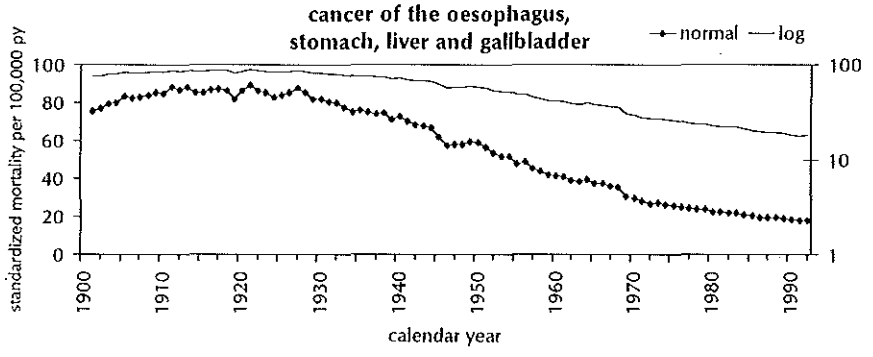

unknown and ill-defined causes of death
normal
log
standardized mortality per 100,000 py
300
250
200
150
100
50
0
1000
100
10
1
1875
1885
1895
1905
1915
1925
1935
1945
1955
1965
1975
1985
calendar year

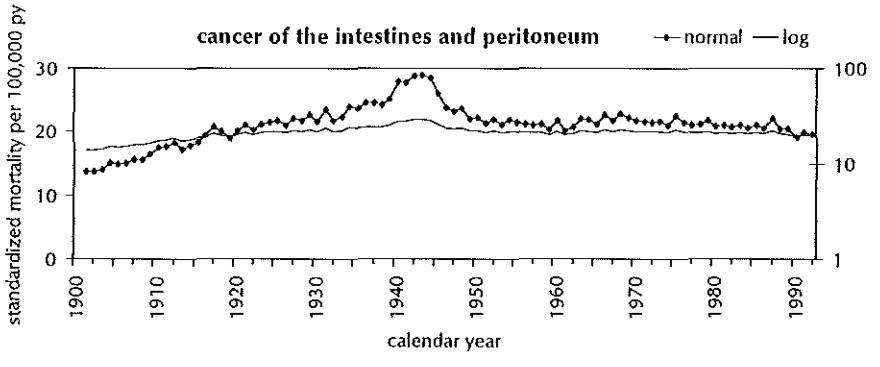
Appendix 2b

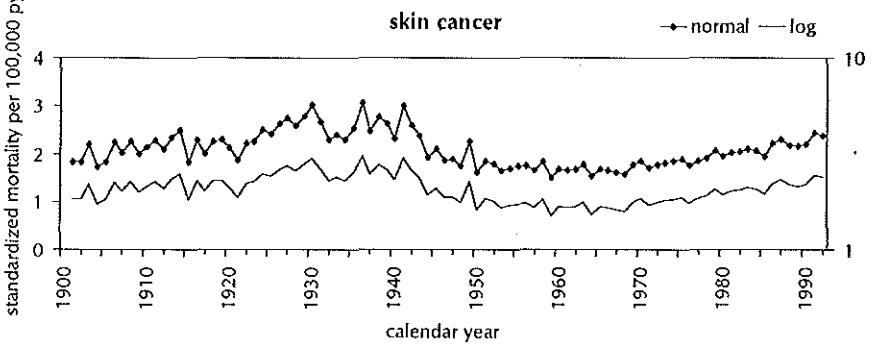
TRENDS OF CAUSES OF DEATH THAT COULD ONLY BE STUDIED FOR THE PERIOD 1901 TO 1992*

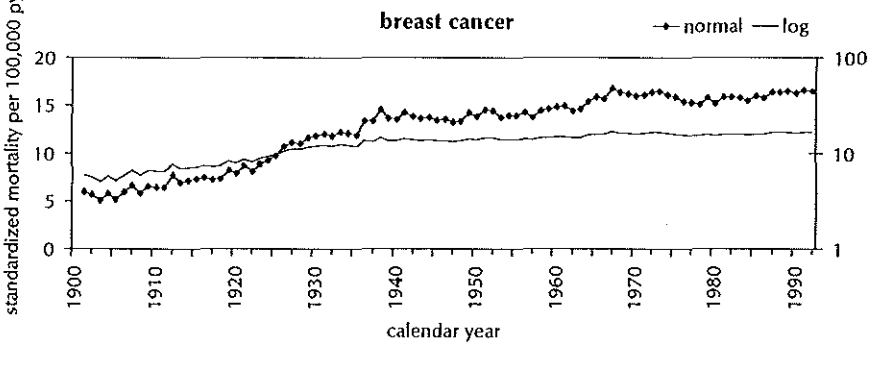
See appendix 1 for the corresponding ICD codes

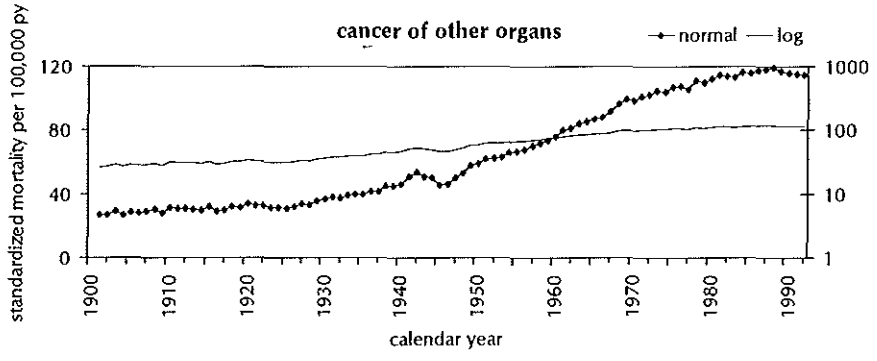
* Age- and sex-standardised mortality rates. Direct standardisation with the average population of 1901-1992 as the reference population.
Trends on the log-scale will be interrupted if the mortality rate is zero.

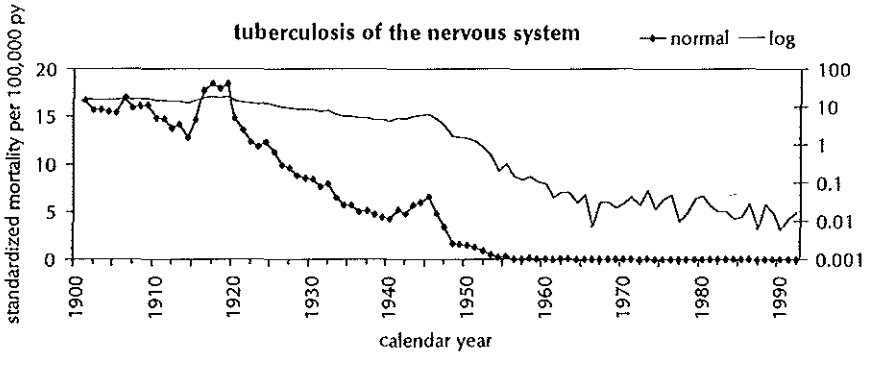

cancer of the oesophagus, stomach, liver and gallbladder
normal
log
standardized mortality per 100,000 py
100
80
60
40
20
0
100
10
1
1900
1910
1920
1930
1940
1950
1960
1970
1980
1990
calendar year

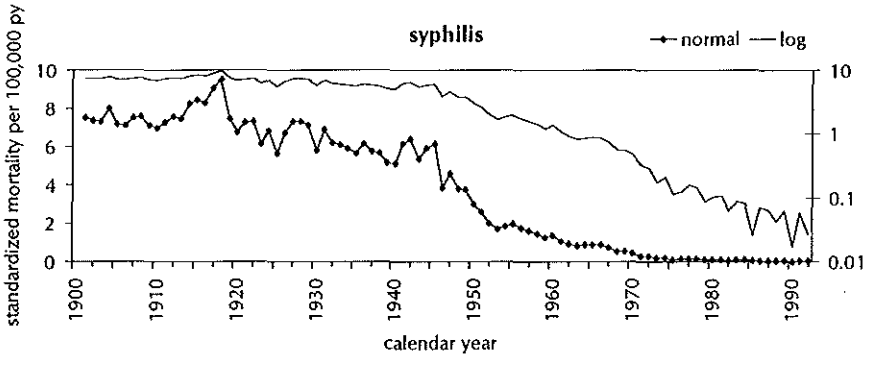

cancer of the intestines and peritoneum
normal
log
standardized mortality per 100,000 py
30
20
10
0
100
10
1
1900
1910
1920
1930
1940
1950
1960
1970
1980
1990
calendar year

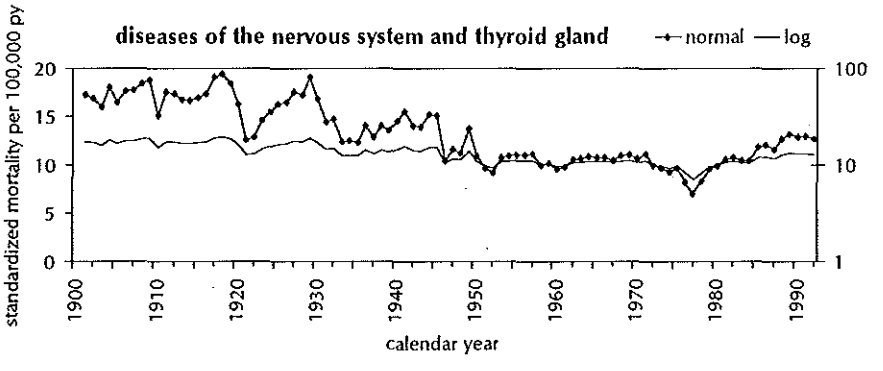

skin cancer
normal
log
standardized mortality per 100,000 py
4
3
2
1
0
10
1
1900
1910
1920
1930
1940
1950
1960
1970
1980
1990
calendar year

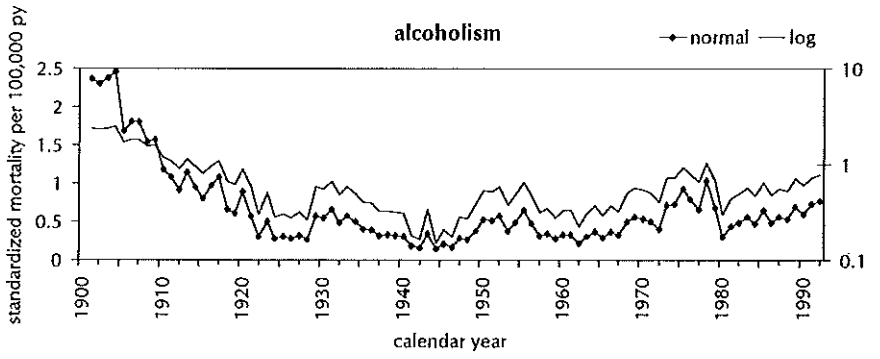

breast cancer
normal
log
standardized mortality per 100,000 py
20
15
10
5
0
100
10
1
1900
1910
1920
1930
1940
1950
1960
1970
1980
1990
calendar year

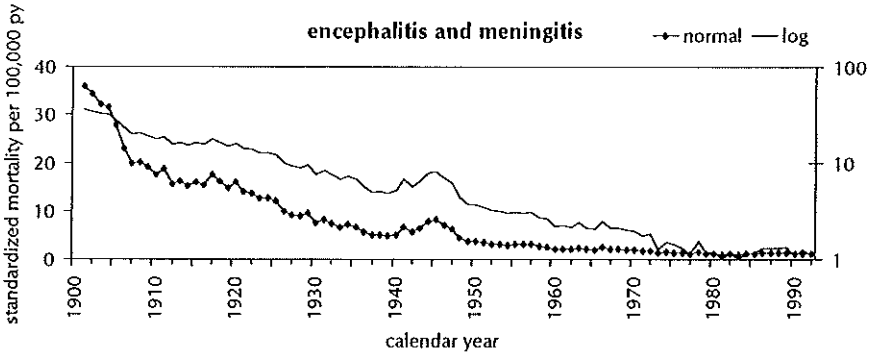

cancer of other organs
normal
log
standardized mortality per 100,000 py
120
80
40
0
1000
100
10
1
1900
1910
1920
1930
1940
1950
1960
1970
1980
1990
calendar year

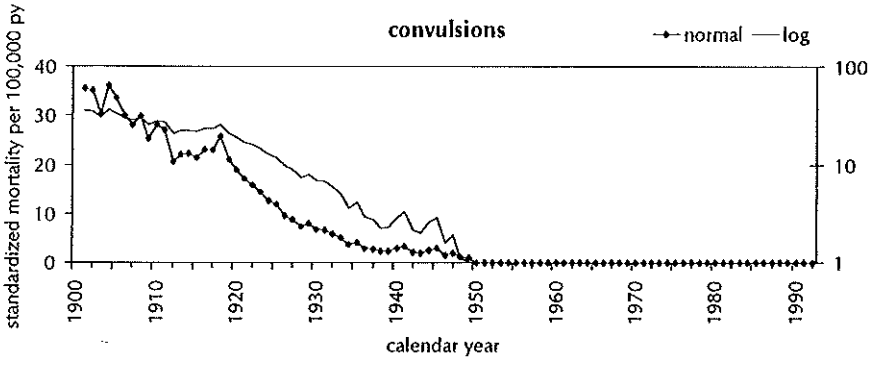

tuberculosis of the nervous system
normal
log
standardized mortality per 100,000 py
20
15
10
5
0
100
10
1
0.1
0.01
0.001
1900
1910
1920
1930
1940
1950
1960
1970
1980
1990
calendar year

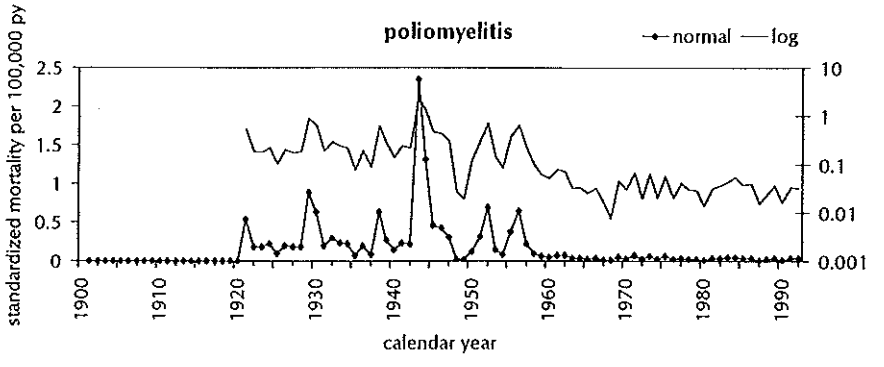

syphilis
normal
log
standardized mortality per 100,000 py
10
8
6
4
2
0
10
1
0.1
0.01
1900
1910
1920
1930
1940
1950
1960
1970
1980
1990
calendar year

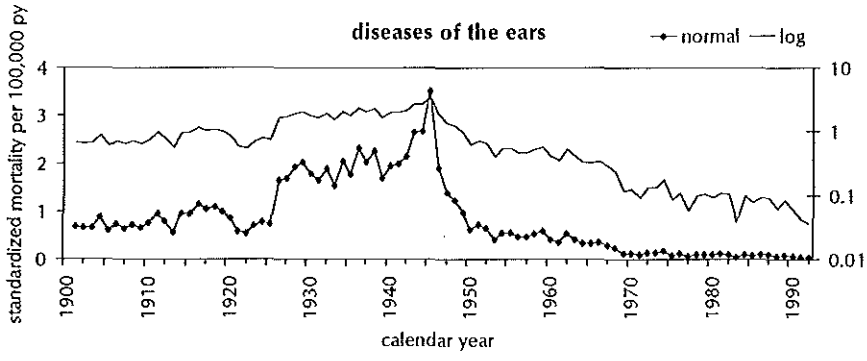

diseases of the nervous system and thyroid gland
normal
log
standardized mortality per 100,000 py
20
15
10
5
0
100
10
1
1900
1910
1920
1930
1940
1950
1960
1970
1980
1990
calendar year

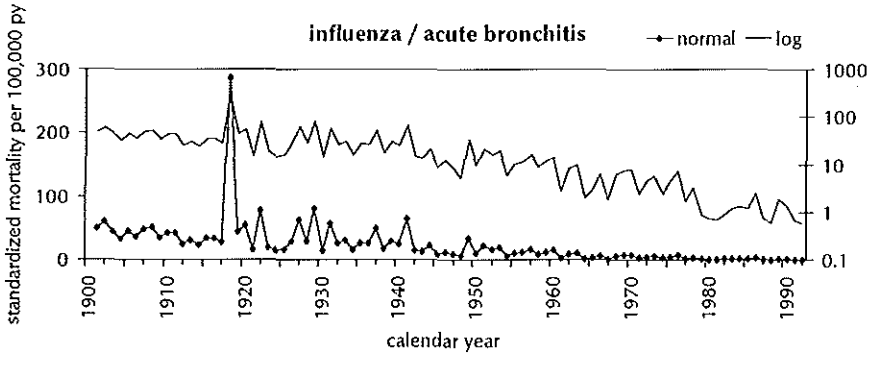

alcoholism
normal
log
standardized mortality per 100,000 py
2.5
2
1.5
1
0.5
0
10
1
0.1
1900
1910
1920
1930
1940
1950
1960
1970
1980
1990
calendar year

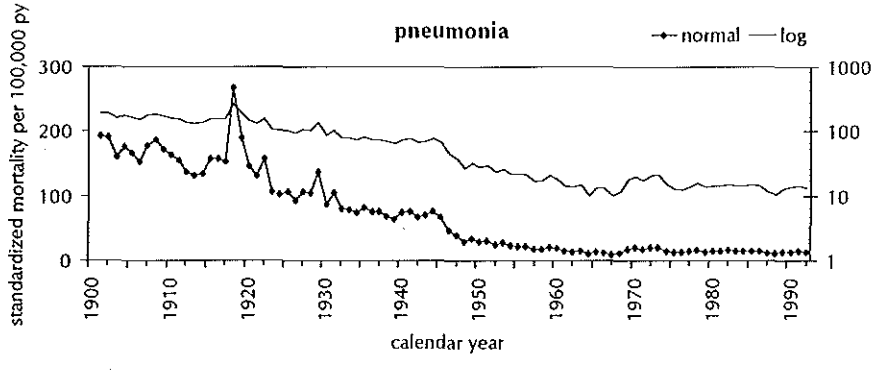

encephalitis and meningitis
normal
log
standardized mortality per 100,000 py
40
30
20
10
0
100
10
1
1900
1910
1920
1930
1940
1950
1960
1970
1980
1990
calendar year

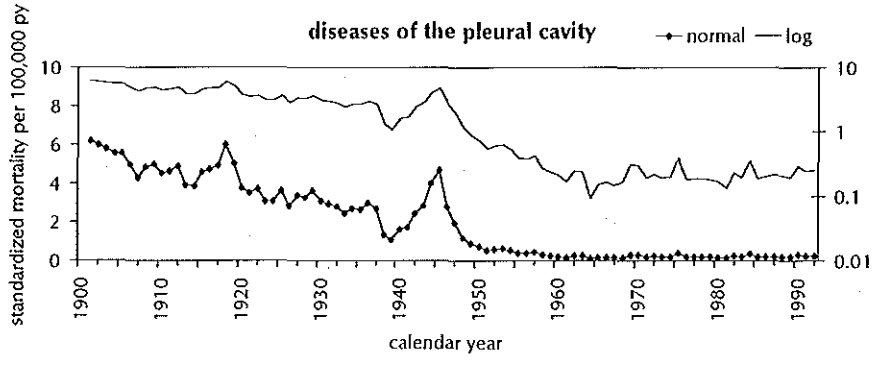

convulsions
normal
log
standardized mortality per 100,000 py
40
30
20
10
0
100
10
1
1900
1910
1920
1930
1940
1950
1960
1970
1980
1990
calendar year

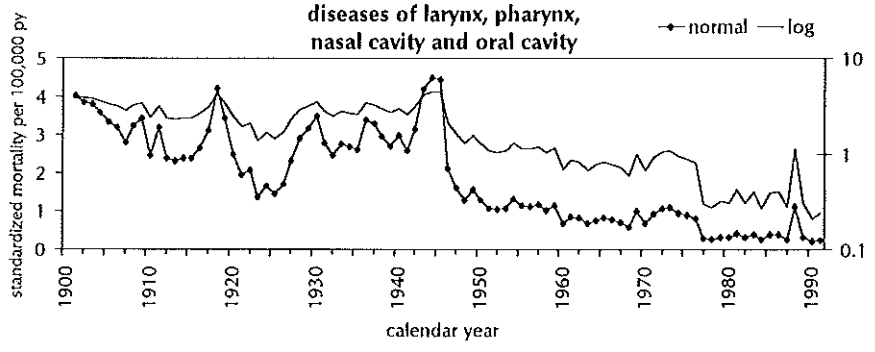

poliomyelitis
normal
log
standardized mortality per 100,000 py
2.5
2
1.5
1
0.5
0
10
1
0.1
0.01
0.001
1900
1910
1920
1930
1940
1950
1960
1970
1980
1990
calendar year

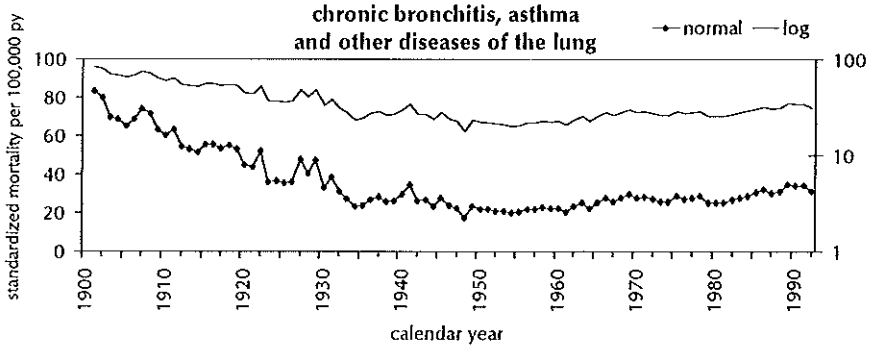

diseases of the ears
normal
log
standardized mortality per 100,000 py
calendar year

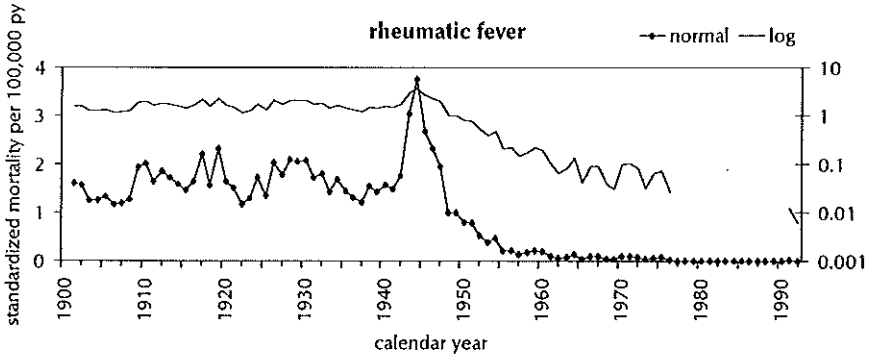

influenza / acute bronchitis
normal
log
standardized mortality per 100,000 py
calendar year

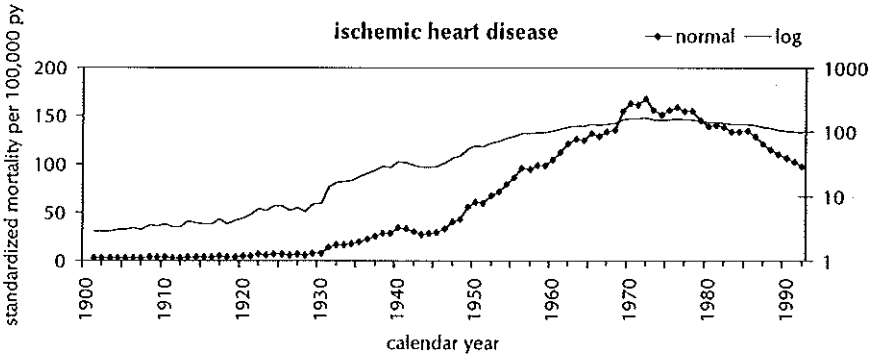

pneumonia
normal
log
standardized mortality per 100,000 py
calendar year

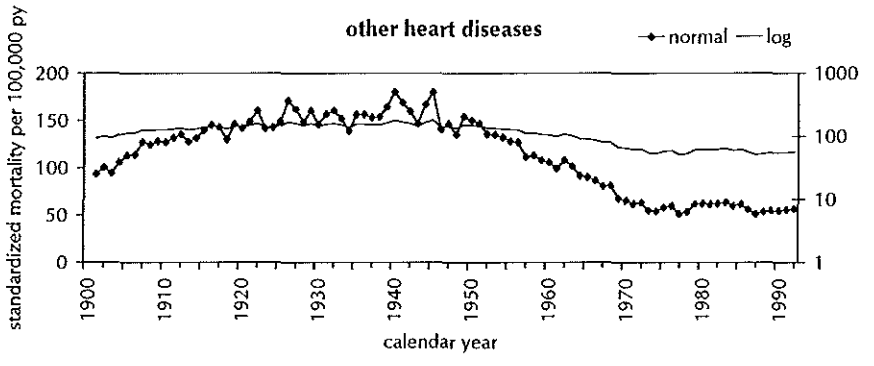

diseases of the pleural cavity
normal
log
standardized mortality per 100,000 py
calendar year

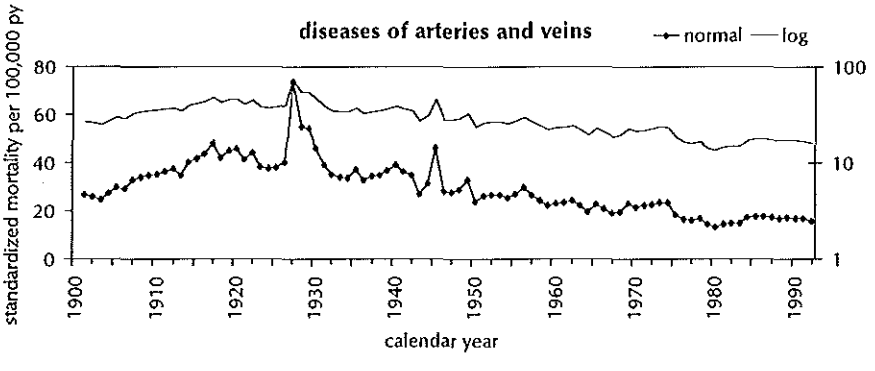

diseases of larynx, pharynx,
nasal cavity and oral cavity
normal
log
standardized mortality per 100,000 py
calendar year

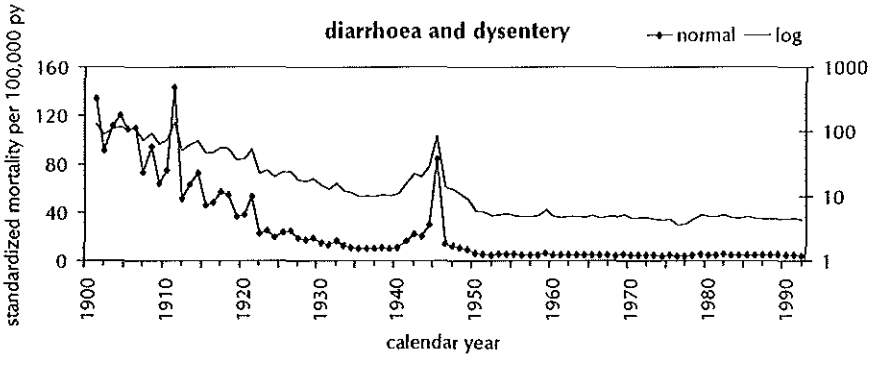

chronic bronchitis, asthma
and other diseases of the lung
normal
log
standardized mortality per 100,000 py
calendar year

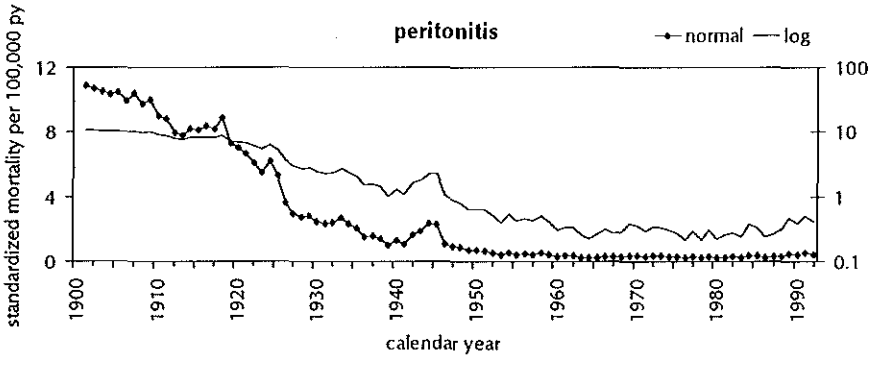

rheumatic fever
normal
log
standardized mortality per 100,000 py
calendar year

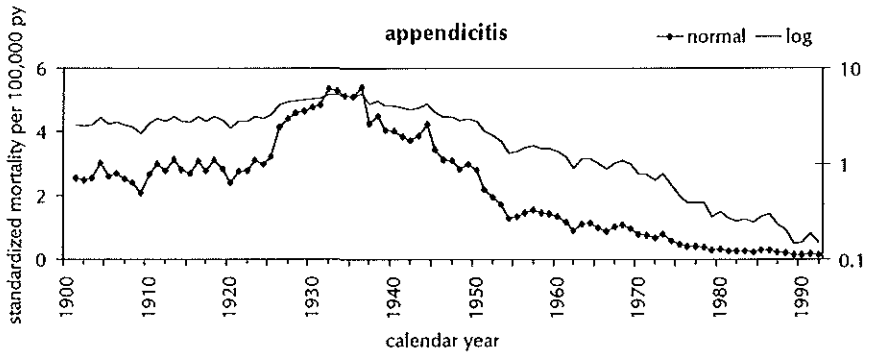

ischemic heart disease
normal
log
standardized mortality per 100,000 py
calendar year

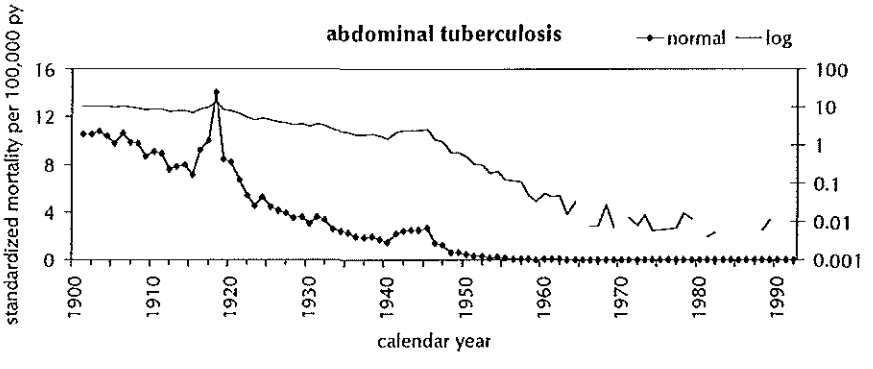

other heart diseases
normal
log
standardized mortality per 100,000 py
200
150
100
50
0
1000
100
10
1
1900
1910
1920
1930
1940
1950
1960
1970
1980
1990
calendar year

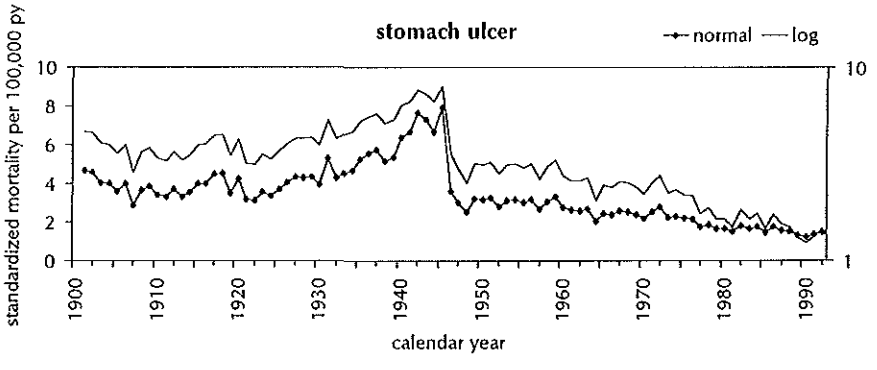

diseases of arteries and veins
normal
log
standardized mortality per 100,000 py
80
60
40
20
0
100
10
1
1900
1910
1920
1930
1940
1950
1960
1970
1980
1990
calendar year

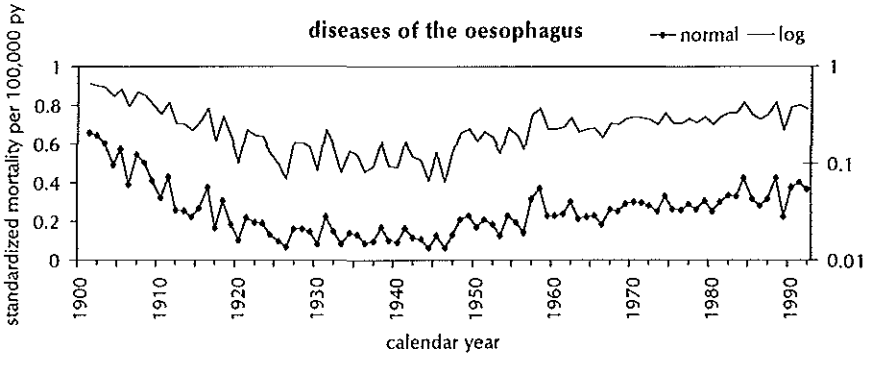

diarrhoea and dysentery
normal
log
standardized mortality per 100,000 py
160
120
80
40
0
1000
100
10
1
1900
1910
1920
1930
1940
1950
1960
1970
1980
1990
calendar year

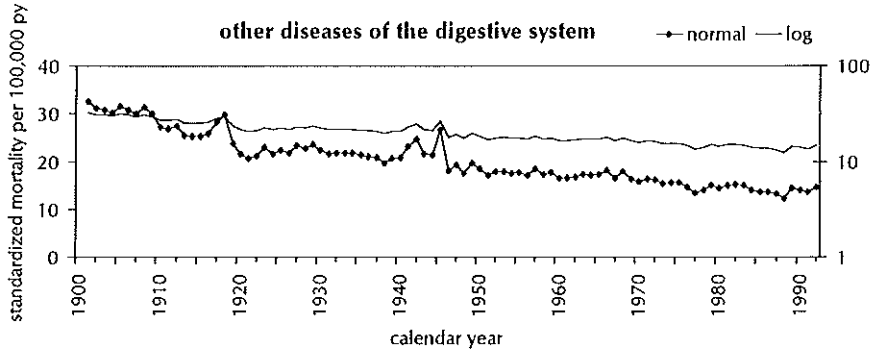

peritonitis
normal
log
standardized mortality per 100,000 py
12
8
4
0
100
10
1
0.1
1900
1910
1920
1930
1940
1950
1960
1970
1980
1990
calendar year

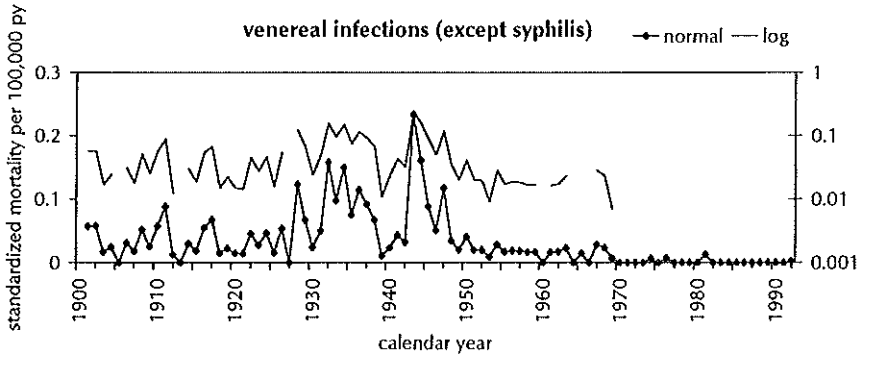

appendicitis
normal
log
standardized mortality per 100,000 py
6
4
2
0
10
1
0.1
1900
1910
1920
1930
1940
1950
1960
1970
1980
1990
calendar year

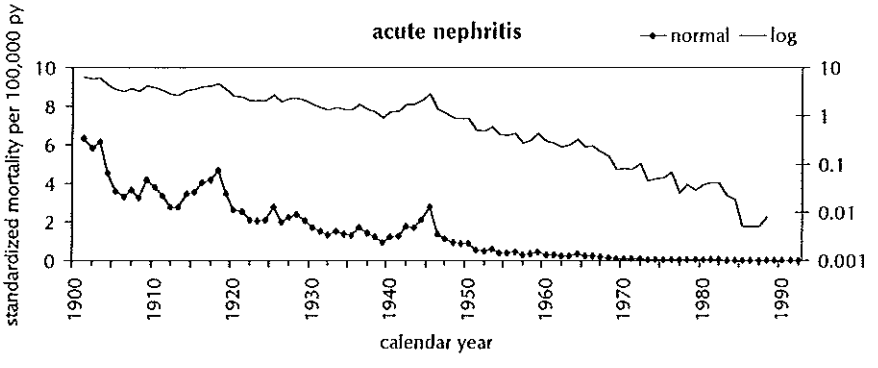

abdominal tuberculosis
normal
log
standardized mortality per 100,000 py
16
12
8
4
0
100
10
1
0.1
0.01
0.001
1900
1910
1920
1930
1940
1950
1960
1970
1980
1990
calendar year

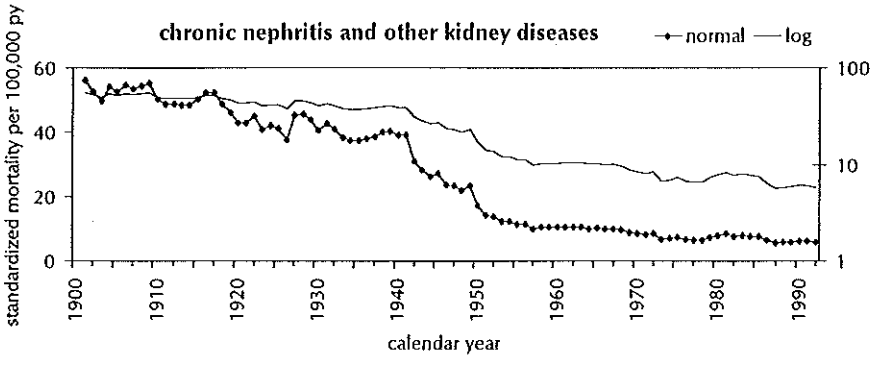

stomach ulcer
normal
log
standardized mortality per 100,000 py
10
8
6
4
2
0
10
1
1900
1910
1920
1930
1940
1950
1960
1970
1980
1990
calendar year

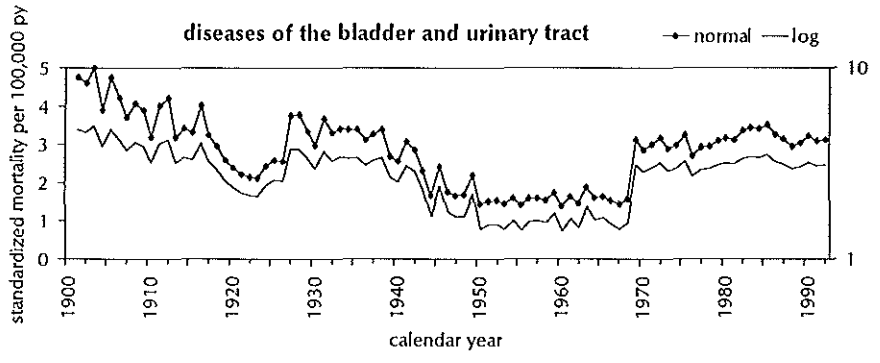

diseases of the oesophagus
normal
log
standardized mortality per 100,000 py
1
0.8
0.6
0.4
0.2
0
1
0.1
0.01
1900
1910
1920
1930
1940
1950
1960
1970
1980
1990
calendar year

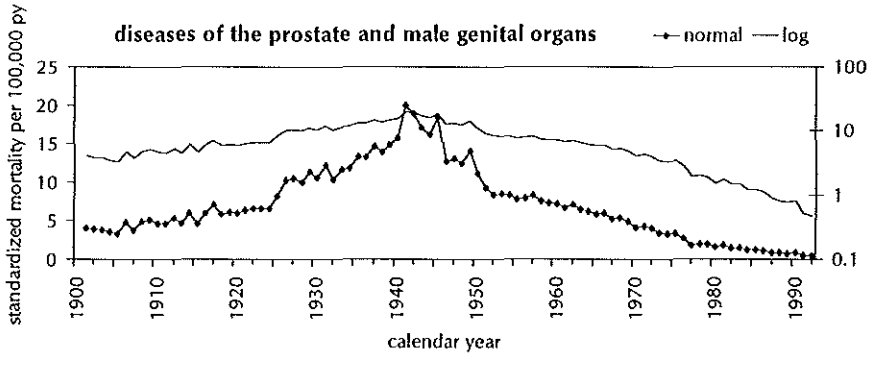

other diseases of the digestive system
normal
log
standardized mortality per 100,000 py
40
30
20
10
0
100
10
1
1900
1910
1920
1930
1940
1950
1960
1970
1980
1990
calendar year

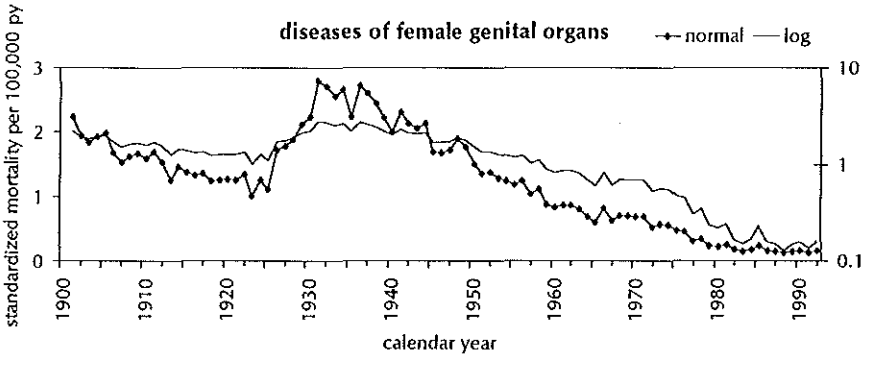

venereal infections (except syphilis)
normal
log
standardized mortality per 100,000 py
0.3
0.2
0.1
0
1
0.1
0.01
0.001
1900
1910
1920
1930
1940
1950
1960
1970
1980
1990
calendar year

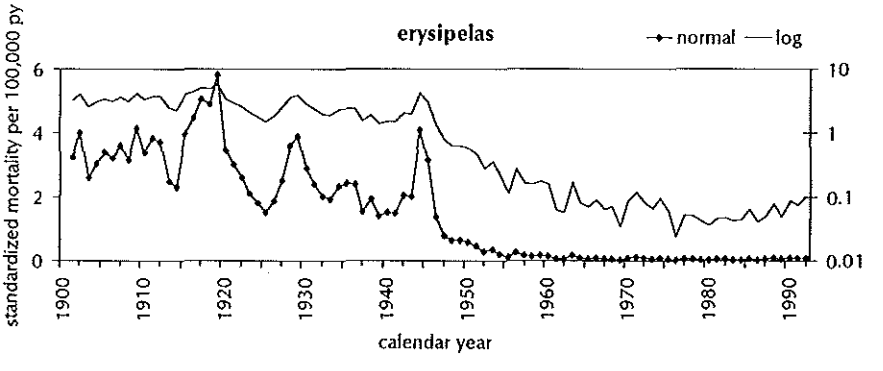

acute nephritis
normal
log
standardized mortality per 100,000 py
10
8
6
4
2
0
10
1
0.1
0.01
0.001
1900
1910
1920
1930
1940
1950
1960
1970
1980
1990
calendar year

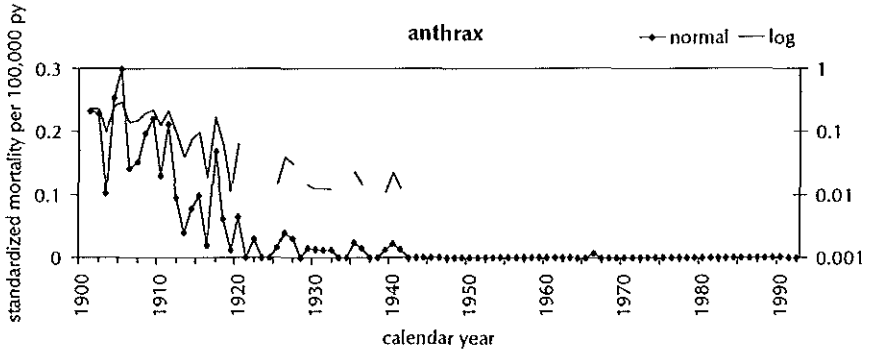

chronic nephritis and other kidney diseases
normal
log
standardized mortality per 100,000 py
60
40
20
0
100
10
1
1900
1910
1920
1930
1940
1950
1960
1970
1980
1990
calendar year

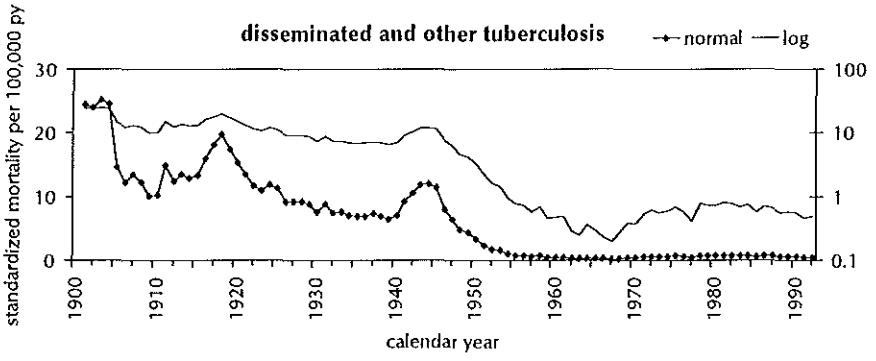

diseases of the bladder and urinary tract
normal
log
standardized mortality per 100,000 py
calendar year

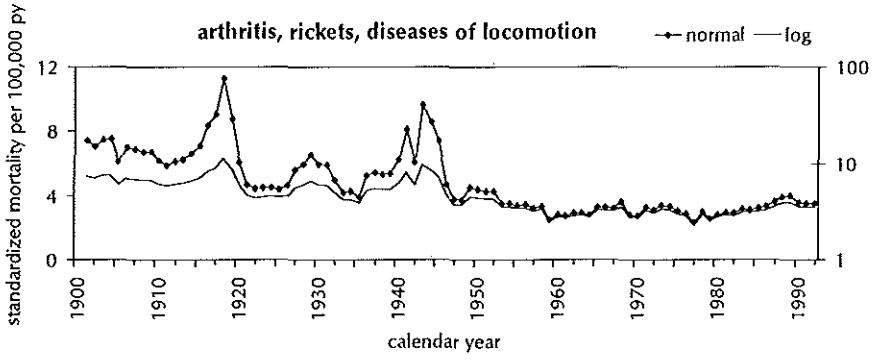

diseases of the prostate and male genital organs
normal
log
standardized mortality per 100,000 py
calendar year

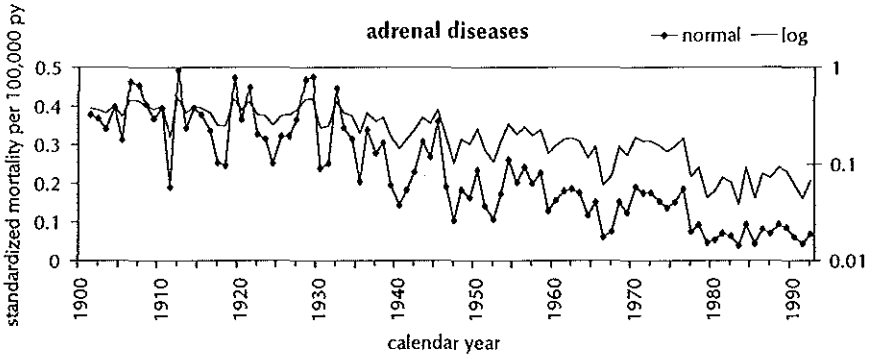

diseases of female genital organs
normal
log
standardized mortality per 100,000 py
calendar year

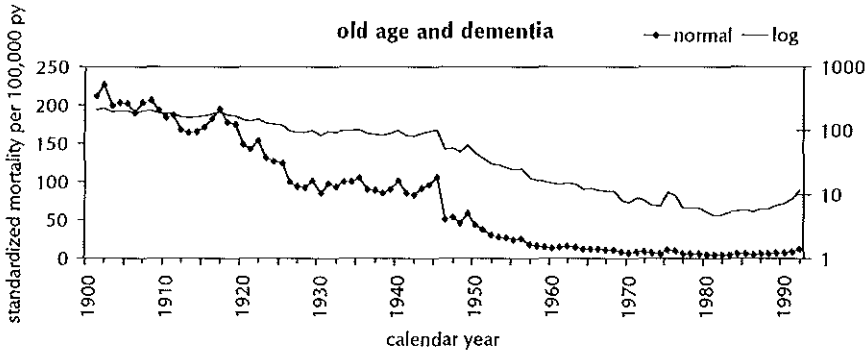

erysipelas
normal
log
standardized mortality per 100,000 py
calendar year

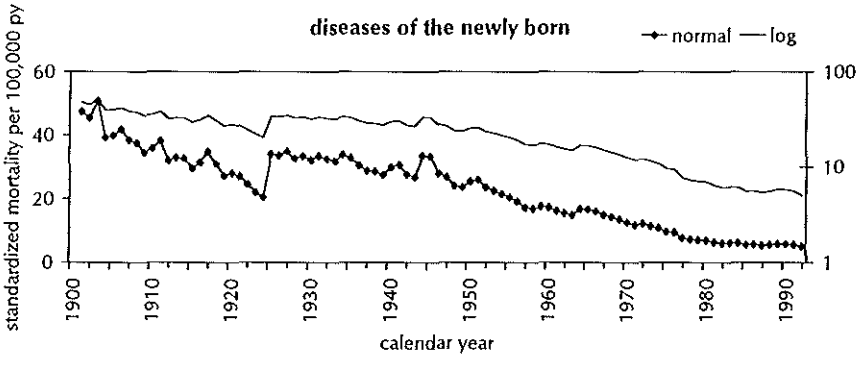

anthrax
normal
log
standardized mortality per 100,000 py
0.3
0.2
0.1
0
1
0.1
0.01
0.001
1900
1910
1920
1930
1940
1950
1960
1970
1980
1990
calendar year

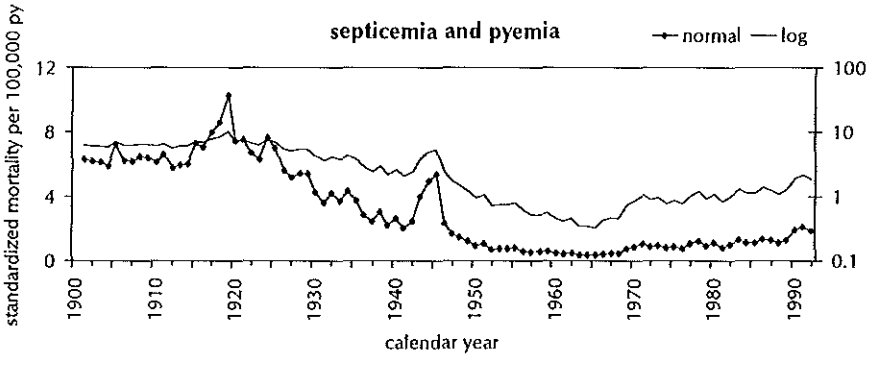

disseminated and other tuberculosis
normal
log
standardized mortality per 100,000 py
30
20
10
0
100
10
1
0.1
1900
1910
1920
1930
1940
1950
1960
1970
1980
1990
calendar year

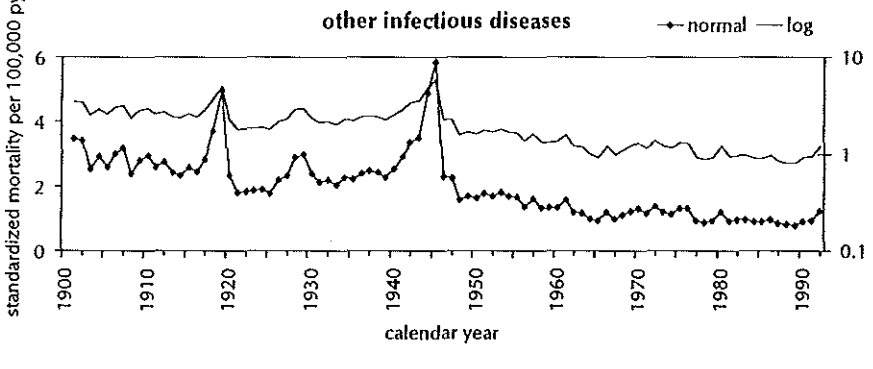

arthritis, rickets, diseases of locomotion
normal
log
standardized mortality per 100,000 py
12
8
4
0
100
10
1
1900
1910
1920
1930
1940
1950
1960
1970
1980
1990
calendar year

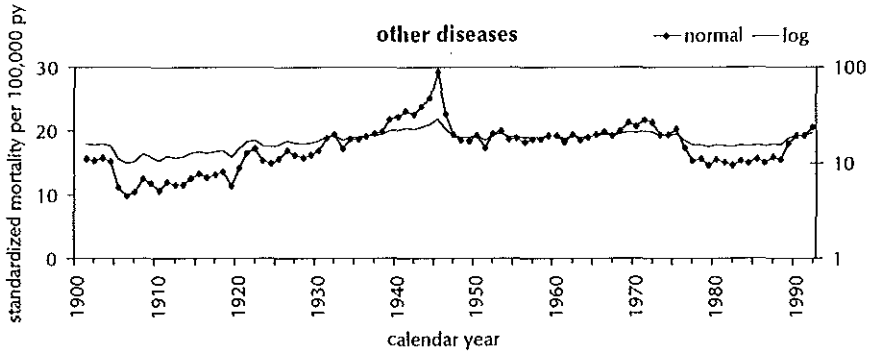

adrenal diseases
normal
log
standardized mortality per 100,000 py
0.5
0.4
0.3
0.2
0.1
0
1
0.1
0.01
1900
1910
1920
1930
1940
1950
1960
1970
1980
1990
calendar year

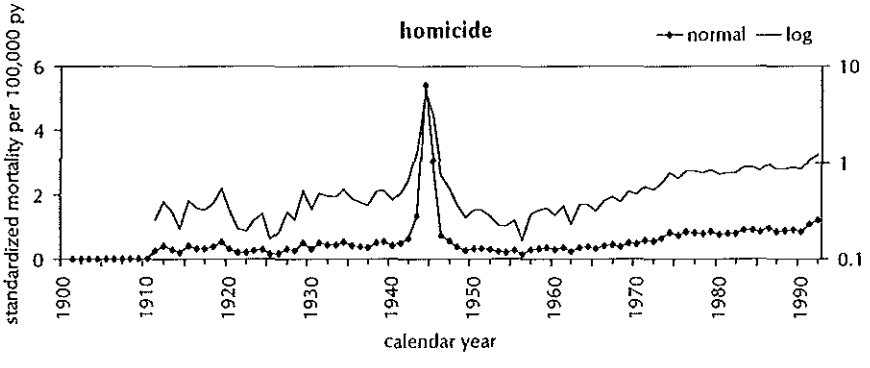

old age and dementia
normal
log
standardized mortality per 100,000 py
250
200
150
100
50
0
1000
100
10
1
1900
1910
1920
1930
1940
1950
1960
1970
1980
1990
calendar year

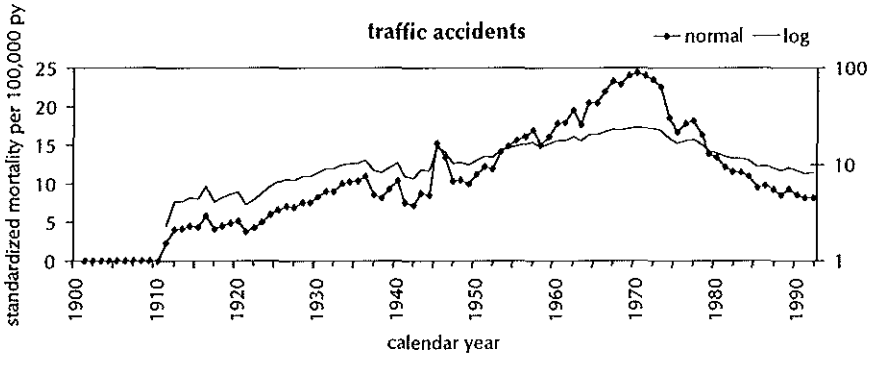

diseases of the newly born
normal
log
standardized mortality per 100,000 py
60
40
20
0
100
10
1
1900
1910
1920
1930
1940
1950
1960
1970
1980
1990
calendar year

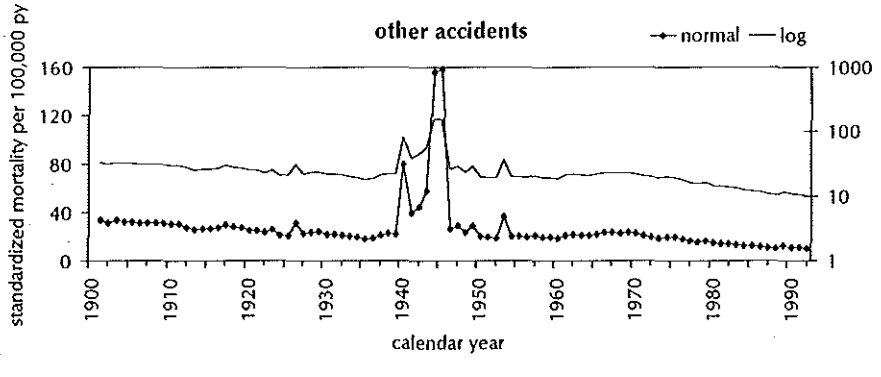

septicemia and pyemia
normal
log
standardized mortality per 100,000 py
12
8
4
0
100
10
1
0.1
1900
1910
1920
1930
1940
1950
1960
1970
1980
1990
calendar year


other infectious diseases
normal
log
standardized mortality per 100,000 py
6
4
2
0
10
1
0.1
1900
1910
1920
1930
1940
1950
1960
1970
1980
1990
calendar year


other diseases
normal
log
standardized mortality per 100,000 py
30
20
10
0
100
10
1
1900
1910
1920
1930
1940
1950
1960
1970
1980
1990
calendar year


homicide
normal
log
standardized mortality per 100,000 py
6
4
2
0
10
1
0.1
1900
1910
1920
1930
1940
1950
1960
1970
1980
1990
calendar year


traffic accidents
normal
log
standardized mortality per 100,000 py
25
20
15
10
5
0
100
10
1
1900
1910
1920
1930
1940
1950
1960
1970
1980
1990
calendar year

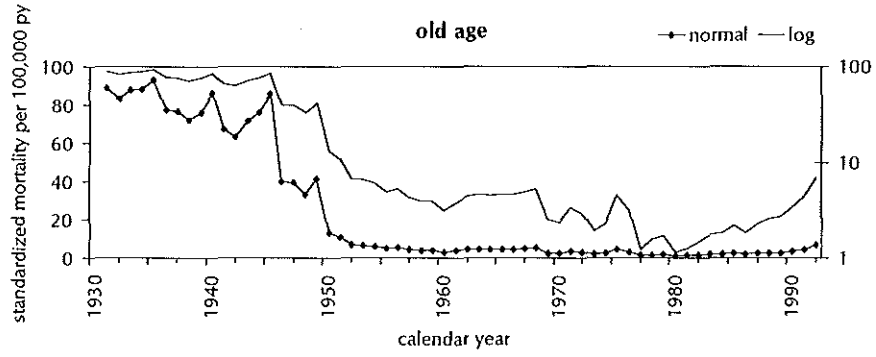

other accidents
normal
log
standardized mortality per 100,000 py
160
120
80
40
0
1000
100
10
1
1900
1910
1920
1930
1940
1950
1960
1970
1980
1990
calendar year

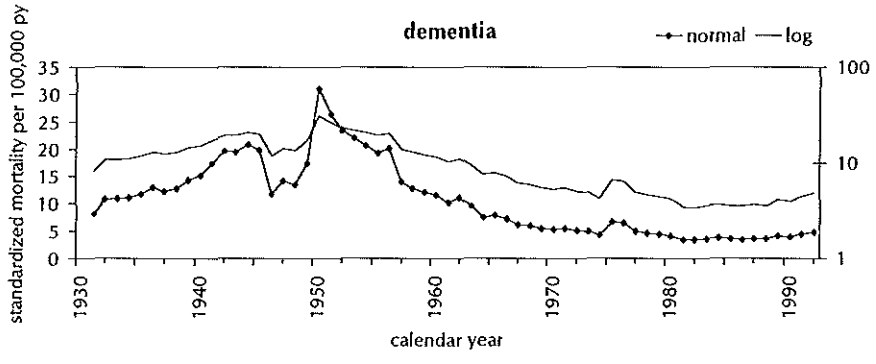
Appendix 2c

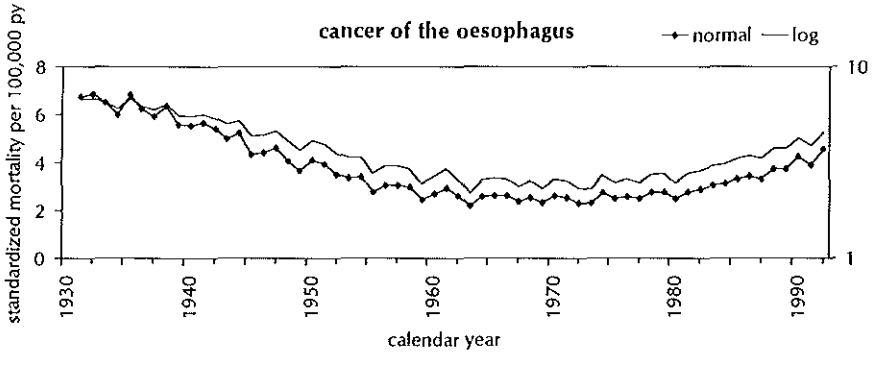
TRENDS OF CAUSES OF DEATH THAT COULD ONLY BE STUDIED FOR THE PERIOD 1931 TO 1992*

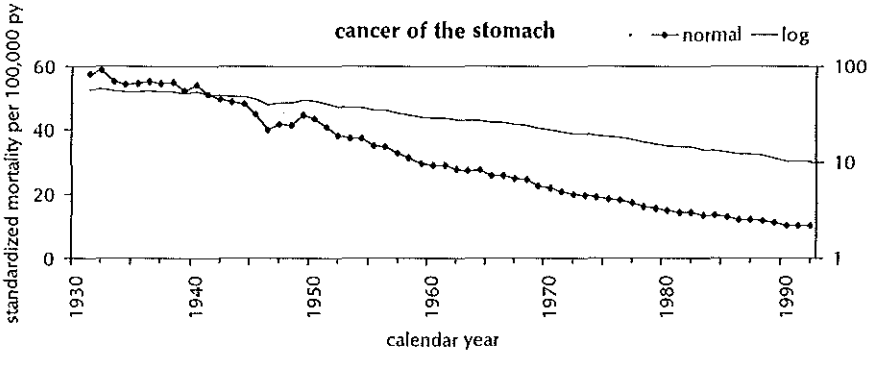
See appendix 1 for the corresponding ICD codes

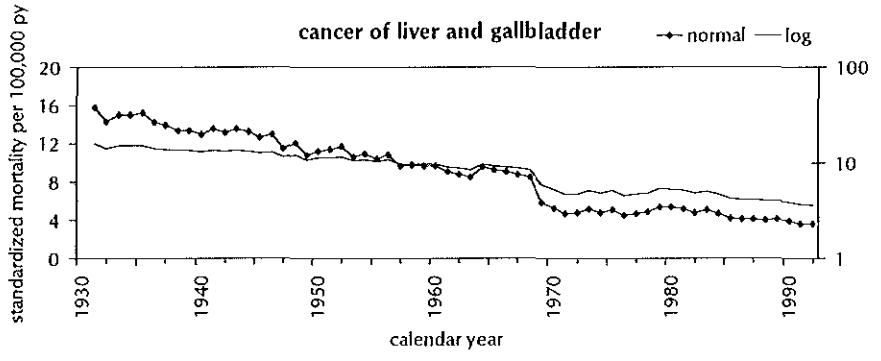
* Age- and sex-standardised mortality rates. Direct standardisation with the average population of 1901-1992 as the reference population.
Trends on the log-scale will be interrupted if the mortality rate is zero.

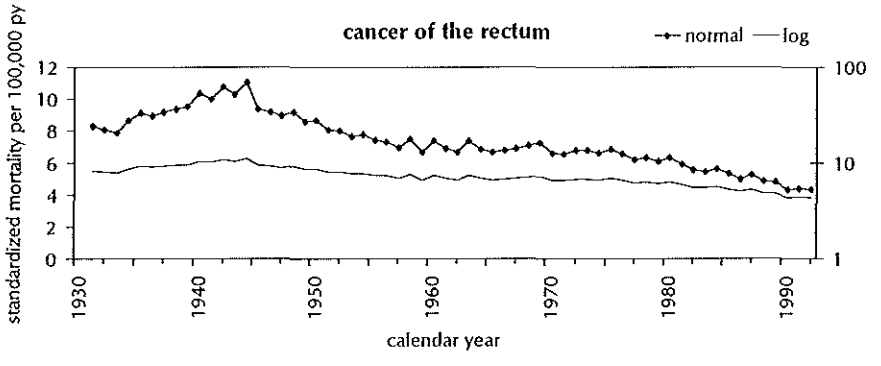

old age
normal
log
standardized mortality per 100,000 py
100
80
60
40
20
0
100
10
1
1930
1940
1950
1960
1970
1980
1990
calendar year

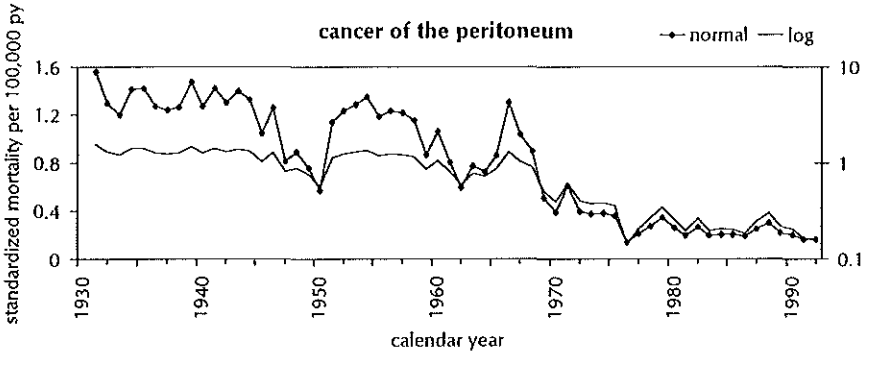

dementia
normal
log
standardized mortality per 100,000 py
35
30
25
20
15
10
5
0
100
10
1
1930
1940
1950
1960
1970
1980
1990
calendar year

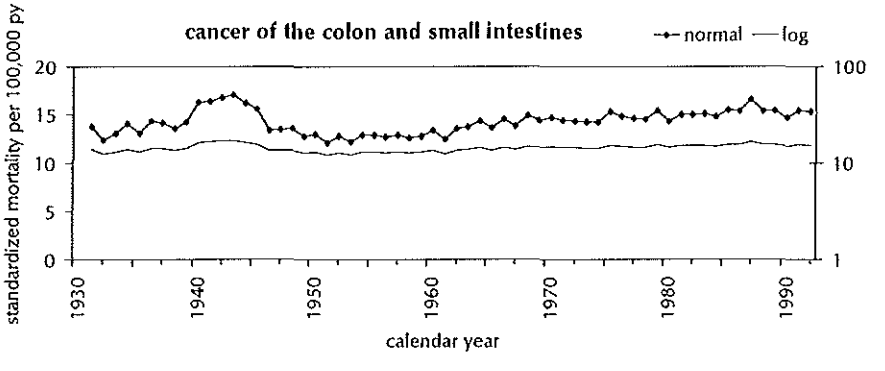

cancer of the oesophagus
normal
log
standardized mortality per 100,000 py
8
6
4
2
0
10
1
1930
1940
1950
1960
1970
1980
1990
calendar year

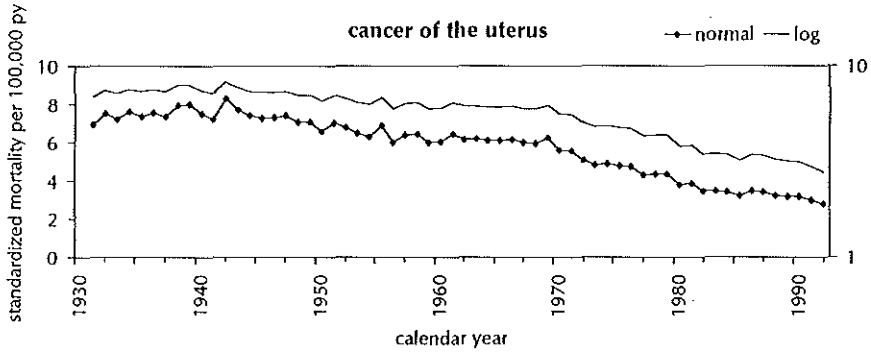

cancer of the stomach
normal
log
standardized mortality per 100,000 py
60
40
20
0
100
10
1
1930
1940
1950
1960
1970
1980
1990
calendar year

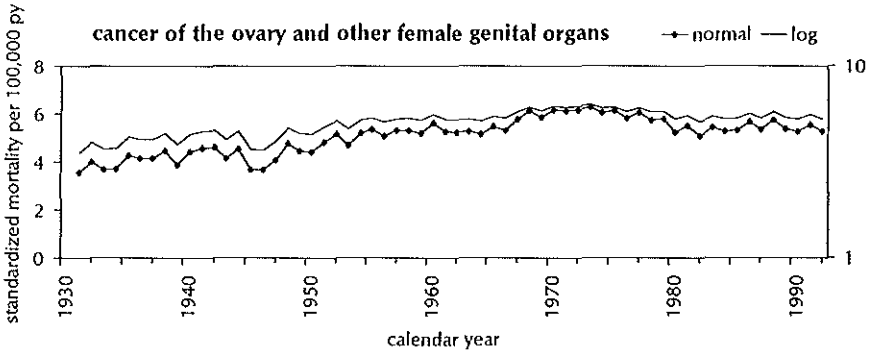

cancer of liver and gallbladder
normal
log
standardized mortality per 100,000 py
20
16
12
8
4
0
100
10
1
1930
1940
1950
1960
1970
1980
1990
calendar year

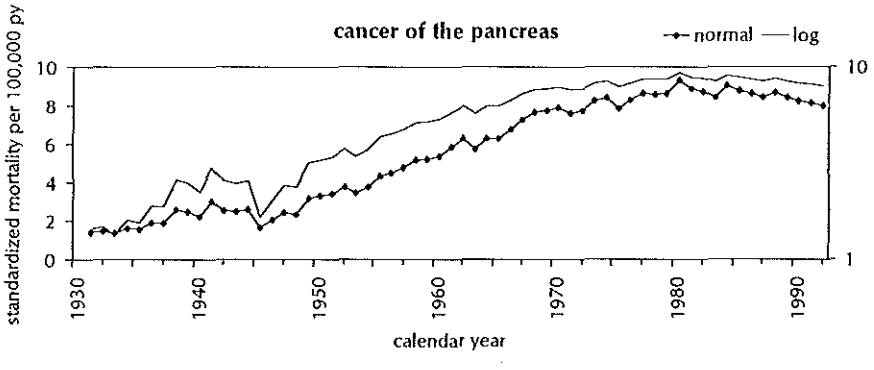

cancer of the rectum
normal
log
standardized mortality per 100,000 py
12
10
8
6
4
2
0
100
10
1
1930
1940
1950
1960
1970
1980
1990
calendar year

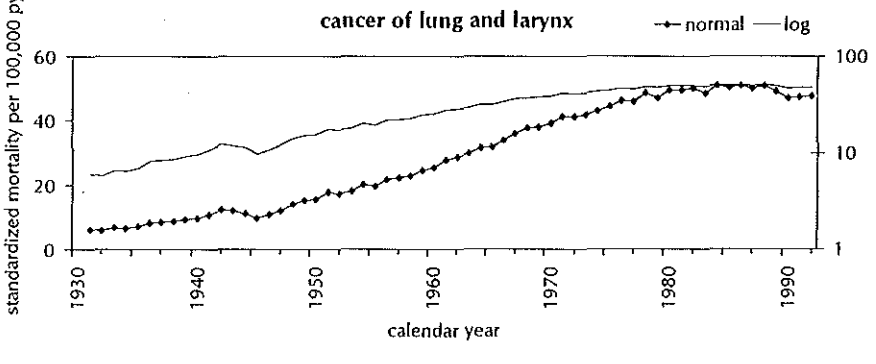

cancer of the peritoneum
normal
log
standardized mortality per 100,000 py
1.6
1.2
0.8
0.4
0
10
1
0.1
1930
1940
1950
1960
1970
1980
1990
calendar year

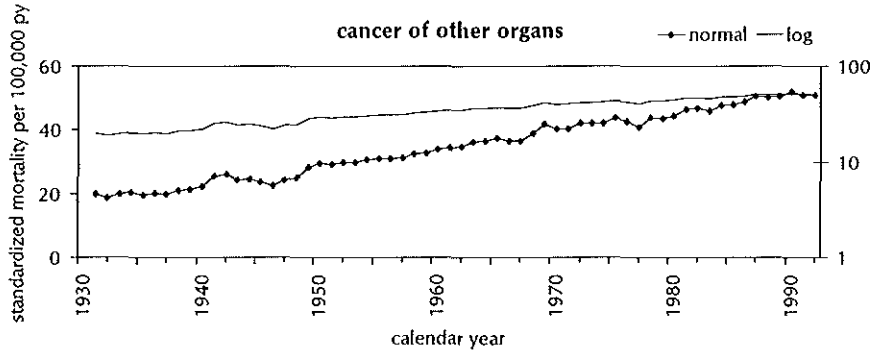

cancer of the colon and small intestines
normal
log
standardized mortality per 100,000 py
20
15
10
5
0
100
10
1
1930
1940
1950
1960
1970
1980
1990
calendar year

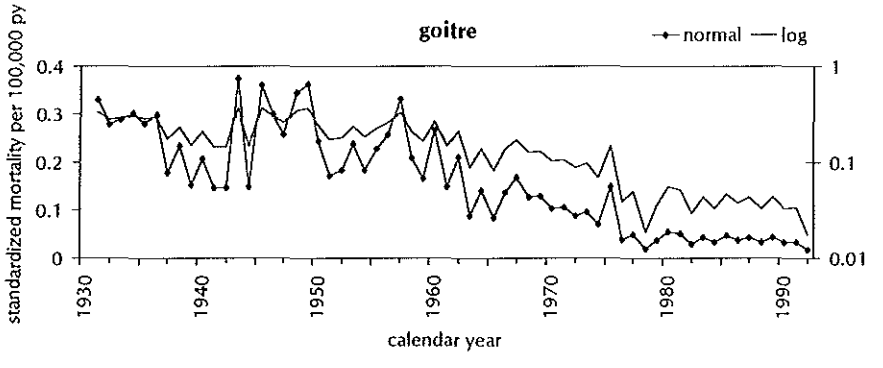

cancer of the uterus
normal
log
standardized mortality per 100,000 py
10
8
6
4
2
0
10
1
1930
1940
1950
1960
1970
1980
1990
calendar year

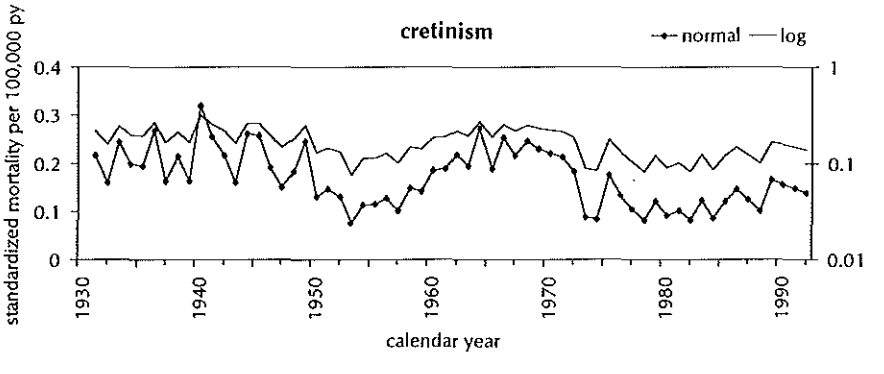

cancer of the ovary and other female genital organs
normal
log
standardized mortality per 100,000 py
8
6
4
2
0
10
1
1930
1940
1950
1960
1970
1980
1990
calendar year

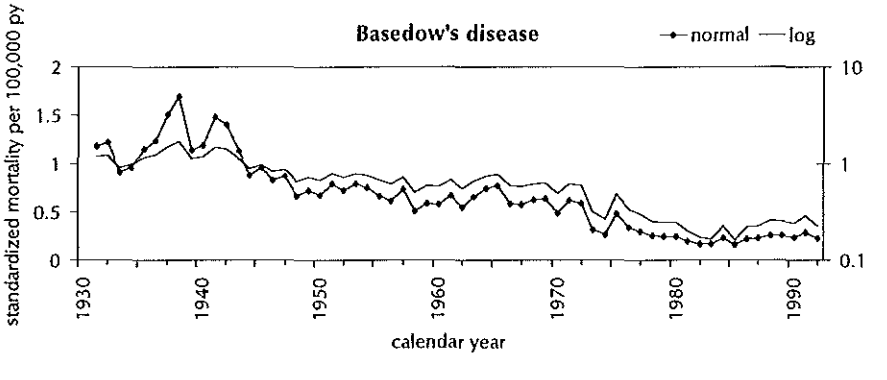

cancer of the pancreas
normal
log
standardized mortality per 100,000 py
10
8
6
4
2
0
10
1
1930
1940
1950
1960
1970
1980
1990
calendar year

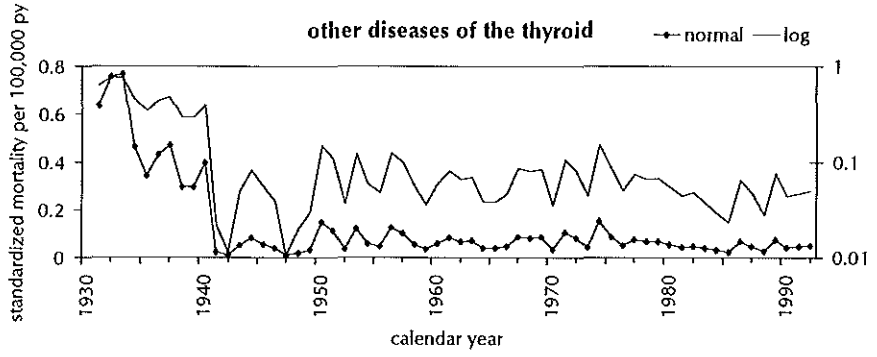

cancer of lung and larynx
normal
log
standardized mortality per 100,000 py
60
40
20
0
100
10
1
1930
1940
1950
1960
1970
1980
1990
calendar year

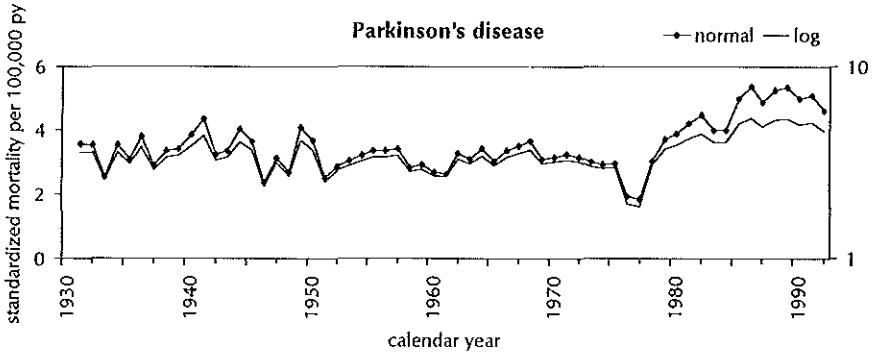

cancer of other organs
normal
log
standardized mortality per 100,000 py
60
40
20
0
100
10
1
1930
1940
1950
1960
1970
1980
1990
calendar year

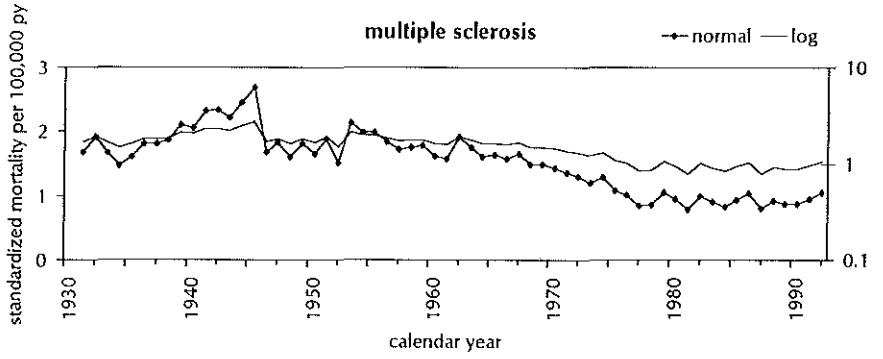

goitre
normal
log
standardized mortality per 100,000 py
0.4
0.3
0.2
0.1
0
1
0.1
0.01
1930
1940
1950
1960
1970
1980
1990
calendar year

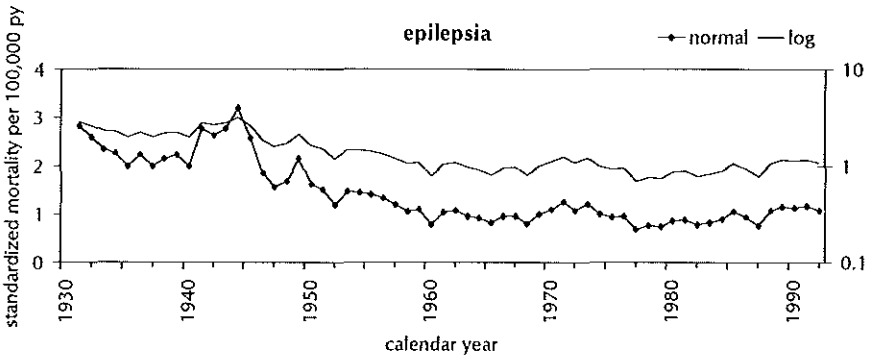

cretinism
normal
log
standardized mortality per 100,000 py
0.4
0.3
0.2
0.1
0
1
0.1
0.01
1930
1940
1950
1960
1970
1980
1990
calendar year

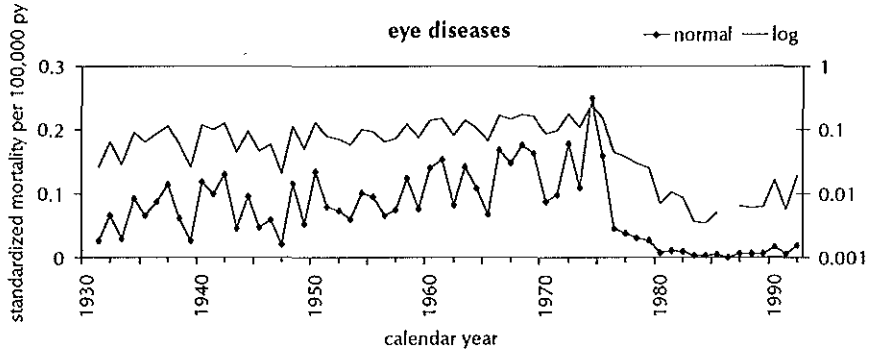

Basedow's disease
normal
log
standardized mortality per 100,000 py
2
1.5
1
0.5
0
10
1
0.1
1930
1940
1950
1960
1970
1980
1990
calendar year

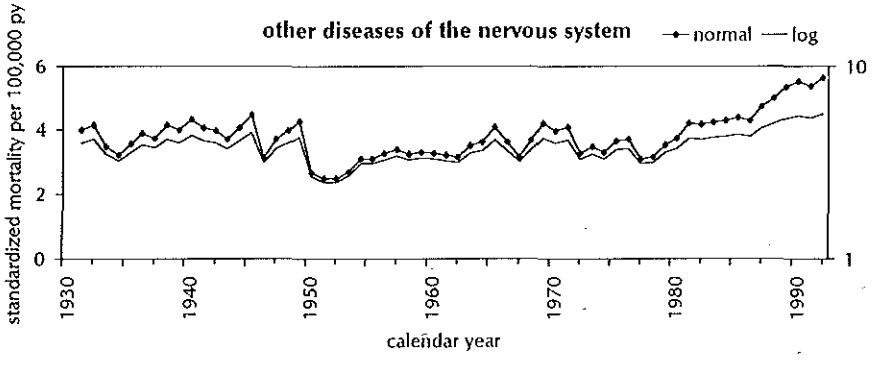

other diseases of the thyroid
normal
log
standardized mortality per 100,000 py
0.8
0.6
0.4
0.2
0
1
0.1
0.01
1930
1940
1950
1960
1970
1980
1990
calendar year

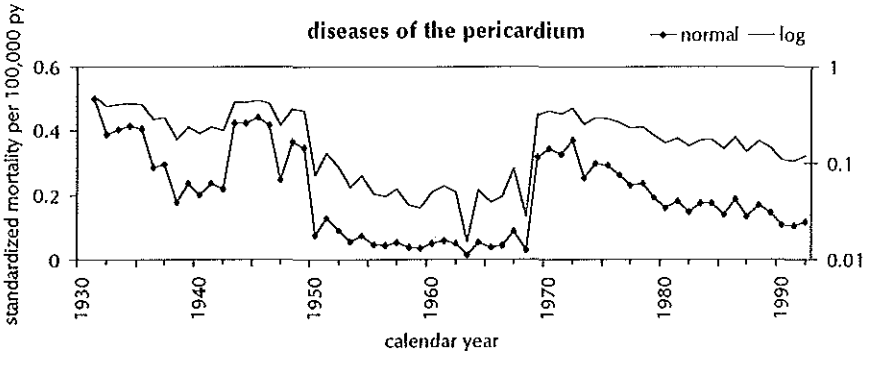

Parkinson's disease
normal
log
standardized mortality per 100,000 py
6
4
2
0
10
1
1930
1940
1950
1960
1970
1980
1990
calendar year

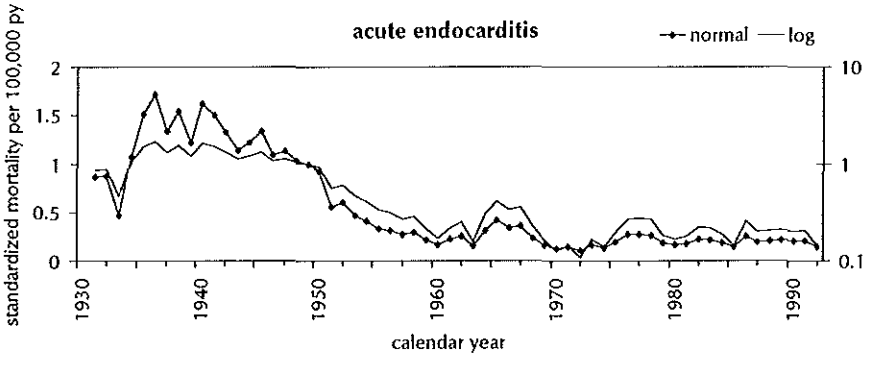

multiple sclerosis
normal
log
standardized mortality per 100,000 py
3
2
1
0
10
1
0.1
1930
1940
1950
1960
1970
1980
1990
calendar year

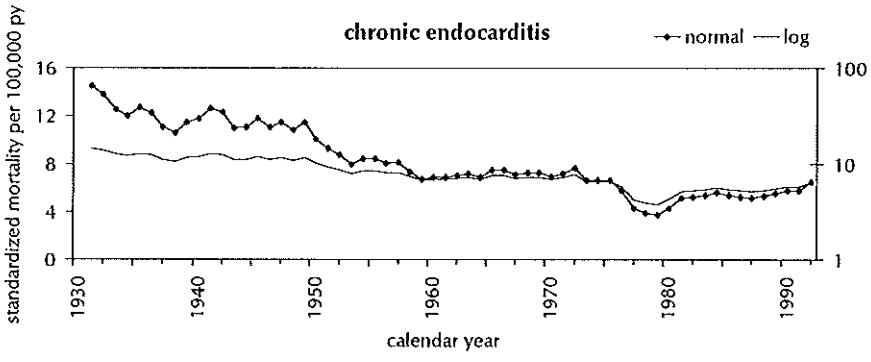

epilepsia
normal
log
standardized mortality per 100,000 py
4
3
2
1
0
10
1
0.1
1930
1940
1950
1960
1970
1980
1990
calendar year

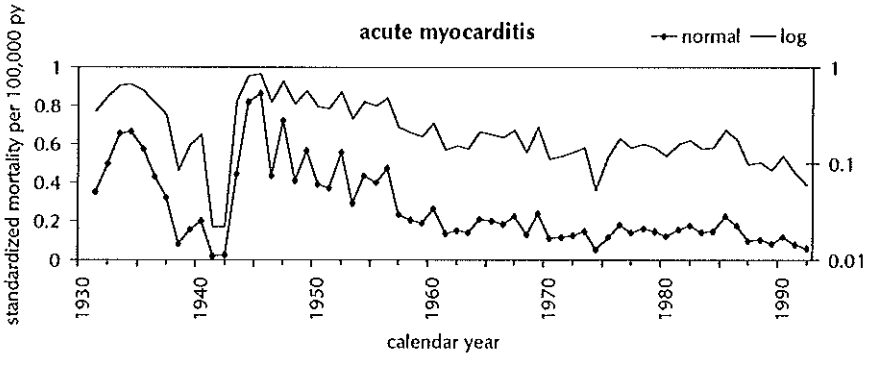

eye diseases
normal
log
standardized mortality per 100,000 py
0.3
0.2
0.1
0
1
0.1
0.01
0.001
1930
1940
1950
1960
1970
1980
1990
calendar year

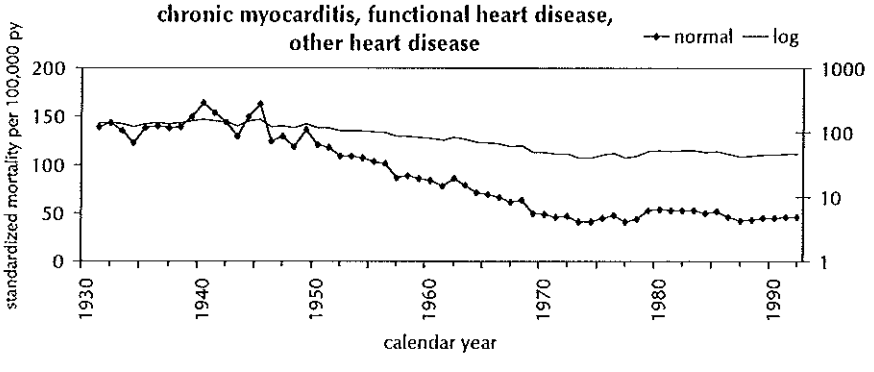

other diseases of the nervous system
normal
log
standardized mortality per 100,000 py
6
4
2
0
10
1
1930
1940
1950
1960
1970
1980
1990
calendar year

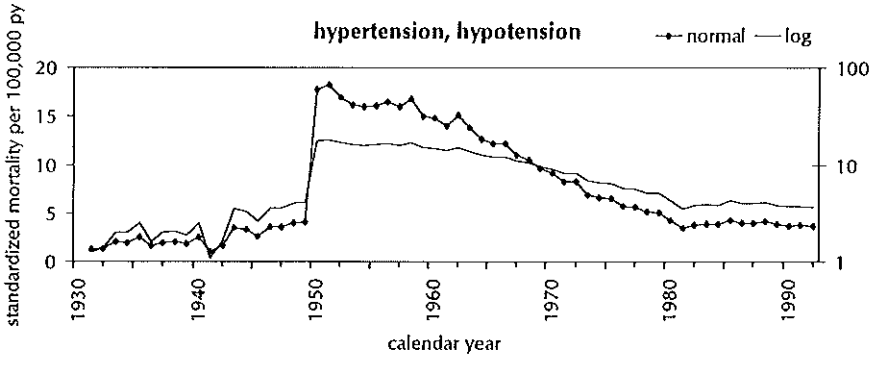

diseases of the pericardium
normal
log
standardized mortality per 100,000 py
0.6
0.4
0.2
0
1
0.1
0.01
1930
1940
1950
1960
1970
1980
1990
calendar year

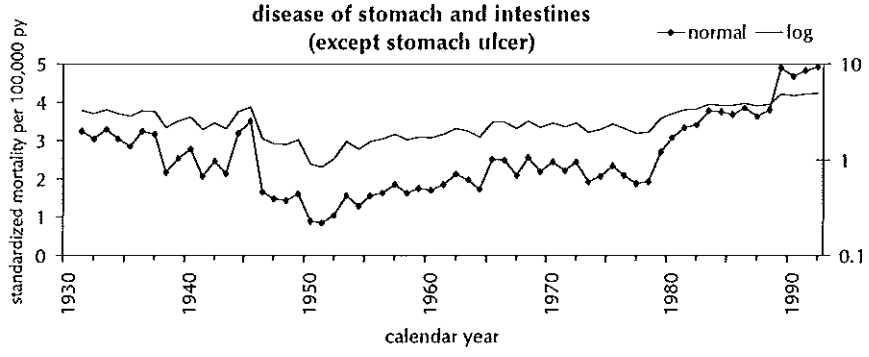

acute endocarditis
normal
log
standardized mortality per 100,000 py
2
1.5
1
0.5
0
10
1
0.1
1930
1940
1950
1960
1970
1980
1990
calendar year

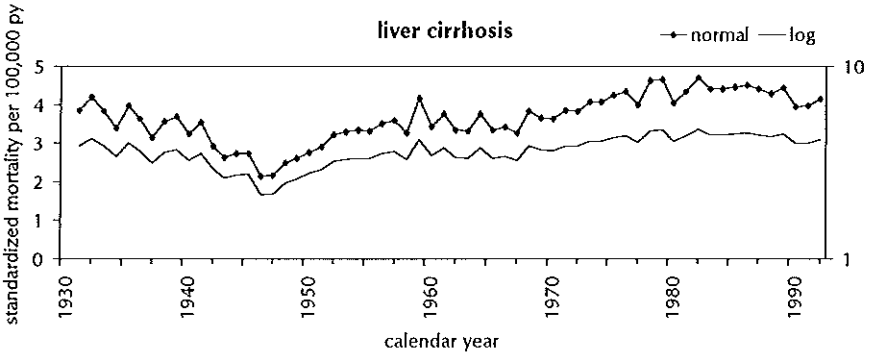

chronic endocarditis
normal
log
standardized mortality per 100,000 py
16
12
8
4
0
100
10
1
1930
1940
1950
1960
1970
1980
1990
calendar year

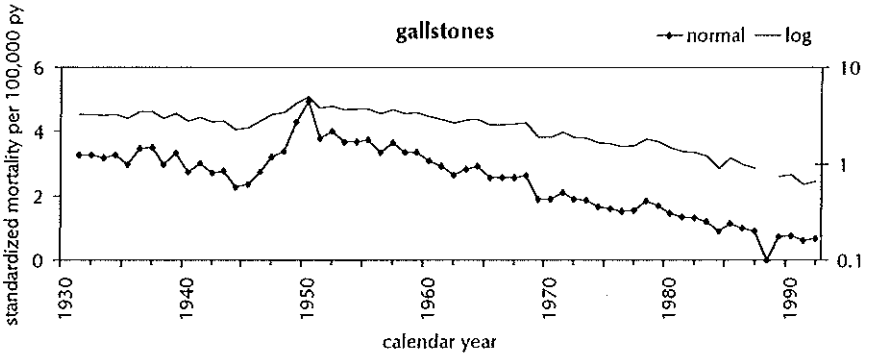

acute myocarditis
normal
log
standardized mortality per 100,000 py
1
0.8
0.6
0.4
0.2
0
1
0.1
0.01
1930
1940
1950
1960
1970
1980
1990
calendar year

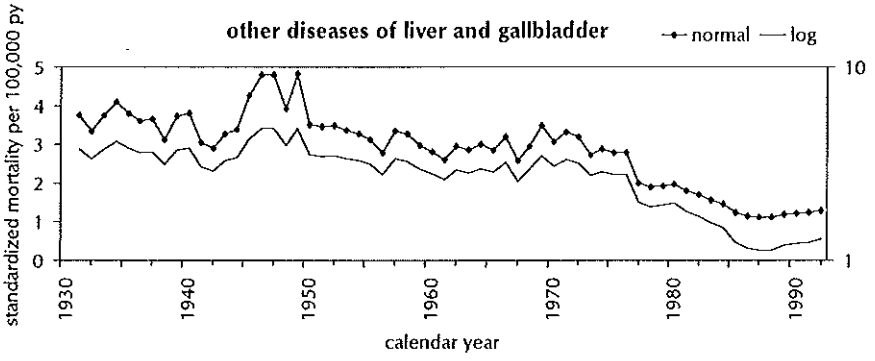

chronic myocarditis, functional heart disease,
other heart disease
normal
log
standardized mortality per 100,000 py
200
150
100
50
0
1000
100
10
1
1930
1940
1950
1960
1970
1980
1990
calendar year

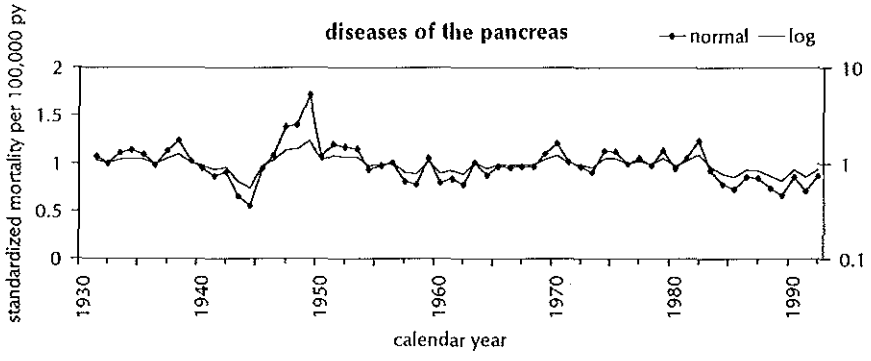

hypertension, hypotension
normal
log
standardized mortality per 100,000 py
20
15
10
5
0
100
10
1
1930
1940
1950
1960
1970
1980
1990
calendar year

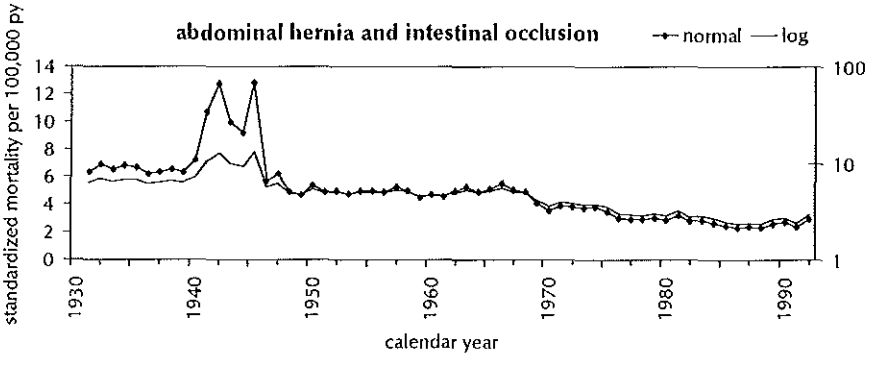

disease of stomach and intestines
(except stomach ulcer)
normal
log
standardized mortality per 100,000 py
0
1
2
3
4
5
10
1
0.1
1930
1940
1950
1960
1970
1980
1990
calendar year


liver cirrhosis
normal
log
standardized mortality per 100,000 py
0
1
2
3
4
5
10
1
1930
1940
1950
1960
1970
1980
1990
calendar year


gallstones
normal
log
standardized mortality per 100,000 py
0
2
4
6
10
1
0.1
1930
1940
1950
1960
1970
1980
1990
calendar year


other diseases of liver and gallbladder
normal
log
standardized mortality per 100,000 py
0
1
2
3
4
5
10
1
1930
1940
1950
1960
1970
1980
1990
calendar year


diseases of the pancreas
normal
log
standardized mortality per 100,000 py
2
1.5
1
0.5
0
10
1
0.1
1930
1940
1950
1960
1970
1980
1990
calendar year


abdominal hernia and intestinal occlusion
normal
log
standardized mortality per 100,000 py
14
12
10
8
6
4
2
0
100
10
1
1930
1940
1950
1960
1970
1980
1990
calendar year

Appendix 3

TWO AGGREGATION LEVELS OF CAUSES OF DEATH:
27 causes for the period 1875-1992 and 65 causes for the period 1901-1992. ICD-9 codes indicate which causes of death are included in the different groups. Infectious disease categories have been marked with an asterisk.

27-Causes (1875-1992)	65-Causes (1901-1992)	ICD-9 codes
1. Congenital anomalies	1. Congenital anomalies	740-759
2. Cancer	2. Cancer of the oesophagus, stomach, liver and gallbladder	150, 151, 155-156
	3. Cancer of the small intestines, large intestines, rectum and peritoneum	152-154, 158
	4. Cancer of the skin	172-173
	5. Cancer of the breast	174-175
	6. Cancer of other organs (including lung)	142, 157, 159-165, 170-171, 179-185, 200, 202-203
3. Scurvy	7. Scurvy	267
4.* Typhus. Typhoid fever	8.* Typhus. Typhoid fever	002
5.* Malaria (including: Intermittent fever. Pernicious fever)	9.* Malaria	084
6.* Smallpox	10.* Smallpox	050
7.* Scarlet fever	11.* Scarlet fever	034
8.* Measles	12.* Measles	055
9. Cerebrovascular disease	13. Cerebrovascular disease	430-438
10.* Brain diseases etc. (Including: Insanity. Syphilis. Convulsions. Diseases of the spinal cord. Paralysis. Trismus. Epilepsy)	14.* Tuberculosis of meninges and central nervous system	013
	15.* Syphilis	090-097
	16. Hereditary and familial diseases of nervous system. Other diseases of central nervous system. Diseases of nerves and peripheral ganglia. Diseases of (para)thyroid gland. Diseases of the eye	076, 240-246, 252330-337, 340-379
	17. Alcoholic psychosis. Alcoholism	291, 303
	18.* Encephalitis. Meningitis	036, 046-049, 062-064, 320-326
	19.* Convulsions	-
	20.* Poliomyelitis	045, 138
	21.* Diseases of the ear	380-389
11.* Respiratory tuberculosis	22.* Respiratory tuberculosis	010-012
12. Diabetes	23. Diabetes mellitus	250
13.* Diphtheria. Croup	24.* Diphtheria	032

27-Causes (1875-1992)	65-Causes (1901-1992)	ICD-9 codes
14.* Whooping cough	25.* Whooping cough	033
15.* Acute respiratory diseases	26.* Acute bronchitis. Influenza	466, 487
	27.* Pneumonia	480-486
	28.* Empyema. Pleurisy	510-511
16. Chronic respiratory diseases	29. Diseases of larynx, pharynx, nasal cavity, oral cavity	460-465, 470, 472-476, 478, 520-529
	30. Chronic bronchitis. Emphysema. Asthma. Other diseases of the respiratory system	415, 477, 490-496, 500-508, 512-519
17. Diseases of circulatory system.	31.* Rheumatic fever. Chorea	390-392
	32. Chronic rheumatic heart disease. Hypertensive disease. Other forms of heart disease (exc. ischemic heart disease)	393-398, 401-405, 416-417, 420-429, 458-459
	33. Diseases of veins. Gangrene	445, 451-456
	34. Angina pectoris. Ischemic heart disease	410-414
18.* Dysentery. Acute diseases of digestive system. Diarrhoeal diseases	35.* Diarrhoeal disease, dysentery, enteritis	004, 006-009, 532, 555-558, 562
	36.* Peritonitis	567
	37.* Appendicitis	540-543
19.* Cholera(Including: Asiatic cholera. Cholera nostras)	38.* Cholera	001
20. Chronic diseases of digestive system	39.* Tuberculosis of intestines, peritoneum and mesenteric glands	014
	40. Stomach ulcer	531, 533-534
	41. Other diseases of digestive system	535-537, 550-553, 560, 564-566, 568-569, 570-579
	42. Diseases of oesophagus	530
21. Diseases of the genito-urinary system	43.* Venereal disease (exc. Syphilis)	098-099
	44.* Acute nephritis	580
	45. Chronic nephritis. Other diseases of kidney	581-591, 593
	46. Diseases of bladder, urethra and other diseases of urinary tract	592, 594-599

27-Causes (1875-1992)	65-Causes (1901-1992)	ICD-9 codes
	47. Diseases of prostate. Other diseases of male genital organs	600-608
	48. Diseases of female genital organs	218-221, 610-611, 614-629
22. Puerperal diseases	49. Complications of pregnancy, childbirth and the puerperium (exc. puerperal fever)	630-639, 640-648, 650-669, 671-676
23.* Puerperal fever	50.* Puerperal fever	670
24. Other diseases(Including: Debility. Several types of tuberculosis. Scrofula. Rickets. Diseases of skin. Abscess. Ulcer. Gangrene. Pyemia. Hemorrhage. Continuous fever)	51.* Erysipelas	035
	52.* Anthrax	022
	53.* Disseminated tuberculosis and tuberculosis of other organs	015-018, 137
	54. Rheumatism. Arthritis. Rickets. Diseases of locomotion	268, 274, 710-739
	55. Diseases of adrenal glands	255
	56. Senility. Dementia	290, 797
	57. Perinatal causes of death	760-779
	58.* Septicaemia. Pyaemia	038
	59.* Other infectious diseases	003, 020-021, 023-027, 030-031, 037, 039-044, 051-054, 056-057, 060-061, 065-066, 070-075, 077-083, 085-088, 100-104, 110-118, 120-136, 139, 681-682
	60. Other diseases	201, 204-208, 210-217, 222-239, 251, 253-254, 256-266, 269-273, 275-289, 292-302, 306-319, 471, 680, 683-686, 690-698, 700-709
25. Violence	61. Homicide	E960-969
	62. Traffic accidents	E800-807, E810-829
	63. Other external causes of death	005, 304-305, E830-838, E840-848, E850-876, E878-888, E890-903, E905-949, E970-978, E980-999

27-Causes (1875-1992)	65-Causes (1901-1992)	ICD-9 codes
26. Suicide	64. Suicide	E950-959
27. Unknown and ill-defined causes of death. Sudden death	65. Unknown and ill-defined causes of death. Sudden death	780-796, 798-799, E904

DANKWOORD

DE AFGELOPEN TIJD zijn al heel wat promovendi uit het cluster Medische en Maatschappelijke Determinanten van Volksgezondheid (MMDV) van het instituut Maatschappelijke Gezondheidszorg (iMGZ) mij voorgegaan met een promotie. Er zijn dus ook al vele dankwoorden aan mijn promotor, Johan Mackenbach, gericht. Een kleine analyse van die dankwoorden laat zien dat de volgende eigenschappen hem veelvuldig worden toegedicht: gestructureerd, kritisch, inspirerend, onheldere redeneringen feilloos detecterend, stukken snel lezend. Ik onderschrijf deze kwalificaties van harte. Ik ben blij dat ik de achterliggende, belangrijke jaren van mijn wetenschappelijke carrière onder de leiding van Johan Mackenbach heb kunnen doorbrengen en dat onze samenwerking in een nieuw project voortgang kon vinden.

Naast Johan Mackenbach is Frans van Poppel van het Nederlands Interdisciplinair Demografisch Instituut (NIDI) nauw bij het onderzoek betrokken geweest. Zijn kennis van de historische demografie is werkelijk fenomenaal. Hij heeft mij geïntroduceerd op de congressen van de Social Science and History Association, wat een goed platform was om mijn onderzoek te presenteren. Op het NIDI was er ook van anderen altijd een grote hulpvaardigheid. Een tweetal personen wil ik met name noemen: Ewa Tabeau, die betrokken is geweest bij de analyses in hoofdstuk 5 en Peter Ekamper, die de kaartjes voor de hoofdstukken 8 en 9 heeft gemaakt.

De analyses die in dit proefschrift gepresenteerd zijn hadden niet gedaan kunnen worden en de plaatjes van de doodsoorzaakspecifieke sterftetrends in appendix 2 hadden niet gemaakt kunnen worden zonder het invoeren van stapels CBS-publikaties. Deze enorme klus werd voornamelijk gedaan door Saskia Drent en Aty Slikkerveer. Het is voor een groot deel aan hen te danken dat er nu voor Nederland een mooi bestand ligt met sterfte naar doodsoorzaak (op 3 cijfer niveau) vanaf 1875.

Anderen die op enige wijze hebben bijgedragen aan het welslagen van dit proefschrift zijn Caspar Looman die mij heeft bijgestaan bij de statistische analyses, Else van den Engel die zorgde voor secretariële ondersteuning, Ingrid Matser die ervoor heeft gezorgd dat het Engels in dit proefschrift gefatsoeneerd werd, Anna Bosselaar die de opmaak van het binnenwerk van dit proefschrift heeft verzorgd en Arjan Schoonhoven die het omslag ontworpen heeft.

Mijn paranimfen en kamergenoten op iMGZ, Inez Joung en Wilma Nusselder, zijn mij voorgegaan op het promotiepad en konden mij daarom zeer goed ondersteunen op het hele traject tot dit proefschrift en zeker bij het dragen van de laatste loodjes. Ik bewonder beiden als wetenschappers en beschouw hen als goede vrienden. Zij hebben een groot aandeel in het feit dat ik al die jaren met veel plezier aan dit proefschrift heb gewerkt.

Mijn ouders bedank ik voor het feit dat ze hun kinderen altijd gestimuleerd hebben om hun talenten te ontplooien. Dat heeft er zeker toe bijgedragen dat ik dit proefschrift voltooid heb.

Ten slotte Rob. Bedankt voor alles! Voor de weekenden en avonden die je zelf moest werken, waardoor het voor mij makkelijker was om ook buiten kantoortijden aan het proefschrift te schrijven. Ook bedankt voor alle ongevraagde adviezen. Ik weet dat ze goed bedoeld waren. Dat je bij die adviezen een proefschrift regelmatig vergeleek met een afstudeerscriptie zij je vergeven.

CURRICULUM VITAE

Judith van den Bosch werd geboren op 21 mei 1969 te Elspeet (gemeente Ermelo). Haar VWO-diploma behaalde zij in 1987 aan het Van Lodenstein-college te Amersfoort. Aansluitend ging zij Gezondheidswetenschappen (later Bio-medische Wetenschappen geheten) studeren aan de Rijksuniversiteit Leiden. Zij studeerde af op een epidemiologische studie: een onderzoek naar de incidentie van bottumoren in Nederland. De doctoraalbul ontving zij in 1992. In datzelfde jaar kwam zij in dienst van het instituut Maatschappelijke Gezondheidszorg (iMGZ) van de Erasmus Universiteit Rotterdam. In opdracht van de Hersenstichting Nederland werd een rapport geschreven over het vóórkomen van ziekten van het zenuwstelsel in Nederland. Dit project werd uitgevoerd in samenwerking met het Centraal Bureau voor de Statistiek, waar zij ten tijde van dat project gedetacheerd was. In 1993 werd zij geregistreerd als epidemioloog. In dat jaar werd ook begonnen met een onderzoek naar de epidemiologische transitie in Nederland, waarvan de resultaten in dit proefschrift zijn beschreven. In de eerste jaren van dat onderzoek heeft zij de MSc-opleiding epidemiologie van het Netherlands Institute of Health Sciences (NIHES) afgerond. Sinds januari 1998 doet zij onderzoek naar de mogelijkheid en wenselijkheid van effectievere preventie van perinatale sterfte in Nederland. Dit onderzoek wordt eveneens uitgevoerd op het iMGZ. Judith van den Bosch is gehuwd met Rob Wolleswinkel.